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Cytoplasmic male sterility and mitochondrial metabolism in plants

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ABSTRACT

Cytoplasmic male sterility (CMS) is a common feature encountered in plant species. It is the result of a genomic conflict between the mitochondrial and the nuclear genomes. CMS is caused by mitochondrial encoded factors which can be counteracted by nuclear encoded factors restoring male fertility. Despite extensive work, the molecular mechanism of male sterility still remains unknown. Several studies have suggested the involvement of respiration on the disruption of pollen production through an energy deficiency. By comparing recent works on CMS and respiratory mutants, we suggest that the "ATP hypothesis" might not be as obvious as previously suggested.

1. Introduction on cytoplasmic male sterility

The occurrence of plants that lost their ability to produce viable pollen grains is frequent in hermaphroditic angiosperm species. As this is usually due to factors coded by the mitochondrial genome, this phenomenon is called cytoplasmic male sterility (CMS). CMS has been extensively exploited in hybrid breeding of crop species. Male sterility is in fact the product of the interaction between the mitochondrial genome and the nuclear genome that can encode for male fertility restorers, which specifically counteract the effect of the mitochondrial sterilizing factors. The link between the mitochondrial molecular phenotype, mitochondrial physiology and pollen sterility remains unknown. In this review, we are comparing recent works on the characterisation of respiratory mutants and CMS lines and their restorers to identify potential mechanisms leading to male sterility.

2. Evolutionary aspects

The evolutionary dynamics happening in species can be understood in light of the concept of genetic conflict between two genomes that do not share the same heredity, maternal for the mitochondrial genome, bi-parental for the nuclear genome (Cosmides and Tooby, 1981). Any mitochondrial gene that favors its own transmission will thus be selected, even at the expense of the nucleus. The evolutionary forces that enable the maintenance of such sexual polymorphism in populations have been investigated in theoretical and empirical studies. Since CMS has gone through the sieve of natural selection, female (male-sterile) plants carrying a sterilizing mitochondrial genome are expected to have a selective advantage that has been called female advantage: more and/or better seeds than her hermaphroditic counterparts. It can be due to the reallocation of the energy saved from pollen production or to the avoidance of inbreeding depression by selfing since females are obligate outcrossers (Dufay and Billard, 2012; Shykoff et al., 2003). Many species exhibit mitochondrial genome diversity with sterilizing and non-sterilizing ("normal") genomes. Theoretical work shows that the maintenance of CMS and non CMS genomes in populations is possible if the sterilizing genome is costly for restored hermaphrodite i.e. if they produce less or lower quality seeds than hermaphrodites carrying a "normal" cytoplasm (Dufay et al., 2007). Last, restorer alleles are expected to bear a cost when they are on the "wrong" cytoplasm i.e. a non-sterilizing cytoplasm or CMS that they cannot restore (Delph et al., 2007). Given these conditions, CMS is predicted to be under a form of selection called balancing selection, under which sterilizing mitochondrial genomes and restorer loci are favored when they are rare, enabling their maintenance for a long period of time (Charlesworth, 2002; Delph and Kelly, 2014; Lahiani et al., 2013).

In conclusion, the mitochondrial dysfunction generated by CMS must be overcome at the seed level. Sterile genes must favor mitochondrial transmission (seed production) and thus affect only pollen production. However as a CMS-associated cost is expected, it can theoretically have mild effects on the overall plant physiology but it should, in any cases, not cause growth retardation.

3. Molecular mechanism of CMS

Studies aiming to identify mitochondrial and nuclear genes involved in CMS have revealed the diversity of mitochondrial sterilizing genes as well as the mechanisms by which restorer genes act (Table 1). This

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Table 1
Mitochondrial male-sterile genes and nuclear male fertility restoration genes.

Species	CMS	CMS gene (sequences from mt genes when chimeric)	Co-transcribed mt gene	CMS gene action	Restorer gene(s)	Restorer effect on CMS factor	References
Bean		*pvs*	–	–	unknown	mt genome rearrangement (Fr), posttranslationnal (Fr2)	(Mackenzie and Chase, 1990)
Beet	CMS-Owen	*preSatp6*?	*atp6*	unknown	Oma1 like (Rf1/X)	Protein–protein interaction?	(Yamamoto et al., 2005) (Matsuhira et al., 2012)
	CMS-E/I-12CMS(3)	*orf129* (*cox2*)	–	unknown	unknown	–	(Yamamoto et al., 2008) (Darracq et al., 2011)
	CMS-G	*G-cox2*?	–	Complex IV dysfunction?	unknown	–	(Darracq et al., 2011; Ducos et al., 2001)
Brassica	Nap	*orf222* (*atp8*)	*nad5c*, *orf139*	–	unknown	Transcript level control	(L'Homme et al., 1997)
	Pol	*orf224* (*atp8*)	*atp6*	–	unknown	Transcript level control	(L'Homme et al., 1997)
Chili pepper		*orf456*	*cox2*	–	–	–	(Kim et al., 2007)
Maize	CMS-C	unknown	–	ROS accumulation, PCD[a]	unknown	–	(Huang et al., 2012)
	CMS-S	*orf355*/*orf77* (*atp9*, *atp4*)	*atp9*		unknown	RNA degradation	(Zabala et al., 1997) (Xiao et al., 2006)
	CMS-T	*T-urf13*	*atp4*	Forming a pore in the inner mitochondrial membrane	ALDH (Rf2)	Detoxification? (Rf2) T-urf13 mRNA control (Rf1)	(Rhoads et al., 1995) (Cui et al., 1996)
Petunia		*Pcf* (*atp9*, *cox2*)	*nad3*	–	PPR (RF-PPR592)	Interaction with *pcf* RNA in a large protein complex	(Bentolila et al., 2002) (Gillman et al., 2007)
Radish	Ogura	*orf138*	*atp8*	Forming a pore in the inner mitochondrial membrane	PPR (Rfo)	Interaction with *orf138* mRNA	(Bellaoui et al., 1999; Duroc et al., 2009) (Brown et al., 2003) (Desloire et al., 2003) (Koizuka et al., 2003) (Uyttewaal et al., 2008)
Rice	BT	*orf79* (*cox1*, *cox2*)	*atp6*	cytotoxic	PPRs (RF1a and Rf1b)	Processing of *orf79-atp6* transcript	(Akagi et al., 1994; Kazama et al., 2008)
	WA	*WA352* (*orf284*, *orf288*)	*rpl5*	Interaction with Complex IV	unknown	Post-transcriptionally (Rf4) and post-translationally (Rf3)	(Luo et al., 2013)
	HL	*orfH79* (*cox1*, *cox2*)	*atp6*	Interaction with complex III	PPR (Rf5)	*atp6-orfH79* RNA processing through the binding of a glycin-rich protein	(Wang et al., 2013) (Hu et al., 2012)
	CW	unknown	–	–	Retrograde Male Sterility gene (Rf17)	Loss of function allele of RMS restores male fertility	(Fujii and Toriyama, 2009)
	LD	unknown	–	–	Glycin Rich Protein (Rf2)	CMS–protein–protein interaction?	(Itabashi et al., 2011)
Sorghum	A3	*orf107* (*atp9*, *BT-orf79*)	–	–	unknown	Transcript processing	(Tang et al., 1996)
Sunflower	PET1	*orf522* (*atp8*)	*atpA*	ATPase activity reduction, PCD	unknown	*atpA-orf522* transcript degradation through polyadenylation	(Balk and Leaver, 2001) (Sabar et al., 2003) (Gagliardi and Leaver, 1999)
Wheat	*timopheevi*	*orf256* (*cox1*)	*cox1*	–	unknown	Transcript processing?	(Hedgcoth et al., 2002)

[a] PCD: Programmed Cell Death.