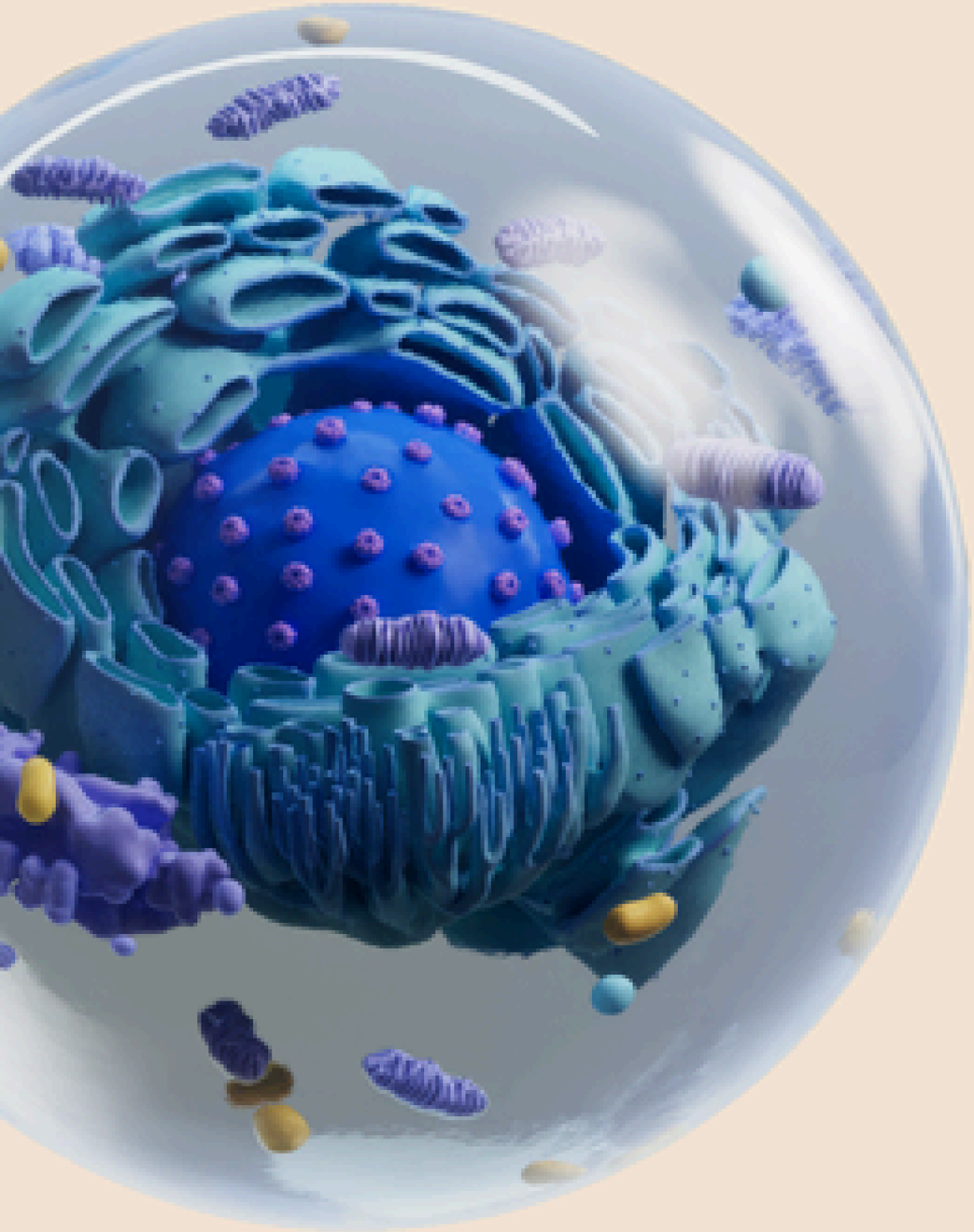


BODYBIO

Optimizing Fertility:
The Impact of Mitochondrial Function on Reproductive Health

Justine Stenger
BodyBio Clinical Educator

Disclaimer

Statements about BodyBio’s Products have not been evaluated by the FDA or any other government agency. BodyBio's Products are not intended to diagnose, treat, cure, mitigate or prevent any disease and BodyBio’s Products are not offered and will not be sold for such purposes.

The health information provided by BodyBio in this webinar is provided as an information resource only, and is not to be used or relied on for any diagnostic or treatment purposes. This information is not intended to be patient education, does not create any patient-physician relationship, and should not be used as a substitute for professional diagnosis and treatment.

Agenda

1. Understanding Infertility, Circadian Biology and Mitochondria
2. Understanding Mitochondrial Heteroplasmy
3. Redox & Fertility
4. The Impact Of Phospholipids and Fatty Acids on Mitochondria For Reproductive Health
5. The Role of Light In Regulating our Biology
6. Phospholipid Therapy and Mitochondrial Restoration
7. Case Studies
8. Lifestyle/Dietary Recommendations

Optimizing Fertility:
The Impact of Mitochondrial Function on Reproductive Health

1

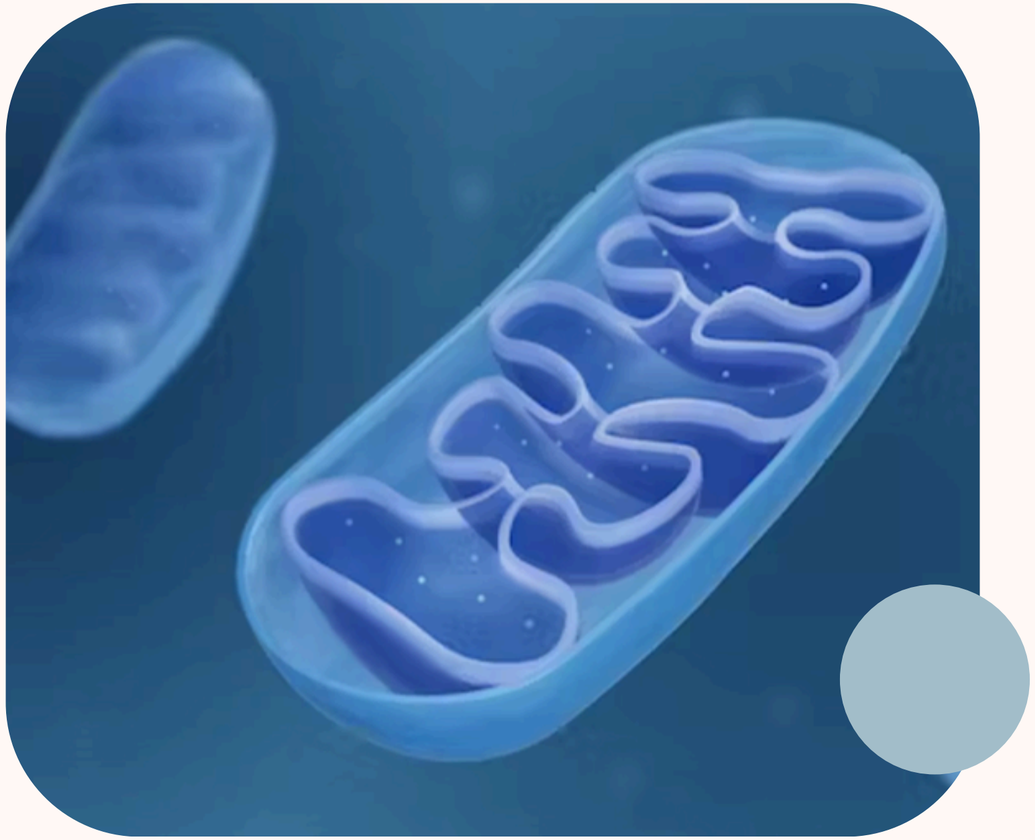
Understanding Infertility

Infertility Rates

Infertility rates suggests that infertility affects an estimated **1 in 4 couples globally**, with certain regions experiencing even higher rates.

In some developed countries, it is estimated that **up to 30% of couples struggle to conceive** within the first year of trying.

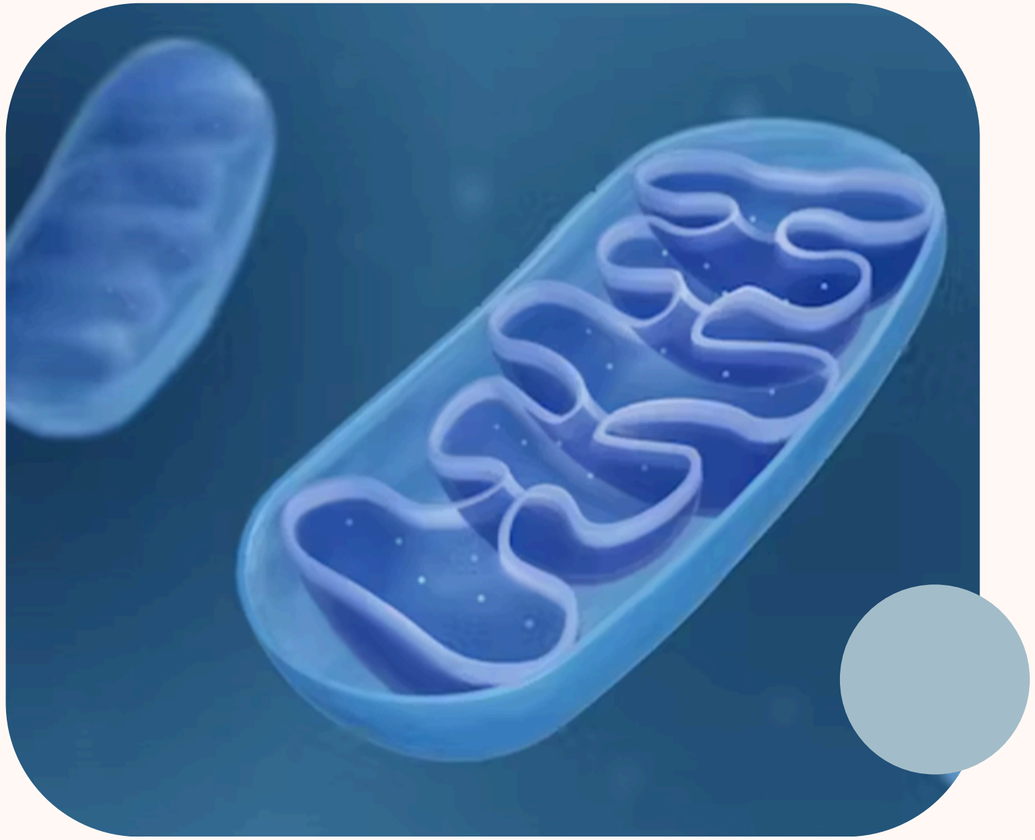
Male fertility has seen a dramatic decline as well, with studies showing that **sperm counts have dropped by 60% over the past 40 years**, a trend that continues to worsen.[8]



What is Infertility?

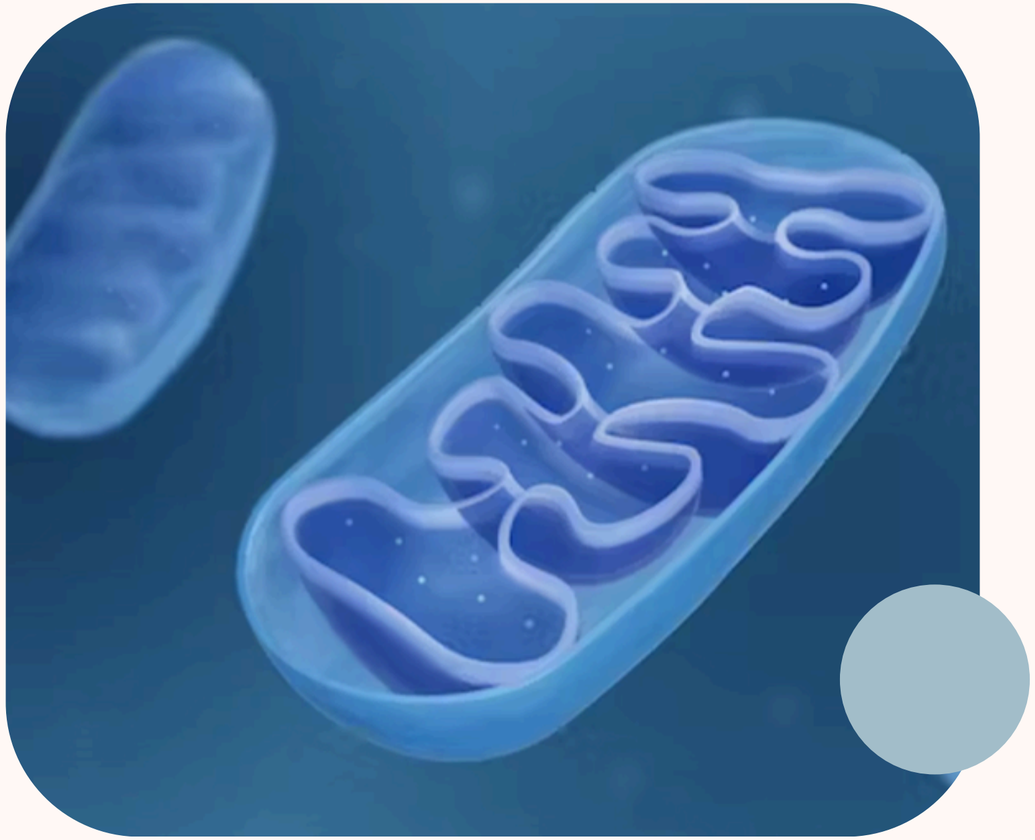
Infertility is traditionally seen as the **inability to conceive after 12 months of regular, unprotected intercourse**, often attributed to hormonal imbalances, structural abnormalities, or ovulatory disorders.

The root of infertility often lies **deeper in mitochondrial dysfunction**, which disrupts the fundamental processes that support reproductive health.



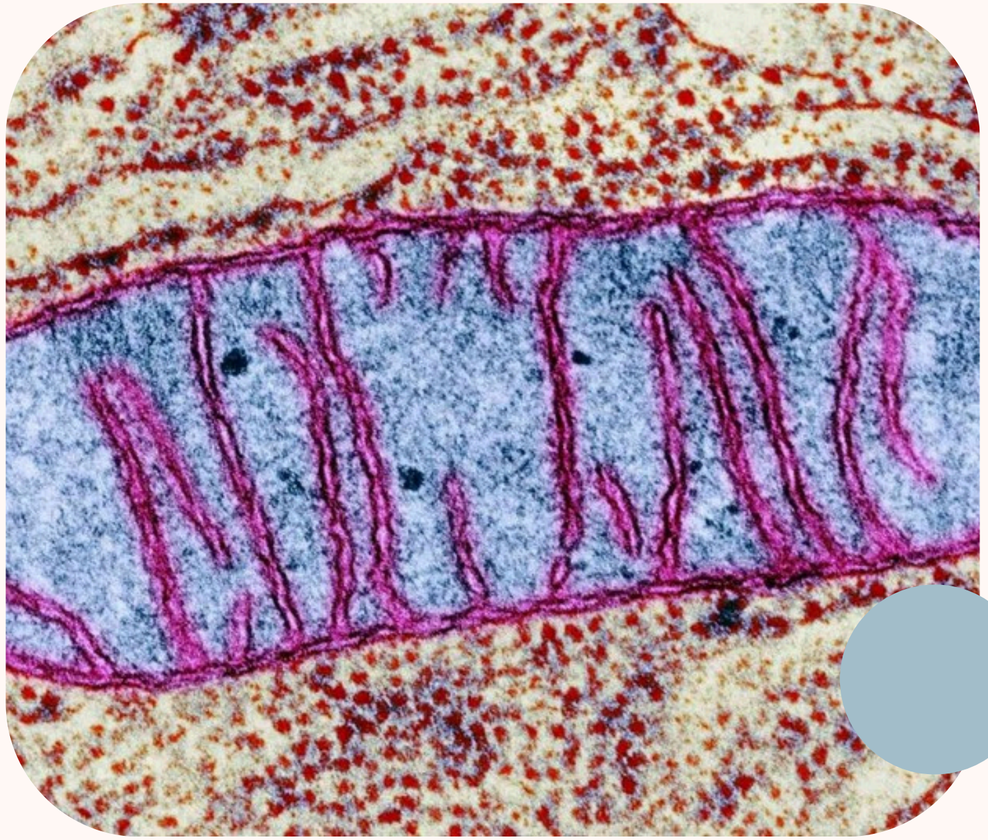
The Fertility–Mitochondria Connection

- **Environmental toxins** like endocrine disruptors, polluted water, and heavy metals damage mitochondria, driving oxidative stress and reducing egg and sperm quality.
- **Modern circadian disruptions** (excess artificial light, insufficient natural light) further impair mitochondrial signaling and hormonal rhythms.
- **Supporting mitochondrial function is essential** for restoring fertility and improving outcomes for healthy conception and pregnancy.[3] [29]



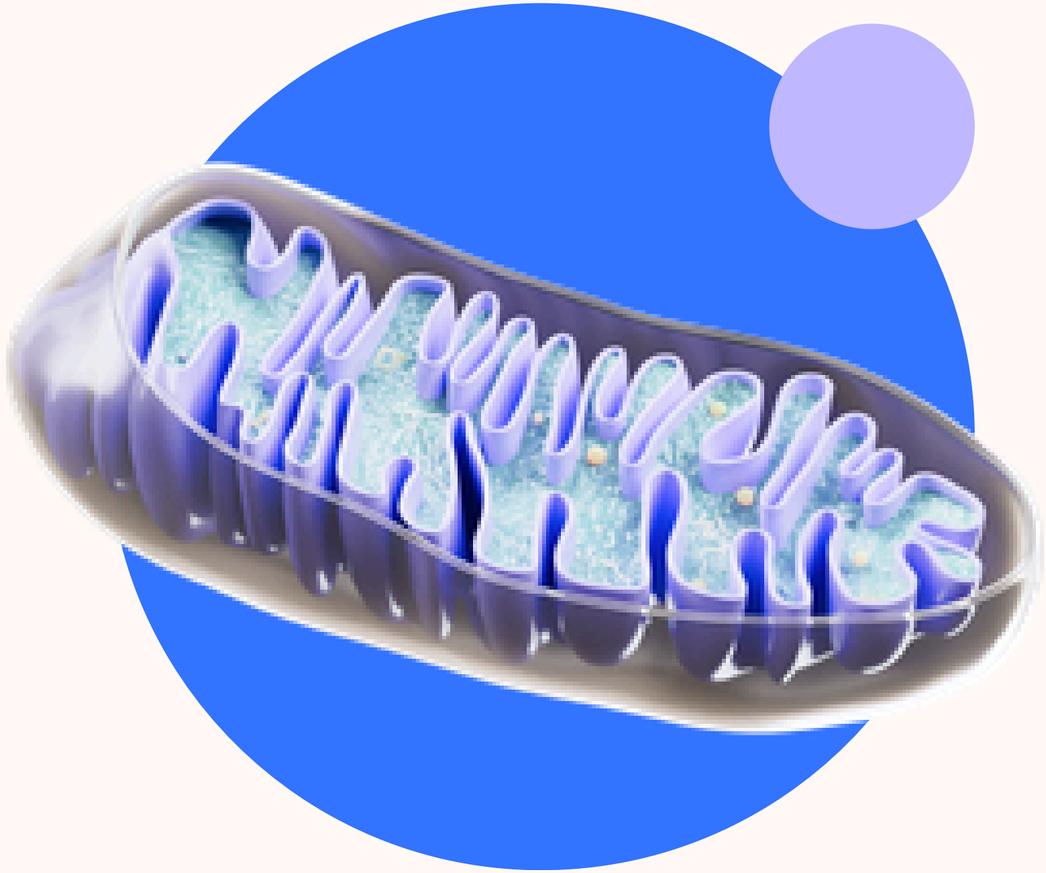
Mitochondrial Dysfunction & Infertility

- Mitochondria directly influence egg and sperm quality, hormonal balance, and embryonic development.
- Dysfunctional mitochondria can lead to inadequate ATP production, increased oxidative stress, and impaired cellular signaling, all of which can contribute to infertility.
- Mitochondrial DNA mutations and issues with mitochondrial biogenesis and mitochondrial dynamics lead to further complications in fertility.
- Supporting mitochondrial function is vital for addressing infertility.[27]



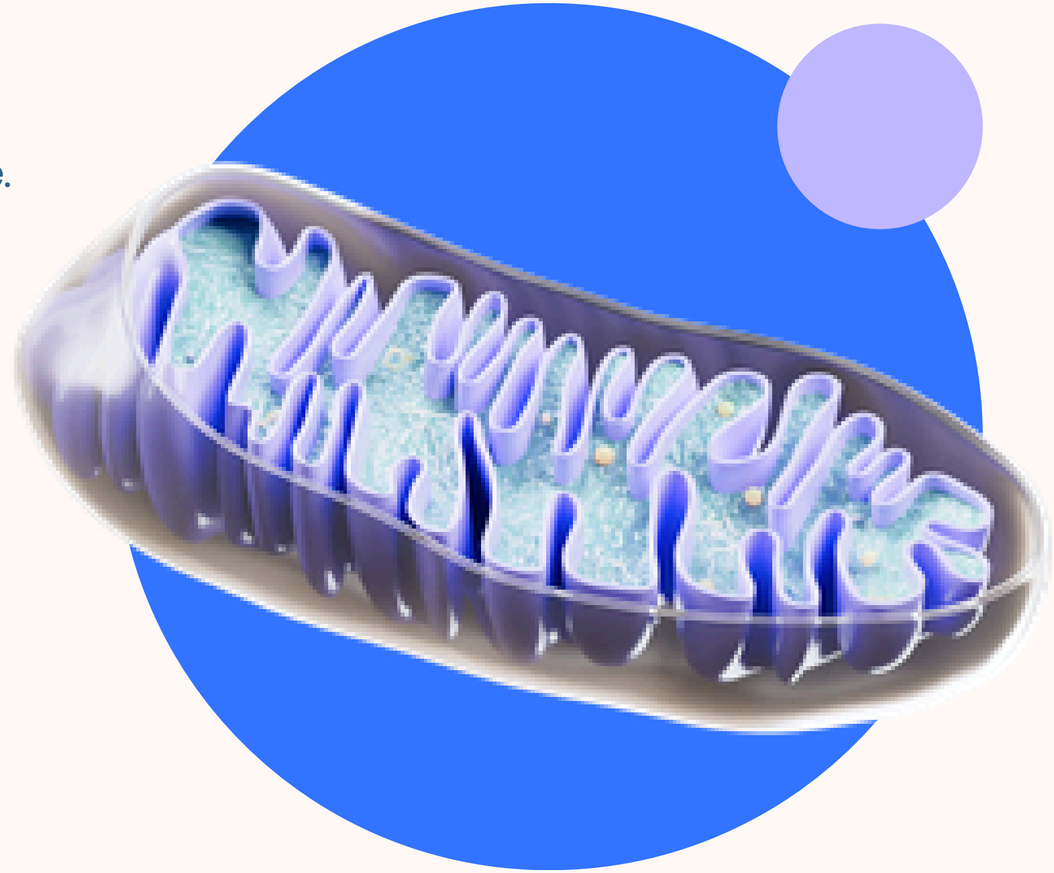
What Are Mitochondria?

- They are organelles
- Come from our Mom
- The “powerhouse of the cell”
- Make ATP
- Also make “byproducts”
  - H20, Co2, Infrared and ROS
  - They are susceptible to damage
  - They can be repaired, replaced and optimized



Core Functions of Mitochondria

- Mitochondria require electrons, oxygen, and red/infrared light to function at their best.
- They produce our master steroid hormone, pregnenolone.
- Central to:
  - Immune function
  - Autophagy
  - Apoptosis
  - Stem cell activity
  - Thyroid regulation
  - Cell signaling
  - Gene expression
- Each cell contains 1,000 to 1 million mitochondria, depending on its energy needs.

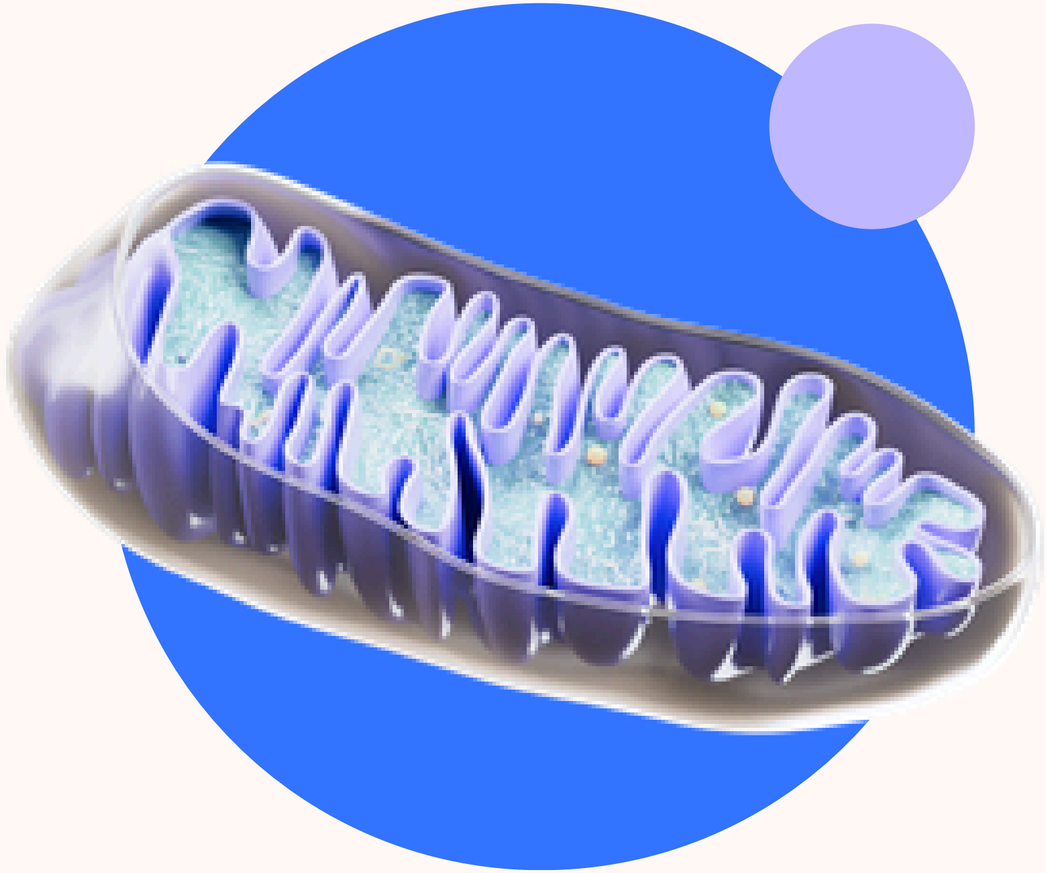


Over 600,000 - 1 Million Mitochondria In Every Egg

This immense concentration of mitochondria reflects the enormous energy demands required for fertilization, early embryonic development, and cellular division.

Mitochondria in the ovum are not only responsible for ATP production but also play a crucial role in regulating calcium signaling, redox balance, and apoptosis all critical processes for a successful conception and embryo viability.

The health and functionality of these mitochondria directly impact the quality of the egg, influencing fertility outcomes, embryonic development, and the long-term health of offspring.



Optimizing Fertility:
The Impact of Mitochondrial Function on Reproductive Health

2

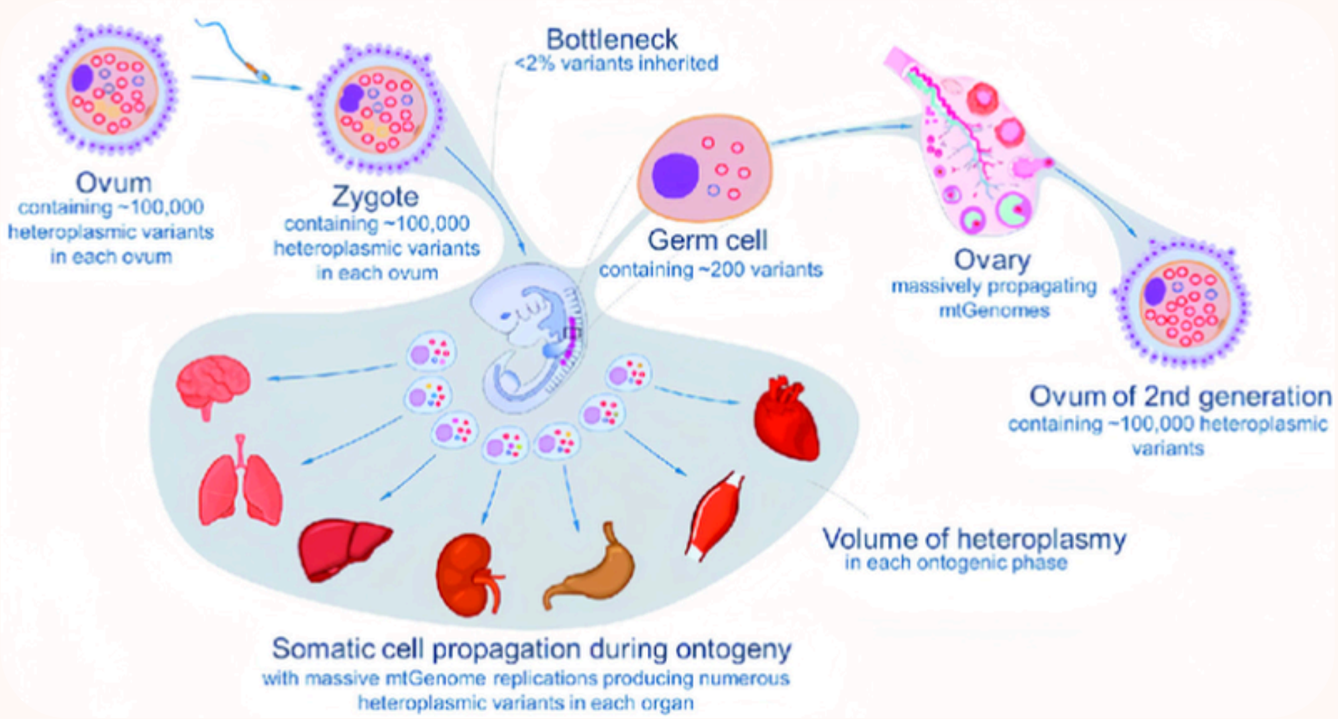
Understanding Mitochondrial Heteroplasmy

What is Mitochondrial Heteroplasmy?

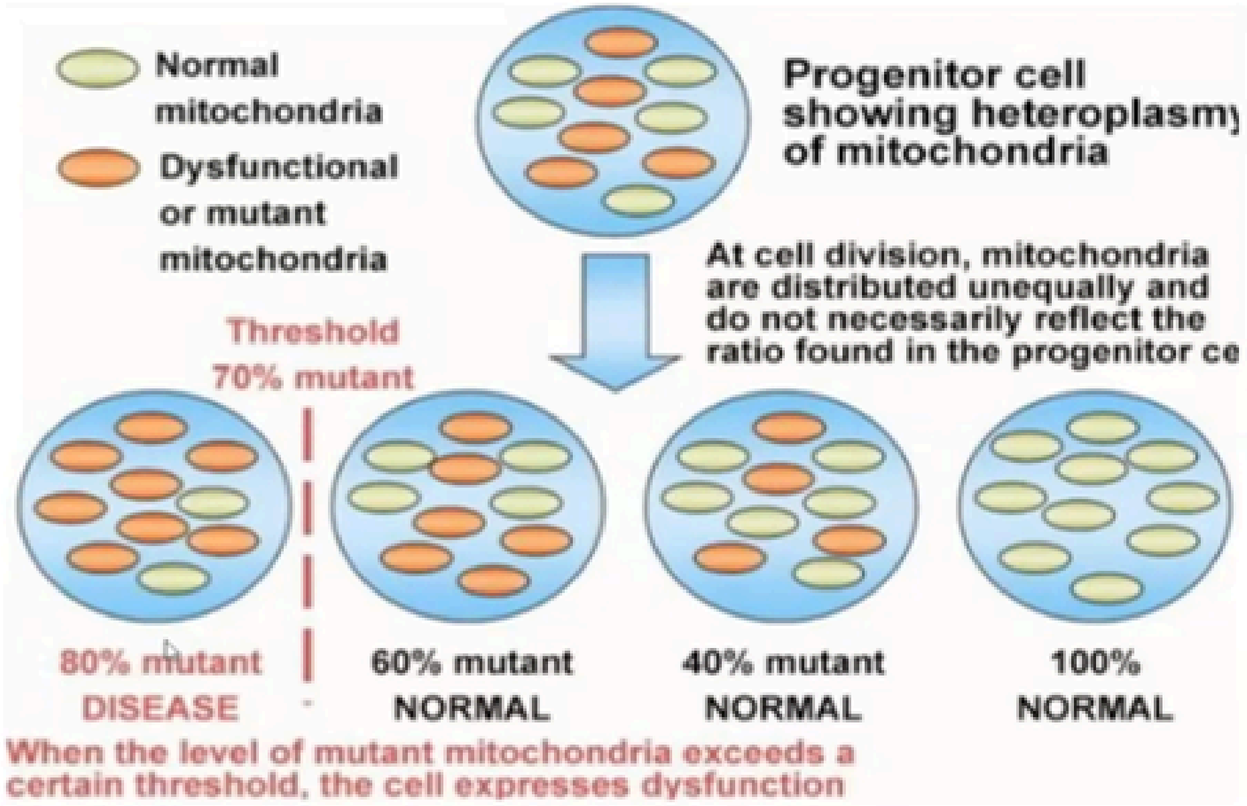
Mitochondrial heteroplasmy refers to the **coexistence of both mutant and healthy mitochondrial DNA (mtDNA)** within the same cell.

The ovary is highly dependent on well-functioning mitochondria.

Variations in mtDNA (heteroplasmy) can lead to **various serious ovarian disorders** affecting the overall health and function of the ovaries, including infertility.[2] [6] [7]



Dr. Doug Wallace's Key Contributions To Mitochondrial Research



Dr. Doug Wallace

Mitochondrial DNA genetics and the heteroplasmy conundrum in evolution and disease

Douglas C Wallace [1], Dimitra Chalkia

Affiliations + expand

PMID: 24186072 PMCID: PMC3809581 DOI: 10.1101/cshperspect.a021220

Abstract

The unorthodox genetics of the mtDNA is providing new perspectives on the etiology of the common "complex" diseases. The maternally inherited mtDNA codes for essential energy genes, is present in thousands of copies per cell, and has a very high mutation rate. New mtDNA mutations arise among thousands of other mtDNAs. The mechanisms by which these "heteroplasmic" mtDNA mutations come to predominate in the female germline and somatic tissues is poorly understood, but essential for understanding the clinical variability of a range of diseases. Maternal inheritance and heteroplasmy also pose major challengers for the diagnosis and prevention of mtDNA disease.

Mitochondria are the Foundation of Female Fertility

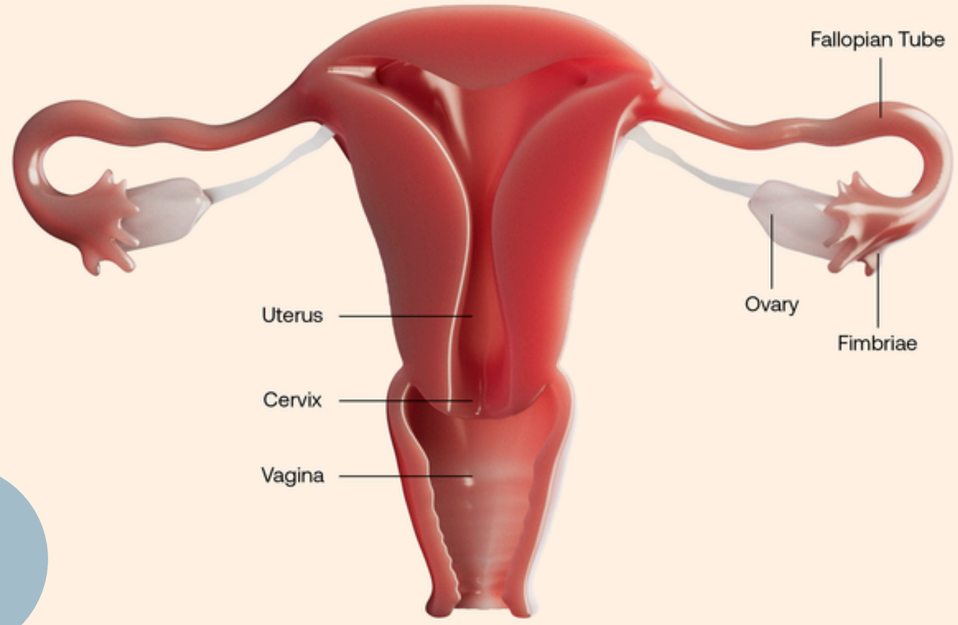
1. The Ovary

Ovarian follicles are among the most mitochondria-dense cells in the human body.

An egg's ability to mature, ovulate, and form a viable embryo depends on:

- high mitochondrial membrane potential
- intact mitochondrial DNA
- sufficient ATP for meiotic division
- stable redox balance

Low mitochondrial output → poor egg quality, poor maturation, chromosomal errors.



Mitochondria are the Foundation of Female Fertility

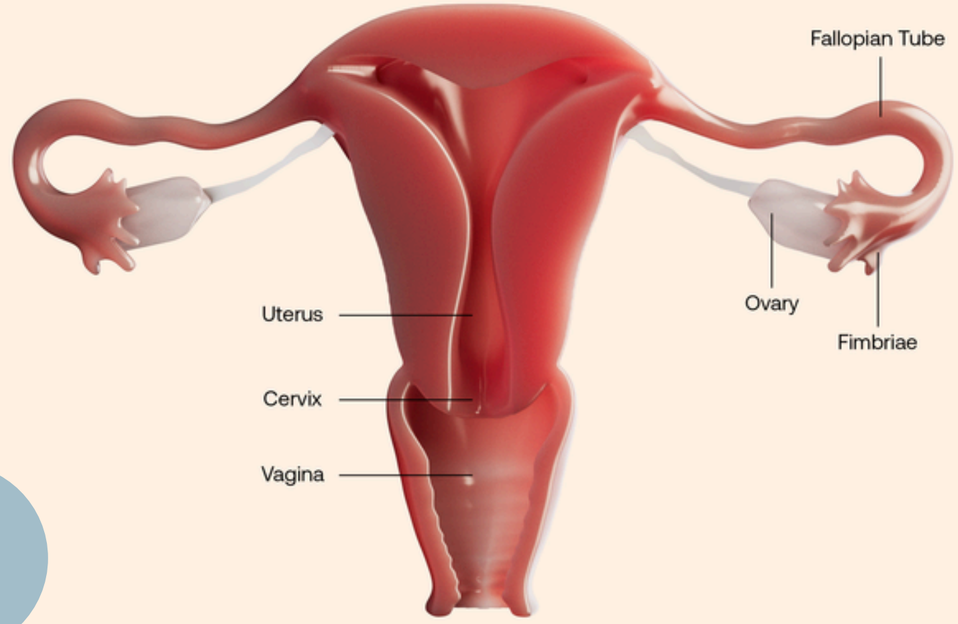
2. The Fallopian Tubes

Fertilization occurs in the tube. The cilia that move the egg toward the uterus require continuous ATP.

If mitochondrial function is impaired:

- ciliary movement slows
- transport becomes dysregulated
- inflammation rises
- fertilization is less likely

This is not a hormone issue - it's a **cellular energy transport issue**.



Mitochondria are the Foundation of Female Fertility

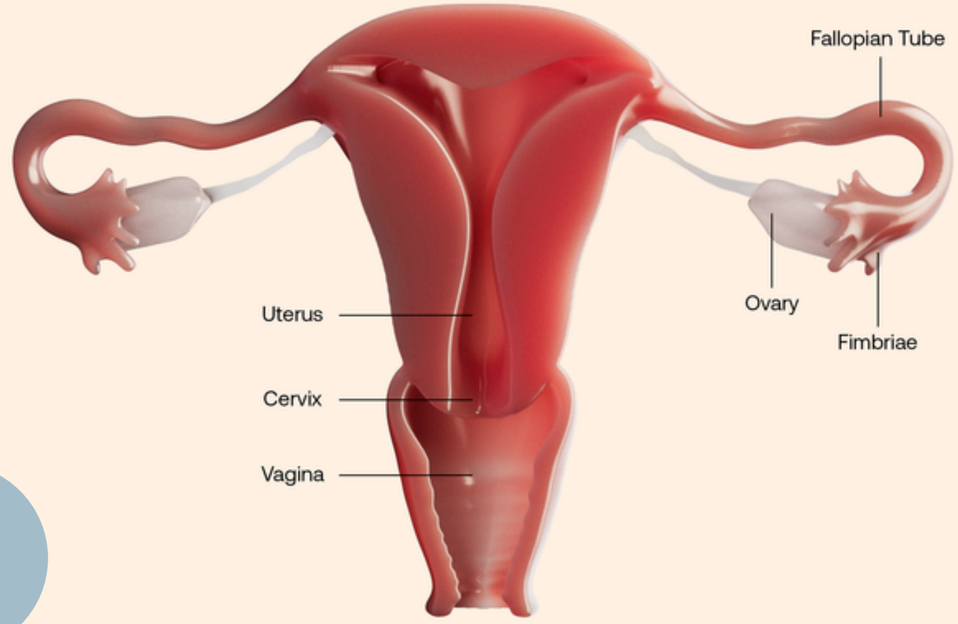
3. The Uterus

Implantation is one of the highest-energy events in reproduction.

The endometrium needs:

- mitochondrial ATP for tissue remodeling
- redox stability for immune tolerance
- membrane integrity for cell signaling
- coherent circadian rhythms to time the implantation window

When mitochondrial charge is low, the endometrium cannot properly prepare for implantation, even when hormones appear “normal.”

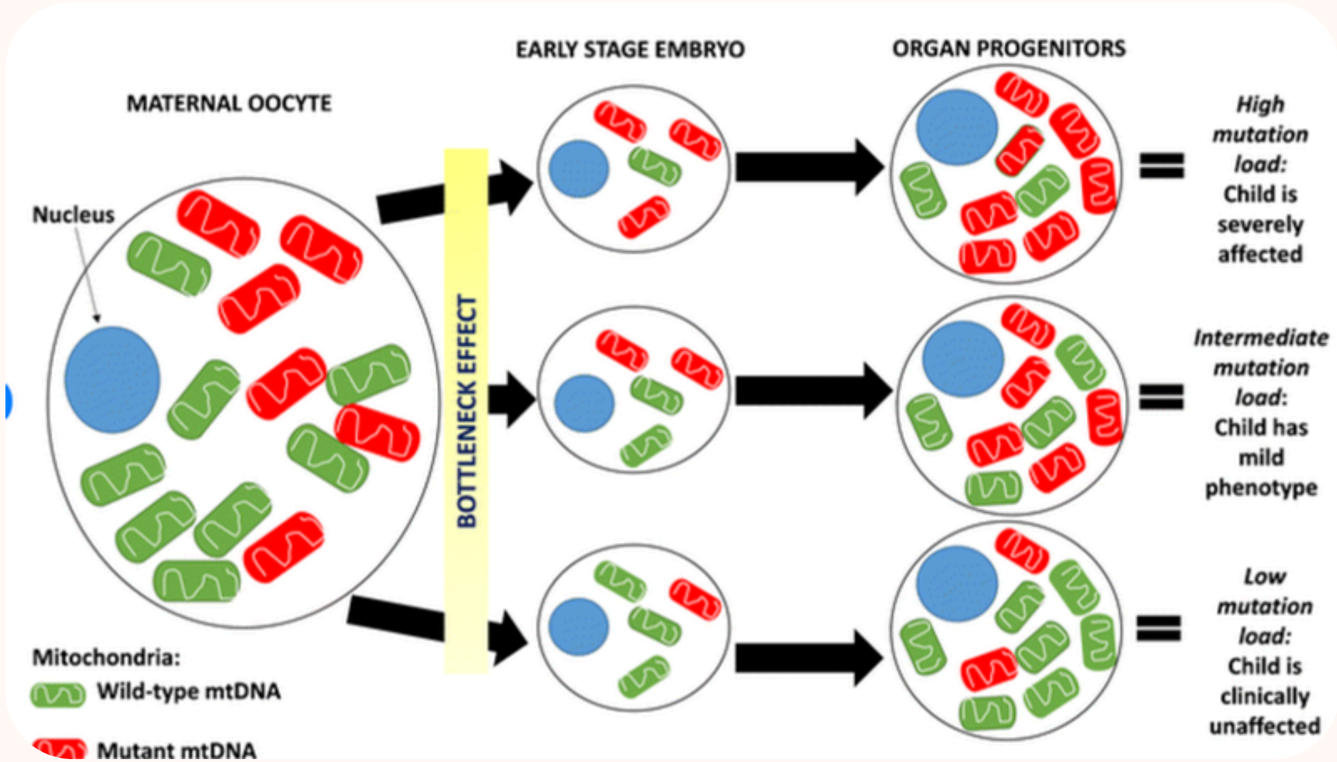


Heteroplasmy In Offspring

When a mother passes on her mitochondrial DNA (mtDNA) to her children, it's a mix of healthy and mutant DNA.

Each egg has over 100,000 mtDNA copies, but early in embryo development, this number drops dramatically to just a few thousand.

This process, known as the "**bottleneck**," can lead to significant variations in the ratio of healthy to mutant mtDNA (heteroplasmy) in different cells of the embryo compared to the mother. (10)

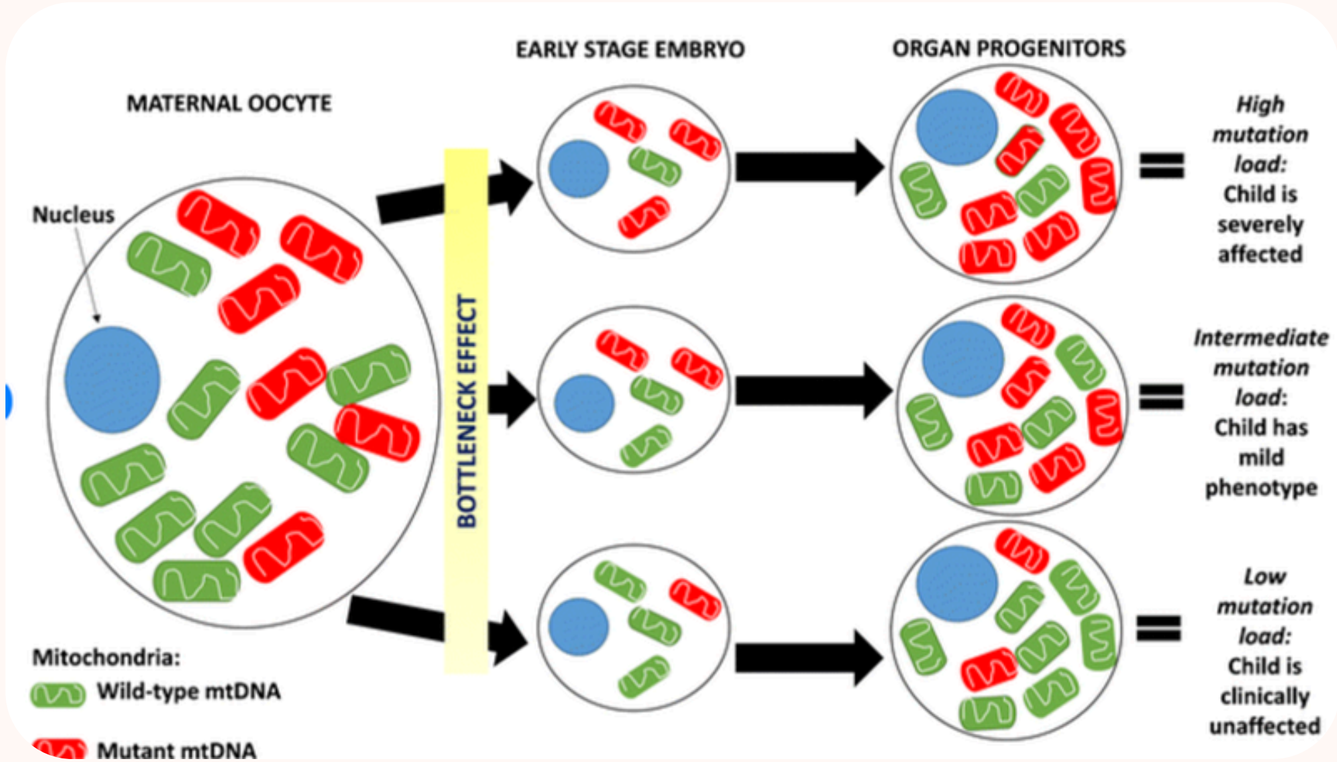


Heteroplasmy In Offspring

As the fetus grows, this ratio can vary widely between different organs.

The **higher the percentage of mutant mtDNA** in an organ, the **more likely the child will develop severe mitochondrial diseases** early in life.

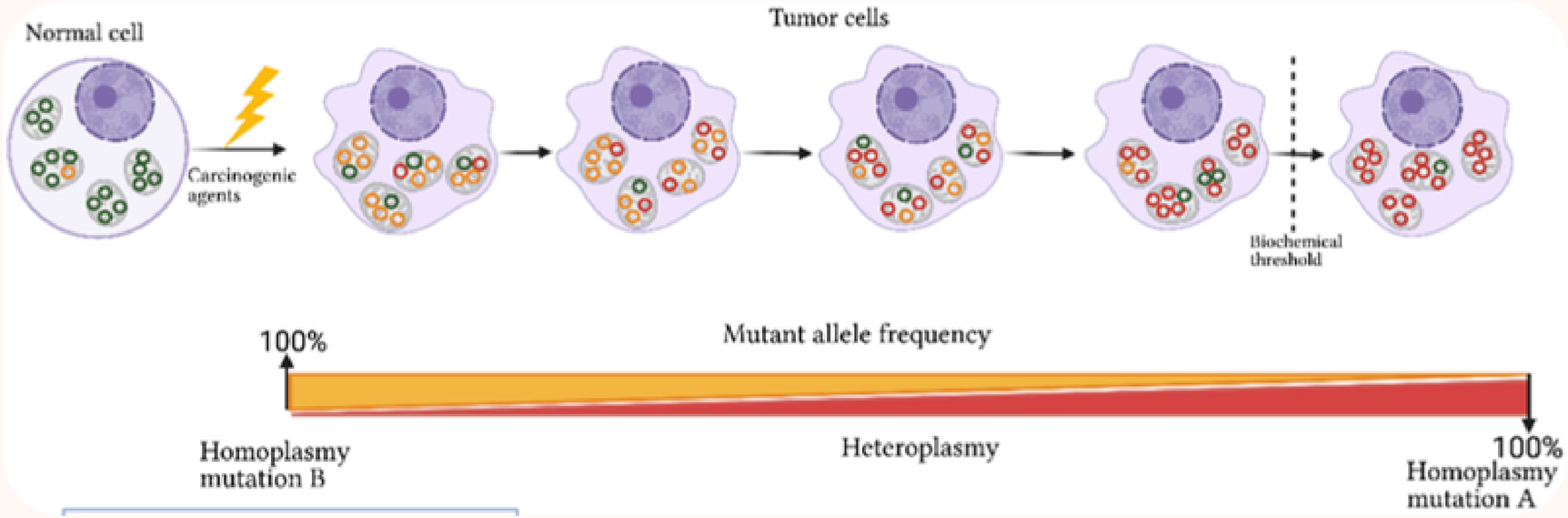
This variability explains the wide range of symptoms seen in mitochondrial diseases and the unpredictable risk of recurrence in future children of mothers with these mutations.[26]



Impact of Environmental Stressors

Mitochondrial **heteroplasmy levels** are a major factor in dysfunction, but **environmental stressors**, oxidative damage, and metabolic imbalances also play significant roles in worsening mitochondrial performance, which also affects fertility.

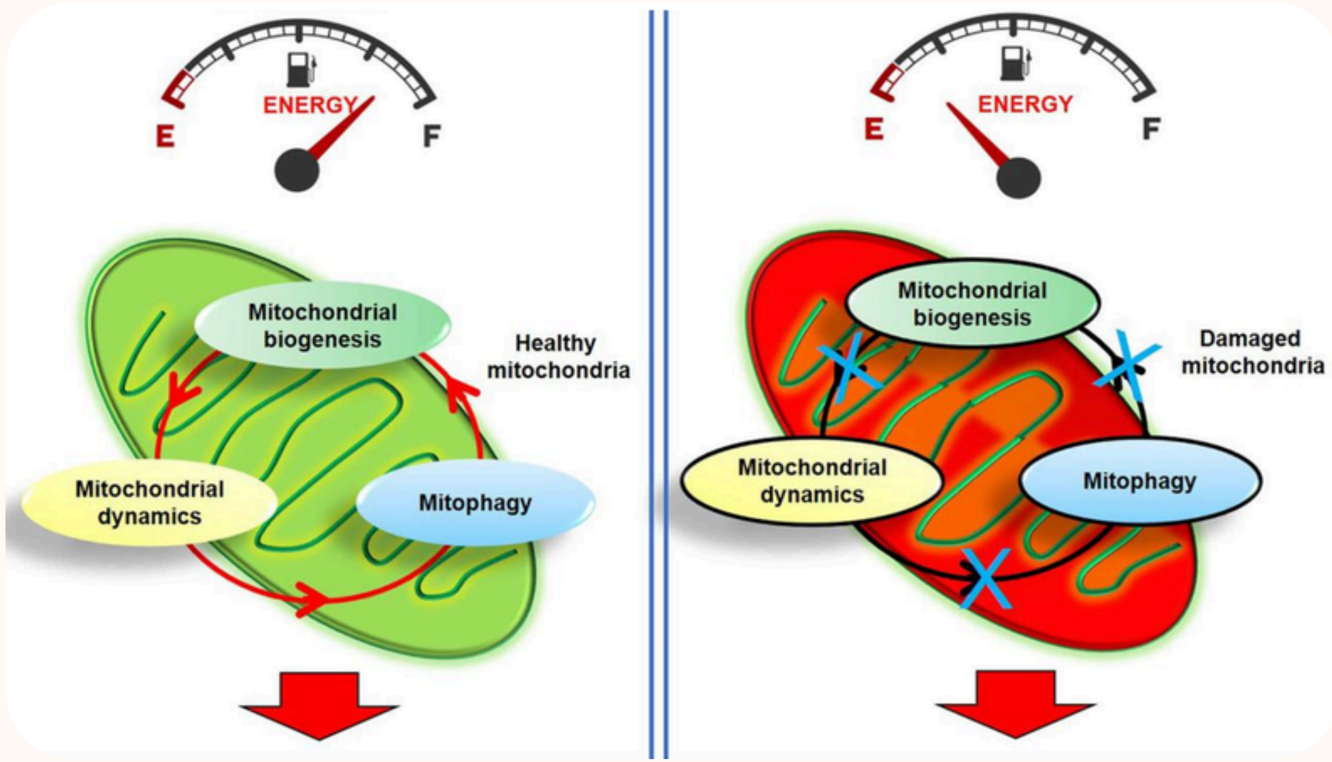
Environmental factors alone can increase oxidative stress and lead to higher heteroplasmy and exacerbate dysfunction in cells already burdened by mutant mtDNA.



How Optimal Mitochondria Function

Energy Production:
Optimal mitochondria produce sufficient ATP, supporting the energy-intensive processes of egg maturation, fertilization, and early embryonic development.

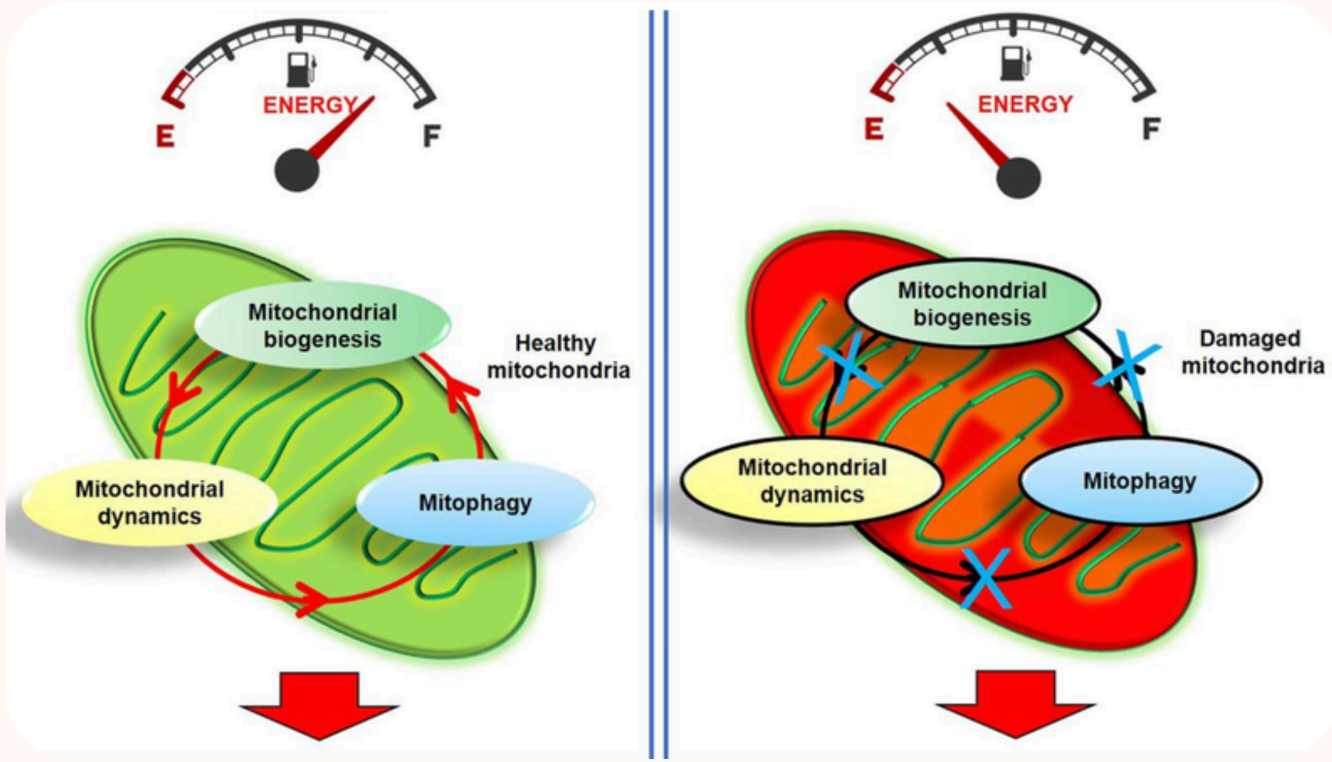
Oxidative Stress Management:
Properly functioning mitochondria manage ROS levels effectively, minimizing oxidative stress and protecting cells from damage.[29]



How Optimal Mitochondria Function

Genetic Integrity:
Lower mutation rates in mtDNA ensure better genetic integrity and reduce the risk of transmitting mitochondrial diseases.

Cellular Function:
Healthy mitochondria support overall cellular function, enhancing the quality of gametes and supporting successful fertilization and embryo development.[29]



Dysfunctional Mitochondria

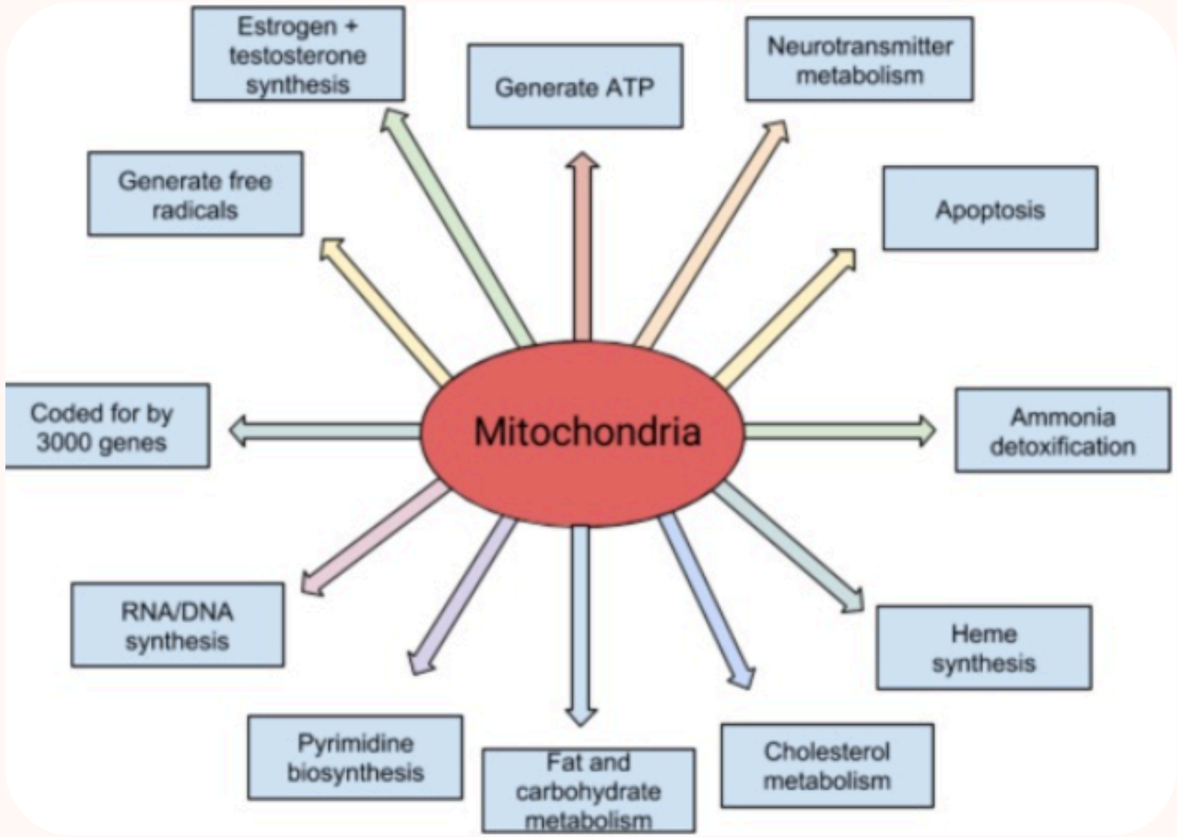
Reduced ATP Generation: Mitochondria produce less ATP, lowering energy availability for egg and sperm quality.

Reduced Water Production: Less metabolic water is produced, affecting cellular hydration and optimal function for reproductive cells.

Reduced CO2 Production: Decreased CO2 disrupts pH balance and oxygen delivery, impacting egg and sperm health.

Reduced Proton Gradient: A weaker proton gradient means less efficient ATP production, leading to energy deficits in cells.

Reduced Redox Potential: Impaired redox balance increases oxidative stress, harming the cellular environment for reproduction.[29]

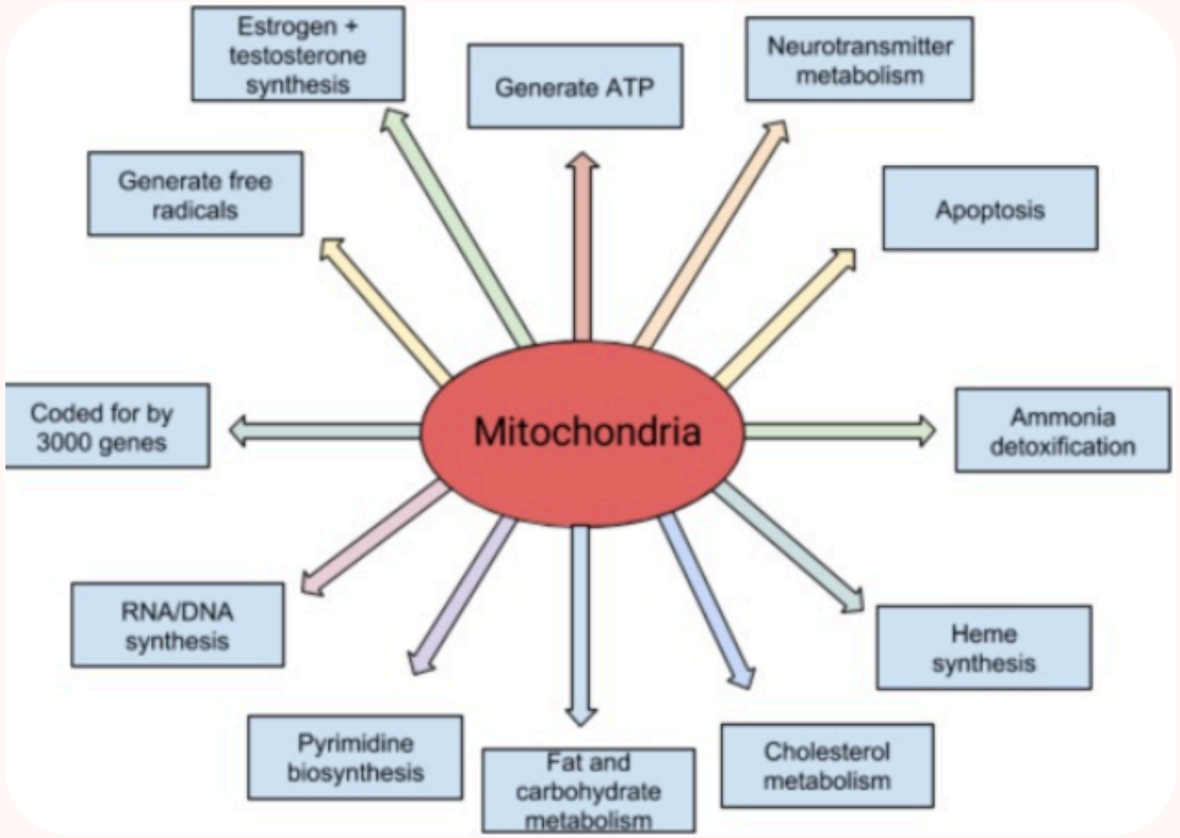


Dysfunctional Mitochondria

Increased ROS: Dysfunctional mitochondria produce higher levels of reactive oxygen species (ROS), leading to oxidative stress and cellular damage.

Mutated mtDNA: Higher rates of mutations in mitochondrial DNA (mtDNA) can impair cellular function and increase the risk of transmitting mitochondrial diseases.

Poor Cellular Health: Overall cellular function declines, affecting the quality of gametes (eggs and sperm) and the early stages of embryonic development.[29]



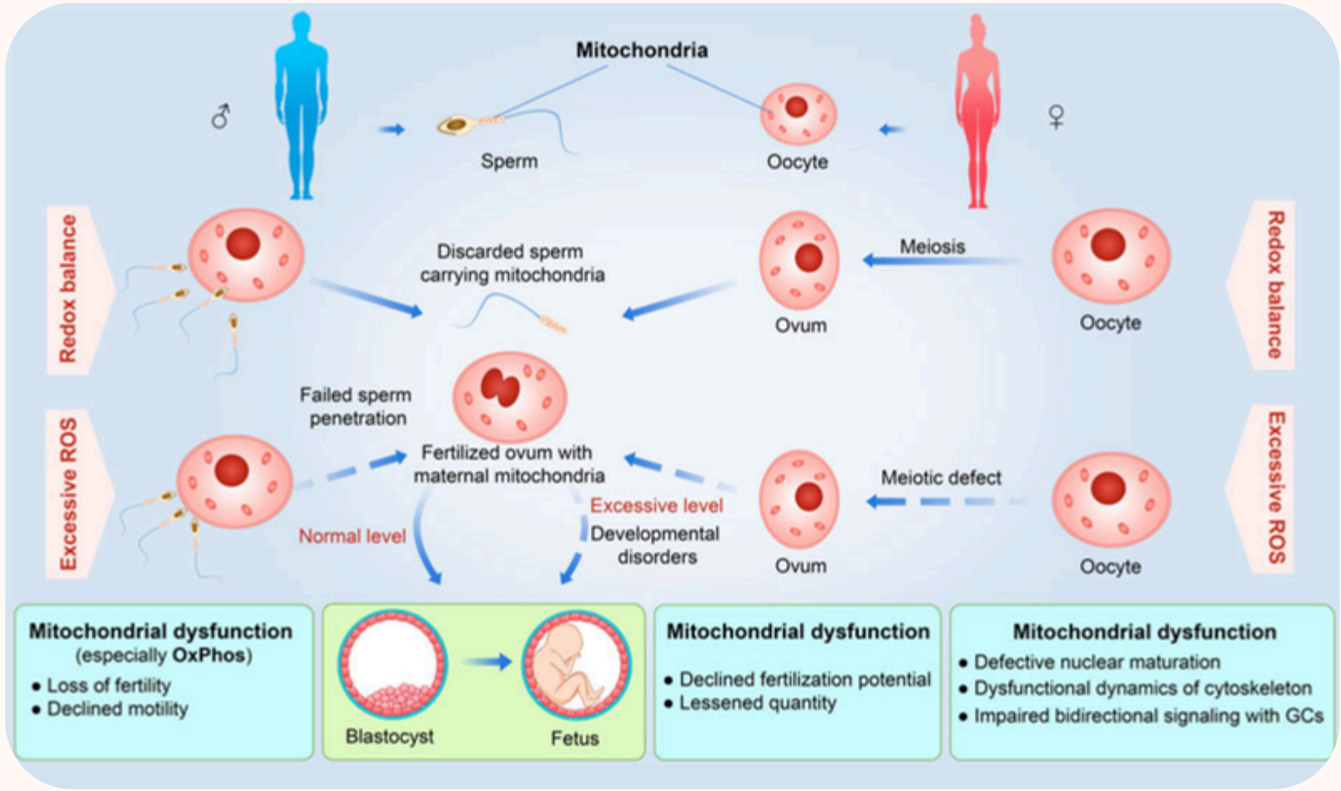
ROS, Mitochondrial Dysfunction & Their Effects on Female Fertility

Detrimental effects of excessive ROS and mitochondrial dysfunction on different reproduction stages from gamete formation to embryonic development in the uterus.

For the female, **excessive ROS and mitochondrial dysfunction**:

- Compromise the entire oogenesis process
- Increase the possibility of lessened quantity of ovum
- Reduce fertilization potential

Otherwise, excessive ROS can **disturb the fetal development and even contribute to developmental disorders**.[15]



ROS, Mitochondrial Dysfunction & Their Effects on Female Fertility

Just as in female fertility, mitochondrial dysfunction and elevated ROS in men have wide-ranging impacts on reproductive potential.

Excessive ROS and mitochondrial dysfunction in males:

- Alters epididymis function
- Disrupts sperm mitochondrial activity
- Leads to structural and cellular defects in sperm at the membrane level
- Compromises genetic stability and damage DNA
- Disrupts intracellular calcium homeostasis
- Impairs OXPHOS, reducing the mitochondria's ability to produce energy
- Triggers apoptosis, programmed cell death [16]

Optimizing Fertility:
The Impact of Mitochondrial Function on Reproductive Health

3

Redox & Fertility

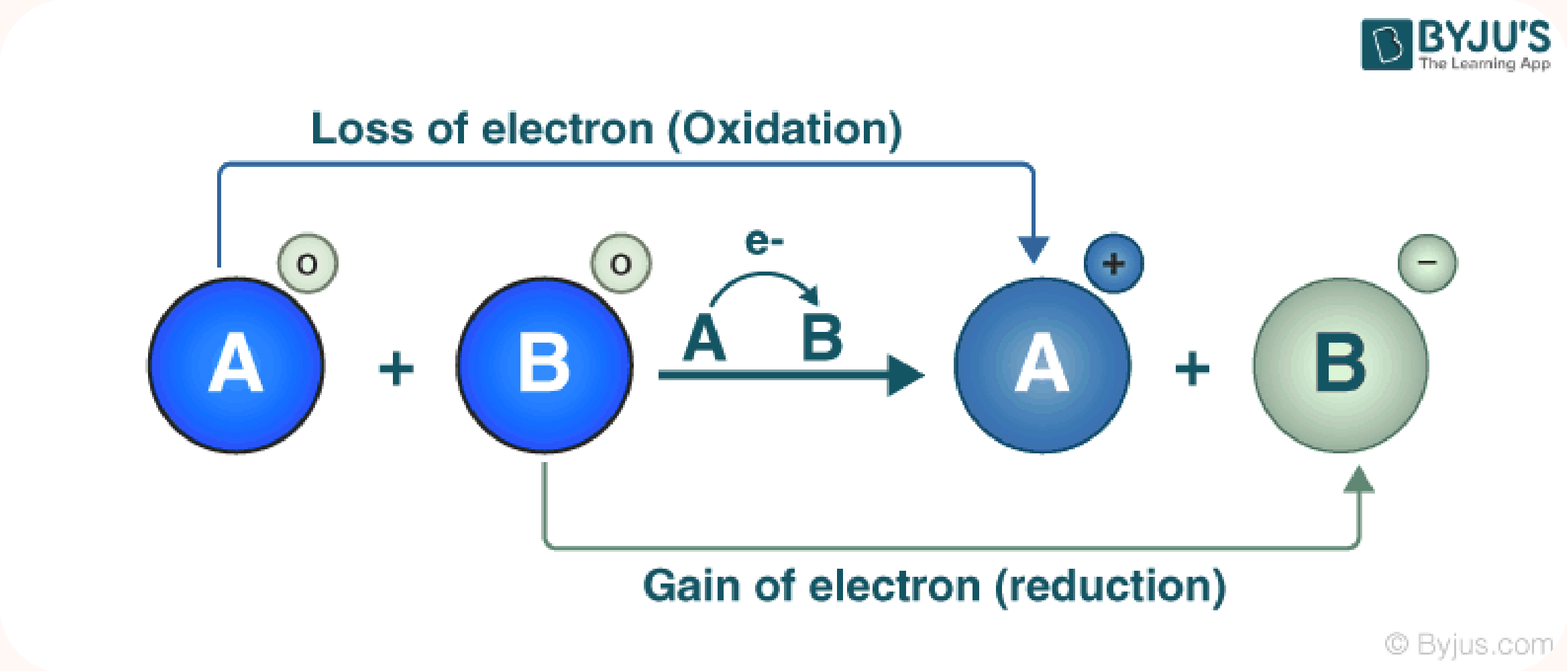
What is Redox?

Redox reactions involve two processes: oxidation and reduction.

- **Oxidation:** The loss of electrons by a molecule, atom, or ion, leading to an increased positive charge (or a decrease in negative charge).
  - **Example:** When a molecule like glucose loses electrons during cellular respiration,
  - it becomes oxidized.
- **Reduction:** The gain of electrons by a molecule, atom, or ion, resulting in a decreased positive charge (or an increase in negative charge).
  - **Example:** When a molecule like NAD+ gains electrons, it becomes reduced to NADH.

These reactions always occur together, with one molecule becoming oxidized (loses electrons) and another becoming reduced (gains electrons). This coupling is critical for biochemical processes, such as cellular respiration where the transfer of electrons facilitates the release or storage of energy necessary for cellular functions.[11]

What is Redox?



These reactions always occur together, with one molecule becoming oxidized (loses electrons) and another becoming reduced (gains electrons). This coupling is critical for biochemical processes, such as cellular respiration where the **transfer of electrons facilitates the release or storage of energy necessary for cellular functions**.[11]

Redox Potential: How Your Body Generates and Uses Electric Charge

Redox potential refers to the **electrical charge created by pools of electrons (negative) or protons (positive)** and reflects the voltage your body can generate and use. It depends on how efficiently you convert food, sunlight, and the earth's magnetic field into usable electrical energy while minimizing electron loss.

Redox is the energy behind health and resiliency, healing capacity, movement of materials, chemical reactions, and LIFE itself.

When your redox is optimized, your biology is full of LIFE.

When your redox is poor, your body lacks the energy to maintain itself. [17]

Measuring Redox

- **Oxidative stress markers:** Levels of reactive oxygen species (ROS)
  - Antioxidant enzyme activity (e.g., superoxide dismutase, glutathione peroxidase)
  - Biomarkers like malondialdehyde (MDA) or 8-oxo-2'-deoxyguanosine (8-oxo-dG)
- pH
- Electrical Charge (membrane potential)

Measuring Redox

- Phospholipid concentrations
- Digestion/GI health
- Immune system function
- Wound healing
- Hair, skin, nail quality
- Sleep quality (dreaming)
- Libido
- Cataracts
- BUN/Creatinine ratio *(When it is high, it means your proteins and mitochondria do not have reverse water micelles around them to transfer energy to and fro to allow for energy production)*
- Intracellular hydration
- Low LDL
- Brain fog
- Neuropathy
- Cardiovascular health
- Hormonal imbalances
- Degenerative disease

4

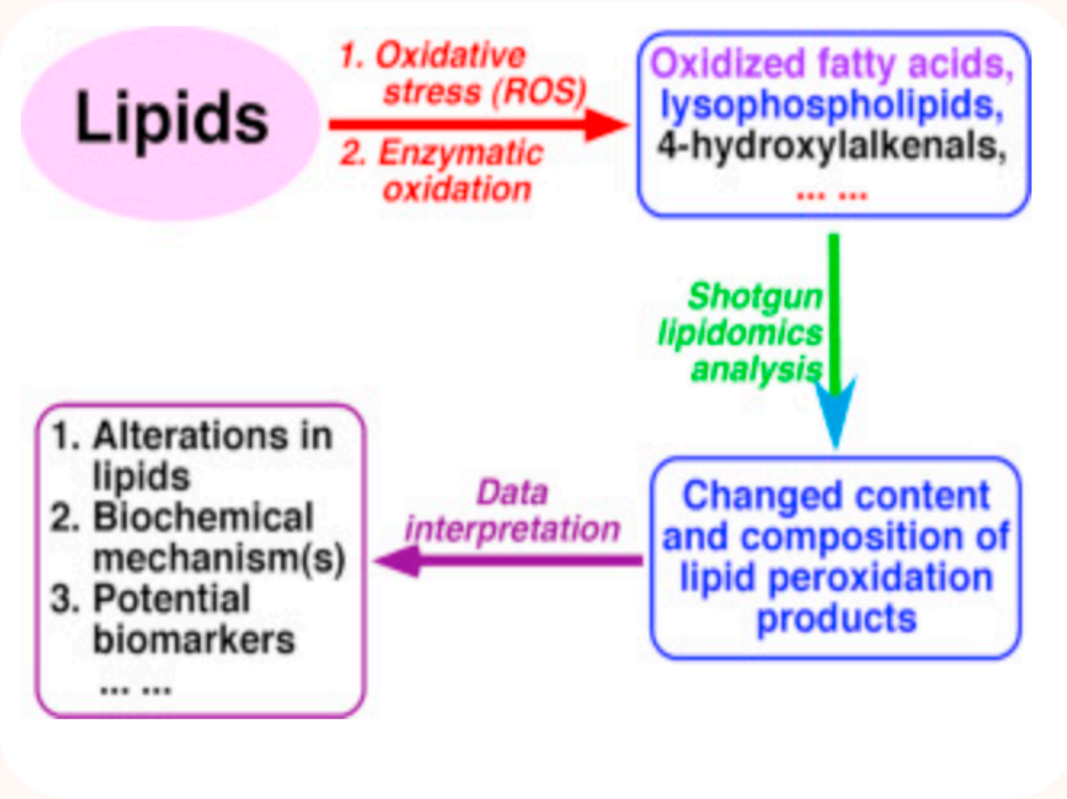
The Impact Of Phospholipids and Fatty Acids on Mitochondria For Reproductive Health

Lipidomics: Lipids & Redox Support

Redox lipidomics examines how lipids influence redox processes in biological systems.

Lipidomics involves the comprehensive analysis of lipids in cells, specifically in the mitochondria.

Redox lipidomics goes further by studying how lipids are modified during oxidative and antioxidative reactions and the impact of these lipid modifications on cellular health and signaling.



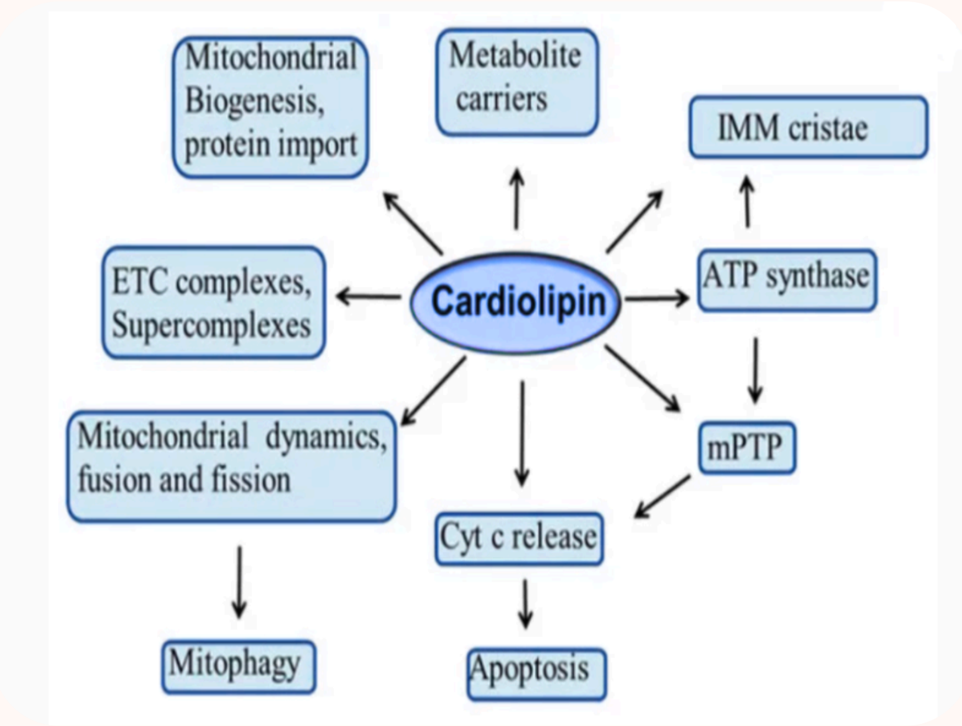
Cardiolipin and Redox

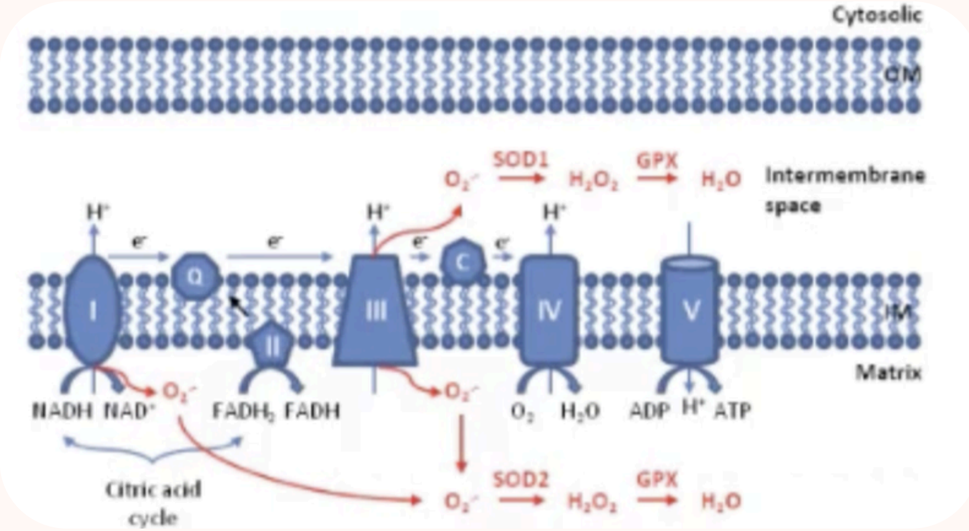
Cardiolipin is crucial for redox balance.
It interacts with key proteins in electron transport.

Cardiolipin **facilitates electron transport** and efficient coupling of electron transport to ATP synthesis.

Phospholipids also play a critical role in **antioxidant defense**.

In particular, **phosphatidylcholine (PC) and phosphatidylethanolamine (PE)** support the body's endogenous antioxidant systems and help maintain overall cellular integrity. Through their functions in membrane structure, signaling, and ROS regulation, PC and PE are essential contributors to sustaining the cell's redox potential.* [22]





Optimizing Fertility:
The Impact of Mitochondrial Function on Reproductive Health

5

The Role of Light In Regulating our Biology

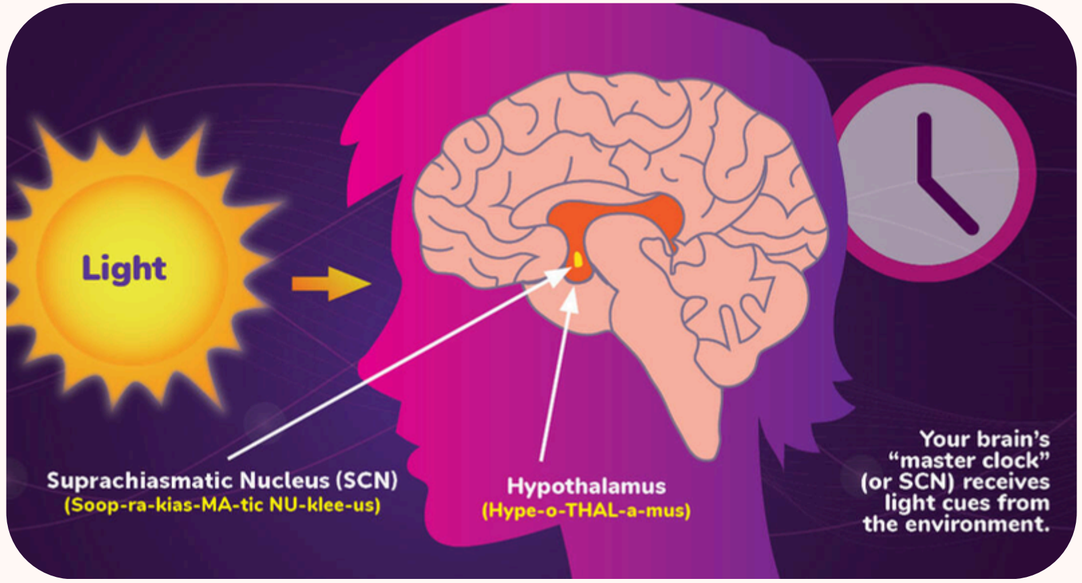
Circadian Biology & The Fertility Link

Circadian rhythms, governed by the master clock (SCN), regulate hormone release; chronobiology shows that light is the strongest cue shaping this clock.

Every cell contains clock genes that influence metabolism, hormonal balance, and overall physiological timing.

When circadian rhythms are disrupted—often through irregular light exposure—hormonal signaling, ovulation, and menstrual cycles become imbalanced.

These disruptions also impair mitochondrial efficiency, reducing oocyte quality and undermining reproductive health.[10]



National Institute of General Medical Sciences. (n.d.). *Circadian rhythms and the SCN* [Infographic]. NIH.

Light Biology Interactions – Melatonin as a Biosensor

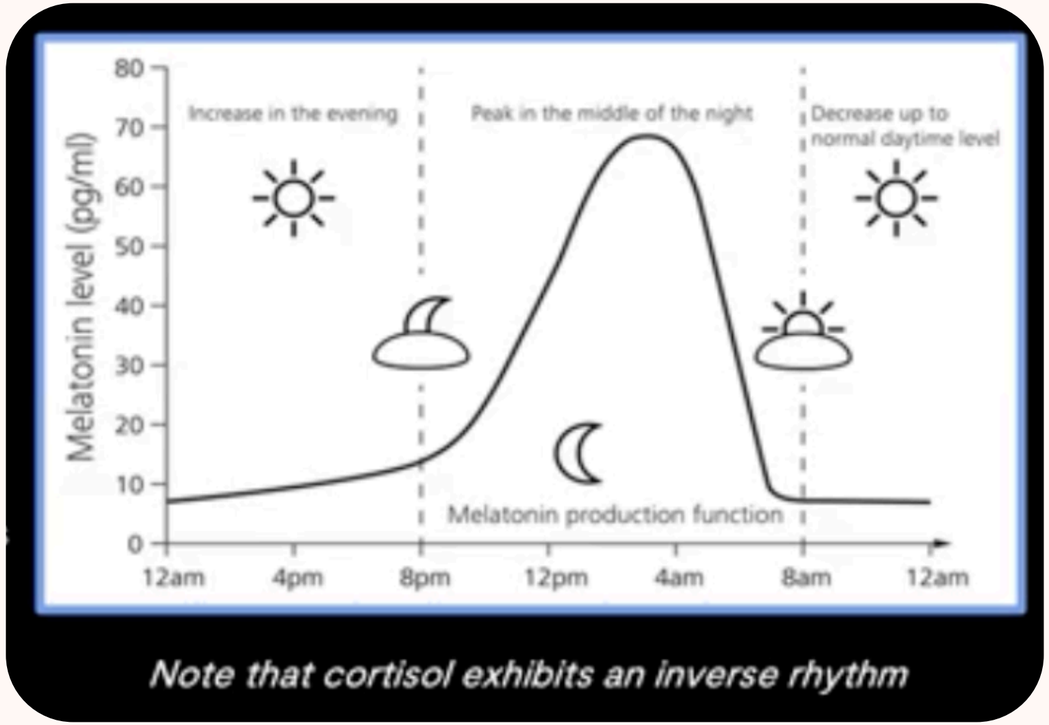
Melatonin is the primary hormone of the pineal gland that induces and maintains sleep.

It enters the bloodstream to synchronize biological clocks throughout the body.

Also **synthesized within mitochondria** in many tissues, where it acts as a potent antioxidant and ROS scavenger (this form stays inside cells).

Blue, green, and bright light inhibit pineal melatonin release; darkness at night triggers its production.

Bright daytime sunlight is essential for building the melatonin needed later for quality sleep.



Note that cortisol exhibits an inverse rhythm

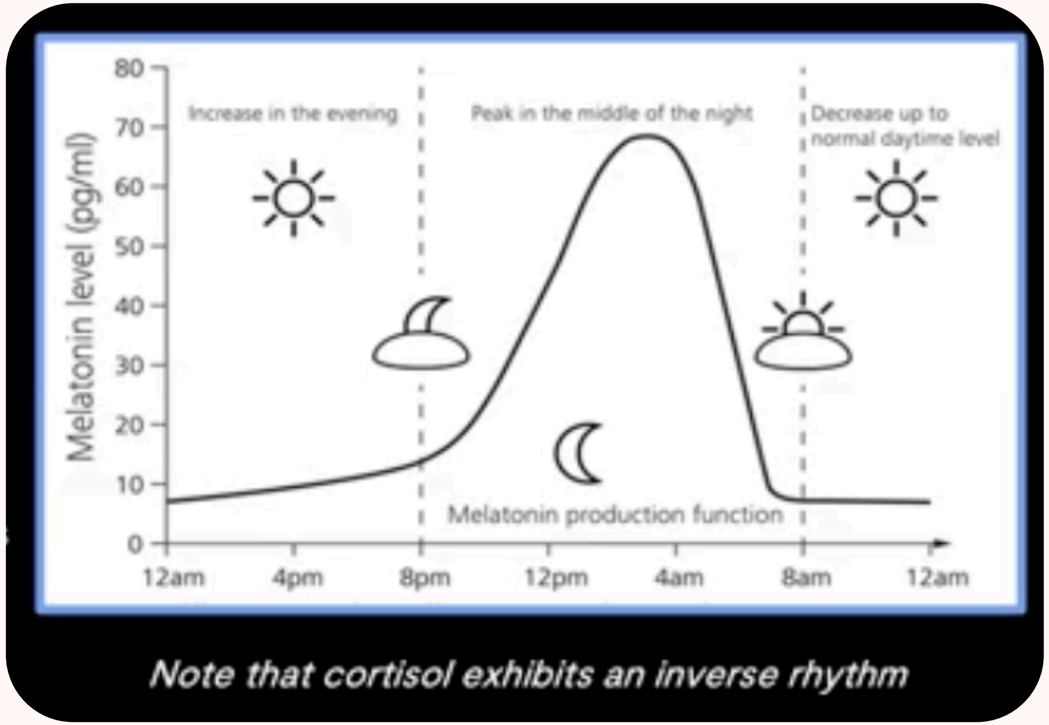
Light Biology Interactions – Cortisol as a Biosensor

Cortisol is a key biosensor of circadian health and a direct influencer of fertility.

It follows a **daily rhythm** — peaking in the morning for alertness and steadily declining to support rest and recovery.

This pattern regulates metabolism, immune function, hormone balance, and mitochondrial activity.

Circadian disruption disturbs cortisol rhythms, impairing reproductive hormones, menstrual cycles, and egg and sperm quality.



Note that cortisol exhibits an inverse rhythm

The HPG Axis and Clock Genes

- **HPG Axis Regulation:** The circadian clock synchronizes the hypothalamic-pituitary-gonadal (HPG) axis. The SCN activates GnRH neurons, driving LH and FSH release for normal reproductive function.

- **Clock Genes in the Ovaries:** Ovarian clock genes regulate hormone production in granulosa and theca cells, shaping estrogen, progesterone, and androgen synthesis essential for oocyte maturation.

- **POMC & α-MSH Pathway:** Leptin activates proopiomelanocortin (POMC) neurons to produce alpha-melanocyte-stimulating hormone (α-MSH), which modulates GnRH and ultimately influences FSH and LH rhythms.

- **Impact of Disruption:** When circadian biology is disrupted, ovarian function, hormone balance, and fertility all decline.

The Leptin-Fertility Connection

Leptin, produced by adipose tissue, is a key energy-sensing hormone that influences metabolism, hormones, and fertility.

It activates POMC neurons, leading to α-MSH production, which modulates GnRH and drives healthy LH/FSH rhythms in the HPG axis.

This pathway links energy status directly to reproductive function - fertility is supported only when energy availability is adequate.

Both **low leptin (low body fat/energy deficiency) and high leptin (obesity/leptin resistance) disrupt this signaling**, impairing ovulation, sperm quality, and overall hormonal balance.

280 nm UVB light supports leptin regulation by influencing the hypothalamus, enhancing leptin receptor expression, and helping align circadian rhythms to improve leptin sensitivity. Optimized leptin signaling via UVB exposure supports healthier energy balance, metabolic function, and fertility.

Balanced leptin levels are essential for optimal fertility. (19, 20)

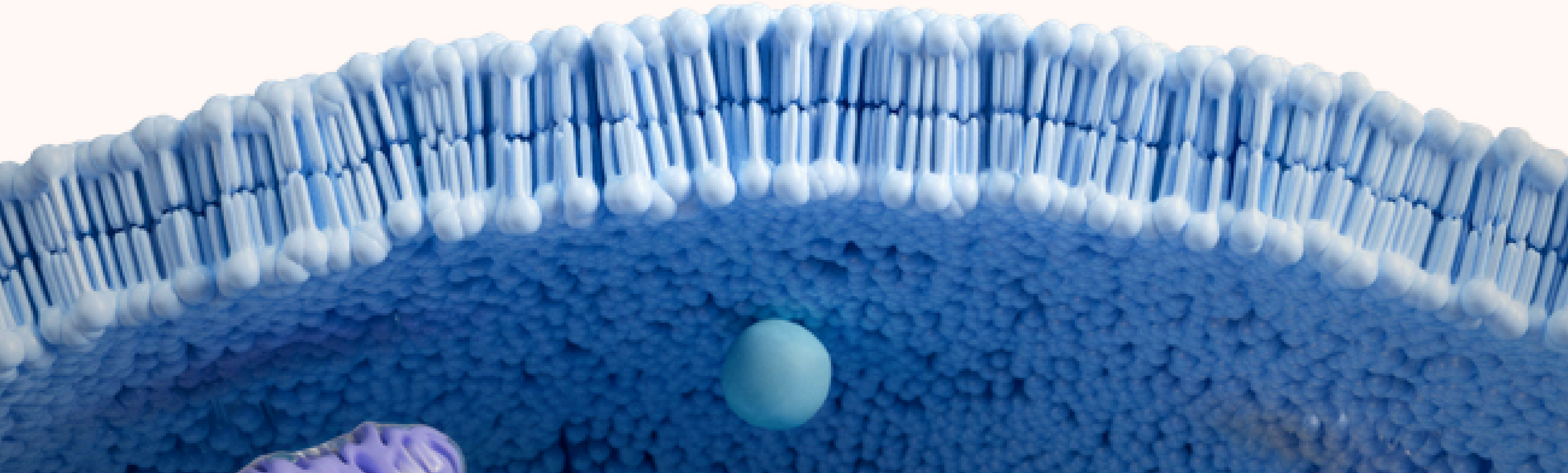
Optimizing Fertility:
The Impact of Mitochondrial Function on Reproductive Health

6

Phospholipid Therapy and Mitochondrial Restoration

Phospholipid Therapy & Mitochondria Restoration

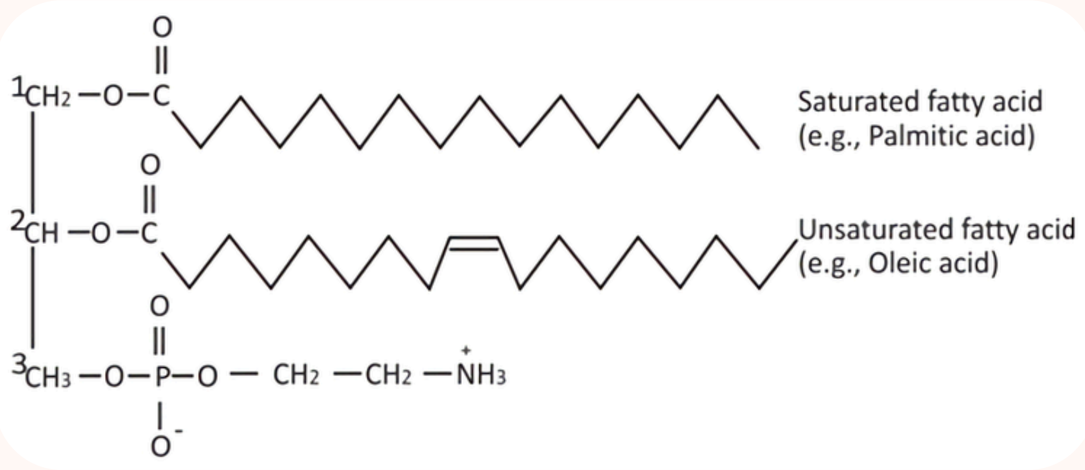
Phospholipids play a crucial role in maintaining and restoring mitochondrial function. They are essential components of cellular membranes, including mitochondrial membranes, and contribute to various mitochondrial functions.



Phosphatidylethanolamine (PE)

Phosphatidylethanolamine (PE), the second most abundant phospholipid found in mitochondria, **is essential for maintaining mitochondrial membrane stability and proper protein folding**.

It also supports key mitochondrial dynamics—**such as fusion and fission**—that are vital for efficient energy production and overall cellular health.



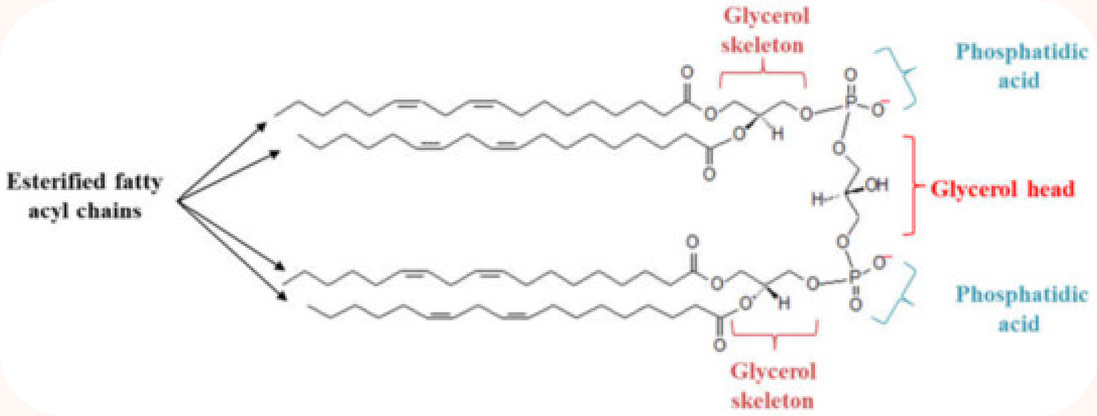
Creative Biolabs. (n.d.). *Phosphatidylethanolamine (PE)*. Creative Biolabs.

Cardiolipin and Linoleic Acid

Cardiolipin is the unique phospholipid of the inner mitochondrial membrane, distinguished by its unique “double phospholipid” structure—two phosphatidic acid units linked by a glycerol backbone, giving it four fatty acid chains instead of the usual two.

This structure is **essential for stabilizing the electron transport chain, supporting ATP synthesis, and maintaining overall mitochondrial membrane architecture**.

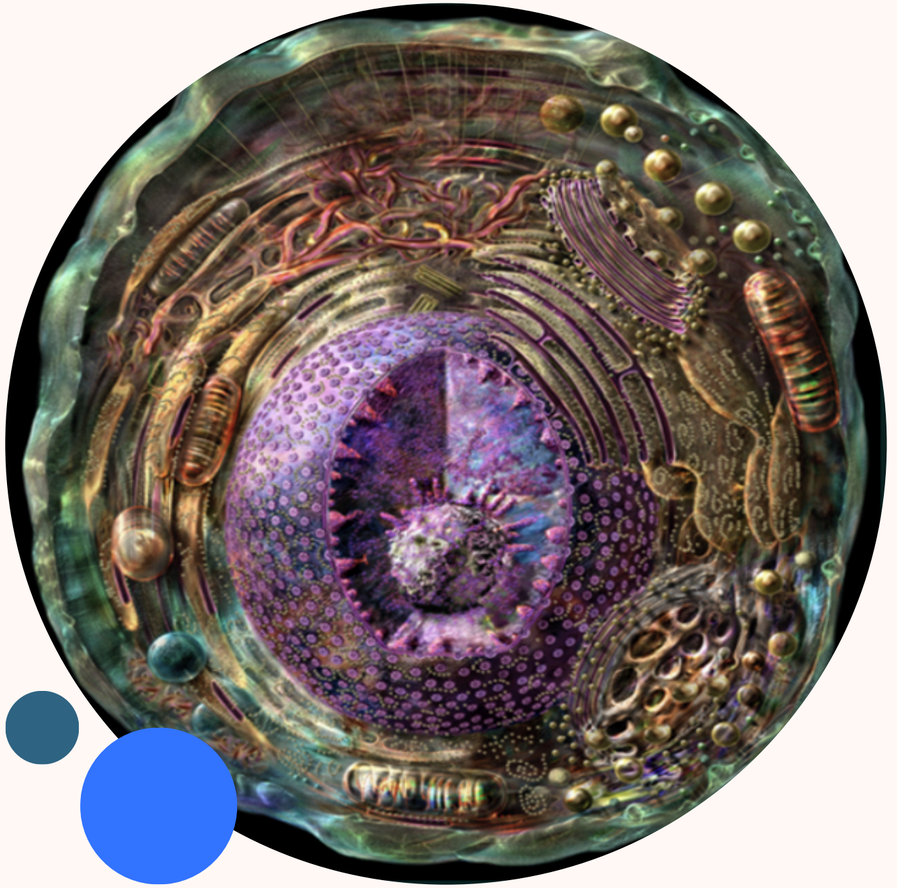
Its integrity **relies heavily on linoleic acid**, a key fatty acid that **preserves cardiolipin’s composition and protects it from oxidative damage**, thereby supporting mitochondrial longevity and preventing dysfunction.



Ahmadpour, S. T., Mahéo, K., Servais, S., Brisson, L., & Dumas, J.-F. (2020). Cardiolipin, the Mitochondrial Signature Lipid: Implication in Cancer. *International Journal of Molecular Sciences, 21* (21), 8031.

How Linoleic Acid Influences Cardiolipin

- **Structural Integrity:** Linoleic acid is a key component of cardiolipin, ensuring proper mitochondrial membrane structure and function.
- **Energy Production:** Linoleic acid helps cardiolipin support the electron transport chain, crucial for DDW water production and ATP production.
- **Protection from Oxidative Stress:** Linoleic acid in cardiolipin enhances the membrane's resistance to oxidative damage, preserving mitochondrial health.
- **Mitochondrial Efficiency:** Linoleic acid’s role in cardiolipin is vital for maintaining efficient energy transfer within the mitochondria.



Phospholipid Therapy & Mitochondria Restoration

1. Energy Production (ATP Synthesis and Water Formation)
2. Regulation of Metabolic Pathways
3. Apoptosis Regulation
4. Calcium Homeostasis
5. Reactive Oxygen Species (ROS) Production and Detoxification
6. Mitochondrial Biogenesis
7. Thermogenesis
8. Synthesis of Steroid Hormones
9. Amino Acid and Protein Synthesis
10. Aging and Longevity/Lifespan Regulation
11. “Sixth Sense” - Environmental Sensing
12. Redox Sensing

Optimizing Fertility:
The Impact of Mitochondrial Function on Reproductive Health

7

Case Studies / IGL Mitochondrial Testing

Before Lipid Replacement Therapy

Test name	Review	Result			optimal reference value [SI units]	
Investigation I Analysis councilarist, curative						
ATP - Profile -mixed leucocytes, whole cells-			(*1-10^6 neutrophils cells)			
ATP [endogenous Mg only]	normal	1,55	nmol/10^6	Aktivity of phosphorylase kinase (PhK)	0,9 - 2,7	nmol/10^6
ATP^{Mg+} [with excess Mg added]	low	1,36	nmol/10^6		1,6 - 2,9	nmol/10^6
Ratio ATP I ATP^{Mg}	normal	1,14	ratio		> 0,65	ratio
Ratio ATP^{Mg} I ATP [PhK activity]	Activation	0,88	ratio		0,6 - 1,0	ratio
ADP to ATP conversion efficiency [whole cells]						
ATP^{Mg+}	low	1,10	nmol/10^6		1,6 - 2,9	nmol/10^6
ATP^{Mg+} [inhibitor present]	normal	0,27	nmol/10^6		< 0,3	nmol/10^6
ATP^{Mg+} [inhibitor removed]	normal	1,82	nmol/10^6		> 1,4	nmol/10^6
ADP to ATP efficiency	normal	186,75	%		> 60,0	%
Blocking of active sites	high	24,55	%		≤ 14,0	%
ADP - ATP Translocator -mt-TL, mitochondria-						
Baseline	normal	445,73	pmol/10^6		290 - 700	pmol/10^6
excess ADP	normal	774,73	pmol/10^6		410 - 950	pmol/10^6
Increase	normal	73,81	change %	in-vitro test reflects ATP supply for cytoplasm	> 35	change %
ADP blocked	normal	219,55	pmol/10^6		140 - 330	pmol/10^6
Decrease	normal	66,53	change %	in-vitro test reflects normal use of ATP on energy demand	55 - 75	change %

Biochemical Report

Mitochondrial Function is...

Laboratory technical report I Clinical findings of end

Cell-free DNA (cfDNA)

Requisition #	1382	Wittbek, October 29th, 2018
Order number	15485 LK 291018 14	
Patient name	Stenger, Justine Leanne	Labor input October 26th, 2018
Date of birth	11.10.1983	Labor output October 29th, 2018
Age of the patient	35 Jahre	Blood collection October 24th, 2018
Doctor I Therapist	Mr. Dr. Bruce Hoffman, M.D.	Acceptance time 08:30 a.m.
Internal reference I Reference-ID	3097	Laboratory Time Code 10:30
Number of parameters	14	Prefabrication available -
Material	Plasma	
DNA Examinations, pathological values	Double checked	
Investigation	Analysis councilarist, curative	

Test name	Review	Result		optimal reference value [SI units]	
cfDNA	-		µg/dl	< 9,5	µg/dl
	-		µg/dl	9,6 - 12,4	µg/dl
	-		µg/dl	12,5 - 14,9	µg/dl
	definite increase	18,55	µg/dl	15,0 - 20,0	µg/dl
	-		µg/dl	> 20,0	µg/dl

Analytical note: Unmodified oligonucleotide of primer, Tris-acetate-EDTA Buffer. PCR amplification & measured in BCA protein assay (Pierce 660nm protein assay).

Wittbek, October 29th, 2018

We are grateful for the Order!

With friendly and collegial greetings

Validated by Dr.rer.nat. Maria Stromiedel

Biochemist / Laboratory manager

Age of the patient	[illegible]	Blood collection October 24th, 2016
Doctor I Therapist	Mr. Dr. Bruce Hoffman, M.D.	Acceptance time 08:30 a.m.
Internal reference I Reference-ID	3097	Laboratory Time Code 10:30
Number of parameters	14	Prefabrication available -
Material	Plasma	
DNA Examinations, pathological values	Double checked	

Test name	Review	Result		Before value	optimal reference value [SI units]	
Investigation I Analysis councilarist, curative						
Membrane lipids that fluorescence-stain as CL	low	15,36	%	Percentage of	19,0 - 28,0	%
CDP-diaclyglycerol	normal	136,00	pmol/l	CL-synthesis precursor	105,0 - 230,0	pmol/l
CL-synthase	low	75,36	U/10^6 mt	-	85,0 - 130,0	U/10^6 mt
CL-synthase manganese sites	normal	16,82	No./10nM	No. per 10nM of membrane	11,0 - 24,0	No./10nM
No. of above occupied	normal	3,09	No./10nM	by metals other than manganese	> 3,0	No./10nM
CL-binding zenobiotics		Mercury	-	metal from above or other substance	Absent	-
Cytochrome-c-oxidase complex	low	1,45	No./10nM	No. Bound to each CL molecule	1,6 - 2,9	No./10nM
Gating action of cytochrome-c-CL complex		poor	-	on pH change	normal	-
Cytochrome-c-CL complex		low	-	Oxidative stress	normal	-
Malondialdehyde, MDA	normal	29,18	pmol/10^6 mt	-	< 32,0	pmol/10^6 mt
Phosphatidylethanolamine, PTE, Kephaline	low	34,45	%	-	> 45	%
Phosphatidyl choline, PTC	normal	127,09	U/10^6 mt	total-(PTE+CL), estimate	> 100,0	U/10^6 mt

Methods: Fluorescence microscopy

Number of parameters	14	Prefabrication available -
Material	Serum, EDTA	
DNA Examinations, pathological values	Double checked	

Test name	Review	Result			optimal reference value [SI units]	
Investigation I Analysis councilarist, curative						
Glutathione-S-tranferase studies						
Glutathione-S-tranferase I.Serum (liver enzyme activity)	normal	26,36	U/ml	GS-FID	12,0 - 46,0	U/ml
Lever abnorm. Enzyme bands		0				
Glutathione-S-tranferase I.EDTA (red cells)	normal	92,18	U/ml	GS-FID	68,0 - 167,0	U/ml
Glutathione (GSH) in red cells	normal	2,09	mmol/l	GS-FID	1,70 - 2,60	mmol/l
Red cells enzyme bands	discrete	0	band(s)	PCR	0	band(s)
that accounts for / is associated	PAHs	7,27	%	PCR	0,00	%
Erythrocytes (RBC)	low	4,64	Mio/µl	cell counter	4,8 - 5,9	Mio/µl
[red blood count]	✔			microskopy controlled		
Glutathione-Peroxidase (GSH-Px)	normal	34,82	U/g Hb	PHOT	27,3 - 73,5	U/g Hb
Reference Hb, haemoglobin (HBG)	normal	25,73	g/dl = mmol/l		22,54 - 28,98	g/dl = mmol/l

NOTE: GSH-Px of Erythrozyten containing selenium in the form of selenocysteine. Since it takes some time, built up currently fed selenium in the GSH-Px in Erythrozytenlysat (hence regarding g Hb) currently measured activity GSH-Px activity reflects the degree of care to about 2 weeks earlier.

Glutathione, GSH-blood	low	681,55	µmol/l		760,0 - 1.200,0	µmol/l

Method: The GSTT1 activity is determined by measuring a substrate acceptance rate.
Gas chromatograph with split-Splitless injector, FID.
Capillary column, Dimethylsiloxan, Fused Silica.

After Lipid Replacement Therapy

Test name	Review	Result			optimal reference value [SI units]	
Investigation I Analysis councilarist, curative						
ATP - Profile -mixed leucocytes, whole cells-			(*1-10⁶ neutrophils cells)			
ATP [endogenous Mg only]	normal	2,45	nmol/10⁶	Aktivity of phosphorylase kinase (PhK)	0,9 - 2,7	nmol/10⁶
ATP^{Mg+} [with excess Mg added]	normal	1,73	nmol/10⁶		1,6 - 2,9	nmol/10⁶
Ratio ATP I ATP^{Mg}	normal	1,42	ratio		> 0,65	ratio
Ratio ATP^{Mg} I ATP [PhK activity]	Activation	0,71	ratio		0,6 - 1,0	ratio
ADP to ATP conversion efficiency [whole cells]						
ATP^{Mg+}	normal	2,00	nmol/10⁶		1,6 - 2,9	nmol/10⁶
ATP^{Mg+} [inhibitor present]	normal	0,18	nmol/10⁶		< 0,3	nmol/10⁶
ATP^{Mg+} [inhibitor removed]	normal	1,83	nmol/10⁶		> 1,4	nmol/10⁶
ADP to ATP efficiency	normal	90,66	%		> 60,0	%
Blocking of active sites	normal	9,00	%		≤ 14,0	%
ADP - ATP Translocator -mt-TL, mitochondria-						
Baseline	normal	290,27	pmol/10⁶		290 - 700	pmol/10⁶
excess ADP	normal	723,18	pmol/10⁶		410 - 950	pmol/10⁶
Increase	normal	149,14	change %	in-vitro test reflects ATP supply for cytoplasm	> 35	change %
ADP blocked	normal	147,45	pmol/10⁶		140 - 330	pmol/10⁶
Decrease	low	44,68	change %	in-vitro test reflects normal use of ATP on energy demand	55 - 75	change %

Biochemical Report

Requisition #	NRF0050135	Wittbek, July 24th, 2020
Order number	32790 LK 28520 03	
Patient name	Stenger, Justine Leanne	Labor input July 222th, 2020
Date of birth	11.10.1983	Labor output July 23th, 2020
Age of the patient	36 Years	Blood collection July 20th, 2020
Doctor I Therapist	Mr. Dr. MD. B. Hoffman	Acceptance time --:-- a.m.
Internal reference I Reference-ID	6558	Laboratory Time Code 10:30
Number of parameters	15	Prefabrication available -
Material	Plasma	
DNA Examinations, pathological values	Double checked	
Investigation	Analysis councilarist, curative	

Test name	Review	Result	optimal reference value [SI units]
	normal	8,89 µg/dl	< 9,5 µg/dl
cfDNA	-	- µg/dl	9,6 - 12,4 µg/dl
	-	- µg/dl	12,5 - 14,9 µg/dl
	-	- µg/dl	15,0 - 20,0 µg/dl
	-	- µg/dl	> 20,0 µg/dl

Analytical note: Unmodifired oligonucloetide of primer, Tris-acetate-EDTA Buffer. PCR ampilfication & measured in BCA protein assay (Pierce 660nm protein assay).

Test name	Review	Result		Before value	optimal reference value [SI units]	
Investigation I Analysis councilarist, curative						
Membrane lipids that fluorescence-statin as CL	normal	21,00	%	Percentage of	19,0 - 28,0	%
CDP-diaclyglycerol	normal	145,91	pmol/l	CL-synthesis precursor	105,0 - 230,0	pmol/l
CL-synthase	normal	97,82	U/10^6 mt	-	85,0 - 130,0	U/10^6 mt
CL-synthase manganese sites	normal	16,64	No./10nM	No. per 10nM of membrane	11,0 - 24,0	No./10nM
No. of above occupied	normal	3,27	No./10nM	by metals other than manganese	> 3,0	No./10nM
CL-binding zenobiotics		-	-	metal from above or other substance	Absent	-
Cytochrome-c-oxidase complex	normal	2,09	No./10nM	No. Bound to each CL molecule	1,6 - 2,9	No./10nM
Gating action of cytochrome-c-CL complex		normal	-	on pH change	normal	-
Cytochrome-c-CL complex		normal	-	Oxidative stress	normal	-
Malondialdehyde, MDA	normal	27,36	pmol/10^6 mt	-	< 32,0	pmol/10^6 mt
Phosphatidylethanolamine, PTE, Kephaline	low	37,82	%	-	> 45	%
Phosphatidyl choline, PTC	normal	113,55	U/10^6 mt	total-(PTE+CL), estimate	> 100,0	U/10^6 mt

Methods: Fluorescence microscopy

Age of the patient	36 Years	Blood collection July 20th, 2020
Doctor I Therapist	Mr. Dr. MD. B. Hoffman	Acceptance time --:-- a.m.
Internal reference I Reference-ID	6558	Laboratory Time Code 10:30
Number of parameters	15	Prefabrication available -
Material	Plasma	
DNA Examinations, pathological values	Double checked	
Investigation	Analysis councilarist, curative	

Match I Adduct found	Review	Result		Gene identified	optimal reference value [SI units]	
Total DNA	normal	42,84	µg/ml		30,0 - 60,0	µg/ml
DNA - Adducts						
#1 NO MATCH FOUND		-	ng/ml	.		
#2 --		-	ng/ml	.		
#3 --		-	ng/ml	-		
#4 --		-	ng/ml	-		
DNA - associated Zinc (Zn)	normal	31,52	ng/ml		21,0 - 74,0	ng/ml

Comments adducts	Gene translations
#1 --	#1 --
#2 --	#2 --
#3 --	#3 --
#4 --	#4 --

DNA Examinations, pathological values Double checked

Test name	Review	Result			optimal reference value [SI units]	
Investigation I Analysis councilarist, curative						
Glutathione-S-tranferase studies						
Glutathione-S-tranferase i.Serum (liver enzyme activity)	normal	14,82	U/ml	GS-FID	12,0 - 46,0	U/ml
Lever abnorm. Enzyme bands		0				
Glutathione-S-tranferase i.EDTA (red cells)	normal	68,18	U/ml	GS-FID	68,0 - 167,0	U/ml
Glutathione (GSH) in red cells	normal	2,18	mmol/l	GS-FID	1,70 - 2,60	mmol/l
Red cells enzyme bands	discrete	0	band(s)	PCR	0	band(s)
that accounts for / is associated	PAHs	6,82	%	PCR	0,00	%
Erythrocytes (RBC)	normal	5,36	Mio/µl	cell counter	4,8 - 5,9	Mio/µl
[red blood count]		✔		microskopy controlled		
Glutathione-Peroxidase (GSH-Px)	low	22,00	U/g Hb	PHOT	27,3 - 73,5	U/g Hb
Reference Hb, haemoglobin (HBG)	normal	22,82	g/dl = mmol/l		22,54 - 28,98	g/dl = mmol/l

NOTE: GSH-Px of Erythrozyten containing selenium in the form of selenocysteine. Since it takes some time, built up currently fed selenium in the GSH-Px in Erythrozytenlysat (hence regarding g Hb) currently measured activity GSH-Px activity reflects the degree of care to about 2 weeks earlier.

Glutathione, GSH-blood	normal	1.051,18	µmol/l		760,0 - 1.200,0	µmol/l

Method: The GSTT1 activity is determined by measuring a substrate acceptance rate.
Gas chromatograph with split-Splitless injector, FID.
Capillary column, Dimethylsiloxan, Fused Silica.

Revitalizing Mitochondrial Function & Leptin for Reproductive Health

8

Lifestyle and Dietary Recommendations

Nutritional Support for Fertility

Emphasize a **high-quality, animal-based diet** with ample **DHA-rich seafood**.

Choose **seasonal, local foods** that align with your circadian environment.

Prioritize **phospholipid- and essential fatty acid–rich foods** to support metabolic and mitochondrial health.

Avoid processed foods, GMOs, corn, mustard, peanuts, vegetable oils, grains, beans, legumes, tropical fruits, roasted nuts/seeds, high-heat cooking/frying, leftovers, and conventionally raised meats.

The Leptin Prescription

Optimizing Macronutrient Composition

- Follow a Paleolithic or Ketogenic-style approach to reduce foods that interfere with leptin receptor function.
- Prioritize a protein- and fat-rich breakfast (40–75 g protein; 0–25 g carbohydrates).

Carbohydrate Management

- Limit carbohydrates to ~25 g/day if overweight by more than 30 lbs; adjust gradually for those with less weight to lose or who are underweight.
- Choose local, seasonal foods, and avoid tropical foods when living at higher latitudes.

The Leptin Prescription

Metabolic & Meal-Timing Strategies

- Eliminate snacking to support liver metabolism and circadian alignment for optimal leptin function.
- Begin with three structured meals, transitioning to two meals as true hunger cues stabilize.
- Prioritize an early breakfast and avoid exercising immediately before or after it.
- Maintain a 4–5 hour window between dinner and sleep.

Evening Light & Pre-Sleep Routine

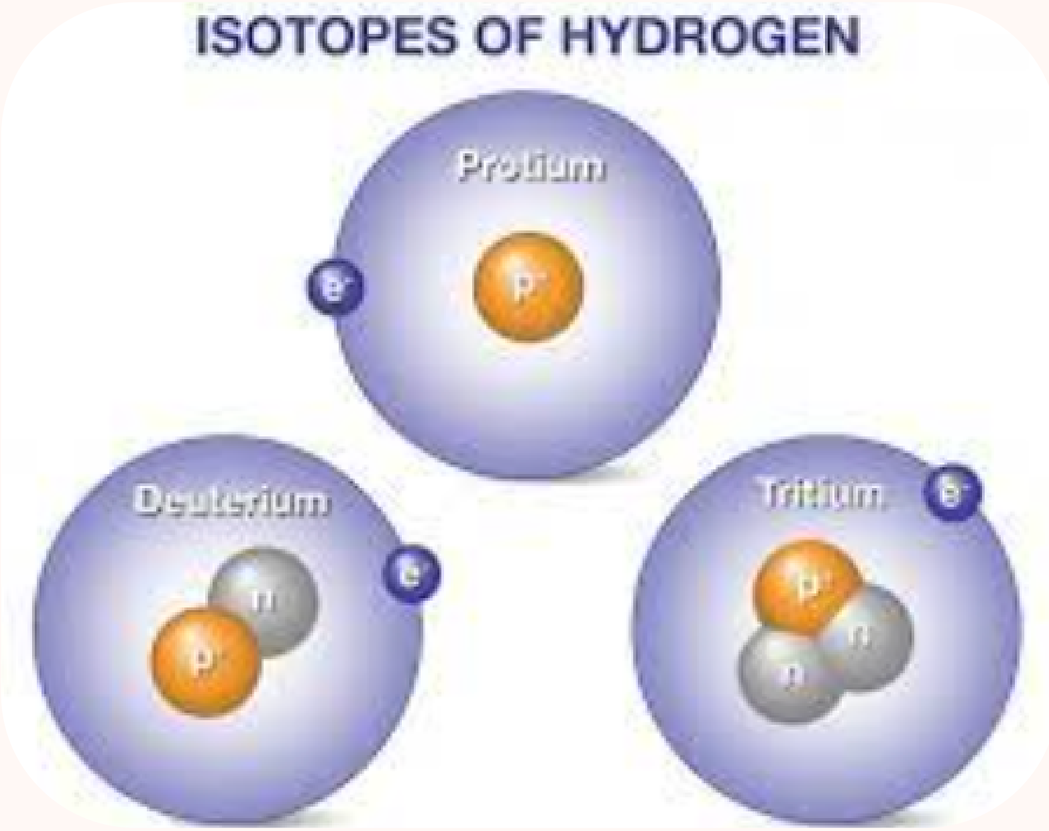
- Remove blue light after sunset (or by 7:00 pm at higher latitudes); use red lighting until bedtime.
- Wear blue-blocking glasses 2 hours before sleep.
- Optional: 3–5 minutes of light bodyweight exercise before bed—skip if evening cortisol is high.

Deuterium Depletion & Fertility

Deuterium is a **heavy isotope of hydrogen** containing both a proton and a neutron, and its added mass **slows mitochondrial function and ATP production**.

Higher-than-normal deuterium levels—often measured in saliva—are associated with **reduced sperm motility and weaker egg bioenergetics**.

Depleting deuterium supports more efficient mitochondria and has been shown in studies, especially animal models, to **improve oocyte development, sperm motility, and embryo implantation**.[12]



BODYBIO

Thank You

Justine Stenger
BodyBio Clinical Educator

References

1. Baldini, G., & Phelan, K. D. (2019). The melanocortin pathway and control of appetite—Progress and therapeutic implications. *Journal of Endocrinology, 241*(1), R1–R33. https://doi.org/10.1530/JOE-18-0596
2. Basu Ball, W., Neff, J. K., & Gohil, V. M. (2018). The role of nonbilayer phospholipids in mitochondrial structure and function. *FEBS Letters, 592*(8), 1273–1290. https://doi.org/10.1002/1873-3468.12887
3. Benkhalifa, M., Ferreira, Y. J., Chahine, H., Louanjli, N., Miron, P., Merviel, P., & Copin, H. (2014). Mitochondria: Participation to infertility as source of energy and cause of senescence. *International Journal of Biochemistry & Cell Biology*, 55, 60–64. https://doi.org/10.1016/j.biocel.2014.08.011
4. Boguenet, M., Bouet, P.-E., Spiers, A., Reynier, P., & May-Panloup, P. (2021). Mitochondria: Their role in spermatozoa and in male infertility. *Human Reproduction Update, 27*(4), 697–719. https://doi.org/10.1093/humupd/dmab001
5. Cacciottola, L., et al. (2021). Oxidative stress, mitochondria, and infertility: Is the relationship fully established? *Fertility and Sterility, 116*(2), 306–308.
6. Childs, G. V., Odle, A. K., MacNicol, M. C., & MacNicol, A. M. (2021). The importance of leptin to reproduction. *Endocrinology, 162*(2), bqaa204. https://doi.org/10.1210/endocr/bqaa204
7. Chicco, A. J., & Sparagna, G. C. (2007). Role of cardiolipin alterations in mitochondrial dysfunction and disease. *American Journal of Physiology–Cell Physiology, 292*(1), C33–C44. https://doi.org/10.1152/ajpcell.00243.2006
8. Chou, S. H., & Mantzoros, C. (2014). 20 years of leptin: Role of leptin in human reproductive disorders. *Journal of Endocrinology, 223*(1), T49–T62. https://doi.org/10.1530/JOE-14-0245
9. Costa, J., Braga, P. C., Rebelo, I., Oliveira, P. F., & Alves, M. G. (2023). Mitochondria quality control and male fertility. *Biology, 12*(6), 827. https://doi.org/10.3390/biology12060827
10. Demain, L. A. M., Conway, G. S., & Newman, W. G. (2017). Genetics of mitochondrial dysfunction and infertility. *Clinical Genetics, 91*(2), 199–207. https://doi.org/10.1111/cge.12896
11. Horvath, S. E., & Daum, G. (2013). Lipids of mitochondria. *Progress in Lipid Research, 52*(4), 590–614. https://doi.org/10.1016/j.plipres.2013.07.002

References

12. Jansen, R. P. S., & Burton, G. J. (2004). Mitochondrial dysfunction in reproduction. *Mitochondrion, 4*(5–6), 577–600. https://doi.org/10.1016/j.mito.2004.07.038
13. Moschos, S., et al. (n.d.). Leptin and reproduction: A review. *Fertility and Sterility, 77*(3), 433–444.
14. Nicolson, G. L. (2014). Mitochondrial dysfunction and chronic disease: Treatment with natural supplements. *Integrative Medicine (Encinitas), 13*(4), 35–43.
15. Osman, C., Voelker, D. R., & Langer, T. (2011). Making heads or tails of phospholipids in mitochondria. *Journal of Cell Biology, 192*(1), 7–16. https://doi.org/10.1083/jcb.201006159
16. Pamplona, R. (2008). Membrane phospholipids, lipoxidative damage and molecular integrity: A causal role in aging and longevity. *Biochimica et Biophysica Acta (Bioenergetics), 1777*(10), 1249–1262. https://doi.org/10.1016/j.bbabio.2008.07.003
17. Scientific Committee on Emerging and Newly Identified Health Risks. (2015). Opinion on potential health effects of exposure to electromagnetic fields (EMF). *European Commission.* https://ec.europa.eu/health/scientific_committees/emerging/docs/scenihr_o_041.pdf
18. Sagalovsky, V. (2023, October 7). Deuterium depletion: A promising drug-free alternative intervention for enhancing fertility. *Gale Academic OneFile.* https://go.gale.com/ps/i.do?id=GALE%7CA771913945
19. Shao, S., Zhao, H., Lu, Z., Lei, X., & Zhang, Y. (2021). Circadian rhythms within the female HPG axis: From physiology to etiology. *Endocrinology, 162*(8), bqab117. https://doi.org/10.1210/endocr/bqab117
20. Song, J., Xiao, L., Zhang, Z., Wang, Y., Kouis, P., Rasmussen, L. J., & Dai, F. (2024). Effects of reactive oxygen species and mitochondrial dysfunction on reproductive aging. *Frontiers in Cell and Developmental Biology,* 12. https://doi.org/10.3389/fcell.2024.1347286
21. Stefano, G. B., & Kream, R. M. (2016). Mitochondrial DNA heteroplasmy in human health and disease. *Biomedical Reports, 4*(3), 259–262. https://doi.org/10.3892/br.2016.590
22. Tesarik, J., & Mendoza-Tesarik, R. (2023). Mitochondria in human fertility and infertility. *International Journal of Molecular Sciences, 24*(10), 8950. https://doi.org/10.3390/ijms24108950

References

23. Toffol, E., Heikinheimo, O., & Partonen, T. (2016). Biological rhythms and fertility: The hypothalamus–pituitary–ovary axis. *ChronoPhysiology and Therapy*, 6, 15–27. https://doi.org/10.2147/CPT.S84855
24. Wallace, D. C. (2005). A mitochondrial paradigm of metabolic and degenerative diseases, aging, and cancer: A dawn for evolutionary medicine. *Annual Review of Genetics*, 39, 359–407.
25. Wallace, D. C., & Chalkia, D. (2013). Mitochondrial DNA genetics and the heteroplasmy conundrum in evolution and disease. *Cold Spring Harbor Perspectives in Biology*, *5*(11), a021220. https://doi.org/10.1101/cshperspect.a021220
26. Wang, J.-J., Wang, S.-X., Tehmina, Feng, Y., Zhang, R.-F., Li, X.-Y., Sun, Q., & Ding, J. (2022). Age-related decline of male fertility: Mitochondrial dysfunction and the antioxidant interventions. *Pharmaceuticals*, *15*(5), 519. https://doi.org/10.3390/ph15050519
27. Webster, W. W., & Smarr, B. (2020). Using circadian rhythm patterns of continuous core body temperature to improve fertility and pregnancy planning. *Journal of Circadian Rhythms*, 18, 5. https://doi.org/10.5334/jcr.200
28. Zeng, X., Huang, Q., Long, S. L., Zhong, Q., & Mo, Z. (2020). Mitochondrial dysfunction in polycystic ovary syndrome. *DNA and Cell Biology*, *39*(8), 1401–1409. https://doi.org/10.1089/dna.2019.5172
29. Zhou, Y., Jin, Y., Wu, T., Wang, Y., Dong, Y., Chen, P., Hu, C., et al. (2023). New insights on mitochondrial heteroplasmy observed in ovarian diseases. *Journal of Advanced Research*. https://doi.org/10.1016/j.jare.2023.11.033