PLANT DEVELOPMENT

Parental conflict overcome

Nir Ohad

In flowering plants, viable seeds result even without two of the mechanisms that normally operate during embryogenesis. This finding illuminates the interplay of male and female factors in the process.

In flowering plants, two identical male gametes from the same pollen grain fuse in the ovule with two female gametes, initiating development of the embryo and the endosperm — the tissue that nourishes the embryo, equivalent to the placenta in mammals. As they describe on page 312 of this issue, Nowack *et al.*[1] find that when only one male gamete triggers embryogenesis, a viable seed can develop if the maternal mechanism controlling endosperm development is disabled. This finding reveals a new layer of conflicting interaction between the maternal and paternal genomes in regulating plant embryogenesis, and supports an early theory concerning endosperm evolution.

Whereas a single fertilization event initiates embryogenesis in animals, in flowering plants two genetically identical gametes from the same pollen grain fertilize the haploid egg cell (chromosome complement n) and the diploid ($2n$) central cell within the ovule. The result is a diploid ($2n$) embryo and a triploid ($3n$) endosperm (Fig. 1a). *In vitro* systems have been used to study the double-fertilization process, but no such *in vivo* studies have been successful so far.

Nowack and co-workers, however, have dissected the regulatory mechanism controlling endosperm and embryo development *in vivo*, and show that seeds with uniparental endosperm of maternal origin are viable. This was achieved using a novel mutant of a model plant, *Arabidopsis thaliana*, that is impaired in the *CDKA;1* gene. Instead of the normal two male gametes, only one gamete develops in the *cdka;1* mutant pollen, which exclusively fertilizes the egg cell[2]. Using the *cdka;1* mutant as a pollen donor, the authors previously showed that this fertilization of the egg cell induces the development of a diploid embryo[2]. And even though the second fertilization event did not take place, endosperm developed. However, this 'homodiploid' ($2n$) endosperm was abnormal and the seeds aborted (Fig. 1b). Endosperm development in the absence of the second fertilization event led the authors[2] to conclude that the fertilized egg cell may provide a signal that can promote the initial stages of this process, thus overcoming the maternal repression within the central cell. Normally, the second fertilization event overcomes this repression.

Such maternal repressive mechanisms act epigenetically. That is, they involve DNA methylation[3] and modification of the histone proteins associated with DNA[4–6], a process partly mediated by complexes of the Polycomb group proteins (PcG)[3–5,7]. Mutations in maternal PcG members (*fie*[8], *fis2*[9] or *mea*[10]) also lead to the proliferation of abnormal ($2n$) endosperm in the absence of fertilization (Fig. 1c). If fertilization does take place in these mutants, normal diploid embryo and abnormal triploid endosperm develop, leading to seed abortion.

Nowack and co-workers[1] have taken dissection of the double-fertilization process a step further, addressing the question of how far the embryo can develop in the absence of the second fertilization event. When the *cdka;1* mutant was used as a pollen donor to fertilize female mutant plants impaired in *fie*, *fis2* or *mea*, homomaternal diploid ($2n$) endosperm developed. Surprisingly, this endosperm developed normally, giving rise to a seed that could germinate and produce a viable plant (although of reduced stature) (Fig. 1d). These results show that even when only the egg cell is fertilized, seed development can be completed if maternal repression of central-cell division is bypassed. This situation occurs when the paternal genome does not contribute genetic input to the endosperm and when the maternal PcG machinery restricting endosperm proliferation is impaired. Remarkably, under such

GENETICS

Run, whippet, run

Whippets are fast — these dogs can run at speeds of more than 60 kilometres per hour, and have long been used in racing. But, recently, owners have been reporting an increase in 'double muscling', a trait characterized by cramping in the shoulders and thighs of otherwise healthy whippets. Dana Mosher and colleagues now show that a mutation in the *MSTN* gene is the culprit (D. S. Mosher *et al. PLoS Genet.* e79.eor doi:10.1371/journal.pgen.0030079.eor; 2007).

The *MSTN* gene encodes myostatin, a protein that affects muscle composition. Mosher *et al.* analysed the sequence of *MSTN* from three categories of whippet: the heavy-muscled 'bully' whippets, which have double muscling; parents of bully whippets; and normal whippets unrelated to bullies.

They found that all bully whippets carry a mutation in both copies of the *MSTN* gene, and all bully-whippet parents have the same mutation in one copy of this gene. This mutation involves deletion of only two DNA base pairs, resulting in premature termination of messenger RNA translation and a truncated form of myostatin.

Looking for a correlation between this *MSTN* mutation and whippets' racing performance, the authors observed a significant association between the dogs' speed, their muscle mass and their genetic profile. Whippets with one mutated copy of *MSTN* were generally the fastest, followed by normal animals. Bully whippets are rarely raced, probably because they are handicapped by an excess of muscle. The increase in the incidence of the *MSTN* mutation is probably due to selective breeding of whippets.

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Mosher *et al.* did not detect this mutation in 14 other heavy-muscled breeds of dog, suggesting that it might be unique to whippets. There is the question of whether a similar mutation in humans could be exploited to enhance athletic performance. However, the authors caution that effects of the *MSTN* mutation on other aspects of health are unknown.

Sadaf Shadan