as well as subunit 9 of ATP synthase (which is absent from humans) and subunit A of ATP synthase (which is in the nuclear genomes of all animals and fungi). In addition, the moss mitochondrial genome contains 16 genes for ribosomal proteins and 29 genes encoding proteins of unknown function.

Thus, although mitochondria in different eukaryotic organisms play similar roles in the conversion of food to energy, evolution has produced mtDNAs with an astonishing diversity in the content and organization of their genes. As we see next, mitochondrial evolution has also led to some remarkable variations on the basic mechanisms of gene expression.

Mitochondrial Transcripts Undergo RNA Editing, a Rare Variation on the Basic Theme of Gene Expression

Researchers discovered the unexpected phenomenon of RNA editing in the mitochondria of trypanosomes. As already noted, these protozoan parasites have a single, large mitochondrion—the kinetoplast—which contains much more DNA than the mitochondria of other organisms; this kDNA exists as a series of interlocking maxi- and minicircles. DNA sequencing shows that the minicircles carry no protein-encoding genes. The detection of transcripts from maxicircle DNA, however, confirms that these larger circles do carry and express genes.

Surprisingly, the sequencing of maxicircle DNA reveals only short, recognizable gene fragments, instead of whole mitochondrial genes. Furthermore, the sequencing of RNA molecules in the kinetoplast revealed both RNAs that looked like the strange fragments of kinetoplast genes and related RNAs that could encode recognizable mitochondrial proteins. From these observations, investigators concluded that kDNA encodes a precursor (the strange fragment observed) for each mRNA. After transcription, the cellular machinery turns these precursors into functional mRNAs through the insertion or deletion of nucleotides.

The process that converts pre-mRNAs to mature mRNAs is **RNA editing.** It is essential for the expression of these mitochondrial genes because without RNA editing, the pre-mRNAs do not encode polypeptides. Some pre-mRNAs lack a first codon suitable for translation initiation; others lack a stop codon for the termination of transcription. RNA editing creates both types of sites, as well as many new codons within the genes.

In addition to the mitochondria of trypanosomes, the mitochondria of some plants and fungi carry out RNA editing. The extent of RNA editing varies from mRNA to mRNA and from organism to organism. In trypanosomes, the RNA editing machinery adds or deletes uracils. In plants, the editing adds or deletes cytosines. At present, researchers understand the general mechanism of uracil editing, but not that of cytosine editing. As Fig. 15.6 shows, uracil editing occurs in stages in which enzymes use an RNA template as a guide for correcting the pre-mRNA. The guide RNAs are encoded by short stretches of kDNA on both maxi- and minicircles, and a structure known as an "editosome" is the workbench where the RNA editing takes place.

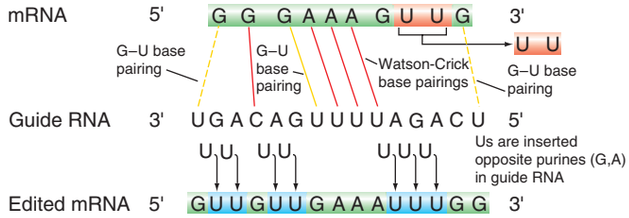

Figure 15.6 RNA editing in trypanosomes. Example of a portion of a pre-mRNA sequence is shown at the *top.* This pre-mRNA forms a double-stranded hybrid with a guide RNA through both standard Watson-Crick A–U and G–C base pairing, as well as atypical G–U base pairing. Unpaired G and A bases within the guide RNA initiate the insertion of U's within the pre-mRNA sequence, bringing about the final edited mRNA.

Translation in Mitochondria Shows That the Genetic Code Is Not Universal

As mtDNA carrying its own rRNA and tRNA genes would suggest, mitochondria have their own distinct translational apparatus. Mitochondrial translation is quite unlike the cytoplasmic translation of mRNAs transcribed from nuclear genes in eukaryotes. Many aspects of the mitochondrial translational system resemble details of translation in prokaryotes. For example, as in bacteria, *N*-formyl methionine and $tRNA^{fMet}$ initiate translation in mitochondria. Moreover, inhibitors of bacterial translation, such as chloramphenicol and erythromycin, which have no effect on eukaryotic cytoplasmic protein synthesis, are potent inhibitors of mitochondrial protein synthesis.

We saw in Chapter 8 that the genetic code is almost, but not quite, universal. The mtDNA sequences of tRNAs and protein-encoding genes in several species cannot explain the sequences of the resulting proteins in terms of the "universal" code. For example, in human mtDNA, the codon UGA specifies tryptophan rather than stop (as in the standard genetic code); AGG and AGA specify stop instead of arginine; and AUA specifies methionine rather than isoleucine (Table 15.3). No single mitochondrial genetic code functions in all organisms, and the mitochondria of higher plants use the universal code. Moreover, while an f-Met-tRNA usually initiates translation in mitochondria by reading AUG or AUA, other triplets, which do not specify methionine, often mark the site of initiation. The genetic codes of mitochondria probably diverged from the universal code by a series of mutations occurring some time after the organelles became established components of eukaryotic cells.

As we see next, chloroplast DNA, although similar in many ways to mtDNA, has some remarkable features of its own.

The Genomes of Chloroplasts

Chloroplasts occur in plants and algae. The genomes they carry are much more uniform in size than are the genomes of mitochondria. Although cpDNAs range in size from 120–217 kb, most are between 120 and 160 kb long (Table 15.4). Chloroplast DNA contains many more genes than mtDNA. Like the genes of bacteria and human mtDNA, these genes are closely packed,