What Is an Adaptive Environmentally Induced Parental Effect?

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Article:

Biologists working in fields as diverse as mammalian behavior, plant ecology, microbial genetics, quantitative genetics, and insect ecology have shown that environmentally induced parenta1 effects can be found in most kingdoms of living organisms. Such effects are diverse, have multiple causes, and can be transmitted via multiple pathways. Historically, and understandably, these effects have been studied by biologists who have focused on a particular group of organisms, such as insects or plants, or who have approached the phenomenon from a particular point of view, such as quantitative genetics, ecology, or behavior. The consequence has been the development of multiple terminologies that are not used consistently across disciplines or kingdoms. I believe these inconsistencies hinder the communication among biologists studying these effects, the development of generalized models of parental effects, and the empirical testing of adaptedness of these effects.

The following essay is my attempt to provide a terminology that can be used consistently across kingdoms, regardless of approach. It is also my attempt to provide a conceptual framework for studying environmentally induced parental effects. First, I briefly discuss the diversity of environmentally induced parenta1 effects. Then I offer general definitions for a parenta1 effect and an environmentally induced parental effect. This is not a trivial exercise because of the present confusion about existing terms. Third. I discuss three general classes of environmentally induced parental effects. Finally. I consider what it means for an environmentally induced parental effect to be "adaptive" and consider which class(es) describes adaptive effects.

4.1 Diversity of Environmentally Induced Parental Effects

Recent books and articles have documented a variety of environmentally induced parental effects that we can observe in nature (e.g., Boyd and Richerson 1985; Roach and Wulff 1987; Clutton-Brock 1991; Mousseau and Dingle 1991a,b; Reznick 1991; Sinervo 1991; Jablonka and Lamb 1995; Bernardo 1996; Lacey 1996; Mazer and Gorchov 1996; Rossiter 1996). For example, in multicellular plants, the parental environment is known to modify such offspring traits as seed size, germination, growth, flowering time, and sexuality (see reviews by Rowe 1964; Roach and Wulff 1987; Gutterman 1992; Wulff)995; see also Galloway 1995; Case et al. 1996). In multicellular animals, it is known to modify such traits as egg size, growth rate, resistance to pathogens, tíme to reproduction, sexuality, behavior, and culture (reviewed by Boyd and Richerson 1985; Mousseau and Dingle 1991a,b; Reznick 1991; Sinervo 1991; Jablonka and Lamb 1995; Bernardo 1996; Rossiter 1996). In unicellular organisms, it is known to modify such traits as cell architecture and the use of potential food sources (reviewed by Jablonka and Lamb 1995). I refer the reader to these cited references for more detailed information about these effects and their proximate causes. What I think needs to be emphasized about these effects is that they can be observed at many levels of biological organization. When the environment stimulates parents to transmit cultural beliefs and practices to a child, the environment produces an environmental effect. When an individual cell is environmentally stimulated to pass nongenetic information to daughter cells, then an environmental effect results. Environmentally induced parental effects can be observed and are being studied at many levels of biological organization, and this is only now being appreciated.

The stimulus for an environmental effect can occur at any time during a parent's lifetime and is transient in that the stimulus usually disappears before an offspring shows the effect of the stimulus. The parental response to the stimulus can be permanent or transient. If the parental response is permanent or persists for a long time, then the environmentally induced parental effect may manifest itself in a group of related individuals. All progeny, and in some cases also a parent, may exhibit the same phenotype. For example, parents may teach all offspring the same foraging techniques or cultural practices. On the other hand, if the parental response to an environmental stimulus is ephemeral, then an environmental change may result in parents producing offspring with multiple phenotypes for the trait affected. For example, diminishing maternal resources may result in variable seed sizes or birth weights within one reproductive season; changing photoperiod during the parental generation may produce offspring with variable degrees of dormancy or diapause.

The pathways by which an environmental stimulus can produce an effect are also diverse. An effect may be transmitted by an individual through word of mouth or by example, as with parental care or the teaching of cultural practices (Boyd and Richerson 1985). It may be transmitted via maternal tissue that is carried along with the offspring tissues into the next generation, for example, the coat of a seed (Roach and Wulff 1987). It may be transmitted via molecular messengers that are transferred from maternal or paternal cell to offspring cell, for example, through the cytoplasm and through proteins that regulate gene expression (e.g., Matzke and Matzke 1993; Jablonka and Lamb 1995).

4.2 What Is an Environmentally Induced Parental Effect?

Defining an environmentally induced parental effect first requires agreement on the definition of a parental effect. Parental effects have traditionally been called "maternal effects" because effects transmitted via the mother were the first to be noticed and because paternal effects, transmitted via the father, were thought to be minimal or nonexistent (e.g., see reviews: Roach and Wulff 1987; Bernardo 1996; Rossiter 1996). Recent studies suggest, however, that paternal effects may be more important than previously thought (e.g., insects: Giesel 1986, 1988; Boggs 1995; Fox et al. 1995; mammals: Clutton-Brock 1991; plants: Lacey 1996; Mazer and Gorchov 1996). Therefore, it seems time to break with tradition and use the term "parental effects" to embrace both maternally and paternally transmitted effects and those effects for which the pathway of transmission is unknown (Lacey 1996). Also, because parents can influence their young in so many ways, it seems important to define a parenta1 effect as broadly as possible so that the term will be generally useful (Bernardo 1996).

I suggest the following definition; *A parental effect is any parental influence on offspring phenotype that cannot be attributed solely to offspring genotype, to the direct action of the nonparental components of the offspring's environment, or to their combination.* This effect is the phenotypic product of the transmission of "information" from parent to offspring above and beyond the parental contribution to offspring nuclear and cytoplasmic genes. Note that I am deliberately excluding the effects of extranuclear (maternal and paternal) inheritance in my definition of a parenta1 effect. I am also excluding random mutations that originate with a parent and are passed to offspring. (I will discuss nonrandom mutations in section 4.2.) Maternal selection (as defined by Kirkpatrick and Lande 1989) is included. A parental effect begins when a parenta1 genotype responds to some signal. That response induces the transmission of information along one or several transmission pathways. Both the response and transmission pathways may have genetic components (e.g., Riska et al. 1985; Kirkpatrick and Lande 1989; Cowley and Atchley 1992). The parental effect is the end product of that transmission, the phenotypic modification of an offspring trait.

There are many definitions of a parental effect in the literature, and suggesting yet another one may seem either audacious or redundant. In spite of these definitions, however, many biologists are still confused about the meaning of a parental effect. This persistent confusion, along with recent studies elucidating the possible mechanisms by which these effects are transmitted, suggest that we try to define a parent effect again. Many biologists have defined a parental effect as a parental influence on offspring phenotype that cannot be attributed to the normal Mendelian transmission of chromosomes (e.g., Mather and Jinks 1971; Riska et al. 1985; Roach and Wulff 1987; Kirkpatrick and Lande 1989; Cowley and Atchley 1992; Platenkamp and Shaw 1993; Carriere

1994; Wulff 1995; Lacey 1996; Mazer and Gorchov 1996; Rossiter, 1996). This definition assumes that a parental effect cannot be controlled by nuclear genes, an untenable assumption. Defining a parental (maternal) effect as "a part of an offspring's phenotype that does not result from the action of its own genes . . ." (Bernardo, 1996) excludes parental effects involving modifications in gene activity, which I believe should be embraced by the term "parental effects". Defining a parental (maternal) effect in terms of the effect of "parental performance" (Cheverud 1984), or "parental phenotype" (Arnold 1994; Bernardo. 1996) or the effect of "genetic and environmenta1 differences in the maternal generation" (Mousseau and Dingle 1991a) on offspring phenotype has resulted in multiple interpretations about what can and cannot be considered a parental effect. For example, none of these definitions explicitly separates parenta1 effects from the parental chromosomal contributions to offspring genotype; these parental chromosomes also partially determine parental phenotype. Some traits that are determined by maternal cytoplasmic genes, such as coiling in snails, are not expressed in the maternal phenotype, and yet these definitions have embraced the effects of cytoplasmic inheritance. To try to resolve the above problems, I have suggested defining a parental effect in terms of offspring genotype and its environment. Also, I have conceptually separated parental effects from extranuclear inheritance.

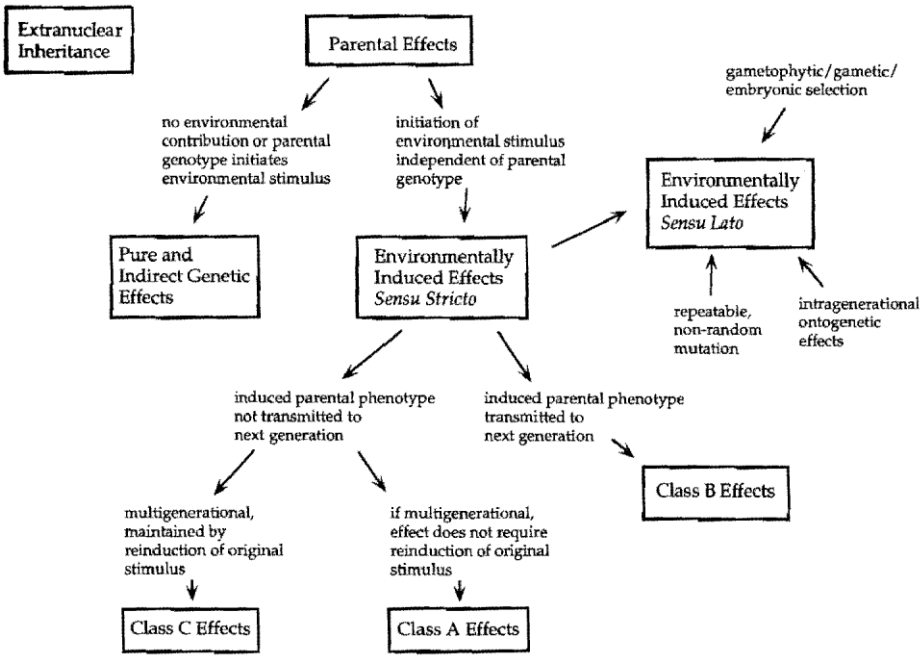

Figure 4.1 Schematic drawing of the relationships among parental effects, different classes of environmentally induced parental effects, and other phenomena that have historically been confounded with these effects. See section 4.2 for further explanation.

A parental effect can be genetically based and/or environmentally induced. Purely genetically based effects, at least given our present understanding of parental effects, include imprinting, in which the sex of the parent determines the expression of genes passed to offspring (e.g., Solter 1988; Surani 1991; Matzke and Matzke 1993). Indirect genetic effects, as they apply to parent-offspring relationships, are contributions that parental genotypes make to offspring phenotype as mediated by parental environment (Moore et al., chap. 2, this volume.). In this case, the parental genotype determines the environmental stimulus that produces the phenotypic effect in the offspring. Environmentally induced parental effects are induced by the parental environment, but the environmental stimulus arises índependently of parental genotype (Figure 4.1).

Quantitative geneticists have often looked for parental effects by measuring the asymmetry of maternal and paternal contributions to offspring phenotype in reciprocal crosses. Unequal parental contributions can only suggest that a parental effect exists, however. Parental effects may be environmentally induced in both parents and, therefore, not manifest themselves. Maternal or paternal inheritance, forms of extranuclear inheritance, will

also produce asymmetrical parental contributions. Therefore, reciprocal crosses alone do not suffice to measure the existence of parental effects.

An environmentally induced parental effect is one that (1) is initiated by an environmental stimulus in the parental generation and (2) cannot be exclusively attributed to parental genotypes. In its simplest form, it is the phenotypic product of the interaction between the parental genotype and that genotype's environment as expressed in the next generation. Initiation of an environmentally induced parental effect begins when a parental genotype responds to an independently derived environmental stimulus; that parent then transmits information to its progeny via one of several possible pathways. Both the ability to respond to a stimulus and the transmission pathway may vary among genotypes. The product is the phenotypic modification of an offspring trait. The underlying genetic bases for the parental ability to respond to the environment and the process by which information is transmitted determine how long an environmentally induced effect persists across generations and how quickly and in what direction the effect evolves.

Because an environmentally induced parental effect reflects one phenotype within a range of offspring phenotypes produced by a range of parental environments, biologists have sometimes viewed these effects as manifestations of intergenerational (also called trans- and cross-generational) phenotypic plasticity (e.g., Lacey 1991; Mousseau and Dingle 1991b; Schmitt et al. 1992). Other biologists have referred to environmentally induced parental effects as intergenerational acclimation (e.g., LeRoi et al. 1994; Fox et al. 1995).

Environmentally induced parental effects are often more complex than I have described above. In reviewing the literature, Rossiter (1996) found a number of studies suggesting that in addition to genetically based differences in parental response to environmental influences, environmentally induced parental effects may manifest themselves to different degrees depending on offspring environment and offspring genotype. In quantitative genetic terms, the interaction terms $V_{G_mE_m}$, $V_{G_mE_mE_o}$, $V_{G_mE_o}$, and $V_{G_oE_m}$ could theoretically all contribute significantly to V_p, the phenotypic variance in offspring, where V_{G_m} is the genetically based maternal variance, V_{G_o} is the variance in offspring genotype, and V_{E_o}, is the variance in offspring environment (Rossiter 1996). Therefore, detecting the existence and documenting the bounds of any one environmentally induced parental effect can be difficult.

Quantifying an environmentally induced parental effect is made even more difficult because of at least three confounding processes whose results mimic parental effects. The first is gametophytic/gametic/embryonic selection (Stephenson et al., 1992; Mazer and Gorchov 1996; Rossiter 1996). Although these types of selection can alter mean offspring phenotype, they involve neither the response of a parent to an environmental stimulus nor the transfer of information from parent to offspring. Therefore, gametophytic/gametic/embryonic selection should be considered a phenomenon that is distinct from an environmentally induced parental effect.

The second confounding process is environmentally induced, repeatable, nonrandom mutation. These mutations are predictable in that they are repeatedly produced by exposure to some identifiable environmental stimulus in the parental generation. The mutation is then transmitted to the offspring. Examples of this type of mutation appear to be rare, but one example appears in flax. In some genotrophs, certain parental fertilizer regimes can cause DNA amplification and RFLP alterations that are transmitted to the offspring and subsequent generations (Schneeberger and Cullis 1991). A parental effect, by definition, includes modifications in gene activity but excludes changes in gene structure. Therefore, these repeatable, nonrandom mutations should be viewed as distinct from parental effects.

The third confounding process is the direct environmental influence on offspring ontogeny. In theory, after formation of an offspring zygote, the postzygotic environment could induce the offspring's maternal parent to modify offspring phenotype. Alternatively, the environment could directly affect embryonic development. The latter is not strictly an environmentally induced parental effect because it is an intragenerational rather than an

intergenerational phenomenon (Lacey 1991). Therefore, direct environmentally induced ontogenic changes also should not be treated as parental effects.

In principle, the boundaries between an environmentally induced parental effect and these three confounding processes are clear; in practice, however, they may be fuzzy. Differentiating between a parental effect and gametophytidgametic/embryonic selection may require restricting experimental studies to homozygous lines (Mazer and Gorchov 1996), as Durrant (1962) has done with flax, or to a single genotype, as LeRoi et al. (1994) have done with *Escherichia coli*. Showing that a postzygotic effect is truly intergenerational requires demonstrating that a parent mediates the effect, for example, through hormones (Mousseau and Dingle 1991a) or oviposition site (Fox et al. 1997), which are maternal in origin, or by demonstrating that the phenotypic effect is transmitted to the offspring's offspring (e.g., Durrant 1962; Miao et al. 1991; Platenkamp and Shaw 1993; Case et al. 1996). Distinguishing between gene activity and structure may require struotural analyses of DNA, and even that may not distinguish structural changes environmentally induced by modification of regulatory genes, for example, through methylation or demethylation. Therefore, differentiating among these three confounding processes and environmentally induced parental effects may be logistically difficult, costly, and time-consuming, as evidenced by the many reports that do not attempt to do so.

For these reasons, it seems useful to distinguish between environmentally induced parental effects in a broad sense and environmentally induced effects in a narrow sense (figure 4.1). Environmentally induced parental effects in the broad sense include gametophytic/gametic/embryonic selection; environmentally induced, repeatable, nonrandom mutations; and direct environmently induced changes in offspring ontogeny. Environmentally induced effects in the narriow do not. Both groups of effects may influence the course of evolution in a population; both may be adaptive or maladaptive. In the rest of this essay, however, 1 will focus on the narrow sense of environmentally induced parental effects.

4.3 Classes of Environmentally Induced Parental Effects

The literature suggests that there are at least three classes of environmentally induced parental effects in the narrow sense. These classes differ from each other regarding phenotypic and environmental covariances across generations (figure 4.2). Class A effects include those produced by an environmental signal that alters the phenotype *j* of trait *B* in the parental generation r; this phenotype, $P_{j^B p}$, then induces phenotype P_i in trait *A* in the offspring generation *t* + 1 (figure 4.2). Thus, the induced parental phenotype is not transmitted aoross generations, and the phenotype of trait *A* in the offspring generation does not covary with the phenotype of trait *A* in the parental generation; rather $P_{iA_{t+1}}$, covaries with $P_{j^B c}$. The covariance may be positive or negative. $P_{iA_{t+1}}$ and the covariance between traits *A* and *B* are expected to disappear at the end of the offspring generation in the absence of environmental reinforcement, that is, unless the original environmental signal reappears independently of the parental effect in subsequent generations. Examples of Class A effects are the maternal con trot of seed germination and the maternal control of offspring sex through choice of oviposition site. Regarding seed germination, the environment may alter the maternally derived seed coat, which then influences germination (e.g., Dome 1981). In turtles, maternal decisions about where to lay eggs influence the sex ratio of her hatchlings (Roosenburg 1996).

Class B effects are those effects produced by an environmental signal that alters the phenotypic expression of trait A in the parental generation. This phenotype, P_{iA_t}, is then transmitted to and expressed in the offspring such that P_{iA_t} and $P_{iA_{t+1}}$ covary (figure 4.2). Theoretically, the phenotypes could positively or negatively covary; however, it is easier to find empirical examples showing positive covariance, which intuitively seems more likely. For example, regarding body size, abundant resources result in larger parents, who then produce larger offspring. Another example of Class B effects is the transmission of cultural beliefs. In the absence of environmental reinforcement, Class B effects should decay after the offspring generation, although the number of generations needed for total decay may vary among genotypes and likely depends on the transmission pathway(s) involved.

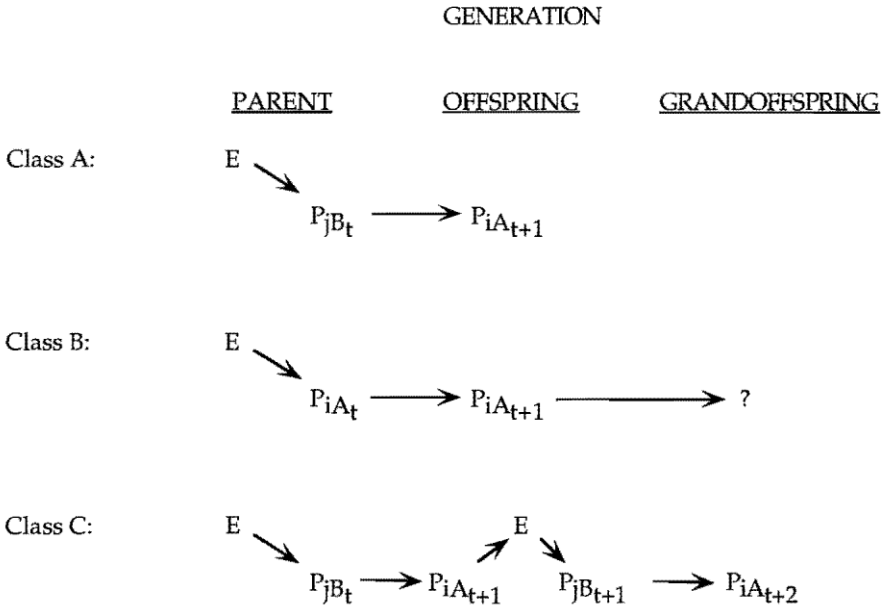

Figure 4.2 Three classes of environmentally induced parental effects, where E = the environmental stimulus, P_{iA} = phenotypic state i for trait A, P_{jB} = phenotypic state j for trait B, and t = generation. The question mark indicates that sometimes Class B effects persist past the offspring generation. See section 4.3 for further explanation.

Class C effects are similar to Class A effects in the parental and offspring generations except that phenotype $P_{iA_{t+1}}$ causes the environmental signal that was first observed in the parental generation to reappear in the offspring generation (figure 4.2). This reappearance induces the reappearance of P_{jB} in the offspring generation, which then induces phenotype P_{iA} in the grandoffspring. Thus, the phenotypes for traits A and B and the environmental states that induced these phenotypes all positively covary across multiple generations. P_{jA} and P_{jB} both reappear in multiple generations because they perpetuate the environmental signal that induces the parental effect (figure 4.1). I must admit that I have found no examples of a Class C effect, However, it is not hard to envision one that involves the photoperiodic control of seed germination or insect diapause. For example. postzygotic photoperiod, that is, the photoperiod during seed development on the maternal parent, is known to influence subsequent seed germination in many plant species (e.g.,, see reviews by Roach and Wulff 1987; Gutterman 1992; Wulff 1995). Also, germination time can influence flowering time in some species (e.g., Arthur et al. 1973; McIntyre and Best 1978). Therefore, it is conceivable that seeds maturing under a particular photoperiod will germinate and consequently flower at a particular time during the next growing season, which causes the same photoperiodic regime to reappear during the development of seeds constituting the next generation. Such a scenario could lead to the evolutíon of populations that are polymorphic for germination and flowering times, as is *Arabidopsis thaliana* (e.g., see review by Rathcke and Lacey)985).

In describing Classes A—C, I have assumed that the influences of offspring genotype and offspring environment are negligible, with the exception of the environmental signal in Class C effects. If these influences are not negligible, then the covariances may deviate from those discussed.

Also, I have described these classes in terms of the "transmissibility" and not the "inheritance" of phenotypes from one generation to the next. I have done this to distinguish between the passage of a phenotype across generations and the pathway by which the phenotype is passed. Words related to "inheritance" have been used in several ways in reports addressing parental effects. For example, "inherited " and "maternal inheritance" have been used very inclusively to embrace extranuclear inheritance and most environmentally induced parental effects, as I have defined them, regardless of whether or not a phenotype is transmítted across generations (e.g., Kirkpatrick and Lande 1989; Rossiter 1996). Others have used "inherited" and "heritable" to indicate the persistence of a particular phenotypic effect across generations (e.g., Hill 1965; Case et al.1996). Geneticists

have severely restricted the use of "inheritance" to the transmission of gene modifications across generations, which includes structural and functional modifications. I have taken the geneticists' approach. Thus, an effect may be transmissible across generations but not necessarily heritable, or inherited. Only if the transmissíon pathway involves gene modification is the effect heritable.

4.4 Adaptive Environmentally Induced Parental Effects

In describing the three classes of environmentally induced parental effects, I have made no assumptions about the ecological or evolutionary consequences of the effects. The classes are purely descriptive. In principle, they can embrace adaptive and/or maladaptive effects. We can, therefore, ask if adaptive or maladaptive effects characterize the classes and if one class of effects is more likely to be adaptive than another class. To address these questions, however, we must first agree on what it means for an environmental effect to be "adaptive."

An environmentally induced parental effect may be adaptive; it may also be an adaptation. Most evolutionary biologists agree that an adaptation is a product of evolution by natural selection (reviewed by Brandon 1990). By this definition, an environmentally induced parental effect is an adaptation only after it has been subjected to natural selection and has increased in frequency in a population. Ideally, evolutionary biologists would like five types of information to establish that a trait is an adaptation: (1) evidence that selection for the trait has occurred, (2) an ecological explanation for this selection, (3) evidence that the trait is heritable, (4) information about patterns of gene flow and selective environment in which the trait is found, and (5) evidence that the trait is a derived phylogenetic character (Brandon 1990). I have found no study that provides all this information for any environmentally induced parental effect in any organism.

The term "adaptive" lacks the historical component and is, therefore, more inclusive. An adaptive environmentally induced parental effect increases the probability of reproductive success of an offspring phenotype relative to others in a population. Brandon (1990) calls thís probability "adaptedness." To establish that an environmentally induced parental effect is adaptive requires only documentation that an environmental effect increases the probability of reproductive success of the phenotype exhibiting the effect over the probability of success of phenotypes not showing the effect. However, one must also establish that the observed phenotypic response in the offspring is truly an environmental effect in the strictest sense, which is not trivial. Thus far, I have found no plant study that has convincingly demonstrated adaptive environmentally induced parental effects. Studies of insect diapause provide the best body of evidence (e.g.. see reviews by Mousseau and Dingle 1991a,b). Many studies have demonstrated that seasonal changes in the parental environment induce changes in parental hormone levels that subsequently affect offspring diapause, all Class A effects. These effects are most likely adaptive. Also, Fox and collaborators (1997) have recently presented strong evidence for an adaptive environmentally induced maternal effect in the seed beetle *Stator limbatus*, another Class A effect. Females lay their eggs on multiple host plants and lay eggs of differing size depending on the host plant. They increase egg size on the host species for which an increase is more likely to improve offspring survivorship and decrease egg size on the species for which an increase has little effect on survivorship. This study demonstrates the existence of an environmentally induced effect, the fitness consequence of the effect, and the transmission of information from parent to offspring that is independent of offspring genotype.

Several studies have failed to detect adaptive effects (e.g., Via 1991; LeRoi et al. 1994; Fox et al. 1995; Bernardo 1996; Donohue and Schmitt, this volume). The experiment by LeRoi and his collaborators is particularly interesting because it shows both adaptive and maladaptive consequences of parental temperature effects. LeRoi et al. used one genotype of *E. coli* that was unable to use arabinose for growth (ara -) and an are mutant of that genotype. They grew both variants separately at both 32°C and 4L5°C for several generations. Then they created all combinations of the acclimated variants, grew the combinations at the two temperatures, and after some time estimated the population size of the variants in each combination. They observed that acclimation to 41.5°C reduced fitness when variants competed at 41.5°C, relative to acclimation to low temperature, regardless of the variant. However, when the variants were grown alone, acclimation to 41.5°C did improve subsequent fitness at 50°C. Thus, intergenerational acclimation, a Class B effect, was both advantageous and disadvantageous, albeit under different conditions.

Three aspects of an environmental fluctuation should influence the intensity of selection for an environmentally induced parental effect, that is, should influence its adaptiveness: amplitude, predictability, and length of the environmental fluctuation, or cycle in the case of predictable fluctuations, relative to the length of the life cycle of the organism involved. The greater the environmental fluctuation, the greater the selection for an environmentally induced effect (Rossiter 1996), regardless of type of effect (Class A or B). One might expect that where a predictable environmental cycle lasts for no more than two generations, there should be selection for short-lived effects, either Class A or B effects. The reason is that there should be selection against effects that persist into the third generation, at which time the adaptive parental information is likely to have become maladaptive. The environment will have changed. For example, any carryover effect of oviposition site in *S. limbatus to* the third generation is likely to lower the fitness advantage of the effect. If, however, the environmental cycle spans more than two generations, as it may for unicellular organisms, then we would expect that selection against the persistence of the effect would be relaxed, and persistence, or a slowing of the decay rate in Class B effects, might even be favored. For unicellular organisms that have rapid generation times, for example, bacteria, Class B effects might be common.

Conclusion

As more biologists have begun to study parental effects, the concept of a parental effect has become muddied. Here /have tried to define a parental effect and an environmentally induced parental effect in ways that are operational and generally useful. The literature indicates that there are at least two classes of environmentally induced parental effect in the narrow sense. These classes differ in the phenotypic and environmental covariances across generations. The relative abundance of classes A and B remains to be determined, as does the degree of adaptedness of these classes. Also, further research is needed to determine if one class applies to some taxonomic groups of organisms better than to others. Based on circumstantial evidence, I proposed a third class of environmentally induced parental effects (Class C). Further research will determine the reality of this class.

It has become clear that natural selection, mutation, and environmental influences on offspring ontogeny can produce effects that mimic environmentally induced parental effects, and teasing apart these processes is often difficult. Therefore, I have proposed distinguishing between narrowly and broadly defined environmentally induced parental effects. Environmentally induced parental effects in the broad sense include these confounding processes; effects narrowly defined do not. Distinguishing between narrow- and broad-sense environmentally induced parental effects serves several purposes. First, it emphasizes that one must be cautious about attributing empirically derived results to environmentally induced parental effects in the narrow sense. Second, I think that it may help us to design experiments that better address the question of the adaptiveness of environmentally induced parental effects strictly defined. Third, it may help us to design experiments that better address the evolutionary and ecological consequences of the confounding processes. Many more studies are needed to identify the environmental conditions under which environmentally induced parental effects in the narrow sense and these confounding processes are adaptive.

Acknowledgments

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References:

Arnold, S. J. 1994. Multivariate inheritance and evolution: A review of concepts. Pp. 17 -48 in C. R. B. Boalce (ed,), Quantitative Genetic Studies of Behavioral Evolution. University of Chicago Press, Chicago.

Arthur, A. E., J. S. Gale, and M. J. Lawrence. 1973. Variation in wild populations of *Papaver dubium.* VII. Germination time. Heredity 30:189-197.

Bernardo, J. 1996. Maternal effects in animal ecology. Am. Zool. 36:83-105.

Boggs, C. L. 1995. Male nuptial gifts: Phenotypic consequences and evolutionary implications. Pp. 215-242 in S. R. Leather and J. Hardie (eds.), Insect Reproduction. CRC Press, New York.

Boyd, R., and P.l Richerson. 1985. Culture and the Evolutionary Process. University of Chicago Press, Chicago.

Brandon, R. N, 1990. Adaptation and Environment, Princeton University Press, Princton, N.J.

Carrière, Y. 1994. Evolution of phenotypic variance: Non-Mendelian parental influences on phenotypic and genotypic components of life-history traits in a generalist herbivore. Heredity 72:420-430.

Case, A. L., E. P. Lacey, and R. G. Hopkins. 1996. Parental effects in *Plantago lanceolata* L. II. Manipulation of grandparental temperature and parental flowering time. Heredity T6:287-295.

Cheverud, J. M. 1984. Evolution by kin selection: A quantitative genetic class illustrated by maternal performance in mice. Evolution 38:766-777.

Clutton-Brock, T. H. 1991. The Evolution of Parental Care. Princeton University Press, Princeton, N.J.

Cowley, D. E., and W. R. Atchley. 1992. Quantitative genetic classes for development, epigenetic selection, and phenotypic evolution. Evolution 46:495-518.

Dorne, A. (1981) Variation in seed germination inhibition of *Chenopodium bonus-henricus* in relation to altitude of plant growth. Can. J. Bot. 59:1893-1901.

Durrant, A. 1962. The environmental induction of heritable change in *Linum.* Heredity 17:27-61.

Fox, C. W., K. J. Waddell, and T. A. Mousseau. 1995. Parental host plant affects offspring life histories in a seed beetle. Ecology 76:402-411.

Fox, C. W., M. S. Thakar, and T. A. Mousseau. 1997. Egg size plasticity in a seed beetle: An adaptive maternal effect. Am. Nat. 149:149-163.

Galloway, L. F. 1995. Response to natural environmental heterogeneity: Maternal effects and selection on life-history characters and plasticities in *Mimulus guttatus.* Evolution 49:1095-1107.

Giesel, J. T. 1986, Effects of parental photoperiod regime on progeny development time in *Drosophila simulans.* Evolution 40:649-65L

Giesel, J. T. 1988. Effects of parental photoperiod on development time and density sensitivity of progeny in *Drosophila melanogaster.* Evolution 42:1348-1350.

Gutterman, Y. 1992. Maternal effects on seeds during development. Pp. 27 -59 in M. Fenner (ed.), Seeds: The Ecology of Regeneration in Plant Communities. CAB International, Wallingford.

Hill, J. 1965. Environmental induction of heritable changes in *Nicotiana rustica.* Nature 207:732-734.

Jablonka, E., and M. J, Lamb. 1995. Epigenetic Inheritance and Evolution: The Lamarckian Dimension. Oxford University Press, Oxford.

Kirkpatrick, M., and R. Lande. 1989. The evolution of maternal characters. Evolution 43:485-50I

Lacey, E. P. 1991. Parental effects on life-history traits in plants. Pp. 735-744 in E. C. Dudley (ed.), The Unity of Evolutionary Biology. Dioscorides Press, Portland, Ore.

Lacey, E. P. 1996. Parental effects in *Plantago lanceolata.* L. I. A growth chamber experiment to examine pre- and post-zygotic temperature effects. Evolution 50:865-878.

LeRoi, A. M., A. F. Bennett, and R. E. Lenski. 1994. Temperature acclimation and competitive fitness: An experimental test of the beneficial acclimation assumption. Proc. Natl. Acad. Sci. USA 91:1917-1921.

Mather, K., and J. L. links. 1971. *Biometrical Genetics.* Chapman and Hall, New York.

Matzke, M., and A. J. M. Matzke. 1993. Genomic imprinting in plants: Parental effects and trans-inactivation phenomena. Annu. Rev. Plant Physiol. Plant Mol. Biol. 44:53-76.

Mazer, S. J., and D. L. Gorchov. 1996. Parental effects on progeny phenotype in plants: Distinguishing genetic and environmental causes. *Evolution* 50:44-53.

McIntyre, G. I., and K. F. Best. 1978. Studies on the flowering of *Thlaspi arvense* L. IV. Genetic and ecological differences between early- and late-flowering strains. Botan. Gaz. 139:190-195.

Miao, S. L., F. A. Bazzaz, and R. B. Primack. 1991. Persistence of maternal nutrient effects in *Plantago major:* The third generation. Ecology 72:1634-1642.

Mousseau, T. A., and H. Dingle. 1991a. Maternal effects in insect life histories. Annu. Rev. Ecol. Syst. 36:511-534.

Mousseau, T. A., and H. Dingle. 1991b. Maternal effects in insects: Examples, constraints, and geographic variation. Pp. 745-761 in E. C. Dudley (ed.), The Unity of Evolutionary Biology. Dioscorides Press, Portland, Ore.

Flatenkamp, G. A. J., and R. G. Shaw. 1993. Environmental and genetic maternal effects on seed characters in *Nemophila menziesii.* Evolution 47:540-555.

Rathcke, B., and E. P. Lacey.)985, Phenological patterns of terrestrial plants. Annu. Rev, Ecol. Syst. 16:179-214,

Reznick, D. 1991. Maternal effects in fish life histories. Pp. 780-793 in E. C. Dudley (ed.), The Unity of Evolutionary Biology. Dioscorides Press, Portland, Ore.

Riska, B., J. J. Rutledge, and W. R. Atchley. 1985. Covariance between direct and maternal genetic effects in mice, with a class of persistent environmental influences. Genet. Res. 45:287-297.

Roach, D. A., and R. Wulff. 1987. Maternal effects in plants: Evidence and ecological and evolutionary significance. Annu. Rev. Ecol. Syst. 18:209-235.

Roosenburg, W. M. 1996. Maternal condition and nest site choice: An alternative for the maintenance of environmental sex determination. Am. Zool. 36:157-168.

Rossiter, M. C. 1996. Incidence and consequences of inherited environmentally induced parental effects. Annu. Rev. Ecol. Syst. 27:451-476.

Rowe, J. S. 1964. Environmental preconditioning, with special reference to forestry. Ecology 45:399-403.

Schmitt, J., J. Niles, and R. D. Wulff, 1992. Norms of reaction of seed traits to maternal environments in *Plantago lanceolata.* Am. Nat. 139:451-466.

Schneeberger, R. G., and C. A. Cullis. 1991. Specific DNA alterations associated with the environmental induction of heritable changes in flax. Genetics 128:619-630.

Sinervo, B. 1991. Experimental and comparative analyses of egg size in lizards: Constraints on the adaptive evolution of maternal investment per offspring. Pp. 725-734 in E.C. Dudley (ed.), The Unity of Evolutionary Biology. Dioscorides Press. Portland, Ore.

Solter, D. 1988. Differential imprinting and expression of maternal and paternal genomes. Annu. Rev. Genet. 22:126-148.

Stephenson, A. G., T. C. Lau, M. Quesada, and J. A. Winsor. 1992. Faotors that affect pollen performance. Pp. 119-136 in R. Wyatt (ed.), Ecology and Evolution of Plant Reproduction. Chapman and Hall, New York.

Surani, M. A. 1991. Genoa& imprinting: Developmental significance and molecular mechamism. Curr. Opin. Genet. Dev. 1: 241-246.

Via, S. 1991. Specialized host plant performance of pea aphid clones is not altered by experience. Ecology 72:1420-1427.

Wulff, R. D. 1995. Environmental maternal effects on seed quality and germination. Pp, 491-505 in J. Kigel and G. Galili (eds.), Seed Development and Germination. Dekker, New York.