INCIDENCE AND CONSEQUENCES OF INHERITED ENVIRONMENTAL EFFECTS

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ABSTRACT
Inherited environmental effects are those components of the phenotype that are derived from either parent, apart from nuclear genes. Inherited environmental effects arise as the product of parental genes and the parental environment, or their interaction, and can include contributions that reflect the abiotic, nutritional, and other ecological features of a parental environment. Separating the impact of inherited environmental effects from inherited genetic effects on offspring phenotype variation has been and continues to be a challenge. This complexity is represented in the presentation of a qualitative model that distinguishes the possible paths of nongenetic cross-generational transmission. This model serves as the framework for considering the nature, in published works, of what was actually measured. Empirical evidence of inherited environmental effects arising from these pathways is documented for a diversity of plant and animal taxa. From these results one can conclude that the impact of inherited environmental effects on offspring can be positive or negative depending on the nature of the contribution and the ecological context in which the offspring exists. Finally, there is a description of theoretical and experimental efforts to understand the consequences of parental effects relative to their impact on population dynamics, the expression of adaptive phenotype plasticity, and character evolution.

INTRODUCTION AND TERMINOLOGY
Inherited environmental effects include those components of an offspring’s phenotype that are derived from the parent, apart from the nuclear genes. This definition serves as the broad designation for the outcome of multiple cross-
generational processes described in the following section. In this review, you will see that inherited environmental effects are documented for species from a broad taxonomic range, and that the external parental environment often plays a significant role in the magnitude and nature of the expression of these effects. Moreover, most of the parental environmental variables known to contribute to inherited environmental effects are permanent components of species’ environments (e.g. seasonal features, density, or food and habitat quality); this fact suggests their sustained importance in ecological and evolutionary processes. Much of the work that addresses the complexity of transmission of inherited environmental effects uses quantitative genetic analysis. I hope to distill, but not to minimize, this complexity by including a general framework that provides a way to think about how previous environmental experience interacts with genes to alter individual and population responses in unexpected ways.

Since an array of terms are used for the processes involved in inherited environmental effects, I first define those used frequently. The most common and most variably defined are “maternal effects” and “paternal effects.” These designations include the inheritance of nuclear genes from the mother or father (84, 134, 211), the inheritance of cytoplasmic genes (mitochondria or plastids) from the mother or father (95, 134), or the transmission of information derived from parental quality or parental environment (11, 40, 116, 161). Sometimes these terms are used without distinguishing the source of impact on offspring phenotype. For example, the presence of a maternal or paternal effect is inferred when offspring from reciprocal crosses differ in mean phenotype (68, 150, 157, 184, 190).

The term “maternal genetic inheritance” or “maternal additive inheritance” is used when a component of the maternal effect is of genetic origin, that is, due to maternal performance genes expressed in the mother and received as an environmental component of offspring phenotype. After the maternal genetic effects are accounted for, the remainder of the inherited environmental effect is often called a “maternal environmental effect” (40, 50, 56, 160, 217, 218). When confirmation of nongenetic cross-generational transmission arises (e.g. from variation in the parental environment due to photoperiod or nutritional quality), the terms “environmentally based” maternal, paternal, or parental effects (69, 168) and “general environmental” and “specific environmental” maternal or paternal effects (180) are used, depending on the experimental design. Durrant (51) found that flax varieties grown under several nutrient regimes responded with changes in their own growth parameters and appeared to transmit these changes to their offspring in the next and later generations, a phenomenon he called maternal “conditioning.”

Inherited environmental effects that extend beyond one offspring generation are called “permanent” (218) or “persistent” (160) environmental effects. For
example, an environmental effect on a grandmother’s maternal performance influences the maternal performance of her daughter and thus affects phenotypic values in grandchildren, and so on (56). Multiple generation reverberation is also called “environmental inheritance” because the maternal performance phenotype of the mother can directly modify the maternal performance phenotype of the daughter, even in the absence of genetic heritability (160).

To facilitate discussion of the evolutionary impact of inherited environmental effects, Kirkpatrick & Lande (91) used the term “maternal inheritance” for the transmission of non-Mendelian contributions from parent to offspring, and “maternal selection” for the effect of this transmission on offspring fitness. Similarly, Lombardi (109) suggested that “maternal influence,” which is the equivalent of “maternal inheritance,” should refer to the source rather than to the phenotypic impact on offspring expressing inherited environmental effects.

**PROCESSES THAT PRODUCE INHERITED ENVIRONMENTAL EFFECTS**

The multiplicity of meanings and underlying assumptions encompassed by the term “maternal effects” likely arises from difficulties in (a) distinguishing inherited environmental effects from direct genetic effects (i.e. transmission of nuclear genes to offspring), and (b) determining the extent to which inherited environmental effects are mediated by the parental genotype. Some of this complexity is represented in Figure 1, which is based on a quantitative genetics model with inherited environmental effects and a variable offspring environment of Eisen & Saxton (53). This figure differs from their model by the addition of a variable parental environment, a condition often measured or manipulated in empirical studies of inherited environmental effects. To describe the processes involved in the production of inherited environmental effects, it is best to start with the offspring phenotype, then consider what sources may contribute to it.

In Figure 1, offspring phenotype $P_o$ may derive from any or all of eight sources shown. Since this figure represents the involvement of only one (either) parent, interactions between maternal and paternal sources (e.g. as occur in plant endosperm) are not addressed. The source of a contribution to offspring phenotype is indicated as genetic (G) or environmental (E), originating in the parental (m) or offspring (o) generation. The parental performance phenotype, $P_m(t)$, represents those traits related to the quality of parenting. $P_m$ arises from parental genes for performance traits, $G_m(t-1)$, whose expression can be modified by the parental environment, $E_m(t-1)$ or offspring environment, $E_o(t)$. 
Figure 1  The components of offspring phenotype \( (P_o) \) expressed in time \( t \), deriving from the direct contribution of nuclear genes by one parent \( (G_o) \), a time-lagged presentation of the parental environment \( (E_m) \), a time-lagged expression of parental performance genes \( (G_m) \) and their interactions with the parental environment to produce the parental performance phenotype \( (P_m) \), plus the offspring’s own environment \( (E_o) \). For simplicity of presentation, \( G \) indicates additive genetic effects with dominance and epistasis assumed to be negligible. The numbered sources indicate possible routes of contribution to the offspring phenotype; Source 8 is any genetic covariance \( (cov) \) between genes expressed in two generations such as \( cov G_m G_o \) or \( cov(G_m E_o)(G_o E_o) \); see text for full description of variables.

The contributions to offspring phenotype typically considered come from: source 1, the offspring genotype, \( V_{G_o} \), which arrives as some fraction (usually, but not always 1/2) of the parental genotype; source 2, the offspring environment, \( V_{E_o} \); and source 3, interaction between offspring genotype and offspring environment, \( V_{G_o E_o} \). Modification of offspring phenotype due to inherited environmental effects comes from sources 4 through 8: source 4, contribution of the parental performance phenotype to offspring phenotype due to parental performance genotype, \( V_{G_m} \) (4a); interaction between the parental performance genotype and parental environment, \( V_{G_m E_m} \) (4b); interaction between parental performance genotype and offspring environment, \( V_{G_m E_o} \) (4c); or interaction between parental performance genotype, parental environment, and offspring environment, \( V_{G_m E_m E_o} \) (4d); source 5, contribution of the parental environment,
INHERITED ENVIRONMENTAL EFFECTS

V_{Em}, source 6, the interaction between parental and offspring environment, V_{EmEo}, source 7, interaction between parental environment and offspring genotype, V_{GoEm}, and source 8, covariance (correlation) between parental performance genes expressed in the parental (t - 1) and in subsequent (t, t+1, ... ) generations. Here, covariance can be simple, \( \text{cov}(G_m, G_o) \), or associated with interaction effects, e.g. \( \text{cov}(G_m, E_o)(G_o, E_o) \). The contribution of these covariance components to offspring phenotype can be positive or negative, a feature of great consequence in interpreting experimental results and predicting the consequences of inherited environmental effects (6, 91, 160, 218).

There is empirical evidence for each of these sources, although a single experimental design seldom assesses all of them: Source 4a: References 98, 142, 180 (plants), 35 (rotifers), 153 (guppies), 6, 43 (mammals); source 4b: 38, 98, 142, 145, 180 (plants), 89 (insects), 35 (rotifers), 64 (daphnia), 9, 165 (rodents), 107 (fish); source 4d, specifically: 180 (inferred, plants), 107 (fish); sources 4c, 4d, or 6: 1, 60, 124, 125, 180, 182, 198, 220 (plants), 58, 59, 65, 66, 67a, 89, 92, 168, 212 (insects), 24 (daphnia), 20 (voles), 87 (frogs), 74, 107 (fish), 29 (cattle); source 5: 180 (plants), 78 (insects), 9 (rats); source 7: 98 (plants); source 8: 19 (insects), 6, 160, 192, 217 (mammals).

While this model can act as a guide to considering the processes involved in the expression of inherited environmental effects, an additional complexity must be recognized. It is often difficult to assess the presence, source, or magnitude of inherited environmental effects (a) when selection acts on the gametophyte, which means that selection acts on the parental genotype, a situation that can give the appearance of inherited environmental effects, or (b) when selection acts during the embryonic stage, a situation often difficult to measure (59, 194). Experiments can be designed to distinguish (60, 119a) or minimize (102, 103, 147, 173, 208) the confounding effects of selection on the measurement of inherited environmental effects.

HOW THE PARENTAL ENVIRONMENT PARTICIPATES

In addition to direct nuclear and cytoplasmic genetic contributions, parents can provide offspring with nutrients. This aspect of inherited environmental effects is the most extensively studied. Data on egg and seed quality often come from studies of parental resource allocation patterns and tradeoffs between propagule size and number, long-standing topics in the study of life-history evolution (12, 37, 76, 153, 183, 188, 191, 201, 210a). Tools and techniques for quantification and manipulation (e.g. 10) provide a tractable approach to studying this aspect of inherited environmental effects. In addition to nutrients, parents can provide offspring with a preregulated genome, defensive agents, symbionts, pathogens, toxins, hormones, enzymes, and cultural conditioning. Depending on the species, parental environmental contributions can be limited...
to gamete packaging (egg or seed production) or extended through prenatal and postnatal periods of care. What follows is a sampling of references that illustrate each type of environmental participation.

**Parental Imprinting**

The notion that the environment might determine regulation in a gametic genome was suggested by Giesel from work on *Drosophila* (61). He offered that a photoperiod-induced paternal effect on offspring development time might be attributed to “memory effects” in the male germline arising from changes in some aspect of gene regulation. Since that time, considerable work has been done on parental imprinting wherein the expression of a gene (or an entire parental genome) in the offspring varies according to its maternal or paternal origin (210b). It has been argued that DNA methylation (which determines the expression or repression of a gene) is the mechanism of regulatory modification (8, 115, 199). To support this argument, Barlow (8) developed a model that assumes that regulatory modifications occur during gametogenesis because that is the only period when maternal and paternal gametic genomes are separate and can be subjected to different influences. Since methylation patterns are heritable, it is reasonable to consider that any environmental influences on DNA methylation patterns in gametes could result in inherited environmental effects. In the absence of any empirical work, Jablonka & Lamb (80) developed a model wherein an environmental stimulus induces heritable chromatin modifications that are specific and predictable, and which might result in an adaptive response to the environmental stimulus.

**Nutritional Factors in the Parental Environment**

The availability of nutrients in the parental generation is known to influence offspring phenotype. For example, minerals such as iron and zinc are taken up from the parental diet and transmitted to eggs (insects) or developing embryos (rodents) (108, 202). In some insect species, the male provides the female with nuptial gifts such as prey items or products of his accessory glands (17) which provide energy sources (104, 114) and minerals (54, 141). While these gifts may be used by the female for her own needs, there have been numerous confirmations that the paternally derived materials end up in the egg itself (17).

In species with extended maternal care, the nutritional status of the mother during prenatal and postnatal care can influence offspring phenotype. In laboratory rats, the impact of alcohol intake by mothers on fetal development was modified by the quality of her nutrition during pregnancy, such that the negative effects of alcohol ingestion on offspring were greater when the mother had a low-protein diet (209). In wild squirrels, juveniles born to mothers who received supplemental food (sunflower seeds left daily at burrow entrances) were
28% heavier compared to offspring of untreated mothers (owing to faster growth versus longer development time), and this mass difference was maintained after juveniles left the natal burrow (205).

**Abiotic and Ecological Factors in the Parental Environment**

The parental thermal experience can influence cold acclimation of offspring in plants (145) and cold tolerance of offspring in *Drosophila* (213). Density of the parental population can influence yolk quality in quail (7) and early behavior of offspring in tent caterpillars (216). From correlations between temporal or spatial environmental heterogeneity and variable provisioning of eggs or seeds by a mother, it has been argued that variable provisions increase the probability of maternal fitness when habitat quality for emerging young is unpredictable (e.g. 47, 153).

**Defensive Agents**

Plant secondary compounds can be sequestered by either parent and passed on to offspring where they serve to defend offspring against natural enemies. Paternal contributions of plant-derived alkaloids are made to offspring in moths (52, 72), butterflies (28), and grasshoppers (22), and terpenoid contributions are made in moths (30).

**Symbionts**

Whether the transmission of symbionts across generations is a form of genetic or environmental inheritance depends on point of view (111). Beyond the familiar cases of mitochondria and chloroplast and other plastid transmission, other examples may be more pertinent to inherited environmental effects. For example, many marine and terrestrial animal species harbor vertically transmitted bacterial or algal symbionts. The influence of these symbionts on offspring vigor can range from mild to complete (31, 204). Cytoplasmically inherited bacteria can be transmitted to offspring from either parent (e.g. 27, 195). In some species, cytoplasmically inherited bacteria interfere with paternal chromosome incorporation into fertilized eggs and cause reproductive incompatibility (an example of selection on the parental genotype), or the bacteria prevent segregation of chromosomes in unfertilized eggs and cause parthenogenesis (196, 197). In these cases, normal sexual reproduction can be reinstated by treatment with antibiotics (23b, 32, 48, 156, 195). Given this potential for “curing,” the transmission of these symbionts through a lineage might be changed in response to the parental environment, for example, by a diet that includes secondary compounds from plants or fungi with molecules active against microbes.

**Parental Gene Products**

The parental performance phenotype is responsible for the passage of parental gene products such as enzymes or proteins (3, 4, 90, 177, 221), hormones
(77, 130, 136, 163), resistance factors (132), self-made defensive compounds (110), or parental mRNA that is essential for offspring development (usually during the early embryonic period) (13, 88, 155). Whether this transmission is subject to modification by the parental environment is less studied. For insects, the parental photoperiod modifies the hormones that are passed on to offspring in locusts and aphids (130). Kerver & van Delden (90) suggested that the parental alcohol experience in *Drosophila* (i.e. parental environment) may influence the quantity or quality of ADH (or its precursor) passed on. Based on comparisons of life history and physiology in viviparous fish, Lombardi (109) hypothesized that the maternal environmental experience can influence offspring phenotype through transfer of disease immunity factors and physiological conditioning of endocrine, osmoregulatory, and thermoregulatory capacity.

**Cultural Conditioning**

Cultural conditioning ranges from the choice of appropriate propagule release sites in limited care species to teaching/learning phenomena in species with extended parental care (41). For many species with relatively immobile early stages and no obvious parental care, choice of oviposition site is a component of the parental performance phenotype, a critical component that determines the first external environment encountered by offspring (148). Choice of oviposition site, an expression of the parental performance phenotype, can be influenced by the quality of the parental environment, for example, faunal composition of ponds (frogs, 149) and floral composition of fields (insects, 189). Postnatal conditioning results from parental behavior which can have both genetic and environmental components. For example, sound reception capacity in infant bats is at least partly determined by the sound reception phenotype of mothers, and the maternal reception phenotype is dictated, in part, by her own environmental experience (83). In birds, evidence from quantitative genetic analysis indicates that socially learned foraging-site fidelity is a component of the correlation between mother and offspring body size (101). Similarly, lambs show socially induced preference for the host plants eaten by their mothers (126), as do mice, even when the mother’s food is less palatable than other available food (207).

**Toxins and Pathogens**

When parental toxicity or infection alters the parental performance phenotype, inherited environmental effects can be expressed. This may or may not involve transmission of the toxin or pathogen itself. In mice, reciprocal crosses between strains characterized by high and low susceptibility to an encephalitis virus indicated that infection was under the influence of the maternal environment (i.e. parental performance phenotype), but not that of the offspring genotype (133).
In molluscs, infectious bacteria from parents were found associated with gametes (158). In the Indian meal moth, sublethal viral infection in either mother or father resulted in reduced egg production and hatchability (178). Heavy metals modify grasshoppers’ parental performance phenotype; heavy metals are also transmitted into eggs, the net effect of which is to modify offspring metabolism and growth (181). For vertebrates, Lewis (106) provided a comprehensive medical/occupational hazard reference on the adverse effects on the fitness of offspring of thousands of substances ingested by parents; substances include drugs, food additives, pesticides, metals, and natural plant products. The evidence of inherited environmental effects is enormous, and depending on the half-life of the ingested substance, reverberations across multiple generations are possible. For example, Swain (200) used data on the half-life of PCBs and the success of their transmission to human offspring through transplacental passage and postpartum nursing to predict that PCB transmission would continue for at least five generations based on the intake of just the original mother.

EMPirical evidence of inherited environmental effects comes chiefly from studies that determined whether there is a relationship between (a) a specific aspect of the external parental environment and offspring quality at the egg or seed stage, (b) a specific aspect of the external parental environment and postnatal offspring quality, or (c) parental performance phenotype and offspring quality. In addition, the presence of maternal or paternal effects has been (d) inferred from breeding experiments, although these lack information about sources of environmental variation in the parental generation, and (e) directly measured after premature separation of mothers and offspring by embryo transplant and cross-fostering.

Parental Environment and Offspring Quality at Egg or Seed Stage

Early fitness traits, particularly propagule quality, are those most likely to express inherited environmental effects (118, 130, 161, 162, 215). Propagule size, often correlated with the level of nutritional provisions supplied to offspring
(18, 119), can contribute to offspring fitness throughout development (119, 167, 169, 198). Egg or seed qualities are desirable traits for experimental work because it is often easiest to quantify an inherited environmental effect, particularly the parental environmental component, before the influence of the offspring environment dominates; some examples are given in Table 1.

**Parental Environment and Postnatal Offspring Quality**

For plants, invertebrates, and vertebrates, excellent demonstrations exist of the degree to which the environment in one generation can influence individual and population quality of the next generation. The examples in Table 2 come from experiments that confirm the presence and magnitude of inherited environmental effects on postnatal traits when the parental environment was manipulated or its natural variation accounted for. In most cases, the experimental designs prevented (or minimized) the possibility that offspring phenotypic patterns were due to selection during the parental generation. The abbreviation of experimental results seen in Table 2 bypasses the creativity and complexity of these experimental designs and analyses. (Original references contain full details; particularly recommended are 60, 79, 107, 124, 142, 180, 213.)

**Parental Performance Phenotype and Offspring Quality**

In contrast to the previous studies in which variation in the external parental environment was measured or manipulated, some studies demonstrate inherited environmental effects without accounting for the external parental environment. Some demonstrations are based on a correlation between the parental performance phenotype and offspring phenotype, after genetic contributions are taken into account. For example, a plant’s maternal provisions to the seed can

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**Table 1** Incidence of inherited environmental effects related to parents’ environmental experience, expressed in propagule features

<table>
<thead>
<tr>
<th>Reference</th>
<th>Organism</th>
<th>Parental environmental variable</th>
<th>Offspring trait with inherited environmental effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>39</td>
<td>Plants</td>
<td>temperature</td>
<td>seed weight, nutrient constitution, germinability</td>
</tr>
<tr>
<td>175</td>
<td>Insects</td>
<td>foliage phenolic levels</td>
<td>egg weight</td>
</tr>
<tr>
<td>174</td>
<td>Insects</td>
<td>host species</td>
<td>yolk provisions (mean &amp; variation around)</td>
</tr>
<tr>
<td>64</td>
<td>Daphnia</td>
<td>food rations</td>
<td>egg weight</td>
</tr>
<tr>
<td>152</td>
<td>Fish</td>
<td>food rations</td>
<td>size, fat reserves (allocation differed by species)</td>
</tr>
<tr>
<td>153</td>
<td>Fish</td>
<td>food rations</td>
<td>number, size, fat reserves</td>
</tr>
<tr>
<td>113</td>
<td>Fish</td>
<td>temperature</td>
<td>egg weight</td>
</tr>
<tr>
<td>137</td>
<td>Chickens</td>
<td>food rations</td>
<td>yolk quality</td>
</tr>
<tr>
<td>7</td>
<td>Quail</td>
<td>density</td>
<td>quantity, quality of yolk provisions</td>
</tr>
<tr>
<td>85</td>
<td>Frogs</td>
<td>temperature, food availability</td>
<td>egg size</td>
</tr>
</tbody>
</table>

*Variation in the parental environmental variable was accomplished through manipulation or measurement of natural environmental variation.*
provide an environment so favorable for early growth that inbreeding effects are temporarily masked (127, 219). Radiolabelling techniques have been used to document the incorporation of nongenetic ejaculate materials into insect eggs (112, 139, 140). In polychaetes, the quality of the offspring changes in terms of survival and juvenile size as a function of the mother’s age (25, 105). In echinoderms, experimental alteration of yolk provisions influences early developmental traits, the most interesting of which is the degree of dependence on exogenous food during the pelagic period, an aspect of a species ecology with implications for life-history evolution (121). In turtles, maternal ability to select a thermally appropriate nest site, relative to egg size, influences the time to reproduction in daughters (166). In birds, parental performance phenotype, in terms of foraging behavior, has a significant influence on offspring growth (146).

Other studies that examine the relationship between parental performance phenotype and offspring quality are based on inference from population genetics principles using reciprocal cross designs, sibling analysis, and parent-offspring regression: (215—plants); (143, 214—insects); (203b—frogs); (144, 179—birds); (21, 71—wild rodents).

Embryo transplantation or cross-fostering experiments have been used to assess the relative contributions of temporally disjunct aspects of the parental performance phenotype on offspring phenotype. For example, Cowley et al (44) showed that regardless of offspring genotype, mice transferred as embryos to the uterus of mothers from an inbred line with large body size had greater body weights, longer tails, and higher growth rates compared to those transferred to mothers from an inbred line with small body size. Bolton (18) showed that although egg size was a component of the maternal performance phenotype that made significant contributions to chick fledgling success, parental care during nesting was of greater importance. Further documentation of inherited environmental effects stemming from prenatal and postnatal care can be found in (5, 34, 120—rodents) and (167—turtles).

ECOLOGICAL AND EVOLUTIONARY CONSEQUENCES

Inherited environmental effects influence survival and fecundity, particularly at the earliest stages of offspring life, a time when mortality and selection are often greatest (154, 215). By virtue of cross-generational transmission, inherited environmental effects may delay the impact of environmental factors on population growth and delay the opportunity for selection to act on the parental performance phenotype and the offspring genotype. These considerations have led to the development of hypotheses and models to predict the consequences
Table 2  Incidence of inherited environmental effects related to parents’ environmental experience, expressed through postnatal stages.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Organism</th>
<th>Parental environmental variablea</th>
<th>Offspring trait with inherited environmental effectsb,c</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>Plants</td>
<td>altitude</td>
<td>frost hardiness</td>
</tr>
<tr>
<td>142</td>
<td>competition intensity</td>
<td>seed weight, time to germination, dormancy</td>
<td></td>
</tr>
<tr>
<td>69</td>
<td>field vs lab</td>
<td>resin production</td>
<td></td>
</tr>
<tr>
<td>60</td>
<td>habitat productivity</td>
<td>flower production, plasticity (#)</td>
<td></td>
</tr>
<tr>
<td>193</td>
<td>microhizal infection or not</td>
<td>germination, vegetative &amp; reproductive yield</td>
<td></td>
</tr>
<tr>
<td>94</td>
<td>microhizal infection or not</td>
<td>development, nutrient content, reproductive allocation</td>
<td></td>
</tr>
<tr>
<td>124</td>
<td>nutrients, pulse intensity, timing</td>
<td>spike biomass for both species tested (#)</td>
<td></td>
</tr>
<tr>
<td>67b</td>
<td>photo period</td>
<td>seed germination</td>
<td></td>
</tr>
<tr>
<td>206</td>
<td>seasonality</td>
<td>salt tolerance variables</td>
<td></td>
</tr>
<tr>
<td>220</td>
<td>soil nutrients</td>
<td>seed weight, growth (#)</td>
<td></td>
</tr>
<tr>
<td>198</td>
<td>soil nutrients</td>
<td>seed and plant size (#)</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>soil nutrients</td>
<td>seed weight, survival &amp; size under nutrient stress</td>
<td></td>
</tr>
<tr>
<td>180</td>
<td>soil quality</td>
<td>seed weight, germination, harvest size (*) (#)</td>
<td></td>
</tr>
<tr>
<td>38</td>
<td>temperature</td>
<td>life history traits through adult stages (#)</td>
<td></td>
</tr>
<tr>
<td>145</td>
<td>temperature</td>
<td>seed weight, nutrient content, cold acclimation</td>
<td></td>
</tr>
<tr>
<td>98</td>
<td>temperature (pre- post zygotic)</td>
<td>seed weight, growth, onset of reproduction(*) (#)</td>
<td></td>
</tr>
<tr>
<td>78, 79</td>
<td>Insects</td>
<td>density at 3 developmental stages</td>
<td>degree of gregarization, coloration (*) (#)</td>
</tr>
<tr>
<td>123</td>
<td>density: pre- &amp; post-natal</td>
<td>wingedness</td>
<td></td>
</tr>
<tr>
<td>117</td>
<td>dietary cadmium</td>
<td>hatch success, enzyme activity</td>
<td></td>
</tr>
<tr>
<td>89</td>
<td>dietary iron availability</td>
<td>survival, development time &amp; stability</td>
<td></td>
</tr>
<tr>
<td>65</td>
<td>dietary quercitin or not</td>
<td>development time and weight (#)</td>
<td></td>
</tr>
<tr>
<td>36</td>
<td>field vs lab</td>
<td>development time, diapause, growth variables</td>
<td></td>
</tr>
<tr>
<td>82</td>
<td>field vs lab</td>
<td>heat resistance</td>
<td></td>
</tr>
</tbody>
</table>

(continued)

of inherited environmental effects on ecological and evolutionary processes. In short, it has been predicted that inherited environmental effects can influence population dynamics and character evolution (see below). Clearly, any significant changes in population dynamics and character evolution owing to inherited environmental effects will be dependent on ecological circumstance and the population’s evolutionary history (172).

The consequences of inherited environmental effects will depend on the transience or sustainability of both environmental input and organism response. Inherited environmental effects that are due to transient environmental input come from experiences that are unique or intermittent (e.g. toxin spill or drought). Sustained environmental input comes from experiences that are predictable such as photoperiod (61), average temperature (222), successive seasonal change (187, 206), or average competition (198). In some cases, the length of a species’ generation time and population structure will determine whether the environmental source of an inherited environmental effect is sustained or transient. For example, the experience of soil contamination by a toxin with a 50-year half-life will
likely be different for a hardwood species than for a soil invertebrate species. Which particular environmental experiences are involved in inherited environmental effects will be species-specific or population-specific. Further, the more variable the magnitude of a sustained environmental input, the more likely will be the adaptation for buffering through inherited environmental effects (33, 36, 46, 75, 107, 122, 129, 169).

Relative to an organism’s response, expression of an inherited environmental effect is transient if it is confined to one offspring generation (161). It is more difficult to decide when transient expression grades into sustained expression. However, multiple-generation reverberation from a single environmental input is known for plants and animals (38, 94, 125, 171, 181, 200, 212, 218), although the intensity usually wanes over time (56, 100). It is conceivable that an inherited environmental effect is initiated by a transient environmental input and sustained indefinitely. From a model of parental imprinting that includes

<table>
<thead>
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<th>Offspring trait with inherited environmental effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>58</td>
<td>host species</td>
<td>survival, development time, body size (*) (#)</td>
<td></td>
</tr>
<tr>
<td>59</td>
<td>host species</td>
<td>survival (#)</td>
<td></td>
</tr>
<tr>
<td>168</td>
<td>host species &amp; quality</td>
<td>pre-dispersal period, development time, pupal weight</td>
<td></td>
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<tr>
<td>181</td>
<td>mercury, cadmium in soil</td>
<td>hatch, development time, adult weight</td>
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<tr>
<td>57</td>
<td>nutritional quality</td>
<td>survival, development time</td>
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<tr>
<td>164</td>
<td>photoperiod</td>
<td>diapause propensity</td>
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<tr>
<td>99</td>
<td>photoperiod</td>
<td>body weight, temperature sensitivity</td>
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</tr>
<tr>
<td>66</td>
<td>photoperiod</td>
<td>age of first reproduction, initial fecundity (#)</td>
<td></td>
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<tr>
<td>92</td>
<td>photoperiod, temperature</td>
<td>diapause propensity</td>
<td></td>
</tr>
<tr>
<td>23a</td>
<td>photoperiod, temperature</td>
<td>diapause propensity</td>
<td></td>
</tr>
<tr>
<td>26</td>
<td>temp, daylength, maternal age</td>
<td>diapause propensity</td>
<td></td>
</tr>
<tr>
<td>57</td>
<td>temperature</td>
<td>age of first reproduction, body size (#)</td>
<td></td>
</tr>
<tr>
<td>223</td>
<td>temperature</td>
<td>body size, territorial success in sons</td>
<td></td>
</tr>
<tr>
<td>212</td>
<td>temperature</td>
<td>cold resistance for both species tested (*) (#)</td>
<td></td>
</tr>
<tr>
<td>213</td>
<td>temperature</td>
<td>cold resistance, development time, fecundity</td>
<td></td>
</tr>
<tr>
<td>45</td>
<td>temperature</td>
<td>heat tolerance</td>
<td></td>
</tr>
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<td>73</td>
<td>temperature</td>
<td>fecundity</td>
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<tr>
<td>35</td>
<td>Rotifiers density</td>
<td>proportion of mictic offspring</td>
<td></td>
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<tr>
<td>107</td>
<td>Fish salinity, food ration</td>
<td>time to hatch, hatch success (#)</td>
<td></td>
</tr>
<tr>
<td>187</td>
<td>Lizards seasonal variation</td>
<td>relationship between egg size &amp; survival</td>
<td></td>
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<tr>
<td>20</td>
<td>Rodents</td>
<td>field density growth rate, age of sexual maturity</td>
<td></td>
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<tr>
<td>9</td>
<td>toxin dose</td>
<td>survival, development, stress response</td>
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<tr>
<td>138</td>
<td>PCB exposure</td>
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<tr>
<td>205</td>
<td>food availability</td>
<td>initial &amp; subsequent body mass</td>
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<tr>
<td>55</td>
<td>pre-reproduc.nutritional quality</td>
<td>physiological variables</td>
<td></td>
</tr>
<tr>
<td>29</td>
<td>Cattle host species</td>
<td>pre-weaning size</td>
<td></td>
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</tbody>
</table>

See Footnote a in Table 1. (*) indicates paternal effect on at least one trait listed. (#) Indicates that offspring response was measured in more than 1 offspring environment.

Table 2 continued
the environmentally induced regulation of germline genes by DNA methylation, Jablonka & Lamb (80) inferred that inherited environmental effects could be responsible for apparent reversible mutations, thus drawing a fine line between genetic and inherited environmental effects. Along similar lines, Koch (93) argues that environmentally induced changes in bacterial gene regulation, including the activation of previously silent genes, are part of an adaptive plasticity response in the face of extreme environmental fluctuation.

Population Dynamics
Inherited environmental effects can generate a delay or acceleration in the response of a population to its environment. (And the environment can include the organism’s own phenotype e.g. parental body size or behavior.) It has been hypothesized that inherited environmental effects could act as a proximal source of time-lagged effects on population dynamics, with the potential to cause cycles or destabilization (either noncyclic outbreak or extinction), depending on other environmental parameters (21, 62, 81, 170, 171, 216). Relative to population ecology, a time lag occurs when the per capita rate of increase of a population is adjusted by the environment experienced in a previous generation(s). If the time-lagged component of population growth arises from a $t^2$ effect (environmental experience two generations prior) or greater, population cycles can occur (14). Inherited environmental effects can produce a time lag of $t^2$ or greater when, for example, they influence fecundity in adult offspring (e.g. 73, 168, 193). The capacity of inherited environmental effects to influence population dynamics is supported by a theoretical model that includes the influence of population quality expressed on a time delay (62), although the model is equally applicable to other proximal sources of time-lagged effects (15, 63). Empirical work, based on extrapolation of laboratory results to the field, supports the hypothesis that inherited environmental effects can influence population dynamics: (142—plants), (25—polychaetes), (21—lemmings), (20, 135—voles), (224—wild mice), (78, 79, 168, 171, 178—outbreak insect species).

Cross-Generational Phenotypic Plasticity and Life History Evolution
It has been hypothesized that as the environmental quality becomes less predictable or more heterogeneous, parents will adopt (i.e. selection will favor) a strategy of producing offspring of variable phenotype to increase the probability of reproductive success. For example, it has been predicted that inherited environmental effects will be important to the initial colonization ability of plants at infertile sites (124), and for early survival in insects with life-history features that preclude or reduce the ability of the parents to place eggs in locations that will predictably maximize the probability of future success (172).
Consequently, inherited environmental effects have been characterized as a form of bet-hedging wherein the production of greater phenotypic variation in offspring phenotype increases the likelihood of survival and reproduction in the face of environmental uncertainty (33, 36, 122, 185). With bet-hedging, a mother (or father) produces a range of offspring phenotypes, presumably to increase the probability that some subset of phenotypes will be appropriate for the environment encountered. The presence of such bet-hedging is interpreted to be a response to unpredictable components of the environment (such as the degree of annual divergence from seasonal means for temperature and rainfall). Inherited environmental effects have also been characterized as a form of plasticity if the parental environmental experience (e.g. photoperiod) produces an average adjustment of offspring phenotype which is directional and repeatable (e.g. 36, 203a), or if it produces a change in the magnitude of offspring phenotypic variation (e.g. 36, 169). Whether inherited environmental effects alter the magnitude of variance around the offspring mean phenotype or shift the offspring mean phenotype in a “programmed” direction, there is no certainty that the effect will be adaptive (i.e. increase the long-term fitness of a lineage), although a positive outcome is often seen (130). Bernardo (11) supplies some examples of nonadaptive maternal effects in viviparous species and cautions researchers about denoting maternal effects as a form of plasticity because the term contains an adaptive connotation for many.

Boggs (16) is working toward the integration of paternally based inherited environmental effects and life-history evolution. She developed a model to predict the consequences of paternal nuptial gifts on life-history evolution as it relates to female reproductive allocation of nutrients and the likelihood of sexual selection, mediated by paternal environmental contributions. Data from the literature corroborated the prediction that male nuptial contributions influenced female fecundity only when her feeding was restricted.

Character Evolution

Scientists have long been aware of the potential for inherited environmental effects to influence evolution. For example, a correlation between the genetic component of the parental performance phenotype and direct genetic effects (source 8 in Figure 1) can accelerate, retard, or change the direction of evolution depending on its magnitude and sign (49, 56, 70, 217, 218). Inherited environmental effects can also alter the correlation between offspring genotype and offspring phenotype (e.g. 36), and thus the response to selection. Evolution will be accelerated or retarded depending on whether sustained inherited environmental effects improve or diminish the relationship between offspring genotype and phenotype (159). Inherited environmental effects can also produce a delay of one or more generations in the response to selection on the
parental phenotype. When this time lag occurs, the trajectory of character evolution cannot be predicted as it would normally be, by defined aspects of selection pressure and inheritance (91, 100, 160). Kirkpatrick & Lande (91) developed a quantitative genetic model for the evolution of multiple traits under maternal inheritance and found that populations could continue to evolve for an indefinite number of generations after selection was relaxed, and that the consequences could be of greater magnitude (+ or −) or in a different direction than expected from simple Mendelian inheritance. Under frequency-dependence and maternal selection, wherein response to selection depends on a fitness function and the degree of resemblance between parents and offspring, evolution away from fitness optima is possible.

The results of empirical work lend support to the general prediction that inherited environmental effects can influence character evolution (128, 198, 213). In particular, from a study to estimate the genetic component of body size, development time, and propensity to diapause in an herbivorous insect, Carriére (36) discovered that inherited environmental effects can adjust both among- and within-family components of variance. He presents data supporting the contention that a change in the relative magnitude of these variance components (and thus a change in the heritability value) would diminish the potential for response to selection. Much the same conclusion was drawn by Galloway (60), who did an experiment on an annual herbaceous plant species from an environment with an unpredictable moisture level. The experiment was designed to determine how the parental environment influenced offspring phenotype—through selection in the parental generation or through transmission of inherited environmental effects. She found that both processes were involved and concluded from the data that the ability of environmentally based maternal effects to reduce phenotypic variation in offspring flower production would slow the rate of evolutionary change.

Groeters & Dingle (66) found that response to selection on reproductive traits in the milkweed bug was modified by a maternal effect; this resulted in a lack of opportunity to select strongly for long delays in the onset of reproduction for two successive generations. From this result they concluded that inherited environmental effects could constrain adaptation toward a genetic optimum but, by providing an override mechanism to the genetically based response of offspring to some environmental condition, could allow greater realization of a phenotypic optimum.

Lacey (98) subjected the parental generation of an herbaceous perennial plant species to several temperature regimes during the pre- and post-zygotic phases of seed production in order to quantify the relative contributions of genetic and inherited environmental effects at sequential stages of development. In the light of the results, she describes the potential for evolutionary change in flowering
time based on the direction of the parental effect (parental temperature hastens or slows offspring flower development) relative to the profile of a population’s genetic variation (frequency distribution of genes for flowering time).

Plantenkamp & Shaw (142) studied an annual plant species where the parental generation was reared under different competition regimes. They found, among other things, the presence of a $G_m \times E_m$ interaction (source 4b on Figure 1) for seed weight. From this and other results they discuss the role of inherited environmental effects on the evolution of reaction norms for seed weight and suggest that such reaction norms may contribute to the maintenance of maternally based genetic variation.

A reading of the papers cited in this section reveals that empirical demonstration of the impact of inherited environmental effects on evolution is challenging, particularly when there are temporal changes in the direction and magnitude of their expression (e.g. 179, 198), and the underlying biology is unknown (91).

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