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Studying Evolution in Action: Foundations for a Transgenerational Comparative Psychology

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The gene centered framework of the modern evolutionary synthesis serves to constrain the contributions to evolutionary knowledge that can be gained from comparative studies of animal development. Contrary to this position, a case is made that understanding the dynamics of ontogenetic processes across generations can illuminate processes of evolution. Examples are provided that show how alterations of developmental contexts in one generation influence patterns of development in subsequent generations. The conceptual foundations and implications of a transgenerational orientation to studying animal development are discussed. By adopting a transgenerational approach, comparative psychologists can study evolutionary processes in action and thus play a more prominent role in discussions of evolution.

> *The many inclusive attempts to determine just how species arose led naturally to a renewed study of the processes by which individuals came into existence, for it seems probable that the principles and causes of the development of individuals will be found to apply also to the evolution of races. . . .indeed there is probably no other subject of such vast importance to mankind as the knowledge of and control over heredity and development* (E. G. Conklin, 1915/1965, p. v).

In its broadest sense, evolution refers to the natural process(es) by which organisms come to differ (morphologically, physiologically, and/or behaviorally) from their ancestors. Though no scientist would dispute the reality of evolution, exactly *how* these transformations take place has been a matter of debate since the 19th century.

Like others working in the life sciences, comparative psychologists have strived to incorporate evolutionary ideas in their pursuits. In these efforts, evolution can be treated either as an historical, completed process or as one that is present and ongoing (Conklin, 1919). For the most part, comparative psychologists have tended to view evolution in the former sense. It is not uncommon, for instance, to ask how behavior has evolved or how aspects of individuals reflect the evolution of that species. In addressing these questions, comparative psychologists have benefited from using phylogenetic data to make sense of similarities and differences between and within species, and such behavioral comparisons can potentially clarify phylogenetic details. Treating evolution as a past process is also found in the growing trend to explain behavior patterns of extant members of our species by appealing to selection pressures operating during the emergence of our species (e.g., Gaulin & McBurney, 2001).

That psychologists have chosen to view evolution in its historical (rather than present) sense is not surprising given the assumptions of the prevailing paradigm of evolution today, the modern synthesis. The picture of life painted in the

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modern synthesis does not leave much room for input from psychologists due in large part to several deeply entrenched assumptions regarding heredity, individual development, and the mechanisms of evolutionary change. Each of these aspects of life are discussed and explained in terms of genes. Indeed, the modern synthesis is more a theory about genes than it is a theory about the generation and evolution of phenotypes (Ho & Saunders, 1979). This genocentrism leads us to view the characteristics of the individual as *effects* of evolution that get realized during ontogeny through internal, hereditary programs (genetic, somatic, neural) that were shaped by natural selection (see Mayr, 1988; Roe & Simpson, 1967). Accordingly, ontogenetic studies, a central concern and mainstay in comparative psychology (Greenberg & Haraway, 2002; Papini, 2002), can only "throw a good deal of light on the processes which *in the past* were responsible for phylogenetic changes" (Maynard-Smith, 2000, p. 310, emphasis added).

Many comparative psychologists, however, are aware that evolutionary thinking is undergoing a modern renaissance of sorts wherein an individual's behavior, development, and experiences take center stage and are allowed to play a leading role in evolutionary change (Bateson, 1988; Gottlieb, 1987; Ho, 1998; Johnston & Gottlieb, 1990; Laland, Odling-Smee, & Feldman, 1999; Oyama, Griffiths, & Gray, 2000; Piaget, 1979; Plotkin, 1988; Stamps, 2003). Because explanations of behavior are the central focus of comparative psychology, and developmental analyses are an accepted component of the latter, it stands to reason that comparative psychologists are well suited to make significant contributions to how the future of evolutionary thinking will proceed.

This essay is not intended to undermine traditional evolutionary approaches in comparative psychology. These pursuits will continue to be important facets of our field. Rather, in the spirit of pluralism, this essay presents an additional way that evolution can be incorporated into comparative psychology, one in which evolution is treated not as an historical, completed process, but rather as an ongoing, present one.

I will argue that the inclusion of a *transgenerational* approach to the study of behavior, in which the development of one generation is studied in relation to the experiences of previous generations, can allow for direct studies of evolutionary mechanisms. Although this line of inquiry was discouraged for much of the 20th century, based on the weight of modern evidence it is no longer inappropriate to pose such questions. In what follows, I provide the conceptual justification and explore the implications of a trangenerational orientation to studying individual development, as well as suggest some general methodological guidelines.

Modern Synthesis Limits Comparative Psychology

Evolutionary theory must explain the mechanisms responsible for the origins of phenotypic change in individuals and the spread of these traits in populations (Endler, 1992). In what follows, it will be shown that the modern synthesis can speak to the spread of traits in a population, but fails to adequately account for the origins of new phenotypes. Moreover, the view of life that emerges from the modern synthesis limits the contributions possible from the study of ontogenetic processes.

The modern synthesis is concerned only with inheritable traits (not phenotypes in general). In some accounts, evolution is even defined as changes in the inheritable traits in a population (e.g., Futuyma, 1986). That aspects of organisms can be divided into parts that are inherited and those that are acquired during the lifetime of the individual reflects a longstanding belief that there are two independent sources of developmental information: one internal, inherited, evolved, and informative (innateness, genes or nature), the other external, transient, and supportive (environment or nurture). As a result, organisms are said to contain a dual nature: an evolved, inherited genetic program (genotype), and an extended body (phenotype) generated under the instructions of the genetic program with the support of environmental factors (Mayr, 1988). Because natural selection can only operate on inheritable traits, one may state that biological evolution involves a change in nature, not nurture (Dobzhansky, 1962).

The architects of the modern synthesis were able to promote this dichotomous view of life by adopting the late 19th century belief that the processes involved in heredity were separate from those involved in development. According to this view, heredity refers exclusively to the transmission of germinal substances (i.e., genes) at the moment of conception, and development to the decoding of the genetic program (Mayr, 1988). Not only does the fertilized egg contain all the information necessary to assemble entire organisms (Simpson, 1967), but those genes present at the moment of fertilization will be the same, unaltered genes transmitted to the next generation. All that is required for a full explanation of heredity is knowledge of the genes present in the parental generation and the laws governing their transmission. The laws of transmission do not depend on and need not contain any reference to the development of organisms (Lewontin, 1992) or the development of the parental generation (Gayon, 1998). In other words, the course of development is predetermined by the genes, and there is no way for the experiences of the growing organism (or errors in the decoding process) to influence the (germline) genes that will be passed on during reproduction. Only genetic mutation and recombination can account for such changes. Hence, the study of ontogenetic processes is rendered superfluous to an understanding of the mechanisms responsible for origins of phenotypes.

The modern synthesis takes an additional step to minimize the gains of studying developing individuals by exclusively treating evolution as a population-level event. The evolution (of populations) and the development (of individuals) are said to operate according to different types of causes (Mayr, 1988). That is, the rules underlying changes in the genetics of a population are distinct from those governing the genetics of individuals (Dobzhansky, 1951).

This dichotomized view of life (organism/environment, nature/nurture, heredity/development, development/evolution) provided the foundation upon which the modern synthesis explained the origins and spread of traits. In brief, a two-step model is proposed in which (1) changes in genes (via mutation, recombination, drift) yield inheritable phenotypic variability and/or novelty, and (2) selections operating on these variants determine which genes persist and which are eliminated (Maynard-Smith, 2000; Mayr, 1988). Even though the two steps are said to be independent (Mayr, 1988), natural selection is often treated as *the* creative process in evolution and is used to account for both the origins and spread of traits in a population. Because genetic changes are thought to be relatively minor, natural selection can drive the accumulation of fortuitously advantageous genetic changes in populations in particular directions. As a result, natural selection is often discussed as the cause of individual development.

It must be kept in mind, however, that natural selection is both a consequence and a cause of developmental patterns (Gottlieb, 1992). Natural selection logically presumes phenotypic variation, and so cannot exclusively explain its origins (Ho & Saunders, 1979; Muller, 1990; Sober, 1995; see Gottlieb, 1992, for historical review). Instead, natural selection influences the maintenance (spread) of phenotypic variation in a population, and only after the variation has emerged in individuals (Gottlieb, 2002). With its emphasis on population genetics, the modern synthesis yields a statistical, non-causal account of how populations come to be comprised of individuals with particular characteristics, but it fails to provide an adequate account of how individuals in populations come to attain these characteristics (Walsh, 2003). A reliance on genetic processes like recombination and mutation as the sole causes of phenotypic change, or as the only *important* sources of change, oversimplifies the number of factors and time-dependent interactions that underlie ontogenetic development. Causal explanations of how individuals in populations develop certain characteristics can only be addressed through the study of ontogenetic processes (Gottlieb, 1992, 2002; Walsh, 2003).

Owing to a commitment to predeterminism and population-genetic thinking, the architects of what would become the predominant theory of evolution in the 20th century were not concerned with including developmental science in their synthesis (Gilbert, Opitz, & Raff, 1996). Still, it would be unfair to claim that the advocates of the modern synthesis completely ignored individual development (Futuyma, 1988) in that it has long been recognized that evolution results from changes in development (e.g., de Beer, 1956; Maynard-Smith, 2000). This admission, however, amounts to little more than saying that evolution brings about changes in genes, and these genetic changes, in turn, alter development. To even attempt explanations of evolution in terms of individual development has been characterized as an “error of misplaced reductionism” (Maynard-Smith, 1985).

Thus, the basic framework of the modern synthesis limits how comparative psychologists can contribute to knowledge of evolution: behavioral and developmental scientists can only speak to identifying the outcomes of evolution, or to the processes in the past that led to divergence between species. On this view, “all the organisms that now live or ever lived, all that they are and all that they do, are outcomes of genetic descent and modification” (Roe & Simpson, 1967, p. 5). Advances in evolutionary theory will no doubt extend the explanatory scope of comparative psychology, but this pathway of influence is decidedly one-sided: comparative psychologists simply cannot address evolution in its present sense, and so cannot illuminate the mechanisms of evolution.

Despite the popularity and achievements of the modern synthesis, there is a growing resistance to the view of life it depicts. Though this resistance is marked by a substantial diversity of opinion, it is generally agreed that the modern synthesis in its current, gene-centered form is far from complete and, at the very least, is in need of substantial revisions (Goodwin, 1982; Gottlieb, 1992; Ho & Fox, 1988; Ho & Saunders, 1979; Jablonka & Lamb, 1995; Lovtrup, 1987; Matsuda, 1979;

Newman & Muller, 2000; Oyama, 2000; Rose, 1998; Walsh, 2003). Whereas the modern synthesis adopted a view of development that allowed its adherents to 'safely sidestep' development, and thereby restrict the contributions that could be generated by studying individuals, modern advances in developmental theory allow analyses of individuals to reenter into discussions of evolutionary processes. Many of these developmental principles are already incorporated into contemporary texts of comparative psychology (e.g., Greenberg & Haraway, 2002; Michel & Moore, 1995; Papini, 2002), so it seems our field is well positioned to gain from and contribute to these advances in evolutionary thinking.

Dynamics of Evolving Developmental Systems

It should be clear from the previous section that how one views individual development has profound effects on how evolution is to be understood. In this section, I argue that many of the assumptions regarding development and heredity that provided a basis for the modern synthesis have failed to find support. These findings necessitate re-thinking how evolution is defined and studied.

Modern developmental theory resoundingly rejects the preformationism lingering behind the idea of pre-scripted developmental programs encoded somewhere in the organism (Godfrey-Smith, 2000; Griffiths & Gray, 1994; Johnston, 1987; Lickliter & Honeycutt, 2003; Mahner & Bunge, 1997; Oyama, 2000). The predetermined view of development has been replaced with a relational one that is focused on the changing (but predictable) dynamics of developmental systems composed of numerous nested factors both internal and external to the organism. All phenotypes, whether described as 'inherited' or 'acquired' are contextually determined. Genes (linear sequences of nuclear nucleotide bases) are an important part of this context, but they cannot be assigned any privileged role or ontological priority. Genes contribute to the control of development, but gene activity and gene structure are in turn regulated by the surrounding cellular and organismic context (Gottlieb, 1998; Johnston & Edwards, 2002). With this in mind, control of development is systemically distributed across the organism-environment system. As a result, stability and disparity in form within a lifetime or across generations cannot be attributed to any single component (like genes) because a whole host of factors related to one another through progressive (temporally nested) events with changing contexts combine to generate form.

Heredity, on this view, *must* involve more than genes and it *cannot* be complete at the moment of fertilization. Instead, inheritance involves numerous resources (DNA, cellular constituents, egg, uterus, nest site, symbionts, conspecifics, artifacts, etc) that are made available at various points throughout the lifespan (Griffiths & Gray, 1994; Ho, 1986; Johnston, 1987; Lickliter & Honeycutt, 2003; Oyama, 2000; West & King, 1987). If one must speak of heredity as transmission, then that which is transmitted includes nothing less than an entire structured context made available across various points of the lifespan, and organisms can be said to inherit these contexts as reliably as they inherit genes. Hereditary relations emerge out of (or more simply, describe) developmental outcomes or processes.

Expanding the temporal scale and number of factors involved in heredity does not accord well with the notion that heredity and development are independent or even different processes. Indeed, prior to the popular acceptance of heredity as germinal substance transmission at the end of the 19th century, heredity and development were considered to operate under a single, unified process (Bowler, 1989; Churchill, 1987). The lack of a clear distinction between heredity and development provided a basis for what Bowler (1989) has called the 'developmentalist' tradition in biology. According to the developmentalists, like Charles Darwin, new characters were believed to result from changes in the process of individual growth and reproduction (Bowler, 1989). Thus, the factors and processes responsible for individual development were thought to be the same factors and processes underlying evolution (Bowler, 2001).

Developmental theorists today, just as the developmentalists of the past, realize that if heredity and development cannot be meaningfully separated, then a strict separation between the processes underlying development and those underlying evolution seems unwarranted. A persistent change in any of the networks and interactions that are involved in the reliable reconstruction of organisms can lead generations of organisms to differ anatomically, physiologically, or behaviorally from their ancestors. *Modifications of many factors besides genes can bring about evolutionary change*.

Evolution in Action, I: Reality of Transgenerational Developmental Effects

For evolution to occur, ontogenetic processes must be altered. Understanding how and when these patterns are likely to change is thus essential to any evolutionary theory. Darwin was aware of the importance of these issues in that he questioned, for instance, how the timing and place of environmental effects experienced by members of one generation was reflected in the development of their descendents (Winther, 2000). Yet despite Darwin's efforts, work has been slow to accumulate given the stigma of being labeled Lamarckian and because a number of early empirical reports in this area were shrouded in controversy and alleged misconduct (see Blacher, 1982; Koestler, 1971, for discussions). Nevertheless, there is now a growing body of evidence to support taking transgenerational influences seriously (Campbell & Perkins, 1988; Rossiter, 1996).

Some of the most striking examples of transgenerational phenomena are physiological effects associated with exposure to toxins and drugs:

(1) Guyer & Smith (1920) obtained blood serum derived from chickens that contained antibodies sensitized to the eye lenses of rabbits (i.e., anti-lens serum). When the fowl-derived anti-lens serum was injected into pregnant rabbits, many of the exposed fetuses (Generation 1) were born with mal-formed eyes (reduced size, discolorations, or even complete disappearance of eyeballs). When these rabbits (Generation 1) were bred with one another, the appearance of eye defects continued to appear in some of the offspring (Generation 2) despite a lack of anti-lens serum exposure, and these abnormalities grew more pronounced through the sixth

generation. Breeding-treated males with unrelated, untreated females was also found to yield eye defects in descendents.

(2) Prenatal alcohol exposure can also lead to ocular defects. Ismail and Jan-jua (2004) found that about 30% of rats prenatally exposed to ethanol showed ocular defects. When these rats were crossbred, about 40% of their offspring, and 45% of their grandoffspring continued showing ocular defects in the absence of alcohol exposure.

(3) Waddington (1956) found that when fruit fly pupa were exposed to ether, many of the flies developed an extra set of wings (a bithorax copy). After repeating this procedure for 12 generations, he found that many flies con-tinued to develop the bithorax phenotype even in the absence of ether ex-posure. Waddington also showed similar transgenerational effects follow-ing heat shock on vein patterning (Waddington, 1953) and salt exposure on anal pupae (Waddington, 1959). At least for the bithorax phenotype, the transgenerational persistence of the extra wings can occur even in the ab-sence of selective breeding (Ho, Tucker, Keeley, & Saunders, 1983).

(4) Oh, Gelardi, & Cha (1991) produced drug-induced hyperglycemia in preg-nant rats that resulted in hyperglycemia and accelerated growth in their offspring (Generation 1). The effects of the single treatment in Generation 1 were observed in the third generation of offspring (Generation 3), who also showed accelerated growth, higher insulin levels, and greater glucose intolerance than control third-generation rats (for similar examples, see Goldner & Sperger, 1972).

(5) Pavelka and Koudelova (2001) followed the offspring of a mutant strain of Mediterranean flour moth that contained an autosomal recessive mutation associated with the development of short antennae. It was found that the appearance of short antennae in the mutant strain could be suppressed by incubating the pupa at a higher than normal temperature (25º C rather than 20º C). That is, adult moths of the mutant strain that were reared in a higher temperature during certain larval and pupal stages did not display the short antennae: Their antennae were indistinguishable from the anten-nae of the wild-type strain. The appearance of normal-sized antennae was found to persist in the next 5 generations of offspring even when the off-spring were maintained in a constant temperature (20º C). Similar effects in the transgenerational suppression of short antennae were associated with exposure to lithium or alternating electric currents.

These examples underscore the reality and potential prevalence of transgenerational phenomena. Each example shows that an unusual experience (e.g., temperature shock) of one generation can impact the development of subsequent generations even when the latter individuals do not encounter the unusual experience (no temperature shock). Although the mechanisms underlying the transgenerational persistence of such effects are not known, they likely involve a form of ‘cellular memory’ known as epigenetic markings (chromatin packaging and methylation patterns; see Jablonka & Lamb, 1995). These markings, formerly believed to be erased during reproduction, are now known to persist across generations (Jablonka & Lamb, 1995; Rakyan, Preis, Morgan, & Whitelaw, 2001).

Other examples of transgenerational phenomena may be mediated in part by changes in behavior:

(1) Zamenhof and colleagues (reviewed in Zamenhoff & van Marthens, 1978) fed pregnant rats a diet deficient in protein. The newborn offspring (Gen-eration 1) of these rats showed significantly lower body weights, fewer brain cells, and reduced brain protein than newborns born to mothers on different diets. When the Generation 1 pups were raised on a normal diet, allowed to mate, and reproduced, their offspring (Generation 2) continued to display significantly lower brain and body measures and also showed learning deficits as adults.

(2) Stewart and colleagues (1975; 1980, cited in Drake and Walker, 2004) maintained colonies of rats on either a control diet or on a diet marginally deficient in protein for 12 generations. Offspring of the malnourished col-ony showed significantly reduced birth weights, a trend that became more amplified in later generations. Even when animals from the malnourished colony (several generations later) were given the control diet, their off-spring continued to show growth deficits for the next three generations.

(3) In a series of studies, Barnett and colleagues (see Barnett, 1973, for re-view) maintained strains (both wild and laboratory) of mice in cold (-3° C) or warm (21° C) laboratory environments. After several generations, the offspring of mice in the cold environment were heavier at birth than mice in the warm environment. After ten generations in the cold environment, some mice were transferred into the warm environment, allowed to breed, and their offspring were followed for two generations. Compared to mice maintained in the warm environment throughout, the offspring of the transferred mice continued to be substantially heavier at birth.

(4) First and second generation offspring of rats that were handled during in-fancy display reliable differences in their behavioral and hormonal re-sponses to stress (Denenberg & Rosenberg, 1967; Denenberg & Whimbey, 1967) and brain development (Meaney, 2001).

Here again, the transgenerational mechanisms responsible for these results are not well understood. Besides any direct environmental effects on gamete composition or uterine qualities that could be attributed to the initiating factor (e.g., protein deficient diets, extreme cold), the transgenerational persistence seen in each of the above examples could involve alterations in the behavior of the dams (or pups) before and/or following birth. That behavior can contribute to transgenerational phenomena has been most clearly shown in the effects of handling infant rat pups. Handling appears to influence styles of maternal behaviors (e.g., nursing patterns) which contribute to the transgenerational recurrence of altered stress responses and brain development (Francis, Diorio, Liu, & Meaney, 1999).

One can only speculate as to the role of behavior in the remaining examples. In some cases (Zamenhof's work; Barnett's work), postnatal maternal effects were thought to be unimportant in that the cross-fostering of pups between dams of different conditions yielded no discernable effects. It could be, however, that characteristics of the pups elicited different maternal behaviors (e.g., less anogenital licking; fewer milk letdowns) even from foster dams. It is not possible to address this issue without quantifying maternal behaviors. Also not ruled out in this work is the potential role of altered activity levels of dams during pregnancy. Lowered activity levels (associated with poor nutrition, extreme cold, stress/emotionality) during pregnancy may generally understimulate the fetuses by reducing levels of fetal sensory stimulation and movement, reducing blood-flow to the fetuses, or altering circulating hormones. Thus, there are numerous and potentially non-obvious ways that behavior can contribute to the transgenerational maintenance of phenotypes.

All of the transgenerational effects reviewed above involve exposing one generation of individuals to an unusual event and tracing its effects in subsequent generations who develop in the absence of this event. A case was made that modifications in the activity of organisms could play an important role in the persistence of these effects across generations. If so, such behaviorally-based mechanisms may stand as an additional way in which the activity of organisms is involved in maintaining phenotypes across generations besides notions of 'social heredity' or 'cultural traditions' (e.g., Freeberg & White, 2006; see Avital & Jablonka, 2000; Heyes & Galef, 1996, for review).

In this section, I presented evidence that the developmental experiences of one generation can in some instances influence developmental patterns of their offspring and their offspring's offspring. In most cases, the mechanisms underlying these effects are not well understood, but it seems that modifications in the activity of organisms may be responsible in part. Thus, behavior can be discussed as both an effect of ancestral conditions (e.g., learning deficits due to grandparental malnutrition), as well as a mechanism responsible for maintaining phenotypes across generations (e.g., patterns of maternal behavior; cultural traditions).

Evolution in Action, II: Environmental Effects on Phenotypes

When evolution is defined in terms of the processes by which organisms come to differ (behaviorally, physiologically, or anatomically) from their ancestors, we must consider the direct influences of environmental factors on phenotypic development and evolution. Drastic changes in environment in one generation can influence the development of generations to come. Understanding the re-organization of developmental resources and interactions across generations stands at the very heart of understanding the mechanisms of evolution.

The importance of direct environmental effects on the development of individuals has been downplayed in modern accounts of evolution. The little attention paid to such effects seems surprising given the widespread belief that early stages of evolutionary transitions involve environmental modifications (e.g., habitats, climate, diet, nesting sites, etc.), and it is even more surprising when one considers that modern evolutionary theory promotes an externalist view in which the environment is seen as primarily causing organic form (Sterelny, 2005).

Despite the direct and formative influence of environmental factors known to play a role in ontogeny, most evolutionary theorists consider environmental changes to have only an indirect role in phenotypic evolution. Environmental factors are most often discussed in terms of their selective effects. Occasionally, external conditions are said to alter phenotypic evolution by influencing genetic mutation rates. More recently, attention has returned to another indirect way that environmental changes can influence evolution. I am referring here to the century old issue of "organic selection" put forth at around the same time by J. M. Baldwin, H. F. Osborn, and C. Lloyd Morgan (e.g., Avital & Jablonka, 2000; Weber & Depew, 2003). Organic selection refers to the idea that animals can modify their behavior to adaptably accommodate environmental alterations, and that these behaviors are socially transmitted across generations long enough for the accumulation of those genetic mutations that predispose the development of the adaptive behavior patterns. In other words, changes in social environments allow animals to temporarily adapt to the demands of environmental alterations, but true evolutionary change does not happen until the genes of these animals have taken over. Thus, to a greater or lesser extent, environmental factors can play an "indirect" role in shaping phenotypic development in that all environmental effects on phenotypic evolution are mediated by genetic changes.

The reality of transgenerational effects on development speaks to the importance of considering direct environmental effects in our accounts of evolution. When organisms come to occupy new habitats or live differently in existing ones, predicting the course of evolution will require that we take into account any new selection and mutation pressures, but will also require that we look for any direct effects that new environmental factors might have on the development of individuals and their progeny. Phenotypes of individuals in a population could systematically change if all members of the population share a particular environmentally-based developmental resource. Thus, if we want to understand the dynamics of phenotypic evolution we must consider direct environmental effects and realize that these effects may influence development of generations to come.

That direct environmental effects may influence evolutionary patterns is not a new idea, and was discussed by both Lamarck and Darwin. In modern times, direct environmental influences on phenotypic development and evolution underscore the notion that phenotypic modifications can arise prior to (or without) changes in gene structure, which is traditionally treated as the sign and basic mechanism of evolution (Gottlieb, 1987, 1992, 2002; Ho, 1998; Johnston & Gottlieb, 1990; Newman & Muller, 2000; Slobodkin & Rappaport, 1976). In other words, genetic change is not required for phenotypic evolution.

Evolution in Action, III: Methodological Suggestions

Identifying and explaining transgenerational phenomena are developmental issues and thus require developmental analyses. Comparative psychology has a long tradition devoted to documenting how ontogenetically early experiences influence subsequent outcomes throughout the lifespan of individuals (Gottlieb, 1978; Michel & Moore, 1995). Adopting a transgenerational orientation requires shifting the basic unit of analysis from individual organisms to lineages of organisms, and broadening the scope of developmental studies to include multiple generations of organisms. Questions can then be posed to determine how experi-ences of members of one generation in a lineage influence developmental patterns in subsequent members of that lineage. At least initially, the goal of such studies is to determine whether developmental patterns of one generation are reliably related to contextual factors encountered by earlier members of that lineage. The actual developmental patterns shown by the earlier and later generations need not be the same; they need only be systematically related. In other words, progeny may appear similar or increasingly different from their ancestors due the experiences of the latter. Both cases constitute "hereditary" effects, but the fact that progeny can be increasingly different calls into question the colloquial use of heredity in reference to how "like begets like" (e.g., Darwin, 1988).

Through extensive experimentation, attention must be paid to how transgenerational effects are brought about and sustained through a lineage. As described earlier, it is possible that a phenotypic change can be initiated (e.g., via handling) and maintained (via maternal behaviors) through different mechanisms, so that the factors involved in the appearance of altered phenotypes in subsequent generations may not be the same factors that initiated the change in the first generation.

Moreover, different lineages may differ in terms of susceptibility and resilience to perturbations. What accounts for these differences? How can we bring about or eliminate differences between lineages? In addressing these issues, our efforts could be more closely aligned with traditional population-level measures of evolution by comparing lineages in terms of the relative viability of its members, mating patterns, and reproductive output (see Freeberg & White, 2006).

As a final methodological suggestion, I believe the experimental investigation of transgenerational phenomena would benefit by drawing upon the methods commonly employed in studies of selective breeding and domestication. The lat-ter fields were at one time prominent traditions within comparative psychology and can still provide powerful concepts and techniques for comparative analyses of animal development across generations (Lickliter & Ness, 1990).

Conclusions

This essay promotes the general idea that knowledge of the dynamics of developmental processes can illuminate mechanisms of evolutionary change. With this in mind, it seems reasonable to ask how developmental patterns change across generations. It does not seem reasonable to place limits on whether or how to go about empirically addressing this issue based solely on *a priori* conceptual commitments that hold such pathways of influence to be impossible or irrelevant to evolutionary analyses. Transgenerational effects are real: alterations in the developmental contexts of one generation can have persisting influences on subsequent generations in that lineage even in the absence of the initiating event. They are not mere curiosities that have no bearing on knowledge of evolution. To the contrary, the examples reviewed earlier can be considered instances, however small and perhaps non-adaptive, of evolution in its most general sense. The history, methods, and available concepts in the field of comparative psychology render comparative psychologists well positioned to pursue this line of inquiry. I hope that by exploring the extent and nature of transgenerational changes in development, comparative psychologists will have a louder voice in discussions of evolutionary processes past and present.

References

Avital & Jablonka (2000). *Behavioural traditions: Behavioral inheritance in evolution*. Cambridge, UK: Cambridge University Press

Barnett, S. A. (1973). Maternal processes in the cold-adaptation of mice. *Biological Reviews*, **48**, 477-508.

Bateson, P. (1988). The active role of behavior in evolution. In M-W. Ho & W. Fox (Eds.), *Evolutionary processes and metaphors* (pp. 191-207). London, UK: Wiley.

Blacher, L. I. (1982). *The problem of the inheritance of acquired characteristics: a history of a priori and empirical methods used to find a solution*. Washington, DC: Amerind Publishing Co.

Bowler, P. J. (1989). *The Mendelian revolution: The emergence of hereditarian concepts in modern science and society*. New York: Blackwell.

Bowler, P. J. (2001). Evolutionary ideas: The eclipse of Darwinism. In: *Encyclopedia of Life Sciences*. Chichester, UK: Wiley. Retrieved from http://www.else.net

Campbell, J. H., & Perkins, P. (1988). Transgenerational effects of drug and hormonal treatments in mammals: a review of observations and ideas. In G. J. Boer, M. G. P. Feenstra, M. Mimiran, D. F. Swaab, & F. Van Haaren (Eds.), *Progress in brain research,* Vol 73. (pp. 535-552). Amsterdam, The Netherlands: Elsevier.

Churchill, F. B. (1987). From heredity theory to *Vererbung*.: The transmission problem, 1850-1915. *ISIS*, **78**. 337-364.

Conklin, E. G. (1919). The mechanism of evolution in the light of heredity and development. *Scientific Monthly*, **7**, 481-505.

Conklin, E. G. (1965). *Heredity and environment in the development of men* (6th ed). New York: Johnson Reprint Corp.

Darwin, C. (1988). Variation of animals and plants under domestication (Vol. 1). In P. H. Barrett & R. B. Freeman (Eds.), *The works of Charles Darwin* (Vol. 19). London, UK: William Pickering. (Original work published in 1875).

DeBeer, G. (1940). *Embryos and ancestors*. Oxford, UK: Clarendon Press.

Denenberg, V. H., & Rosenberg, K. M. (1967). Nongenetic transmission of information. *Nature*, **216**, 549-550.

Denenberg, V. H., & Whimbey, A. E. (1963). Behavior of adult rats is modified by the experiences their mothers had as infants. *Science*, **142**, 1192-1193.

Dobzhansky, T. (1951). *Genetics and the origin of species*. New York: Columbia University Press.

Dobzhansky, T. (1962). *Mankind evolving: the evolution of the human species*. New Haven, CT: Yale University Press.

Drake, A. J., & Walker, B. R. (2004). The intergenerational effects of fetal programming: Non-genomic mechanisms for the inheritance of low birth weight and cardiovascular risk. *Journal of Endocrinology*, **180**, 1-16.

Endler, J. A. (1992). Natural selection: Current usages. In E. F. Keller & E. A. Lloyd (Eds.). *Keywords in evolutionary biology* (pp. 220-224). Cambridge, MA: Harvard University Press.

Francis, D., Diorio, J., Liu, D., & Meaney, M. J. (1999). Nongenomic transmission across generations of maternal behavior and stress responses in the rat. *Science*, **286**, 1155-1158.

Freeberg & White (2006). Social traditions and the maintenance and loss of geographic variation in mating patterns of brown-headed cowbirds. *International Journal of Comparative Psychology, **19***, 206-222.

Futuyma, D. J. (1986). *Evolutionary Biology* (2nd ed.). Sunderland, MA: Sinauer.

Futuyma, D. J. (1988). *Sturm and drang* and the evolutionary synthesis. *Evolution*, **42**, 217-226.

Gaulin, S. J. C., & McBurney, D. H. (2001). *Psychology: An evolutionary approach*. Upper Saddle River, NJ: Prentice Hall.

Gayon, J. (1998). *Darwin's struggle for survival – Heredity and the hypothesis of natural selection*. Cambridge, UK: Cambridge University Press.

Gilbert, S. F., Opitz, J. M., & Raff, R. A. (1996). Resynthesizing evolutionary and developmental biology. *Developmental Biology*, **173**, 357-372.

Godfrey-Smith, P. (2000). On the theoretical role of genetic coding. *Philosophy of Science*, **67**, 26-44.

Goldner, M. G., & Spergel, G. (1972). On the transmission of alloxan diabetes and other diabetogenic influences. *Advances in Metabolic Disorders*, **6**, 57-72.

Goodwin, B. C. (1982). Development and evolution. *Journal of Theoretical Biology*, **97**, 53-55.

Gottlieb, G. (1978). *Studies on the development of behavior and the nervous system: Early influences*. New York: Academic Press

Gottlieb, G. (1987). The developmental basis of evolutionary change. *Journal of Comparative Psychology*, **101**, 262-271.

Gottlieb, G. (1992*). Individual development and evolution: The genesis of novel behavior*. New York: Oxford University Press.

Gottlieb, G. (1998). Normally occurring environmental and behavioral influences on gene activity: From central dogma to probabilistic epigenesis. *Psychological Review*, **105**, 792-802.

Gottlieb, G. (2002). Developmental-behavioral initiation of evolutionary change. *Psychological Review*, **109**, 211-218.

Greenberg, G., & Haraway, M. M. (2002). *Principles of comparative psychology*. Boston, MA: Allyn and Bacon.

Griffiths, P.E., & Gray, R.D. (1994). Developmental systems and evolutionary explanation. *The Journal of Philosophy*, **91**, 277-304.

Guyer, M. F., & Smith, E. A. (1920). Studies on cytolysins. II. Transmission of induced eye-defects. *Journal of Experimental Zoology*, **31**, 65-82.

Heyes, C. M., & Galef, B. G. (Eds.). (1996). *Social learning in animals: The roots of culture*. New York: Academic Press.

Ho, M-W. (1986). Heredity as process: Towards a radical reformulation of heredity. *Rivista di Biologia*, **79**, 407-444.

Ho, M-W. (1998). Evolution. In G. Greenberg and M. M. Haraway (Eds.), *Comparative psychology: A handbook* (pp. 107-119). New York: Garland Publishing.

Ho., M-W., & Fox S. W. (Eds.). (1988). *Evolutionary processes and metaphors*. New York: John Wiley.

Ho, M-W., & Saunders, P. T. (1979). Beyond neo-Darwinism: an epigenetic approach. *Journal of Theoretical Biology*, **78**, 573-591.

Ho, M-W, Tucker, C., Keeley, D., & Saunders, P. T. (1983). Effects of successive generations of ether treatment on penetrance and expression of the bithorax phenocopy in *Drosophila melanogaster*. *Journal of Experimental Zoology*, **225**, 357-368.

Ismail, M., & Janjua, M. Z. (2004). Ocular defects in first generation of ethanol exposed albino rats and its penetrance into third generation. *Journal of the Ayub Medical College Abottabad*, **16**, 77-79.

Jablonka, E., & Lamb, M. J. (1995). *Epigenetic inheritance and evolution*. New York: Oxford University Press.

Johnston, T. D. (1987). The persistence of dichotomies in the study of behavioral development. *Developmental Review*, **7**, 149-182.

Johnston, T. D., & Edwards, L. (2002). Genes, interactions, and development. *Psychological Review*, **109**, 26-34.

Johnston, T. D., & Gottlieb, G. (1990). Neophenogenesis: A developmental theory of phenotypic evolution. *Journal of Theoretical Biology*, **147**, 471-495.

Koestler, A. (1971). *The case of the midwife toad*. New York: Random House.

Laland, K. N., Odling-Smee, F. J., & Feldman, M. W. (1999). Niche construction, biological evolution, and cultural change. *Behavioral Brain Sciences*, **23**, 131-175.

Lewontin, R. C. (1992). Genotype and phenotype. In E. F. Keller & E. A. Lloyd (Eds.). *Keywords in evolutionary biology* (pp. 137-144). Cambridge, MA: Harvard University Press.

Lickliter, R. L., & Honeycutt, H. G. (2002). Developmental dynamics: Toward a biologically plausible evolutionary psychology. *Psychological Bulletin*, **129**, 819-835.

Lickliter, R. L., & Ness, J. W. (1990). Domestication and comparative psychology: Status and strategy. *Journal of Comparative Psychology*, **104**, 211-218.

Lovtrup, S. (1987). *Darwinism: Refutation of a myth*. Beckenham, England: Croom Helm.

Mahner, M, & Bunge, M. (1997). *Foundations of biophilosophy*. New York: Springer

Matsuda, R. (1979). *Animal evolution in changing environments*. New York: Wiley.

Maynard Smith, J. (1985). Evolution and development. In B. Goodwin, N. Holder, & C.C. Wylie (Eds.). *Development and evolution* (pp. 33-45). Cambridge, UK: Cambridge University Press.

Maynard Smith, J. (2000). *The theory of evolution* (3rd ed). New York: Cambridge University Press.

Mayr, E. (1988). *Toward a new philosophy of biology: Observations of an evolutionist*. Cambridge, MA: Harvard University Press.

Meaney, M. J. (2001). Maternal care, gene expression, and the transmission of individual differences in stress reactivity across generations. *Annual Review of Neuroscience*, **24**, 1161-192.

Michel, G. & Moore, C. (1995*). Developmental psychobiology: An integrative science*. Cambridge, MA: MIT Press.

Muller, G. B. (1990). Developmental mechanisms and the origin of morphological novelty: A side-effect hypothesis. In M. H. Nitecki (Ed.), *Evolutionary innovations* (pp. 99-130). Chicago, IL: University of Chicago Press.

Newman, S. A., & Muller, G. B. (2000). Epigenetic origins of character origination. *Journal of Experimental Zoology*, **288**, 304-317.

Oh, W., Gelardi, N. L., & Cha, C. J. (1991). The cross-generation effect of neonatal macrosomia in rat pups of streptozotocin-induced diabetes. *Pediatric Research*, **29**, 606-610.

Oyama, S. (2000). *Evolution's eye*. Durham, NC: Duke University Press.

Oyama, S., Griffith, P. E., & Gray, R. D. (Eds.). (2001). *Cycles of contingency: Developmental systems and evolution*. Cambridge, MA: MIT Press.

Papini, M. R. (2002). *Comparative psychology: Evolution and development of behavior*. Upper Saddle River, NJ: Prentice hall.

Pavelka, J., & Koudelova, J. (2001). Inheritance of a temperature-modified ohenotype of the short antennae (sa) mutation in a moth, *Ephestia kuehniella* (Lepidoptera Pyralidae). *Journal of Heredity*, **92**, 234-242.

Piaget, J. (1978). *Behavior and evolution*. New York: Pantheon Books.

Plotkin, H. C. (Ed.). (1988). *The role of behavior in evolution*. Cambridge, MA: M.I.T. Press.

Rakyan, V. K., Preis, J., Morgan, H. D., Whitelaw, E. (2001). The marks, mechanisms and memory of epigenetic states in mammals. *Biochemistry Journal*, **356**, 1-10.

Roe, E., & Simpson, G. G. (Eds.). (1967). *Behavior and evolution*. New Haven, CT: Yale University Press.

Rose, S. (1998). *Lifelines: Biology beyond determinism*. New York: Oxford University Press.

Rossiter, M. C. (1996). Incidence and consequence of inherited environmental effects. *Annual Review of Ecology and Systematics*, **27**, 451-476.

Simpson, G. G. (1967). Behavior and evolution. In A. Roe & G. G. Simpson (Eds.), *Behavior and evolution* (pp. 507-535). New Haven, CT: Yale University Press.

Slobodkin, L. B., & Rapoport, A. (1974). An optimal strategy of evolution. *Quarterly Review of Biology*, **49**, 181-200.

Sober, E. (1995). Natural selection and distributive explanation: A reply to Neander. *British Journal for the Philosophy of Science*, **46**, 384-397.

Stamps, J. (2003). Behavioural processes affecting development: Tinbergen's fourth question comes of age. *Animal Behaviour*, **66**, 1-13.

Sterelny, K. (2005). Made by each other: Organisms and their environment. *Biology and Philosophy*, **20**, 21-36.

Stewart, R. J., Preece, R. F., Sheppard, H. G. (1975). Twelve generations of marginal protein deficiency. *British Journal of Nutrition*, **33**, 233-253.

Stewart, R. J., Sheppard, H. G., Preece, R. F., & Waterlow, J. C. (1980). The effect of rehabilitation of different ages of development of rats marginally malnourished for ten to twelve generations. *British Journal of Nutrition*, **43**, 403-412.

Waddington, C. H. (1953). Genetic assimilation of an acquired character. *Evolution*, **7**, 118-126.

Waddington, C. H. (1956). Genetic assimilation of the *bithorax* phenotype. *Evolution*, **10**, 1-13.

Waddington (1959). Canalization of development and genetic assimilation of acquired characters. *Nature*, **183**, 1654-1655.

Walsh, D. M. (2003). Fit and diversity: Explaining adaptive evolution. *Philosophy of Science*, **70**, 280-301.

Weber, B. H., & Depew, D. J. (Eds.). (2003). *Evolution and learning: The Baldwin effect reconsidered*. Cambridge, MA: MIT Press.

West, M. E., & King, A. P. (1987). Settling nature-nurture into an ontogenetic niche. *Developmental Psychobiology*, **20**, 549-562.

Winther, R. G. (2000). Darwin on variation and heredity. *Journal of the History of Biology*, **33**, 425-455.

Zamenhoff, S, & Van Marthens, E. (1978). Nutritional influences on prenatal brain development. In G. Gottlieb (Ed.), *Studies on the development of behavior and the nervous system*: *Early Influences* (pp. 149-186). New York: Academic Press

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