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Review Article

RNA EDITING AND ITS ROLE IN CROP IMPROVEMENT: A REVIEW

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Abstract: RNA editing is a posttranscriptional modification to nuclear, mitochondrial or chloroplast genome-encoded transcripts and distinct from the events of RNA splicing, capping or polyadenylation and occurs in a wide range of organisms. It was discovered in 1986 in Trypanosoma brucei where uridines were inserted at specific sites in the mitochondrial (kinetoplast) cytochrome C oxidase II (coxII) transcript to restore the proper protein-coding sequence mediated by guide RNA (gRNA), followed by a report that described deletion of uridines in coxIII mRNA. RNA editing by cytidine (C) to uridine (U) conversions is widespread in plant mitochondria and was discovered in chloroplasts two years later. In some plant taxa, "reverse" U-to-C editing also occurs in flowering plants. In Arabidopsis, 43 sites are edited in the chloroplast and 619 in the mitochondria. In many cases, amino acid alterations caused by RNA editing are essential for the expression of functional proteins. Most RNA editing events can restore the evolutionarily conserved amino acid residues in mRNAs or create translation start and stop codons. Therefore, RNA editing is an essential process to maintain genetic information at the RNA level. RNA editing expands the genetic information, thus making the environment more adaptable to the organisms. RNA editing played an important role in the normal mitochondrial function. Cytoplasmic male sterility was induced after transferring unedited atp9 gene into fertile tobacco, and this confirmed the correlation between the RNA editing of atp9 gene and tobacco cytoplasmic male sterility. Individual RNA editing sites are recognized by plant-specific pentatricopeptide repeat (PPR) proteins that are encoded in the nuclear genome. These PPR proteins are characterized by repeat elements that bind specifically to RNA sequences upstream of target editing sites and rapid expansion in number of PPR genes permitted RNA editing on a large scale.

Key words: RNA, Crop improvement

INTRODUCTION

The most important discoveries concerning modified nucleosides in nucleic acids (mostly RNA) before 1948, naturally occurring nucleic acid polymers (DNA and RNA) were thought to contain only four canonical nucleosides: the ribo or deoxyribo derivatives of adenine, cytidine, guanine and uracil or thymine . Hotchkiss [1] reported the first evidence for presence of trace amounts of a non-canonical nucleoside in DNA. This nucleoside was identified as deoxy 5-methylcytosine (dm5C) [2]. Soon after Cohn and Volkin [3] also detected small amounts of another compound designated as pseudouridine (abbreviated in Ψ). Pseudouridine results from enzymatic isomerisation of the genetically encoded U into Ψ, catalyzed by RNA pseudouridine synthases. To date, many distinct RNA pseudouridine synthases

have been identified and several of them (mostly from *Escherichia coli*) have been obtained in crystallized forms [4]. Soon after the discovery of Ψ in RNA, a great deal of efforts have been made in many laboratories (1950-60) to identify other 'rare ' or 'minor' nucleosides in RNA. They were 2'-*O*-methylribose derivatives (Cm, Gm, Um, Am), 5-methylribouridine (m5U, also named riboT) and 5-methylribocytosine (m5C). Between 1955, at least 35 well-characterized modified nucleosides had been identified in both DNA (in fact only dm5C at that time) and RNA (34 new structures) (Fig.1). These included inosine (a deaminated form of adenosine abbreviated in I. The development of methods for purifying individual RNA species (isoacceptor tRNAs, different rRNAs, and later also various snRNA) from diverse organisms of the three domains of life (Bacteria, Archaea, and Eukarya) and methods for sequencing them, more than 100 modified nucleosides have been identified. Among them, over 80 distinct modified nucleosides have been found to occur naturally in tRNAs and about an additional 20 were shown to be present in other types of RNA (rRNA, mRNA, snNA, and even chromosomal RNA). Information about modified nucleosides is generally limited and only a few examples of modified nucleosides, the most popular one being inosine because of its presence in the anticodon of yeast tRNA (anticodon IGC), the first tRNA that was sequenced [5]. The famous Wobble Hypothesis stating that inosine in position 34 of tRNA could base pair with A in the third position of a codon has been only recently demonstrated [6].

RNA editing in plant organelles: RNA editing sites in translated regions can be predicted by a comparison of amino acid sequences deduced from genomic DNA sequences from various plant species. Subsequently, RNA editing can be verified by cDNA sequence analysis. A number of editing sites identified in various land plant mitochondria and chloroplasts are listed in Table 1. There are 20 to 60 editing sites in chloroplasts and 300 to 600 sites in mitochondria of most flowering plants. In seed plants, all these editing events are of C-to-U type. Most of the sites in translated regions are efficiently edited, with 90%–100% efficiency, in green leaves. On the other hand, the efficiency of C-to-U editing events that create a translation initiation codon (by an ACG to AUG change) has been surprisingly low. For instance, the editing efficiency at the ndhD-1 site in the Arabidopsis chloroplast ndhD transcript is 45% and that of the rps14-C2 site in the moss *Physcomitrella* patens chloroplast rps14 mRNA is 70% in filamentous *protonemata*, which reduces further to only 20% in leafy tissues. This suggests that editing at this site may regulate translation in chloroplasts. RNA editing efficiency varies in different tissues and organs, developmental stages, or different mutant lines. Recent high-throughput RNA-seq analyses have revealed minor RNA editing events in untranslated regions and intron sequences as well as in protein-coding regions. For instance, in addition to the 34 already known editing sites in Arabidopsis chloroplasts, nine novel sites have been identified that are edited at a low level (5% to 12%). Among the 635 identified editing sites in Nicotiana tabacum mitochondria, five sites are in tRNAs and 73 in non-coding regions. Across the plant kingdom, the total number of C-to-U editing sites in chloroplasts varies from 0 in the liverwort Marchantia polymorpha to 3415 in the spike moss *Selaginella uncinata*. Out of 3415 sites identified in 74 S. uncinata chloroplast mRNAs, 428 are silent editing events, 74 have been identified in four group II introns, 52 create start codons and 31 create stop codons. A total of 2139 editing sites in 18 mRNAs were identified in *S. moellendorffii* mitochondria. Of these, 424 are silent, whereas the others result in 1488 codon changes. In addition, 13 sites are in the two rRNAs. To date, RNA editing sites can be predicted by plant RNA-editing prediction and analysis computer tools PREPACT 2.0 and PREP-Mt. Some 1800 C-to-U editing sites have been predicted in the *S. moellendorffii* chloroplast, 460 sites in the quillwort *Isoetes flaccida* (chloroplast) and 340 sites in *Huperzia lucidula* chloroplasts. Therefore, the organellar transcripts in *Selaginella*, one of the early vascular plant lycopods, seem to be most commonly edited. In case of the bryophyte (early non-vascular land plants) *P. patens*, where there are only two identified C-to-U editing sites in chloroplasts, there are 11 such site in its mitochondria. On the other hand, hornworts such as *Anthoceros* and *Phaeoceros laevis* undergo substantial RNA editing However, no editing event has so far been reported in green algae, including *C. vulgaris* (stonewort), suggesting that the process of RNA editing may have evolved only after the plants established themselves on the land.

Factors in plant organelles responsible for RNA editing:

PPR (pentatricopeptide repeat) proteins as a

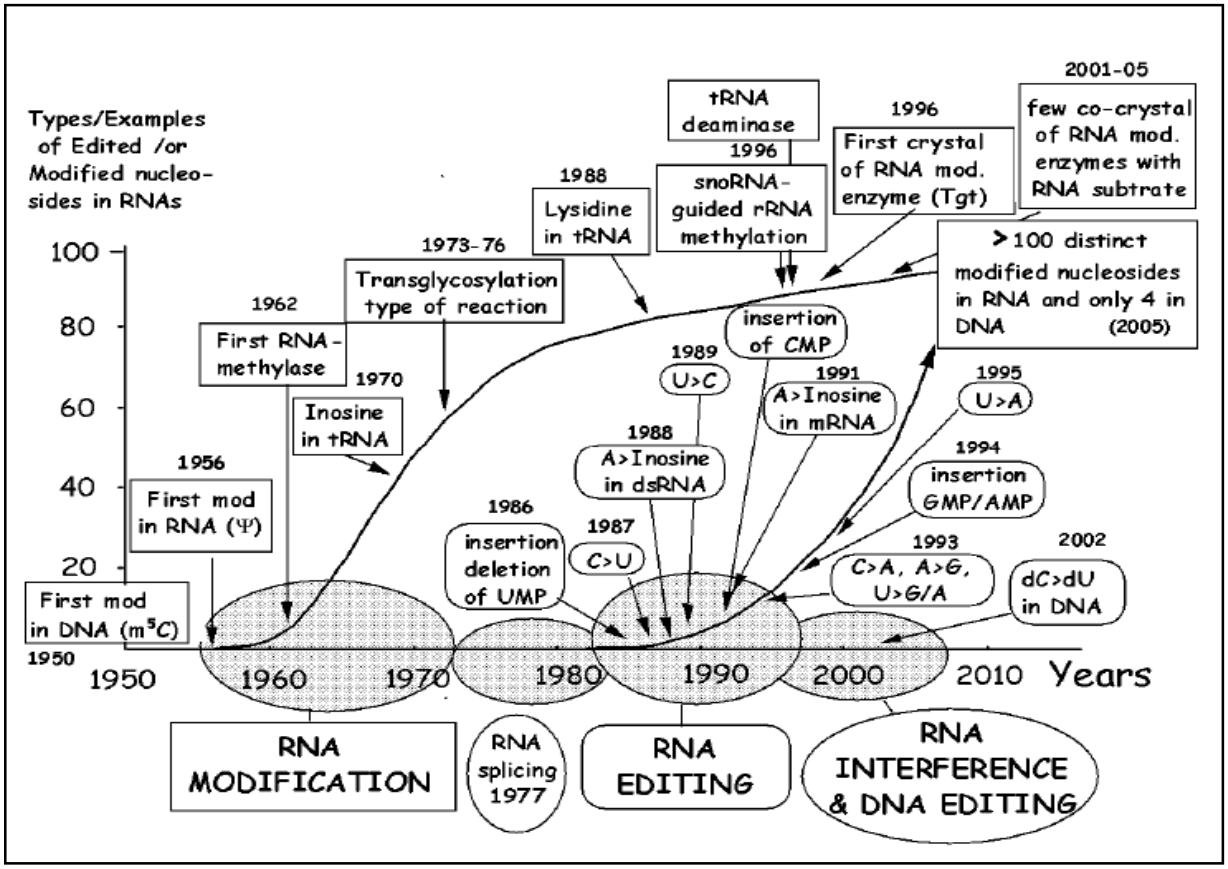

Fig. 1: Milestones discoveries related to post-transcriptional modification, splicing, editing and interference of nucleic acids (DNA and RNA). Gray circles correspond to the various periods of the greatest scientific excitements concerning due to the novelty of the discoveries: 1) the identification of numerous modified nucleosides in RNA hydrolysates and in newly sequenced RNAs (period 1955-1970); 2) the discovery of intron splicing phenomena (period 1975-1985); 3) the RNA Editing phenomena (period 1985-1995), and 4) more recently the discoveries related to RNA interference and DNA editing processes (period 1995 - present). (Henri Grosjean 2005)

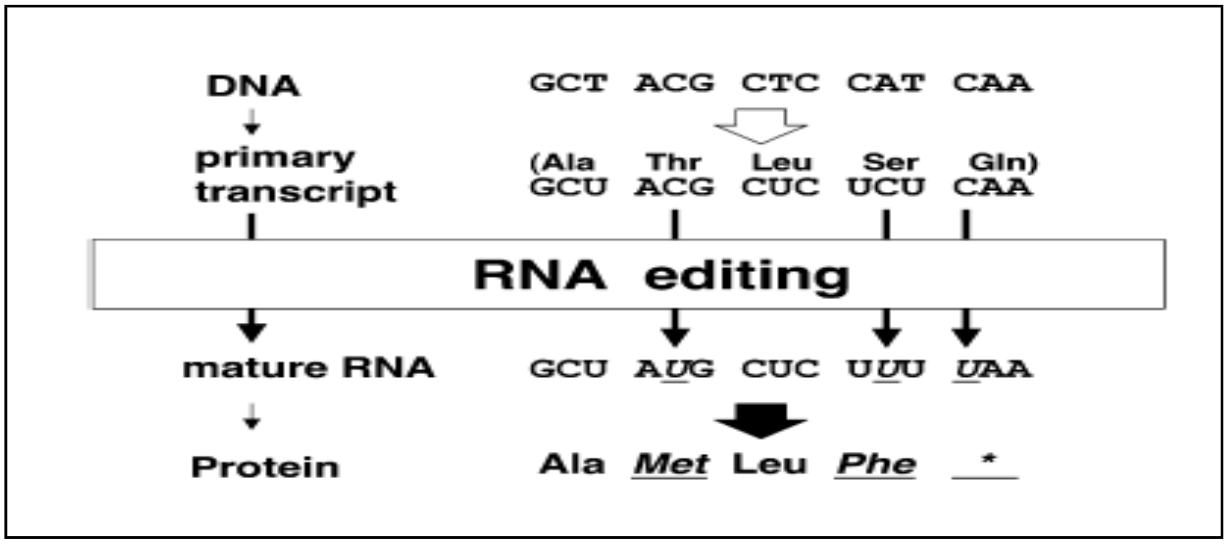

Fig 2. RNA editing in plant mitochondria which changes selected cytidines (C) in the primary transcript to uridines (U) in the mature mRNA (Gene regulation). Ichinose and Sugita [7].

Table 1. The numbers of RNA editing sites in chloroplasts and plant mitochondria. Ichinose and Sugita [7].

Plant species (common name)	RNA editing type	
	C-to-U	U-to-C
Chloroplasts		
Seed plants (monocotyledonous angiosperms)		
Oryza sativum (rice)	21	0
Zea mays (Maize)	26	0
Seed plants (dicotyledonous angiosperms)		
Arabidopsis thaliana (Thale cress)	43	0
Nicotiana tabacum (Tabacco)	34	0
Cucumis sativus (cucumber)	51	0
Seed plants (Gymnosperms)		
Cycas taitugensis (Emperor sago)	85	0
Ferns		
Adiantum capillus-veneris (southern maidenhair fern)	315	35
Ophioglossum californicum (California adder's tongue fern)	297	3
Psilotum nudum (whisk fern)	27	0
Lycophytes		
Selaginella uncinata (spike moss)	3415	0
Bryophytes		
Anthoceros angustus (hornwort)	509	433
Physcomitrella patens (moss)	2	0
Marchantia polymorpha (liverwort)	0	0
Plant species (common name)	**RNA editing type**	
	C-to-U	**U-to-C**
Mitochondria		
Seed plants (monocotyledonous angiosperms)		
Oryza sativum	491	0
Seed plants (dicotyledonous angiosperms)	619	0
Arabidopsis thaliana		
Brassica napus L. (rapeseed)	427	0
Beta vulgaris (sugarbeet)	357	0
Phoenix dactylifera L. (date palm)	445	0
Seed plants (Gymnosperms)		
Cycas taitugensis	565	0
Lycophytes		
Isoetes engelmannii (Engelmans quillwort)	1560	0
Selaginella moellendorffii (spike moss)	2152	0
Bryophytes		
Physcomitrella patens	11	0

site-recognition factor: PPR proteins constitute a large family of nuclear-encoded proteins comprising of 100 to over 1000 members in land plants. However, there number varies from only several to 20 members in fungi, protists, and animals. Almost all the PPR proteins are localized in either chloroplasts or mitochondria, or both where these proteins participate in different facets of RNA metabolism such as RNA splicing, RNA editing, RNA stability, and translational initiation. PPR proteins are characterized by tandem arrays of the degenerate 31 to 36-amino acid PPR motif that folds into a pair of anti-parallel alpha helix, which have been suggested to specifically bind to RNA sequence targets. To elucidate the molecular mechanism of RNA editing in plant organelles, *in vitro*, *in vivo*, and in organello studies have extensively been performed using flowering plants such as wheat, tobacco, pea, and cauliflower. These studies have helped in identifying cis-acting elements adjacent to editing sites and discovering putative site-

specific proteins that interact with these elements. In all such instances, the cis-elements comprise stretches of 20 to 25 nucleotides upstream of the editing sites. The hypothesis was that this cis-element was recognized by a trans-factor that recruited the RNA editing machinery to the site. It was also possible that this trans-factor was a component of the RNA editing machinery. Because many RNA editing sites are in protein coding regions, the sequences surrounding the sites are not highly conserved One. The trans-factor was finally discovered in *Arabidopsis chlororespiratory* reduction 4 (crr4) mutants defective in activity of the chloroplast NADH dehydrogenase-like (NDH) complex, which is a multi-subunit complex in the thylakoid membrane. The loss of NDH complex was correlated directly to the loss of a C-to-U editing event that otherwise creates the start codon AUG in ndhD mRNA. crr4 mutants are specifically defective in the RNA editing that generates the translational initiation codon of the ndhD gene. Because of this defect, crr4 mutants do not translate ndhD and consequently do not accumulate the chloroplast NDH complex. The defect was monitored as a specific alteration in chlorophyll fluorescence pattern. The CRR4, a member of the pentatricopeptide repeat (PPR) protein family, binds to a 36 nucleotides (-25 to +10) region surrounding its target editing site. This suggested that CRR4 could be the bona fide trans-acting factor essential for recognizing this RNA editing target site. Following this discovery, several other PPR proteins were identified as site recognition factors affecting editing in chloroplasts and mitochondria. Many editing PPR proteins were found to be responsible for only a single editing site, whereas, some PPR proteins could recognize multiple sites with similar cis-element sequences. The PPR family has an extraordinarily large number of members, especially in angiosperms. In *Arabidopsis*, the family contains approximately 450 members. The vast majority of fertility-restorer genes identified so far belong to the pentatricopeptide repeat (PPR) family [8]. In plants, two subfamilies of PPR genes can be distinguished based on the structure of the encoded protein: the P family contains canonical PPR domains characterized by the repetition of a 35-amino acid motif, and the PLS subfamily contains related shorter (S) and longer (L) motifs arranged as repeating triplets. PPR proteins have been shown to be sequence-specific RNA-binding. proteins potentially involved in different stages of organellar gene expression . PPR proteins are found in all eukaryotes and are particularly numerous in plants, ranging from 103 in the moss Physcomitrella patens to 450 and 477 in *Arabidopsis* and rice, respectively [9]. There is a remarkable coincidence between the occurrence of RNA editing and the phylogenetic distribution of the PLS subfamily in plants. In fact, several proteins involved in plant mitochondrial RNA editing have been recently identified as PPR proteins belonging to the PLS subfamily [10]. The PLS family is specific to land plants and is not present in the closest algal relatives, which do not undergo RNA editing. This raises the question of the involvement of PLS PPR in plant-specific processes. Moreover, some of these PPR proteins contain a domain with conserved amino acids that match the active site of nucleotide deaminases, leading to the hypothesis that one or several of these proteins could act as the catalytic factor for C-to-U conversions on organellar transcripts.

Role of RNA editing (RNA editing helps in regulation of gene expression): RNA editing in mitochondria of flowering plants is a posttranscriptional process which changes selected cytidines (C) in the primary transcript to uridines (U) in the mature mRNA. The amino acid sequence encoded by the fully edited, mature mRNA is different from the protein sequence encoded by the genomic DNA and the primary transcript. In this hypothetical sequence several examples of such amino acid changes are included. About 400–500 such C to U RNA editing events are observed in the mRNA and tRNA population in plant mitochondria of a given angiosperm species. Since only some of the cytidines are altered by RNA editing, For example, the introduction of an AUG translational start codon from an ACG codon could make an mRN rapidly accessible for translation. This would be much faster than the complete de novo synthesis of the affected transcript. (Fig 2) .

RNA editing affects tRNA maturation and RNA splicing: RNA editing in plant organelles mostly affects mRNAs, thus providing the means to correct genetic information for proper protein function. In addition, editing affects some tRNAs and rRNAs encoded in the organellar genomes. In bean and potato mitochondria, a C-to-U editing event corrects a C:A mismatch base pair into a U:A base pair in the acceptor stem of tRNA. In larch, three C-to-U editing events restore U:A base pairs in the acceptor, D and anticodon stem, respectively, in mitochondrial tRNA. In the lycophyte I. engelmanni mitochondria, ten

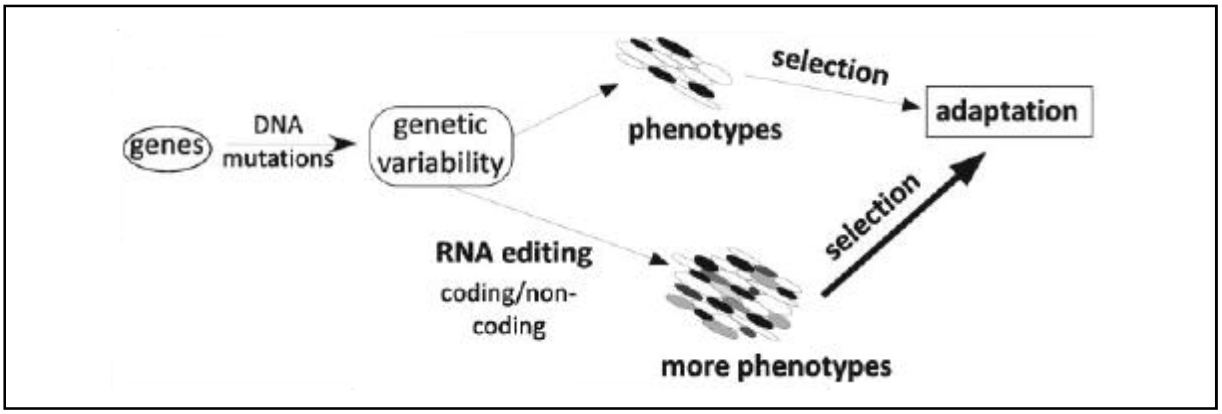

Fig 3. Basic mechanism for increased genetic variability through post-transcriptional RNA editing. Willemijn et al. [16].

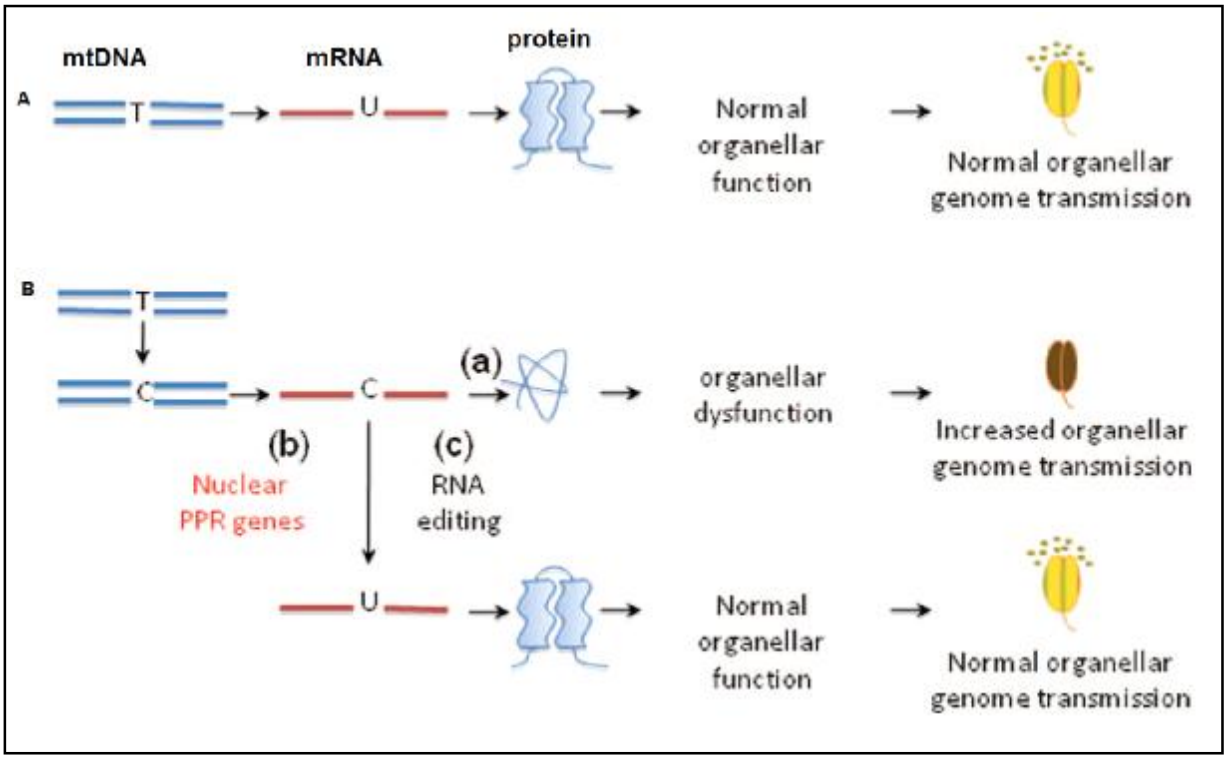

Fig. 4: (A) Before appearance of RNA editing, organellar genes encode for functional proteins leading to a wild-type organellar phenotype. Plants produce normal pollen and organellar and nuclear genomes transmission is balanced. (B) When T-to-C mutations are established in the organellar DNA, the corresponding mRNAs are translated into altered proteins (a). When mitochondrial genes are involved, mitochondrial dysfunction and pollen abortion (male sterile plant) can result, and the transmission of organellar genomes is increased. (b) The spread of these mutations in the offspring creates selective pressure to restore male function mobilizing nuclear PPR genes (c). Specialized PPR proteins participate in correcting the mutations at the RNA level by changing C into U (RNA editing). Translation of the corrected genetic information restores normal organellar function and the balance between the transmission of nuclear and organelle. Benoit and Alejandro [17].

tRNAs are edited to improve base pairing in stem regions. Thus, editing events in pre-tRNAs help in restoring the RNA secondary structure by removing mismatches in the double-stranded stem region and are a prerequisite for their processing into functional tRNAs. Also editing can improve the base pairing required for splicing. Self-splicing in vitro is observed only in the edited (A:U basepair) form, indicating that this editing event is a prerequisite for splicing thereby regulating the availability of functional RNAs. RNA editing in exons close to splice sites may also affect intron splicing or vice versa. For instance, the spinach

chloroplast ndhA mRNA is edited at two sites, one of which is located only 12 nucleotides downstream of the 30 intron-exon splice site. To assess if RNA editing occured after or before splicing, short "spliced" and "unspliced" ndhA gene fragments were introduced and transcribed within tobacco chloroplasts. The subsequent cDNA analysis showed that only spliced ndhA mRNAs were edited [7].

RNA editing leads to fertility restoration or deficiency of RNA editing leads to sterility: Cytoplasmic male sterility (CMS) is a widespread phenomenon observed in >150 flowering plant species. CMS is a maternally inherited trait and is often associated with unusual open reading frames (ORFs) found in mitochondrial genomes, and in many instances, male fertility can be restored specifically by nuclear-encoded, fertility restorer (Rf) genes. Rf genes are members of a recently defined large gene family encoding pentatricopeptide repeat (PPR)–containing proteins as a site-recognition factor for RNA editing. CMS/Rf systems have long been exploited for hybrid breeding to enhance the productivity of certain crops. The expression in transgenic tobacco plants of an exogenous wheat mitochondrial atp9 DNA sequence seems to have no effect on most phenotypic characters. The size, growth rate, node number, shape, and size of leaves and flowers are similar in transgenic and control plants. However, dramatic effects were observed at the level of male reproductive organs when unedited wheat atp9 sequence was expressed in tobacco plants. Indeed, transformation experiments performed with plasmid and unedited and edited forms of wheat atp9 coding sequences have shown that the chimeric genes are integrated into the nucleus of the host plant and expressed, as shown by Northern and immunoblot analyses. The plasmid construct bearing the unedited form of atp9 severely affects male fertility in tobacco plants. This important observation implies that the introduction of modified mitochondrial information, engineered to be addressed to the organelle, may induce male sterility through pollen abortion. This is an innovative way to introduce such a phenotype. Moreover, this approach constitutes an exciting model for studies concerning the function of the RNA editing process in plants. An atp6 mRNA-editing defect was found linked to pollen abortion, and the restoration of male fertility co-segregated with the ability to edit atp6 mRNA in *Sorghum* bicolour. These results suggest a link between the male sterile phenotype and the lack of editing. In this model, RNA editing ''corrects'' two highly conserved codons whose mutations are involved in human mitochondrial diseases [11]. Similarly, the gene B-atp6 is unprocessed and unedited in CMS rice plants carrying the [cms-bo] phenotype [12]. In rice, the ogr1 mutant is defective in seven specific RNA-editing sites in five mitochondrial mRNAs: nad2, nad4, cox2, cox3, and ccmc, which are associated with abnormalities in pollen grains. In the rice wild abortive (WA)-CMS system, sterility is correlated with the lack of editing of the orfB transcript [13]. Additional evidence that the lack of editing can lead to a male sterile phenotype comes from experimentally engineered plants. Expression of the unedited version of the wheat atp9 (u-atp9), but not the edited one, results in male sterility [14].

Molecular Diversity through RNA editing: RNA editing can directly or indirectly affect the expression or function of many genes. Alteration of amino acid codons, splice patterns, stability or localization of protein-coding transcripts, modulation of regulatory RNA biogenesis and function, as well as crosstalk of RNA editing with RNA processing and silencing pathways provides a rich resource for the generation of molecular diversity and for gene regulation. RNA editing by adenosine-to-inosine conversion (A-to-I editing) can introduce codon changes in mRNAs and hence generate structurally and functionally different isoforms of proteins. By changing codons at the level of mRNA, protein function can be altered, resulting in change in phenotype. RNA editing changes a codon, which in turn leads to a protein with amino acid substitution and altered functional properties . A-to-I editing may be particularly relevant for generating genetic variability as a basis for adaptive evolution. A-to-I RNA editing involves the specific modification of single adenosine nucleotides in RNA molecules to inosine *via* hydrolytic deamination. The resulting inosine is interpreted as guanosine by the translational machinery. A-to-I editing within the protein-coding region of an mRNA can therefore result in codon alterations that lead to an amino acid substitution in the protein product [15].

RNA editing as driving force for adaption: The RNA editing refers to the molecular processes in which the genetic information of DNA is altered through mRNA base insertion, deletion or replacement after gene transcription, thus resulting in changes of amino acid sequence and encoded protein and expands the genetic information, thus

making the environment more adaptable to the organisms. Genetic variability is considered a key to the evolvability of species. The conversion of an adenosine (A) to inosine (I) in primary RNA transcripts can result in an amino acid change in the encoded protein, a change in secondary structure of the RNA, creation or destruction of a splice consensus site, or otherwise alter RNA fate. RNA editing may be a principal contributor to the evolution of phenotypic complexity. With the widespread prevalence of A-to-I editing, the functional variation generated through editing and the fact that editing position and extents evolve through small inheritable changes in genomic sequences, A-to-I editing exhibits key properties to support a general evolutionary role (Fig. 3). It has been shown that organisms with higher complexity tend to be more robust due to increased functional redundancy. The evolution of RNA editing sites involves heritable genetic variation in the form of genetic changes in the genome, which is a critical prerequisite for this process to play a role in adaptation. However, any phenotypic variation is expressed only indirectly on the epigenetic level in form of RNA modification events that alter transcriptome and proteome composition [16].

RNA editing acts as a correcting mechanism for mutations: Mutation is an alteration in the genetic material of a cell that is transmitted to the cell's offspring. We assume that mutations in organellar genomes were established before the emergence of the correcting mechanism, that is, RNA editing. This was possible because these mutations increased the transmission of the organellar genome at the expense of the nuclear one. The appearance of the RNA-editing reaction was the response of the nuclear genome to restore its own transmission by correcting the organellar mutations, (Fig 4) [17]. A general consequence of the RNA-editing reaction is the restoration of highly conserved amino acid sequences or RNA secondary structures. Thus, it has been proposed that RNA editing evolved to correct organellar mutations.

Roles for RNA editing in leaf development: RNA editing alters transcripts to differ from the DNA sequence they were transcribed from, and thus breaks one of the central tenets of molecular biology - that protein sequences can be predicted from the genes that encode them. Over 600 cytidines in Arabidopsis organellar transcripts are specifically deaminated to uridine by this process. In the last year, 14 pentatricopeptide repeat (PPR) proteins have identified, that specify editing of target nucleotides in chloroplast or mitochondrial mRNAs. A failure to edit a specific organellar RNA can give rise to phenotypes that are unobtainable by any other means, given the intractability of Arabidopsis organelle genomes to the usual genetic tools. The extent of editing of some sites varies from 0% - 100% depending on tissue-type, developmental stage or growth conditions, suggesting RNA editing may be a novel means of controlling gene expression. As just one example, the PPR protein *FLAVODENTATA (FLV)* is required for editing of *rpoC1*, encoding a subunit of the plastid RNA polymerase. A failure to edit *rpoC1* leads to delayed chloroplast biogenesis in leaf margins and characteristic alterations in leaf morphology and symmetry. The target site for *FLV* shows variable editing in wild-type plants, and the extent of editing can be manipulated by altering *FLV* expression. The results imply previously unsuspected routes by which nuclear and chloroplast gene expression are coordinated.

RNA editing helps in the formation of functional enzymes: RNA editing is one of the most interesting and universal RNA-processing mechanisms known to affect gene regulation. This process has been detected in a variety of organisms, such as trypanosomes and mammals. [18]. In chloroplasts, RNA editing is a widespread processing event that creates start and stop codons and, most frequently, alters coding sequences.47–51) RNA editing in chloroplasts is mostly a cytosine-to-uracil change at the second nucleotide position of the triplet.[19]. In transcripts of the tobacco plastid genome, 0.13% of cytosine is changed to uracil. Comparing the accD gene sequence in the pea with its cDNA sequence, we found a cytosine-to-uracil change: the second nucleotide of UCG (serine) is converted to a uracil, and the resultant UUG triplet encodes a leucine. Multiple alignment of the amino acid sequences deduced from the accD gene of 15 land plants suggests the occurrence of similar changes in 6 plants. In such plants that do not have a leucine codon at the position, editing was shown to take place so as to create the leucine codon. The requirement of a leucine codon at a specific position suggests that accD editing is necessary for several plants. This proposition has been verified [20] by compared the CT activity of recombinant enzymes containing edited or unedited subunits and found that the edited enzyme, but not the unedited one, is active. On the basis of

the crystal structure of yeast CT,we predicted the structure of pea CT; this prediction suggests that the edited residue is present in the core region of the monomer. ACCase is an essential enzyme, indicating that the editing is essential. Probably, the cytosine-to-uracil change is caused by enzymatic deamination, but we do not know why deamination to a specific cytosine occurs. There are several cases in which both unedited and edited mRNAs produce different functional proteins, indicating the biological role of RNA editing.

CONCLUSION

In plants, post-transcriptional modification of transcripts includes C-to-U, U-to-C and A-to-I editing. RNA editing in plants is essential, with many mutants impaired in editing of specific sites exhibiting strong deleterious phenotypes, even lethality. The majority of editing in plants occurs in mitochondrial and plastid transcripts, however, A-to-I editing also occurs in cytosolic tRNAs. Editing usually restores amino acids that are phylogenetically more conserved with organisms that do not edit their RNA. RNA editing by cytidine (C) to uridine (U) conversions is widespread in plant mitochondria and chloroplasts. In some plant taxa, “reverse” U-to-C editing also occurs. However, to date, no instance of RNA editing has yet been reported in green algae and the complex thalloid liverworts. RNA editing may have evolved in early land plants 450 million years ago. However, in some plant species, including the liverwort, Marchantia polymorpha, editing may have been lost during evolution. Most RNA editing events can restore the evolutionarily conserved amino acid residues in mRNAs or create translation start and stop codons. Therefore, RNA editing is an essential process to maintain genetic information at the RNA level. Extensive editing takes place in the transcript of both nadh and atp9 genes of crop plants and these edit sites are conserved across the species. Individual RNA editing sites are recognized by plant-specific pentatricopeptide repeat (PPR) proteins that are encoded in the nuclear genome. These PPR proteins are characterized by repeat elements that bind specifically to RNA sequences upstream of target editing sites. PPR proteins are part of the eukaryotic machinery for regulating organelle gene expression in plants. PPR proteins are the specificity factors that bind the RNA and target the C to be edited and rapid expansion in number of PPR genes permitted RNA editing on a large scale.

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