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Parent-of-origin specific gene expression and dispersal

Thomas J Hitchcock and Andy Gardner

Genes can behave in ways that are conditional upon their parent-of-origin. The best understood form of this is genomic imprinting (GI), which typically involves the silencing of a gene originating from one parent and the expression of its homologue originating from the other parent. A number of hypotheses have been proposed to explain GI, which may be grouped into those based on asymmetries of genetic interest versus those based on asymmetries of genetic information. Dispersal patterns can drive both of these asymmetries and modulate the costs and benefits of imprinting. GI may also have consequences for dispersal of individuals and genes, by driving imprinting of loci underpinning dispersal, altering the fitness consequences of dispersal, and affecting rates of introgression.

Address
School of Biology, University of St Andrews, St Andrews KY16 9TH, UK

Corresponding authors: Hitchcock, Thomas J (thomasjameshitchcock@gmail.com), Gardner, Andy (andy.gardner@st-andrews.ac.uk)

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Introduction

Genes can behave in ways that are conditional upon their parent of origin. This was first recognized in the 1920s with paternal genome elimination in Arthropods [1,2] and referred to as 'imprinting' by Helen Crouse in her work on Sciarid flies [3,4]. Evidence for imprinting has now been reported in a wide range of taxa, including insects [5,6], mammals [7], and angiosperms [8].

'Genomic imprinting' (GI) refers specifically to when a gene's level of expression depends on its parent of origin, and usually involves the silencing of a gene originating from one parent and the expression of its homologue originating from the other. Although GI represents a form of mono-allelic expression, and may result in maternal or paternal effects, these are separate phenomena that may alternatively arise through random gene silencing [9] or transgenerational inheritance [10]. Recent years have seen the development of new and improved experimental and statistical methods to detect GI and distinguish it from these other processes [11–13]. This has led to a new wave of interest into surveying the extent of imprinting across different species, and its mechanistic underpinning.

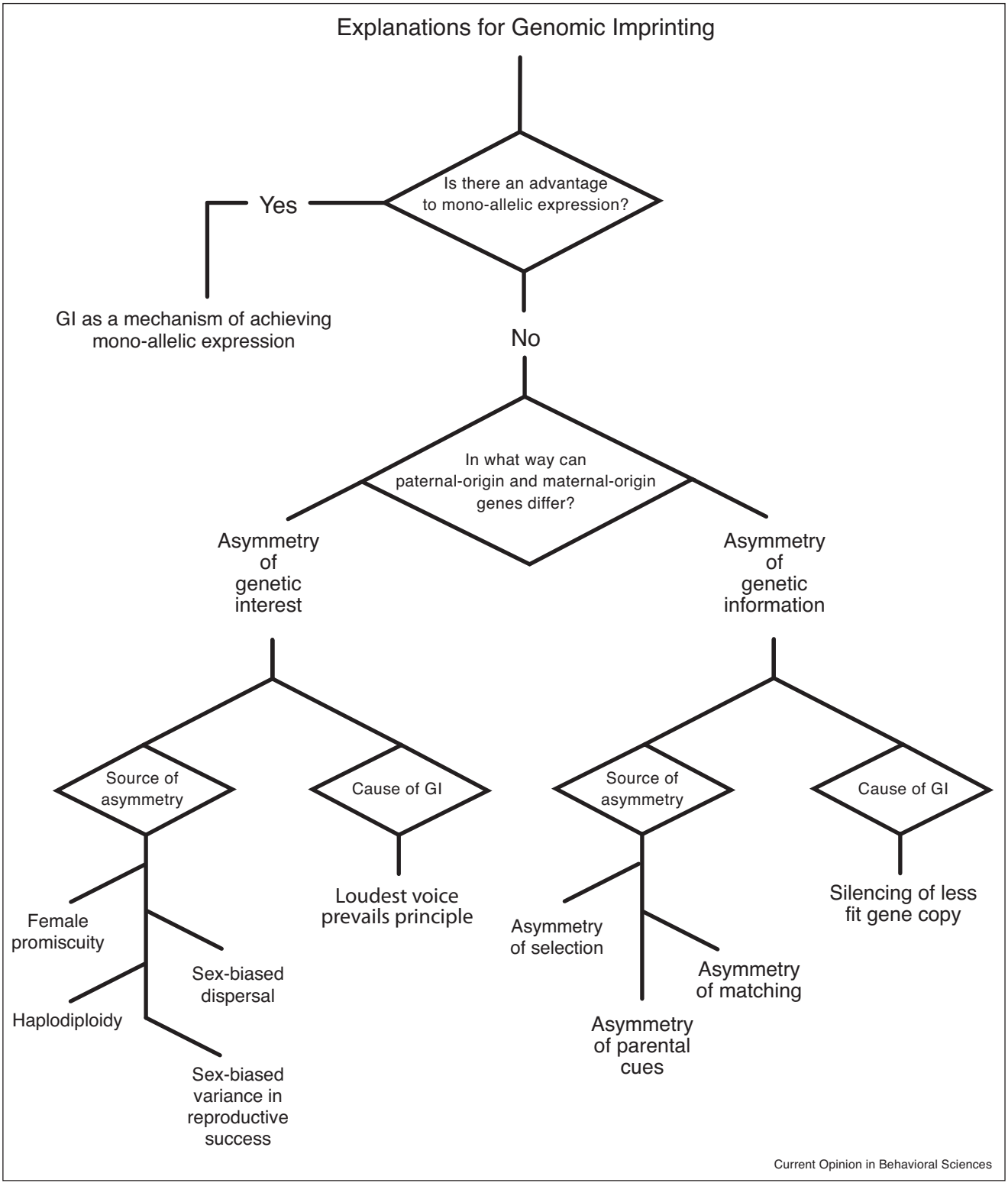
From an evolutionary perspective, GI is paradoxical in that it may render the individual functionally haploid at affected loci, and hence more vulnerable to the effects of recessive deleterious mutations [14]. Indeed, GI has been implicated in a wide range of human pathologies, including growth and developmental disorders, cancers, and infertility [15]. Accordingly, evolutionary biologists have sought explanations as to why an organism would systematically silence a gene inherited from one of its parents (Figure 1). Two basic explanations have emerged: one concerning an asymmetry of genetic interests [16], and another concerning an asymmetry of genetic information [17,18••] (Figure 2).

Much of the evolutionary literature on GI has emphasized how female promiscuity may drive asymmetries between maternal-origin versus paternal-origin genes [16]. However, focus has recently shifted onto dispersal as a driver of such asymmetries. Here we consider the roles dispersal may play in the evolution of GI and the ways in which GI may, in turn, influence dispersal. These ideas intersect with a number of ongoing controversies in evolutionary biology, in relation to epigenetics [19], phenotypic plasticity [18••] and the fundamental units of adaptation [20–22].

Dispersal as a modulator of GI

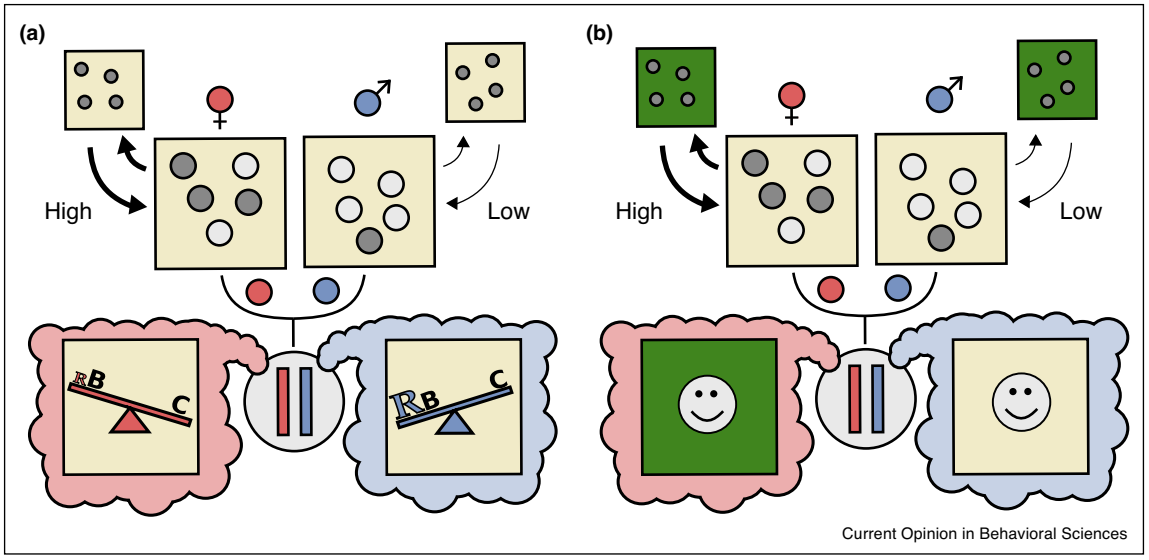
Dispersal and asymmetries of interest — Conventionally, the individual organism is viewed as a unified entity which, through the action of natural selection, comes to appear as if designed to maximize its inclusive fitness [23,24]. That is, it is favoured to maximize the total reproductive success of all its relatives — including itself — with each increment or decrement of reproductive success being weighted by the degree of genetic relatedness to that relation [25]. However, Haig has pointed out that the genes the individual obtains from its mother may disagree with the genes obtained from its father over how related they are to the individual's maternal versus paternal relatives, leading the genes to have different inclusive-fitness interests when it comes to social interactions with those relatives [16]. He suggested that this asymmetry of genetic interests could drive the evolution of GI.

Figure 1



Hypotheses for the evolution of genomic imprinting.

Figure 2



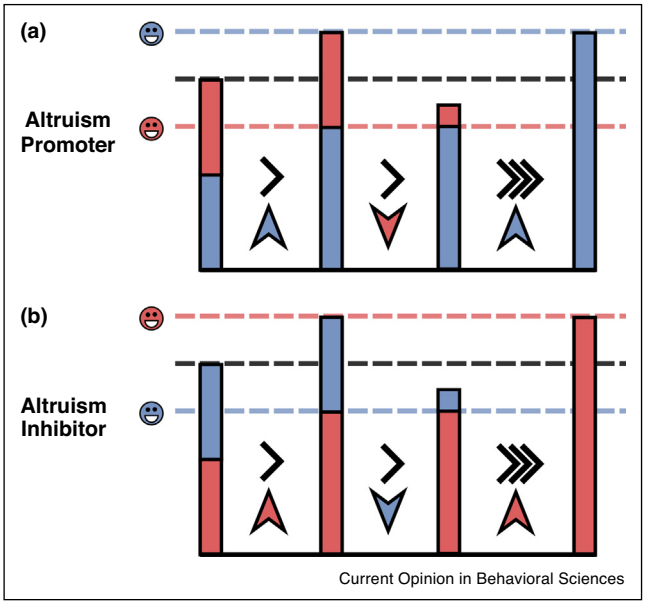
Asymmetries of interest and information. **(a)** Asymmetries of genetic interest: Different rates of dispersal between the sexes results in genes from parents of the less-dispersing sex being more related, on average, to their carriers' social partners. In this case, female-biased dispersal results in social partners being more related via their fathers than their mothers. Thus, higher levels of altruism favoured by paternal-origin genes, and higher levels of selfishness favoured by maternal-origin genes. **(b)** Asymmetries of genetic information: Local adaptation coupled with sex-biased dispersal may result in genes from parents of the less-dispersing sex being fitter, on average, in their carriers' current environments. In this case, less-dispersing fathers provide genes that are more adaptive for the local environment, whereas more-dispersing mothers provide genes that are less adaptive for the local environment.

Take for example a group-structured population in which females are more likely than males to disperse away from their natal group to start a family elsewhere, such that individuals will tend to be more related to their group-mates via their fathers than via their mothers [26–29]. If individuals may behave in individually costly but group-beneficial ways — for instance, by participating in cooperative care of the group's young — then their paternal-origin genes will tend to favour such behaviour, whilst their maternal-origin genes will tend to disfavour it (Figure 2a). In such cases, genes residing at loci where increased expression leads to more group-beneficial behaviour are favoured to increase their expression when of paternal-origin and decrease their expression when of maternal-origin, which may lead to the self-imposed silencing of the maternal-origin gene (Figure 3; [30]). Conversely, genes residing at loci where increased expression leads to less group-beneficial behaviour are favoured to decrease their expression when of paternal-origin and increase their expression when of maternal-origin, leading to the silencing of the paternal-origin gene.

Accordingly, sex-biased dispersal may drive the evolution of GI in relation to loci that modulate social interactions between group-mates, perhaps accounting for the extensive imprinting of loci in the brains of mice and humans [31–33]. Sex-biased dispersal has been suggested to explain GI in *Nesp* and *Grb10*, involved in mouse behaviour [34,35,36••], with their opposing imprinting patterns possibly explained by their opposite effects on impulsive decision making [35,36••]. In humans, sex-biased dispersal has been implicated in known imprinted genes underpinning menopause [37] and language [38–40], is potentially of relevance to known imprinted genes underpinning menarche [41] and sleep [42], and has also been suggested to drive GI in relation to intrasexual [43] and intersexual [44] conflict traits. Moreover, even when sex-biased dispersal is not responsible for asymmetry in relatedness, it has been suggested to modulate the intensity of the corresponding intragenomic conflict, for example in relation to soldier development in polyembryonic parasitoid wasps [45].

Dispersal and asymmetries of information — Alternatively, GI might evolve in scenarios where maternal-origin versus paternal-origin genes differ in the quality of information they possess. In variable environments, organisms may maximize their inclusive fitness by integrating information from a range of different sources — including genomic cues [18••] — in deciding which of a range of different phenotypes to employ, termed 'phenotypic plasticity' [46]. Accordingly, if alleles encoded by maternal-origin genes are associated with systematically better

Figure 3



The 'loudest voice prevails' principle of GI. The expression levels of two genes coevolve over time, each gene attempting to push the total expression level closer to their personal inclusive-fitness optima. Here, the paternal-origin gene (blue) favours a more-altruistic phenotype, whereas the maternal-origin genes (red) favours a less-altruistic phenotype. **(a)** If a higher level of expression at this locus leads to an increase in altruistic behaviour (altruism-promoter locus), then the paternal-origin gene favours a higher level of expression and the maternal-origin gene favours a lower level of expression, such that the maternal-origin gene ultimately silences itself and the paternal-origin gene expresses at its optimal level (maternally silenced and paternally expressed locus). **(b)** If a higher level of expression at this locus leads to a decrease in altruistic behaviour (altruism-inhibitor locus), then the paternal-origin gene favours a lower level of expression and the maternal-origin gene favours a higher level of expression, such that the paternal-origin gene ultimately silences itself and the maternal-origin gene expresses at its optimal level (paternally silenced and maternally expressed locus).

adaptation to environmental circumstances than are alleles encoded by paternal-origin genes, or vice versa, then the individual may choose to silence genes derived from one parent.

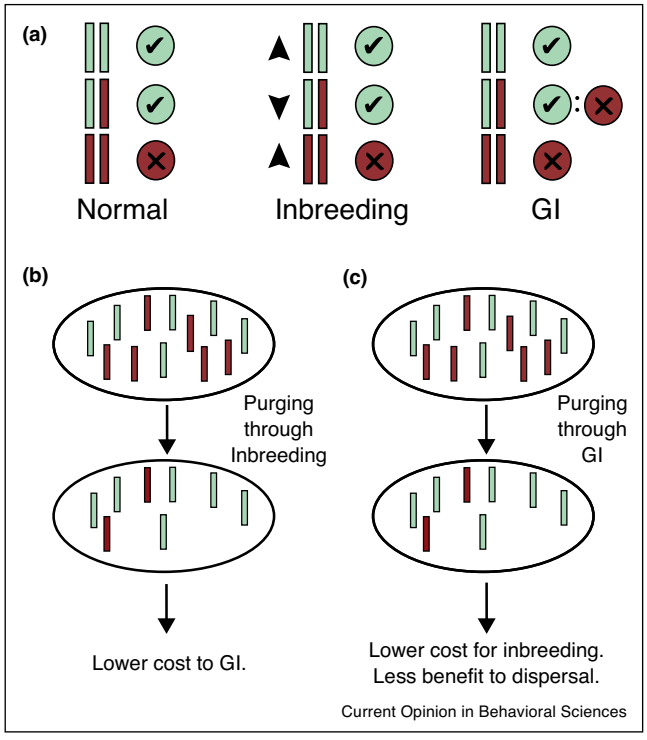
But why would the quality of genetic information differ in this way? One possibility is an asymmetry in the selective environment previously experienced by maternal-origin versus paternal-origin genes. Taking again the example of a group-structured population characterized by female-biased dispersal, if the environment is spatially heterogeneous then there may be different selective pressures in different groups, resulting in local adaptation. Accordingly, genes deriving from an individual's father will, on average, be better adapted to the individual's current environment than those genes deriving from the individual's mother, such that the individual may benefit from silencing its maternal-origin genes (Figure 2b) [47,48]. Local adaptation in the face of gene flow has been documented in several taxa [49] — for example, cryptic coat coloration in deer mice [50] — and sex-biased philopatry has been highlighted as an important factor in maintaining allelic polymorphism [49].

Similar scenarios include situations in which selection is: stronger in one sex [51], in which case genes derived from parents of that sex are better quality for both sons and daughters; sex-limited [52,53], in which case genes derived from parents of that sex are better quality for offspring of the same sex; or sexually antagonistic [54], in which case genes derived from parents of each sex are better quality for offspring of the same sex and lower quality for offspring of the opposite sex. Also, when genes interact with other, 'uniparental' genes inherited from a particular parent — such as cytoplasmic or, indeed, imprinted genes — are more likely to work well in conjunction with the other genes inherited from that parent (this parent having clearly managed to successfully reproduce), favouring silencing of the genes deriving from the other parent [51,55–57]. Finally, if social partners expressing the same alleles leads to beneficial (or harmful) effects, then individuals might be favoured to silence genes derived from the parent via whom they are less (or more) related to their social partners, to maximize the probability of expressing the correct allele [58,59•]. Such matching effects might occur in relation to parent-offspring interactions [60,61] and could involve genes at a single locus [62] or several linked loci [60].

Although these latter scenarios do not highlight dispersal as a driver of asymmetry in information quality of maternal-origin versus paternal-origin genes, there is a possible role for dispersal to modulate selection of GI via its impact on the allelic diversity of evolving populations. This is because these scenarios propose that GI is an adaptation to make improved use of the allelic variation segregating at a locus, and so its adaptive value disappears in the absence of such segregating variation. Various models have shown that weak migration in structured populations helps maintain allelic diversity [63,64], and gene flow has also been shown to be important in maintaining polymorphism in wild populations [65–67].

Dispersal and the fitness consequences of GI — In addition to modulating the benefits of GI via its impact on asymmetries of inclusive-fitness interests and information quality of maternal-origin versus paternal-origin genes, dispersal may also have consequences for the evolution of GI by modulating its fitness consequences. For example, as described above, an obvious cost of GI is that the resulting functional haploidy renders the individual particularly vulnerable to the effects of recessive, deleterious alleles [51]. The frequency with which such alleles segregate may be lower in populations with lower rates of dispersal and hence higher rates of inbreeding, as inbreeding

Figure 4



Mutual reinforcement of inbreeding and GI. **(a)** Inbreeding and GI both expose a higher proportion of deleterious recessive alleles to selection, by either increasing the amount of homozygosity (inbreeding) or functional haploidy (GI). **(b)** Greater exposure of deleterious recessive alleles to selection leads to purging of such alleles from inbreeding populations, which reduces the cost of functional-haploidy and hence promotes the evolution of GI. **(c)** Similarly, greater exposure of deleterious recessive alleles to selection leads to purging of such alleles at loci exhibiting GI, which reduces the cost of inbreeding depression and hence promotes the evolution of inbreeding.

increases the frequency of homozygotes, increasing the exposure of such alleles to selection, and thereby purging them from the population [68]. Accordingly, individuals in low-dispersal, high-inbreeding populations are expected to suffer lower costs in relation to functional haploidy and hence might more readily evolve GI [51] (Figure 4).

GI as a modulator of dispersal

GI of dispersal loci — An individual's proclivity to disperse often has a strong genetic basis [69] and, accordingly, the loci underpinning dispersal may themselves exhibit GI. One reason for why this may occur is that, by dispersing, an individual may relax competition for reproductive resources among those non-dispersing neighbours that have been left behind. If neighbours are more related via one parent, then genes originating from this parent may be more inclined to have the individual disperse than are genes originating from the other parent [70•]. However, it is unclear what effect, if any, this would have on the level of dispersal exhibited by individuals: although genes originating from the first parent are expected to win the intragenomic conflict against their homologues at dispersal-promoting loci, raising the level of dispersal above the individual's inclusive-fitness optimum, genes originating from the second parent are expected to win at dispersal-inhibiting loci, reducing the level of dispersal, and, on average, these opposing effects might be expected to cancel each other out [70•,71]. It is also feasible that GI driven by asymmetries of information quality could occur in relation to loci underpinning dispersal, but as far as we are aware this remains to be investigated.

GI and the fitness consequences of dispersal — GI might also modulate the evolution of dispersal by altering its fitness consequences. One factor thought to promote the evolution of dispersal (in particular, sex-biased dispersal) is inbreeding depression [72], with the deleterious consequences of consanguineous mating particularly penalising individuals who fail to disperse away from their place of origin. Just as inbreeding may lead to purging of recessive deleterious alleles that incur costs in relation to the functional haploidy associated with GI, so too may GI lead to purging of these same alleles that incur costs in relation to the inbreeding-depression associated with consanguineous mating [51,73,74]. Accordingly, by reducing the cost of inbreeding, GI may inhibit the evolution of dispersal (Figure 4).

GI as a barrier to allelic dispersal — Hybrid dysfunction is an important barrier to introgression (that is, the dispersal of alleles) between hybridizing populations, and hence may play a role in the formation and maintenance of incipient species. GI has long been suggested to play an important role in hybrid dysfunction for both mammals [75,76], and flowering plants [77,78], and may do so through multiple mechanisms [79,80].

In flowering plants, endosperm failure — resulting in inviable seeds — is known to be caused by imprinted genes for certain interploidy crosses [81,82], and similar processes appear to underlie interspecies seed inviability [83,84]. Furthermore, the endosperm-balance-number hypothesis may be underpinned by GI [85,86], potentially explaining patterns of hybridization in wild populations [87•].

Similarly, in mammals, hybrids often have growth abnormalities dependent on the direction of the cross [80,88]. The loss of imprinting causing misregulation of growth was proposed [76,80], and has been tested in multiple mammalian species [88–93]. Whilst there is mixed evidence for loss of imprinting being the cause [88], disruption of imprinted genes is nonetheless strongly linked to abnormal hybrid growth [93].

Conclusion

GI may be driven by asymmetries in the adaptive information content and evolutionary interests of maternal-origin versus paternal-origin genes. These different types of asymmetry are not mutually exclusive but likely drive GI in different types of gene. Dispersal may play an important role in driving asymmetries between maternal-origin and paternal-origin genes and, in the process, modulate the costs and benefits of GI. Conversely, GI may have consequences for dispersal, both by presenting a barrier to gene flow and also by modulating the costs and benefits of dispersal. However, the interaction between dispersal and GI remains underexplored, both theoretically and empirically, and hence represents a valuable avenue for future research.

Conflict of interest statement

Nothing declared.

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