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What is a paternal effect?

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Maternal effects are now universally recognised as a form of nongenetic parental influence on offspring but, until recently, paternal effects were regarded as an anomaly. Although it is now clear that paternal effects are both widespread and important, their proximate basis and evolutionary consequences have received little attention and remain poorly understood. In particular, because many paternal effects are mediated by maternal responses such as differential allocation, the boundary between paternal and maternal effects is sometimes blurred. We distinguish here three basic types of paternal effect and clarify the role of maternal responses in these effects. We also outline key questions that can serve as a road map for research on the proximate basis and evolutionary implications of paternal effects.

An unexpected source of heritable variation

Paternal effects (the influence of fathers on the features of their offspring via mechanisms other than the transmission of alleles) have long been regarded as a rare phenomenon confined to species exhibiting paternal care. However, a rapidly growing body of evidence now shows that such effects occur in a variety of organisms, can be mediated by cellular and physiological processes that characterise all sexually reproducing eukaryotic species, and affect a broad range of phenotypic traits in the next generation (reviewed in [1–5]).

The importance of paternal effects in evolutionary ecology derives from the fact that such effects represent a source of variation in phenotype and fitness. Theory suggests that paternal effects can have unique evolutionary consequences (e.g., [4,6,7]), and it is therefore important to identify paternal effects and distinguish them from other sources of variation. Nevertheless, whereas the nature and role of maternal effects has been examined by several authors [8–14], the distinct nature of paternal effects has received little consideration, and there is as yet no clear consensus on how paternal effects are to be defined, or differentiated from maternal effects. The lack of a clear framework for differentiating paternal effects from maternal effects is particularly evident in relation to a common class of paternal effects that are mediated by maternal responses. This is a potential source of confusion in many studies and, in particular, complicates the distinction between maternal differential allocation and paternal condition transfer.

We clarify here the definition of a paternal effect, distinguish several distinct routes of paternal influence on offspring, and show that paternal effects differ from maternal effects in fundamental ways. We also outline some key questions to guide research on paternal effects.

The nature of paternal effects

Parents contribute in many ways to the development of their offspring but, by conventional definition, a paternal (or maternal) effect can be said to occur when variation in the paternal (or maternal) genotype or phenotype is causally associated with variation in offspring phenotype, and this effect cannot be accounted for by offspring genotype [14]. It has been recognised for a long time that the causal link between parents and offspring –that forms the basis of all forms of heredity – is the transmission of some factor across generations [15]. By focusing on the nature of the transmitted factor, we can distinguish hereditary effects mediated by the transmission of genetic alleles (genetic inheritance) from effects mediated by the transmission of other factors (nongenetic inheritance, which encompasses parental effects in the broadest sense) [3]. Nongenetic parental effects can be mediated by the transmission of epigenetic, somatic, morphological, behavioural, or environmental variants [3,16,17]. Thus, a paternal effect can be said to occur when a nongenetic factor is transmitted from a male to his offspring, resulting in effects on offspring development. The nature of this nongenetic factor can be influenced by paternal genotype (paternal indirect genetic effect), paternal environment (paternal environmental effect), or a combination of both. The term 'paternal effect' has sometimes been used to refer to direct genetic effects (i.e., the transmission of alleles from males to their offspring) (e.g., [18–20]) or genomic imprinting effects (e.g., [21]), but this usage should be avoided because, in these cases, variation in offspring phenotype reflects variation in offspring genotype [14].

How then are nongenetic factors transmitted from a male to his offspring? It is easy to see how mothers can transmit biomolecules (e.g., nutrients or hormones), environmental influences (e.g., temperature or natal environment), or behaviour (e.g., maternal care or anxiety) to their offspring. Likewise, various channels of father–offspring influence are available in species that exhibit substantial paternal investment, such as ejaculate-borne defensive alkaloid compound transfer in the moth *Utetheisa ornatrix* [22], direct transfer of antimicrobial compounds to brooded eggs in the blenny *Ophioblennius atlanticus* [23], or postnatal paternal care in the mouse *Peromyscus californicus* [24]. However, paternal

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Box 1. Three types of paternal effect that are mediated to varying degrees by maternal responses

Type A

In species where males interact directly with their offspring, such as some vertebrates and arthropods that exhibit complex forms of paternal care, paternal effects can occur via direct effects of males on their offspring. Such effects can, for example, involve paternal behavioural influences on offspring development [24]. Nonetheless, paternal effects can interact with maternal effects in such systems (Figure I).

Type B

In external fertilisers, paternal effects can occur via male influences on eggs outside the body of the female. Such effects can be mediated by sperm- or ejaculate-borne factors [26–28], or by secretions from somatic glands [74]. There has been little contention in classifying Type B effects as paternal effects because, even though the egg phenotype can influence the effect, and different egg phenotypes can respond differently to sperm produced by different males, there is no opportunity for females to adjust egg phenotype after eggs have been released: the causal pathway is limited to responses by the egg cell itself. However, an interaction between paternal and maternal effects can still occur, and affect relative allocation to different offspring traits. For example, in an external fertiliser, eggs fertilised by males of phenotype p_1 might grow more quickly but hatch at a smaller body size, whereas eggs fertilised by males of phenotype p_2 might grow less quickly but hatch at a larger body size. Such responses might represent a facultative maternal or paternal strategy or, alternatively, a nonadaptive interaction.

Type C

In internal fertilisers, male phenotype can exert an influence on the female body, and this influence can, in turn, manifest as an effect on offspring development. Such paternal effects can encompass a complex chain of maternal responses involving the nervous, endocrine, and other physiological systems [1,5,24]. Type C paternal effects can be mediated by female differential allocation of total resources (e.g., making larger or higher-quality offspring in response to particular male phenotypes [35,36]) or, potentially, differential relative allocation to different offspring traits or fitness components (e.g., producing offspring that grow faster but are less viable in response to particular male phenotypes). Type C effects might be especially challenging to interpret because in many cases it will be difficult to determine how the effect is mediated and whether it represents a male strategy, a female strategy, or a combination of both.

(A) Post-natal effects
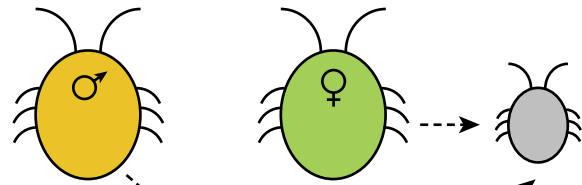
(B) External fertilization
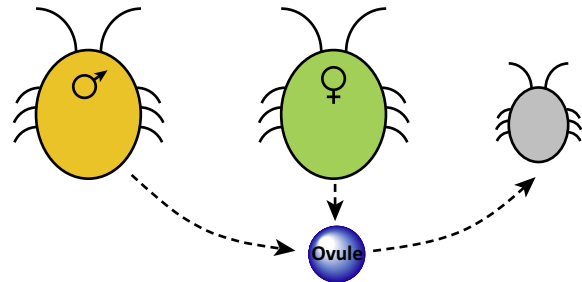
(C) Internal fertilization
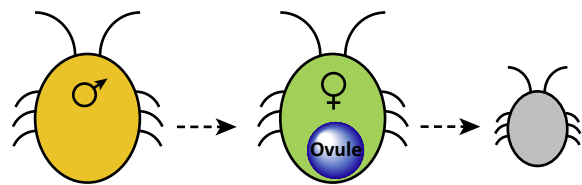
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Figure I. Three basic types of paternal effect. Broken lines represent pathways whereby fathers (♂) and mothers (♀) influence their offspring. **(A)** Postnatal effects. **(B)** External fertilization. **(C)** Internal fertilization.

investment is low in most species [25], and the resulting lack of opportunity for father-offspring influence was long regarded as a constraint on the occurrence of paternal effects. Nonetheless, recent evidence shows that sperm can convey many nongenetic (e.g., cytoplasmic or epigenetic) factors [26,27], and that ejaculates contain a complex blend of proteins and lipids that can influence offspring development [28]. For example, in golden hamsters, ejaculate components produced by the male accessory glands influence offspring embryonic development, postnatal growth, and adult responses to olfactory and auditory cues [29–31]. Sperm- and ejaculate-borne factors have the potential to mediate paternal effects in all sexually reproducing species.

Importantly, however, many pathways of father–offspring influence are mediated by maternal responses. The nature of these pathways reflects a fundamental asymmetry between maternal and paternal investment, and represents a basic difference between a typical maternal effect and common types of paternal effects (Box 1). Given that eggs contribute a larger quantity of cytoplasm to the zygote than do sperm, and the egg controls early development [32], we suggest that the maternally derived composition and structure of the egg will often play a role in mediating paternal effects. Moreover, in taxa with

internal fertilization, paternal effects are likely to be mediated by a complex chain of maternal physiological or behavioural responses. For example, in mice, the social upbringing of a male was found to influence the nursing behaviour of its mate, and this maternal response in turn affected the growth rate of the offspring [33]. By contrast, mothers typically exert direct effects on egg content and structure and, in many organisms, on embryonic (and sometimes postnatal) development as well. The evolutionary consequences of these fundamental differences between maternal and paternal effects have not been fully explored.

Paternal or maternal?

When paternal influences on offspring are mediated by maternal responses (Box 1), we run into an obvious conundrum: should these effects be regarded as paternal effects or maternal effects? The logic of analysis of variance (ANOVA) suggests the most straightforward decision rule. Statistically, an effect occurs when variation in an independent variable is associated with variation in the response (dependent variable). Note that, although a key assumption of such analysis is that the parent–offspring correlation is causal [14], the nature of the causal pathway


Box 2. Distinguishing male condition transfer effects from female differential allocation effects: two examples

Example 1. Consider an insect species, with or without conventional forms of paternal care, in which paternal diet influences offspring phenotype (e.g., [38,75,76]). Although this effect could be mediated by female physiological responses, it might be most natural to regard this as a paternal effect mediated by the content of the male ejaculate {e.g., diet-dependent quantities or types of proteins (accessory gland proteins, ACPs) or other biomolecules that influence female effects on eggs, or directly alter the pattern of development of eggs with which they come into contact within the female body [28,58]} or sperm epigenotype (e.g., diet-dependent cytosine methylation patterns [1]). However, if it can be shown that the effect results not from variation in limiting resources transferred in the sperm or ejaculate, but instead from female assessment of male phenotype (whether visual, chemical or tactile), then the effect will represent maternal differential allocation [36,77].

Example 2. Consider a bird species in which paternal colouration influences offspring phenotype, and this effect is observed even if male colour is manipulated experimentally [36,77]. The most plausible mechanism mediating this effect is female differential resource allocation to eggs on the basis of female assessment of male attractiveness [35,36]. However, it is also possible that males may allocate resources to females or their offspring, and the quantity or quality of the allocated resources might be associated with male colouration (which might be condition-dependent, and could be assessed by males themselves) [78,79]. Of course, female differential allocation and male condition transfer might both occur and interact within the same species.

These examples show that a detailed knowledge of the proximate basis of observed effects is necessary to differentiate

paternal condition transfer from maternal differential allocation (Figure I).

(A) Paternal condition transfer

(B) Maternal differential allocation

Assessment

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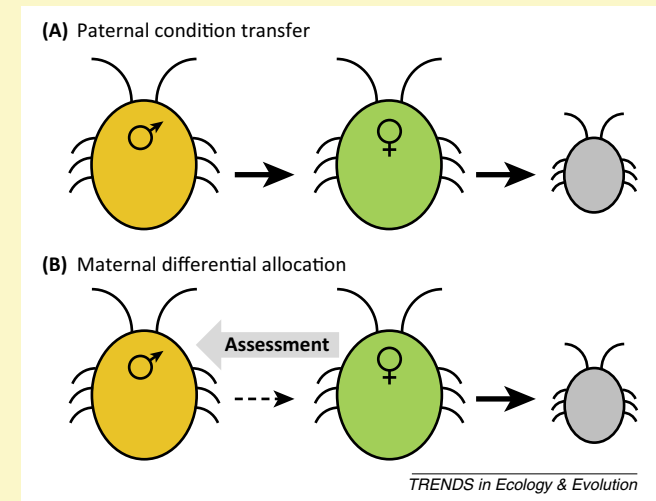
Figure I. Male condition transfer or female differential allocation? **(A)** Under paternal condition transfer, males transmit limiting resources or condition-dependent epigenetic factors (thick arrow) directly or indirectly (i.e., via the female) to their offspring. **(B)** Under maternal differential allocation, females assess male quality and use their own pool of resources to preferentially provision offspring of some males.

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between the independent and dependent variables is treated as a black-box in the statistical model. Thus, if an effect on offspring is observed when the paternal phenotype is manipulated while holding sources of maternal variation constant (reflected in a 'sire' effect in ANOVA), then the effect will generally be considered paternal. Conversely, if an effect on offspring is observed when the maternal phenotype is manipulated while holding sources of paternal variation constant (reflected in a 'dam effect' in ANOVA), then the effect could be considered maternal. If both sire and dam effects or a sire × dam interaction are detected, then both maternal and paternal effects could be said to occur. Such data can be obtained via experimental manipulation of environment in one or both parents [34].

Based on this logic, it is straightforward to differentiate paternal effects of type A or B (Box 1) from maternal effects because the role of maternal responses in mediating such effects is limited. However, in the case of type C effects, the boundary between maternal and paternal effects is less distinct. Thus, in some cases, it might be challenging to determine whether the association between male condition and offspring phenotype is mediated by the transfer of male condition (which is most readily interpreted as a paternal effect) or by maternal differential allocation (Box 2). If differential allocation occurs, variation in male condition can be modelled as a male phenotype effect (i.e., paternal effect), or as an effect of variation in the environment experienced by females (i.e., a maternal effect), although we suggest that the term 'maternal effect' should be restricted to cases where different females respond differently to variation in male phenotype.

The distinction between paternal condition transfer effects and female differential allocation effects is important because the evolutionary implications of these effects

might be very different. Differential allocation of resources by females to offspring on the basis of male 'attractiveness' or quality [35–37] is a facultative maternal investment strategy that represents an expression of existing female preferences [35,36]. By contrast, paternal effects that involve the transfer of acquired paternal condition to offspring [38,39], or indirect genetic effects on the quality of paternal investment [40,41], can drive the evolution of novel, costly female preferences because such effects represent additional benefits for females of mating with high-condition males [4,6,7]. The key difference is that, in the former case, females draw on their own pool of resources to provision differentially the offspring of particular males whereas, in the latter case, some males furnish additional resources to females or their offspring (Box 2). This reasoning can be extrapolated to maternal and paternal effects in general: because maternal and paternal contributions of limiting resources (i.e., parental investment) and other fitness-influencing factors to offspring are expected to influence the course of sexual coevolution in different ways [25,42], we can predict that paternal and maternal effects will usually have different evolutionary consequences.

Questions to guide research on paternal effects

We outline below some key questions that can serve as a guide to research on the physiology, behavioural ecology, and evolutionary biology of paternal effects.

Is the effect genetic or environmental?

Paternal environmental effects can be detected by comparing the offspring of males reared or housed under different conditions, and mated to standard females. Experimental studies in rodents have demonstrated transgenerational

effects of male exposure to toxins such as alcohol, including reduced birth weight and cognitive impairment (reviewed in [1]), and there is increasing evidence for transgenerational effects of other environmental manipulations such as paternal stress [43,44], social environment [33,45] and physical exercise [46]. For example, male mice stressed by chronic exposure to a dominant rival sired offspring that exhibited depression- and anxiety-like behaviour [43], whereas male mice reared in a complex social environment sired offspring that exhibited increased rates of growth [33]. Environmental effects can also interact with the paternal genome: for example, in the bellflower (*Campanula americana*), paternal light level affected offspring germination success, but this effect varied among genotypes [47]. Such transgenerational genotype × environment effects could be investigated using replicate clones or inbred lines, cross-fostering, *in vitro* fertilisation, and/or split-clutch and split-ejaculate designs [48–51]. By contrast, paternal indirect genetic effects can be detected by examining the phenotypes of offspring produced by males of varying genotype, taking care to exclude the possibility of effects mediated by allele transmission. This can be done most simply by examining effects of sex-chromosome-linked variation on offspring that do not inherit those chromosomes. For example, recent studies have shown that the Y chromosome of male mice can affect the physiology and behaviour of their daughters [52], and the X chromosome of male *Drosophila* can affect the egg-to-adult survival of their sons [53].

What is the proximate mechanism mediating the paternal effect?

Identifying the proximate mechanism mediating paternal influence on offspring has become the 'holy grail' for paternal-effect studies, but in most cases the mechanism remains unknown. This question is intriguing because in most species fathers do not interact with their offspring beyond transferring an ejaculate to their mother and, thus, mechanisms of paternal effects in these species might be general and widespread. However, from an ecological or evolutionary perspective, the proximate mechanism mediating an effect might be of less interest than its pattern of transmission and its consequences for variation in phenotypic features and fitness [4,54].

With the discovery that epigenetic modification of the genome can, in some cases, be stably transmitted down the male line, the focus of paternal-effect studies has shifted to epigenetic effects [1,2]. Paternal effects of a high-fat diet in rats, resulting in impaired insulin secretion and glucose sensitivity in daughters, have been linked to epigenetic modification (reduced methylation) of the *Il13ra2* (interleukin 13 receptor, $\alpha 2$) gene [55], and paternal effects of cocaine use in rats, resulting in increased levels of BDNF (brain-derived neurotrophic factor) protein in the medial prefrontal cortex of sons, are associated with epigenetic modification (acetylation) of a histone protein associated with the promoter of the *Bdnf* gene in sperm [56]. However, in addition to epigenetic modifications, sperm also transmit oocyte activation factor, centrosomes, and various cytoplasmic RNAs [26,27], all of which play a role in development and, therefore, could potentially mediate nongenetic paternal effects.

Moreover, seminal fluid often comprises a substantial portion of the ejaculate, and contains a suite of proteins, lipids, and other molecules that could mediate paternal effects [28]. These seminal fluid products can be strategically allocated by males in response to mating conditions [57–59], and can have complex effects on female fitness and behaviour [57,60,61]. Thus, seminal fluid might be altered by the paternal environment, with differential effects on females causing downstream effects on offspring development and phenotype. Seminal proteins might also directly influence the development of offspring [62] independently of any effects on the female. The list of potential mechanisms mediating nongenetic paternal effects has grown rapidly in recent years, and we suspect that more mechanisms are yet to be discovered.

Is the effect a facultative response on the part of the father or mother?

A paternal effect can represent an evolved, adaptive (facultative) strategy on the part of the male [63] or a non-adaptive effect (e.g., a transgenerational manifestation of pathology or senescence) [64]. If the paternal effect is a fitness-enhancing strategy for the father, it might or might not also increase maternal fitness. For example, a paternal effect could enhance offspring fitness without cost to the female (e.g., via the transfer of paternal resources or condition-dependent epigenetic states), or it might elevate female investment in the immediate batch of offspring at the expense of future offspring and female net fitness (similar to the 'toxic ejaculate' effects thought to mediate sexual conflict in some species [65]). In addition, a maternal response to variation in male phenotype can represent a facultative female strategy (e.g., differential allocation as described above) or a non-adaptive response (which could nonetheless represent an adaptive male strategy that enhances male fitness at the expense of the female). In other words, a paternal effect can be facultative or antagonistic from the perspective of the male, female, or both.

Females are predicted to adjust their reproductive investment in response to mate quality, either to take advantage of high male quality (differential allocation hypothesis [35,36]) or to compensate for low male quality (compensation hypothesis [66]). Differences in male phenotype can act as a cue that females actively respond to. In such cases it is also likely that different females will respond differently to male cues, such that a male × female interaction will be observed. However, there might be cases where male-derived substances directly influence offspring development within the female body, and the female can be regarded as a passive conduit for such effects.

In practice, it might often be difficult to separate out whether an effect on offspring is directly induced by the male or is mediated by female responses to the male (Box 2). Facultative female responses might appear unlikely when the paternal effect is negative (e.g., high-fat diet or smoking) because it is unlikely that a mother would actively reduce the health of her offspring (although see 'selfish maternal effects' [12]). However, it is possible that some females are better able than others to mitigate paternal harm to offspring. By contrast, if the paternal effect has a positive influence on offspring health, it could

result from female differential allocation, from a paternal investment strategy (e.g., condition transfer), or a combination of both. In some systems it is possible to exclude some forms of female sensory and/or behavioural responses to male phenotype using artificial insemination techniques [67], thereby reducing the opportunity for facultative allocation of resources (although this does not exclude post-copulatory selection on the basis of chemical cues associated with the ejaculate, or effects mediated by eggs themselves). It might also be possible to test for facultative female differential allocation effects by comparing egg investment in females mated to males of different phenotypes to egg investment in females exposed to males of different phenotypes without mating, and later mated to standard males.

Does the paternal effect increase offspring fitness?

Offspring might have differing interests from their parents [68]. For example, males might increase fertilization success [69] or offspring number [45] at a cost to offspring quality. If a father can predict the environment their offspring are likely to experience, they might adaptively alter the phenotype of their offspring to increase offspring fitness in the local environment (e.g., [63]). Given that these effects might often be context-dependent, to test for adaptive paternal effects it might be necessary to manipulate both the paternal and offspring environment and to quantify relative (in addition to absolute) offspring fitness [70,71]. An interaction between parental and offspring environments, whereby offspring have higher fitness when experiencing the same environment as their parent, is known as an anticipatory parental effect [12]. However, it is also possible for offspring of males in high condition to perform better in all environments [39]. Finally, as noted above, many paternal effects might be non-adaptive, reflecting the transmission across generations of pathology or senescence [64].

Concluding remarks and future directions

Mounting evidence from studies on a variety of organisms indicates that paternal effects are widespread and important. Indeed, because the paternal germline appears to be highly susceptible to environment-induced epigenetic reprogramming [55,56,72,73], paternal effects could turn out to be as commonplace as maternal effects. However, the fundamental asymmetry between maternal and paternal influences on offspring, and the mediating role of maternal responses in many paternal effects, complicate the interpretation of paternal effects at both proximate (mechanistic) and evolutionary levels. Future research should address the sources of paternal variation (genetic and/or environmental) affecting offspring phenotype, the chain of proximate effects (encompassing both paternal and maternal responses) that mediate paternal influence on offspring, and the consequences of such effects for paternal, maternal, and offspring fitness.

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