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Генетические системы пластид и митохондрий. Механизмы наследования генов в митохондриях

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Mitochondria and Plastids Genetic Systems.
Mechanisms of Genes Inheritance in Mitochondria

Аннотация. Данная статья посвящена генетическим системам, которые принадлежат митохондриям и пластидам. В статье представлена информация о генетических системах в общем, основные принципы их работы и ныне открытые механизмы наследования. Основная цель статьи – собрать информацию в целом о генетике митохондрий и пластид, а также обозначить ее особенности.

Ключевые слова. Генетика, биотехнологии, генетические системы, митохондрии, пластиды.

Abstract. This article is devoted to genetic systems which belong to mitochondria and plastids. The article presents information on genetic systems in general, the main principles of their work and current inheritance mechanisms. The main purpose of the article is to collect information on genetic systems of mitochondria and plastids as a whole and to identify their features.

Key words. Genetics, biotechnologies, genetic systems, mitochondria, plastids.

Introduction

It is believed that mitochondria and plastids developed from bacteria that were absorbed by nucleated ancestral cells. As a relic of this past, types of organelles contain their own genomes, as well as their own biosynthetic mechanism for the production of RNA proteins and organelles. Mitochondria and plastids are never created from scratch, but instead arise from the growth and division of an existing mitochondria or plastid. On average, each organelle should double in weight in each generation of cells and then be distributed to each daughter cell. Even non-dividing cells must replenish organelles that degrade as part of the continuous process of organelles turnover, or produce additional organelles as needed. The process of growth and proliferation of organelles is complex because mitochondrial and plastid

proteins are encoded in two places: the nuclear genome and individual genomes contained in the organelles themselves.

Mitochondria and chloroplasts contain complete genetic systems

The biosynthesis of mitochondria and plastids requires the participation of two separate genetic systems. Most proteins in mitochondria and chloroplasts are encoded by special genes isolated for this purpose in nuclear DNA. These proteins are imported into organelles from the cytosol after they have been synthesized on cytosolic ribosomes. Other organelle proteins are encoded by organelle DNA and synthesized on the ribosomes inside the organelle using the mRNA produced by the organelles to determine their amino acid sequence. The movement of the protein between the cytosol and these organelles seems to be unidirectional, since none of the known proteins is exported from mitochondria or chloroplasts to the cytosol. An exception occurs under special conditions when the cell is about to undergo apoptosis. The release of intermembrane space protein (including cytochrome c) from mitochondria through the outer mitochondrial membrane is part of the signaling pathway that is triggered in cells undergoing programmed cell death.

The processes of transcription of DNA organelles, protein synthesis and DNA replication occur where the genome is located: in the mitochondrial matrix and in the stroma of chloroplasts. Although the proteins that mediate these genetic processes are unique to organelles, most of them are encoded in the nuclear genome. This is all the more surprising because the mechanism of protein synthesis of organelles resembles that of bacteria, not eukaryotes. The similarity is especially noticeable in chloroplasts. For example, chloroplast ribosomes are very similar to E. coli ribosomes in their structure and sensitivity to various antibiotics (such as chloramphenicol, streptomycin, erythromycin and tetracycline). In addition, protein synthesis in chloroplasts begins with N-formylmethionine, as in bacteria, and not with methionine, used for this purpose in the cytosol of eukaryotic cells. Although mitochondrial genetic systems are much less similar to those of modern bacteria than the chloroplast genetic systems, their ribosomes are also sensitive to antibacterial

antibiotics, and protein synthesis in mitochondria also begins with N-formyl methionine.

Mitochondrial genes are inherited by the non-Mendelian mechanism

Many experiments on the mechanism of mitochondrial biogenesis have been conducted with Saccharomyces cerevisiae (baker's yeast). There are several reasons for this preference. Firstly, when grown on glucose, this yeast has the ability to live only due to glycolysis and survive with defective mitochondria, which cannot cause oxidative phosphorylation. This allows you to grow cells with mutations in mitochondrial or nuclear DNA that disrupt mitochondrial function; such mutations are fatal to many other eukaryotes. Secondly, yeast is simple unicellular eukaryotes that are easy to grow and include biochemicals. Finally, these yeast cells usually reproduce asexually by budding. During sexual reproduction, two haploid cells mate and merge to form a diploid zygote, which can either grow mitotically or divide to form new haploid cells. The ability to control asexual and sexual reproduction in laboratories greatly facilitates genetic analysis. Mutations in mitochondrial genes are not inherited in accordance with the Mendelian rules that govern the inheritance of nuclear genes. Therefore, the genotype of mitochondrial genes can be sequenced, genetic studies have revealed which of the genes are involved in the functions of mitochondrial yeast, are in the nucleus, and which are in the mitochondria. When a chloramphenicol-resistant haploid cell mates with a wild-type chloramphenicol-sensitive haploid cell, the resulting diploid zygote outlines a mixture of the mutant and wild-type genomes. Mitochondrial networks of twito fuse in a zygote, creating one continuous network that contains the genomes of the corresponding parent cells. When a zygote is exposed to mitosis, copies of both wild-type mitochondrial and mutant DNA are secreted into the diploid daughter cell. In the case of nuclear DNA, each daughter cell receives exactly two copies of each chromosome. In contrast, in the case of mitochondrial DNA, the daughter cell can inherit or copy the DNA of mutant DNA. Successful mitotic divisions can further enrich any DNA, so that as a result there are many cells that contain mitochondrial DNA of only one genotype. This random process is called mitotic segregation. When diploid cells, which thus

distribute their mitochondrial genomes, turn into mesosis with the formation of four haploid daughter cells, each of the four daughters receives the same mitochondrial genes. This type of inheritance is called non-Mendelian or cytoplasmic inheritance in order to contrast it with the Mendelian inheritance of nuclear genes. This shows that this gene is located outside the nuclear chromosomes. Although clusters of mitochondrial DNA molecules (nucleoids) are relatively immobile in the mitochondrial network due to their attachment to the inner membrane, individual nucleoids sometimes come together. This often occurs during the formation of the zygote. Nucleic acids can cause genetic recombination. This recombination can lead to mitochondrial genomes that contain DNA from parent cells that are stably inherited after their mitotic segregation.

The genome of higher plant chloroplasts contains about 120 genes

Currently, more than 20 chloroplast genomes have been sequenced. The genomes of even remotely related plants (such as tobacco and liverwort) are almost identical, and even the genomes of green algae are closely related. Chloroplast genes are involved in four main types of processes: transcription, translation, photosynthesis and biosynthesis of small molecules such as amino acids, fatty acids and pigments. Genes of plant chloroplasts also encode at least 40 proteins whose functions are still unknown; in addition, about twice as many genes of unknown function are present in the chloroplasts of some algae. Paradoxically, all known proteins encoded in the chloroplast are part of larger protein complexes that also contain one or more subunits encoded in the nucleus. We will discuss the possible causes of this paradox later. The similarity between the genomes of chloroplasts and bacteria is striking. Basic regulatory sequences, such as transcription promoters and terminators, are almost identical in both cases. Amino acid sequences of proteins encoded in chloroplasts are clearly recognized as bacterial, and several clusters of genes with related functions (e.g., encoding ribosomal proteins) are equally organized in the genomes of chloroplasts, Escherichia coli and cyanobacteria. Further comparisons of a large number of homologous nucleotide sequences should help

clarify the exact evolutionary path from bacteria to chloroplasts, but several conclusions can already be made:

1. Chloroplasts in higher plants arose from photosynthetic bacteria.

2. The genome of chloroplasts is stably maintained for at least several hundred million years, the estimated time of divergence of the liver and tobacco.

3. Many of the genes of the original bacterium are currently present in the nuclear genome, where they are integrated and stably maintained. For example, in higher plants, two-thirds of the 60 or so chloroplast ribosomal proteins are encoded in the cell nucleus; these genes have a clear bacterial pedigree, and chloroplast ribosomes retain their original bacterial properties.

Symbiogenesis of mitochondria and plastids

The endosymbiotic theory, which holds that eukaryotic mitochondria and plastids arose from the engulfment and integration of a bacterium by another cell, has long been a matter of controversial debate, but growing evidence over time has led to the substantiation and universal acceptance of the theory. Recent genetic and biochemical analyses have provided detailed insights into the fundamental events that happened more than a billion years ago.

In 1905, Konstantin Mereschkowsky, a prominent Russian biologist, published his piece of work in “The nature and origins of chromatophores in the plant kingdom” where he proposed that the photosynthetic organelles of plants (plastids, chromatophores) originate from unicellular algae that live in a symbiotic relationship with their host cells.

Plastids are symbionts rather than cell organelles

Mereschkowsky was a leading lichenologist and his theory was inspired by his work on lichens where he had shown that lichens are symbiotic organisms composed of fungi and algae. Four years later in 1909, Mereschkowsky presented his theory of symbiogenesis according to which higher, i.e. more complex cells evolve from the symbiotic relationship between less complex ones. He came up with some astonishing conclusions: "Chlorophyll bodies (i.e. plastids) can grow and divide independently of the nucleus, and produce substances synthetically; in short they do

not behave like organs at all, but like independent organisms and must therefore either be regarded as such or as symbionts."

Mereschkowsky based his theory on the symbiogenesis of the plastids of green plant cells (chloroplasts) on the work of the German botanist Andreas Schimper (1856-1901) who had previously observed that chlorophyll bodies in plant cells do not develop de novo, but reproduce by dividing (in the same way as yeast cells) and are distributed to the daughter cells when the plant cell divides. The power of observation, patience and perspicacity of the 19th century cell researchers is without a doubt highly admirable, and led them to discoveries that were only confirmed much later before being considered self-evident by many people. The 19th century researchers used rather primitive devices with a resolution that was much less effective than a light microscope. This makes their findings even more admirable. Mereschkowsky's and Schimper's research and interpretation was initially dismissed and ignored and over the years fell virtually into oblivion.

Symbiogenesis models

There are many modern organisms that can be used as model systems for the symbiogenesis of mitochondria and plastids. The primitive fungus Geosiphon occasionally forms structures (bladders) that enable it to phagocytose certain cyanobacteria (Nostoc punctiforme) that then take over the function of chloroplasts and cover the fungus' energy requirements for fixing nitrogen from the atmosphere. Many organisms, including unicellular Paramecium bursaria, corals and giant saltwater clams (Tridacna), take up chloroplast-containing algae that then carry out photosynthesis for the host organism. However, the two organisms do not live in obligatory symbiosis, they can also subsist on their own. This is in contrast to mitochondria and plastids, which have long ago lost their autonomy and underlie the control of the cell nucleus of their host cell.

The study of such symbiogenesis models has also contributed to clarifying the somewhat confusing relationships between the different algal groups: the plastids of red and green algae, including ones in higher plants that descended from red and green algae, are the result of a primary endosymbiotic event (such as described

above) between a cyanobacterium and another free-living cell. All other algae have plastids that are surrounded by more than two, i.e. three or even four membranes, and some of them even contain nuclear material. These plastids have arisen through secondary (or tertiary) endosymbiosis, in which a eukaryote already possessing plastids (green or red algae) is engulfed by a second eukaryote.

Dinoflagellates have evolved by way of tertiary endosymbiosis involving the engulfment of a secondary endosymbiont, a situation reminiscent of Russian Matrjoschka dolls: a flagellate cell engulfed a haptophytic alga which itself arose from a red alga being engulfed by a unicellular organism while the red alga arose from a (presumably eukaryotic) host cell that had taken up cyanobacteria and turned them into its plastids.

It appears that the symbiogenesis of mitochondria occurred before the primary endosymbiosis of cyanobacteria. Mitochondria are genetically more strongly integrated into the host cell than plastids; and all plastid-containing cells also have mitochondria. So the question arises as to when did the steps that were crucial to the evolution of higher life occur? Molecular clocks, which are based on the mutation rates of gene sequences, are not reliable enough for estimating the timescale of events that happened such a long time ago and for which no reference systems are available; estimates range between 850 million and 2 billion years ago. Over the last few years, a growing number of microfossils has been found in Precambrian rock. These fossils are far from easy to interpret. State-of-the-art knowledge suggests that cyanobacteria existed as many as 2.7 billion years ago. Multicellular fossils (e.g. Bangiomorpha pubescens) have been found in rock dating back 1.2 billion years, and these fossils closely resemble the modern red algae Bangia. To date, this is the oldest fossil eukaryote known. It can therefore be safely assumed that the symbiogenetic events that gave rise to mitochondria and plastids, which are key steps in the evolution of higher life, took place before then.

Mitochondrial disease

Mitochondrial disease, also called mitochondrial disorder, is any of several hundred hereditary conditions resulting from functional deficiency of mitochondria,

such as a cell organelle. Mitochondrial diseases can occur at any age and are extremely diverse in their clinical and molecular features. Their severity ranges from a relatively mild disease that affects only one organ to a debilitating, and sometimes fatal, disease that affects several organs. A wide range of symptoms poses serious challenges in the diagnosis of conditions associated with mitochondrial dysfunction. At least 1 out of every 5,000 people in the world is affected by mitochondrial disease.

The mitochondrial respiratory chain consists of five multisubunit protein complexes that produce most of the energy that stimulates cellular ...

Although some mitochondrial diseases are caused by mutations in the mitochondrial genome (mtDNA), most conditions are the result of mutations in the genes in the nuclear genome, which encodes a series of proteins that are exported and transported to the mitochondria in the cell. Proteins gather in the mitochondria to form the electron transport chain (ETC), the primary energy apparatus of cells. The transfer of electrons from one protein component to another ultimately allows cells to produce energy in the form of adenosine triphosphate (ATP), which is the main form of energy used by cells and organs in the body. A deficiency in any of the proteins that make up ETC can impair ATP production and lead to the accumulation of unused intermediates (the original sugar and fat molecules that make up ETC) and reactive oxygen species (ROS; free radicals containing oxygen). Unused intermediates can react with other molecules, which leads to the formation of harmful by-products, such as lactic acid, while ROS can react with various cellular molecules, causing oxidative stress and cell death.

Signs and symptoms of mitochondrial disease vary depending on the organ or organ systems involved in the process. Possible symptoms include stunted growth, decreased growth, fatigue, migraine, muscle weakness, muscle pain, cardiomyopathy, liver failure, blindness, optic atrophy (optic nerve degeneration), hearing loss, diabetes, and seizures. Often there are signs and symptoms indicating a discrete syndrome. For example, in people aged three months to two years, the inability to develop, progressive neurological degeneration (with reduced muscle tone, inconsistent movements and involuntary and repeated muscle contraction), as well as

vision, breathing and heart problems are characteristic of Lee syndrome, Kearns syndrome Saire, on the other hand, is characterized primarily by progressive weakness or paralysis of the eye muscles and retinopathy (damage to the photosensitive retina), which can lead to omitted Ekam and vision loss; onset usually before the age of 20 years.

The diagnosis of mitochondrial disease is based on clinical features and, if possible, the results of genetic testing. The medical history in the maternal family can provide important diagnostic information, since hereditary mitochondrial diseases are transmitted from the mother to her offspring and are transmitted strictly through the maternal family. Persons affected by mitochondrial diseases can receive genetic counseling to assess the risk of transmission of a hereditary disease.

The treatment of mitochondrial diseases is supportive. Optical devices, including lens replacement, and hearing aids, such as cochlear implants, can help people with visual or hearing impairments. Implanted pacemakers or defibrillators may be helpful for some patients. Supportive care for deficiencies in certain ETC components may include oral administration of substances such as coenzyme Q10, L-creatine (creatine monohydrate), or riboflavin. Exercise can also help relieve symptoms in some people.

Conclusion

Why do mitochondria and chloroplasts have their own genetic systems?

Why do mitochondria and chloroplasts need their own genetic systems, while other organelles that have the same cytoplasm, such as peroxisomes and lysosomes, do not need? The question is not trivial because maintaining a separate genetic system is expensive: more than 90 proteins, including many ribosomal proteins, aminoacyltRNA synthases, DNA and RNA polymerases, as well as RNA processing and RNA modifying enzymes, must be encoded by nuclear genes specifically for this goal. The amino acid sequences of most of these proteins in mitochondria and chloroplasts differ from the amino acid sequences in the nucleus and cytosol, and these organelles appear to have relatively few common proteins with the rest of the cell. This means that the nucleus must provide at least 90 genes only to maintain the

genetic system of each organelle. The reason for such an expensive device is not clear, and the hope that the nucleotide sequences of the mitochondria and chloroplasts genomes will give an answer turned out to be unfounded. We cannot think of the good reasons why proteins produced in mitochondria and chloroplasts should be made there and not in the cytosol. At one time, it was suggested that some proteins should be made in the organelle, because they are too hydrophobic to get into their site in the membrane from the cytosol. More recent studies, however, make this explanation implausible. In many cases, even highly hydrophobic subunits are synthesized in the cytosol. Moreover, although individual protein subunits in various mitochondrial enzyme complexes are highly conserved in evolution, their synthesis site is not. The diversity in the arrangement of genes encoding subunits of functionally equivalent proteins in different organisms is difficult to explain by any hypothesis that postulates a specific evolutionary advantage of modern mitochondrial or chloroplast genetic systems. Perhaps the organelle's genetic systems are an evolutionary dead end. From the standpoint of the endosymbiont hypothesis, this will mean that the process by which endosymbionts transfer most of their genes to the nucleus stops before it ends. Further transfers can be ruled out for mitochondria by recent changes in the mitochondrial genetic code that made the remaining mitochondrial genes non-functional if they were transferred to the nucleus.

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