RNA is complementary to the DNA sequence from which it is transcribed. Therefore, interactions between DNA and RNA provide a simple mechanism of genetic self-detection within nuclei. Imprinted RNAs could enable alleles of maternal and paternal origin to detect whether they are the same (homozygous) or different (heterozygous), and thereby provide strategic information about expected relatedness to siblings.

'The situations in which a species discriminates in its social behaviour tend to evolve and multiply in such a way that the coefficients of relationship involved in each situation become more nearly determinate.' [1, p. 24]

1. Introduction

The probabilities that factor into calculations of relatedness can be parsed into probabilities, given a genealogy and uncertainties of genealogy. Genealogy may be uncertain, for instance, because a littermate is sometimes a full-sib, sometimes a half-sib, or because a herd contains kin of different degrees, but members cannot distinguish the categories [2]. Hamilton proposed that natural selection favours reduction of genealogical uncertainty. He further proposed that natural selection favours ‘discrimination of those individuals which do carry one or both of the behaviour-causing genes from those which do not’ ([1], pp. 24–25). Here, he considered the possibility of genes ‘recognizing’ their own copies and directing benefits on the basis of this privileged information [1].

Dawkins called gene-based discrimination ‘the Green-Beard Altruism Effect’. He envisaged a gene that encoded both a phenotypic label, the green beard, and the tendency to be nice to green-bearded individuals [3]. Kinship is a major cause of identity by kind but the label is independent of genealogy. Green-beard effects were considered implausible because genetic self-recognition and altruism were viewed as complex behaviours unlikely to be encoded by a single gene or tightly linked cluster of genes. Kinship seemed a much stronger basis for altruism. The situation is reversed at the genic level. It is much simpler to imagine a gene, or its products, preferentially interacting with identical genes, or their products, than to envisage a gene that recognizes half-cousins [4,5].

The interaction between labels of genic identity (green beards) and parental origin (genomic imprinting) makes possible a novel form of discrimination. I first review effects of genomic imprinting on estimates of genic relatedness, then describe the unusual evolutionary properties of imprinted green beards.

2. Parent-specific relatedness

The standard way to calculate the probability that a gene in A has an identical-by-descent copy in B is to multiply by one-half for each generation back from A to a common ancestor C, by one-half for each generation forward from C to B, and then sum these products for all distinct paths linking A to B. Ascending and descending factors of one-half arise from different sources of uncertainty. For
3. Imprinted green beards

Complementarity between the strands of a double helix, and between DNA and the RNA transcribed from its sequence, allow allele-specific interactions within nuclei between different copies of the same gene. A diverse fauna of small RNAs viewed as a simple form of genetic self-recognition that makes possible intranuclear green-beard effects.

24-nucleotide small-interfering RNAs (siRNAs) of Arabidopsis cause RNA-directed DNA methylation (RdDM) and transcriptional silencing of DNA sequences with motifs complementary to the siRNA. The template for synthesis of siRNAs is probably the sequence subject to RdDM [8]. Dosage-sensitive responses to an siRNA would allow a sequence to ‘count’ its copies within a nucleus. Imprinted expression of an siRNA would allow alleles of maternal origin to signal their presence to alleles of paternal origin or vice versa. A silent allele can ‘hear’ what the other allele has to say.

An abundant class of maternally expressed siRNAs (m esiRNAs) are expressed from madumal (maternally derived) chromosomes of endosperm [9]. MesiRNAs target genes that delay onset of endosperm cellularization and prolong endosperm proliferation [10], consistent with theoretical predictions that maternally expressed imprinted genes should inhibit endosperm growth [11].

Consider the introduction of m esiDNA (a motif that encodes m esiRNA) into a previously unimprinted gene encoding a growth enhancer. Transcription of Am, the established allele without m esiDNA, is expected to be a compromise between a lower level favoured as a madumal allele and higher level favoured as a padumal (paternally derived) allele. By contrast, A′, the initially rare allele with m esiDNA, will be transcribed at the same level as A when it is a padumal allele because the siRNA is not expressed, but at lower levels than A when it is a madumal allele because the siRNA is expressed. Thus, A′ behaves as a padumally silent allele when heterozygous. It will increase in frequency at the expense of A because it makes finer discriminations of relatedness.

By contrast to its behaviour in heterozygotes, A′ mRNA is transcribed from neither allele in homozygotes, because both alleles are silenced by the m esiRNA. The siRNA produced by madumal A′ inforces padumal A′ that the seed contains an A′A′ embryo rather than an AA′ embryo and that the mother carries at least one copy of A′. Therefore, at least half of other embryos will receive A′ from their mother (in addition to those that receive A′ from their father). Thus, m esiRNA signals a doubling of ‘relatedness’ to self and increased ‘relatedness’ to littermates (rt). If rt more than doubles, the balance of benefits to self and costs to littermates for padumal A′ shifts in favour of production of less growth enhancer, as occurs in the presence of m esiRNA.

Whether rt is doubled will depend on allele frequencies, the frequency of selfing and the number of fathers per brood. A complete analysis of this problem is beyond the scope of this letter, although some insight can be gained by considering effects when A′ is rare. In an outbreeding population, A′ will be transmitted predominantly by AA′ parents, with padumal A′ expressed when mothers are AA but inhibited by m esiRNA in 50% of seeds when mothers are AA′. MesiRNA signals a doubling of rt for single paternity of a mother’s offspring and more than doubling for multiple paternity. Therefore a reduction in seed size, with concomitant increase in seed number, would appear to benefit A′. MesiRNA functions as a ‘secret hand-shake’ that allows padumal A′ to recognize its allelic partner as self and to reduce its own transcription for the benefit of madumal A′ in littermates.

MesiRNA could also function as an adaptive signal of self-fertilization. Selfing shifts the optimal trade-off between...
seed size and number to smaller seeds for genes expressed in filial tissues [12]. If mothers sometimes self, padumnal A’ will be more likely to encounter mesiRNA in selfed seeds than outcrossed seeds, especially when A’ is rare. Therefore, padumnal A’ will promote smaller seeds on selfing and larger seeds when outcrossed.

A’ produces less mRNA in homozygotes than is optimal for an unimprinted allele expressed in offspring. Therefore, near fixation of A’, rare alleles (such as A) that are expressed more than A’ will be favored by natural selection. This suggests that A and A’ will be maintained at a polymorphic equilibrium.

Green-beard altruism is vulnerable to ‘cheats’ who flaunt the label, receive its benefits, but do not reciprocate [13]. A’, a version of A that retains mesiRNA but is insensitive to its inhibitory action, would increase in frequency at the expense of A because padumnal A’ induces padumnal A’ to reduce demand, benefiting A’, but A’ does not reciprocate when padumnal and padumnal roles are reversed. Once A’ eliminates A’, the population is primed for the introduction of A*, an allele that possesses a beard of a different colour (new mesiRNA) [14]. Such an iterative process (figure 1), in which successively introduced mesiRNAs are only transiently effective, could explain the diversity of mesiRNAs, their rapid evolutionary turnover and mild effects [9,15].

Imprinted gene clusters of mammals contain many non-coding RNAs [16]. DNA–DNA associations and RNA–DNA interactions within these clusters may be facilitated by somatic pairing of madumnal and padumnal chromosomes [17,18]. MicroRNAs processed from maternally expressed antiPeg11 cause mRNA degradation of paternally expressed Peg11 [19]. Mutations of madumnal Rasgrf1 silence the padumnal copy of a neighbouring non-coding RNA [20]. Disruption of madumnal Ube3a upregulates padumnal Ube3a-antisense [21]. Such examples suggest the possibility of green-beard effects at mammalian imprinted loci.

References