EFECTOS MATERNALES

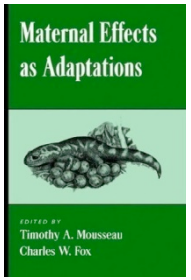
Definición

el fenotipo de un individuo viene determinado no sólo por su propio genotipo y las condiciones ambientales que experimenta durante su desarrollo sino también por el fenotipo y las condiciones ambientales de sus padres u otros conespecíficos.

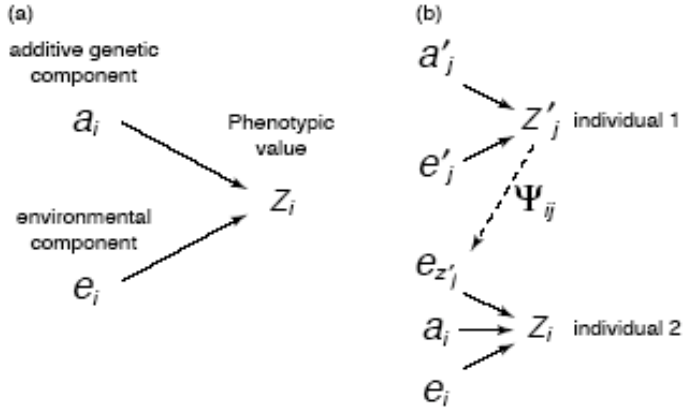
Efectos indirectos ambientales (IEE): Efectos parentales fruto del ambiente en el que se desarrolla los padres.

Efectos genéticos indirectos (IGE): fruto de características sociales o genéticas de las padres. Se llaman efectos genéticos indirectos porque los genes influencian el rasgo indirectamente.

Mousseau y Fox 1998. Maternal effects as adaptations



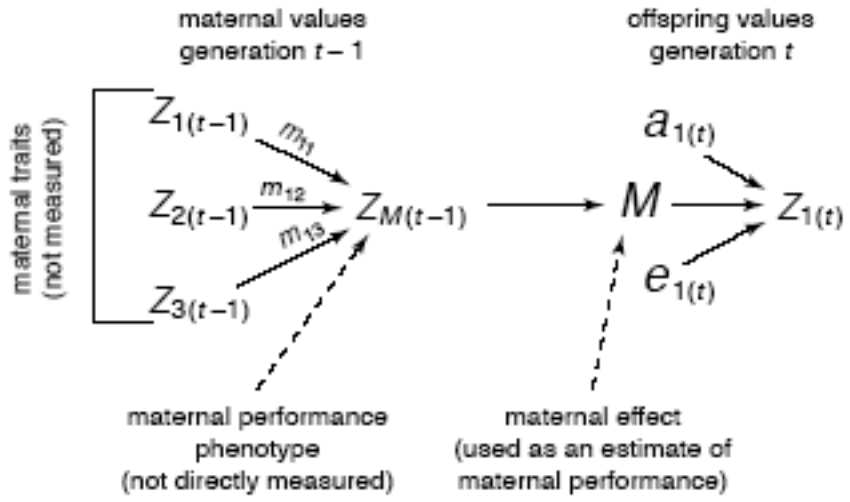
Box 2. The contribution of direct genetic effects, indirect genetic effects and environmental effects to the phenotype



(a) The standard quantitative genetic partitioning of the phenotype (z_i) into additive genetic (a_i) and environmental values (e_i). (b) The indirect genetic effect of phenotype z'_j in individual 1 on the phenotype z_i of individual 2. Primes indicate values for characteristics of individuals other than the focal individual. The effect of the environment provided by individual 1 on the expression of trait i in the focal individual, 2, is denoted $e'_{z'_j}$. ψ_{ij} is a coefficient that measures the effect that z'_j has on the expression of z_i (see Refs 6,7 for details). (c) The special case for maternal effects. Trait z_j expressed by the mother in the previous generation, $t-1$, affects the expression of trait z_i in the offspring in the current generation, t. The coefficient m_{ij} describes the degree to which the maternal trait z_j contributes to the expression of the offspring trait z_i (see Ref. 23).

Box 3. Performance characters (illustrated by maternal performance)

Shown are the three components contributing to the offspring phenotype: the direct genetic effect $a_{1(t)}$, the environmental effect $e_{1(t)}$ and the maternal effect M. The maternal effect is caused by maternal performance of the mother that is itself composed of the contributions of three maternal traits. The effect of the maternal trait on the expression of the offspring trait is given by m_{ij} where i is a trait in the offspring (in this case $i=1$) and j is the maternal trait ($j=1$, 2 or 3). Additional details are explained in Box 2.



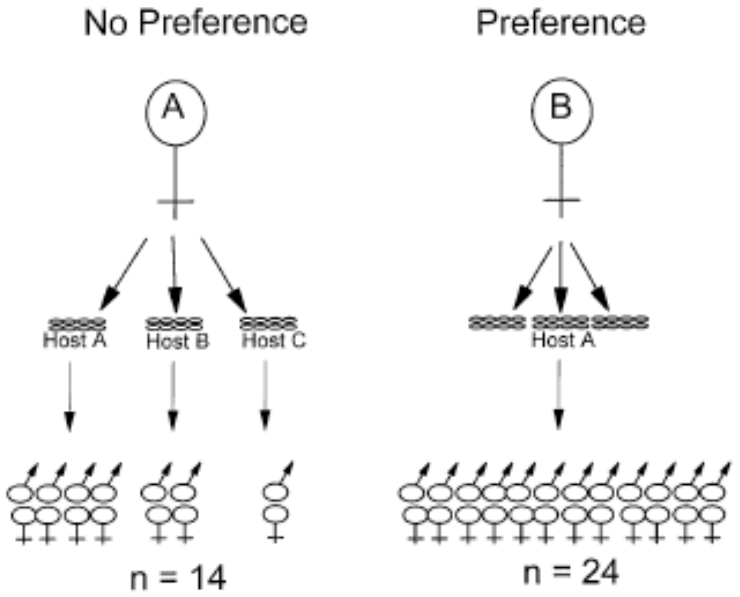
Cuando se expresan los efectos maternales

1-**Efectos maternales prezigóticos**: ocurre cuando el fenotipo de la madre, especialmente las condiciones nutricionales de la madre, afectan a la calidad y tamaño gamético.

2-**Efectos maternales postzigóticos prenatales**: ocurre debido a los efectos nutricionales de la madre durante el desarrollo embrionario o la maduración.

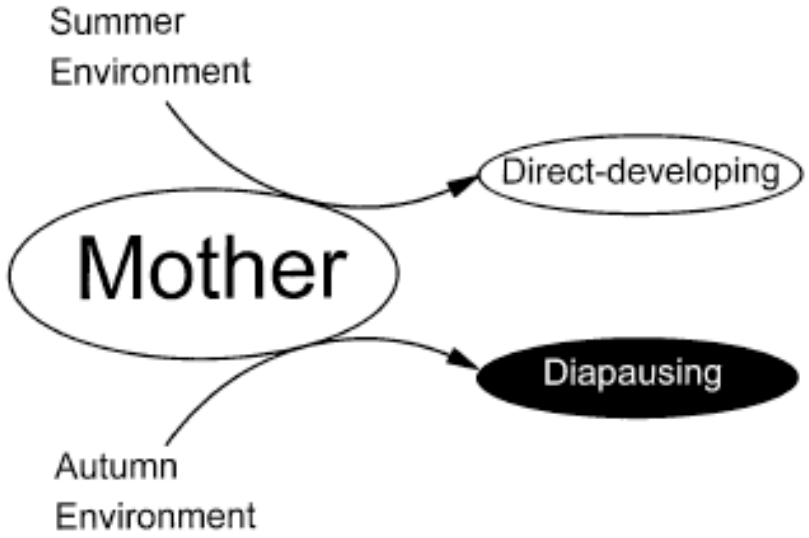
3-**Efectos maternales postzigóticos postnatales o postgerminativos**: ocurre cuando el cuidado maternal y otros factores similares afectan al fenotipo de los descendientes.

Box 3. Maternal host/mate preferences often influence offspring fitness



The figure illustrates the potential effects of maternal host or mate preference on maternal and offspring fitness. Variation in maternal preferences for hosts that vary in their suitability for growth and survival frequently result in maternal effects. Maternal host preferences are expected to evolve when offspring fitness varies with host because a female who places her eggs randomly among hosts (female A) will have lower fitness than a female with the ability to discern among potential hosts (female B). Relationships between maternal-host preference and offspring performance are frequently observed in host–parasite and plant–insect interactions[17]. Similarly, there is mounting evidence that females often show preferences for mates who positively affect offspring fitness in some way[22,40,43]. Females choosing to mate with high quality males often obtain resources (e.g. nuptial gifts and parental care) that are translated into increased offspring quality (e.g. increased size and/or increased survival), making female mate choice an adaptive maternal effect.

Box 2. Environmentally modulated transgenerational plasticity in offspring development



The figure illustrates the influence of maternal environment on offspring development. In many insects and plants, the expression of diapause or dormancy is regulated by maternal environmental experience[5,6]. In most cases, the photoperiod experienced by a mother will influence whether an offspring enters a diapausing or dormant state, although temperature and host availability have also been found to affect diapause. Often, the environmental cue to which females are receptive is a good predictor of future environmental conditions (i.e. short photoperiods and cooling temperatures are good indicators of an impending winter). In many species, a female will change from producing diapausing (or dormant) offspring to directly developing offspring (and vice versa) in response to a change in environmental conditions[5]. Similar responses are observed for maternal effects on flight and wing polymorphisms[8,14].

Table 1. A selected set of empirical examples to illustrate the breath that maternal effects can take, focusing on studies that have also investigated effects of variable maternal and/or offspring environment on maternal effects (note that environmental induction does not preclude a genetic basis).

Species	Offspring trait	Maternal trait	Environment-dependent plasticity	Reference
Daphnia pulicaria	Propensity for resting stage production	Reproductive mode	Maternal and offspring environment	Alekseev & Lambert (2001)
Daphnia cucullata	Inducible defences	Inducible defences	Maternal and offspring environment	Agrawal, Laforsch & Tollrian (1999)
Daphnia magna	Parasite resistance	Per offspring investment	Maternal and offspring environment	Mitchell & Read (2005)
Soil mite (*Sancasania berlesei*)	Hatching time, Traits at maturation	Fecundity, age	Maternal and offspring environment	Beckerman *et al.* (2006)
Burrower bug (*Sehirus cinctus*)	Solicitation pheromones	Provisioning	Offspring environment	Kölliker *et al.* (2006)
Soap berry bug (*Jadera haematoloma*)	Morphology	Host plant choice	Offspring environment	Carroll *et al.* (2001)
Drosophila serrata	Survival	Age, grandmothers age	Offspring environment	Hercus & Hoffmann (2000)
Yellow dungfly (*Scatophaga stercoraria*)	Life-history traits	Mating pattern	Maternal environment	Tregenza *et al.* (2003)
Three species of poeciliid fishes	Offspring size and fat content	Offspring size and number	Maternal environment	Reznick, Callahan & Llauredo (1996)
Brown trout (*Salmo trutta*)	Survival, growth	Propagule size	Offspring environment	Einum & Fleming (1999, 2000)
Fire-bellied toad (*Bombina orientalis*)	Growth	Propagule size	Maternal and offspring environment	Kaplan & King (1997), Kaplan & Phillips (2006)
Moor frog (*Rana arvalis*)	Survival, growth	Egg capsule Propagule size	Offspring environment	Räsänen *et al.* (2003b, 2005)
House finch (*Carpodacus mexicanus*)	Survival, growth	Sex-biased laying sequence Onset of incubation	Maternal and offspring environment	Badyaev *et al.* (2002)
Field vole (*Microtus agrestis*)	Growth	Sex allocation	Maternal environment	Koskela *et al.* (2004)

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Maternal effects and evolution at ecological time-scales

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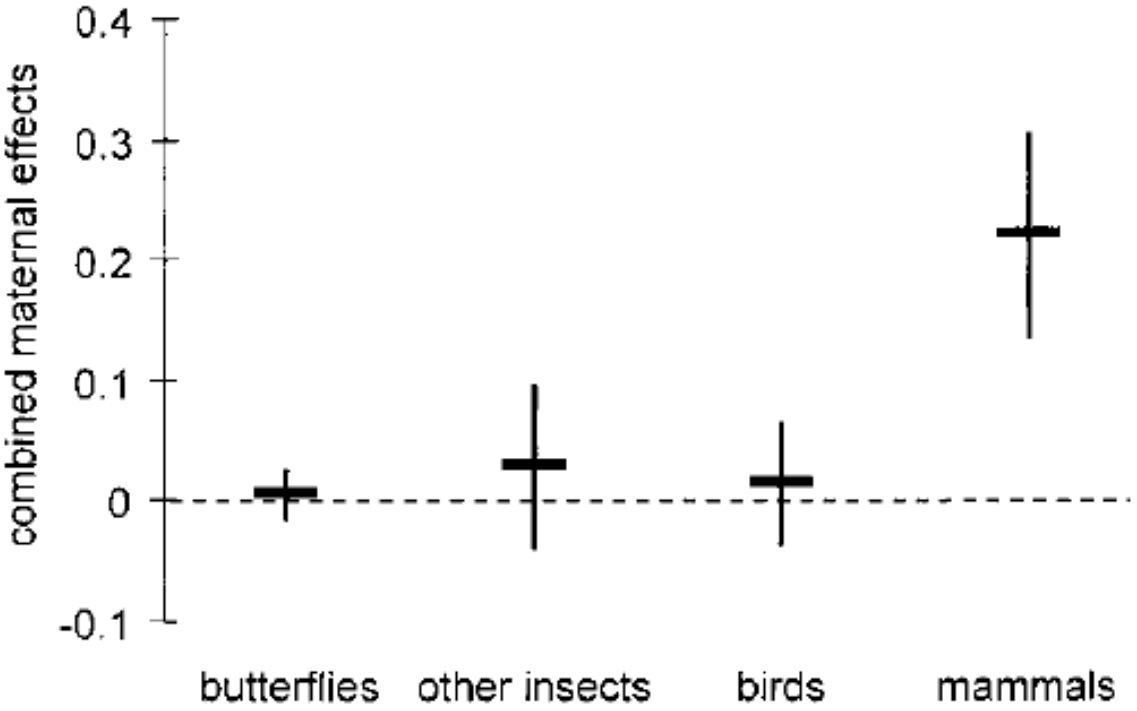
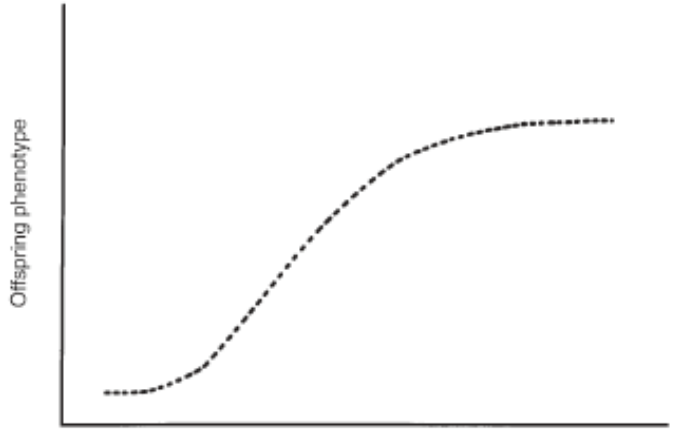


Figure 1. Comparison of average maternal effects between the examined four different animal groups. The horizontal bars denote the arithmetic mean, the vertical bars denote the standard error, and the hatched line indicates the expected value when maternal effects are absent.

Efectos maternales y Plasticidad fenotípica transgeneracional

Box 1. Maternal effects as transgenerational phenotypic plasticity

Many maternal effects can be visualized as a reaction norm describing the influence of maternal environment or behavior on an individual offspring's phenotype (as in the figure). Maternal effects occur when a mother's phenotype influences her offspring's phenotype independently of the female's genetic contributions to her offspring. Many maternal effects can be modeled as environmentally modulated transgenerational phenotypic plasticity, in which environmental variation (e.g. temperature, photoperiod and nutrients) experienced by mothers is translated into phenotypic variation in offspring. Similarly, maternal behavior (e.g. host choice, oviposition behavior and parental care) will often influence offspring phenotype and fitness.



In many cases, maternal effects can be visualized as reaction norms that extend across generations. For example, variation in maternal photoperiod often influences the expression of diapause in insects[5] and dormancy in plants[6], and the temperature of a nest chosen by many female reptiles can influence the sex of offspring[18,19]. Similarly, female choice of male secondary sexual characters can influence aspects of offspring fitness[12,22,40,43], and female host choice by herbivores and parasites can dramatically influence offspring growth and survival[17,24,36]. In addition, many aspects of female environmental experience and behavior can influence offspring fitness via effects on propagule (i.e. eggs or seeds) size[27,28,32,33,35,36] or through competitive interactions among offspring (i.e. clutch size effects)[23–25].

Efectos maternales y Plasticidad fenotípica transgeneracional

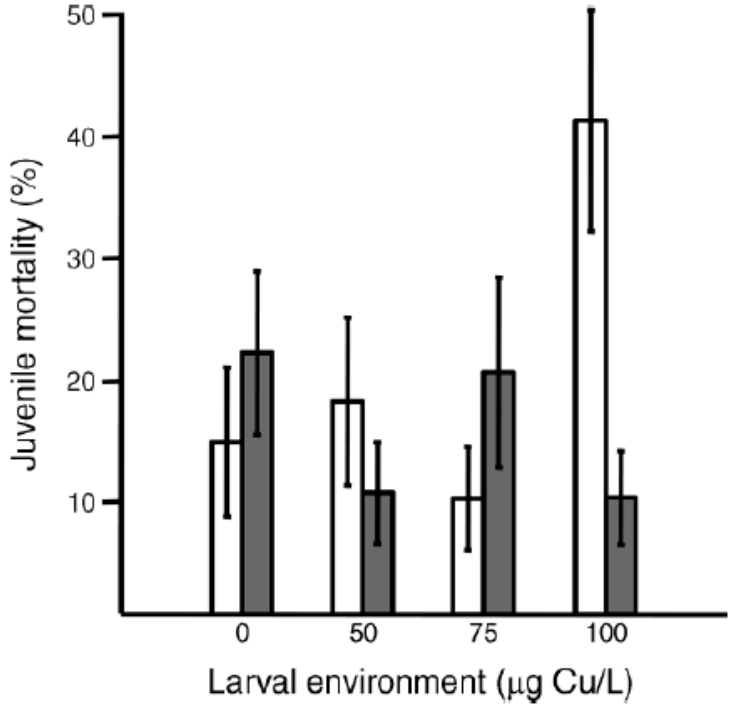


FIG. 3. Effect of maternal exposure history and larval environment on the mortality (mean ± SE) of *Bugula neritina* juveniles after 48 hours in the laboratory. Open bars indicate mortality of juveniles from toxicant-naïve mothers, and closed bars indicate mortality of juveniles from mothers exposed to 300 µg Cu/L a week before spawning. On the *x*-axis are the different environments that juveniles were exposed to as larvae (0 = control).

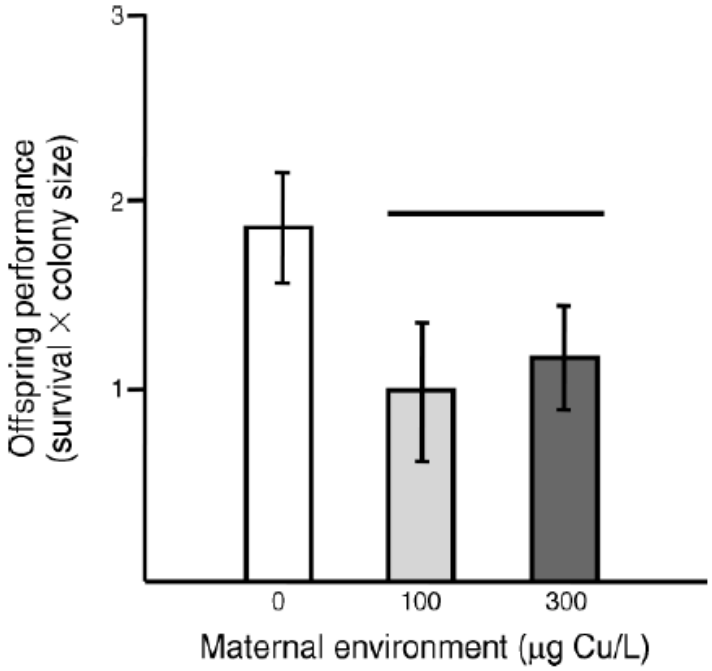


FIG. 4. Effect of maternal exposure history on the performance of *Bugula neritina* colonies after six weeks in the field. Bars represent performance (mean ± SE) of offspring from toxicant-naïve mothers (as indicated by open bars) and offspring from mothers that were exposed to either 100 (light-shaded bar) or 300 µg Cu/L (dark-shaded bar) a week before spawning. The horizontal bar indicates no significant difference between 100 and 300 µg Cu/L exposure histories using planned comparisons.

Relación heredabilidad-efectos maternales

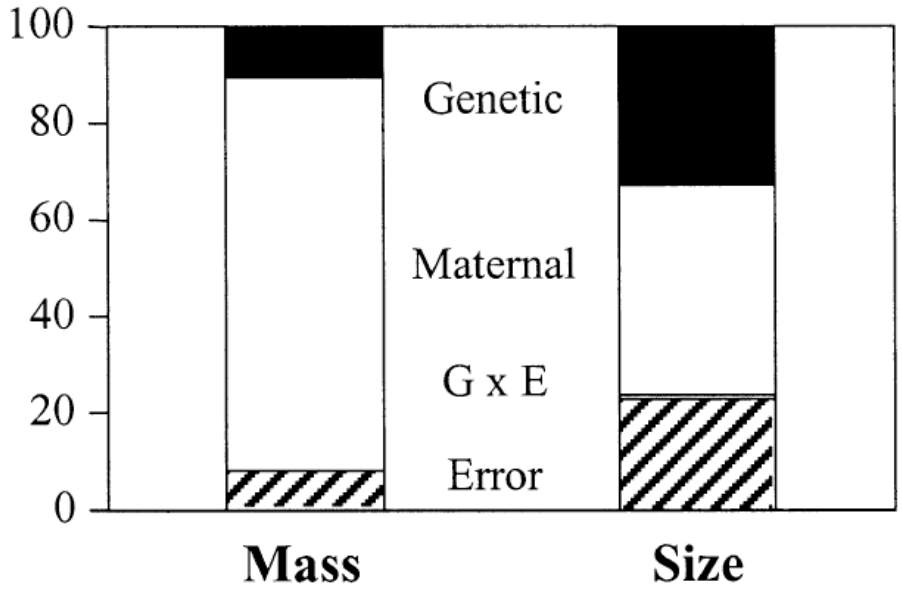


FIG. 1. Sources of variation as percentages of phenotypic variation in growth in body mass and body size in cross-fostered red squirrels. The black, white, gray, and hatched areas within each bar represent the percentage of total phenotypic variation within dyads that was due to genetic (V_{AO} assuming single paternity), maternal (V_M), interaction (genotype × environment, G × E), and error variances, respectively. Total phenotypic variation in growth of body mass and body size were 0.151 $(g/day)^2$ and 5.32×10^{-6} $(day)^{-2}$ respectively.

Relación heredabilidad-efectos maternales

TABLE 1. The potential for evolution and sources of variation in growth in body mass and growth in body size of nestling red squirrels (±SE) based on direct genetic effects alone (h^2) and including maternal genetic effects (h_t^2). Sources of variation for growth in body mass and size are reported as (g/day)^2 and $\times 10^{-6}$ (day^{-2}), respectively. V_{PO}, V_{AO}, V_M, and $Cov\ (A_O, A_M)$ were estimated from the cross-fostering experiment. V_{PO} represents the total phenotypic variance within dyads. V_{AM} was calculated as the product of the phenotypic variance due to maternal performance from the cross-fostering experiment (V_M) and the heritability of maternal performance (h_m^2; see Table 2). h_t^2 was calculated following Dickerson (1947). Standard errors were generated by jackknifing at the level of the dyad.

	Growth in body mass	Growth in body size
Total phenotypic variance (V_{PO})	0.151 ± 0.001	5.32 ± 0.04
Direct effects		
Direct genetic variance (V_{AO})	0.016 ± 0.0001	1.77 ± 0.01
Heritability (h^2)	0.10 ± 0.001	0.33 ± 0.005
Maternal effects		
Maternal variance (V_M)	0.123 ± 0.001	2.30 ± 0.04
Maternal genetic variance (V_{AM})	0.016 ± 0.0001	0.24 ± 0.004
Direct-maternal genetic covariance ($Cov\ [A_O, A_M]$)	0.020 ± 0.001	0.02 ± 0.06
Total heritability (h_t^2)	0.36 ± 0.01	0.36 ± 0.02

Relación heredabilidad-efectos maternales

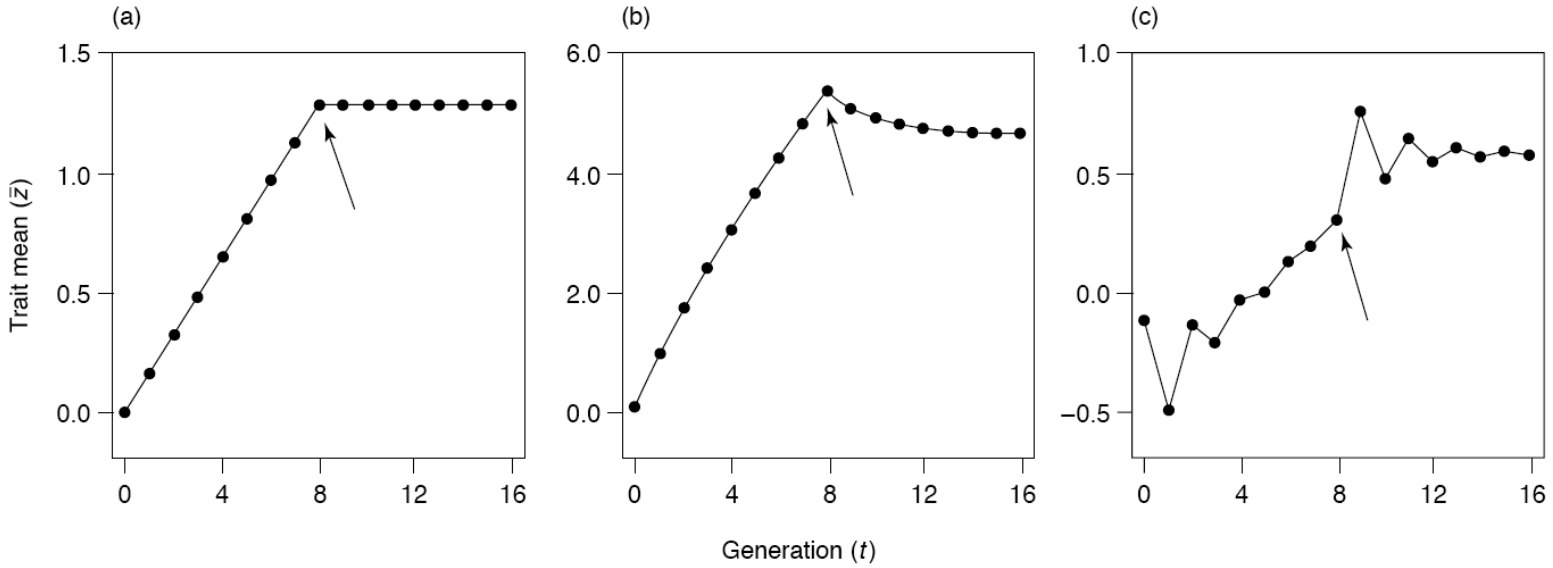


Fig. 1. Evolutionary trajectories showing the mean value of a maternal trait that affects the expression of the same trait in the offspring. Trajectories are calculated using the equation for the change in the mean of the character across one generation; $\Delta\bar{z}(t) = [C_{az} + mP]\beta_{(t)} + m\Delta\bar{z}_{(t-1)} - mP\beta_{(t-1)}$ (cf. Ref. 23, eqn 3), where P is the phenotypic variance of the trait, β is a directional selection gradient[22], and t refers to the current generation. All other symbols are given in the text. For all trajectories, $\beta = 0.4$, $G = 0.4$ and $E = 1.0$ (where E is the environmental variance); using these values, C_{az} is calculated as in the text. P can be calculated using eqn A14 in Ref. 23. Directional selection was applied for the first 8 generations. The generation where selection ceased is indicated by an arrow. (a) Shows the case for no maternal effect ($m = 0$), (b) shows the case for a positive maternal effect ($m = 0.6$), and (c) shows the case for a negative maternal effect ($m = -0.6$).

The thrifty phenotype as an adaptive maternal effect

Thrifty phenotype: in poor nutritional conditions, a pregnant female can modify the development of her unborn child such that it will be prepared for survival in an environment in which resources are likely to be short.

Epigenetics: The study of mitotically and/or meiotically heritable changes in gene function that cannot be explained by changes in DNA sequence

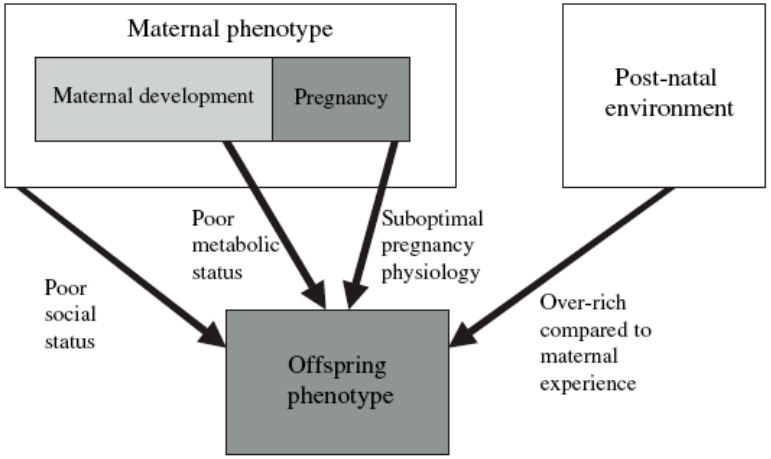


Fig. 4. Pathways through which the relationship between maternal phenotype and environmental quality may invoke adverse phenotype in the offspring. Low social status, abnormal metabolic status, and pregnancy physiological dysfunction may all compromise offspring development, while these effects may be exacerbated if the offspring subsequently encounters an environment different from that to which maternal phenotype is adapted. These routes are in addition to the direct maternal transmission of infectious disease.

Human diseases in adulthood are increasingly associated with growth patterns in early life, implicating early-life nutrition as the underlying mechanism. The thrifty phenotype hypothesis proposed that early-life metabolic adaptations promote survival, with the developing organism responding to cues of environmental quality by selecting an appropriate trajectory of growth. Recently, some authors have proposed that the thrifty phenotype is also adaptive in the longer-term, by preparing the organism for its likely adult environment. However, windows of plasticity close early during human development, and subsequent environmental changes may result in the selected trajectory becoming inappropriate, leading to adverse effects on health. This paradox generates uncertainty as to whether the thrifty phenotype is indeed adaptive for the offspring in humans. The thrifty phenotype should not be considered a dichotomous concept, rather it refers to the capacity of all offspring to respond to environmental information during early ontogenetic development. This article argues that the thrifty phenotype is the consequence of three different adaptive processes - niche construction, maternal effects, and developmental plasticity - all of which in humans are influenced by our large brains. While developmental plasticity represents an adaptation by the offspring, both niche construction and parental effects are subject to selection on parental rather than offspring fitness. The three processes also operate at different paces. Human offspring do not become net calories-producers until around 18 years of age, such that the high energy costs of the human brain are paid primarily by the mother, even after weaning. The evolutionary expansion of human brain volume occurred in environments characterised by high volatility, inducing strong selective pressure on maternal capacity to provision multiple offspring simultaneously. The thrifty phenotype is therefore best considered as a manipulation of offspring phenotype for the benefit of maternal fitness. The information that enters offspring phenotype during early development does not predict the likely future environment of the offspring, but rather reflects the mother's own developmental experience and the quality of the environment during her own maturation. Offspring growth trajectory thus becomes aligned with long-term maternal capacity to provision. In contemporary populations, the sensitivity of offspring development to maternal phenotype exposes the offspring to adverse effects, through four distinct pathways. The offspring may be exposed to (1) poor maternal metabolic control (e.g. gestational diabetes), (2) maternally derived toxins (e.g. maternal smoking), or (3) low maternal social status (e.g. small size). Adverse consequences of these effects may then be exacerbated by (4) exposure either to the "toxic" western environment in postnatal life, in which diet and physical activity levels are mismatched with metabolic experience in utero, or at the other extreme to famine. The rapid emergence of the epidemic of the metabolic syndrome in the 20th Century reflects the rapid acceleration in the pace of niche construction relative to the slower physiological combination of developmental plasticity and parental effects.

Wells, JCK 2007. *The thrifty phenotype as an adaptive maternal effect*. Biological Reviews 82: 143-172