**COVID Vaccines and Your DNA – Fact Check**

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Introduction

You may have heard that the COVID vaccine doesn’t alter your DNA, and isn’t gene therapy, because it doesn’t enter the cell’s nucleus where your DNA is found. And although many people involved with the manufacturing and promotion of these vaccines have said they alter DNA, the statement about DNA only being found in the nucleus seemed to be a slam dunk, disproving the information those conspiracy theorists posted online (information they got from people involved with the manufacturing of the vaccines). This is what we’re going to take a look at.

**Facebook CEO Mark Zuckerberg Takes ‘Anti-Vax’ Stance in Violation of His Own Platform's New Policy**  
<https://www.bitchute.com/video/oXWREk7MVqQ/>

Alternative sources:  
<https://www.youtube.com/watch?v=oXWREk7MVqQ>  
<https://www.bitchute.com/video/32vYKWaWsZ8w/>  
<https://www.bitchute.com/video/mXEvAPKHjeQ8/>  
<https://www.bitchute.com/video/Bt6N21dSm3NV/>  
<https://www.bitchute.com/video/1js0kq1dEONO/>  
<https://www.bitchute.com/video/H7FMGQIBjebA/>

The Adenovirus COVID Vaccine

First, there is actually no “COVID vaccine,” as in one type of COVID vaccine. There are actually several different types, and I don’t mean brands. According to the CDC there are three types of COVID vaccines: mRNA, protein subunit, and vector (Source: [*Understanding How COVID-19 Vaccines Work*](https://www.cdc.gov/coronavirus/2019-ncov/vaccines/different-vaccines/how-they-work.html))

“A coronavirus vaccine known as ChAdOx1 nCoV-19 or AZD1222 was developed by the University of Oxford and AstraZeneca to treat severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection (the cause of COVID-19). In this vaccine, a modified version of a chimpanzee adenovirus (ChAdOx1) is used which can enter human cells but not replicate inside. A gene for the coronavirus vaccine was added into the adenovirus DNA, allowing the vaccine to target the spike proteins that SARS-CoV-2 uses to enter human cells. The vaccine was given emergency authorization by the UK in December 2020 of the pandemic, and India authorized a variation of it the same month. As of February 2021, versions of the vaccine produced in India and South Korea gained approval for emergency use by the World Health Organization (WHO), which should improve accessibility and ease of approval for other nations. Other adenovirus-based SARS-CoV-2 vaccines in development include the Sputnik V vaccine and the Johnson & Johnson vaccine.”  
Source: *What are Adenovirus-Based Vaccines?*  
<https://www.news-medical.net/health/What-are-Adenovirus-Based-Vaccines.aspx>

“But here with Sputnik, with the AstraZeneca and the J&J, they're using slightly different adenoviruses. So, there are a number of adenoviruses. You can take an adenovirus from a chimpanzee, and that's what they're using at AstraZeneca. They took a chimp adenovirus, the original ChAdOx. What the Russians are doing and I think this is clever, is they're using two different adenoviruses. One for the delivery of the initial and then a different one for the boost. And then J&J is actually just using one adenovirus, because you know, they're hoping for a one-shot, one and done vaccine.”  
Source: *What Do We Really Know About Adenovirus Vectors for Vaccines?*  
<https://www.medpagetoday.com/podcasts/trackthevax/91323>

“The Oxford-AstraZeneca vaccine is based on the virus’s genetic instructions for building the spike protein. But unlike the Pfizer-BioNTech and Moderna vaccines, which store the instructions in single-stranded RNA, the Oxford vaccine uses double-stranded DNA. The researchers added the gene for the coronavirus spike protein to another virus called an adenovirus. Adenoviruses are common viruses that typically cause colds or flu-like symptoms. The Oxford-AstraZeneca team used a modified version of a chimpanzee adenovirus, known as ChAdOx1. It can enter cells, but it can’t replicate inside them. … After the vaccine is injected into a person’s arm, the adenoviruses bump into cells and latch onto proteins on their surface. The cell engulfs the virus in a bubble and pulls it inside. Once inside, the adenovirus escapes from the bubble and travels to the nucleus, the chamber where the cell’s DNA is stored. The adenovirus pushes its DNA into the nucleus. The adenovirus is engineered so it can’t make copies of itself, but the gene for the coronavirus spike protein can be read by the cell and copied into a molecule called messenger RNA, or mRNA.”  
Source: *How the Oxford-AstraZeneca Vaccine Works*  
(Updated March 22, 2021)  
<https://www.nytimes.com/interactive/2020/health/oxford-astrazeneca-covid-19-vaccine.html>

\*An archived copy can be found here: <http://archive.today/scxBD>

You should also take a look at the images in that article.

“The Johnson & Johnson vaccine is based on the virus’s genetic instructions for building the spike protein. But unlike the Pfizer-BioNTech and Moderna vaccines, which store the instructions in single-stranded RNA, the Johnson & Johnson vaccine uses double-stranded DNA. … The researchers added the gene for the coronavirus spike protein to another virus called Adenovirus 26. Adenoviruses are common viruses that typically cause colds or flu-like symptoms. The Johnson & Johnson team used a modified adenovirus that can enter cells but can’t replicate inside them or cause illness. … After the vaccine is injected into a person’s arm, the adenoviruses bump into cells and latch onto proteins on their surface. The cell engulfs the virus in a bubble and pulls it inside. Once inside, the adenovirus escapes from the bubble and travels to the nucleus, the chamber where the cell’s DNA is stored. The adenovirus pushes its DNA into the nucleus. The adenovirus is engineered so it can’t make copies of itself, but the gene for the coronavirus spike protein can be read by the cell and copied into a molecule called messenger RNA, or mRNA. The mRNA leaves the nucleus, and the cell’s molecules read its sequence and begin assembling spike proteins.”  
Source: *How the Johnson & Johnson Vaccine Works*  
<https://www.nytimes.com/interactive/2020/health/johnson-johnson-covid-19-vaccine.html>

\*An archived copy can be found here: <https://archive.vn/6o0n2>

You should also take a look at the images in that article.

Now take a look at #2 in the following JAMA article. The adenovirus enters cells and releases its viral DNA where?

**The Johnson & Johnson Vaccine for COVID-19**  
<https://jamanetwork.com/journals/jama/fullarticle/2777172>

Here’s the image:  
<https://cdn.jamanetwork.com/ama/content_public/journal/jama/0/jpg210009fa_1614377158.3683.png?Expires=1621094993&Signature=wcwmO94nyQzu4jcG9CMFO6mnjoqOFuEBZnMp61HXxrYzNKkoIaUz7vGfNYnTn4KQSnev-su25P5AYJUOCUXXeT~0jj7kQFfrFPf3gcY8zr~1CeH4qRc4Mildjl0EZkgp~EzGrY-NcpvCUYoxD8FyPLYbZwt9igVnU9httpeZ62k0ABMTlaxgOr~8IVLQ2DUoGCKIZXxPnJy-bZr3ML6WUqcJXdaSheeNsC7pyJ4QPkcnAF1RVLM-SIk99svqoq77VO6~yryAQMDe6ILo3b4NkDws3gezl9IueU1olODeQPwHyFJFS7bP-bjTcr8cltGREB0B4knqcxuTlXvI4Qz8Zg__&Key-Pair-Id=APKAIE5G5CRDK6RD3PGA>

**Coronavirus Vaccines - An Introduction**  
<https://www.youtube.com/watch?v=KMc3vL_MIeo>

So, two of the main vaccines that we keep hearing about work by placing genetic material into the nucleus of the cell. So, you have been lied to. What a shocker!

**What Are The Adenovirus Vector & mRNA Shots? Gene Therapies? Vaccines?**  
<https://www.bitchute.com/video/icK7RJLUUTc0/>

**Gene Therapy**  
<https://en.wikipedia.org/wiki/Gene_therapy#/media/File:Gene_therapy.jpg>

Now let’s see GAVI explain how these vaccines work.

**There are four types of COVID-19 vaccines: here’s how they work**  
<https://www.youtube.com/watch?v=lFjIVIIcCvc>

Take a closer look at the portion of the video where they talk about the adenovirus vaccines (2:41 - 3:15).

- 2:54 – “… to get the code into the **cells**…” Where is the code going? Only into the cells.

- 3:06 - 3:15 – Notice how they show “the code” leaving the virus and entering the cell, and then they cut it right there. This, along with the statement about “the code” entering the cells, shows that they want the viewer to only believe that it enters the cell and that’s it. But what about the part where it enters the nucleus? I thought they were showing us how it works. And for the record, I’ve seen several other videos that do the same, and have read several articles that leave out the same.

And if you want, you can take a look at these and see how the CDC conveniently leaves out the nucleus as well:

**Understanding How COVID-19 Vaccines Work**  
<https://www.cdc.gov/coronavirus/2019-ncov/vaccines/different-vaccines/how-they-work.html>

**Understanding Viral Vector COVID-19 Vaccines**  
<https://www.cdc.gov/coronavirus/2019-ncov/vaccines/different-vaccines/viralvector.html>

The mRNA COVID Vaccine

The adenovirus COVID vaccines are the only vaccines that go into the nucleus; the mRNA vaccines do not and therefore cannot alter your DNA, and are not gene therapy? Before we get to this, let’s take a quick look at the “machinery” they are claiming the mRNA COVID vaccines take over – the machinery you are trusting them to control.

“Suppose that you have a very precious piece of information. Let’s imagine that this piece of information is a blueprint. In fact, it’s not just a blueprint for a house, or a car, or even a top-secret fighter jet. It’s a blueprint for an entire organism – you – and it not only specifies how to put you together, but also provides the information that enables every cell in your body to keep functioning from moment to moment. Sounds important, right? You’d probably want to keep information this valuable in a secure spot, perhaps in a protected vault where you can keep an eye on it. In fact, that’s exactly what eukaryotic cells do with their genetic material, placing it in a membrane-enclosed repository called the nucleus. Eukaryotic DNA never leaves the nucleus; instead, it’s transcribed (copied) into RNA molecules, which may then travel out of the nucleus.”  
Source: *Nucleus and ribosomes*  
<https://www.khanacademy.org/science/biology/structure-of-a-cell/prokaryotic-and-eukaryotic-cells/a/nucleus-and-ribosomes>

“The complete set of your DNA is called your genome. It [contains](https://www.genome.gov/about-genomics/fact-sheets/Deoxyribonucleic-Acid-Fact-Sheet) 3 billion bases, 20,000 genes, and 23 pairs of chromosomes! You inherit half of your DNA from your father and half from your mother. This DNA comes from the [sperm](https://www.healthline.com/health/how-is-sperm-produced) and egg, respectively. Genes actually make up very little of your genome — only [1 percent](https://www.genome.gov/about-genomics/fact-sheets/Deoxyribonucleic-Acid-Fact-Sheet). The other 99 percent helps to regulate things like when, how, and in what quantity proteins are produced. Scientists are still learning more and more about this ‘non-coding’ DNA. … DNA contains the instructions that are necessary for an organism — you, a bird, or a plant for example — to grow, develop, and reproduce. These instructions are stored within the sequence of nucleotide base pairs. Your cells read this code three bases at a time in order to generate proteins that are essential for growth and survival. The DNA sequence that houses the information to make a protein is called a gene. … So far, we’ve learned that DNA contains a code that gives the cell information on how to make proteins. But what happens in between? Simply put, this occurs via a two-step process: First, the two DNA strands split apart. Then, special proteins within the nucleus read the base pairs on a DNA strand to create an intermediate messenger molecule. This process is called transcription and the molecule created is called messenger RNA (mRNA). mRNA is another type of nucleic acid and it does exactly what its name implies. It travels outside of the nucleus, serving as a message to the cellular machinery that builds proteins. In the second step, specialized components of the cell read the mRNA’s message three base pairs at a time and work to assemble a protein, amino acid by amino acid. This process is called translation.”   
Source: *DNA Explained and Explored*  
<https://www.healthline.com/health/what-is-dna>

“Proteins are probably the most important class of material in the body. Proteins are not just building blocks for muscles, connective tissues, skin, and other structures. They also are needed to make enzymes. Enzymes are complex proteins that control and carry out nearly all chemical processes and reactions within the body. The body produces thousands of different enzymes. Thus, the entire structure and function of the body is governed by the types and amounts of proteins the body synthesizes. Protein synthesis is controlled by genes, which are contained on chromosomes.”  
Source: *Genes and Chromosomes*  
<https://www.merckmanuals.com/home/fundamentals/genetics/genes-and-chromosomes>

Even if their claims of what it does inside your body are true, you would have to be insane to trust drug companies with this type of power over your body. And you don’t even know what they’re not telling you, or the consequences they don’t even know about themselves.

Mitochondria – The Other Source of Cellular DNA

Now, mRNA COVID vaccines and your DNA – there’s no way it can alter your DNA because it doesn’t enter the nucleus of a cell? And they are not gene therapy?

“Although the vast majority of [DNA](https://www.ncbi.nlm.nih.gov/books/n/mcb/A7315/def-item/A7455/) in most [eukaryotes](https://www.ncbi.nlm.nih.gov/books/n/mcb/A7315/def-item/A7488/) is found in the [nucleus](https://www.ncbi.nlm.nih.gov/books/n/mcb/A7315/def-item/A7692/), some DNA is present within the [mitochondria](https://www.ncbi.nlm.nih.gov/books/n/mcb/A7315/def-item/A7650/) of animals, plants, and fungi and within the chloroplasts of plants.”  
Source: *Molecular Cell Biology. 4th edition.*  
<https://www.ncbi.nlm.nih.gov/books/NBK21574/>

“DNA, or deoxyribonucleic acid, is the hereditary material in humans and almost all other organisms. Nearly every cell in a person’s body has the same DNA. Most DNA is located in the cell nucleus (where it is called nuclear DNA), but a small amount of DNA can also be found in the mitochondria (where it is called [mitochondrial DNA](https://medlineplus.gov/genetics/chromosome/mitochondrial-dna/) or mtDNA).”  
Source: *What is DNA?*  
<https://medlineplus.gov/genetics/understanding/basics/dna/>

“Comprised of one small chromosome and less than 17 thousand base pairs, mitochondrial DNA is [very short](http://www.majordifferences.com/2015/05/difference-between-mitochondrial-dna.html#.WDW2JvnyvIU) in comparison with nuclear DNA, which includes three billion bases on 23 chromosome pairs. Nevertheless, every cell contains a single nucleus – but numerous mitochondria: in some cells, there are thousands of these small structures, and thus such cells contain thousands of copies of mitochondrial DNA.”  
Source: *DNA Hanging by a Hair - What type of DNA is found in hair? What is mitochondrial DNA? Can it be used to identify criminals? Q&A*  
<https://davidson.weizmann.ac.il/en/online/askexpert/dna-hanging-hair>

“Each cell contains hundreds to thousands of mitochondria, which are located in the fluid that surrounds the nucleus (the cytoplasm). Although most DNA is packaged in chromosomes within the nucleus, mitochondria also have a small amount of their own DNA. This genetic material is known as mitochondrial DNA or mtDNA. In humans, mitochondrial DNA spans about 16,500 DNA building blocks (base pairs), representing a small fraction of the total DNA in cells. Mitochondrial DNA contains 37 genes, all of which are essential for normal mitochondrial function. Thirteen of these genes provide instructions for making enzymes involved in oxidative phosphorylation. Oxidative phosphorylation is a process that uses oxygen and simple sugars to create adenosine triphosphate (ATP), the cell's main energy source. The remaining genes provide instructions for making molecules called transfer RNA (tRNA) and ribosomal RNA (rRNA), which are chemical cousins of DNA. These types of RNA help assemble protein building blocks (amino acids) into functioning proteins.”  
Source: *Mitochondrial DNA*  
<https://medlineplus.gov/genetics/chromosome/mitochondrial-dna/>

\*The transfer RNA and ribosomal RNA they mentioned are mitochondrial.

And as you’ll see later, yes, mRNA can enter the mitochondria, and has previously been designed by scientists to do so.

“The genes encoding mt tRNAs are highly susceptible to point mutations, which are a primary cause of mitochondrial dysfunction and are associated with a wide range of pathologies.”  
Source: *Human mitochondrial tRNAs: biogenesis, function, structural aspects, and diseases*  
<https://pubmed.ncbi.nlm.nih.gov/21910628/>

**Extrachromosomal DNA**

**Extrachromosomal DNA** (abbreviated ecDNA) is any [DNA](https://en.wikipedia.org/wiki/DNA) that is found off the [chromosomes](https://en.wikipedia.org/wiki/Chromosome), either inside or outside the [nucleus](https://en.wikipedia.org/wiki/Cell_nucleus) of a [cell](https://en.wikipedia.org/wiki/Cell_(biology)). Most DNA in an individual [genome](https://en.wikipedia.org/wiki/Genome) is found in [chromosomes](https://en.wikipedia.org/wiki/Chromosomes) contained in the nucleus. Multiple forms of extrachromosomal DNA exist and serve important biological functions,[[1]](https://en.wikipedia.org/wiki/Extrachromosomal_DNA#cite_note-Rush-1) e.g. they can play a role in disease, such as ecDNA in cancer.[[2]](https://en.wikipedia.org/wiki/Extrachromosomal_DNA#cite_note-:1-2)

In [prokaryotes](https://en.wikipedia.org/wiki/Prokaryote), nonviral extrachromosomal DNA are primarily found in [plasmids](https://en.wikipedia.org/wiki/Plasmids) whereas in [eukaryotes](https://en.wikipedia.org/wiki/Eukaryote) extrachromosomal DNA are primarily found in [organelles](https://en.wikipedia.org/wiki/Organelle).[[1]](https://en.wikipedia.org/wiki/Extrachromosomal_DNA#cite_note-Rush-1) [Mitochondrial DNA](https://en.wikipedia.org/wiki/Mitochondrial_DNA) are a main source of this extrachromosomal DNA in eukaryotes.[[3]](https://en.wikipedia.org/wiki/Extrachromosomal_DNA#cite_note-Kuttler-3) The fact that this organelle contains its own DNA supports the hypothesis that mitochondria originated as bacterial cells engulfed by ancestral eukaryotic cells.[[4]](https://en.wikipedia.org/wiki/Extrachromosomal_DNA#cite_note-4) Extrachromosomal DNA are often used in research of [replication](https://en.wikipedia.org/wiki/DNA_replication) because they are easy to identify and isolate.[[1]](https://en.wikipedia.org/wiki/Extrachromosomal_DNA#cite_note-Rush-1)

Although [extrachromosomal circular DNA](https://en.wikipedia.org/wiki/Extrachromosomal_circular_DNA) (eccDNA) are found in normal eukaryotic cells, extrachromosomal DNA ([ecDNA](https://en.wikipedia.org/wiki/EcDNA)) are a distinct entity that have been identified in the nuclei of cancer cells and have been shown to carry many copies of driver oncogenes.[[5]](https://en.wikipedia.org/wiki/Extrachromosomal_DNA#cite_note-Targeted_therapy_resistance_mediate-5)[[6]](https://en.wikipedia.org/wiki/Extrachromosomal_DNA#cite_note-:2-6)[[7]](https://en.wikipedia.org/wiki/Extrachromosomal_DNA#cite_note-:0-7) ecDNA are considered to be a primary mechanism of gene amplification, resulting in many copies of driver oncogenes and very aggressive cancers.

… [Inheritance](https://en.wikipedia.org/wiki/Inheritance) of extrachromosomal DNA differs from the inheritance of nuclear DNA found in chromosomes. Unlike chromosomes, [ecDNA](https://en.wikipedia.org/wiki/EcDNA) does not contain [centromeres](https://en.wikipedia.org/wiki/Centromere) and therefore exhibits a non-Mendelian inheritance pattern that gives rise to heterogeneous cell populations. In humans, virtually all of the cytoplasm is inherited from the egg of the mother.[[40]](https://en.wikipedia.org/wiki/Extrachromosomal_DNA#cite_note-Griffiths_NCBI-40) For this reason, organelle DNA, including mtDNA, is inherited from the mother. [Mutations](https://en.wikipedia.org/wiki/Mutations) in mtDNA or other cytoplasmic DNA will also be inherited from the mother. This [uniparental inheritance](https://en.wikipedia.org/wiki/Uniparental_inheritance) is an example of [non-Mendelian inheritance](https://en.wikipedia.org/wiki/Non-Mendelian_inheritance). Plants also show uniparental mtDNA inheritance. Most plants inherit mtDNA maternally with one noted exception being the redwood *Sequoia sempervirens* that inherit mtDNA paternally.[[41]](https://en.wikipedia.org/wiki/Extrachromosomal_DNA#cite_note-Sato-41)

… Mitochondrial DNA can play a role in the onset of disease in a variety of ways. [Point mutations](https://en.wikipedia.org/wiki/Point_mutations) in or alternative gene arrangements of mtDNA have been linked to several diseases that affect the heart, [central nervous system](https://en.wikipedia.org/wiki/Central_nervous_system), endocrine system, gastrointestinal tract, eye, and kidney.[[18]](https://en.wikipedia.org/wiki/Extrachromosomal_DNA#cite_note-Chinnery-18) Loss of the amount of mtDNA present in the mitochondria can lead to a whole subset of diseases known as mitochondrial depletion syndromes (MDDs) which affect the liver, central and [peripheral nervous](https://en.wikipedia.org/wiki/Peripheral_nervous_system) systems, smooth muscle and hearing in humans.[[19]](https://en.wikipedia.org/wiki/Extrachromosomal_DNA#cite_note-Dimmock-19) There have been mixed, and sometimes conflicting, results in studies that attempt to link mtDNA copy number to the risk of developing certain cancers. Studies have been conducted that show an association between both increased and decreased mtDNA levels and the increased risk of developing [breast cancer](https://en.wikipedia.org/wiki/Breast_cancer). A positive association between increased mtDNA levels and an increased risk for developing kidney tumors has been observed but there does not appear to be a link between mtDNA levels and the development of [stomach cancer](https://en.wikipedia.org/wiki/Stomach_cancer).[[42]](https://en.wikipedia.org/wiki/Extrachromosomal_DNA#cite_note-Thyagarajan-42)  
Source: *Extrachromosomal DNA*  
<https://en.wikipedia.org/wiki/Extrachromosomal_DNA>

“Mitochondria have another important characteristic. In human and animal cells, they’re the only location of DNA outside the nucleus. An error in mitochondrial DNA or in the nuclear DNA that controls the mitochondria may have serious consequences in our bodies. The damage can stop the organelles from working properly and cause hundreds of different disorders. These disorders are collectively known as mitochondrial diseases. … Another unusual feature of mitochondria is that they can reproduce independently of the cell that contains them. A human cell may contain hundreds or even thousands of mitochondria. … The DNA molecules in the nucleus contain about 20,000 to 25,000 genes. About 1500 of these genes control the function of the mitochondria. According to one theory, the genes migrated to the nucleus from the organisms that became mitochondria. Mutations (changes) in mitochondrial DNA or in the nuclear genes that affect the mitochondia can result in a mitochondrial disease. A huge number of biological processes and chemical reactions are constantly occurring in our cells and in the tissues and organs that they form. Many of these processes require added energy, so malfunctioning mitochondria may sometimes have serious and widespread effects in our bodies. Some ATP molecules can be made outside the mitochondria, but not enough are produced in this location to keep us alive. A person's mitochondria are inherited from his or her mother. During fertilization, the sperm penetrates the egg. The genetic material of the egg and sperm join, but the rest of the sperm—including its mitochondria —disintegrates. The egg contains the mitochondria that the baby will inherit. Once the process of fertilization has finished, the egg is known as a zygote. This is the first cell of the new individual. If the egg's mitochondria contained mutated DNA, the zygote's will as well. The zygote contains genetic material from both the mother and the father. If the genes controlling the mitochondria are mutated in either of these sources, the zygote will inherit them. Unfortunately, although it's sometimes possible to predict the probability that a child will inherit mutated genes related to mitochondrial disease, it's not always possible to predict the effect of this inheritance. There are many variables involved. If the mitochondria in the zygote contain a mutation, the child that develops may experience no symptoms, mild symptoms, or serious ones. If only some of the mitochondria in a zygote contain a mutation, the child will contain a mixture of normal and mutated organelles. This may affect the symptoms that he or she experiences. The nature of the mutations and the parts of the body where mitochondria with specific mutations are located will also affect the symptoms. When a cell divides to make new cells during the production of a baby, a selection of mitochondria go to each daughter cell instead of a complete set. The distribution of the mitochondria appears to be random. This means that we can't predict where specific versions of the organelles will end up. … An inherited mitochondrial problem is present at birth and is known as a primary condition. If this problem produces symptoms, they may appear immediately after birth. They may not appear until later, however. They may even be delayed until adulthood, as they were for the patient in the video above. Mitochondrial dysfunction may develop during a person's lifetime without the presence of a relevant mutation. This condition is known as secondary mitochondrial disease. One cause of the condition is exposure to toxins that enter the body from the outside or are made inside the body. If the toxins damage the mitochondria, organelle dysfunction may develop. The condition may also arise after a serious health problem such as a heart attack or as a result of the aging process.”  
Source: *Mitochondria Facts and Disease: DNA Outside the Nucleus*  
<https://owlcation.com/stem/Mitochondrial-DNA-and-Disease-Genes-Outside-the-Nucleus>

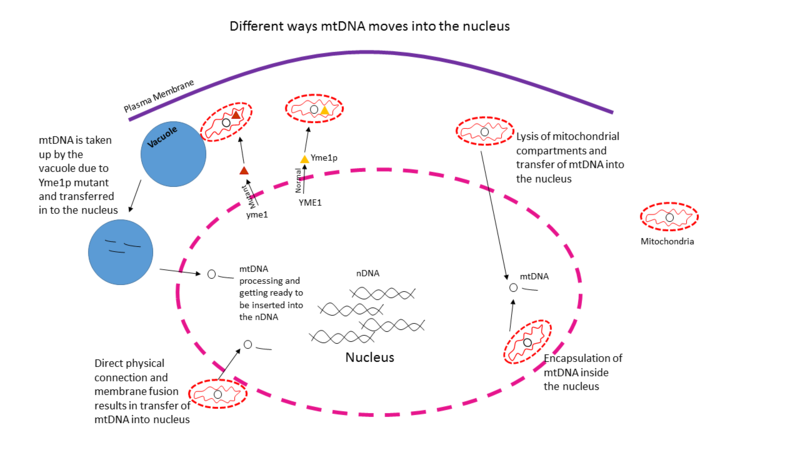
“Mitochondrial dysfunction may develop during a person's lifetime without the presence of a relevant mutation. This condition is known as secondary mitochondrial disease. One cause of the condition is exposure to toxins that enter the body from the outside or are made inside the body.” Toxins that enter the body from the outside? Oh, like what happens when you inject newborn babies with vaccines that contain toxic chemicals? And if an inherited mitochondrial problem is present at birth, similar to toxins given to a child after birth, but symptoms could take until adulthood to manifest, what do you think is going on with all vaccines whether they affect the mitochondria or immune system, causing autoimmune diseases? Somehow when it comes to vaccines the symptoms and conditions have to manifest immediately or else the vaccine is not the cause. Interesting.

“For plant and animal cells, the vast bulk of their DNA is tightly packaged and tucked away within a storage and processing facility inside their cells known as the nucleus. However, cells also carry a small number of genes in specialized compartments, called organelles, which lie outside of the nucleus. These are the mitochondria, which generate energy for plant and animal cells, and chloroplasts, which carry out photosynthesis in plant cells. A new study now finds that organellar genes can have a much larger effect on a cell's metabolism than would be expected based on their numbers. The finding, published online this month in the journal *eLife*, could have implications for future treatments of inherited diseases in humans, scientists said. ‘Whenever you see a study where the scientists say they've linked this gene with this disease, or that they've found the gene that controls this behavior, they almost always looked solely at genes within the nucleus,’ said the study's leader [Daniel Kliebenstein](http://bit.ly/1bXnP0c), a plant geneticist at the University of California, Davis. In their study, Kliebenstein and his team studied how variation in 25,000 nuclear and 200 organellar genes affected the concentrations of thousands of individual chemicals, or metabolites, in leaf tissues of *Arabidopsis*, a small flowering plant related to cabbage and mustard. The experiment involved more than 300 *Arabidopsis* plants, all of which had the same nuclear genetic diversity, but differing chloroplast and mitochondrial genes. When the scientists measured different metabolite concentrations in the plants — everything from sugars and amino acids, to fat molecules — they found that organellar genes appeared to influence the concentrations of about 80 percent of them, and that the effects could range from 25 to 200 percent. Edward Morrow, a biologist at the University of Sussex in the U.K., said that scientists have suspected for nearly 20 years that organellar genes can ‘punch above their weight,’ but the current research is the one of the most comprehensive studies of the phenomenon to date. The paper revealed volumes of amazing detail of the effects of organellar genes on nuclear genes, said Morrow, who was not involved in the study. ‘[It's] quite stunning, really.’”  
Source: *Genes Outside of Nucleus 'Punch Above Their Weight'*  
<https://www.insidescience.org/news/genes-outside-nucleus-punch-above-their-weight>

“Mitochondrial DNA (mtDNA) is a type of DNA located outside the nucleus in the liquid portion of the cell (cytoplasm) and inside cellular organelles called mitochondria. … Each mitochondrion in a cell can have multiple copies of the mtDNA [genome](https://embryo.asu.edu/search?text=genome). In [humans](https://embryo.asu.edu/search?text=humans), the mature [egg](https://embryo.asu.edu/search?text=egg) cell, or [oocyte](https://embryo.asu.edu/search?text=oocyte), contains the highest number of mitochondria among human cells, ranging from 100,000 to 600,000 mitochondria per cell, but each mitochondrion contains only one copy of mtDNA. In human embryonic development, the number of mitochondria, the content of mtDNA in each mitochondrion, and the subsequent mtDNA activity affects the production of the oocytes, [fertilization](https://embryo.asu.edu/search?text=fertilization) of the oocytes, and early embryonic growth and development. Mitochondria were once free-living bacteria that took up residence inside a primitive eukaryotic cell in the process called endosymbiosis. Much of the evidence for the claim is in the mtDNA [genome](https://embryo.asu.edu/search?text=genome) and the nuclear [genome](https://embryo.asu.edu/search?text=genome). The genomes co-evolved, and control of mitochondria involves exchange of information between the [nucleus](https://embryo.asu.edu/search?text=nucleus) and the many copies of the mtDNA. In the developing embryo, ninety-nine percent of the mitochondria, and therefore the mtDNA, come from the mother. Point mutations and deletions in the mtDNA can lead to serious developmental mitochondrial diseases.”   
Source: *Mitochondrial DNA (mtDNA)*  
<https://embryo.asu.edu/pages/mitochondrial-dna-mtdna>

“For a long time, biologists thought our DNA resided only in the control center of our cells, the nucleus. Then, in 1963, a couple at Stockholm University discovered DNA outside the nucleus. Looking through an electron microscope, Margit and Sylvan Nass noticed DNA fibers in structures called mitochondria, the energy centers of our cells. Our mitochondrial DNA accounts for a small portion of our total DNA. It contains just 37 of the 20,000 to 25,000 protein-coding genes in our body. But it is notably distinct from DNA in the nucleus. Unlike nuclear DNA, which comes from both parents, mitochondrial DNA comes only from the mother. Nobody fully understands why or how fathers’ mitochondrial DNA gets wiped from cells. An international team of scientists recently studied mitochondria in the sperm of a roundworm called C. elegans to find answers. Their results, [published](http://science.sciencemag.org/lookup/doi/10.1126/science.aaf4777) this week in the journal Science, show that paternal mitochondria in this type of roundworm have an internal self-destruct mechanism that gets activated when a sperm fuses with an egg. Delaying this mechanism, the scientists found, led to lower rates of embryo survival. Down the road, this information could help scientists better understand certain diseases and possibly improve in vitro fertilization techniques. … Before this research, it had been thought that maternal inheritance was orchestrated by processes in the mother’s egg cells, said Ding Xue, a professor at the University of Colorado Boulder and one of the authors of the paper. Large structures called autophagosomes, for instance, are known to engulf paternal mitochondria shortly after a sperm penetrates an egg. Dr. Xue and his colleagues found, however, that the paternal mitochondria in the roundworms actually started to break down before any autophagosomes reached them. ‘It’s like a suicide mechanism,’ said Byung-Ho Kang, a professor at the Chinese University of Hong Kong and another author of the paper. The researchers identified a gene, called cps-6, that seemed to initiate the breakdown process within paternal mitochondria. They found that deleting cps-6 caused paternal mitochondria to linger longer in the embryo. It also led to higher rates of embryonic death. ‘This paper provides the first experimental data suggesting that it’s not good to keep sperm mitochondrial DNA,’ said Vincent Galy, a researcher at Pierre and Marie Curie University in Paris, who was not involved in the study.”  
Source: *Why Do We Inherit Mitochondrial DNA Only From Our Mothers?*  
<https://www.nytimes.com/2016/06/24/science/mitochondrial-dna-mothers.html>

The Relationship Between Mitochondrial DNA and Nuclear DNA

  
Source: <https://en.wikipedia.org/wiki/File:Different_ways_mtDNA_moves_into_the_nucleus.PNG>

“Many mitochondrial and plastid proteins are derived from their bacterial endosymbiotic ancestors, but their genes now reside on nuclear chromosomes instead of remaining within the organelle. To become an active nuclear gene and return to the organelle as a functional protein, an organellar gene must first be assimilated into the nuclear genome. The gene must then be transcribed and acquire a transit sequence for targeting the protein back to the organelle. On reaching the organelle, the protein must be properly folded and modified, and in many cases assembled in an orderly manner into a larger protein complex. Finally, the nuclear copy must be properly regulated to achieve a fitness level comparable with the organellar gene. Given the complexity in establishing a nuclear copy, why do organellar genes end up in the nucleus? Recent data suggest that these genes are worse off than their nuclear and free-living counterparts because of a reduction in the efficiency of natural selection, but do these population–genetic processes drive the movement of genes to the nucleus? We are now at a stage where we can begin to discriminate between competing hypotheses using a combination of experimental, natural population, bioinformatic and theoretical approaches.”  
Source: *Organellar genes: why do they end up in the nucleus?*  
<https://www.sciencedirect.com/science/article/abs/pii/S0168952500020539>

“In eukaryotes, DNA is exchanged between endosymbiosis-derived compartments (mitochondria and chloroplasts) and the nucleus. Organelle-to-nucleus DNA transfer involves repair of double-stranded breaks by nonhomologous end-joining, and resulted during early organelle evolution in massive relocation of organelle genes to the nucleus. A large fraction of the products of the nuclear genes so acquired are retargeted to their ancestral compartment; many others now function in new subcellular locations. Almost all present-day nuclear transfers of mitochondrial or plastid DNA give rise to noncoding sequences, dubbed nuclear mitochondrial DNAs (NUMTs) and nuclear plastid DNAs (NUPTs). Some of these sequences were recruited as exons, thus introducing new coding sequences into preexisting nuclear genes by a novel mechanism. In organisms derived from secondary or tertiary endosymbiosis, serial gene transfers involving nucleus-to-nucleus migration of DNA have also occurred. Intercompartmental DNA transfer therefore represents a significant driving force for gene and genome evolution, relocating and refashioning genes and contributing to genetic diversity.”  
Source: *DNA transfer from organelles to the nucleus: the idiosyncratic genetics of endosymbiosis*  
<https://pubmed.ncbi.nlm.nih.gov/19014347/>

**NUMT**

**NUMT**, pronounced "new might," is an acronym for "nuclear mitochondrial DNA" segment coined by evolutionary geneticist, [Jose V. Lopez](https://en.wikipedia.org/wiki/Jose_V._Lopez), which describes a transposition of any type of cytoplasmic mitochondrial DNA into the nuclear genome of [eukaryotic](https://en.wikipedia.org/wiki/Eukaryotic) organisms.[[1]](https://en.wikipedia.org/wiki/NUMT#cite_note-Gaziev-1)[[2]](https://en.wikipedia.org/wiki/NUMT#cite_note-:0-2)[[3]](https://en.wikipedia.org/wiki/NUMT#cite_note-3)

More and more NUMT sequences, with different size and length, in the diverse number of Eukaryotes, have been detected as more [whole genome sequencing](https://en.wikipedia.org/wiki/Whole_genome_sequencing) of different [organisms](https://en.wikipedia.org/wiki/Organisms) accumulate.[[4]](https://en.wikipedia.org/wiki/NUMT#cite_note-Nomiyama-4) In fact, NUMTs have often been unintentionally discovered by researchers who were looking for mtDNA ([mitochondrial DNA](https://en.wikipedia.org/wiki/Mitochondrial_DNA)).[[5]](https://en.wikipedia.org/wiki/NUMT#cite_note-Hazkami-Covo-5) NUMTs have been reported in all studied eukaryotes, and nearly all mitochondrial genome regions can be integrated into the nuclear genome.[[6]](https://en.wikipedia.org/wiki/NUMT#cite_note-:1-6)[[7]](https://en.wikipedia.org/wiki/NUMT#cite_note-7) However, NUMTs differ in number and size across different species.[[6]](https://en.wikipedia.org/wiki/NUMT#cite_note-:1-6)[[8]](https://en.wikipedia.org/wiki/NUMT#cite_note-8)[[9]](https://en.wikipedia.org/wiki/NUMT#cite_note-9) Such differences may be accounted for by interspecific variation in such factors as [germline](https://en.wikipedia.org/wiki/Germline) stability and mitochondria number.[[10]](https://en.wikipedia.org/wiki/NUMT#cite_note-Richly-10) After the release of the mtDNA to the [cytoplasm](https://en.wikipedia.org/wiki/Cytoplasm), due to the mitochondrial alteration and [morphological](https://en.wikipedia.org/wiki/Morphology_(biology)) changes, [mtDNA](https://en.wikipedia.org/wiki/MtDNA) is transferred into the nucleus by one of the various predicted methods[[1]](https://en.wikipedia.org/wiki/NUMT#cite_note-Gaziev-1)[[5]](https://en.wikipedia.org/wiki/NUMT#cite_note-Hazkami-Covo-5) and are eventually inserted by double-stranded break repair processes into the [nuclear DNA](https://en.wikipedia.org/wiki/Nuclear_DNA) (nDNA).[[1]](https://en.wikipedia.org/wiki/NUMT#cite_note-Gaziev-1) Not only has any correlation been found between the fraction of noncoding DNA and NUMT abundance in the genome[[10]](https://en.wikipedia.org/wiki/NUMT#cite_note-Richly-10)[[11]](https://en.wikipedia.org/wiki/NUMT#cite_note-Rogers-11)[[12]](https://en.wikipedia.org/wiki/NUMT#cite_note-Tsuji-12) but NUMTs are also proven to have non-random distribution and a higher likelihood of being inserted in the certain location of genome compare to others.[[12]](https://en.wikipedia.org/wiki/NUMT#cite_note-Tsuji-12) Depending on the location of the insertion, NUMTs might perturb the function of the genes.[[1]](https://en.wikipedia.org/wiki/NUMT#cite_note-Gaziev-1) In addition, De novo integration of NUMT pseudogenes into the nuclear genome has an adverse effect in some cases, promoting various disorders and aging.[[13]](https://en.wikipedia.org/wiki/NUMT#cite_note-Dayama-13)[[14]](https://en.wikipedia.org/wiki/NUMT#cite_note-HAzkani-Covo-14)[[15]](https://en.wikipedia.org/wiki/NUMT#cite_note-Chatre-15)[[16]](https://en.wikipedia.org/wiki/NUMT#cite_note-bensasson2001-16)

…

**General characteristics of NUMT**

As the number of mitochondria and their functional level differs across eukaryotic organisms, the length, structure, and sequence of NUMTs vary dramatically.[[26]](https://en.wikipedia.org/wiki/NUMT#cite_note-antunes1-26) Researchers have found that the recent NUMT insertions are derived from different segments of the mitochondrial genome, including the [D-loop](https://en.wikipedia.org/wiki/D-loop) and, in some extreme cases, a number of, nearly, the full-length mitochondrial genome.[[10]](https://en.wikipedia.org/wiki/NUMT#cite_note-Richly-10)[[13]](https://en.wikipedia.org/wiki/NUMT#cite_note-Dayama-13) The sequence, frequency, size distribution,[[10]](https://en.wikipedia.org/wiki/NUMT#cite_note-Richly-10) and even the difficulties of finding these sequences in the genome vary substantially among species.[[1]](https://en.wikipedia.org/wiki/NUMT#cite_note-Gaziev-1)[[5]](https://en.wikipedia.org/wiki/NUMT#cite_note-Hazkami-Covo-5) The majority of DNA fragments transferred from mitochondria and plastids into the nuclear genome are less than 1 kb in size.[[1]](https://en.wikipedia.org/wiki/NUMT#cite_note-Gaziev-1)[[13]](https://en.wikipedia.org/wiki/NUMT#cite_note-Dayama-13) Yet, extremely large fragments of organelle DNA are found in some the plant genomes.[[5]](https://en.wikipedia.org/wiki/NUMT#cite_note-Hazkami-Covo-5)

As the genome evolves and alters over time by mutation, the number of NUMT in the genome differs over the course of evolution.[[5]](https://en.wikipedia.org/wiki/NUMT#cite_note-Hazkami-Covo-5) NUMT enters the nucleus and inserts in the nDNA at different stages of the time. Due to constant mutation and instability of NUMT, the resemblance of this genome stretch to the mtDNA varies widely across the kingdom [Animalia](https://en.wikipedia.org/wiki/Animalia) and even within the certain genome.[[1]](https://en.wikipedia.org/wiki/NUMT#cite_note-Gaziev-1)[[5]](https://en.wikipedia.org/wiki/NUMT#cite_note-Hazkami-Covo-5) For instance, the latest number of NUMT recorded in the human genome is 755 fragments which range from 39 bp to almost the entire mitochondrial sequence in size.[[13]](https://en.wikipedia.org/wiki/NUMT#cite_note-Dayama-13) There are 33 paralogous sequences with over 80% sequence similarity and of a greater length than 500 bp.[[34]](https://en.wikipedia.org/wiki/NUMT#cite_note-Ramos-34) Moreover, not all the NUMT fragments in the genome are the result of mtDNA migration; some are the outcome of amplification after insertion.[[13]](https://en.wikipedia.org/wiki/NUMT#cite_note-Dayama-13) Old NUMTs are found to be more abundant in the human genome than the recent integrants, indicating that mtDNA can be amplified once inserted.[[13]](https://en.wikipedia.org/wiki/NUMT#cite_note-Dayama-13) Dayama et al. developed a high yield new technique for the exact detection of the number of NUMT in the human genome called *dinumt*.[[13]](https://en.wikipedia.org/wiki/NUMT#cite_note-Dayama-13) This method enables her and her team members to identify NUMT insertions, of all sizes, in the whole genomes sequenced using paired-end sequencing technology with a greater sensitivity. They applied *dinumt* to 999 individuals from the [1000 Genomes Project](https://en.wikipedia.org/wiki/1000_Genomes_Project) and [Human Genome Diversity Project](https://en.wikipedia.org/wiki/Human_Genome_Diversity_Project) (HGDP) and conducted an updated enrichment analysis in humans using these [polymorphic](https://en.wikipedia.org/wiki/Genetic_polymorphism) insertions.[[13]](https://en.wikipedia.org/wiki/NUMT#cite_note-Dayama-13) Further investigation and genotyping of the discovered NUMT also analyses age of insertion, origin, and sequence characteristics. Finally they assessed their potential impact on ongoing studies of mitochondrial heteroplasmy.[[13]](https://en.wikipedia.org/wiki/NUMT#cite_note-Dayama-13)

As previously mentioned, mtDNA is inserted into the nuclear genome only when a DSB is produced by endogenous or exogenous damaging factors.[[1]](https://en.wikipedia.org/wiki/NUMT#cite_note-Gaziev-1) However, mtDNA is not inserted at any location within the genome.[[12]](https://en.wikipedia.org/wiki/NUMT#cite_note-Tsuji-12) Moreover, there is no correlation between the fraction of noncoding DNA and NUMT abundance;[[10]](https://en.wikipedia.org/wiki/NUMT#cite_note-Richly-10)[[11]](https://en.wikipedia.org/wiki/NUMT#cite_note-Rogers-11)[[12]](https://en.wikipedia.org/wiki/NUMT#cite_note-Tsuji-12) In addition, Antunes and Ramos found that old NUMTs are inserted preferentially into the known and predicted loci, as inferred for recent NUMTs in the human genome, during their vigorous work on NUMT sequence in fishes using BLASTN analysis method.[[26]](https://en.wikipedia.org/wiki/NUMT#cite_note-antunes1-26) Therefore, based on these studies, the insertion of NUMT in nuclear genome is found to be non-random. One of the best studies proving the non-random distribution and insertion of NUMTs in the nuclear genome is done by Tsuji and his teammates.[[12]](https://en.wikipedia.org/wiki/NUMT#cite_note-Tsuji-12) Using the LAST method instead of BLAST, which makes computing E-value with higher accuracy possible and does not under-represent the repetitive elements in NUMT flanks, Tsuji and his teammate became able to characterize the location of NUMT insertion precisely.[[12]](https://en.wikipedia.org/wiki/NUMT#cite_note-Tsuji-12) They found out that NUMT fragments tend to be inserted in the regions with high local DNA curvature or bendability and high A+T rich oligomers, especially TAT.[[12]](https://en.wikipedia.org/wiki/NUMT#cite_note-Tsuji-12)[[13]](https://en.wikipedia.org/wiki/NUMT#cite_note-Dayama-13) Moreover, NUMTs are mostly inserted into open chromatin regions.[[12]](https://en.wikipedia.org/wiki/NUMT#cite_note-Tsuji-12) Using the same method, Tsuji showed that NUMTs are not usually clustered together and the NUMTs produced by D-loop are usually under-represented which evident more vividly in monkey and human compare to rats and mouse due to the total length of their NUMTs.[[12]](https://en.wikipedia.org/wiki/NUMT#cite_note-Tsuji-12) However Tsuji also found that retrotransposon structure is highly enriched in NUMT flanks and most NUMTs are inserted in close proximity of [retrotransposon](https://en.wikipedia.org/wiki/Retrotransposon) while only a few, 10 out of 557 NUMTs, were inserted within a retrotransposon, they could not find any clear relation the size of non-coding DNA and the number of NUMT.[[12]](https://en.wikipedia.org/wiki/NUMT#cite_note-Tsuji-12)

**Consequences of De Novo Integration of NUMT Inserts**

NUMTs are not utterly functionless and certain functions are being associated with them.[[1]](https://en.wikipedia.org/wiki/NUMT#cite_note-Gaziev-1) Although the insertion of NUMTs was previously considered functionless pseudogenes, recent human NUMTs are shown to be a potentially mutagenic process that could damage the functional integrity of the human genome.[[26]](https://en.wikipedia.org/wiki/NUMT#cite_note-antunes1-26) The accumulation of mutation in NUMT, post-insertional alteration, mutagenic mechanism of NUMT insertion, MMEJ and NHEJ, DSB, as well as the place in which insertion [hot spot](https://en.wikipedia.org/wiki/Recombination_hotspot) is located can cause mutation and dramatic alterations of the genome structure at the integration site, interfere with the function of the genome, and exert substantial effects on the expression of genetic information.[[1]](https://en.wikipedia.org/wiki/NUMT#cite_note-Gaziev-1) Moreover, Integration of mtDNA sequences substantially affects the spatial organization of nDNA and may play an important role in the evolution of eukaryotic genomes.[[1]](https://en.wikipedia.org/wiki/NUMT#cite_note-Gaziev-1) In addition to the negative effect of mtDNA, those conserved old NUMTs in the genome are likely to represent evolutionary successes and they should be considered as a potential evolutionary mechanism for the enhancement of genomic coding regions.[[26]](https://en.wikipedia.org/wiki/NUMT#cite_note-antunes1-26) Moreover, Chatre and Ricchetti with the utilization of [Two-dimensional gel electrophoresis](https://en.wikipedia.org/wiki/Two-dimensional_gel_electrophoresis), [plasmid](https://en.wikipedia.org/wiki/Plasmid) construct, [mutagenesis](https://en.wikipedia.org/wiki/Mutagenesis), in a sillico analysis of ACS [motifs](https://en.wikipedia.org/wiki/Sequence_motif), and plasmid loss rate assay found that migratory mitochondrial DNAs can impact the replication of the nuclear region in which they are inserted.[[15]](https://en.wikipedia.org/wiki/NUMT#cite_note-Chatre-15) Through their functional evidence, they showed that sequences of mitochondrial origin promote nDNA replication in *Saccharomyces cerevisiae* . NUMTs are rich 11-bp [ARS](https://en.wikipedia.org/wiki/Autonomously_replicating_sequence) core-A consensus sequence (ACS), which its presence in the matches to these consensus motifs, in the *Saccharomyces cerevisiae* origin of replication, is necessary but not sufficient for the function of replication origin and any mutation in this consensus causes the reduction or loss of DNA replication activity.[[15]](https://en.wikipedia.org/wiki/NUMT#cite_note-Chatre-15) Given the high density of ACS motifs, some NUMTs appear essentially as ACS carriers.[[15]](https://en.wikipedia.org/wiki/NUMT#cite_note-Chatre-15) In contrast, replication efficiency is higher in those yeast strains that have plasmids containing both NUMT and ARS.[[15]](https://en.wikipedia.org/wiki/NUMT#cite_note-Chatre-15) They also found that some NUMTs can work as an independent replication fork and late chromosomal origins and NUMTs located close to or within ARS provide key sequence elements for replication. Thus, NUMTs can act as the independent origins, when inserted in an appropriate genomic context or affect the efficiency of pre-existing origins.[[15]](https://en.wikipedia.org/wiki/NUMT#cite_note-Chatre-15)

**Disease and Disorders:** NUMT insertion into the genome can be problematic. Transposition of NUMTs into genome has also been associated with human diseases.[[13]](https://en.wikipedia.org/wiki/NUMT#cite_note-Dayama-13)[[14]](https://en.wikipedia.org/wiki/NUMT#cite_note-HAzkani-Covo-14)[[15]](https://en.wikipedia.org/wiki/NUMT#cite_note-Chatre-15) De novo integration of NUMT pseudogenes into the nuclear genome has an adverse effect in some cases, promoting various disorders and aging.[[1]](https://en.wikipedia.org/wiki/NUMT#cite_note-Gaziev-1) MtDNA integration into coding genes in the germline cells has dramatic consequences for embryo development and, in many cases, is lethal.[[1]](https://en.wikipedia.org/wiki/NUMT#cite_note-Gaziev-1) Few NUMT pseudogenes associated with diseases are found within exons or at the exon–intron boundaries of human genes.[[1]](https://en.wikipedia.org/wiki/NUMT#cite_note-Gaziev-1) For example, the patients with [mucolipidosis](https://en.wikipedia.org/wiki/Mucolipidosis) syndrome inherit a mutation caused by insertion of a 93bp fragment of mitochondrial ND5 into exon 2 of the R403C mucolipin gene. This is the first case of a heritable disorder due to the NUMT insert.[[1]](https://en.wikipedia.org/wiki/NUMT#cite_note-Gaziev-1) Despite the small treatment group, Stem Cell transplant found to be effective and lysosomal enzyme levels seemed to normalize after transplant in at least one case.[[35]](https://en.wikipedia.org/wiki/NUMT#cite_note-35) The [Pallister–Hall syndrome](https://en.wikipedia.org/wiki/Pallister%E2%80%93Hall_syndrome), a developmental disorder, in another example, where a functional disorder of a key developmental gene results from a [*de novo*](https://en.wikipedia.org/wiki/Mutation#By_inheritance) [insertion](https://en.wikipedia.org/wiki/Insertion_(genetics)) of a 72bp mtDNA fragment into *GLI3* [exon](https://en.wikipedia.org/wiki/Exon) 14 in [chromosome 7](https://en.wikipedia.org/wiki/Chromosome_7),[[1]](https://en.wikipedia.org/wiki/NUMT#cite_note-Gaziev-1) which results in central and postaxial [polydactyly](https://en.wikipedia.org/wiki/Polydactyly), bifid epiglottis, imperforate anus, renal abnormalities including cystic malformations, [renal hypoplasia](https://en.wikipedia.org/wiki/Renal_hypoplasia), ectopic ureteral implantation, and pulmonary segmentation [anomalies](https://en.wikipedia.org/wiki/Genetic_anomaly) such as bilateral bilobed lungs.[[36]](https://en.wikipedia.org/wiki/NUMT#cite_note-biesecker-36) A splice site mutation in the human gene for plasma factor VII that causes severe plasma factor VII deficiency, bleeding disease, results from a 251-bp NUMT insertion.[[5]](https://en.wikipedia.org/wiki/NUMT#cite_note-Hazkami-Covo-5) As the last known example, a 36-bp insertion in exon 9 of the USH1C gene associated with Usher syndrome type IC is the NUMT.[[5]](https://en.wikipedia.org/wiki/NUMT#cite_note-Hazkami-Covo-5) No certain curse has yet found for [Usher](https://en.wikipedia.org/wiki/Usher_syndrome) syndrome, however, a current clinical study on 18 volunteers is taking place to determine the influence of UshStat both in a short and a long-term period. This study has been started in September 2013 and is estimated to be done by October 2023.[[37]](https://en.wikipedia.org/wiki/NUMT#cite_note-37)

**Aging:** Several studies indicated that de novo appearance of NUMT pseudogenes in the genome of somatic cells may be of [etiological](https://en.wikipedia.org/wiki/Etiological) importance for carcinogenesis and aging.[[1]](https://en.wikipedia.org/wiki/NUMT#cite_note-Gaziev-1)[[13]](https://en.wikipedia.org/wiki/NUMT#cite_note-Dayama-13) To show the relation between aging and NUMT in the nuclear genome, Cheng and Ivessa used *yme1-1* mutant strains of Saccharomyces Cerevisiae that have a higher rate of mtDNA migration.[[38]](https://en.wikipedia.org/wiki/NUMT#cite_note-Ivessa-38) The method is exactly the same as the method Thorsness and Fox used to determine the important mechanisms and factors for mtDNA migration into the nucleus.[[29]](https://en.wikipedia.org/wiki/NUMT#cite_note-Thorsness_1993-29)[[38]](https://en.wikipedia.org/wiki/NUMT#cite_note-Ivessa-38) They found out the yeast strains with elevated migration rates of mtDNA fragments to the nucleus showed accelerated chronological aging, whereas, strains with decreased mtDNA transfer rates to the nucleus exhibited an extended CLS, chronological life span [[38]](https://en.wikipedia.org/wiki/NUMT#cite_note-Ivessa-38) which could possibly be due to the effect of NUMT on nuclear processes including DNA replication, recombination, and repair as well as gene transcription.[[15]](https://en.wikipedia.org/wiki/NUMT#cite_note-Chatre-15)[[38]](https://en.wikipedia.org/wiki/NUMT#cite_note-Ivessa-38) The effect of NUMT on the higher Eukaryotic organisms was investigated by Caro and his teammates in the rats as a model organism. Using a real-time PCR quantification, in situ hybridization of mtDNA to [nDNA](https://en.wikipedia.org/wiki/NDNA), and comparison of young and old rats, Caro and his crew not only could determine the high concentration of [cytochrome oxidase](https://en.wikipedia.org/wiki/Cytochrome_oxidase) III and 16S [rRNA](https://en.wikipedia.org/wiki/RRNA) from mtDNA in both young and old rats, but they also could find out the increase in the number of mitochondrial sequences in nDNA as the rat gets older.[[39]](https://en.wikipedia.org/wiki/NUMT#cite_note-Caro-39) Thus, based on these findings, mitochondria can be a major trigger of aging, but the final target could also be the nucleus.[[38]](https://en.wikipedia.org/wiki/NUMT#cite_note-Ivessa-38)[[39]](https://en.wikipedia.org/wiki/NUMT#cite_note-Caro-39)

**Cancer:** The most dreadful impact of NUMT insertion happens when the mtDNA is inserted into the regulatory region or nuclear structural genes and disrupts or alters the vital cell processes.[[1]](https://en.wikipedia.org/wiki/NUMT#cite_note-Gaziev-1)[[31]](https://en.wikipedia.org/wiki/NUMT#cite_note-Campbell-31) For instance, in primary low-grade brain neoplasms, fluorescent in situ hybridization analysis helped with the recognition of mtDNA localized in the nucleus in correlation with an overall increase in mtDNA content in the cell.[[40]](https://en.wikipedia.org/wiki/NUMT#cite_note-Liang-40) This ontogenically early event is important in the etiology of these tumors.[[40]](https://en.wikipedia.org/wiki/NUMT#cite_note-Liang-40) Similarly, in [hepatoma](https://en.wikipedia.org/wiki/Hepatoma) cells mtDNA sequences are present in the nuclear genome at a higher copy number in contrast with the normal tissues.[[18]](https://en.wikipedia.org/wiki/NUMT#cite_note-Ju-18)[[31]](https://en.wikipedia.org/wiki/NUMT#cite_note-Campbell-31) Another example would be [HeLa](https://en.wikipedia.org/wiki/HeLa) nDNA that contains sequences which hybridize with mtDNA fragments of approximately 5 kb. An analysis showed that nDNA of malignant cells contains sequences of the mitochondrial *cytochrome oxidase I*, *ND4* , *ND4L* , and 12S rRNA genes.[[18]](https://en.wikipedia.org/wiki/NUMT#cite_note-Ju-18) Based on these findings, mtDNA fragments were assumed to act as a mobile genetic element in the initiation of [carcinogenesis](https://en.wikipedia.org/wiki/Carcinogenesis).[[1]](https://en.wikipedia.org/wiki/NUMT#cite_note-Gaziev-1) [Southern blotting](https://en.wikipedia.org/wiki/Southern_blotting) is the method used to determine the frequency of mitochondrial insertion in nDNA of the normal and the tumor cells of mice and rats, which proved that the mtDNA sequences are far more numerous and abundant in nDNA of rodent tumor cells in comparison with normal cells.[[1]](https://en.wikipedia.org/wiki/NUMT#cite_note-Gaziev-1) Using FISH probes, PCR and data sequencing, mapping and comparison, Ju and his teammate found that the mitochondrial-nuclear genome fusions occur at a similar rate per base pair of DNA as interchromosomal nuclear rearrangements, indicating the presence of a high frequency of contact between mitochondrial and nuclear DNA in some somatic cells.[[18]](https://en.wikipedia.org/wiki/NUMT#cite_note-Ju-18) Also, Ju and his teammates investigated the timing of somatic mtDNA integration into the nuclear genome by assessing cases in which a metastatic sample had been sequenced in addition to the primary tumor.[[18]](https://en.wikipedia.org/wiki/NUMT#cite_note-Ju-18) In some cases, mtDNA transfers into the nucleus in somatic cells are very frequent and can occur after neoplastic formation and during the course of subclonal evolution of cancer which suggest that this event occurs in the common ancestral cancer clones or in normal somatic cells prior to the neoplastic change.[[18]](https://en.wikipedia.org/wiki/NUMT#cite_note-Ju-18) These findings demonstrated that the presence of direct correlation between NUMT and cancer in different body organs.[[16]](https://en.wikipedia.org/wiki/NUMT#cite_note-bensasson2001-16)[[18]](https://en.wikipedia.org/wiki/NUMT#cite_note-Ju-18) Understanding the relation, the timing of the NUMT insertion, location of the insertion, and disrupted genes would help with producing more powerful and effective medicine.[[5]](https://en.wikipedia.org/wiki/NUMT#cite_note-Hazkami-Covo-5)   
Source: *NUMT*  
<https://en.wikipedia.org/wiki/NUMT>

“Mitochondria contain a separate protein-synthesis machinery to produce the polypeptides encoded in mitochondrial DNA (mtDNA), and many mtDNA disease mutations affect this machinery. In humans, the mitochondrial rRNAs and tRNAs are encoded by mtDNA, whereas all proteins involved in mitochondrial translation are encoded by nuclear genes. Recently, several articles have discussed the identification of pathological mutations in nuclear genes encoding components of this protein-synthesis machinery, suggesting that these types of mutation are a frequent cause of human genetic diseases.”  
Source: *Nuclear genes and mitochondrial translation: a new class of genetic disease*  
<https://pubmed.ncbi.nlm.nih.gov/15922826/>

Can mRNA be Engineered to Enter the Mitochondria?

“Mitochondria contain hundreds of proteins but only a few are encoded by the mitochondrial genome. The other proteins are nuclear-encoded and imported into mitochondria. These proteins can be translated on free cytosolic polysomes, then targeted and imported into mitochondria. Nonetheless, numerous cytosolic mRNAs encoding mitochondrial proteins are detected at the surface of mitochondria in yeast, plants and animals. The localization of mRNAs to the vicinity of mitochondria would be a way for mitochondrial protein sorting. The mechanisms responsible for mRNA targeting to mitochondria are not clearly identified. Sequences within the mRNA molecules (cis-elements), as well as a few trans-acting factors, have been shown to be essential for targeting of some mRNAs. In order to identify receptors involved in mRNA docking to the mitochondrial surface, we have developed an in vitro mRNA binding assay with isolated plant mitochondria. We show that naked mRNAs are able to bind to isolated mitochondria, and our results strongly suggest that mRNA docking to the plant mitochondrial outer membrane requires at least one component of TOM complex.”  
Source: *Targeting of cytosolic mRNA to mitochondria: Naked RNA can bind to the mitochondrial surface*  
<https://www.researchgate.net/publication/258765552_Targeting_of_cytosolic_mRNA_to_mitochondria_Naked_RNA_can_bind_to_the_mitochondrial_surface>

“Mitochondria harbor their own genetic system, yet critically depend on the import of a number of nuclear-encoded macromolecules to ensure their expression. In all eukaryotes, selected non-coding RNAs produced from the nuclear genome are partially redirected into the mitochondria, where they participate in gene expression. Therefore, the mitochondrial RNome represents an intricate mixture of the intrinsic transcriptome and the extrinsic RNA importome. In this review, we summarize and critically analyze data on the nuclear-encoded transcripts detected in human mitochondria and outline the proposed molecular mechanisms of their mitochondrial import. … Mitochondria possess their own genome (mtDNA), which, in humans, encodes 11 mRNAs, 2 ribosomal, and 22 transfer RNAs required for the synthesis of 13 proteins of the oxidative phosphorylation complexes ([Figure 1](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6468882/figure/cells-08-00286-f001/)). However, this is far from being sufficient to perform all their functions, which necessitate more than 1000 proteins encoded by the nuclear DNA, synthetized in the cytosol and imported into mitochondria [[1](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6468882/#B1-cells-08-00286)]. These include structural components, enzymes, and all the protein factors required for the maintenance and expression of the small mitochondrial genome. Additionally, in all groups of eukaryotes, some non-coding RNAs have been predicted or experimentally demonstrated to translocate into the mitochondria; this import pathway is often essential for the mitochondrial function [[2](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6468882/#B2-cells-08-00286),[3](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6468882/#B3-cells-08-00286),[4](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6468882/#B4-cells-08-00286)]. In many cases, this is to be expected: the absence of an RNA gene whose product is strictly required for mitochondrial translation from the mitochondrial genome is usually considered a strong indication for the existence of a compensatory mitochondrial RNA import pathway. Indeed, in some species, tRNAs for select amino acids are not encoded in mtDNA and must be imported from the cytosol, ranging from just a few tRNAs in plants to the complete tRNA set in such protists as *Trypanosoma brucei* [[5](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6468882/#B5-cells-08-00286)] and *Leishmania tarentolae* [[6](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6468882/#B6-cells-08-00286)]. However, even in species in which all required tRNAs are encoded by the mitochondrial genome, tRNA import from the cytosol has been observed and is likely to become essential under particular stress conditions.”  
Source: *Import of Non-Coding RNAs into Human Mitochondria: A Critical Review and Emerging Approaches*  
<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6468882/>

“Recent findings of high rates of transfer of organelle DNA to the nucleus [[1](https://genomebiology.biomedcentral.com/articles/10.1186/gb-2005-6-5-110#ref-CR1)], and of high rates of functional gene transfer from organelles to the nucleus [[2](https://genomebiology.biomedcentral.com/articles/10.1186/gb-2005-6-5-110#ref-CR2)–[5](https://genomebiology.biomedcentral.com/articles/10.1186/gb-2005-6-5-110#ref-CR5)], demonstrate that the endosymbiotic origin of organelles was a major determinant in defining eukaryotic nuclear genomes and was probably a defining event for the formation of the eukaryotic cell [[1](https://genomebiology.biomedcentral.com/articles/10.1186/gb-2005-6-5-110#ref-CR1), [6](https://genomebiology.biomedcentral.com/articles/10.1186/gb-2005-6-5-110#ref-CR6)]. … The steps required for a gene to be transferred from an organelle to the nucleus. (a) The gene must be transferred from the organelle, either as a fragment of organellar DNA or as a cDNA, and (b) integrated into a nuclear chromosome. (c) The gene must then acquire the signals for expression, including promoter, terminator, and polyadenylation signals, and also a signal to target the protein back to the organelle. These events may occur together or separately. (d) The expressed gene may be translated on free polysomes to produce a protein that is targeted to mitochondria, or alternatively the mRNA may be targeted to mitochondria to be translated on the surface. (e) The targeting signal must be removed and (f) the protein has to be assembled in order for it to function. Assembly may require re-sorting to the correct location within the organelle and additional processing of sorting signals.”  
Source: *Why genes persist in organelle genomes*  
<https://genomebiology.biomedcentral.com/articles/10.1186/gb-2005-6-5-110>

“… to produce a protein that is targeted to mitochondria, or alternatively the mRNA may be targeted to mitochondria to be translated on the surface.”

“As previously established in yeast, two sequences within mRNAs are responsible for their specific localization to the mitochondrial surface—the region coding for the mitochondrial targeting sequence and the 3′UTR. This phenomenon is conserved in human cells. Therefore, we decided to use mRNA localization as a tool to address to mitochondria, a protein that is not normally imported. For this purpose, we associated a nuclear recoded ATP6 gene with the mitochondrial targeting sequence and the 3′UTR of the nuclear SOD2 gene, which mRNA exclusively localizes to the mitochondrial surface in HeLa cells. The ATP6 gene is naturally located into the organelle and encodes a highly hydrophobic protein of the respiratory chain complex V. In this study, we demonstrated that hybrid ATP6 mRNAs, as the endogenous SOD2 mRNA, localize to the mitochondrial surface in human cells. Remarkably, fusion proteins localize to mitochondria in vivo. Indeed, ATP6 precursors synthesized in the cytoplasm were imported into mitochondria in a highly efficient way, especially when both the MTS and the 3′UTR of the SOD2 gene were associated with the re-engineered ATP6 gene. Hence, these data indicate that mRNA targeting to the mitochondrial surface represents an attractive strategy for allowing the mitochondrial import of proteins originally encoded by the mitochondrial genome without any amino acid change in the protein that could interfere with its biologic activity.”  
Source: *mRNA localization to the mitochondrial surface allows the efficient translocation inside the organelle of a nuclear recoded ATP6 protein*  
<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1484424/>

“mRNA subcellular distribution and translational control are key player mechanisms for development, cellular differentiation and synaptic plasticity. mRNA localization is also implicated in mitochondria biogenesis. Two sequences within the transcripts are involved in their mitochondrial localization: the region coding for the mitochondrial targeting sequence (MTS) and the 3'UTR. Therefore, we decided to use mRNA localization as a tool to address to mitochondria a protein that is not normally imported. We have chosen to construct nuclear versions of the mtDNA encoded ATP6 and ND1 genes to which we appended the signals of COX10 gene, whose transcript is sorted to the mitochondrial surface. Thus, by directing a hybrid mRNA to the mitochondrial surface, we significantly improved the feasibility of the allotopic approach for the mitochondrial genes examined.”  
Source: *[mRNA localization to the mitochondrial surface: a tool to treat retinal pathologies due to mitochondrial DNA mutations]*  
<https://pubmed.ncbi.nlm.nih.gov/17762826/>

“Here, we report that a 20-ribonucleotide stem-loop sequence from the *H1* RNA, the RNA component of the human RNase P enzyme, appended to a nonimported RNA directs the import of the resultant RNA fusion transcript into human mitochondria. The methodology is effective for both noncoding RNAs, such as tRNAs, and mRNAs. The RNA import component, polynucleotide phosphorylase (PNPASE), facilitates transfer of this hybrid RNA into the mitochondrial matrix. In addition, nucleus-encoded mRNAs for mitochondrial proteins, such as the mRNA of human mitochondrial ribosomal protein S12 (*MRPS12*), contain regulatory sequences in their 3′-untranslated region (UTR) that confers localization to the mitochondrial outer membrane, which is postulated to aid in protein translocation after translation. We show that for some mitochondrial-encoded transcripts, such as *COX2*, a 3′-UTR localization sequence is not required for mRNA import, whereas for corrective mitochondrial-encoded tRNAs, appending the 3′-UTR localization sequence was essential for efficient fusion-transcript translocation into mitochondria. In vivo, functional defects in mitochondrial RNA (mtRNA) translation and cell respiration were reversed in two human disease lines. Thus, this study indicates that a wide range of RNAs can be targeted to mitochondria by appending a targeting sequence that interacts with PNPASE, with or without a mitochondrial localization sequence, providing an exciting, general approach for overcoming mitochondrial genetic disorders.”  
Source: *Correcting human mitochondrial mutations with targeted RNA import*  
<https://www.pnas.org/content/109/13/4840>

“We report on the validation of a mitochondrial gene therapeutic strategy using fibroblasts from a Leigh syndrome patient by the mitochondrial delivery of therapeutic mRNA. The treatment involves delivering normal ND3 protein-encoding mRNA as a therapeutic RNA to mitochondria of the fibroblasts from a patient with a T10158C mutation in the mtDNA coding the ND3 protein, a component of the mitochondrial respiratory chain complex I. The treatment involved the use of a liposome-based carrier (a MITO-Porter) for delivering therapeutic RNA to mitochondria *via* membrane fusion.”  
Source: *Validation of a mitochondrial RNA therapeutic strategy using fibroblasts from a Leigh syndrome patient with a mutation in the mitochondrial ND3 gene*  
<https://www.nature.com/articles/s41598-020-64322-8>

“The majority of mitochondrial proteins are products of nuclear genes and are synthesized in the cytosol, then translocated into the mitochondria.[3](https://www.ahajournals.org/doi/full/10.1161/circresaha.112.267732#B3),[4](https://www.ahajournals.org/doi/full/10.1161/circresaha.112.267732#B4) … MicroRNAs (miRNAs) are small noncoding RNA molecules that regulate gene expression by inhibiting mRNA translation and/or inducing mRNA degradation.[5](https://www.ahajournals.org/doi/full/10.1161/circresaha.112.267732#B5),[6](https://www.ahajournals.org/doi/full/10.1161/circresaha.112.267732#B6) Several groups have proposed that miRNAs play critical roles in cardiovascular physiology and disease pathogenesis.[7](https://www.ahajournals.org/doi/full/10.1161/circresaha.112.267732#B7)[–](https://www.ahajournals.org/doi/full/10.1161/circresaha.112.267732#B8)[9](https://www.ahajournals.org/doi/full/10.1161/circresaha.112.267732#B9) Powerful cardioprotective interventions, such as ischemic preconditioning, also induce changes in miRNAs.[10](https://www.ahajournals.org/doi/full/10.1161/circresaha.112.267732#B10),[11](https://www.ahajournals.org/doi/full/10.1161/circresaha.112.267732#B11)The goal of this study was to determine if miRNA, like protein, could translocate into the mitochondria and regulate mitochondrial function with possible pathophysiological implications in cardiac myocytes. Others have found miRNA in the mitochondria of liver cells, HeLa cells, and human myoblasts,[12](https://www.ahajournals.org/doi/full/10.1161/circresaha.112.267732#B12)[–](https://www.ahajournals.org/doi/full/10.1161/circresaha.112.267732#B13 B14)[15](https://www.ahajournals.org/doi/full/10.1161/circresaha.112.267732#B15) but the significance and functional consequences were not characterized.”  
Source: *Nuclear miRNA Regulates the Mitochondrial Genome in the Heart*  
<https://www.ahajournals.org/doi/full/10.1161/circresaha.112.267732>

So, you’re giving the companies who get paid to create vaccines, medicine, and therapeutics the ability to create sickness and disease in your body?

**Moderna boss: mRNA jabs are "rewriting the Genetic Code" we call it "information therapy" (Ted 2017)**  
<https://www.youtube.com/watch?v=lfXItfe07Wc>

Alternative source:  
<https://www.bitchute.com/search/?query=moderna%20boss&kind=video>

So, this new form of medicine/treatment was already a goal.

“Within our platform, we develop technologies that enable the development of mRNA medicines for diverse applications. When we identify technologies that we believe could enable a new group of potential mRNA medicines with shared product features, we call that group a ‘modality.’ While the programs within a modality may target diverse diseases, they share similar mRNA technologies, delivery technologies and manufacturing processes to achieve shared product feature. The programs within a modality will also generally share similar pharmacology profiles, including the desired dose response, the expected dosing regimen, the target tissue for protein expression, safety and tolerability goals, as well as pharmaceutical properties. Programs within a modality often have correlated technology risk, but because they pursue diverse diseases they often have uncorrelated biology risk.”   
Source: *mRNA Technological Solutions to Create New Medicines*<https://www.modernatx.com/modalities-mrna-vaccines-therapeutics-and-immuno-oncology>

One of the six modalities Moderna claims to have is mRNA Systemic Intracellular Therapeutics. Of this modality they state, “We designed our systemic intracellular therapeutics modality to increase levels of intracellular proteins, using cells in the human body to produce proteins located in the cytosol or specific organelles of the cell to achieve a therapeutic effect in one or more tissues or cell types. The goal of this modality is to provide intracellular proteins, such as intracellular enzymes and organelle-specific proteins, as safe, tolerable, and efficacious therapies. Our initial focus within this modality is on rare genetic diseases. This modality currently has three programs.”

Nanocarriers

“Lipid nanoparticles (LNPs) are the most clinically advanced non-viral gene delivery system. Lipid nanoparticles safely and effectively deliver nucleic acids, overcoming a major barrier preventing the development and use of genetic medicines. Genetic medicine has many different applications such as gene editing, rapid vaccine development, immuno-oncology and treatment of rare genetic and undruggable diseases; all of which are usually hindered by nucleic acid delivery inefficiency.”  
Source: *Lipid Nanoparticles*  
<http://www.precisionnanosystems.com/workflows/formulations/lipid-nanoparticles>

“Nanotechnology offers unique tools and materials to target therapeutic agents to mitochondria. As discussed in this paper, a variety of functionalized nanosystems including polymeric and metallic nanoparticles as well as liposomes are more effective than plain drug and non-functionalized nanosystems in delivering therapeutic agents to mitochondria. Although the field is in its infancy, studies to date suggest the superior therapeutic activity of functionalized nanosystems for treating mitochondrial defects.”  
Source: *Functionalized Nanosystems for Targeted Mitochondrial Delivery*  
(Published online Nov 23, 2011)  
<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3299903/>

“In the recent years, targeting mitochondria emerged as an attractive strategy to control mitochondrial dysfunction related diseases. Despite the desire to direct therapeutics to the mitochondria, the actual task is more difficult due to the highly complex nature of the mitochondria. The potential benefits of integrating nanomaterials with properties such as biodegradability, magnetization, fluorescence, and near-infrared absorption into a single object of nanoscale dimensions can lead to the development of hybrid nano-medical platforms for targeting therapeutics to the mitochondria. Only a handful of nanoparticles based on metal oxides, gold nanoparticles, dendrons, carbon nanotubes, and liposomes were recently engineered to target mitochondria. Most of these materials face tremendous challenges when administered *in vivo* due to their limited biocompatibility. Biodegradable polymeric nanoparticles emerged as eminent candidates for effective drug delivery. In this review we highlight the current advancements in the development of biodegradable nanoparticle platforms as effective targeting tools for mitochondrial medicine.”  
Source: *Targeted Nanoparticles in Mitochondrial Medicine*  
(Published online Oct 27, 2014)  
<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4397104/>

“Effective targeting of mitochondria has emerged as an alternative strategy in cancer chemotherapy. However, considering mitochondria's crucial role in cellular energetics, metabolism and signaling, targeting mitochondria with small molecules would lead to severe side effects in cancer patients. Moreover, mitochondrial functions are highly dependent on other cellular organelles like nucleus. Hence, simultaneous targeting of mitochondria and nucleus could lead to more effective anticancer strategy. To achieve this goal, we have developed sub 200 nm particles from dual drug conjugates derived from direct tethering of mitochondria damaging drug (α- tocopheryl succinate) and nucleus damaging drugs (cisplatin, doxorubicin and paclitaxel). These dual drug conjugated nanoparticles were internalized into the acidic lysosomal compartments of the HeLa cervical cancer cells through endocytosis and induced apoptosis through cell cycle arrest. These nanoparticles damaged mitochondrial morphology and triggered the release of cytochrome c. Furthermore, these nanoparticles target nucleus to induce DNA damage, fragment the nuclear morphology and damage the cytoskeletal protein tubulin. Therefore, these dual drug conjugated nanoparticles can be successfully used as a platform technology for simultaneous targeting of multiple subcellular organelles in cancer cells to improve the therapeutic efficacy of the free drugs.”  
Source: *Dual drug conjugated nanoparticle for simultaneous targeting of mitochondria and nucleus in cancer cells*  
(Publication Date: March 26, 2015)  
<https://pubs.acs.org/doi/10.1021/am5090226>

“Owing to the mitochondrial special bilayer structure and highly negative potential nature, therapeutic molecules have multiple difficulties in reaching mitochondria. To overcome multiple barriers for targeting mitochondria, the researchers developed various pharmaceutical preparations such as [liposomes](https://www.sciencedirect.com/topics/pharmacology-toxicology-and-pharmaceutical-science/liposome), polymeric [nanoparticles](https://www.sciencedirect.com/topics/pharmacology-toxicology-and-pharmaceutical-science/nanoparticle) and inorganic nanoparticles modified by mitochondriotropic moieties like [dequalinium](https://www.sciencedirect.com/topics/pharmacology-toxicology-and-pharmaceutical-science/dequalinium) (DQA), triphenylphosphonium (TPP), mitochondrial penetrating peptides (MPPs) and [mitochondrial protein](https://www.sciencedirect.com/topics/pharmacology-toxicology-and-pharmaceutical-science/mitochondrial-protein) import machinery that allow specific targeting. The targeted formulations exhibited enhanced pharmacological effect and better therapeutic effect than their untargeted counterpart both *in vitro* and *in vivo*. [Nanocarriers](https://www.sciencedirect.com/topics/pharmacology-toxicology-and-pharmaceutical-science/nanocarriers) may be used for bio-therapeutic delivery into specific mitochondria that possess a great potential treatment of mitochondria related diseases.”  
Source: *Nanopreparations for mitochondria targeting drug delivery system: Current strategies and future prospective*  
(Available online 24 May 2017)  
<https://www.sciencedirect.com/science/article/pii/S1818087617302751>

“Here, we report on validating a mitochondrial gene therapy by delivering nucleic acids to mitochondria of diseased cells by a MITO-Porter, a liposome-based carrier for mitochondrial de-livery. … To date, the transport of several RNA sequences from the cytoplasm to mitochondria has been reported.[7–9](https://www.cell.com/molecular-therapy-family/nucleic-acids/pdf/S2162-2531(20)30112-8.pdf#%FE%FF%00b%00i%00b%007) However, it has been shown that RNA is not transported to mitochondria by itself, and a cytosolic endogenous protein for mitochondrial transport is required. Since the level of expression of this protein is rate limiting, the efficiency of transport to mitochondria is a very low level. In order to transport much larger amounts of RNA to mitochondria, a MITO-Porter, a liposome-based carrier for the mitochondrial delivery, was developed in our laboratory,[10–16](https://www.cell.com/molecular-therapy-family/nucleic-acids/pdf/S2162-2531(20)30112-8.pdf#%FE%FF%00b%00i%00b%001%000) and was found to be a useful strategy. The MITO-Porter is internalized into cells and delivers encapsulated molecules to mitochondria via membrane fusion, a process that is independent of its size and physical properties. Therefore, this system could be used for the direct mitochondrial transfection of nucleic acids.”  
Source: *Validation of Gene Therapy for Mutant Mitochondria by Delivering Mitochondrial RNA Using a MITO-Porter*  
(June 2020)  
<https://www.cell.com/molecular-therapy-family/nucleic-acids/pdf/S2162-2531(20)30112-8.pdf>

“mRNA vaccines rely on the delivery of mRNA into the cytoplasm of host cells, where it can be transcribed into antigenic proteins to trigger the production of neutralizing antibodies. However, mRNA is three to four orders of magnitude larger than molecules that readily diffuse into cells; in addition, the dense negative charge of mRNA electrostatically repulses the anionic cell membrane, preventing its uptake. Therefore, mRNA vaccines require a delivery vehicle that not only protects the nucleic acid from degradation but allows the mRNA to get into cells. BioNTech/Pfizer’s and Moderna’s mRNA vaccines both use lipid nanoparticles as mRNA carriers. The impressive speed at which these vaccines could be developed is partly owed to the fact that nucleic acid delivery by lipid nanoparticles has long been investigated and optimized by the nanomedicine community, who thoroughly studied lipid nanoparticle chemistry, structure, surface, injection routes, uptake, endosomal escape, cargo release, dosage, clearance and, importantly, safety. This interest in lipid nanoparticle research has been driven by the emergence of promising new mRNA-based therapies and gene editing technologies for a variety of diseases, the success of which depends on the availability of a safe and efficient delivery vehicle.”  
Source: *Let’s talk about lipid nanoparticles*  
(Published: February 09, 2021)  
<https://www.nature.com/articles/s41578-021-00281-4>

“This interest in lipid nanoparticle research has been driven by the emergence of promising new mRNA-based therapies and gene editing technologies…” They could target your nuclear DNA or mitochondrial DNA, and you wouldn’t even know it. And they could be telling you this is all being done to fight disease when it’s really, or also, being done to edit your genes, and you wouldn’t even know it. A Trojan horse?

Gene-editing Using mRNA/RNA

“The CRISPR/Cas9 gene-editing tool has revolutionized biomedical research and has tremendous potential for use in the clinic. There are still many challenges that need to be addressed before it’s safe for people, however. A team of scientists from Tufts University and the Chinese Academy of Sciences has now improved the way CRISPR/Cas9 tools are delivered to cells where they’re supposed to make the intended edits. This approach utilizes synthetic and biodegradable lipid nanoparticles, which deliver reagents that make the edits with about 90 percent efficiency. The nanoparticles, [reported in Advanced Materials](http://dx.doi.org/10.1002/adma.201902575), can help scientists create a tool that is useful in the clinic as a therapeutic. One problem with CRISPR/Cas9, which is described in the video, is its size. It requires an enzyme called a nuclease that cuts the genome, in this case, it’s Cas9, as well as a specific RNA molecule that can guide the nuclease to the precise point in the genome where the cut should be made. The genome is located in the nucleus of the cell, and the size of the CRISPR/Cas9 tool can prohibit it from entering. Scientists have tried packaging it into viruses, polymers, or other kinds of nanoparticles with limited success. In this work, the researchers turned to lipid nanoparticles to carry messenger RNA encoding for the Cas9 nuclease, which is translated when it enters a cell, instead of the nuclease itself. The synthetic lipids composing the nanoparticles contain disulfide bonds, which break open when it gets inside of a cell; the contents of the nanoparticles then get rapidly released there. … ‘The lipid nanoparticles are one of the most efficient CRISPR/Cas9 carriers we have seen,’ said co-corresponding study author Ming Wang, a professor at the Chinese Academy of Sciences, Beijing National Laboratory for Molecular Science.”  
Source: *Lipid Nanoparticles Improve the Delivery of CRISPR/Cas9 Into Cells*  
<https://www.labroots.com/trending/genetics-and-genomics/15202/lipid-nanoparticles-improve-delivery-crispr-cas9-cells>

“RNA can also be engineered, as Jennifer Doudna and others discovered, to target genes for editing. Using the CRISPR system adapted from bacteria, RNA can guide scissors-like enzymes to specific sequences of DNA in order to eliminate or edit a gene. This technique has already been used in trials to cure sickle cell anemia. Now it is also being used in the war against COVID. Doudna and others have created RNA-guided enzymes that can directly detect SARS-CoV-2 and eventually could be used to destroy it. More controversially, CRISPR could be used to create ‘designer babies’ with inheritable genetic changes. In 2018, a young Chinese doctor used CRISPR to engineer twin girls so they did not have the receptor for the virus that causes AIDS. There was an immediate outburst of awe and then shock. The doctor was denounced, and there were calls for an international moratorium on inheritable gene edits. But in the wake of the pandemic, RNA-guided genetic editing to make our species less receptive to viruses may someday begin to seem more acceptable.”  
Source: *mRNA Technology Gave Us the First COVID-19 Vaccines. It Could Also Upend the Drug Industry*<https://time.com/5927342/mrna-covid-vaccine/>

“RNA can also be engineered, as Jennifer Doudna and others discovered, to target genes for editing. Using the CRISPR system adapted from bacteria, RNA can guide scissors-like enzymes to specific sequences of DNA in order to eliminate or edit a gene. … Now it is also being used in the war against COVID. Doudna and others have created RNA-guided enzymes that can directly detect SARS-CoV-2…”

And it was called “inheritable gene edits.”

If you wanted, you could make human beings into whatever you wanted them to be. This would work well when starting from scratch with humans, getting them to believe there are no genders, and convincing them to erase all that is human by getting them to stay away from one another, eventually banning human-to-human contact.

**How CRISPR lets us edit our DNA | Jennifer Doudna**  
<https://www.youtube.com/watch?v=TdBAHexVYzc>

3:35 – “And importantly, those bits of DNA are passed on to the cell’s progeny.”

Alternative source:  
<https://www.bitchute.com/video/7SXbxJ9eD8AW/>

**What you need to know about CRISPR | Ellen Jorgensen**<https://www.youtube.com/watch?v=1BXYSGepx7Q>

Additional info:

CRISPR gene-editing tool causes unintended genetic mutations  
<https://newatlas.com/crispr-gene-editing-causes-mutations/49762/>

CRISPR-induced mutations – what do they mean for food safety?  
<https://gmwatch.org/en/news/latest-news/17657-crispr-induced-mutations-what-do-they-mean-for-food-safety>

Bill Gates and others have been talking about administering quantum dot tattoos to identify who has had the virus.  
  
“There is growing concern about the safety of engineered nanoparticles, which are produced for various industrial applications. Quantum dots are colloidal semiconductor nanoparticles that have unique luminescence characteristics and the potential to become attractive tools for medical imaging. However, some of these particles can cause oxidative stress and induce cell death. The objective of this study was to explore quantum dot-induced metabolic changes, which could occur without any apparent cellular damage.”  
Source: *Nanoparticles can induce changes in the intracellular metabolism of lipids without compromising cellular viability*  
(accepted August 24, 2009)  
<https://febs.onlinelibrary.wiley.com/doi/pdfdirect/10.1111/j.1742-4658.2009.07324.x>

Now what do you think about the claim that the COVID vaccine can’t alter your DNA, and isn’t gene therapy, because it doesn’t enter the cell’s nucleus where your DNA is found?

If you want, compare what you’ve just learned to what the media, fact-checkers and others have been telling you. Here’s some of their deceit:

<https://www.mskcc.org/coronavirus/myths-about-covid-19-vaccines>

<https://www.cdc.gov/coronavirus/2019-ncov/vaccines/facts.html#:~:text=No.%20COVID%2D19,DNA%20in%20any%20way>.

<https://www.reuters.com/article/factcheck-vaccine-dna/fact-check-the-covid-19-vaccine-does-not-change-recipients-genetic-makeup-idUSL2N2L23EM>

<https://www.reuters.com/article/factcheck-moderna-mrna/fact-check-modernas-chief-medical-officer-did-not-say-mrna-vaccines-alter-dna-idUSL1N2M10IV>

<https://factcheck.afp.com/vaccines-dont-change-your-dna>

<https://www.snopes.com/fact-check/mrna-alter-dna/>

<https://www.factcheck.org/2021/03/scicheck-texas-doctor-spreads-false-claims-about-covid-19-vaccines/>

<https://www.newswise.com/factcheck/covid-vaccines-aren-t-gene-therapy>

The Verdict: FALSE