CRT

Cellular Regeneration Therapy

An insight into our cells for Healthy Aging and Rejuvenation Module

presented by;

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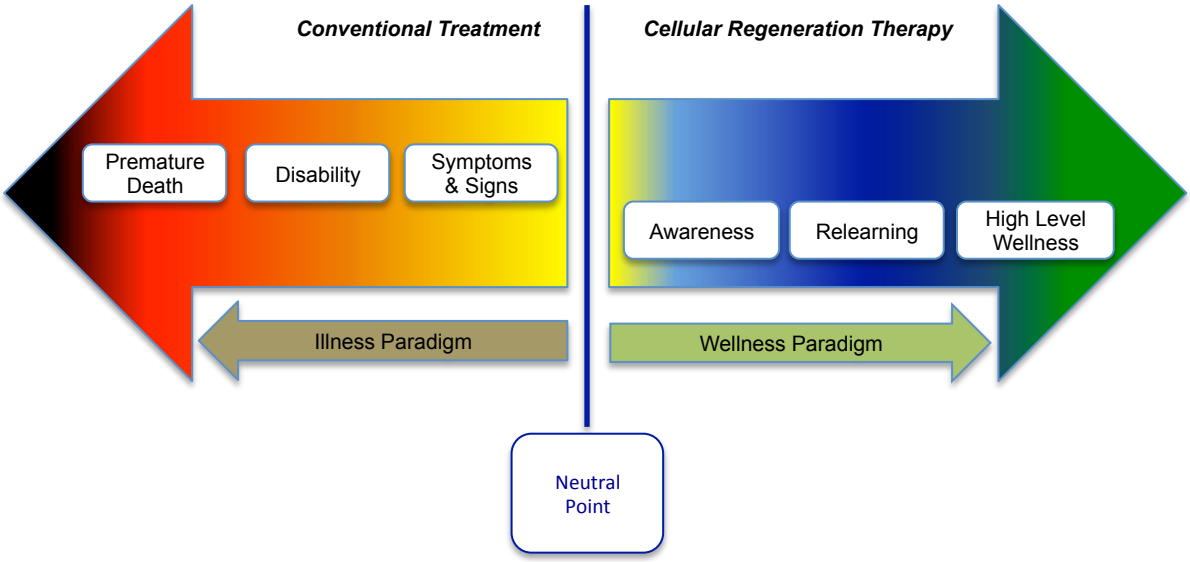
The Concept Of Illness To Wellness Paradigm



There are actually many degrees of wellness, just as there are many degrees of illness.

The Illness–Wellness Continuum illustrates the relationship of the treatment paradigm to the wellness paradigm. An individual can move beyond the "neutral" point to increasingly higher levels of wellness.

Moving from the center to the left shows a progressively worsening state of health. Moving to the right of center indicates increasing levels of health and wellbeing.

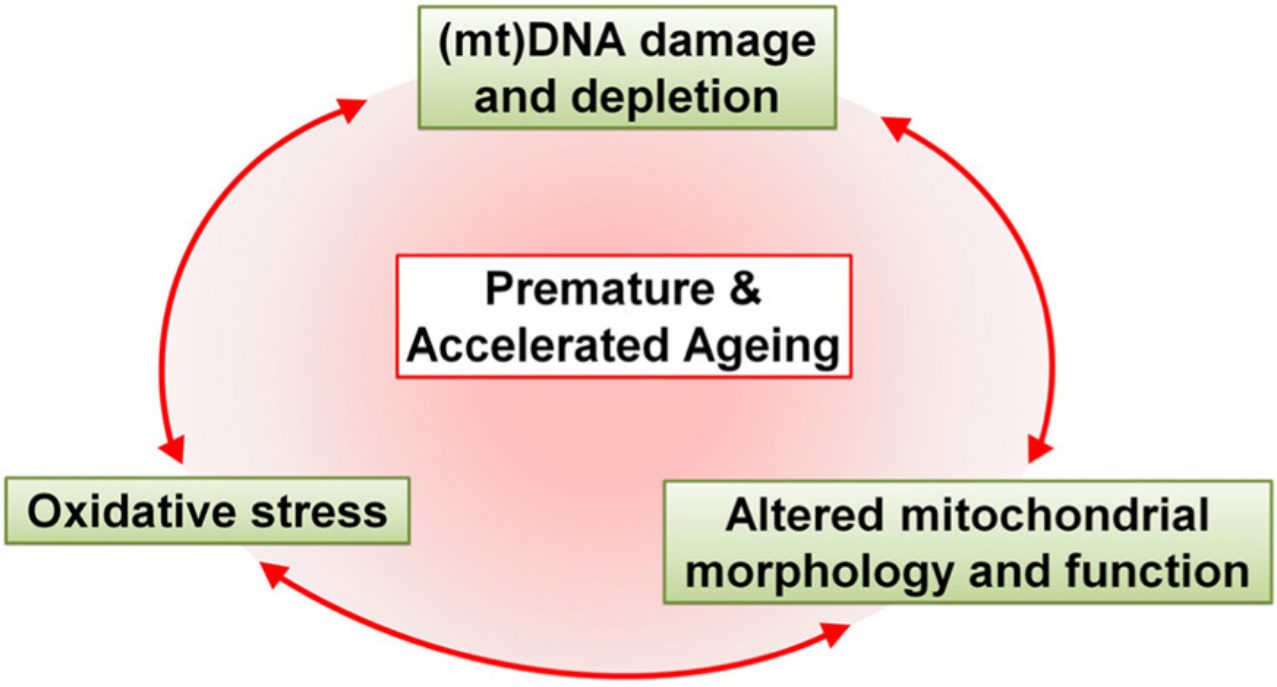
The treatment paradigm (drugs, surgery, psychotherapy, and so on) can bring you up to the neutral point, where the symptoms of disease have been alleviated. That's all it's designed to do.

On the other hand, the Wellness Paradigm, which can be used at any point on the continuum, helps you move toward higher levels of wellness.

The wellness paradigm directs you beyond the neutral point and encourages you to move as far toward wellness as possible.

If you are ill, then treatment is important, but don't stop at the neutral point. Use the wellness paradigm to move toward high-level wellness! This makes all the difference in quality of life!

What causes Premature and Accelerated Aging?



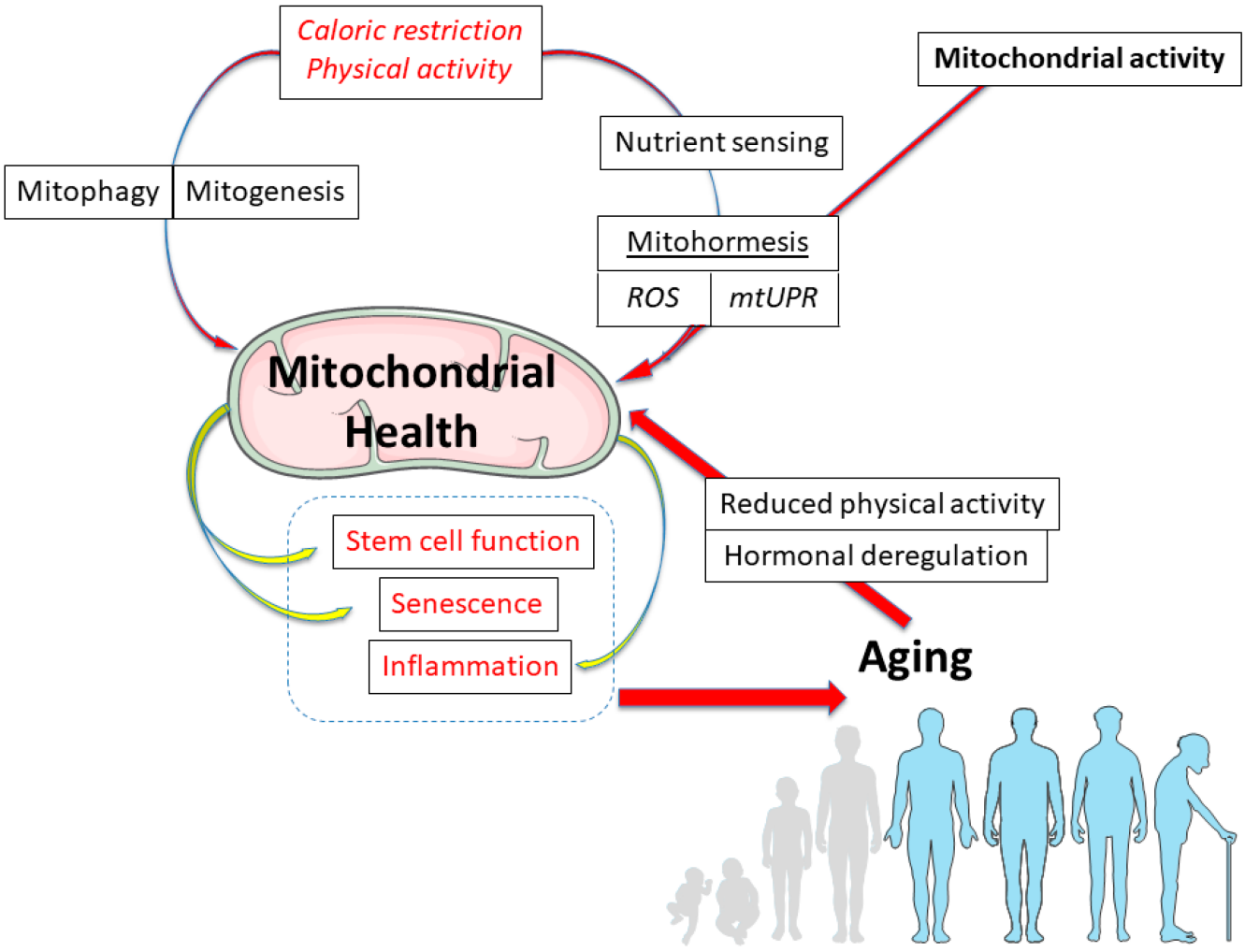
As shown in the illustration above, the Premature and Accelerated Aging are caused by 3 key cellular processes;

- *OXIDATIVE STRESS* –
  This process produces *free radicals* –which interact with the molecules within our cells resulting in damage (or stress) to nearby cells, mitochondria, and DNA (our genes).

- *ALTERED MITOCHONDRIAL FUNCTION* –
  Mitochondrial dysfunction can *affect* key cellular functions, result in a variety of diseases and *altered* mitochondrial DNA (MtDNA) levels have been reported in a wide range of human disease.

- *MITOCHONDRIAL DNA (mtDNA) DAMAGE* –
  MtDNA damage can result from endogenous, cytoplasmic, and environmental sources, and the persistence of **mtDNA damage** can cause **mtDNA** degradation. Faulty repair or replication can result in **mtDNA** point mutations.

Why Mitochondrial Health is influenced by Aging Process?



As shown in the illustration above, the mitochondrial health is influenced directly by aging process. The continuous deregulations at molecular and cellular levels directly exerts diseases or illnesses manifestation and these indirectly accelerates the aging process and it is a continuous vicious cycle that we focus to reduce.

Our CRT is proprietary prepared formulation using cell penetrating peptides after many years of research in the field of mitochondrial health and circular energy.

What Is Mitochondria And its Cellular Function?

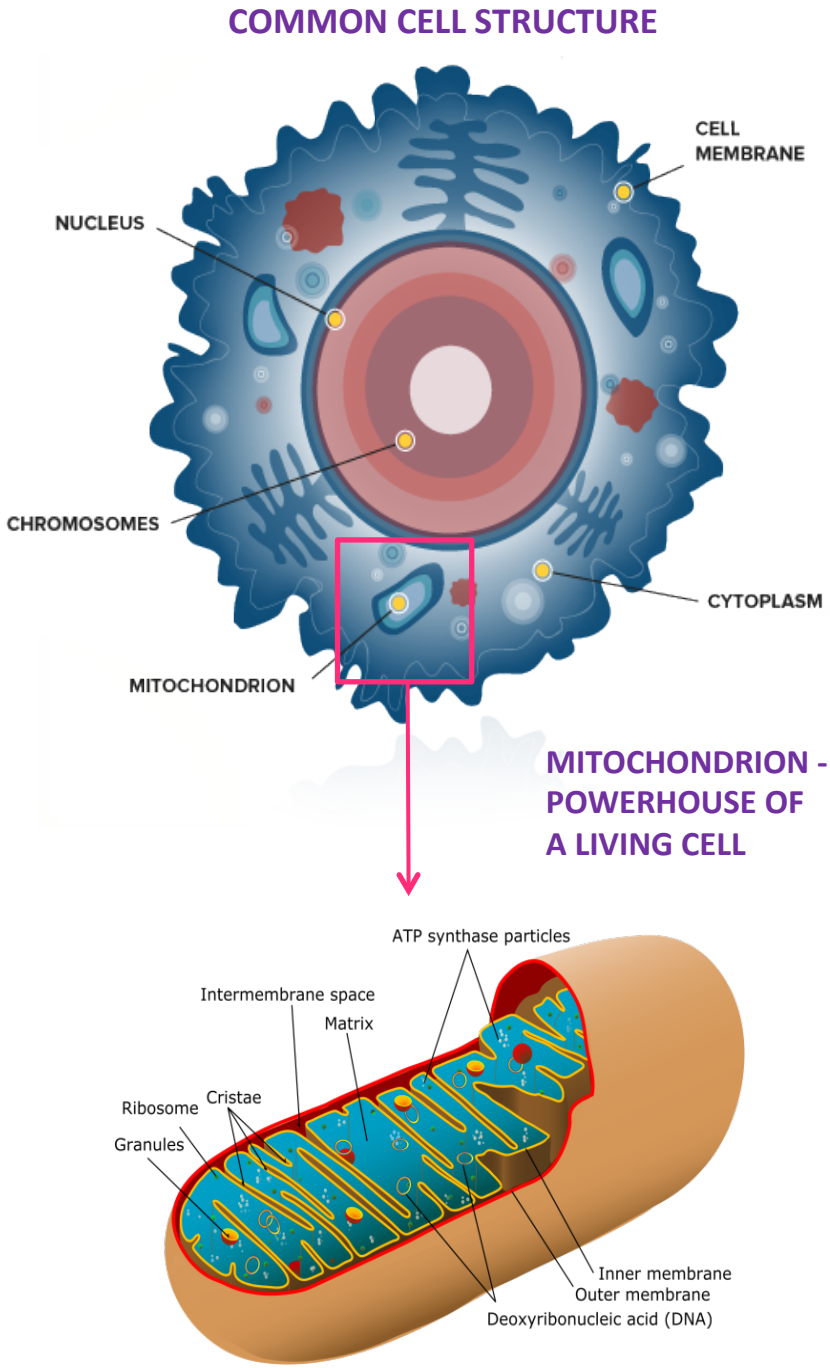
Cellular Regeneration Therapy

Mitochondria are structures within cells that convert the energy from food into a form that cells can use. Each cell contains hundreds to thousands of mitochondria, which are located in the fluid that surrounds the nucleus (the cytoplasm).

Mitochondria produce energy through a process called *oxidative phosphorylation*. This process uses oxygen and simple sugars to ***create adenosine triphosphate (ATP)***, the cell's main energy source.

The mitochondria **GENERATE ENERGY** for the cell as a whole by processing the nutrients we ingest into charged molecules, ultimately producing **ATP MOLECULES**.

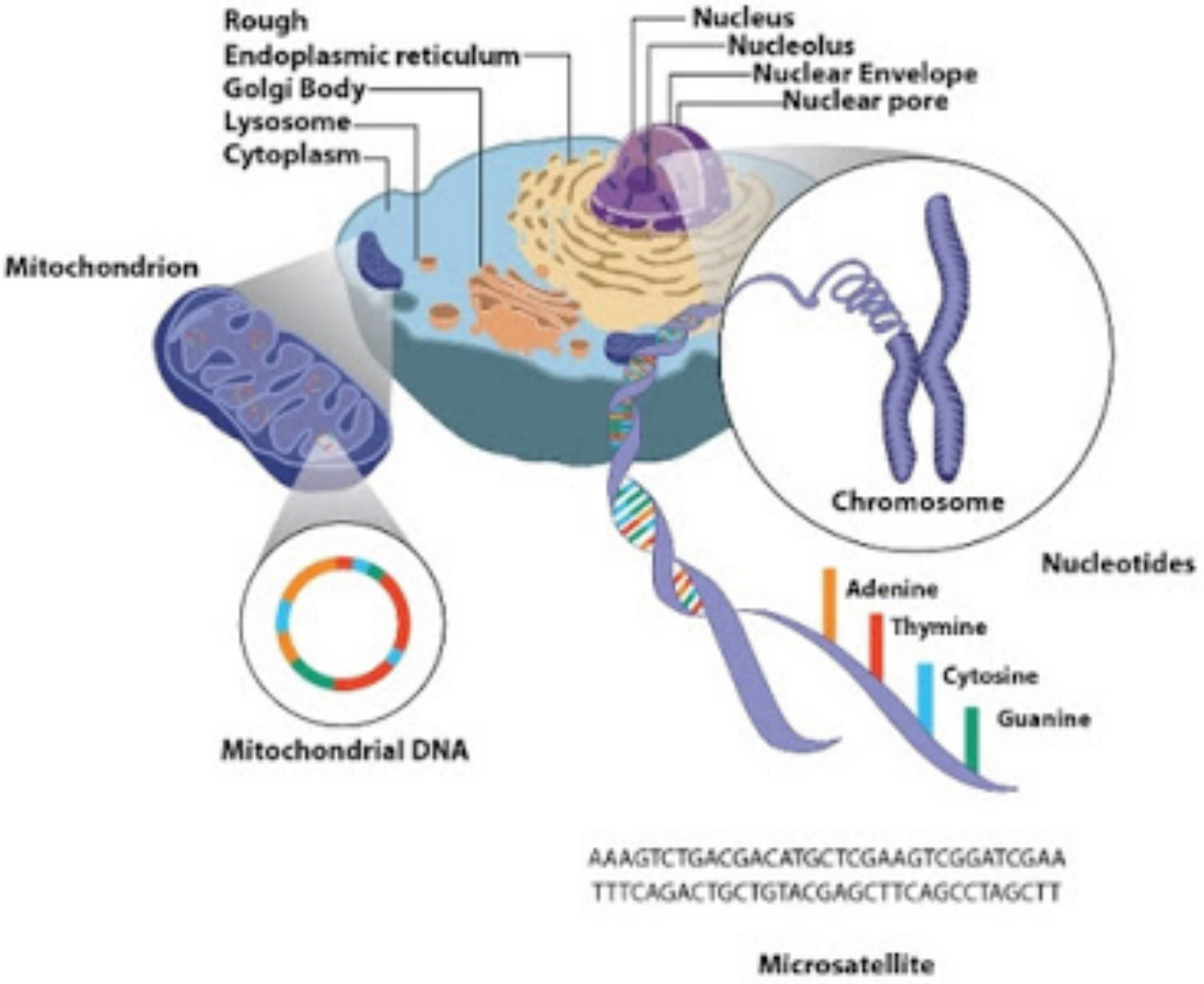
A set of enzyme complexes, designated as complexes I–V, carry out oxidative phosphorylation within mitochondria.



These mitochondrion are the **POWER STORAGE AND ENERGY PRODUCTION UNIT**.

Mitochondria also contribute to other cellular functions including the cell cycle, cell growth, apoptosis (cell death), cell differentiation and thermogenesis (heat production)

What Is Mitochondrial DNA (mtDNA) and its Cellular Function?



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.

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. The remaining genes provide instructions for making molecules called transfer RNAs (tRNAs) and ribosomal RNAs (rRNAs), which are chemical cousins of DNA. These types of RNA help assemble protein building blocks (amino acids) into functioning proteins.

CELLULAR ENERGY

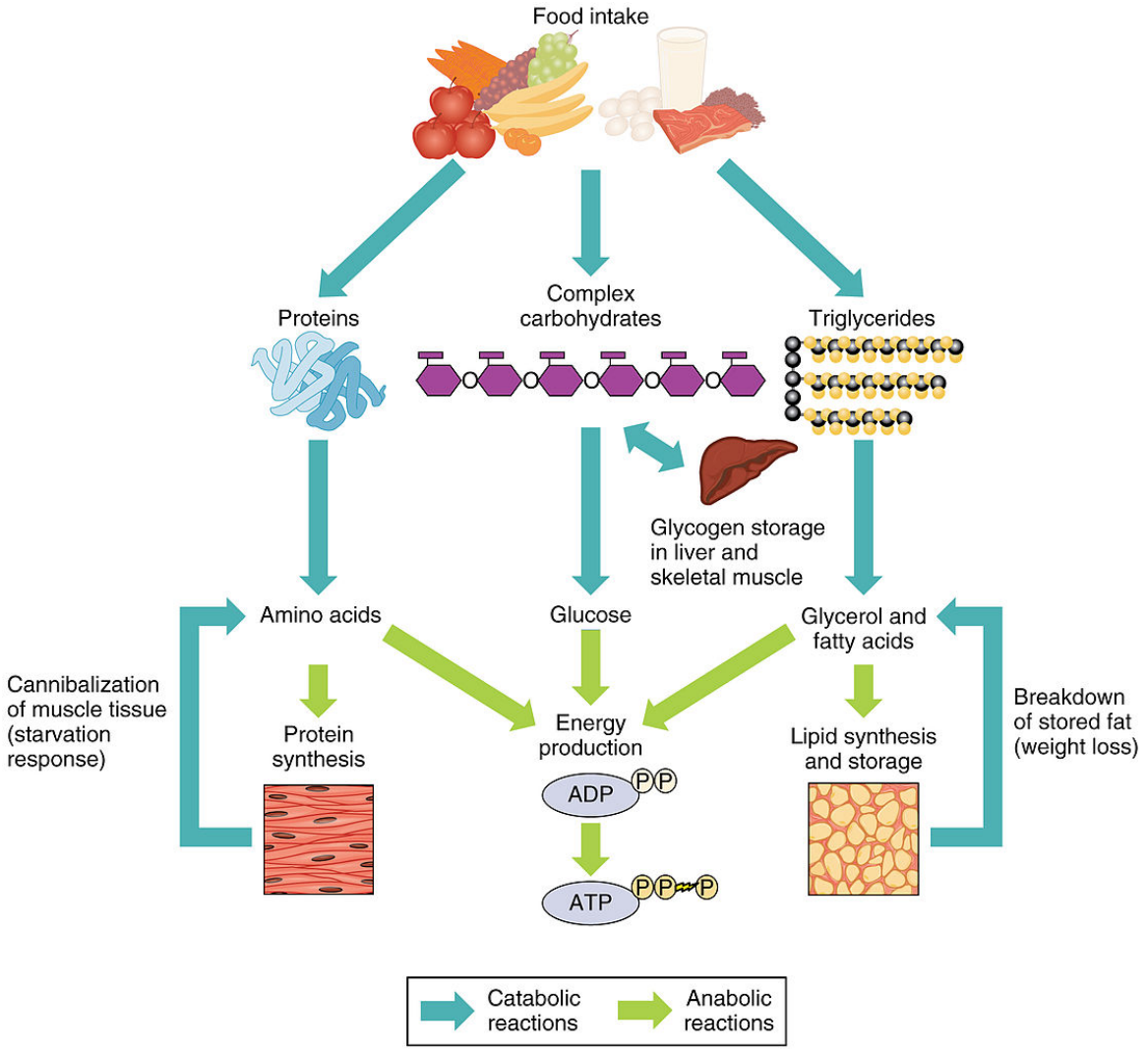
There are basically 2 types of cellular energy –

a. Metabolic Energy
b. Electrophysiological Energy

What is Metabolic Energy?

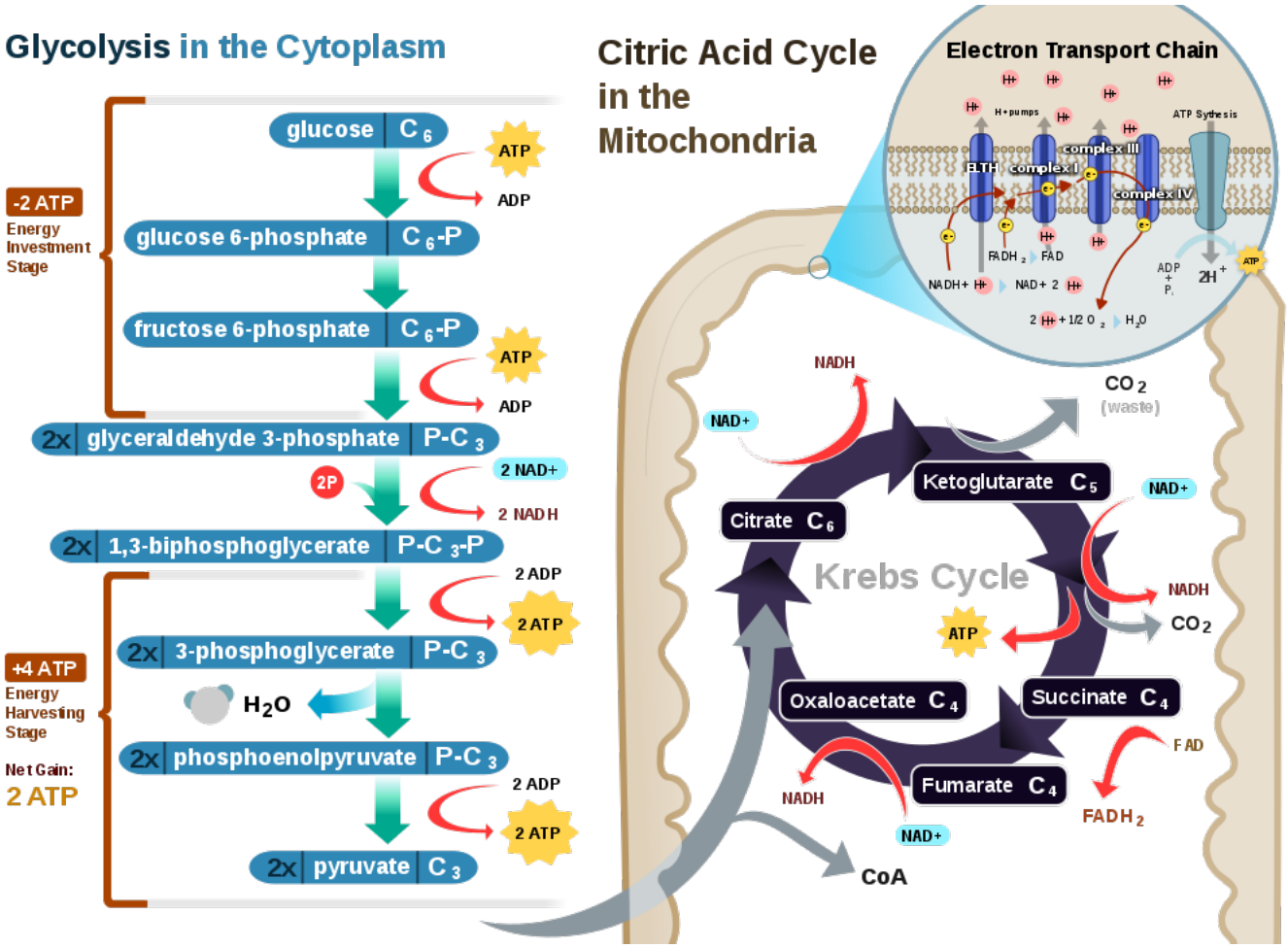
Cells are constantly carrying out thousands of chemical reactions needed to keep the cell, and your body as a whole, alive and healthy. These chemical reactions are often linked together in chains, or pathways. All of the chemical reactions that take place inside of a cell are collectively called the cell's metabolism.

In the metabolic web of the cell, some of the chemical reactions release energy and can happen spontaneously (without energy input). However, others need added energy in order to take place. Just as you must continually eat food to replace what your body uses, so cells need a continual inflow of energy to power their energy-requiring chemical reactions. In fact, the food you eat is the source of the energy used by your cells.



CELLULAR ENERGY

Breaking down glucose: Cellular respiration



Cellular Metabolism

$C_6H_{12}O_6$ + 6 (O_2) → 6 (CO_2) + 6 (H_2O) + ATP

Glucose — Oxygen — Carbon Dioxide — Water — Energy

CELLULAR ENERGY

What is Electrophysiology Energy?

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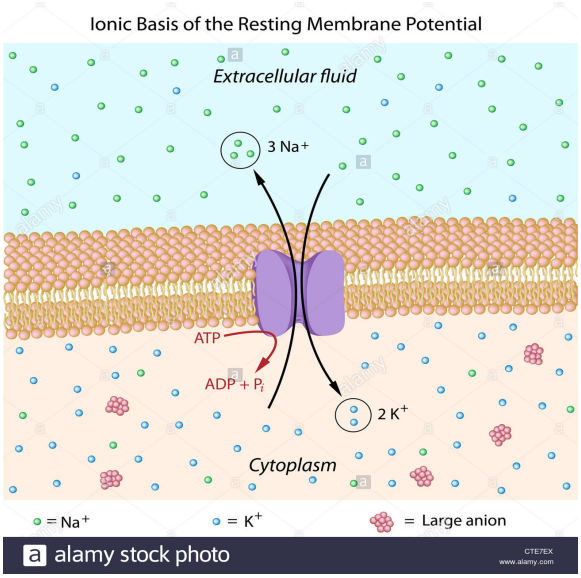
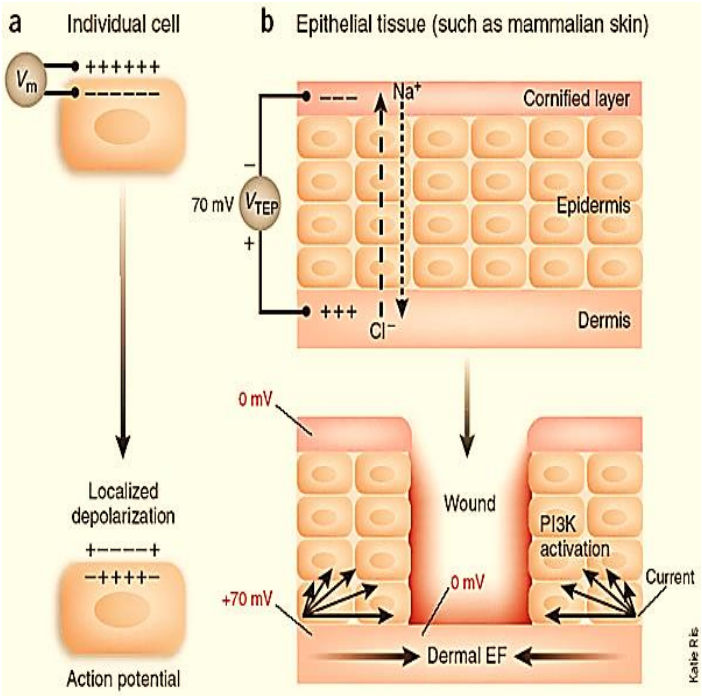
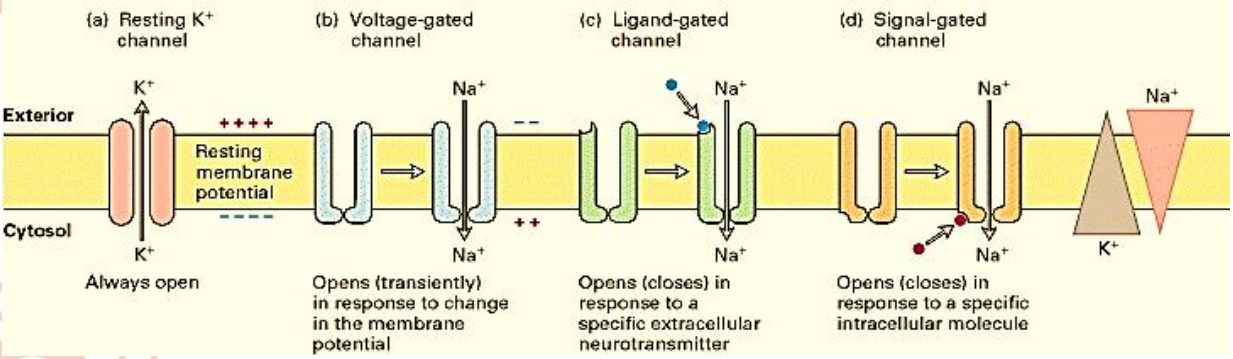
Electrophysiological energy is the RESTING MEMBRANE POTENTIAL of the cell.

The human organism is composed of multiple cells, all of them with different components and therefore with different **resting membrane potentials**. Some of these cells are excitable (e. g.: cells; neurons; muscle fibers), generating an ***ACTION POTENTIAL*** when subjected to an external stimulus, causing its membrane depolarization. The ***RESTING MEMBRANE POTENTIAL*** **(RMP)** is due to changes in membrane permeability for potassium, sodium, calcium, and chloride, which results from the movement of these ions across it. Once the membrane is *polarized*, it acquires a voltage, which is the difference of potentials between intra and extracellular spaces.



The Na+/K+ ATPase pump creates a concentration gradient by moving 3Na+ out of the cell and 2K+ into the cell. Na+ is being pumped out and K+ pumped in against their concentration gradients. Because this pump is moving ions against their concentration gradients, it requires energy.





In most nerve and muscle cells, the ELECTRICAL ACTIVITY **or RESTING MEMBRANE POTENTIAL** is about -**70 mV**, negative on the inside; the potential is due mainly to the relatively large number of open K+ channels in the membrane.

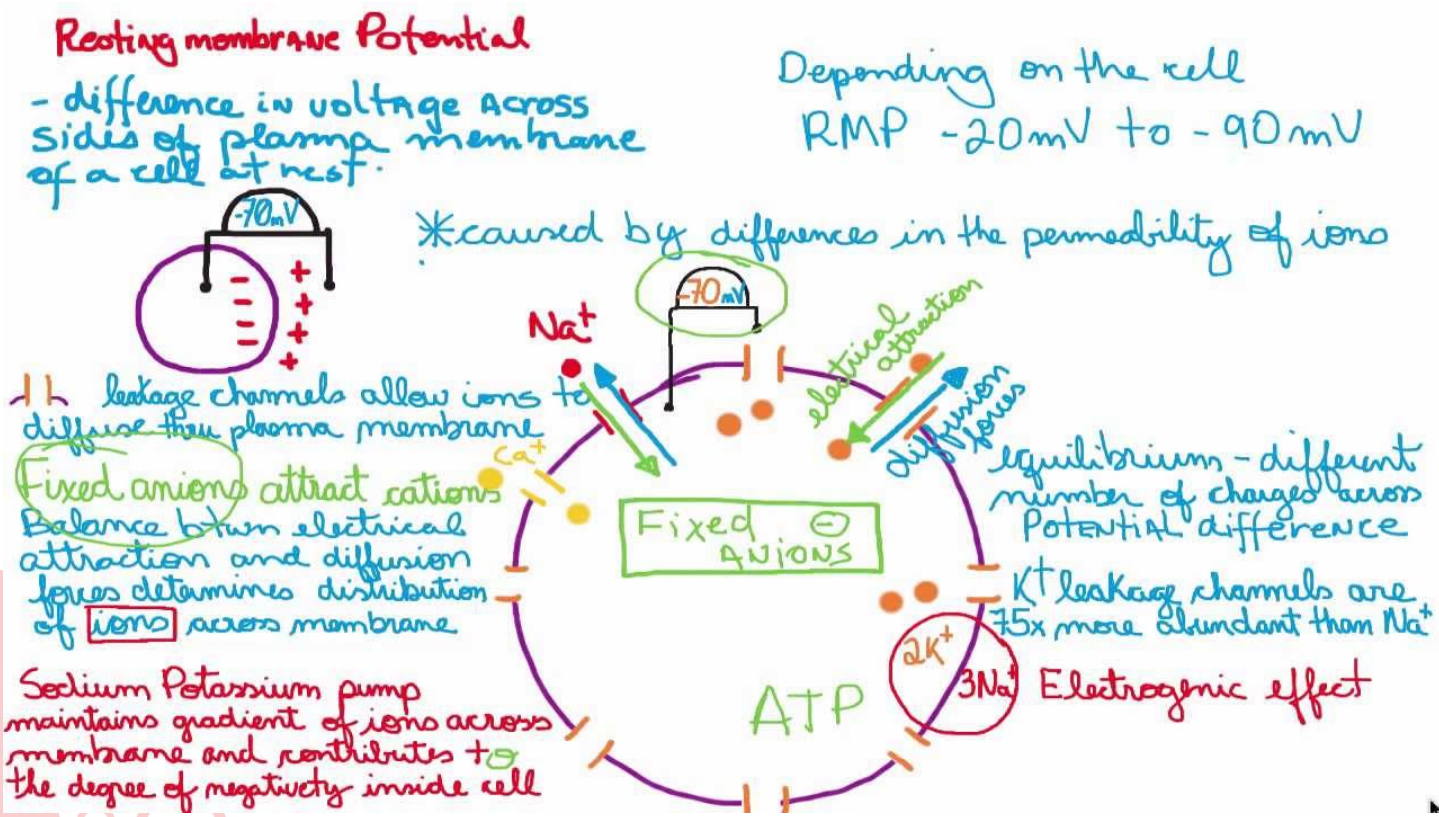
CELLULAR ENERGY

Resting Membrane Potential

ELECTRICAL ACTIVITY OF THE CELL MEMBRANES is needed as it controls most of the ionic channels for cellular activity and functions

The cell membrane contains protein channels that allow ions to diffuse passively without direct expenditure of metabolic energy. These channels allow Na + and K+ to move across the cell membrane from a higher concentration toward a lower. As these channels have selectivity for certain ions, there are potassium– and sodium– selective ion channels. All cell membranes are more permeable to K+ than to Na+ because they have more K+ channels than Na+

RMP is created by the distribution of ions and its diffusion across the membrane. Potassium ions are important for **RMP** because of its *active transport*, which increase more its concentration inside the cell.



Resting membrane potential varies according to types of cells;

Skeletal muscle cells: −95 mV
Smooth muscle cells: −50 mV
Erythrocytes: −12 mV
Astrocytes: −80/−90 mV
Neurons: −70 mV

EFFECTS OF LOSS OF CELLULAR ENERGY?

An inevitable consequence (if not a precursor) of aging is a slow, insidious decline in cellular energy levels. The outward effects often present as a sense of overall fatigue, depression, sexual dysfunction and a variety of diseases of aging.

The internal effect of a cellular energy deficit is a greater vulnerability to a host of degenerative diseases.

A chronic decrease in cellular metabolic energy is an underlying cause of many seemingly unrelated, age-related diseases. As humans grow older, systemic energy depravation can inflict devastating degenerative effects throughout the body. This fact is often overlooked by the medical establishment, yet persuasive scientific evidence exists that correcting chronic cellular energy production may enable many of the infirmities of aging to be prevented or reversed.

The prime reason cells lose their energy-producing ability is that the powerhouses of the cells - the mitochondria - become dysfunctional due to nutritional deficiencies.

Research has shown that carnitine, ribose, coenzyme Q10, acetyl l-carnitine and alpha-lipoic acid are critical to maintaining optimal mitochondrial function and supporting high energy production.

Dangers of cellular energy depravation should not be ignored. Every organ - from the heart to the kidneys to the skin; every process in the body - from walking to breathing to immune function to vision - is driven by energy produced in the cell's mitochondria.

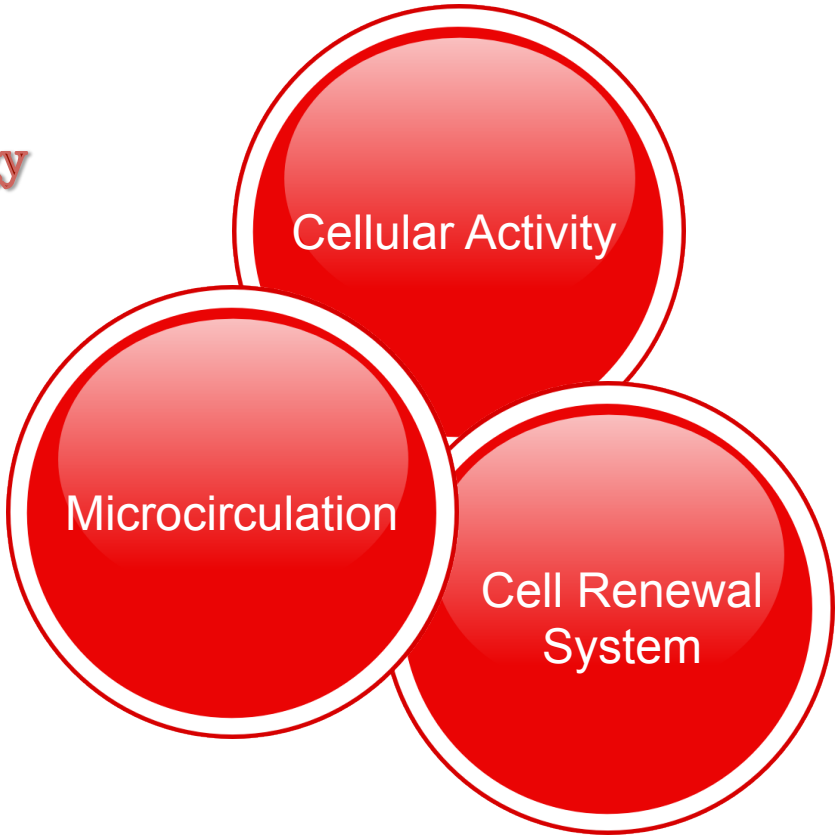
Proven ways exist to increase the effectiveness of cellular respiration and reduce the effects of aging. By following specific diet and supplement protocols, energy production can be restored and maintained.

Loss of CELLULAR ENERGY

Under the regenerative medicine perspective, we need to relearn and focus on the following ongoing complex molecular and cellular metabolism and activity and correct any irregular activities of the cell that are responding to various stimulus to maintain the illness – wellness continuum.

In recent years a lot of research is done in the field of regenerative medicine and finally able to summarize to the three key areas of concern that when corrected had actually healed the diseased or damaged cells.

Loss of Cellular Energy directly affects 3 KEY AREAS



The loss of cellular energy due to impairment of the mitochondrial health affects the 3 key areas as shown above. Mitochondrial health is mainly affected due to accelerated aging process greatly influenced by DNA Methylation and Oxidative Phosphorylation processes.

Our CRT is designed to reduce this processes by reenergizing and reactivating the mitochondrial health.

Dr Kals Ice Cubes Model

CRT
Cellular Regeneration Therapy

Origin of Diseases

due to '*loss of cellular energy*'

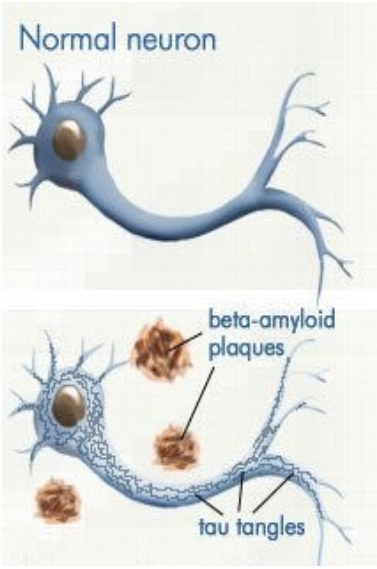
Extracellular Junk

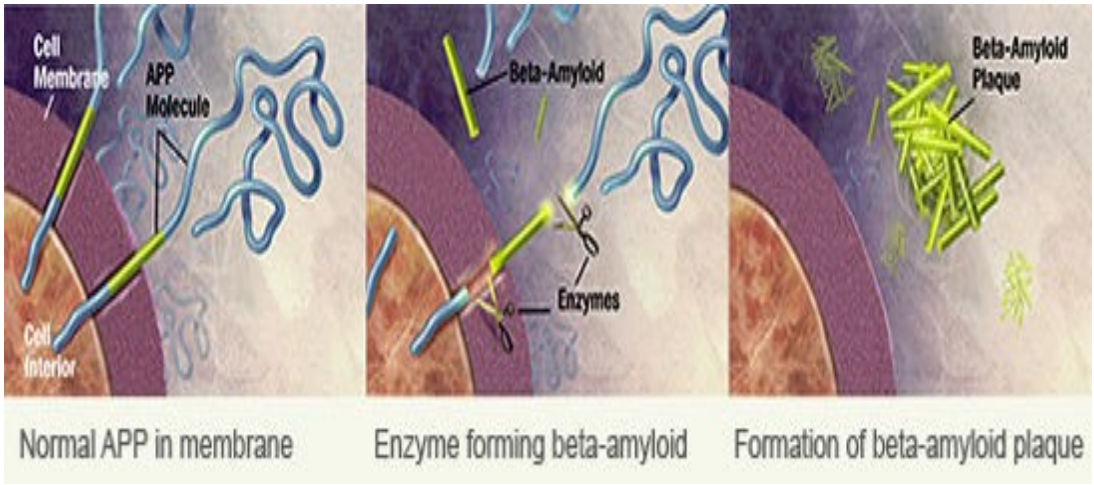
Removing junk from between cells

Extracellular junk is accumulations of sticky, malformed proteins that no longer serve their function, but instead impair cell or tissue function by their presence. Extracellular junk is different from extracellular cross-linking, which is a form of damage that occurs between structural proteins and impairs their ability to move. Most extracellular junk is termed "amyloid" of one variety or another.

The most well-known form of extracellular junk is beta - amyloid the stifling web-like material that forms plaques in the brains of patients with Alzheimer's disease, and also (more slowly) in everyone else's, and impairs cognitive function. There are also a variety of similar aggregates that form in other tissues during aging and contribute to age-related diseases, including islet amyloid in Type II diabetes and senile cardiac amyloidosis, which is a major contributor to heart failure.

Neurodegenerative Diseases
&
Metabolic Syndromes





Death Resistance Cells

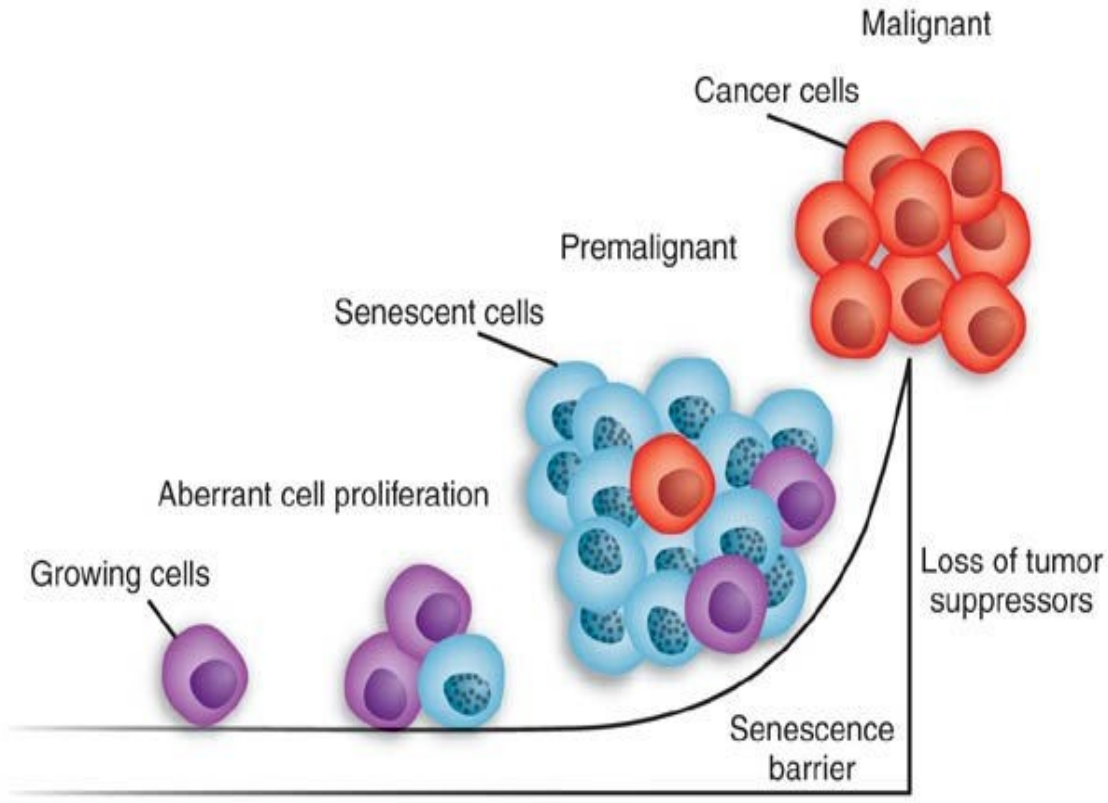
Removing dysfunctional cells

Our cells have built-in programming that sometimes veers them far away from their normal fate. Some of this programming watches out for conditions emerging within the cell that could put the rest of the body at risk; similar systems exist because the body no longer has need for the function that the cell normally fulfills. Pushing these cells to undergo such transformations is favored by evolution because it meets short-term needs, and having a few of these abnormal cells in the body for is nearly harmless. But the number of these cells in our tissues gradually rises over time, until by our fifth decade or so they begin to reach levels that are harmful to normal tissue function.

A – Classic Senescent Cells

The original and most well-studied sort of cells of this type are what are usually called senescent cells. Senescent cells began their existence as skin cells, or as related cells that normally play supporting roles in other organs, but were forced into an abnormal state where they lost the ability to divide and reproduce themselves as a protective response to some danger. For instance, the senescence program is activated in cells that undergo risky changes in their DNA expression that put them on a path toward becoming cancerous ; it is also activated in some cells involved in the wound response, to keep them from overstepping their bounds and generating an overgrowth of fibrous connective tissue.

But in addition to halting growth, senescent cells secrete abnormally large amounts of proteins that inflame the immune system and degrade the normal supporting tissue architecture. The relatively small number of such cells in a youthful tissue is so small as to be harmless, but after decades of accumulation, the number becomes large enough that their abnormal metabolic state begins to pose a threat to surrounding, healthy tissues. Larger numbers of senescent cells in a tissue make it more vulnerable to the spread of cancer,



Death Resistance Cells

Cellular Regeneration Therapy

Removing dysfunctional cells

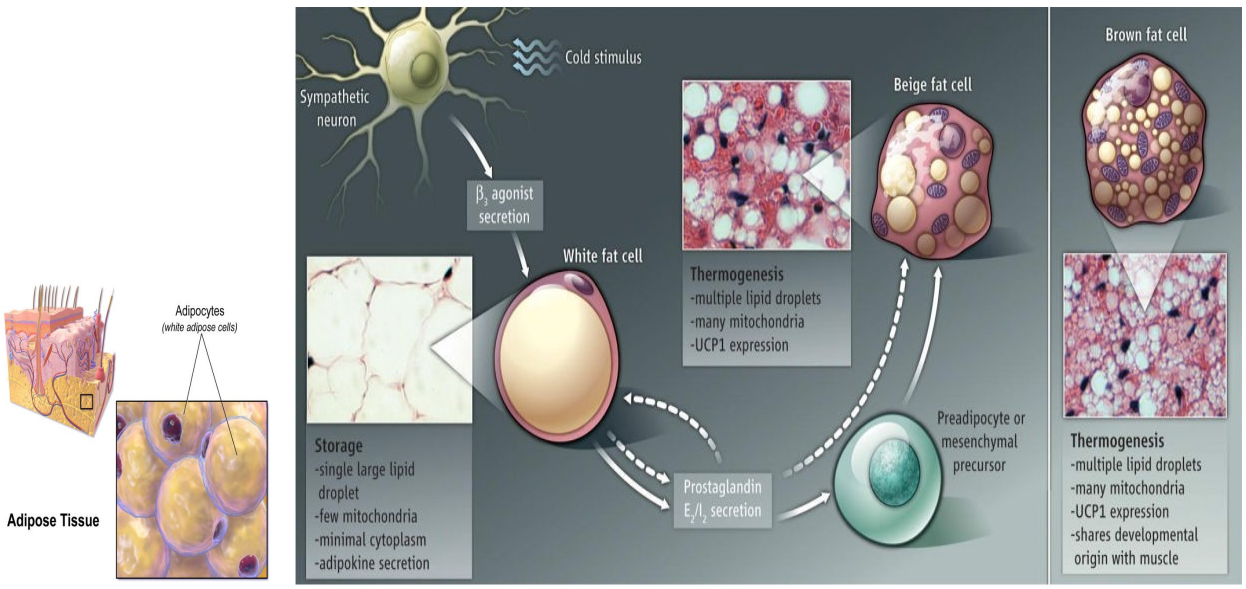
B – Cells in Fat Tissue

The reasons why fat tissue goes haywire during degenerative aging do not lie in the fat cells per se (adipocytes – the ones that store up excess Calories). Instead, the culprits are two other kinds of cells that reside in fat tissue; Preadipocytes and Visceral Adipose Tissue Macrophages [ATM's]

Preadipocytes are the precursor cells from which fat cells are formed. They begin to behave abnormally in all people during degenerative aging, whether they are slim or overweight: their expression of their genes becomes altered; they release more inflammatory factors; and they fail to reproduce themselves and to develop into mature fat cells, which may be one reason why the level of unhealthy free fatty acids circulating in the blood rises with age. Aging preadipocytes also develop a large droplet of abnormally-stored fat molecules within themselves, which may be part of why they and their progeny become more insulin resistant than the corresponding cells in young people's fat tissue.

The other unhealthy change is that occurs in fat tissue over time occurs in the so-called visceral fat – the fat tissue that surrounds the gut and liver. It's become widely understood that most of the metabolic harm that occurs as a result of obesity is the result of having too much fat in the visceral depot in particular (making people "apple- shaped" rather than "pear-shaped"). The degenerative aging process leads to a higher percentage of one's total fat being shifted into the visceral fat tissue, as well as into aberrant storage in the muscles and the liver.

The reason why excess visceral fat is so metabolically toxic is that it causes the accumulation of a kind of immune cells called Adipose Tissue Macrophages to multiply in the visceral fat tissue.. Like senescent preadipocytes, ATMs are also highly inflammatory cells, and their accumulation in visceral fat is probably one key reason why obese people become insulin resistant even when they have not yet undergone other degenerative aging changes. It is also possible that degenerative aging itself has effects on the function of ATMs that go beyond those attributable to the sheer mass of visceral fat and number of ATMs in the tissue



Obesity

Death Resistance Cells

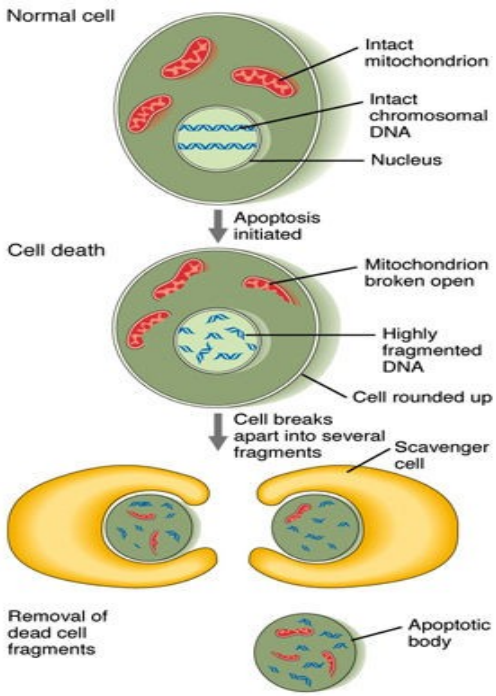
Removing dysfunctional cells

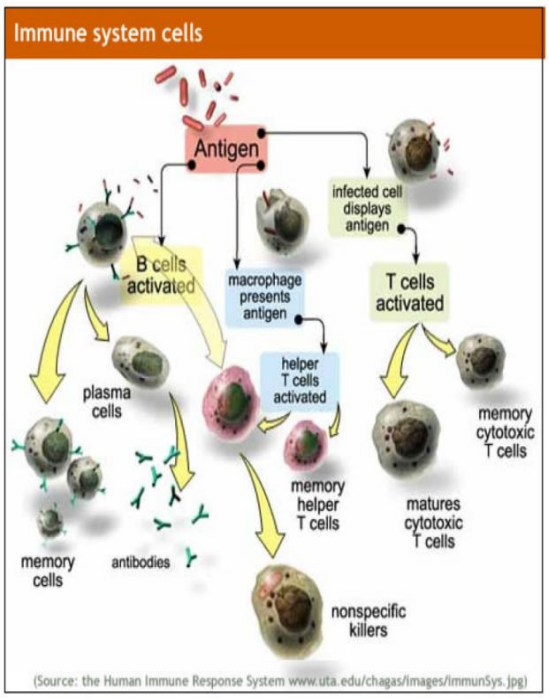
Cellular Regeneration Therapy

C – Immune Cells

The CD8+T-Cells [or Killer T-Cells] are a kind of immune cell that specializes in destroying cells that have been hijacked by viruses or by cancer. Killer T-cells first emerge as naïve cells that are out on the lookout for entirely new threats, but in order to do their job, they must assume a specialty, being trained by other immune cells to recognize, seek out, and eliminate a very particular threat. But while the total number of invaders that the body has encountered increases with every year of life, the total number of killer T-cells cannot: the sum total of all the different specialized cells, plus the naïve cells, is held constant over time. So an increase in the number of killer T-cells with one particular specialization can only come at the expense of a decrease in cells with different specializations (and naïve cells) .

As part of the degenerative aging process, an imbalance in the killer T-cell population occurs. Specific subsets of killer T-cells refuse to cull their numbers to make room for other subsets, and instead begin to occupy more and more of the limited immunological "space" – literally crowding out cells that are equipped to fight other infections. This crowding-out effect is thought to be one of the main reasons for the weakened immune responses of people as they age, which is why so many people over the age of 65 die or are hospitalized each winter by influenza or pneumonia, while younger people can bounce back after a couple of days in bed. This A – Classic Senescent Cells crowding-out effect is also one reason why vaccines are less effective in older people than in younger



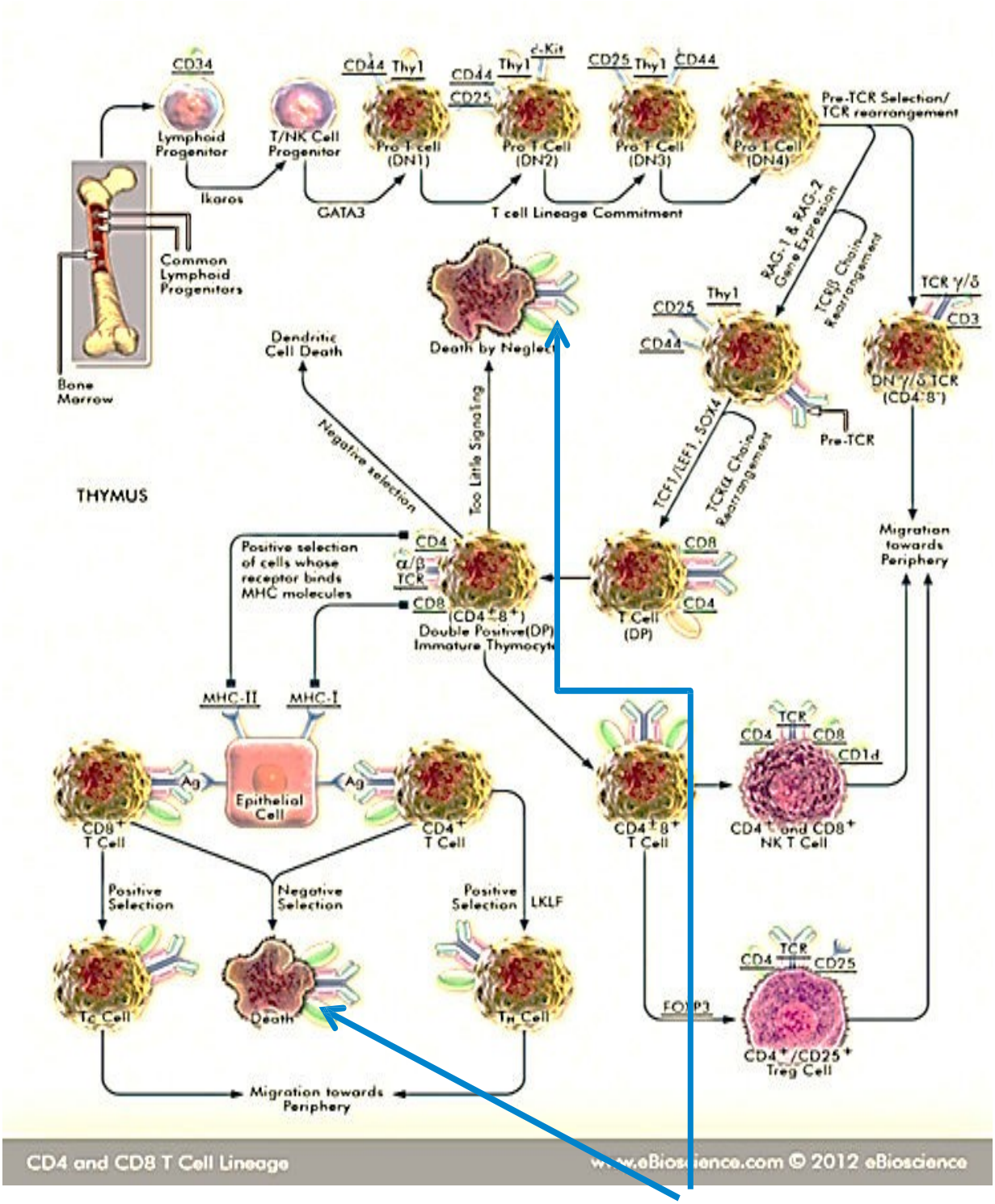


Poor Immune Response

Death Resistance Cells

Cellular Regeneration Therapy

Removing dysfunctional cells



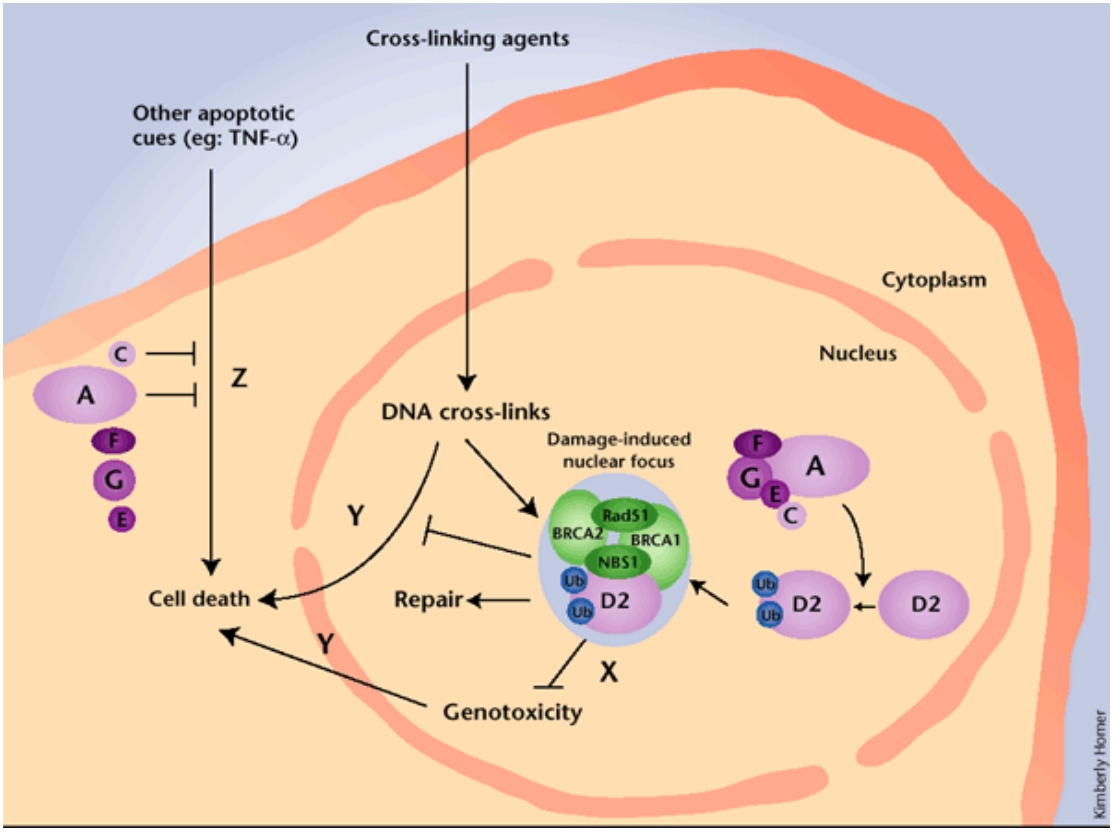
Resisting Cell Death Post Activation

Extracellular Crosslinks

Breaking Extracellular Crosslinks

Many of the major structural features of the body are built out of proteins that are laid down early in our life, and then more or less have to last for a lifetime: they are never recycled or replaced, or are only recycled over the course of many decades slowly. The healthy functioning of these tissues relies on these constituent proteins maintaining their proper structure. Such proteins are responsible for the elasticity of the artery wall, the transparency of the lens of the eye, and the high tensile strength of the ligaments, for example. But occasionally, blood sugar (and other molecules in the fluids in which these tissues are bathed) will react with these proteins, creating chemical bonds called crosslinks.

Crosslinks act like molecular "handcuffs," taking two neighboring proteins that were previously able to move independently of one another and binding them together, impairing their function in the same way that occurs to participants in a three-legged race. In the case of the artery wall, for instance, the crosslinking of strands of the protein collagen prevents them from spreading apart from one another to accommodate the surge of the pulse being driven forward by the pumping action of the heart. As more and more strands of collagen become cross-linked together over time, the blood vessels to become ever more rigid, leading to a gradual rise in systolic blood pressure with age. With the loss of the cushioning effect provided by free-moving collagen in the blood vessels, the force of the surge of blood that is driven into the arteries by the pumping action of the heart is carried directly to organs like the kidneys and the brain, damaging to the structures that filter our blood and that connect the functional regions of our brain, and putting us at risk of a stroke



Impairing Protein Functions

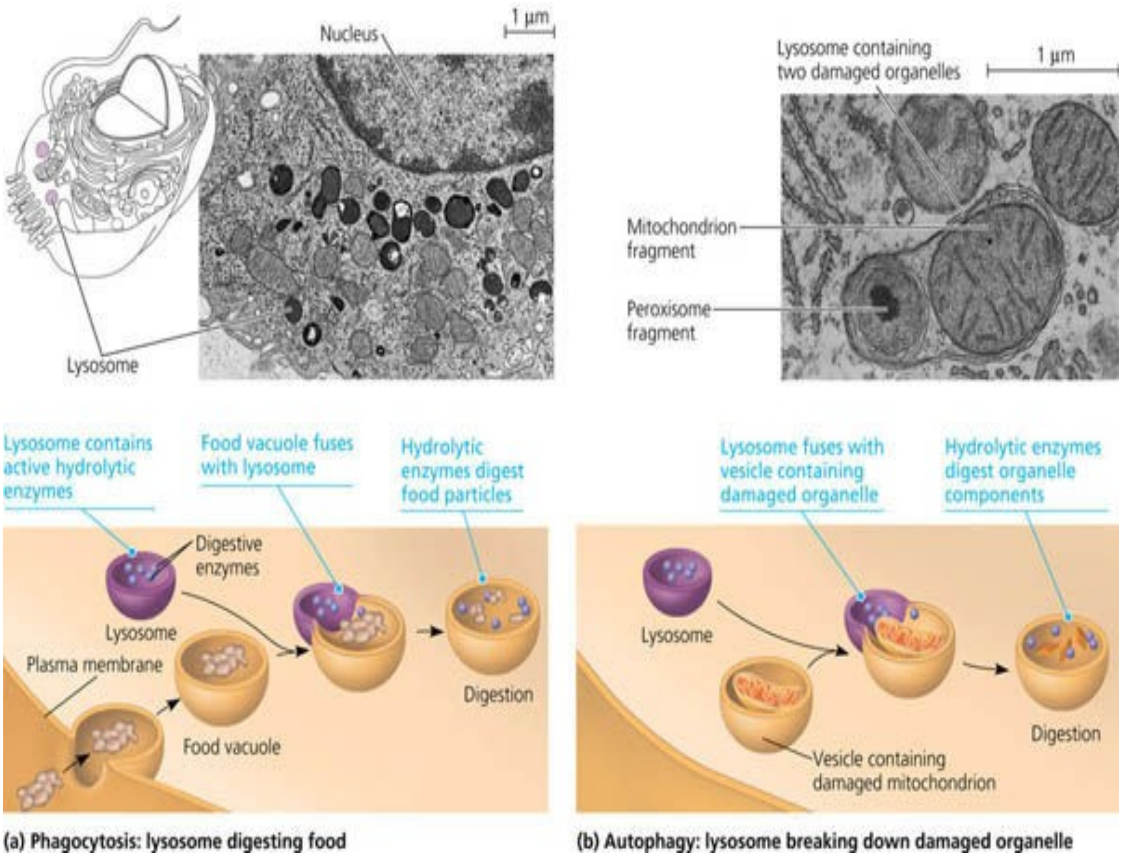
Intarcellular Aggregates

Clearing waste accumulation out of cells

The proteins and other constituents of our cells are all eventually damaged as the result of biochemical accidents that occur during normal metabolism, or simply outlive their usefulness. Cells have a variety of systems for breaking down and recycling such unwanted materials, allowing them to clear garbage out of the way and reuse the raw materials.

One such system is the lysosome a kind of cellular "incinerator" that contains the most powerful enzymes in the cell for breaking mangled molecules down into manageable pieces. However, sometimes these constituents are so badly fused together that not even the lysosome is able to tear them apart. And if something can't be broken down in the lysosome, there's nowhere else for it to go: it just stays there until either the lysosome disastrously ruptures, or the cell itself is destroyed. Over time, the material that the lysosome has been unable to break down accumulates inside of it, and eventually the rising buildup of such material begins to interfere with the lysosome's function.

This is an especially big problem for cells that have to last for a lifetime in our bodies, such as the cells that make up heart, the back of the eye, and nerve cells in the brain and elsewhere. And of course, when the cell's ultimate garbage disposal system starts malfunctioning, it impairs the function of the cell as a whole. And as more and more cells become dysfunctional over time, tissue function is impaired, and age-related disease sets in and the vicious cycle of accelerated aging process continues.



(a) Phagocytosis: lysosome digesting food

(b) Autophagy: lysosome breaking down damaged organelle

Increased Cell Toxicity

Mitochondrial Mutations

Preventing damage from mitochondrial mutations

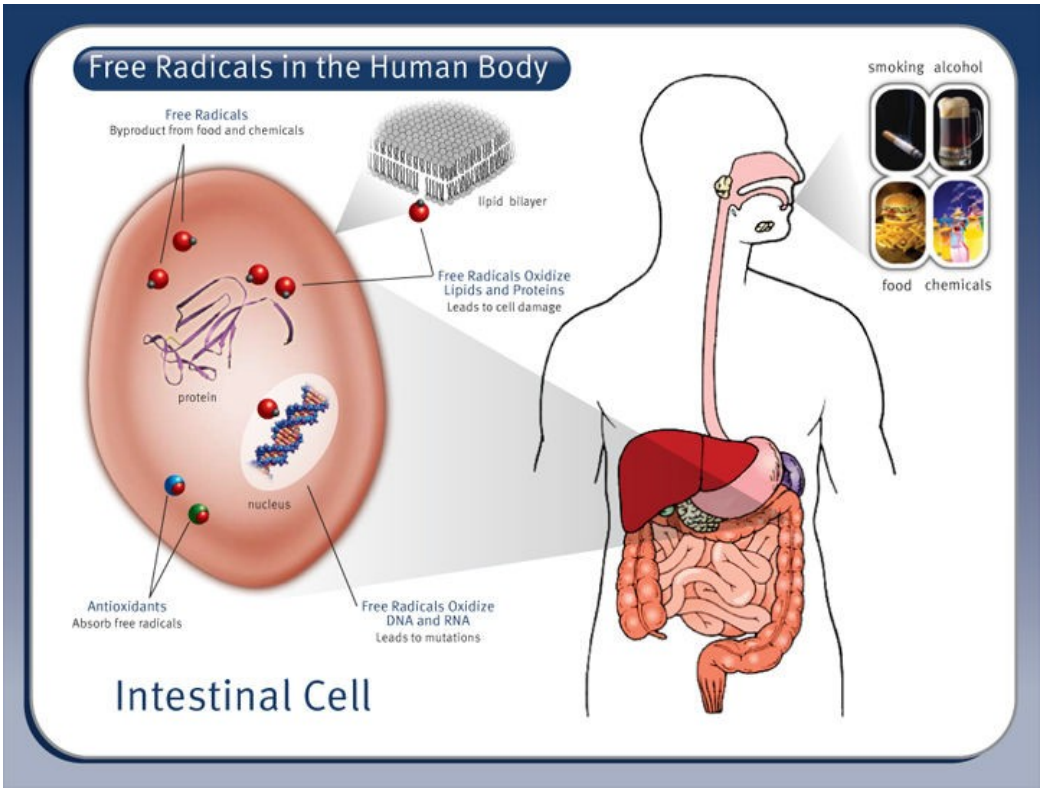
Cellular Regeneration Therapy

Mitochondria are the living machines within cells that act as their "power plants," converting the energy-rich nutrients in our food into ATP, the form of energy that directly powers biochemical reactions in the cell. Unlike any other part of the cell, mitochondria have their own DNA (mtDNA) separate from the DNA in the cell's nucleus, where all the rest of our genes are kept.

Just like real power plants, mitochondria generate toxic waste products in the process of "burning" food energy as fuel – in this case, spewing out highly-reactive molecules called Free Radicals which can damage cellular structures. And the mtDNA is especially vulnerable to these free radicals, because it is located so close to the center of its production. At worst, a free radical "hit" to the mtDNA can cause major deletions in its genetic code, eliminating the mitochondria's ability to use the instructions to make proteins that are critical components of their energy-generating system.

Lacking the components needed to produce cellular energy the normal way, these mutant mitochondria enter into an abnormal metabolic state to keep going -- a state that produces little energy, while generating large amounts of waste that the cell is not equipped to metabolize.

Perversely, the cell tends to hang onto these defective, mutant mitochondria, while sending normal ones to the recycling center, so if just one mitochondrion suffers a deletion, its progeny quickly take over the entire cell. Although this happens to just a few cells in our body, those few cells wind up doing disproportionate damage to the body as a whole, because they dump the waste that their mutant mitochondria generate into the circulation, poisoning their environment by causing oxidative stress to rise all over the body.



Damaged DNA Synthesis

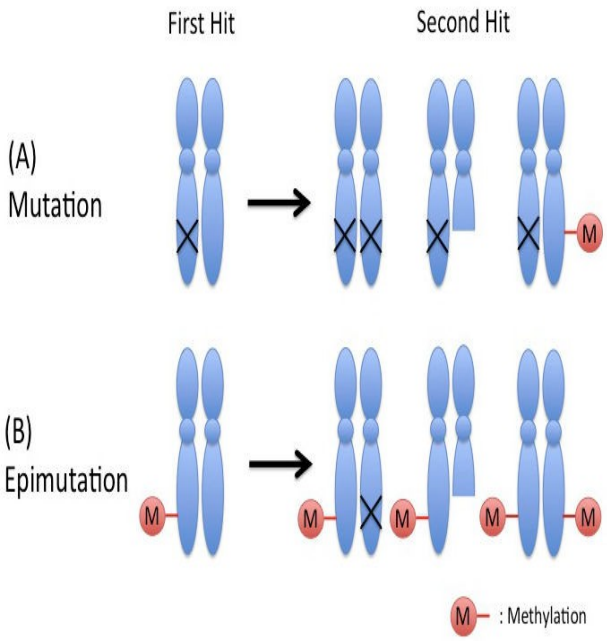
Nuclear Mutations

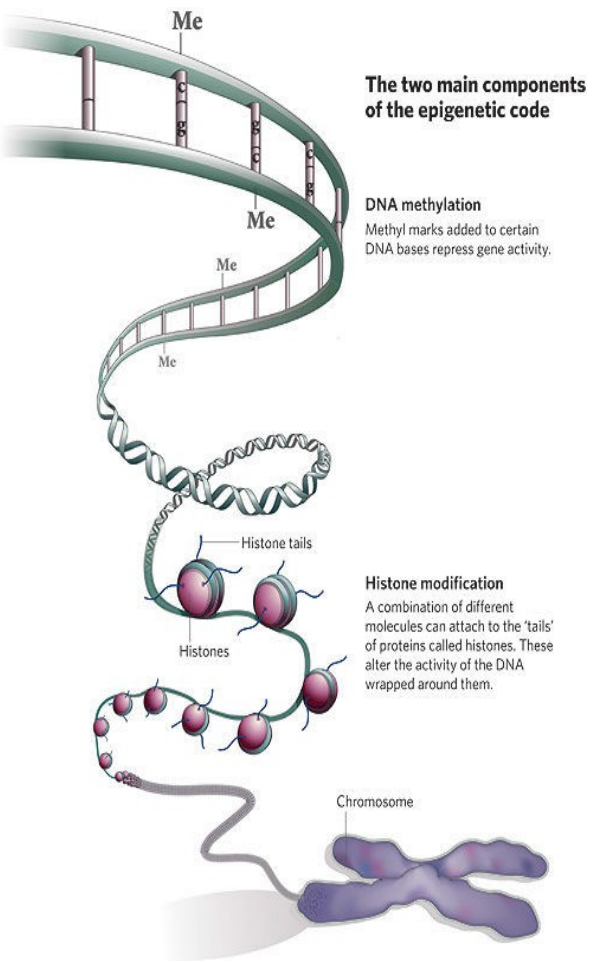
Making cancerous mutations harmless

Cellular Regeneration Therapy

Two types of damage accumulate in our genes as we age: mutations and epimutations. Mutations are damage to the DNA sequence itself, whereas Epimutations are damage to the "scaffolding" of that DNA, which controls how and when genes get turned on in the cell. For practical purposes, both mutations and epimutations ultimately harm us in the same way: by causing abnormal gene expression, whether it's by increasing or decreasing the amount of a protein encoded by a gene being produced, by altering in the conditions under which that production is activated, or by altering the structure and function of the ensuing protein.

So what kind of harm can the changes in gene expression resulting from epimutations cause? The one that most people know about is cancer, which is the result of a series of epimutations that happen in sequence in the cell, leading to its uncontrolled growth. Other kinds of epimutations also occur in our cells over time, and some scientists have worried that these non-cancer-causing epimutations might also contribute in different ways to age-related disease and disability.





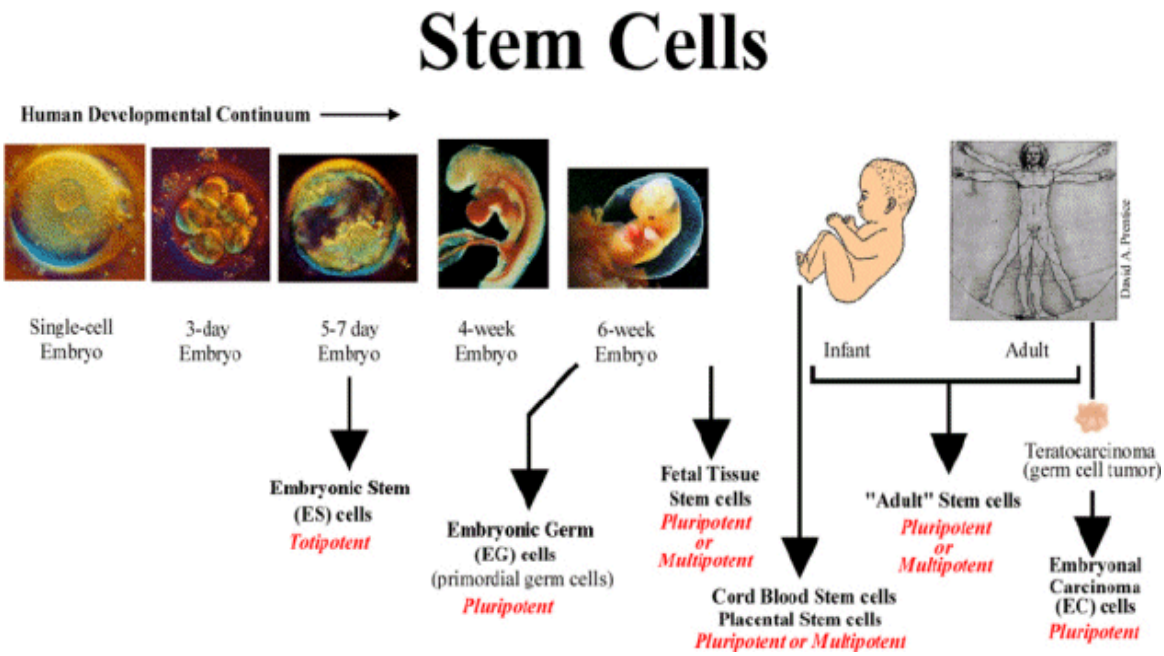
Abnormal Gene Expressions

Cell Loss and Atrophy

Replacing Lost Cells

Every day, our cells are damaged by both tiny molecular-level insults and by obvious trauma. Some of these damaged cells are repaired, but others are either destroyed, or forced into a dysfunctional 'senescent' state where they can no longer divide, or commit 'cellular suicide' (apoptosis) for the greater good of the body. Some of the lost cells are replaced by the pools of specialized, tissue – specific stem cells, but the degenerative aging process makes these stem cell pools less effective at repair over time.

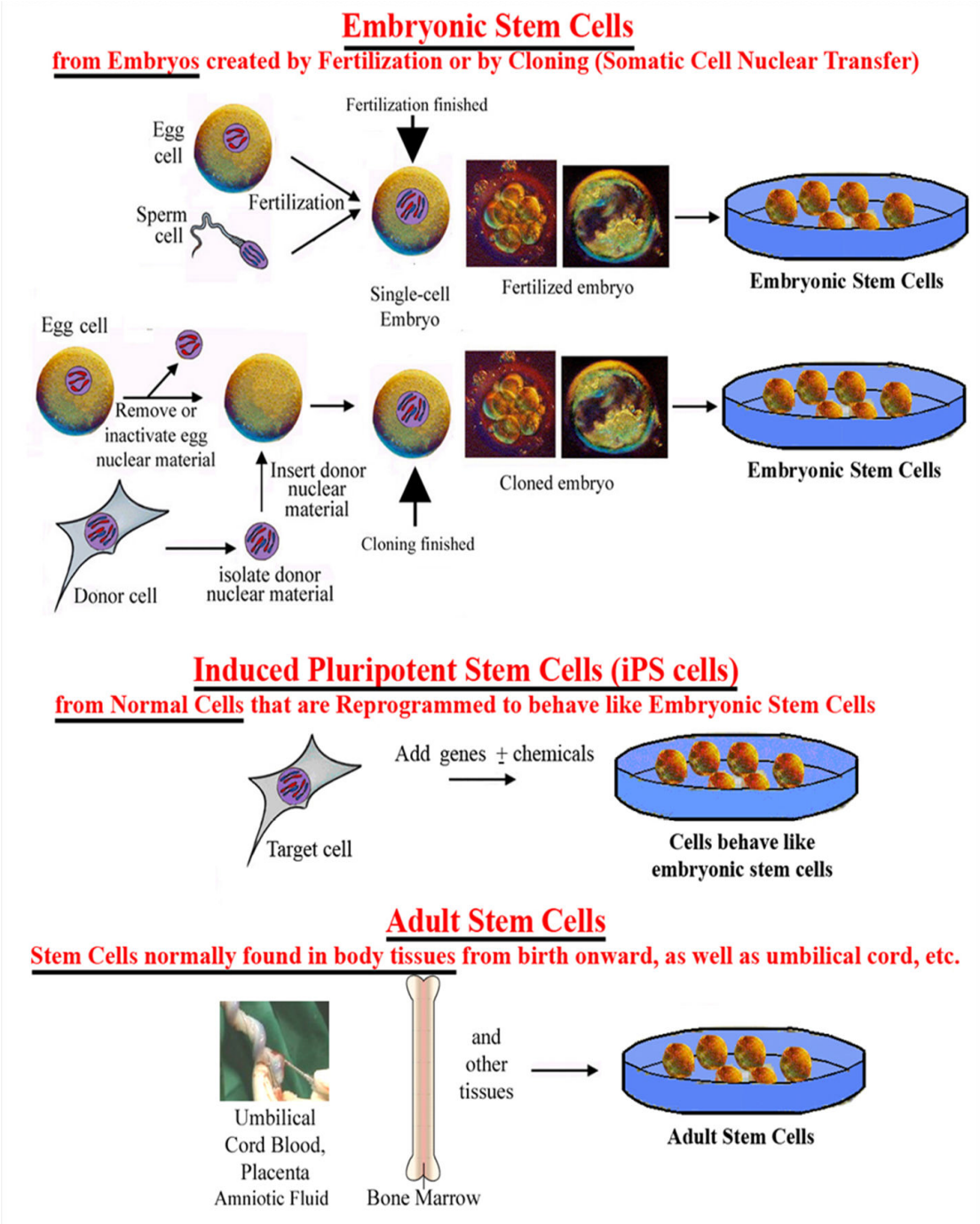
The net result is that over the course of many decades, long-lived tissues like your brain, heart, and skeletal muscles begin to progressively lose cells, and their function becomes increasingly compromised. Muscles weaken, and don't respond properly to exercise or injury. The brain loses neurons, contributing to cognitive decline and dementia, as well as to loss of control over fine muscle movements (a process that ends in Parkinson's disease). The thymus – the gland in your breastbone where a major class of immune cells mature – shrinks, leaving you more vulnerable to infectious disease as fewer fresh immune cells are produced.



Depletion with Aging

Cell Loss and Atrophy

Replacing Lost Cells



Delivery Systems

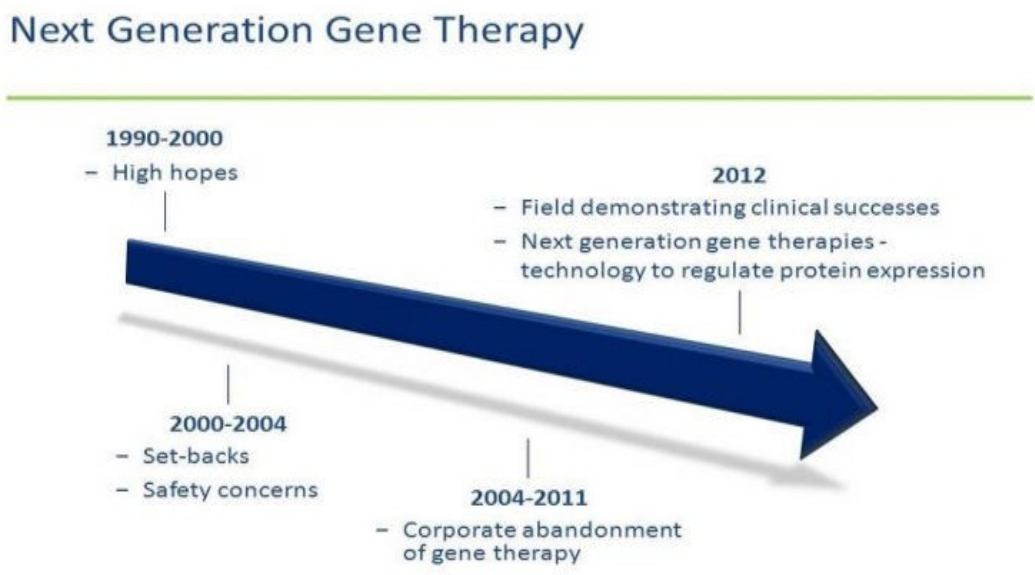
Building In Rejuvenation Biotechnologies

The workhorse molecules of many rejuvenation biotechnologies are not classical small-molecule drugs, but proteins – and protein based therapies face special challenges. For one thing, proteins usually can't be given in pill form, but have to be injected, which can be inconvenient and cause discomfort. Also, most proteins are quickly degraded by the cell's recycling machinery and other metabolic processes, making it very difficult in many cases to deliver the quantity of protein required to have a therapeutic effect, and to keep the local tissue concentration up to therapeutic levels. And there are similar problems with delivering RNA, the "working copies" of genetic instructions that tell cells how to build proteins on their own.

An additional challenge comes from the sheer number of different protein- (or RNA-) based therapies, each of them removing or repairing a different kind of aging damage, that will be required to fully maintain the healthy vigor of youth – and to keep the full range of age-related diseases and disabilities at bay. If each of these many protein-based therapies had to be delivered via periodic injection, the complete panel of rejuvenation biotechnologies could entail an impractically-frequent regimen of trips to rejuvenation clinics to keep up with a person's full schedule of regeneration sessions.

Even more challengingly, the sheer size of proteins would in many cases make it difficult to get protein-based therapies into the tissues where they're needed to repair and rejuvenate the local cellular and molecular structures. The most important such case is the brain, which is protectively shielded from the rest of the body's circulation; this shielding will likely present a barrier to the enzymes that will be needed to clear harmful wastes out of brain cells, as part of reversing age-related cognitive decline and preventing neurological diseases such as Alzheimer's and Parkinson's.

As an alternative to delivering therapeutic proteins (or RNA) via pills or injections, many rejuvenation biotechnologies can be produced directly in the very tissues where they're needed. There are two primary ways to do this: transplantation of cells and tissues that have been engineered with the ability to produce the needed proteins built into them, and somatic gene therapy to engineer that ability into our existing tissues.



Delivery Systems

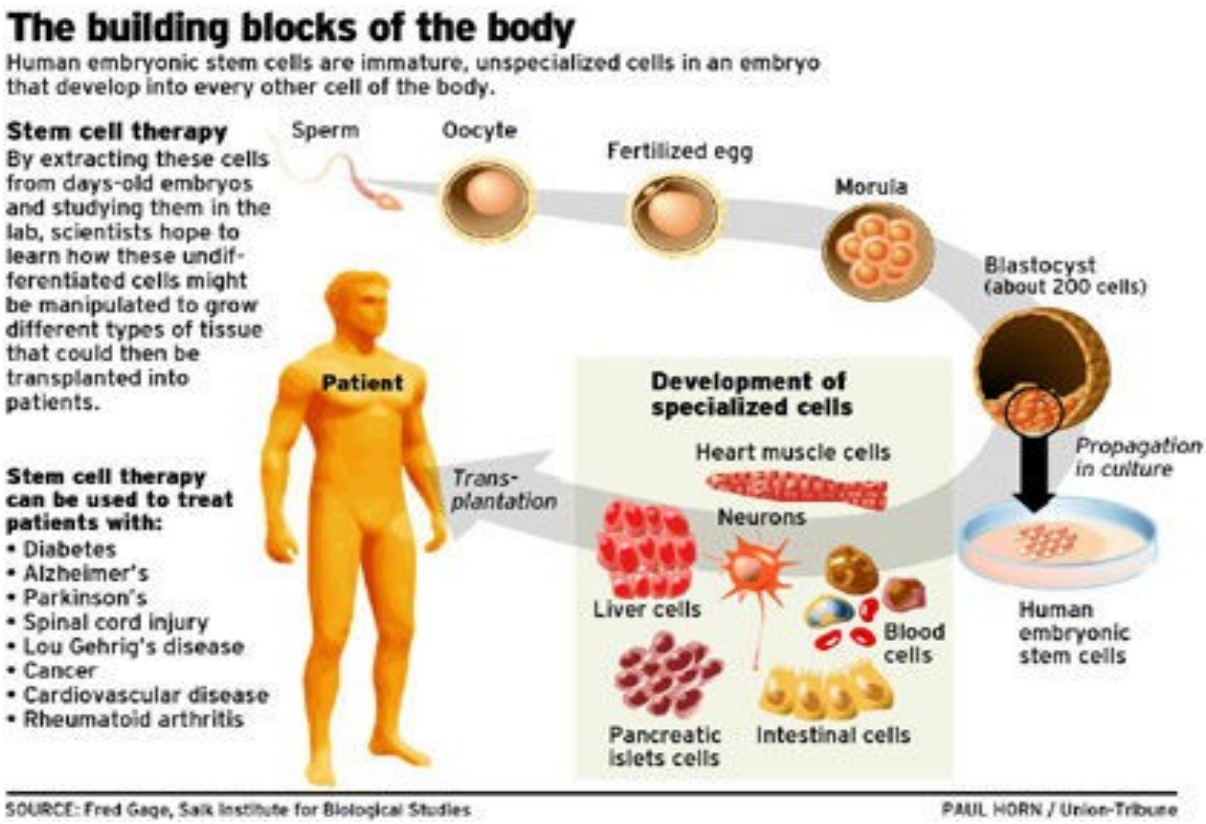
Building In Rejuvenation Biotechnologies

A – Transplantation

In any case where rejuvenating a tissue already requires replacing cells, tissues, or organs lost or irreparably damaged by aging, it will be a relatively simple matter to build into them the ability to express therapeutic proteins before transplanting them into the patient. This will be especially easy and uncomplicated in cases where we are able to use cells that can be taken out of a person, nudged to multiply in a lab, and then transplanted back in: such cells will be engineered to make the needed proteins when they are first taken out of a patient, and then all of those original cells legions of progeny will inherit the therapeutic genes and be delivered into their target tissues in the act of being transplanted back into the original patient/donor. In fact, in those tissues (like the skin, the blood, or the gut) that continuously lose cells are therefore continuously repopulated by pools of stem cells, this approach will be used to gradually upgrade the entire tissue, until all of its cells are fortified with the ability to make therapeutic proteins.

But in many cases, the cells in a tissue needing therapeutic proteins will not be amenable to this simple cycle (extract the cells harmlessly; build in the ability to express therapeutic proteins; nudge the enhanced cells to multiply; return the enhanced cells to the tissue en masse). Some of the target cells will be lacking in the tissue, or too few or too difficult to safely harvest; some will be unable to divide; and some are the very cells that will need to be therapeutically removed, because they are stuck in a permanent abnormal metabolic state that is harmful to the surrounding tissues.

In such cases, we can create the needed cells and tissues using the rapidly-progressing techniques of cell therapy and tissue engineering– a key rejuvenation biotechnology in its own right, which in these cases can serve an additional therapeutic purposes.



Delivery Systems

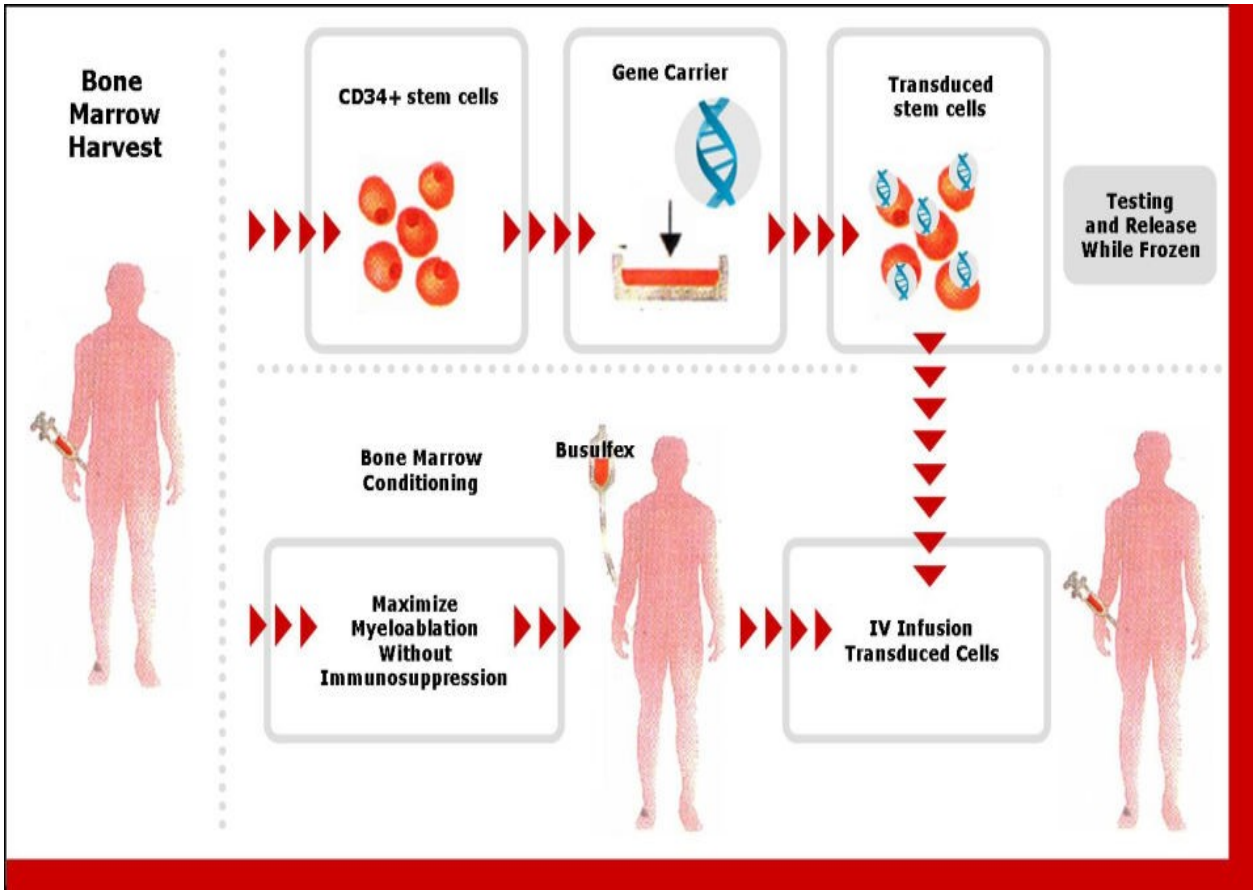
Building In Rejuvenation Biotechnologies

B – Somatic Gene Therapy

While it is convenient to be able to engineer new therapeutic properties into cells and tissues while they are in a Petri dish or bioreactor vat and then transplant them into a person who needs both cells and therapeutic proteins, several regenerative therapies for the diseases and disabilities of aging will almost certainly require that we introduce new genes into the cells that are already in the tissue and are not going to be replaced – so-called somatic gene therapy. This is a much harder task, for several reasons.

First, when scientists engineer a new gene in a cell or tissue in isolation in the lab, they can check whether the therapeutic gene was properly taken up by the cell (and that nothing harmful happened in the process) before the cells are transplanted into the recipient. By contrast, the existing techniques of somatic gene therapy are somewhat scattershot in terms of which and how many cells they modify in a target tissue, and in the process, they sometimes cause unwanted alterations in the genes surrounding the new, therapeutic genes.

These challenges have so far relegated gene therapy to a highly experimental and risky procedure for patients, which has held back its enormous potential to cure diseases caused by inherited mutations, such as sickle cell anemia, cystic fibrosis, Tay-Sachs disease, and even the BRCA1 mutation that gravely increases the risk of early breast and ovarian cancers



Intense R&D

CRT

Cellular Regeneration Therapy

Clinical Application Protocol

Phase 1 – Cellular Optimization

Phase 2 – Cell Renewal

Phase 1 - Cellular Optimization

How It Works?

Cellular Regeneration Therapy

There are only a few molecules vital for the function of cells to maintain human life. – Proteins, peptides, and your body's specific DNA. These biological structures carry all the information necessary for life. DNA is simply a matrix; it is a molecule that, by itself, performs no function. Only when a relevant protein or peptide connects with a corresponding segment of the DNA, will it stimulate the synthesis of specific proteins. This interaction between the two is the key to life! By stimulating the action potential of the mitochondria using mtCPP 1,8 and 9 will optimize the cellular energy of the cell for full functionality.

Our Cellular Regeneration Therapy (CRT) Phase 1 – Cellular Optimization Protocol has developed specific protocols utilizing Cell Penetrating Peptides mainly mtCPP 1,8, and 9 to treat variety of metabolic, neurological and autoimmune diseases, along with cirrhosis and various forms of cancer. is is accomplished through the introduction of specific proteins, peptides, growth factors and neurotropic factors that are carrying encoded information to a specific type of receptor, on the membrane of a targeted cell type.

The signals carried by these families of molecules passes through the nucleus of the cell to reach the DNA. These signals stimulate the DNA through a process know as protein transcription. Transcription begins with a bundle of factors assembling at the start of a gene to process the protein's information. e double helix is unzipped and one of the strands is copied. e strand that is produced, as the DNA is unzipped is called RNA, which is a close chemical cousin of DNA.

When the RNA is complete it begins to extend to the outer part of the cell. When this occurs, all the components of a molecular factory, called a Ribosome, lock together around the RNA. It then translates the genetic information within the RNA into a string of amino acids that will become a protein.

The molecular information is passed to the cell through signal transduction pathways, which then either stimulate, regulate or inhibit various functions of the cell, in order to produce a series of intercellular reactions. The production of these specific protein families is fundamental to treating the targeted disease. The functional protein, when necessary, exits the cell and inserts itself into neighboring cells that are unable to synthesize functional proteins, allowing the organ or tissue to begin the regeneration process. Continued applications of these molecules will progressively boost the repair process and promote stabilization within the targeted cell cycle.

(Phase 2 - Cell Renewal)
How It Works?

Moving Stem Cells Into The Clinic

Clinical translation is the process used to turn scientific knowledge into real world medical treatments. Researchers take what they have learned about how a tissue usually works and what goes wrong in a particular disease or injury and use this information to develop new ways to diagnose, stop or fix what goes wrong. Before being marketed or adopted as standard of care, most treatments are tested through clinical trials.

Have stem cells already been used to treat diseases?

Yes, doctors have performed stem cell transplants, also known as bone marrow transplants. In stem cell transplants, stem cells replace cells damaged by chemotherapy or disease or as a way for the donor's immune system to fight some types of cancer and blood-related diseases, such as leukemia. These transplants use adult stem cells or umbilical cord blood.

About Stem Cells

Stem cells are the foundation of development in plants, animals and humans. In humans, there are many different types of stem cells that come from different places in the body or are formed at different times in our lives.

Stem cells are defined by two characteristics:

- They can make copies of themselves, or *self-renew*
- They can *differentiate*, or develop, into more specialized cells

Our Cellular Regeneration Therapy (CRT) Phase 2 – Cell Renewal Protocol utilizes the Umbilical Cord Blood derived Mesenchymal Stem Cells from the cGMP laboratory in Malaysia that has numerous clinical studies published with proven safety and efficacy standards.

Umbilical Cord Blood derived Mesenchymal Stem Cells (UC-MSC)

UC-MSC is currently the most widely used stem cells due to its zero-immunogenicity that enables us to use them for our stem cell therapy.

CRT - PROTOCOL

Cellular Regeneration Therapy

Based on the case studies and paper publications from multi-centered clinical research done world wide by numerous research scientists who are leaders in the field of regenerative medicine, we have noticed a growing number of successful treatment protocols established over the past ten years using cellular therapy and stem cells. We have taken only clinically proven and established safety standards of the cellular therapy protocols for our study.

Using the knowledge obtained from this studies, we have designed our own therapeutic protocol and have been successfully treated many medical conditions that failed to respond to conventional medicines or drugs in the past three years.

Our Treatment Protocol –

We have established our treatment protocol as two phase therapy;

a. ***Cell Penetrating Peptide Complex*** – focuses on protein and peptide based compounds to optimize cellular function.

b. ***Stem Cells (UC-MSC)*** focuses on stem cells to replenishing and replacing damaged cells.

PHASE 1 Therapy –

Cell Penetrating Peptide Complex with proprietary formulated small molecules will be infused intravenously to the recipient patient on twice per week regime for five weeks.

PHASE 2 Therapy –

Umbilical cord derived Mesenchymal Stem Cells will be infused intravenously to the recipient patient only once on the sixth week.

Thus the full cycle of CRT is completed.

CRT

Cellular Regeneration Therapy

CRT is ideally given and suggested for all conditions that failed conventional treatment. It has more than 50 active cell penetrating peptides and small molecules required by our cells for its cellular metabolism, cytoplasmic substance formation, mitochondrial reactivation, cell membrane receptor specific resensitization, free radical neutralization, reduces reactive oxygen species replication by modification of oxidative process mechanics and restabilizes the cellular specific activity by inhibiting the genetic modification of the mitochondria.

Our patients that have improved post CRT for the following medical conditions –

Fact Sheet

Molecules

Proteins – are molecules composed of linear chains of amino acids. e word protein comes from the French word "protéine" and this from the Greek "π ρ ω τ ε ι □ ο ς (proteios)", meaning: "prominent, premium". Proteins fold acquiring a three-dimensional structure that allows them to perform thousands of functions.

Peptides – (from the Greek "π ε π τ ο □ς", peptós di- gested) are a class of molecules formed by the union of several amino acids through peptide bonds. Peptides, as well as proteins, are present in nature and are responsible for a large number of functions. e union of a small number of amino acids results in a peptide, and when there is a union of more than 100 amino acids it results in a protein.

Oligopeptide: from 2 to 10 amino acids
Peptide: between 10 and 100 amino acids.
Protein: more than 100 amino acids.

Cell-penetrating peptides (CPPs) –

CPPs are short peptides with a sequence of up to 30 amino acids that are able to cross the cellular membrane and transport bioactive cargo into cells in a nontoxic and efficient way. These peptides are amphipathic, carry a positive charge, and show both hydrophilic and lipophilic properties.

Mitochondrial Cell-penetrating peptides (mtCPPs) -

mtCPPs are tetrapeptide, and its sequence was designed to display at least 3 characteristics known to be important to target the mitochondria: positive charge, lipophilicity, and alternating aromatic residues and basic amino acids.

Growth Factors

Growth Factors, Neurotrophic factors, and Fibroblast Growth Factors are naturally occurring, regulatory molecules.

Growth factors are proteins that bind to receptors on the cell surface with the primary result of activating cellular proliferation and / or differentiation. Many growth factors are quite versatile , stimulating cellular division in numerous different cell types , while others are specific to a particular cell type.