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Research Article

Intelligible Intelligence of Immune Neuroplasticity: Influence of Drugs and Vaccines on Mitochondria and Genomic Stability in Disease Processes

Mahin Khatami

Inflammation, Cancer and Vaccine Sciences, Retired, NCI/NIH, Bethesda, MD.

***Corresponding Author:** Mahin Khatami, PhD, Inflammation, Cancer and Vaccine Sciences, Retired, NCI/NIH, Bethesda, MD.

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Abstract:

Background-Aim. Efforts to understand the intricate molecular and electrochemical dynamics of immune neuroplasticity, which safeguard health, have highlighted a significant public health shift in 20th century toward induced diseases. In the United States, prevalence of cancers, autoimmune and neurodegenerative illnesses rose dramatically, reaching rates of 50-80%, for younger and older populations, compared to the much lower rates of 1-5% for congenital or inherited conditions in last century. After the 1986 National Childhood Vaccine Injury Act (NCVIA) granted vaccine manufacturers immunity from liability, vaccine promotion surged, particularly targeting fetuses, newborns, and immunocompromised individuals. This perspective suggests that the rise in induced diseases to be closely tied to significant increase in childhood vaccination.

Material-Methods. Human birth is viewed as a continuation of fetal development outside the womb, marked by a state of vulnerability or 'helplessness'. During first two years of life, as newborn is exposed to atmospheric oxygen, body's organs and immune system, including mitochondria, continue completing their development. Defence mechanisms, governed by biological rhythms, require differential bioenergetic for ATP production from mitochondria and cytoplasm to maintain health. An effective immune system balances tumoricidal (Yin, apoptosis, degeneration) and tumorigenic (Yang, wound healing, regeneration) processes, characterized by self-limiting and protective nature of acute inflammation. Validity of heavily focusing on genetics or 'heredity' as origin of diseases (eg, cancers, autoimmune, and neurodegenerative disorders) is also being challenged.

Results. Autonomic and intelligible intelligence of immune neuroplasticity can be affected, weakened, or damaged by both external and internal factors. These include repeated infections, genotoxins, low-level carcinogens, defective cells or proteins, high-energy electronic devices (wearables), aging, substance abuse, or pathogen-specific vaccines and ingredients or synthetic-engineered and modified mRNA-DNA spike proteins encased in lipid nanoparticles.

Conclusions: Over-vaccination of unborn babies, newborns, infants, or those with compromised immunity might disrupt immune neuroplasticity, harm mitochondrial and histamine functions, and activate genes that could lead to abnormal expression and co-expression of receptors, surface molecules, metabolites or mediators. Imbalanced tissue bioenergetics and immune responses likely trigger or worsen diseases. Permanent loss of immune neuroplasticity in ancestors and parents is believed to play a major role in onset of diseases, challenging the concept that genetics alone is origin of diseases. Deep understanding of immune neuroplasticity could lead to accurate risk assessment tools/formula, universal safe vaccines, and strategies to reduce environment-gene-immune interactions, toward preventing diseases and fostering a healthier society.

Call to Action: The author calls on policymakers to reinstate liability laws for vaccine manufacturers.

Key words: Autism; inflammation; bioenergetics; cancer; covid-19; electromagnetics; foetus; genomics; histamine; immune disorders; inheritance; intelligence; mitochondria; pathogens; spike protein; sympathetic and parasympathetic; trophoblast; vaccines; yin-yang

Introduction

If there is no enemy within, the enemy without can do us no harm. Winston Churchill.

Human immunity is an incredibly complex system that has adapted over millions of years to handle diverse, hostile and challenging environments. It operates as a dynamic network of electrochemical-electromagnetic and molecular signals, influenced by biological clock (biorhythms) and biofeedback systems. Endogenous oscillators within these systems are crucial for health, aging, and disease processes. From the conception and fetal development to adulthood and aging, electrochemical signal communication pathways across organ systems shape human immunity [1-4].

Amid debates and controversies about whether inflammation prevents or triggers diseases like cancer, the author highlights that after birth, inflammation and mitochondria are crucial for either maintaining health or causing disease. The article discusses how disruptions in tissue and organ bioenergetics (biological rhythms) at different life stages, by drugs and pathogen-specific vaccines (eg, MMR, pneumonia, tetanus, HPV, flu, hepatitis, shingles, or synthetic COVID mRNA or DNA spike protein vaccines in lipid nanoparticles), engineered crops, processed foods, environmental chemical or biological hazards, including high-energy producing electronic devices (wearables) might lead to mitochondrial and immune dysfunctions. These immune disruptors (antigen overload) could contribute to the development and progression of acute and chronic inflammatory or infectious diseases, as well as carcinogenesis and angiogenesis [4-7].

In this article, the author provides evidence that the signal transduction mechanisms in multi-organ systems shaping human immunity continuously evolve from conception through fetal development, birth, adulthood, and aging. The intricate molecular network of autonomic immune neuroplasticity (sympathetic-parasympathetic) regulates biological processes like degeneration, regeneration, and recycling to protect the body from internal and external threats to its survival [4-7].

An overview of literature [7-59] in biomedical fields of developmental biology (embryogenesis), infections, toxicology, genetics, and drug or vaccine abuse highlights various manifestations of health conditions. The illnesses range from acute, chronic and age-related problems, site-specific cancers and immune disorders of varying severity (eg, allergies, asthma, anaphylaxis, autism, diabetes, cardiovascular problems, muscular dystrophy, blood clots, neurodegenerative and autoimmune diseases), including vaccine-related injuries. Advanced disease outcomes typically fall into three interconnected categories:

a) vascular and lymphatic channel disorders,

b) tissue necrosis, and

c) tissue growth.

Central to the above disease categories are mitochondrial dysfunction (tissue bioenergetics or biological rhythms) and vascular complications, which disrupt immune response profiles and may cause defects in nano-electrochemical-electromagnetic spacing critical for immune-metabolic-neuronal-hormonal-genetic communications for the proper functioning of the body's multi-organ systems.

A review of Burnet's insightful theory of immune surveillance [15] and related literature prompted me to re-analyze and expand our original and accidental studies that were established in 1980s, at the University of Pennsylvania on experimental models of acute and chronic inflammatory diseases that resulted in tumorigenesis and angiogenesis [4-7,10,21-24, 30,31,50-53]. These efforts led to new definitions of acute and chronic inflammation and the sympathetic-parasympathetic nature of autonomic immune neuroplasticity in health and disease [4-7,22-24,30].

Since 1986, when the National Childhood Vaccine Injury Act (NCVIA), H.R.5546, 99th Congress, was enacted [60], vaccine manufacturers have been indemnified. This has resulted in increased vaccine promotion targeting vulnerable groups like unborn babies, newborns, and immunocompromised individuals. Governmental agencies within HHS have supported this promotion, fueling debates about the safety and efficacy of pathogen-specific vaccines and their ingredients. The NCVIA also raised concerns about the profit motives of vaccine manufacturers [7, (manuscript in preparation)].

Recently, the author suggested that pathogen-specific vaccines and their components might cause "antigen overload," potentially leading to mini electric shocks (electrochemical sinkholes or spatial defects) in the sympathetic-parasympathetic immune response, potentially contributing to an increase in childhood diseases [7]. In this article, the validity of decades of heavy investments on theory of genetics 'inheritance' as the origin of diseases is also challenged. Evidence is presented that over stimulation of immune system (sustained oxidative stress) is the prime suspect in the destabilization of chromosomal components, increased nuclear and mitochondrial DNA (mtDNA) mutations and altered genomic repair mechanisms that would change tissue physiology toward induction of immune disorders.

1. Insights into Molecular Intelligible Intelligence of Immune Neuroplasticity:

Time and energy-dependent interactions within human immunity involve molecular, electrochemical and electromagnetic intelligence. These interactions occur between immune and non-immune systems, encompassing vascular, hormonal, neuronal, metabolic, genetic, and lipid components. Collectively, this is known as cell-mediated and humoral immunity (CMI, HI) [4-7, 21-24,30,31]. Our accidental discoveries using experimental models of acute and chronic ocular inflammatory diseases indicate the only evidence of a direct link between inflammation and the time-course kinetics of developmental phases of immune dysfunction, which contributed to multistep tumorigenesis and angiogenesis [4-7, 10,30,50-53]. Further analyses of related data support our systematic studies and findings on the role of mast cells, mitochondria, and histamine biology in maintaining health or triggering immune disorders [4-7].

a. Definitions of Yin-Yang of Acute or Chronic Inflammation: Differential Energy Requirements

Effective immunity relies on the polarization-depolarization dynamics of self-terminating acute inflammation. This process involves differential bioenergetics (biorhythms) to defend against both exogenous and endogenous threats. Acute inflammation is characterized by two biologically opposing arms termed Yin and Yang [20,21]. Analyses of data in multidisciplinary fields of developmental biology, childhood or age-associated illnesses, cancer bioenergetics, drug abuse and vaccine sciences directly or indirectly support our original studies and recent definitions of effective immunity, including the role of mast cells, mitochondria and histamine biology in maintaining health or inducing immune disorders as outlined below [4-7,10,21-24]:

i. Yin Events (Sympathetic Arm of Acute Inflammation) [4-7, 21-24]:

a) Apoptosis, pro-inflammatory responses, degeneration, tearing, tumoricidal actions, or catabolism.

b) High energy expenditure (ATP hydrolysis) from mitochondrial oxidative phosphorylation in activated immune and non-immune cells (eg, MCs, DCs, MΦs, NKs, T and B cells).
c) Activation of Yin pathways is essential for expression of danger molecule, release of oxidants, and production of pro-inflammatory cytokines.

ii. Yang Events (Parasympathetic Arm of Acute Inflammation):

a) Post Inflammatory processes, wound healing, tissue regeneration, or anabolism.
b) Utilizes low energy (ATP) from cellular glycolysis.
c) Expression of reducing mediators, decoy receptors, growth factors, and enzymes (eg, VEGF, SODs, IFN-γ) occurs during regeneration and remodeling.

iii. Major outcomes of an acute inflammation [4-7]**:**

(a). **Yin pathways** (Dressed to Kill) are responsible for destruction- degeneration of foreign elements and injured or infected host tissue. Examples include apoptosis, pro-inflammatory responses, and catabolism. Pathways are high-energy (ATP requiring) processes.

(b). **Yang pathways** are required for generation of wound healing (anti-inflammatory) or regeneration mediators and reducing enzymes for tissue repair and remodeling. Pathways utilize low energy from cellular glycolysis that enable mitochondrial shut down and regeneration of TCA cycle intermediates.

(c)**. Biosynthesis of antigen-specific antibodies** (IgE, IgGs, IgM, IgA) and generation of memory B and T cells. Upon next exposure to similarly structure foreign elements the body is prepared to unleash appropriate immune responses for defeating any exogenous or endogenous entities that threaten body's survival.

(d). **Recycling and Autophagy** of proteins and lipids of phagocytized materials, expression of lysosomal hydrolases and proteases.

iv. Differential Effects of Acute Inflammation:

Acute inflammation differentially affects the tissues that are immune-responsive (eg, epithelial, endothelial, fibroblasts, mucus secreting, vasculature), or immune-privileged (eg, BBB, CNS, cornea, neuroretina, reproductive system) [4-7, 20,21].

The body's innate neuroplasticity and immune defenses are vital for preserving health and reacting to immune challenges. The underlying mechanisms are intricate and influenced by a range of factors such as individual genetic makeup, overall health status, age, predispositions, as well as the nature and intensity/potency of the stimulus exposure, and/or the type and vulnerability of the target tissue [4-7,21-24,30,41].

b. Chronic Inflammation: Over-Stimulation of Immune Neuroplasticity and Induction of Inflammatory Diseases.

Unresolved inflammation (sustained oxidative stress, sub-clinical) and over-, or under-stimulation of immune response profiles was suggested to be a common denominator in the genesis and progression of all acute and chronic illnesses (eg, sepsis, anaphylaxis, allergies, asthma, thyroiditis, Hashimoto, arthritis, atherosclerosis, gastritis, hepatitis, myo- carditis, pancreatitis, prostatitis, MS, hypertension, obesity, diabetes and cardiovascular complications, neurodegenerative and autoimmune diseases) or site-specific cancers [4,7,21-24].

The author suggested that wound healing processes (the Yang arm of inflammation) share similarities with the orderly growth of the embryo-fetus and the disorderly growth of cancerous cells. During both fetal development (before birth) and carcinogenesis, mitochondria and the Yin arm of inflammation are either incomplete (not necessary) or dysfunctional, respectively [4-7].

2. Cancer, Drug and Vaccines: 'Antigen Overload', creating Sink Holes-Defects in Tissue Electrochemical Spaces: Altered Histamine and Mitochondrial Functions.

Immunologically, it's now widely recognized that site-specific cancers primarily result from an imbalance between tumoricidal processes (apoptosis and mitochondrial function) and tumorigenic processes (wound healing, regeneration) [4-7,10,15,21-24,29-33,58,61,65 (NCI/NIH documents 1998/1999). In immune-response tissues, the disruption (breakdown) of self-limiting properties of acute inflammation, combined with mitochondrial dysfunction, can trigger an "immune tsunami" (unresolved inflammation) in vulnerable tissues. This drives the expression and co-expression of inflammatory mediators, encouraging tissue growth, multistep carcinogenesis, and angiogenesis [4-7,10,15,21-24,29,30,31,33,41-47,58,61,65 (NCI/NIH scientific documents 1998/1999 and 2006)].

a. **Cancer: Severe Hypersensitivity Reactions**? Cancer was suggested as a delayed and severe hypersensitivity reaction in susceptible tissues, where low-level of histamine release (independent of IgE-fc receptor binding) was a blueprint in tumor growth and angiogenesis [4-7,30].
b. **Increased Induced Diseases (Cancer) in 20th Century:** Cancer emerged as an induced ['genetic'] disease in the 20th century, particularly after the American public was exposed to filterable live viruses, such as SV40, which contaminated polio vaccines in the 1950s and 1960s [4,7,21,24,41].
c. **Mitochondrial Dysfunction and Immune Disorders**. In the following sections, the author explains that mitochondrial dysfunction and disrupted tissue biorhythms (catabolism versus anabolism) represent an imbalance in the Yin-Yang dynamics of acute inflammation's protective properties. Mitochondrial dysfunction (mitophagy) likely leads to decreased ATP production from mitochondrial oxidative phosphorylation (aerobic glycolysis or the Warburg effect). This affects mitochondrial genesis or exhaustion (mitophagy) and may increase mutations in nuclear and mitochondrial DNA (mtDNA). Over the past four generations, this has contributed to genetic predisposition and heightened vulnerability to immune disorders, including cancers [6,7,29-31, 33,39, 41-48, 54,59, 61-76].
d. **Shift toward Increased Induced Diseases:** In the 20th century, classic disease categories (congenital, inherited, neonatal) that occurred in 1 to 5%, shifted toward increased induced diseases up to 54% in children and young adults [4-7 (manuscript in preparation)].
e. **Cancer, an Immune-Inflammation Problem:** Contrary to the popular notions, we demonstrated that cancer is not 100, 200, or 1000 distinct diseases; cancer represents severely disrupted autonomic immune neuroplasticity [4-7].

3. Biological Oscillators: Molecular Intelligible Intelligence of Immune Neuroplasticity in Living Cell- Roadmap into Diverse Mitochondrial Function.

The body's routine electrochemical communications, or biological rhythms, exhibit cycles of positive and negative response switches. The main pacemakers of these complex electrochemical interactions, known as the suprachiasmatic nucleus (SCN), the primary circadian pacemaker in the brain hypothalamus, are influenced by various peripheral circadian rhythms [1-5]. The timeframe for energy-dependent biogenesis and degeneration-regeneration processes in tissues ranges from fractions of a second to minutes, hours, days, months, and even years. An overview of data on diverse immune disorders suggests different degrees of alterations in tissue bioenergetics affecting mitochondria, glycolysis, or alternative cellular energy sources (like hydrogen bonds) could result in defects in the nano-electrochemical spaces, altering the behavior of adaptive and horizontal immune neuroplasticity [4-7,30,32].

Routine cellular activities during respiration include spontaneous and highly regulated electrochemical contributions of subcellular organelles (eg, mitochondria, ER, and Golgi apparatus) and proteins, enzymes, lipids, neurons, and genetic materials (DNA/RNA, epigenetic modifications, hypo- and hypermethylation) are necessary for continuous repair and maintenance. Optimal performance also demands precise nano-electrochemical spacing among specific trace elements; a balance of cations and anions in charged proteins, substrates, vitamins, nutrients, and co-factors for immediate cellular activities [4-7].

4. Mitochondria: Not just an Energy Producing Organelle

Review of data on embryogenesis, the orderly fetus growth during organogenesis and vasculogenesis, and related topics of acute and chronic inflammatory conditions or carcinogenesis, led author to suggest that the completion of organ development and functional immunity; and the Yin (apoptotic/degenerative) arm of acute inflammation along with mitochondrial functionality occurs after birth and the newborn's exposure to air oxygen and environmental conditions [6,7,21-24,30,41]. As detailed in the following sections, the concept that organ and effective immunity including mitochondrial function are completed within two years after birth is bolstered by analysis of data across multidisciplinary fields of developmental biology, models of inflammatory diseases or cancers, childhood diseases, mitochondrial and metabolic diseases, pathophysiology of aging, carcinogenesis and angiogenesis, drug abuse or vaccination of newborn by pathogen-specific vaccines (Figures 1 and 2) [4-7,19-24,38-49,54-59,74-82].

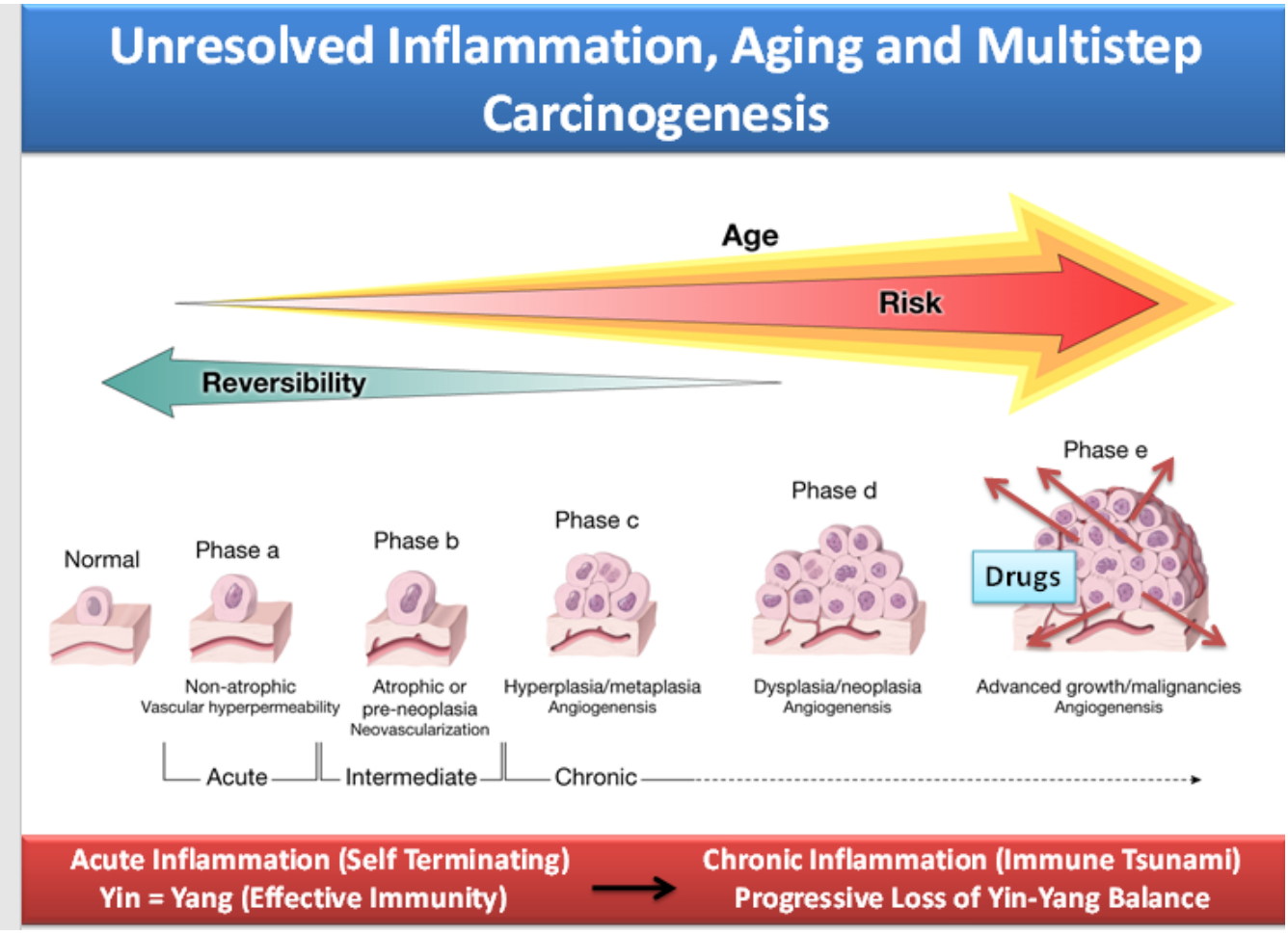

Figure 1: Schematic representation that inflammation and aging are co-risk factors in developmental phases of immune dysfunction in multistep tumorigenesis and angiogenesis. The left panel depicts initial stages of our 'accidental' discoveries on inflammation-induced identifiable immune dysfunction in ocular tissue responses during (a) acute phase responses or self-terminating inflammation (reversible); (b) intermediate phase, down-regulation phenomenon accompanied with mild tissue atrophy and neovascularization (potentially reversible); and (c) chronic phase, induction of massive lymphoid hyperplasia and tumorigenesis and angiogenesis (irreversible?). The right panel represents chronic inflammation and continued stages of tissue growth (d, e), advancing to cancer malignancies and angiogenesis in site-specific tissue. The complex scheme demonstrates that

majorities of translational medicine and clinical trials are conducted in identification of endless damaged molecules at advanced stages of carcinogenesis for drug development and therapy (red arrows in phase e, 'cancer tsunami'). Modified from Ref 23 (2011), with permission, All Rights reserved.

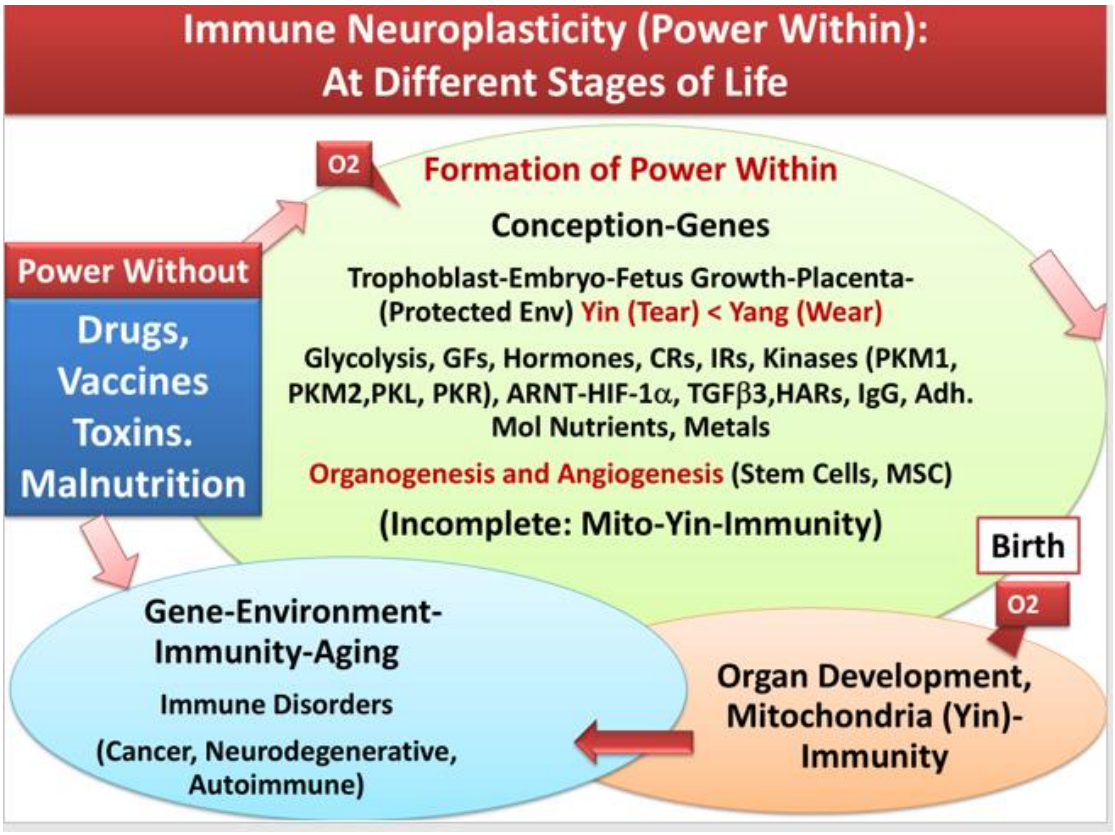

Figure 2: Schematic representation of immune neuroplasticity (power within, biological sovereignty) at different stages of life. It depicts that fetus organogenesis and angiogenesis occur under hypoxia where glycolysis is the main source of energy for unidirectional orderly growth (Yang) of embryo within protected environment of placenta. It represents that mitochondria and tumoricidal (Yin) arm of immunity are not completely developed during fetus growth. Power within (effective immunity) and completion of organ development and immunity (gene-environment-immunity, balance between Yin and Yang) are depicted to be completed within 2 years after birth and exposure to atmospheric oxygen. Expression of constituent or inducible receptors (CRs, IRs), growth factors (GFs), epithelial-mesenchymal transition (EMT), are shown to be influenced by environmental factors (power without) at different stages of life. Immune triggers such as vaccines and ingredients, infections, low level carcinogens are depicted as seeds of immune destruction affecting mitochondrial bioenergetics and immunity that alter chromosomal/genomics stability; bases for cause, exacerbation (aggregation) or consequence of induced diseases. See text.

The primary functions of mitochondria include routine cellular respiration in multifunctional organ systems and defense mechanisms. Mitochondrial homeostasis, balancing degeneration and biogenesis, is essential for the energy-dependent endogenous circadian cycles that regulate the clearance of biological components such as proteins/peptides, cancerous cells, lymphocyte complexes, senescent cells, and DNA/RNA mutations. The bidirectional cooperation of mitochondria with the endoplasmic reticulum (ER), or the functionality of the mitochondria-associated membrane (MAM) is evident in a variety of biological activities listed below [4-7,35,36,41-49,62-68,80,82,99-114]:

a. Biosynthesis of amino acids and structural proteins.

b. Protein folding and degradation.

c. Lipid metabolism.

d. Generation of free radicals.

e. Calcium homeostasis and cell cycle regulation.

f. Degeneration (necrosis or Yin) and ER pathways.

g. Receptor signaling and thermogenesis.

4-1. Bird's eye view of mitochondrial morphology, electrofusion, mtDNA and tissue bioenergetics at stages of life:

The extracted data highlights the critical roles of mitochondria as signaling organelles in reproduction, oocyte development during pregnancy, feto-maternal immunity transfer, and immune tolerance, while addressing challenges like germ line selection and maternal age. It also explores the fetus's transition from intrauterine to extrauterine life at birth and the maturation of the newborn's organ-immune system. The overview of data is essential for better understanding the mitochondria's contributions in oxidative phosphorylation, the apoptotic (tumoricidal) properties of acute inflammation for required ATP production, and the TCA cycle recovery period during tumorigenic (wound healing, post-inflammatory) processes [4-7, 41-49, 80,82, 99-114 (manuscript in preparation)]:

i. Approximately 2,000 proteins and enzymes, mainly encoded by the nuclear genome, are involved in the biogenesis and degradation of mitochondrial complex structures. In humans, an average of 700

(±50) proteins is allocated to mtDNA, primarily participating in oxidative phosphorylation and the production of high energy through ATP hydrolysis by ATPase. The mitochondrial genome is a double-stranded circular structure of about 16.5 kbp, containing few non-coding bases.

ii. Maintaining the stability and integrity of mtDNA, as well as the ability to retain adequate mtDNA template levels, is essential for proper bioenergetics and the body's defense. The circular structure of mitochondria appears to render mtDNA ten times more susceptible to oxidative damage than nuclear DNA. This may be due to the functionality of the mitochondrial matrix and the need for flexibility and accessibility of the inner and outer membranes to mtDNA, facilitating interactions with external lipid nanoparticles or genotoxins (eg, lipophilic and positively charged components) during the production of oxidative phosphorylation for rapid defense mechanisms.

iii. The loss of mitochondrial homeostasis, both biogenesis and degradation, seems to be linked with minor or major alterations in the structure and function of the inner mitochondrial membrane (IMM), outer mitochondrial membrane (OMM), and/or mtDNA.

iv. Alterations in mitochondrial function can include defects in the biosynthesis or degradation of various pathways (eg, ROSs, SODs, cytochrome p450, Ca+2 release and binding, heat shock protein family D (Hsp60), regenerative processes in TCA cycles, biosynthesis of structural proteins for cell contact inhibition). The morphology and behaviors of mitochondria dynamically change through cycles of fusion and fission, affecting mitochondrial homeostasis. The expression of several fusion factors (eg, Opa1, Mfn1, Mfn2) is necessary for binding to the structures of IMM and OMM; fission factors.

v. The expression of several fusion factors, such as Opa1, Mfn1, and Mfn2, is essential for binding to the inner mitochondrial membrane (IMM) and outer mitochondrial membrane (OMM) structures. Fission factors, which facilitate binding to the OMM to form a 'ring-like' structure around mitochondria, include dynamin-related protein 1 (Drp1). Drp1 allows the separation of IMM and OMM and is necessary for connecting to the endoplasmic reticulum (ER) through adaptor molecules such as Mff, Fis1, Mid49, and Mid51, as well as for the attachment of Drp1 to the OMM.

vi. Mitochondria and mitochondrial DNA (mtDNA) are primarily inherited from the mother, although occasionally both parents contribute to the genomic transfer to offspring. The impact of inheriting mtDNA from both parents on mtDNA mutations or heteroplasmy is not yet understood.

vii. Oogenesis is linked to a significant decrease in mtDNA numbers, known as the germline mtDNA bottleneck. The mtDNA of offspring appears to derive from a small population of maternal mitochondrial genomes (30-35 in humans). Thus, mechanisms that reduce heteroplasmy at this bottleneck are crucial for decreasing the transmission of mtDNA mutations and the prevalence of mitochondrial diseases, such as neuronal dysfunction or carcinogenesis.

viii. Eleven distinct sites are associated with mitochondrial complexes I, II, and III during respiratory complexes for the utilization of NAD+ in electron transfer and the coordinated generation of ROS-H2O2 in the OMM-IMM and redox signaling (under hypoxic conditions and ER stress) between the mitochondrial matrix and cytosol. An altered balance in exchanges between mitochondrial complexes (I, II, III) may contribute to various mitochondrial diseases, including those affecting skeletal muscle bioenergetics (fatigue), cardiovascular health, neurological function, and metabolic processes.

ix. Damage to mitochondrial quality control mechanisms, the integrity of the outer or inner mitochondrial membrane structures, or the expression of oxidoreductases related to the MAM domain of the endoplasmic reticulum, often following tumoricidal events and the concurrent expression of anti- inflammatory mediators (such as PINK1 or PGAM5) or mutations in their receptors, has been linked to mitochondrial autophagy and neurological diseases, including Parkinson's disease.

x. Aging is linked to a decline in mitochondrial homeostasis, which is a potential factor in the increase of heteroplasmies and shows a positive correlation with maternal age and its transmission to offspring.

xi. Increased mutations in mitochondrial DNA significantly impact the function of the nervous system and the high energy demands of the polar structures in neuronal tissues necessary for signal transduction in the electro-neuronal network.

xii. Defects in the electrobiology of mitochondria are associated with instability, increased mutations or deletions in mitochondrial DNA ('heteroplasmic'), leading to childhood-onset diseases such as Leigh and Alpers syndromes, autism, ataxia-neuropathy spectrum disorders, epilepsy, progressive external ophthalmoplegia, as well as clinical fatigue, Alzheimer's, Parkinson's, and site-specific cancers in adults.

xiii. Patients tested for mtDNA dysfunction may not show signs of disease, possibly due to the compensatory mechanisms of heteroplasmy that could affect test results. However, advanced mtDNA dysfunction may lead to an accumulation of mutated mtDNA, enough to impair overall health, cause progressive fatigue, and trigger clinical diseases.

xiv. The primary sources of mtDNA instability and mitochondrial diseases appear to stem from defects in loci that encode proteins/enzymes involved in mtDNA replication. These proteins/enzymes mainly include a large family of three complex polymerases and subunits, such as the catalytic subunit of mtDNA polymerases (with 3’-5’ exonuclease activity to remove mis-incorporated nucleotides and 5’ dRP lyase for base excision repair), DNA polymerase gamma (for proofreading and mtDNA repair), POLG2/a dimer (the accessory subunit of DNA polymerase gamma for tight DNA binding and holoenzyme formation), and TWNK/PEO1 (mtDNA helicase).

xv. Genotoxins seem to induce mutations, deletions, and damage to mtDNA and the outer and inner mitochondrial membrane structures at a faster rate than damage to nuclear DNA.

xvi. mtDNA replication occurs in nucleoids attached to the inner mitochondrial membrane (IMM), close to the oxidative phosphorylation site, which not only generates ATP but also reactive oxygen species (ROS). These ROS can damage proteins, lipids, and nucleic acids. Consequently, mtDNA polymerases may be harmed by frequent ROS production and the incorporation of oxidized nucleosides and nucleotides or mutations, such as deoxyribonucleoside triphosphate (dNTP) or 7,8-dihydro-8-oxo-2′-deoxyguanosine (8-oxo-dG) into mtDNA by normal DNA polymerase gamma. If not corrected, repaired, or removed, these oxidized agents can cause G to T transversion mutations. Chemicals, uncouplers, or toxicants may also promote mtDNA instability.

xvii. The inherent and endogenous supply of antioxidants or natural regenerative and repair mechanisms (eg, glutathione recycling, SODs, NAC, NADH/NAD, ascorbate-semi-dehydrogenase, ser/thr

kinase) seems to inhibit and protect tissues, to some extent, from chemically- induced increases in mtDNA mutations. Review of related topics on the consumption of antioxidants, nutrients, vitamins, and trace elements (eg, vitamins C, D3, B complex, Zn, Mg, glutathione, quercetin or NAC) is thought to support, to varying degrees, the integrity and defence capabilities of mtDNA at different stages of life and under diverse inflammatory conditions including age-induced chronic diseases (Figure 3) [4-7,35,41,62-70,80-114 (manuscript in preparation)].

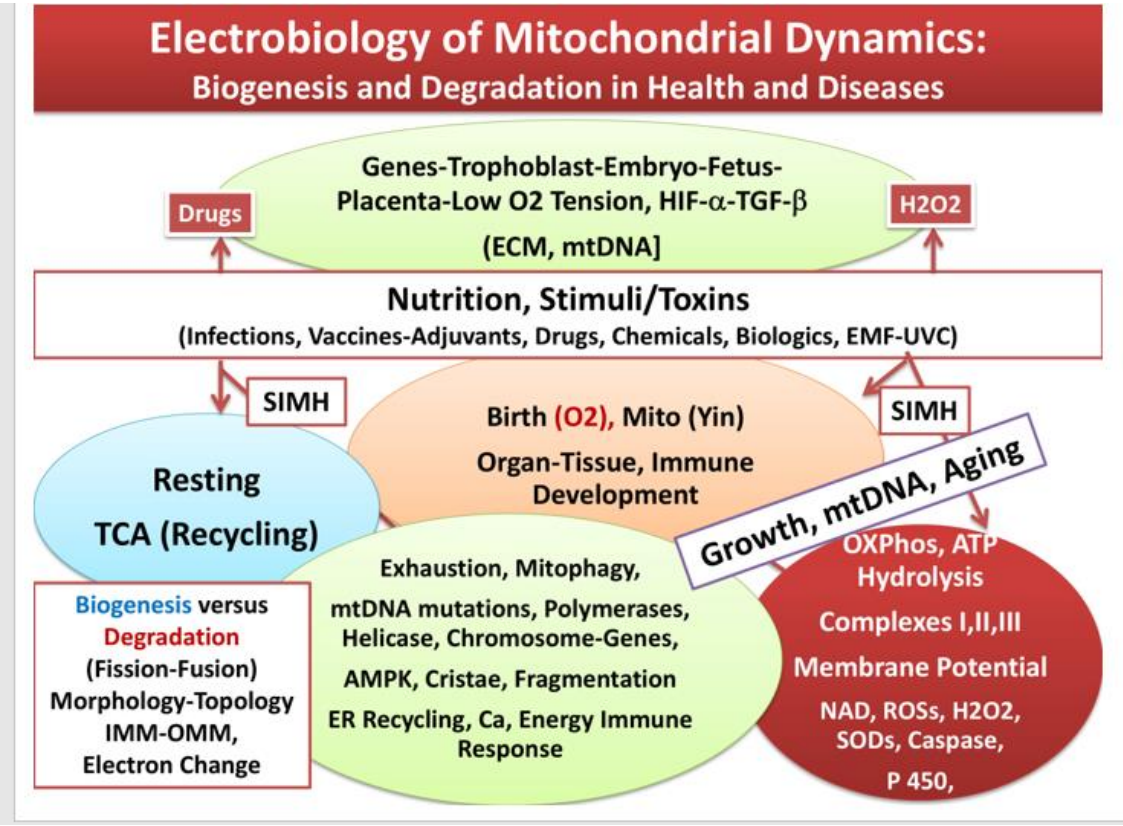

Figure 3: Schematic representation of mitochondrial biogenesis and degradation at different stages of life. It depicts that during pregnancy embryonic growth and mitochondrial DNA (mtDNA) are influenced by the quality of nutrition or exposures to toxins, chemicals, drugs or vaccines. Complex scheme represents that stimulus or stress-induced mitochondrial hyperfusion (SIMH) during pregnancy and/or immediately after birth and exposure to air oxygen alter organ tissue development and defense mechanisms. Exhaustion of mitochondria (mitophagy) could lead to diverse changes in tissue respiration, oxidative phosphorylation (OxPhos) impaired complexes I, II, II, impaired generation and balance in production and utilization of SOD/H2O2, oxidizing or reducing enzymes [eg, superoxide dismutases (SODs), caspases, cytochrome P450]. Mitophagy is associated with mtDNA mutations, altered polymerases, helicase, instability of nuclear chromosomal-genomic components, adenosyl-monophosphate kinase (AMPK) phosphorylation and Ca fluxes. See text.

5. Immune Neuroplasticity: Formation of Body's Sovereignty (Power Within) At Different Stages of Life: Influence of Drugs and Vaccines (Power Without).

During the initial 10-11 weeks of gestation, the earliest patterns of embryonic growth are characterized by the fluctuating expression of hypoxia-induced factor 1 (HIF-1) and transforming growth factor-β [4-7, 11,20,30,41, 78-83, 86-94, 97-100]. The early rise in HIF-1 levels, a factor also involved in cancer progression, likely hinders the initial differentiation of the trophoblast, while promoting the development of the trophoblast's extravillous structures necessary for fetal outgrowth. The expression of HIF-1 precedes the establishment of thermal regulation and the induction of immune tolerance within the placenta's protective environment. In general, key early events in embryonic vasculogenesis and organogenesis, such as the influence of maternal age on germline mitochondrial and metabolic activities, growth survival, and various pregnancy complications (eg, severe preeclampsia, intrauterine growth restriction), as well as failures in the differentiation of human placental trophoblasts and safety-related issues, are outlined below (Figures 2-4) [7,8,28,41,85-94 (manuscript in preparation)]:

a. Proliferation, differentiation, fusion, and formation of multinucleated syncytiotrophoblasts.

b. Fragmentation of the syncytium at various sites of the non-polarized extravillous trophoblast.

c. Physical attachment and integration of the embryo with the uterine wall, and its penetration through the myometrium.

d. Creation of vascular endothelial and smooth muscle cells necessary for vasculogenesis.

e. Transformation of arteries into enlarged utero-placental arteries, ensuring an adequate nutrient-O2 supply through blood flow to the placenta for fetal growth.

f. Transmission of maternal IgG antibodies through the placenta; providing immediate protection against pathogens when the neonate/newborn is exposed to atmospheric oxygen, a time during

which organ development and effective immunity are finalized outside the uterus.

g. Trafficking of maternal immune cells across the placenta, during normal pregnancy is minimal. This may be in part due to the incomplete functionality of mitochondria and the low degenerative capacity of Yin events, such as the expression of inducible receptors for TNF-α, interleukins, HLA, which helps to minimize or prevent miscarriage. On rare occasions, fetal graft-versus-host disease may occur, where the induction of IgG antibodies (eg, IgG isozymes, IgG4) is linked to fetal immunodeficiency and potential postnatal complications (manuscript in preparation).

It was proposed that within the protected environment of the placenta, the mitochondria and the Yin (tumoricidal) arm of immunity are not fully operational (unnecessary), because immune tolerance is crucial for the growth and development of the embryo-fetus at this early life stage (Figure 4) [4,7,41].

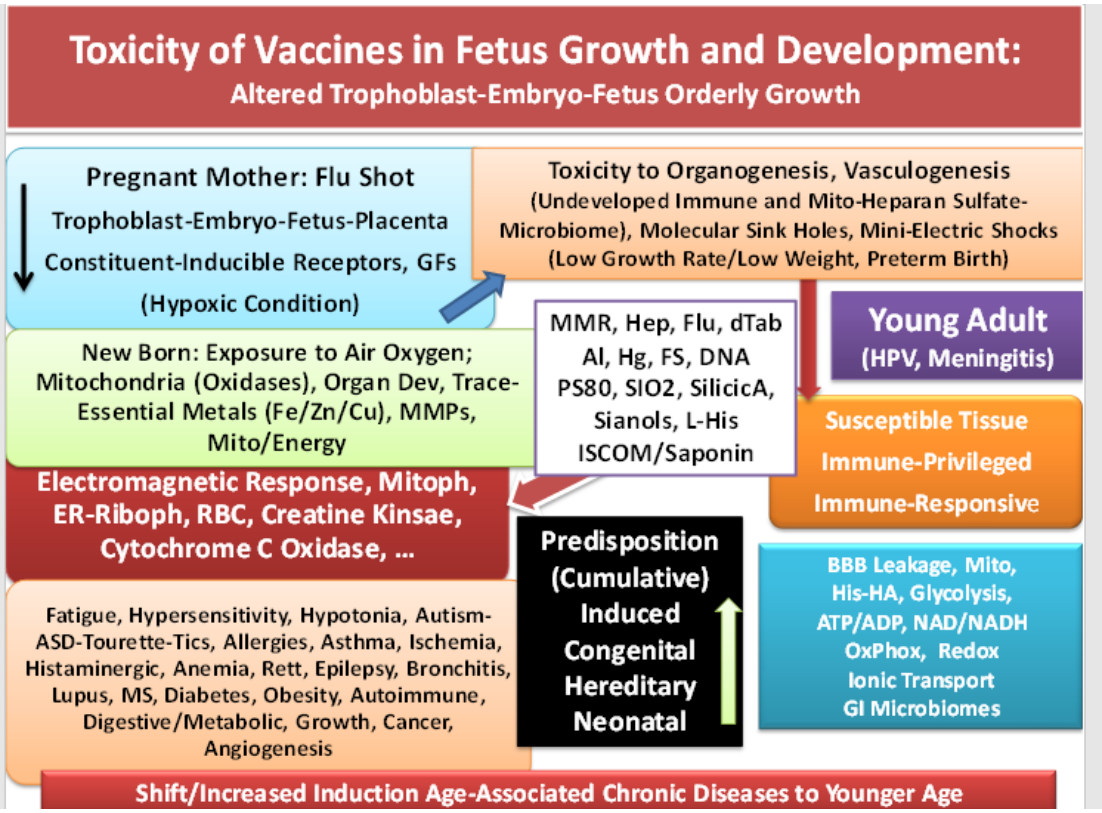

Figure 4: Schematic representation of toxicities of vaccines altering fetus growth and development and bases for immediate or long-term immune disorders, at different stages of life. The complex scheme represents that current pathogen-specific vaccines (MMR, Hep, Flu, dTab, HPV, meningitis) and adjuvants [Al, Hg, detergents, solvents, growth factors (PS80, Silica A, fetal serum/tissue segments)] would alter immune electromagnetic response profiles in tissues and damage differential bioenergetics of mitochondrial oxidative phosphorylation, leading to diverse immune disorders. It also depicts that vaccination of unborn/newborn and infant lead to shifted disease categories (congenital, hereditary, neonatal or induced) to increase the induced diseases (black box). Reproduced from Khatami [7], all rights reserved.

6. Heterogeneity of Immune Response Dynamics toward Oxidative Stress.

In general, exploring how oxidative stress impacts immune response profiles, particularly mitochondrial function, highlights their variability, shaped by various intrinsic and extrinsic factors that often influence health outcomes. These factors can be summarized as follows (Figures 2-4) [4-7,12-14,20-24,29-31,41,72-76,101-114]

a. Frequency of exposure to single or combined immune disruptors [eg, drugs, chemical, biological, or environmental toxins, infections, pathogen-specific vaccines and their adjuvants, electromagnetic fields and devices (electronic 'wearables') or prenatal ultrasounds];

b. Age and health status of individuals in relation to frequent stimulus exposure;

c. Potency and nature of immune disruptors;

d. Effectiveness, integrity, and functionality of innate and adaptive immune responses, as well as electrochemical signals from non-immune systems (eg, vascular, metabolic, neuronal, hormonal);

e. Genomic susceptibility of target tissues;

f. Types of susceptible host tissues (eg, immune-responsive or immune-privileged, insulin-dependent or insulin-independent tissues for glucose transport and utilization);

g. Presence of other chronic diseases that could affect immune response profiles, especially in an aging body.

7. Immune Neuroplasticity: Role of Constituent and Inducible/Acquired Pattern Recognition Receptors:

As mentioned in previous articles [4-7], the stimulation of immune neuroplasticity is linked to the expression of a wide range of inherent/constituent and inducible receptor molecules, potentially leading to either tissue growth or necrosis. While exploring the vast array of

receptor molecules is beyond the scope of this article, it's worth noting that the immune response incorporates various receptors, such as those for danger or death signals, toll-like receptors, pattern recognition receptors, decoy receptors (e.g., IL-1dR and TNF-α dR), and numerous others. These include surface molecules for insulin, other hormones, or enzymes like phosphatases, kinases, ATPases, hydrolases, dismutase, and catalase. For instance, certain receptors are involved in pyruvate kinase activities, located both inside and outside mitochondria. Examples include PKM1 (linked to muscle, heart, or brain function), PKM2 (present in embryonic tissues), PKL (liver), and PKR (erythrocytes) [4-7, 26,28,81,109].

8. Challenging Validity of Theory of 'Inheritance-Genetics' as Origin of Diseases: Immune Neuroplasticity (Adaptive, Horizontal) v. Genetic (Innate, Perpendicular).

Theories on aging biology, such as oxidative stress, genomics, telomere shortening, and immunity, have been explored in other reports [4,5]. This section takes a closer look at the strong emphasis on genetic or 'inheritance' theories, which are regarded as the root cause, or origin of many diseases and have heavily influenced research funding. Despite significant public and private investments over decades to identify countless genetic mutations and develop costly, highly specific technologies aimed at curing diseases like progeria, sickle cell anemia, autism, and cancers, the outcomes have often been inconsistent, leading to debates, setbacks, and frequent failures [4-7,10,59,93,94,112,124-127, (manuscript in preparation)].

Nearly all traditional disease categories, like congenital, inherited, neonatal, or induced, which previously had rates of 1%-5% in the last century, have now shifted significantly towards induced diseases, with rates climbing to 50% to 80% in the 20th century. A review of scientific evidence reveals a notable rise in induced diseases over the past four generations, which is briefly discussed in this section [4-7].

Exploring the connection between genetic mutations and various health conditions (infections, allergies, asthma, sickle cell anemia, progeria, autism, obesity, lysosomal and digestive disorders, neuronal or autoimmune diseases, diabetes, cardiovascular problems, hypertension, and site-specific cancers) or vaccine injuries, raises questions about the influence of genetic inheritance in disease development. It also sparks debate over the definitions of vaccination versus immunization, as well as the safety and effectiveness of pathogen-specific vaccines and their components. Studies on disease promotion suggest that chromosomal instabilities, nuclear and mitochondrial DNA mutations, altered RNA/mRNA translations, or changes in oncogene and repair mechanisms (like P53 or epigenetic shifts such as hypo- or hyper-methylation) might result from overstimulation or improper use of adaptable immune neuroplasticity. This could stem from frequent infections, drug abuse, pathogen-specific vaccines, cancer 'targeted' therapy, 'precision' or 'personalized' medicine, exposures to other immune triggers like GMOs, glyphosate, or electronic devices ('smart wearables') that generate high levels of electrochemical activity, potentially disrupting the electromagnetic properties of intelligible intelligence of immune neuroplasticity [4-7, 21-23,72,73,115-123 (manuscript in preparation)].

The author proposed [4,7] that traditionally inherited diseases result from inflammation-induced irreversible damage to the genomic structure and stability of parental chromosomes. This damage leads to permanent changes in the expression of receptors or surface molecules, affecting tissue functions. Such irreversible alterations in immune response profiles (immune suppression) can cause chromosomal instability, increasing mutations in nuclear and mitochondrial DNA, which are likely passed on to offspring at conception.

This concept is supported by integrating data from developmental biology, genetics of infectious diseases, nuclear and mitochondrial DNA mutations (oxidative stress), environmental toxicology, immune disorders, vaccine biology and vaccine injuries, cancer drugs, and the health status of Americans across different age groups. Studies highlight that mitochondrial oxidative phosphorylation and TCA cycle metabolites (eg, acetyl-CoA, succinate, fumarate, alpha-KG) play roles in regulating stem cell function, chromosomal changes, DNA methylation and demethylation, histone acetylation or deacetylation, and epigenetic modifications throughout life [4-7,29-31,41,50,59, 61-66,69,72,77,115 (manuscript in preparation)].

Additional data, including our unexpected findings from experimental models of acute and chronic ocular inflammatory diseases, revealed the development of multistep immune dysfunction leading to tumorigenesis and angiogenesis [4-7]. These observations question the idea that inheritance is the primary cause of diseases, as outlined below (Figures 3-6) [4-7,118,119,154-161]:

After birth, biological signals trigger interactions between genes-environment-immune system, showcasing adaptive behaviors through intricate electrochemical and electromagnetic responses. The author recently suggested [7,41] that immune neuroplasticity, which mirrors neuronal brain function, plays a key role in shaping the body's innate 'biological power' (its sovereignty and inner strength). Immune responses, with both sympathetic and parasympathetic traits, are adaptive, flexible, and operate horizontally. The author also argued that the adaptability of immune neuroplasticity cannot be fully attributed to genomics, which is vertical and inherent, and therefore should not be seen as the root cause of 'inherited' diseases [4,7,41].

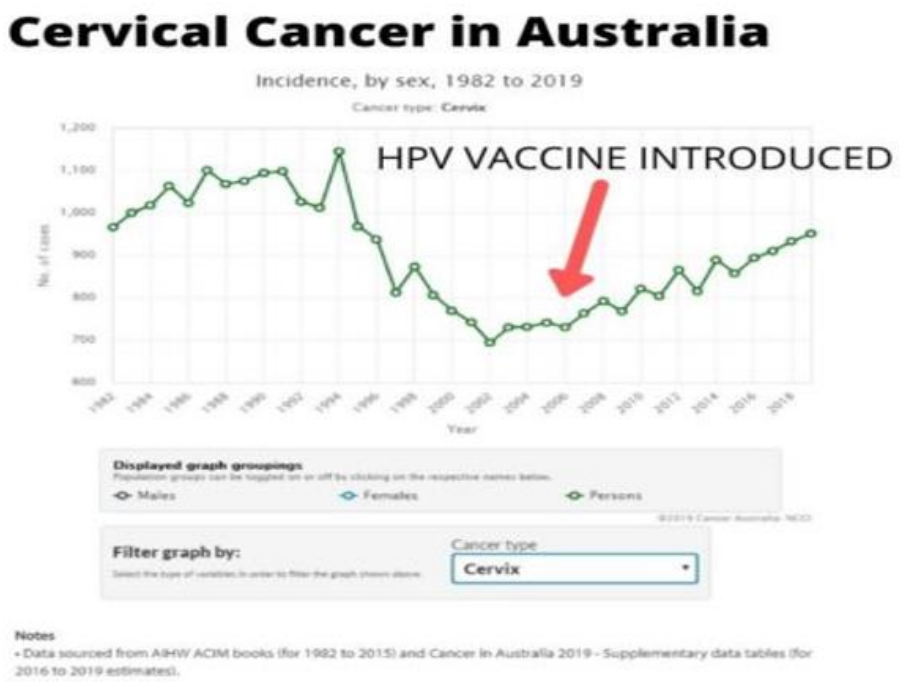

Figure 5: Incidence of Cervical Cancer before and after HPV Vaccination in Australia from 1982 to 2019. Source Internet—Accessed May 2021.

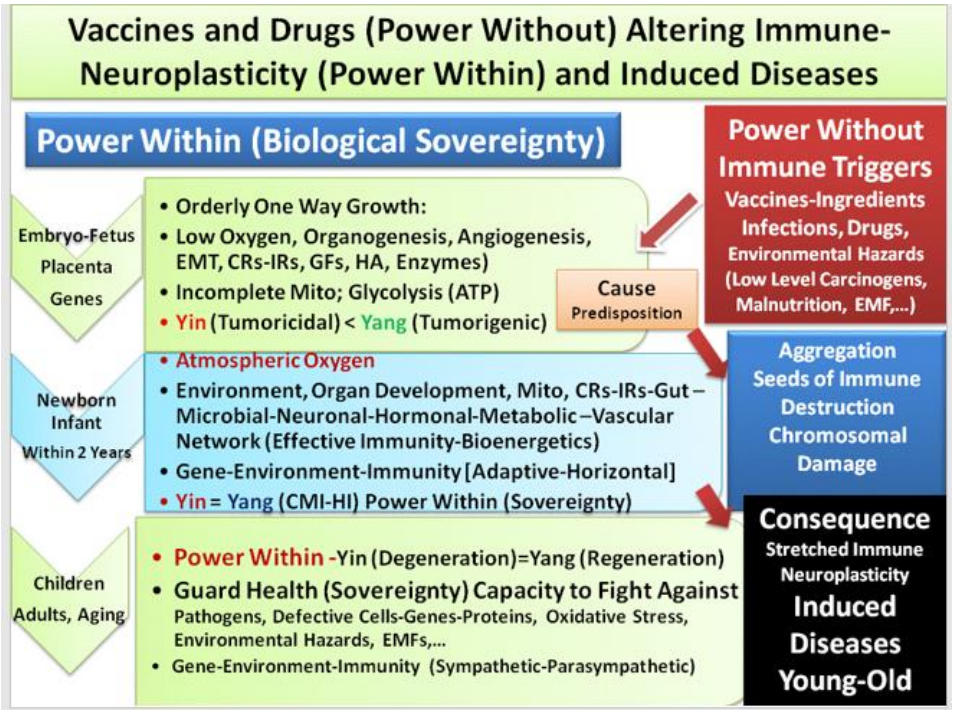

Figure 6: Schematic representation of influence of vaccines and drugs (power without) in weakening immune neuroplasticity (power within), at different stages of life. The complex scheme depicts that fetus organogenesis and vasculogenesis-angiogenesis occur under hypoxia where glycolysis is the main source of energy for unidirectional growth (Yang) within protected environment of placenta. Mitochondria and tumoricidal (Yin)arm of immunity are not completely developed or functional during fetus growth. Power within (effective immunity) and completion of organ development and immunity (gene- environment-immunity, or balance between Yin and Yang of acute inflammation) are completed within 2 years after birth and exposure of new born to atmospheric oxygen. Expression of constituent or inducible receptors (CRs, IRs), growth factors (GFs), epithelial/mesenchymal transition (EMT) are depicted to be influenced by environmental factors (power without) at different stages of life. Immune triggers such as vaccines and ingredients, infections, low level carcinogens are demonstrated as seeds of immune destruction affecting mitochondrial bioenergetics and immunity that alter chromosomal/genomics stability; bases for cause, exacerbation (aggregation) or consequence in induction of diseases. Modified from CIO [41].

Further evidence supporting the idea that impaired immune neuroplasticity may secondarily and permanently destabilize chromosomal and genomic structures—potentially causing the emergence or acquisition of various so-called 'inherited' diseases—comes from our unexpected findings on inflammation-induced multistep tumorigenesis and angiogenesis, along with a summary review of relevant data as outlined below [4-7,21-24,30,31,34,41,51-53,59,69-74,79, 104-110, 123,129,132.136-140, 160-165, (manuscript in preparation)]:

a. At different stages of life, disruptions in the interactions between environment, genetics, and immune response profiles are clinically identified as congenital, neonatal, or induced illnesses, varying from mild to severe immune disorders.
b. In our experiments, guinea pigs showing strong local hypersensitivity reactions in their eyes often displayed wheezing-like symptoms. This suggests that B/plasma cells and mast cells in the lungs, or potentially other distant tissues, were sensitized and activated through circulating antibodies, like antigen-specific IgE [4-7].
c. The offspring of sensitized guinea pigs showed ocular reactions to the first or second exposure to the antigen (FLOA), much earlier than the usual 9-12 days needed for acute/immediate type I reactions, which are marked by redness, itching, and the release of histamine or PGF-1α. This indicates a genetic predisposition due to the sensitization and activation of mast cells and B/plasma cells in fetal or neonatal tissues.
d. Combining the antigen (FLOA) with tumor-promoting agents (TPAs, phorbol esters) led to an earlier onset of ocular tumorigenesis and hyperplasia, occurring within 6 months instead of the 12-30 months observed with the antigen alone. This suggests that additional immune stimuli accelerate tissue growth promotion.

These and related studies suggest that genomic destabilization might be a secondary outcome of reduced/altered immune neuroplasticity. There are also accounts of adverse reactions to pathogen-specific vaccines, including increased injuries or deaths linked to synthetic mRNA spike protein segments in LNPs used in coronavirus vaccines (see below). Early observations point to the accelerated growth of certain cancers, often called 'turbo cancer,' in vulnerable population, along with other autoimmune and neurological issues (eg, myocarditis, fatigue, dementia and higher adult mortality rates) [4-7,30,39-41,50-53,77,115-122, 126,127,141-159, 163,171 (manuscript in preparation)].

Furthermore, the rapid tissue growth or 'turbo cancer,' after mRNA or DNA vaccine injections could be tied to overstimulation of immune cells [eg, mast cells, macrophages (TAM), and CD8 cells)]. The overstimulation of tissue immune system may lead to chromosomal-genomic instability and the expression or co-expression of pro-, and anti-inflammatory mediators, causing unresolved inflammation in tissue under low-oxygen environments (due to reduced mitochondrial function) enhancing wound healing processes. The condition is likely to change the ratios of anabolic (wound healing, tumorigenic) to catabolic (apoptosis, tumoricidal) properties of effective immunity in favor of growth stimulation, increasing cytoplasmic glycolysis (low ATP production) and accelerated growth. Mitochondrial 'antigen overload' could overstimulate immune neuroplasticity, resulting in immune-mitochondrial exhaustion. Unresolved inflammation amplifies genetic mutations in nuclear and mitochondrial DNA, and disrupts the proper expression of growth and apoptotic factors, receptors and surface molecules, potentially driving rapid tissue growth [4,7, 30,31,41 (manuscript in preparation)].

In summary, misusing the natural abilities of immune neuroplasticity can mess with chromosomes and cause DNA mutations in both the nucleus and mitochondria. These mutations might throw off the balance between pro-inflammatory and anti-inflammatory responses, resulting in chronic oxidative stress and a higher chance of immune disorders, including certain cancers. This implies that 'inheritance' or genomics alone may not fully explain where diseases come from [7 (manuscript in preparation)].

9. Toxicities of Vaccines and Adjuvants: Weaken Immune Neuroplasticity (Power Within): Causes, Exacerbations and Consequences of Mild, Moderate and Severe Immune Disorders

Gaps in knowledge, ongoing controversies, and reductionist approaches in cancer and vaccine sciences have greatly hindered progress in cancer research, treatments, and the creation of safe and effective vaccines.

While the direct link between pathogen-specific vaccines and the rise in childhood or adult diseases remains unclear, the author attempts to explore this connection. Evidence from natural immunity studies, comparisons between vaccinated and unvaccinated groups, and vaccine-related injury reports indicates that current government policies promoting widespread vaccination, particularly for the unborn, newborns, and infants, may be contributing to an increase in immune disorders (asthma, autism, neurogenerative and autoimmune diseases, cancers) over three to four generations in America [4,7,10-14,28,39,113-120,124,125,141,148,155-158,161,162,168 (Khatami M, legal briefs and opinions on immunology of vaccine cases, manuscript in preparation)].

After birth, genetically programmed receptor molecules are influenced by environmental signals, adapting to the quality of nutrition (beginning with mother's milk) and exposure to bioactive agents like microorganisms, environmental hazards, or vaccines. These factors continually shape the adaptive and flexible nature of immune neuroplasticity [4-7,10-14,22,41]. Extracted relevant data suggest that administering drugs or vaccines during different stages of embryo-foetus development can disrupt and destabilize the orderly processes of organogenesis and vasculogenesis, as well as the developing immunity, either immediately after birth or later in life. Vaccinating the unborn may alter or harm the delicate biology of the trophoblast-embryo-foetus-placenta complex or the epithelial-mesenchymal transition, impacting the timely expression or suppression of constituent or inducible receptors (Figure 6) [4-7, 30, 41,52,74,85-90,97-99,134-169].

According to vaccine manufacturers' inserts, current vaccines and their ingredients include various chemicals, hormones, and agents like metals (aluminium and mercury salts), detergents and stabilizers (CTAB, formaldehyde, Tween 80/100), gelatin, polysorbate, hydrogel, graphene oxides, and L-His. Genetically engineered synthetic vaccines, such as SARS-CoV-2 modified mRNA or DNA, and naked viral genes (spike or S-protein), are encapsulated in lipid nanoparticles (eg, phospholipids and cholesterol) containing graphene oxides to aid cellular transport. Pathogen-specific vaccines have been associated with various acute and chronic inflammatory conditions, tissue necrosis, or abnormal growth. Specifically, reported Covid-19 vaccine injuries include anaphylaxis, heart issues (eg, pericarditis), blood clots, fatigue, and brain damage (skull-meninges-brain axis). These issues may arise from altered immune and non-immune responses (excessive activation of MCs, MΦs, and vasculature), involving mitochondrial exhaustion that would lead to abnormal neurological necrosis or rapid tumor growth (turbo cancer) [4,5,7,24,39,40,116-122,143,144,148,168-171 (unpublished data)].

Vaccines that are injected at different stages of pregnancy are likely to alter fetus growth and development (eg, increased nuclear and mitochondrial mutations, heteroplasmy in mature oocytes, disrupt proper expression of constituent and inducible factors, histamine/histaminase

pathways) in the neonate-newborn organ development that would alter the complex network of sympathetic-parasympathetic mechanisms of body's defense capacity in immune and non-immune systems (Figures 2-4) [4-7].

10. Emergence of Infections and Over-vaccination: Serious Health Concerns and Future Research Considerations

Over the past few decades, the rising risk of infections like measles, pertussis, shingles, herpes, flu, HIV, Ebola, Zika, SARS, MERS, coronavirus and its variants (delta, omicron), and monkeypox has highlighted the urgent need for pathogen-specific vaccines. This increased risk, along with increase in site-specific cancers, has raised serious concerns, ongoing debates, and controversies about the safety and motives behind vaccines or drug use, the state of American health, and the reliability of 'evidence-based medical sciences' [4-7, 38-41, 115-119, 127,141,142, 145-159, 163, 169-173]. Below is a summary of biological concerns about how drugs or pathogen-specific vaccines might influence immune disorders, like immune tolerance or intolerance, along with ideas for future research [4-7 (manuscript in preparation)]:

i. Vaccine-related reports have indicated the detection of live filterable viruses, such as those resembling SV-40 (found in virus-contaminated polio vaccines) in vaccine culture media. The live viruses in vaccine media may overstimulate the immune system, potentially leading to antibody-dependent enhancement (ADE) of responses to pathogens in vaccines, which could result in viral mutations and shedding. This might also activate other opportunistic or dormant pathogens, like herpes, shingles, CMV, or meningitis, increasing the risk of various diseases.

ii. Messenger RNA (mRNA) vaccines might lead to immune tolerance by causing mitochondrial exhaustion and disrupting mitochondrial metabolites (eg, mutations in isocitrate dehydrogenase and increased hydroxyglutarate levels). This disruption could weaken the body's defense mechanisms and potentially contribute to immune disorders, including multistep carcinogenesis.

As noted above, data on pathogen-specific vaccines, including coronavirus injections, suggest they might overstimulate immune cells (eg, mast cell activation syndrome) and cause low-level histamine release. This could disrupt acid-base balance (oxidative stress) and interfere with feedback mechanisms in mitochondria-ER enzymes (hydrolases, dehydrogenases, dismutase, kinases), or affect ROS-H2O2 production, ion fluxes, ATP/ADP/AMP ratios, and mitochondrial complexes I, II, III. These biological changes may result in tissue necrosis, growth promotion, and immune disorders, including brain and heart tissue damage (myocarditis) or increased mortality [4-7,118,119,170,171 (manuscript in preparation].

Discussion: Evolutionary Nature of Human Immunity (Power Within)

In a state of health, people are shut off from the invasion of germs. Louis Pasture

Interference with fetal growth and development caused by pathogen-specific vaccines, drugs, or electromagnetic fields (EMF) could have lasting impacts on human biology and behavior. The use of even 'safe' vaccines during pregnancy and early childhood might be linked to the emergence, worsening, and outcomes of immune dysfunction, including pre-eclampsia, low birth weight, allergies, asthma, autism, infections, neurodegenerative and autoimmune diseases, or cancers. Additionally, drugs and vaccines, along with aging process, could contribute to electrochemical irregularities in mitochondrial function, changes in receptor expression or surface molecules affecting tissue function, and shifts in electromagnetic spacing crucial for regulating biological rhythms.

These disruptions might alter the balance between tumoricidal (Yin, degeneration or catabolic) and tumorigenic (Yang, regeneration or anabolic) properties of immune neuroplasticity (sympathetic-parasympathetic), potentially triggering or worsening immune disorders.

During the pandemic, it was concerning how science seemed to lose its way. The truth felt elusive, as government authorities' heavily publicized stances on lockdowns and masks often didn't add up, nor did many scientific studies hold water. This could be linked to the close ties between government, industry, and venture capitalists (referred to as 'philanthropists' or disease investors), which undermined medical ethics and conflict-of-interest standards in taxpayer-funded projects. For instance, in 2021, the CDC reportedly withheld data on deaths following Covid-19 vaccinations (with 1,170 deaths reported, while VAERS data suggested twice that number, though likely still underestimated) [172, 173 (Kulldorff M, Gupta S, Bhattacharya J. and Co-signers: Great Barrington Declaration, December 4, 2020, Great Barrington Declaration; Senate Hearing (The vigilant Fox, May 21, 2025, COVID Vaccine "Safe and Effective" Narrative Collapses on Camera); Health Impact News, February 13, 2021; manuscript in preparation)].

Concluding Remarks.

The genetic makeup of parents, fundamental and inherent, acts as the 'hardware' or building blocks of inheritance passed to offspring at conception. This genetic framework in insects, animals, and humans has remained remarkably stable for thousands of years. Unlike species like insects, birds, horses, and guinea pigs, which quickly become self-sufficient and able to find food shortly after birth, human newborns continue their development outside the womb, remaining helpless and dependent for a prolonged period. Achieving self-reliance in humans, including various survival skills like physical, neural, cognitive, mechanical, and social abilities, requires an extended learning period that spans many years.

If organized medicine had explored the intricate natural intelligence of autonomic immune neuroplasticity that protects health, the mysteries of cancer biology and treatment might have been solved years ago, lessening the financial strain on taxpayers. Future studies should focus on mitigating gene-environment-immune hyper-activation to strengthen the body's defenses against external and internal threats. Examining the interactions triggered by stimuli between immune and non-immune cells, along with tissue oxido-redox potentials (like mitochondria and histamine biology), could shed light on the early signs of immune surveillance failure. Researching the initial stages of immune dysfunction might help develop better risk assessment tools, safer vaccines, and effective strategies to boost natural immune neuroplasticity, paving the way for a healthier society.

It's important to comprehend that, under normal circumstances, internal pressure or 'power from within' during fetal development supports life and promotes health. In contrast, external pressures or 'power without', such as 'antigen overload' of pathogen-specific vaccines or drugs, likely to weaken or disrupt life. Even introducing 'safe' vaccines to unborn or newborns could interfere with the proper development of human organs and immunity.

For improving public health (making America Healthy Again-MAHA), the author urges policymakers to reinstate liability laws for vaccine manufacturers.

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Conflict of Interest: None.

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