ARTICLE

The genetic battle of the sexes

Sofia Deleniv considers the implications of genomic imprinting for social behaviour and mental health

When we see a toddler showing the first signs of personality, how often are we tempted to say 'She must get that from her mother'? Such assumptions are quite sensible – after all, our genetic makeup is composed of two copies of 23 chromosomes, one copy inherited from each parent. But is there any scientific reason to believe that some aspects of our behaviour could be inherited from *one* of our parents, as opposed to both of them?

Over the past few decades, researchers have observed this phenomenon throughout much of the animal kingdom, leading some theoretical biologists to suggest that this might be the evolutionary outcome of our paternal and maternal genes competing for their own survival. At first thought, this doesn't make sense. If the genes of mothers and fathers are equally 'interested' in the survival of their children, why would they ever compete? According to one prominent school of evolutionary biology, we should blame non-monogamous mating habits for providing the playground for the genetic battle of the sexes (Haig, 2000; Moore & Reik, 1996). To understand why, let's take some time to examine what it means to be genetically successful.

Seeds of conflict

It's widely accepted that evolution is driven by the propagation of some genes to future generations, and our intuition is often that genetic success is entirely driven by the survival of our offspring. However, according to a revolutionary hypothesis that has become a central tenet of modern biology, this view isn't entirely accurate, since your children are not the sole carriers of your genes (Hamilton, 1964). Instead, your genetic success is defined by your inclusive fitness – that is, the survival of your genetic material contained in your children, as well as your kin, whose contribution to your fitness is weighted by the strength of their relatedness to you (eg. the survival of your cousin is equivalent to 12.5 per cent of you, since that is the portion of genes you share). So, if your blood relatives survive and reproduce, their input into the future gene pool is also a genetic triumph for you – after all, a portion of your genes have made their way to the next generation, even if not through your own offspring.

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If the genes of mothers and fathers are equally 'interested' in the survival of their children, why would they ever compete?

The idea that your evolutionary success is propelled purely by the survival of your own genes raises an intriguing and fundamental issue. And that is, your interests don't actually lie in the survival of a relative or child as an individual, but rather merely in the survival of the portion of their genetic material that is also yours. Let's consider that each child is a mosaic of two genomes derived from two distinct individuals. Years ago, the respected evolutionary biologist David Haig suggested that a child's inclusive fitness can be separated into two independent components, representing the success of that child's matrilineal and patrilineal genes (Haig, 2000). This means that the genetic material donated by a child's mother can 'pursue' the success of her own bloodline without regard for how the father's genes fare, and vice versa. In some cases, genes inherited from the two parents end up competing inside their own offspring.

Competition begins when the two parents have conflicting interests. This is found in species that practise polygamy or serial monogamy – so, most primate species, including humans. As a female mates with several males during her lifetime, she will often give birth to children of multiple paternities, and subsequently raise them on her

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own, or with her current partner. Depending on the time these children take to raise, they will likely overlap in time and have to live with each other, sharing resources as well as maternal attention. This means that family groups in non-monogamous societies are primarily composed of asymmetrically related children, who share their mother's genes, but are unlikely to be related via the paternal line. This sows the seeds of conflict.

First, let's examine the male's perspective. When he mates with a female, who is likely to eventually mate with others, his genes enter a world of uncertainty. If he has successfully impregnated her, his offspring will probably be raised in a family group that already does or will at some point include other children who don't carry his genes. These other children will presumably compete with his offspring for maternal attention and resources, and as ruthless as this might sound, the welfare of these children is none of his concern. They do nothing to propagate his genes. In light of this, the male would benefit from transmitting genes that encourage his children to exploit the mother's care and resources to the detriment of her other children.

This, however, doesn't suit the female's interest, as all the children she is raising are equal carriers of her genes, regardless of paternity. If the genes that the female donated to her children produced exploitative behaviours, this would only cause some children to outcompete others and harm the net survival of her genetic material. In light of this, the mother's genetic fitness would increase if her genes influenced her offspring in ways that could oppose the resource-grabbing genes of the males with whom she mates. This maximises the probability that all her children receive an equal portion of care and chance of survival.

Given the evidence for fundamental tension between the sexes, evolutionary biologists have suggested that non-monogamy has shaped the evolution of a special class of genes, called imprinted genes. These genes stand in stark contrast to most of our genes, which become expressed into proteins from both gene copies that we inherit from our two parents. Imprinted genes are distinct because they carry so-called imprints, or markings, that inform our cells about the sex of the parent who donated a particular gene copy. Subsequently, our cells use this sex-of-origin information to allow only one parent's gene copy to be active throughout the child's life, depending on whether it came from the mother or the father. This scenario, whereby one gene copy speaks while the other remains mute, provides an avenue for our maternal and paternal genomes to go their separate ways in pursuing genetic success. This is because at its core, silencing a gene copy donated by one parent is an effective method for preventing it from having an influence on the child's physiology and behaviour, while handing over the power to the other parent's gene. Why is that? Quite simply, a silent gene copy doesn't produce any protein. Thus, if its DNA sequence undergoes any changes it will have no functional implications for the child's phenotype and chances of reproductive success.

The active gene copy, on the other hand, does make a difference, which allows it to shape the phenotype of the individual. Ultimately, the fact that the gene copy originating from one parent has the exclusive rights to making its protein product means that it's also in charge of controlling whatever function is underpinned by that protein. This could concern any aspect of the organism, from hormonal and cardiovascular to metabolic and neurological function.

Ruthless agents

How could this scenario have arisen? Let's consider a situation in which a female mouse pup inherits two copies of a particular gene, of which the paternal copy is highly active, while the maternal copy results in less efficient protein expression. What if, perchance, the protein coded by this gene influenced the

Meet the author

'My foray into genomic imprinting began last year, during my MSc in Neuroscience at Oxford. As I dug into the scientific literature on the various ways in which our genes can "remember" the sex of their donor parent, I began to wonder why they would do this at all. An imprinted gene is unlike most genes, because only the gene copy donated by one of our parents is permitted to produce protein throughout our lives, while the copy donated by our other parent remains eternally silent. At first thought, this arrangement makes no biological sense! After all, it only takes a mutation to disrupt our one active gene copy before its protein production suffers. This contrasts with most of our other genes, which often require a "double hit" to both gene copies before protein production is impaired (since one "hit" would still leave room for the second active gene copy to compensate). So why do imprinted genes exist at all, given that they appear to make us more vulnerable to genetic anomalies? Trying to answer this question for myself led me discover some fascinating theories about the evolutionary forces that have given rise to imprinting. I hope that, in reading about them, you too will come to appreciate this phenomenon.'

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pup in a way that gave her patrilineal genes an edge over the matrilineal ones? To take a purely hypothetical example, this protein could make her innately more drawn to subtle pheromone cues secreted by her paternal relatives, and thus more likely to socialise and share her food with them, while robbing her maternal relatives of the same courtesy (or sometimes outright robbing their food stores). According to the prominent evolutionary biologist David Haig, in the case that a gene would benefit the father's genetic fitness to the detriment of the mother's, evolution will come to favour an arrangement in which the paternal copy of the gene becomes maximally active while the copy donated by the mother becomes silent. The mother, after all, does not benefit from contributing to the production of a protein that jeopardises her own genetic success. The imprinted gene becomes the father's ruthless agent, increasing the likelihood that his children fight for survival at the cost of harming the prospects of their matrilineal kin. In multiple-paternity families, this often concerns half-siblings.

Of course, genes would have no need to compete with each other if the only means to success were cooperation. This could apply to members of largely monogamous species, such as beavers, which tend to raise most of their children with the same life-long partner. In these populations, it is not in a father's interest to transmit genes that make children exploit their mother so much that she becomes incapable of raising more. After all, any cost that his genes would inflict on the female's residual capacity to reproduce is a cost he will also have to bear himself, as all her future children would carry his own genetic material. Here, both parents increase their genetic fitness if all children receive equal nutrition and care, which theoretically leaves no room for conflicting interests.

I cautiously refer to this as a theoretical possibility purely because researchers question whether any species truly practises strict lifetime monogamy. Detailed investigations of the mating habits of oldfield mice – classic beacons of monogamy – have revealed that females in fact exchange partners in roughly 10–20 per cent of cases (Foltz, 1981). It appears that most animal species are sufficiently unfaithful to generate parental conflicts of interest that give rise to pressure for the evolution of imprinted genes.

How do cells remember the parental origins of genes?

The pervasive existence of imprinting is rooted in the ability of some genes to retain a lifelong molecular memory of the donor parent's sex. Such memories are created during the development of sex cells, when certain genes may acquire particular markings, called imprints, depending on whether they originate from an ovum or sperm (Villar et al., 1995). Imprints often take the form of methylation, which involves the transfer of methyl groups to segments of DNA or to the proteins around which the DNA winds (E. Li et al., 1993; Rose & Klose, 2014; Wagschal et al., 2008). The presence or absence of these imprints, which carries an implicit signal about the sex-of-origin of a particular gene copy, has substantial implications for how maternally and paternally derived genes are treated once they are donated to a child. As an example, let's examine a maternally expressed gene – one that is only active on the copy inherited from the mother, while silent on the copy donated by the father.

> "This could mean genetic mind-control at its most rudimentary level"

During the development of the male germline, the chromosome site containing the relevant gene acquires an imprint, which is placed on each copy of that gene in every sperm cell. When one of this male's spermatozoa goes on to fertilise an egg and its 23 chromosomes become part of the developing offspring's genome, the presence of the imprint at this particular site prevents the underlying DNA sequence from being used to make protein. In essence, the gene is silenced. This can occur through many possible mechanisms – for instance, the aforementioned methylation marks can obstruct the underlying DNA sequence and attract various molecules that modify the structure of the protein that organises the DNA, making it more compact, and hence less accessible for reading out. Ultimately, imprints tend to conceal the gene from the machinery that reads out its DNA sequence and translates the genetic code into a protein.

This particular gene might be treated quite differently in the female's developing ovaries, where it would not become marked with an imprint. Thus, when that gene is transmitted to a developing child, its DNA sequence can be expressed into protein. Such a gene is said to be maternally expressed and paternally silent, or imprinted. Of course, the reverse cases also exist – genes that are paternally expressed and maternally silent, because of obstructive imprints being placed on such genes in the mother's ovaries.

At the moment, roughly 145 genes are known to be imprinted in mice, while the number is so far slightly smaller in humans (Barbaux et al., 2012). While this is a mere fraction of our total of ~20,000 genes, the fact that they exist at all has yielded some fascinating examples of the conflict between some of our maternal and paternal genes.

From womb to brain

The parental conflict within us starts in the womb. The first imprinted gene to be discovered, which codes for insulin-like growth factor II (Igf2), is active only on the copy inherited from the father (De Chiara et al., 1991). When this protein

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Potential evolutionary implications of the effects of paternally expressed genes are fascinating and far-reaching

becomes expressed from the genome of a developing embryo, it promotes placental growth, increasing the nutritional supply to the developing fetus at the expense of the pregnant mother's health. In light of this, it's perhaps unsurprising that the maternal copy of this gene is normally silent, coated in obstructive imprints that prevent the underlying DNA from being read out and used to produce more Igf2 protein. From the mother's perspective, maintaining her copy of the Igf2 gene silent is critical for cushioning the effects of the paternal protein's demanding nature. Indeed we know that increasing activity of the Igf2 gene in the lab results in oversized embryos that, to an extent, drain the mother of nutrients (Constancia et al., 2002). Thus, in encouraging the developing fetus to exploit its mother during pregnancy, the paternally active Igf2 gene has been interpreted as effectively serving the father's interest of maximising the resources allocated to his developing child without regard for the mother's remaining reproductive capacity. Her future children are unlikely to be his own, after all.

Now, given the universal existence of the Igf2 gene in placental mammals, it's easy to wonder why all babies aren't born gigantic. It just so happens that the maternal genome appears to have a system of opposition to temper the gluttonous effects of the paternally active Igf2 gene. One such gene, expressed only when inherited from the mother, codes for the Igf2 receptor, which consumes and reduces levels of paternal Igf2 protein floating around during pregnancy (Wutz et al., 2001). This acts to suppress placental growth, preventing a female's developing fetus from exploiting her to the fullest and preserving her capacity to recover and produce more children in the future. Since their discoveries, the reverse imprinting of the Igf2 and the antagonistic Igf2 receptor genes has been adopted as one of the primary lines of evidence in support of the idea that genomic imprinting could be a manifestation of an evolutionary conflict between the sexes (Haig, 1997).

Interestingly, imprinted genes are particularly common in the brain, which suggests that the longstanding battle of the sexes may have benefited from hijacking the most sophisticated organ of all (Davies et al., 2005). By contributing to neuronal function, imprinted genes are able to exercise control over something much more complicated than nutrient uptake through the placenta – our behaviours, and perhaps personality traits. This could mean genetic mind-control at its most rudimentary level. Importantly, the abundance of such genes in our nervous system makes us quite sensitive to neurological abnormalities brought on by dysfunction of imprinted genes, perhaps more so than to any other physical anomaly. This is evidenced in both animals and humans.

One imprinted gene that is paternally expressed in the brain, Peg3, has received some attention in the scientific literature due to its critical importance for priming female brains for maternal care, as well as producing normal breastfeeding habits in newborns. Over a decade ago, researchers found that mouse pups who inherited disruptive mutations on the Peg3 gene specifically from their fathers are less capable of seeking out breast milk, likely due to disrupted appetite, and thus fail to gain weight and grow after birth (Curley et al., 2004). Even when raised by healthy wild-type mothers, these pups had a mortality rate of 32 per cent shortly after birth. Their survival rates were even lower when living in litters where some pups did not inherit Peg3 mutations from the fathers and thus produced normal levels of the protein.

Interestingly, inheriting Peg3 mutations from the father can have profound effects on the maternal skills of his female pups when they themselves have children. Researchers have found that these females tend to be strikingly bad mothers, as they fail to increase their appetite during pregnancy, produce poor amounts of breast milk, and often neglect feeding, grooming and sheltering their pups in the early stages of life when they are incapable of producing sufficient body heat to sustain themselves (L.L. Li et al., 1999). Indeed, the brains of mutated females are known to have fewer oxytocin-producing neurons, which are essential for the transformative emergence of maternal behaviours, such as protectiveness and enhanced appetite, as well as lactation (Pedersen & Prange, 1979). It comes as no surprise, perhaps, that the pups of Peg3 mutant mothers are less likely to make it past childhood.

A more puzzling recent discovery is that females don't even need to inherit the Peg3 mutation themselves to have compromised maternal skills! All it takes is that the fetuses they carry in their womb have a mutated paternal copy of the Peg3 gene (Champagne et al., 2009). The researchers behind this finding hypothesise that the effect might be rooted in disrupted Peg3 protein production in the mother's placenta, which contains the same genome as the embryo. This perturbation, caused by the child's own mutation, might degrade the normal hormonal signalling that takes place between the placenta and various regions of the mother's brain, such as the hypothalamus, which regulate appetite, milk production in response to suckling, and maternal instincts.

The potential evolutionary implications of the effects of paternally expressed genes are fascinating and far-reaching. In the case of Peg3, researchers argue that offspring inheriting a functional copy of the gene from their father and thus benefiting from quality maternal care 'would themselves be both well provisioned and genetically predisposed towards good mothering when adult' (Curley et al., 2004). Ultimately, this transmits a father's genes to future generations, increasing his genetic fitness along the way.

Our species is, of course, not immune to the detrimental effects of disrupted

mouse placenta. *Molecular and Cellular Biology, 28*, 1104–1113.

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imprinting, especially in our brains. This is evident when we look at neurodevelopmental conditions resulting from events that upset the normal function or inheritance of imprinted genes. As an example, patients born with Prader–Willi syndrome tend to have delayed mental development, extremely poor appetite control (often leading to obesity) and a great propensity for temper tantrums (Cassidy & Driscoll, 2009; Tunnicliffe et al., 2014). This rare syndrome, which affects roughly one in 15,000 children, results from dysfunction of the 15q11-q13 cluster of imprinted genes specifically on the paternal copy of chromosome 15. This might occur in a variety of ways. Most often (~70 per cent of cases) the disruption is due to a massive accidental deletion of this gene cluster, or a mutation, taking place within the sperm cell that goes on to impregnate a female. In the remainder of patients, compromised function of the 15q11-q13 cluster might be brought on by maternal uniparental disomy, whereby individuals inherit both copies of the critical gene cluster, or the entire chromosome 15, from their mother. Regardless of its precise genetic aetiology, Prader–Willi syndrome is characterised by a lifelong silence of a collection of paternally expressed genes (Hasegawa et al., 2012).

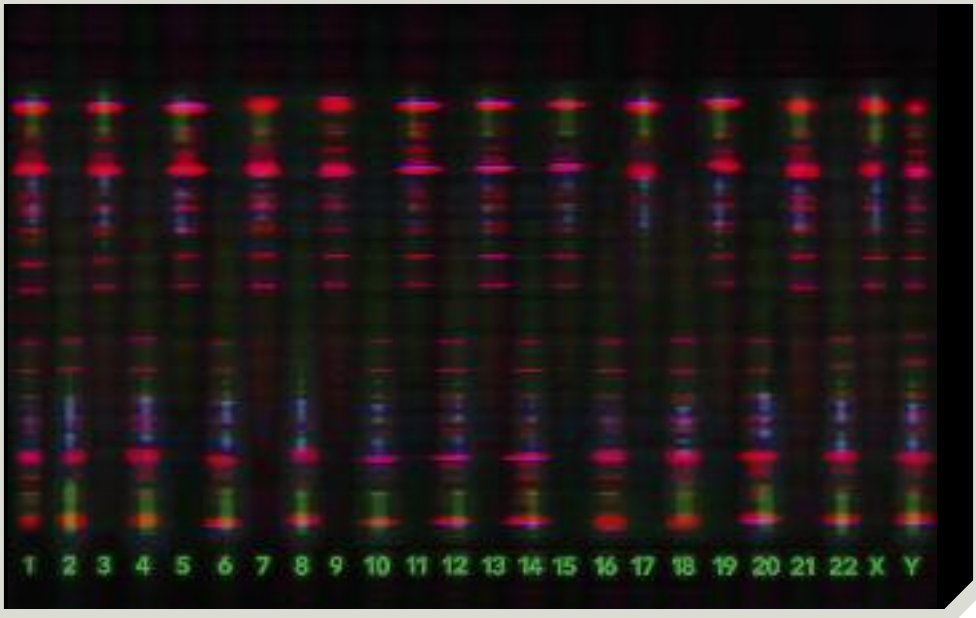
A different neurodevelopmental disorder, Angelman syndrome, often considered to be the 'sister' of Prader–Willi syndrome, is caused by the reverse genetic disruption (Cassidy et al., 2000; Mabb et al., 2011). From the moment of conception, patients with this disorder suffer from an absence of maternally expressed genes on the exact same imprinted gene cluster 15q11-q13. This can result from the occurrence of mutations on the maternal copy of chromosome 15, which disrupts activity of the relevant imprinted genes. In rarer cases, the syndrome can be caused by the inheritance of both copies of chromosome 15 from the father, which means that the imprinted genes which tend to be maternally active remain mute throughout the child's life (Poyatos et al., 2002). Children with Angelman syndrome tend to present with severe developmental delays, stereotypical jerky movements, speech impairments and an inappropriately friendly demeanour, which inspired the disorder's original, and somewhat demeaning, name of 'happy puppet syndrome' (Williams & Frias, 1982).

A more widespread role?

The phenotypes of both Prader–Willi and Angelman syndromes are extremely complex, rooted in disruptions of multiple genes involved in endocrine function, neuronal plasticity and the differentiation and migration of inhibitory interneurons during cortical development (Hasegawa et al., 2012; Muscatelli et al., 2000; Sato & Stryker, 2010). Collectively, these perturbations can explain several facets of these disorders, including deficient appetite control, learning disability and proneness to epileptic seizures. But importantly, they also highlight the severely detrimental outcomes of disturbing the function of imprinted genes, many of which have widespread effects on the developing brain.

In fact, recent explorations suggest that the influence of imprinted genes on brain and behaviour might be more far-reaching than previously imagined. Some theorists have argued that imprinting dysfunctions might have a role to play in some more widespread disorders, such as autism and psychosis (Badcock, 2011; Skuse, 2000). Only several months ago, a publication in the high-profile journal *Cell* reported that researchers examining neural cultures derived from the stem cells of severely autistic patients found robust over-activation of the imprinted gene FOXG1, which positively correlated with symptom severity (Mariani et al., 2015). Although the evidence for an association between autism and deficient imprinting isn't entirely consistent across population studies, and it's clear that imprinted genes are droplets in the ocean of genes already implicated in this disorder, the association is perhaps something worth our attention and research efforts. Ultimately, it's quite possible that our vulnerability to various neurodevelopmental conditions is an inevitable price to pay for the evolutionary battle of the sexes taking their competition to our brains.

It is comparatively early days for this area of research. We have yet to understand just how often imprinted genes lie at the root of various neurodevelopmental and psychiatric conditions, as well as how strongly they might be contributing to these phenotypes alongside non-imprinted genes that make up the bulk of our genome. It's also important to acknowledge alternative explanations for the origins of imprinting that do not invoke parental conflict (eg. Spencer & Clark, 2014). Ultimately, one notable strength of the conflict theory is that we have the opportunity to test its predictions across a range of species with differing levels of partner exchange – something that can be expected to produce varying levels of conflict between the parental genomes. I would encourage readers to try personally picking apart this fascinating scientific battleground, by familiarising themselves with Haig's (1997, 2000) conflict-based framework, as well as its competition (Spencer & Clark, 2014).



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