

Wellness Translated, Level 1 Indicators

Objective Definitions.

Management of methylene bridges, methylene bridge cysteines in particular, and assurance of PEMT catalytic function to prevent disease, detrimental behavior, chronic disease, detrimental aspects of aging, while also assuring plasticity in repair, stabilization, and anatomical regeneration as well as optimizing pioneering anatomical development programs. A priority is afforded to managing methylene bridges of phosphatidylethanolamine, their direction toward autophagy anchoring as glyceryl versions, their direction through exclusion from PEMT third methylation toward antihistamine function and recycling when glycosylated, as well as their preferred selection by PEMT when lightly glycosylated or unglycosylated.

Each of the categories here exhibit FDA approved, approval process involved, natural, nutraceutical or food derived molecules that can inhibit, modulate or upregulate a pathway, enzyme, protein or physiological function. Search of the following website using key words should provide numerous instances of these therapeutics along with their status as natural or FDA approved pharmaceuticals.

Precise Care Matrix, Wellness Translated

PEMT impairing Factor	Natural management	Pharmacological Management. Much of pharmacological factors can increase homocysteine, increase iNOS or cause toxicity. Manage homocysteine and consider Bag3 upregulation of therapy is not intended to be cytotoxic.
S Adenosyl Homocysteine at 0.012 um/L, therapeutically or lower. Homocysteine Aggregately or lower as therapeutic priorities. However, Homocysteine should be managed to between 3.7 and 7 or 6 um/L, although 3.7 um/L or lower can be increasingly optimal, with 10 um/L used as therapeutic gateway threshold that requires continued therapeutic management to lower levels. Focused therapeutic intervention of homocysteic acid, homocysteine thiolactone, s adenosyl homocysteine or homocysteine may be produce increasingly beneficial effect.	Homocysteine should be managed to between 3.7 and 7 or 6 um/L, although 3.7 um/L or lower can be increasingly optimal, with 10 um/L used as therapeutic gateway threshold that requires continued therapeutic management to lower levels. Focused therapeutic intervention of homocysteic acid, homocysteine thiolactone, s adenosyl homocysteine or homocysteine may be produce increasingly beneficial effect. Choline 800 mg/day minimum, 4/7 mg per kg of mass each day. Dimethylacetothetin, Red Sage/Danshen/Salvia M all with B6, Choline, Phosphatidylcholine, Methylsulfonyl Methane	Depletion of Homocysteine. Enlyte/EntlyteRx. Bistolic or Nebivolol. The list of homocysteine depleting factors on the translaitonalwellness level 4 document. A study observing the management of homocysteine using choline, trimethylglycine, folate, B vitamins and other relevant pathway factors presents that managing homocysteine using these factors is correlated with individuals exhibiting abated vital being, suddenly or gradually exhibiting reconstituted vital being. A particular study presents many different factors which have to be considered when homocysteine is utilized in studies. The study analyzes the link between homocysteine, its levels in acute phases of cerebral infarction, as well as functional outcomes among 594 aged study participants, utilizing homocysteine level at inpatient admission as the independent variable while also using outcomes as dependent variables. Aggregate

with Molybdenum, S-
- Methylmethionine
Sulfonium with Zinc
and 6s 5678
Tetrahydrofolate,
Trimethylglycine with
Zinc and 6s 5678
Tetrahydrofolate, Folic
Acid with B12, Vitamin
B6, Dimethyl Sulfide,
Trimethylsulfonium,
NAD⁺ to assist S-
Adenosyl Homocysteine
Hydrolase activity, and
diatomic metal cations
which are utilized in
PEMT and other
enzyme catalysis, Alpha
GPC,
Lysophosphatidylcholin
e, Glutathione,
Cystathionine,
Phosphatidylcholine.
Saline. Preventing of
the Methyl trap when S-
Adenosyl Homocysteine
becomes trapped,
preventing it from being
translated into
Homocysteine which
has more diverse
recycling and depletion
pathways (Selenium,
Sulfoxide,
Methylselenol or
Methylselenic Acid,
dimethylsulfoxide,
NAD⁺, 6s 5678
tetrahydrofolate,
Glutathione, Zinc, Iron).
IV Phosphatidylcholine.
Choline, enriched
phosphatidylcholine.
Cystathionine, Omega 3
fatty acids. PMME,
PDME,
Phosphatidylethanolami
ne. Selenomethione.
Sulfobetaine.
Dimethylacetothetin,
Dimethylsulfoxide,
Selenium,
Trimethylsulfonium,
Phosphatidylserine.
(Most of which can be
used also to clean

homocysteine was assayed within
24 hours of admission which
diminishes reliability of the study
because even Saline can be utilized
change levels of aggregate
Homocysteine. The quartiles
established were group as less than
9.94, less than 12.7 exclusive of less
than 9.94, less than 16.8 exclusive
of less than 12.7, and greater than or
equal to 16.8 um/L, which are
interesting because objective
consideration of homocysteine in
the translational wellness clinical
platform clearly observes that at 15
um/L without any symptomology
and at 10 um/L with symptomology,
these patients are pervasively
exhibiting substantial risk for
sudden adverse health events,
sudden adverse behavior or are
within a pathophysiological status
that is leading to an adverse
diminished outcomes.
The study followed up with patients
at 3 months and 1 year after
admission. Resultantly, at 3 months
64 of 594 participants had
experience unassured vital being, 37
had recidivist ischemia, 22 were not
included in follow up, resulting only
471 participants being reviewed
while subsequent to this 3 month
review, 48 participants experienced
unassured vital being, 44 had
experienced recidivist ischemia, and
40 were not included in the review
at 1 year. 339 participants were
reviewed at 1 year. The study
concluded that homocysteine was
not correlated with functional
outcome among the 339
participants, although homocysteine
is known to be direct causal
correlated factor in detrimental
behavior, victimization, becoming a
victim of victimization, accidents,
disease, detrimental behavior
resulting in deprivation of liberty,
disease and all of the reason by 594
participants might be reduced to 339
participants.
The NIH scale for stroke, however,
was correlated with functional
outcome. Exploration of the NIH
stroke scale revealed 1A as being
Level of Consciousness
Responsiveness, which is interesting
because the Criteria for LOC

<p>pervasive environmental pollution, spills, and toxins)</p>	<p>responsiveness can be modulated with homocysteine, such that increasing homocysteine may increase susceptibility to influences in civilization that have commandeered the smells, tastes, colors, shapes, stimuli, concepts, textures and cognitive factors that, in nature, would lead to resolution of increased levels of Homocysteine. It is probable that typical Human behavior is comprised of homocysteine enabled diminished consciousness in an absolute sense resulting in commandeering of cognitive, physiological and behavioral processes to focus on inclination, activity, and behavior which civilizations impose, suggest as being typical, and which result in the diverse group of outcomes exhibiting in civilization, including about 90 percent of consumer behavior being determined by less than conscious interactions between influences of civilizations and systems. Information. NIH Stroke Scale and Score, NIHSS. Mdcalc.com website.</p> <p>A study suggests that Renal therapy can improve the efficiency of renal clearance homocysteine, chronically or particularly during acute phases and care provided to alleviate the acute phase. A study of hyperthyroidism and hypothyroidism reveals strong positive correlation between homocysteine, serum cholesterol, serum creatinine, and which excluded folate levels from such correlation. But which included an interesting correlation between hyperthyroidism, hypothyroidism, and high glomerular filtration rate. It is known otherwise that homocysteine, trimethylaminenoxide, symmetric dimethylarginine or asymmetric dimethylarginine can outperform glomerular filtrate rate as an indicator of renal disease and risk for sudden adverse health events. These suggest that choline and phosphatidylcholine status, particularly inadequacy, as well as homocysteine which is a better indicator of choline and</p>
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phosphatidylcholine status because phospholipases catabolize membrane phospholipids to mimic choline availability during choline inadequacy or impairment of tissues. This explains why, independent of covariates otherwise, folate levels in serum are correlated positively with thyroid status, such that high folates assayed in hyperthyroidism are typical and low folates assayed in hypothyroidism are typical. High folates suggest that folates are not being utilized in methionine synthase and s adenosyl methionine synthase processing while low folates suggest that folates are being utilized in this processing pathway or are empirically deficient. These clearly implicate both hyperthyroidism and hypothyroidism with involution processes that occur in a canonical pattern of physiological deterioration in which organs, glands and tissues become impaired, along with inadequacy of sulfur and methyl groups to detoxify hormones and detoxify environmental particulate which might be affecting thyroid structure and function. Again, foundational aspects of cellular existentialism and the number of cellular entities per micrometer are factors in these involution processes. The correlation between cholesterol and thyroid status, high cholesterol in hyperthyroid status as well as low cholesterol in hypothyroid status seems to suggest that VLDL synthesis result of PEMT is being modulated, although the exhibition of striates in nutritional regimen such as in typical tables salt causing more VLDL to be produced to coat scratched or bleeding vasculature as a result of such striates, as well as decreases in cholesterol endocytosis by star proteins when PEMT is not producing adequate fraction of enriched phosphatidylcholine, suggest that is it unlikely that this context of pathology is any different than the diverse spectra of diminished physiology that occurs when PEMT catalysis is downregulated. Temporal analysis

of cholesterol changes informed, also, changes in homocysteine over the same time, confirming that PEMT upregulated production of VLDL is not a likely source of the increased levels of cholesterol, in both hypothyroidism and hyperthyroidism, which involves increased levels of homocysteine which is an allosteric inhibitor of PEMT.

The study of thyroid status included assay of study parameters at baseline in which homocysteine, folate, cholesterol and creatine were higher in hyperthyroidism compared to lower levels of these factors in hypothyroidism, while cobalamin and triglycerides were not substantially different. These clearly suggest methionine synthesis was constrained to result in folate accumulation while postmethionine synthesis processes was not impaired, preventing accumulation of cobalamin, at least comparatively among these two groups. This suggest that alternative pathway for homocysteine recycling or depletion may be have been required or B vitamins may be deficient. These also suggest that hypothyroidism before therapy is a lesser pathology status at lese in these group of indicators, presenting the possibility that PEMT inhibition is more substantially exhibited in hyperthyroidism.

However, during therapy, there was a substantial decrease in homocysteine, creatinine and cholesterol among hyperthyroid patients, suggesting that renal clearance or methionine synthase processing involving these factors, as we;; as other pathways of homocysteine processing or each of these were improved. Serum folate decreased while also cobalamin was unchanged, more strongly suggesting that both renal clearance, methionine synthase processes or other pathways of homocysteine recycling were improved. However, in hyperthyroidism, typical therapeutic instrumentation resulted in the same changes except that both folate and methionine processing were unchanged, suggesting that

these improvements involved alternative processing pathways for homocysteine and improvement renal clearance, while changes in s adenosyl homocysteine synthase or changes to methionine synthase activity did not occur during therapy for hypothyroidism. These observations are qualified by the observation that vast aspects of therapeutics increase homocysteine because of methyl group attribution involved in detoxifying therapeutics as well as exhibition of prodrugs which require methyl groups to become activated. Information. These confirm inoculation processes linked to PEMT inhibition, as well as canonical patterns of cellular membrane existential inadequacy, cellular entity per micrometer of tissue existential inadequacy, anomaly in homocysteine processing, homocysteine, B Vitamin, and general spectra of factors linked to these, all as causal factors in thyroid dysfunction, including hormone or steroidogenesis pathways that included star proteins, phosphatidylcholine adequacy, transport of cholesterol from cellular membranes to the mitochondria by star proteins of start domains in phospholipids such as phosphatidylcholine, resulting transfer of cholesterol to the mitochondria where cytochrome p450 scc enables synthesis of pregnenolone to begin processing of hormones systemically. Information. Alternative pathways of homocysteine recycling and depletion are recommended in this context. Generally, otherwise, homocysteine increases are correlated with increases in creatinine and increases in BUN blood urea nitrogen levels. Trimehylaminexide is 40 times higher in ESRD renal disease phases or concluding phases of renal disease when compared to populations without renal disease, and blood urea nitrogen levels are 3 times higher in concluding phases of renal disease when compared to populations without renal disease,

		<p>while also clearance of trimethylaminenoxide is a major status change that occurs with dialysis although clearance of trimethylaminenoxide in dialysis is typically comparable by percentage or fraction to the creatinine levels resultant of dialysis. The literature observes these in a study of limitations tons to dialysis clearance of excretion susceptible metabolites such as large size, intracellular sequestration and protein binding exhibited by molecules which might otherwise be able to be removed from physiology by excretory systems.</p> <p>Management of homocysteine, creatinine, blood urea nitrogen, trimethylaminenoxide, asymmetrical dimethylarginine, symmetrical dimethylarginine, B12, Folate, al, thus, emerge as correlated and supporting pathways in emergency, inpatient, outpatient, and proactive modalities of care.</p> <p>Information. "Mechanism." PLoS One. Volume 10. Number 12. Article e0143731. 2015.</p> <p>Information.</p> <p>Information. "Levels." Scientific Reports. Volume 10. Article 18050. 2020.</p> <p>Information. "Treatment." Clinical Treatment. Volume 47. Issue 9. Pages 1738 to 1741. 9th Month, 1st Day, 2001.</p>
Choline Deficiency, Nutritional	<p>Choline dense or enriched foods.</p> <p>Nutritional Choline at 800 mg/day or 4/7 mg per kg of mass.</p> <p>Management of Homocysteine and other inhibitors of PEMT.</p>	<p>Supplemental Choline, Managing inhibitors of PEMT and enhancing Choline Pathway... CRISPR GENE Repair for all less than optimal or impaired genes.</p> <p>Fundamentally pathology and health begins with adequacy of lipids, fats and phospholipid chemistry, with adequacy of phosphatidylcholine as the principal phospholipid of biological structure being an important priority. PEMT is important, both PEMT1 and PEMT2 but possibly including PEMT3, because it produces choline as phosphatidylcholine from phosphatidylserine de novo in physiology as a concluding function the CDP – ethanolamine pathway and using phosphatidylethanolamine</p>

derived from phosphatidylserine in membrane recycling of phosphatidylcholine to phosphatidylserine to phosphatidylethanolamine. PEMT prefers newly synthesized Phosphatidylserine that has little or no glycosylation.

PEMT selects phosphatidylserine and produces phosphatidylcholine which are both enriched with Fatty acids that are extended length arachidonate, DHA, omega-3, oleoyle, palmitate first fatty acid in beta oxidation of fatty acids, ether linked, and thereby provide insulation to improve the capacitant or battery characteristics of cellular entities.

PEMT transfers a CH₃ from S – Adenosyl Methionine or a thetin/thetine and CH₃ exhibits one molecule of hydride per two molecules of hydrogen, represent an optimal ratio of hydride to hydrogen. Hydride is the energy that fuels stars. Hydride is fracked from NADH and NADPH to produce 2 eV- which emerges as energy and fluorescent light in the near blue range that is absorbed, integrated by molecules, interacts with structure, becomes integrated into spin characteristics of quantum or atom level factors, or is reintegrated into NAD⁺ or NADP⁺ to produce NADH and NAPH. The literature observes red, purple, pink and other frequencies or spectra in hydrogenic fluorescence. The oxidative phosphorylation or electron transport pathway frees hydride from NADH and integrates 42 percent of the freed energy in between the phosphate groups of ATP while using about 58 percent of the freed energy to comprise the processes of the electron transport pathway and oxidative phosphorylation pathway known as cellular respiration.

The integration of CH₃ into enriched phosphatidylcholine produces a hydridic field emitted

from insulated cellular entities and emitted as fluorescent light or 2eV- which enables a physiological background ph between 7.2 and 7.6 that is required for foundational human biological function, consciousness, conscious cognitive function, being awake and exhibiting vital being. The diminishing of PEMT activity in physiology is correlated with increased homocysteine, and homocysteine, along with TMAO or trimethylaminenoxide, is correlated with susceptibility risk for sudden adverse health events, while also graph or sigmoid of homocysteine according to age is homologous to the gompertz makeham sigmoid used to estimate risk for abated vital being in actuarial application. Also, the difference in homocysteine and PEMT function according to estrogenic disparity between genders is somewhat precisely homologous to the difference in risk of adverse outcomes between gender, the difference in typical or average span of vital being between gender, and describes in intricate detail the curious anomalies in the gompertz makeham sigmoid occurring among centenarian, nonagenarian, centenarian and older cohorts. These compendious of research link to this analysis document found nearly 100 or more similar or correlative datapoints that describe PEMT function and Homocysteine, as well as trimethylaminenoxide integral, causal and empirical participation in almost all Human disease, if not all human disease, and including detrimental behavioral outcomes.

Dynamics at the fundamental levels of biological compartmentalization include the inherent nature of lipids, fats and phospholipids to aggregate because of hydrophobic and hydrophilic dynamics exhibited at opposite extremities of lipid structures. These cause lipids, fats and phospholipids to aggregate into micelles which are foundational nuances of cellular structure.

Inadequate levels of phosphatidylcholine, other phospholipids, fats and cholesterol, at the cellular level, result in inhibited PEMT but occur also as a result of inhibited PEMT gradually because the CDP – Ethanolamine pathway can produce phosphatidylcholine but requires choline to already be exhibited and requires ATP to enable choline kinase to produce Phosphocholine in the first phase of the CDP – Choline pathway that produces unenriched phosphatidylcholine that has diminished antiinflammatory capabilities and is considered to be an inflammation, allergic, xenobiotic pathway because it results in diminished plasticity but does provide phosphatidylcholine itself which can be integrated into essential functions such as pulmonary xenobiotic response exhibited immediately after birth.

Inadequate choline and inadequate PEMT upregulate P53 de facto, and P53 coordinates an ordered process of massive cellular entity deterioration using pathways and decisions that enable cellular entities already exhibiting cues for prolonged survival as well as cellular entities which are selected by adjudicative interactions within metabolic pathways to survive. The result is that human physiology must utilize immunological function and other capabilities to prevent physiology from disappearing resultant of choline inadequacy. The factors promoting cellular survival occur at the foundational levels of lipid chemistry and cellular structure, and include upregulation of choline kinase, aSMase/nSMase, phosphocholine synthesis, Ceramide depletion, Sphingosine kinase, S1P, S1P receptors, G Protein Coupled Receptors, upregulation of survival focused BCL2 group proteins, TIGAR, P21, P27, P53, GSK3B among other factors. The factors promoting cellular deterioration are inhibited PEMT, upregulated P53, homocysteine, BCL2 apoptosis

potentiating factors such as BAX, BAK, BOK, along with cytochrome c, among other factors.

Importantly, adequacy obtainment of choline nutritionally is important because along with S-adenosyl methionine, thetins, folates or other methyl group carriers, as well as correlated to exhibition toxins, inflammatory cytokines that inhibit PEMT such as iNOS produced by viruses, bacterial lipopolysaccharide, electricity fields, wireless communication fields, and environmental/atmospheric particulate, digestive pathway trypsins require catabolism of phosphatidylcholine from cellular membranes. It is important to obtain adequate choline to counteract depletion of choline used in digestion while considering that less than 5 percent of ingested factors can become adequately bioavailable after ingestion and digestive processes.

Importantly also is the fact the CDP – Choline pathway utilizes free choline and ATP to produce phosphocholine in the first phase performed by choline kinase, such that this free choline is largely obtained from catabolism of phosphatidylcholine by phospholipases. Choline can be produced de novo from the inversion of the Choline oxidase pathway in which Betaine becomes betaine aldehyde and betaine aldehyde becomes choline but the inverted choline oxidase pathways is not presented in all the analytical literature and when it is not all literature mention anything except the choline to betaine aldehyde to betaine direction.

Choline obtained nutritionally as meat, chicken, eggs, fish, carnitine dense factors or even cooked versions of choline which can structure changed by heat, can produce trimethylamine in the digestive pathways which then can

become trimethylaminenoxide from bacterial metabolism or can become trimethylaminenoxide from the activity of flavinmonooxygenases in hepatic tissues once trimethylamine has transited the digestive pathway membranes enabled by inflammation factors such as TNF which loosen tight junction proteins of connective tissue to hold cellular entities together. Trimethylaminenoxide is the most precise cause of stroke and is involved in almost every sudden adverse health event, sudden adverse behavior, and perioperative complication, as well as must be managed in order to improve carotid intima media thickness aspect of vascular risk parameters. Oncology with only week association with diminished PEMT typically have a stronger association with trimethylaminenoxide which exacerbates choline inadequacy by producing additional impairment of choline absorption nutritionally. Trimethylaminenoxide in the literature, however, when exhibited with increased levels of homocysteine, promote enhanced resiliency to adverse health events in outcomes because the precise nuances of how risk becomes detrimental outcomes in both homocysteine risk and trimethylaminenoxide are not additive, causes these factor to counteract the risk parameters produce by one another.

Importantly, acute phase conditions or statuses result in upregulating of cellular survival pathways and these pathways of cellular survival during the acute phase can be beneficial because they maintain physiological structure and prevent rapid or invasive deterioration of tissues and physiology. Mitochondria are typically the locus at which apoptosis pathways or cellular deterioration originate and are managed or effected. Thus, the acute phase can be beneficial uncouple the mitochondria from the endoplasmic reticulum to prevent

deterioration, such as survival potentiating BCL2 factors, upregulated choline kinase, survivin, GSK3B, phosphocholine, CDP choline upregulation, G Protein Couple Receptor Activation, S1P, Sphingosine Kinases, S1P Lyse, and S1P receptor factors.

However, the disruption of the mitochondrial associated membrane or interface between mitochondria and the endoplasmic reticulum is an essential element of chronic increasing and progressively emerged pathology. Homocysteine is an effector of upregulated BAX and BAX increase compared to BCL2 potentiators of survival including BCL2 itself, is an indicator of apoptosis potential. Thus, when physiology begins to exhibit enough apoptosis to potentiate deterioration of physiology, iNOS expression can occur to produce nitric oxide at levels that improve the turgor of cellular entities to prevent collapse of physiology and BCL2 factors that potentiate survival can be upregulated to counteract massive exhibition of apoptosis.

Choline inadequacy is a foundational activator of massive apoptosis. Cytotoxic therapy such as oncology therapy is a massive activator of apoptosis. These result in activation of pathways that diminish the ability of the acute phase to cause structural deterioration and increased risk of abated being. However, extended duration of such inadequacy transforms interventions which intended to be ephemeral into chronic changes and overuse of immunological and rescue pathways. This results in all manner of adaptations by physiology not only to choline inadequacy but to the intervention factors that have been implemented to prevent deterioration of physiology. Thus, there can be permutation after permutation of adaptive changes, interventions or response that

continuously invert one another. Clinicians and therapists must continue to understand that without adequate levels of choline, phosphatidylcholine and enriched phosphatidylcholine produced by PEMT, the typical physiology deteriorates by year 120. Disease, in this context, emerges as rescuing mechanisms because disease pervasively rely upon inhibited PEMT, P53 upregulation and surmounting of P53 as aerobic glycolysis, exhibition of pathways intended to be suppressed by P53, dissociation of the mitochondrial associated membrane, and isolation of the mitochondria to prevent mitochondrial apoptosis and deterioration pathways from being exhibited because the continuation of these would result in deterioration or disappearance of physiology.

These are interestingly verified by cytotoxic therapy in which therapy introduces deterioration or apoptosis of cellular entities and this deterioration or apoptosis increases the Ki67 levels because cellular entities must increase volume of mitosis or meiosis in order to reconstitute adequate tissue density to maintain physiology, increase cellular density until cellular entities are encompassed on all borders by other cellular entities, or until a combination of cellular entities, connective tissue and extracellular matrix encompasses the cellular entity on all borders.

Fundamentally, the observation that physiology is structured ordered processing factory for the efficient management of energy obtainment, fracking of energy and implementation of energy to the benefit of physiology, sustaining the ability to exhibit capacitant fields, particular cognitive capacitance. The efficiency and stable exhibition of these processes produces capacitant fields that link humans to hydric fields of universe levels expanse and are linked to optimal

cognition, physiology and behavior. Correlatively, BAX and BAK which promote apoptosis among cellular entities, when ablated in t cellular entities, produces Ca²⁺ signal processing impairment and impaired Ca²⁺ metabolism, impaired antigen enabled proliferation, dysfunctionl T Cellular Receptor function, dysfunctional Inositol processing, impaired IP3 enabled CA²⁺ mobilization, and dysfunctional endoplasmic reticulum function. Reenablement or reintroduction of BAX improved these statuses. TCR reliaent Ca²⁺ signals reintroduced by BAX reintroduction were observed to stimulate increased mitochondrial NADH synthesis at levels much higher than that utilized for or required for ATP synthesis. BAK and BAX were required for sustained overproduction of NADH. These demonstrate that proapoptosis pathways at least, but perhaps both proapoptotic and antiapoptotic survival signal promote upregulated NADH production, resulting in impairment of the ratio of hydride to hydrogen as well as disruption of the NAD⁺ to NADH ratio. However, NADH is not produced by the oxidative phosphorylation pathway, hydride is fracted from NADH to produce ATP in which 58 percent of energy from Hydride is utilized to fuel oxidative phosphorylation while 42 of hydridic is maintained between the phosphate groups of ATP. A likely scenario includes the potential that BAX upregulation and BAK increases are inhibiting Oxidative Phosphorylation as well as enabling mitochondrial membrane pores to open, enabling aggregation of NADH in the mitochondria because it is not being depleted by oxidative phosphorylation as well as enabling NADH to escape from the mitochondria through opening mitochondrial membrane permeability pores. A study confirms that BAX can inhibit oxidative phosphorylation. Molecular and cellular Biology. Volume 20. Number 10. Pages 3590

		<p>to 3596. 6th Month, 2000. PMCID PMC85651.</p> <p>These provide strong information that suggests homocysteine enabled upregulation of BAX and Cytochrome C release along with mitochondrial membrane permeability pore activation provide a persist pathway by which oxidative phosphorylation can be inhibited, ATP production de novo is inhibited, cellular respiration is diminish, and NADH may escape the mitochondria to be exhibited in other areas of intracellular environment as well as extracellular environment to promote pathology and supply substrate for pathogenic energetics.</p>
SP1	<p>Curcumin. Mithramycin A or Mith, Diterpenes(Coffee, Cafestol and Kahweol as sources). Tripterygium Wilfordii or triptolide(Toxic, requires inhibition of GSK3beta to prevent cytotoxicity) 154 percent increase in curcumin availability was observed in a study at about 2 hours after administration while in the first half hour piperine/chavacine together or piperine results in 2000 percent increase in absorption of curcumin. Curcumin formulations as curcumin phytosome formulation was 790 percent higher than curcumin, while mix of curcumin volatile oils increase availability by 130 percent, although formulation of curcumin with hydrophilic carrier, cellulosic derivatives and natural antioxidants resulted in a 4590 percent increased absorption of curcumin.</p>	<p>SP1 inhibitors. Mithramycin A. Daunorubicin. Doxorubicin. Hedamycin. Elsamicin A. Actinomycin D. Tolfenamic Acid. Aspirin. Arsenic Trioxide. Betulinic Acid. Curcumin. Resveratrol. 154 percent increase in curcumin availability was observed in a study at about 2 hours after administration while in the first half hour piperine/chavacine together or piperine results in 2000 percent increase in absorption of curcumin. Curcumin formulations as curcumin phytosome formulation was 790 percent higher than curcumin, while mix of curcumin volatile oils increase availability by 130 percent, although formulation of curcumin with hydrophilic carrier, cellulosic derivatives and natural antioxidants resulted in a 4590 percent increased absorption of curcumin.</p> <p>Lupus kidney disease is linked to SP1 activation of TGF b1, IL6, NFkB p65 domain and MCP1. The literature suggests that SLE patients should benefit remarkably from inhibition of SP1 by mithramycin as well s other small molecule inhibitors of SP1. Genetic causal origins should can be changed using CRISPR gene repair. Information. "SP1 Transcriptional Factor." American College of</p>

REST is a direct inhibitor of SP1 at the SYN1 promoter in tissues other than neuronal tissue although once differentiation of neuronal cellular entities occurs, there is a decrease in REST and stabilization of SP1 at SYN1 promoters. Information 288. Number 5. Pages 3227 to 3239. February PMID 23250796.

AP1, SP1, Homocysteine, iNOS and s adenosyl homocysteine are all causally link to the viral vector linked to the epidemiological patterns of 2020 and 2021 Betulinic acid inhibits SP1 expression exhibited in oncology through inhibition of sentrin specific protease 1, such that both Betulinic acid and mithramycin A inhibited pulmonary neoplasm expansion along with inhibiting SP1 expression of KRAS pulmonary oncology examples in small nonhuman mammals. Cyclin A2 is inhibited by Betulinic Acid and Mithramycin A, producing also diminished phosphorylation of retinoblastoma protein pRb, all of which resulted in cellular cycle pause at G2/M. Betulinic Acid produced these affects through inhibition of SP1 and SP1 was essential to antineoplasm activity of Betulinic Acid in this context Mol Pharmacol. Volume 82. Number 6. Pages 1115 to 1128. December, PMID 22956772.

Rheumatology, 2007 Annual Scientific Meeting.

Globin exhibits Heme and Globulins are functional blood proteins that do not always or typically exhibit Heme. Fetal globin exhibits different capacity for carrying oxygen than adult globin, while GATA1 upregulation and SP1 upregulation typically occur in adults compared fetal conditions where SP1 is decreased. Hemoglobin exhibits 1 Hemoglobin A and two Hemoglobin B moieties, while hemoglobin are erythroid cellular entity proteins being exhibited in erythrocytes in particular. Ferrous +2 iron, Fe, typically occupies 5 of 6 coordination loci, with four of these occurring in the porphyrin ring and with one loci being in the proximal histidine of the globin polypeptide. Oxygen redox occurs at the 6th proximal loci, which can be reduced by oxygen and also become oxidized to release oxygen. Hemoglobin at this 6th proximal loci can experience reduction and oxidation without chemical activity or ligand action which contrasts with cytochrome which participate in redox that is catalyzed by the oxidation status of the heme iron. Information. Cold Spring Harb Perspect Med. Volume 2. Number 12. Article a011627. 2012. PMID 23543078.

Systemic lupus erythematosus SLE can involve basic leucine zipper transcription factor cAMP responsive element modulator, CREM alpha, upregulation which suppresses IL2 and T cellular Zeta moiety, producing by integrating into the IL2 promoter regions. SP1 integrates into the CRME promoter to enhance CREM transcriptional activation. Protein phosphatase 2A activates SP1 by dephosphorylating SP1 at serine 59, producing enhanced affinity of SP1 for CREM promoter integration, producing a

Monoamine oxidase B inhibitors downregulate dopamine decomposition through antagonism of MAO B activity. SP1 upregulates production of MAO B through the SP1 promoter in the transcriptional sequences of MAO B in genome. SIP was upregulated by MPP + neurotoxin in SH SY5Y cellular entities. Mithramycin preventing MPP+ neurotoxin enabled increased in SP1 integration into the MAO B promoter, Mithramycin or ablation of SP1 mRNA translation resulting resilience in MPP+ neurotoxin exposure, preventing apoptosis among SH SY5Y cellular entities. MPTP instrumentation was also prevented from upregulating MAO B when Mithramycin A was provided before exposure. Particularly, Mithramycin A preventing deterioration of dopaminergic neurons in the substantia nigra pars compact along with preventing correlated behavioral impairment. The substantia nigra are neuronal versions which exhibit neuromelanin and which perform photosynthesis in ranges of energy or influence exhibited in the basal substance of the universes, outside of known electromagnetic spectra. The interactions occurring within the substantia nigra can include exchange of quantum level influences that are essential for spatial recognition, balance,

reliable homologue to pathology enabling metabolic change exhibited in SLE lupus conditions. Information. J Biol Chem. Volume 286. Number 3. Pages 1795 to 1801. 2011. . PMCID PMC3023474.

GATA1 is foundational pioneering and maintenance factor, and the reason for its upregulation in fetal conditions, among others, is that it is an activator of the KLF1 gene which encodes a version of the SP1 Specificity Protin, requires, in experimental conditions, only 1 functional allele in order to produce some erythroid proteins among which is B Globin, Globin B, Globin B, Globin Beta, Globin-B or Globin-Beta. Information. "Transcription Factors." Blood. 2014. Volume 123. Number 20. Pages 3080 to 3088. 2014. .

Inhibition of GATA1, KLF3, and SP1, as well as MYB, during Beta Globin gene expression can result in gamma globin gene expression and expression of fetal hemoglobin/Fetal Hemoglobin or HbF. This can potentiate therapeutic amelioration of conditions or circumstances involving impaired globulin. Information. Cellular J. Volume 17. Number 4. Pages 583 to 592. 2016.

SP1 and AP1 oppositely regulate Telomerase addition of repeats at the extremities of genes in genetic sequences. SP1 activates hTERT or telomerase reverse transcriptase. PMCID PMC4746408.

Omitting of the hyphens and underscores in this complete compendium of research are intend to standardize search capabilities are not intended to imply any detrimental indication, context or nuance. Microbiol Mol Biol Rev. Volume 66. Number 3. September, 2002.

AP1 suppresses the activity of hTERT, or telomerase reverse

	<p>coordination of movement and other functions that are involved in extrasensory interactions with environment and the Universes. Parkinson's Disease, thus, involves or can involve SP1 upregulation of MAO B, thereby producing a dissociation of neuronal capacitance and sensory function from photosynthesis and other activity occurring within and outside of known electromagnetic spectra. J Neurosci Res. Volume 96. Number 10. Pages 1663 to 1676. October, 2018. PMID 3004136.</p>	<p>transcriptase. It should be presented here that telomerase replacement or repair can occur through the activities of ALT. Molecular and cellular Biology. Volume 25. Number 18. Pages 8037 to 8043. October, 2005. PMID 12334330.</p>
AP1	<p>AP1 should be considered along with the MAPK, PI3K and Wnt status in the neoplasm or oncology microenvironment. Inhibiting these or modulating these in the microenvironment can result in even more substantial therapeutic effect by affecting the AP1 cascade directly. Neoplasms. Basel. Volume 11. Number 7. Page 1037. July, 2019. PMCID 31340499.</p> <p>AP1 is a transcription activator and immune checkpoint regulator, activating PD1 and PD L1 transactions, but also can have its cascade modulated to modulate immunological checkpoint function. The PD1 inhibitor dostarlimab has produced nearly 100 percent remission of colorectal oncology in a limited size clinical study.</p>	<p>AP1 inhibitors such as Celecoxib, retinoid SR 11302, is a vitamin of retinol.</p> <p>SP1 and The literature observes that AP1 oppositely regulate Telomerase addition produces upregulation of repeats at choline kinase alpha. This pivotal linkage suggests that the extremities of genes AP1 moiety accompanying cFos has an integration locus in genetic the Choline Kinase Promoter beginning at position negative 875 for 11 or 12 sequences. SP1 activates hTERT or telomerase reverse transcriptase. in the negative direction. Information. Microbiol Mol Biol Rev. Biochim Biophys Acta. Volume 661 171. Number 3. September, 2002.</p> <p>AP1 suppresses the activity of hTERT, or telomerase reverse transcriptase. It should be presented here that telomerase replacement or repair can occur through the activities of ALT. Molecular and cellular Biology. Volume 25. Number 18. Pages 8037 to 8043. October, 2005. PMID 12334330. PMID 17728180.</p>

	<p>Information. NEJM. 6th Month, 5th Day, 2022.</p> <p>Berberine. Mormordin I.</p> <p>Therapeutic inhibition of AP1 has been observed to cause impaired, diseased or oncology exhibiting cellular entities to regress from pathogenic status until exhibiting near embryonic plasticity.</p>	
C-Reactive Protein	<p>Curcumin, Bok Choy, Beets, Pineapple and Celery. 154 percent increase in curcumin availability was observed in a study at about 2 hours after administration while in the first half hour piperine/chavacine together or piperine results in 2000 percent increase in absorption of curcumin. Curcumin formulations as curcumin phytosome formulation was 790 percent higher than curcumin, while mix of curcumin volatile oils increase availability by 130 percent, although formulation of curcumin with hydrophilic carrier, cellulosic derivatives and natural antioxidants resulted in a 4590 percent increased absorption of curcumin.</p>	<p>C-Reactive Protein Inhibitors. Aspirin, Rofecoxib, celecoxib, clopidogrel, abciximab, statins, ezetimibe, niacin, fenofibrate, vitamin E, beta adrenoreceptor receptor antagonists, ramipril, fosinopril, captopril, valsartan, irbesartan, olmeartan, telmisartan, rosiglitazone, pioglitazone, all are known to inhibit C Reactive Protein.</p>
Monocyte Chemoattractant Protein 1	<p>Grapeseed Extract, Apigenin, 1,25-Dihydroxyvitamin D3 Ablation of PD1 expression results in increased tissue infiltration by Monocyte Chemoattractant Protein 1 or MCP1.</p>	<p>33 DMB, Fenofibrate, Clofibrate, Aspirin</p>

Nitrosamine	Garcinia Kola Seed extract	Kolaviron
Homocysteine Thiolactone	<p>Through PON1 by a number of factors.</p> <p>PON1 Translocation through SREBP2 and SP1 integration at the PON1 promoter occurs resultant of Statin, Quercetin and Glucose.</p> <p>PON1 activation through the aryl hydrocarbon receptor occurs resultant of Quercetin, Resveratrol and Aspirin utilization.</p> <p>Berberine, however, induces PON1 through the JNK-c-JUN signaling pathway.</p> <p>Resveratrol is a phytoalexin. trans 3,4,5,4'-tetramethoxystilbene.</p> <p>Pomegranate juice polyphenolics stimulate PON1 expression through the PPARγ-PKA-cAMP signaling pathway.</p> <p>Unknown mechanisms of action enable PON1 upregulation resultant of utilizing Curcumin, Betanin, Isothiocyanates, Licorice Polyphenolics, and olive oil.</p>	Enlyte/EnlyteRx
Homocysteic Acid	<p>Saline along with Alkalinization Therapy.</p> <p>Vitamin K1 and Vitamin K2 as Menaquinone-4.</p> <p>NMDA Receptor inhibitors</p>	Enlyte/EnlyteRx
<p>Inducible Nitric Oxide Synthase. Alleviation of EMF causing Ponderomotive Force.</p> <p>EMF=>Ponderomotive Force (also used as Brachytherapy for neoplasm) causes asymmetric vascular flow, slower flow near vascular wall, deposits of solute and material, vascular pathology. These are involved in all detrimental behavioral/physiological outcomes. iNOS is considered to be the cause of septic shock because microbes pervasively stimulate its expression, although methylglyoxal is also causal indicator of exhibition of septic shock. The involvement of</p>	<p>Curcumin, Irinotecan, other, EMF protection for Homes, communications, phones, electrical infrastructure, wireless Networks, Devices, Wireless Devices, etc.</p> <p>Filtered water without Chlorine/Fluorine/Particulates. Atmospheric cleansing devoid of</p>	<p>iNOS inhibitors. L – canavanine, aminoguanidine, methylguanidine. Transcription iNOS inhibitors and post translational iNOS inhibitors.</p> <p>pyrimidineimidazole-based allosteric dimerization inhibitors of iNOS are emerging as strong and useful inhibitors of iNOS. The diterpenoide lactone derived from plants knowns as andrographolide is an effective inhibitor of iNOS and it</p>

iNOS in pervasively pathology and behavior since the 1700s when environmental particulate, electricity and wireless communications began to emerge, has expanded the causes and exacerbation of pathogenic processes and conditions, interestingly enhancing the expression of iNOS which accompanies sepsis that occurred before such exacerbation and enhancing the expression of iNOS that accompanies PEMT and homocysteine which, before 1800s were the pervasive causes of detrimental human outcomes. Information. Gen. Pharmacol. Volume 29. Number 2. Paes 159 through 166. 8th Month, 1997. PMID 9251894. Information. Biochim Biophys Acta. Volume 14211. Number 2 and Number 3. Pages 437 through 455. May 5, 1999.

Inducible Nitric Oxide synthase is presented in the literature as greenhouse gas that can be cleaned from power plant exhaust and automotive exhaust by methane. This suggest that the massive increases in expression of iNOS among human populations may be a factor in global warming and atmospheric particulate increases. Correlatively, the literature observes that extreme low frequency electromagnetic energy increases or exacerbates iNOS expression levels in diverse organisms include a diverse array of mammalian and nonmammalian organisms. Similarly, another study observes exposure to medium and high powered electromagnetic energy in plants as well as mammals and other organisms. In diverse aspects of the tree of biological taxonomy, iNOS is expressed as injury of distress, impairment, injury and harm, including being expressed with wounds, distress, drought, structural impairment, disease or in other instances. These clearly present that electricity, electromagnetic, satellite, wireless fields and communications, cause harm to biological tissues, organisms of diverse nature and to the environment. iNOS expression is hidden factor in pervasive disease and pathogenic processes as well as detrimental behavior, and conditions or factors linked to aging. Information. Biomed Res Int. Volume 2016. Article 1830262. 2016. Information. Journal of Experimental Botany. Volume 72. Issue 3. Pages 777 through 7780. February 11, 2020. Information. Pathophysiology. Volume 7. Number 2. Pages 131 through 135. July, 2000. "Power Plant Emission." ScienceDaily. ScienceDaily. March 2, 2000.

Plant nutritional content, development, and metabolism were all observed to become impaired and diminished by the modalities of electromagnetic energy exposure produced by

toxic particulates. Hydroxychloroquine. Using EMF protection stickers, blankets, clothing, coverings, material, curtains, etc. Covering electrical power outlets. Turning off wireless networks, communications and other devices. Removing personal information, locations, telemetry services, names, communication information, addresses, etc from internet connected systems and from systems not connected to internet. Avoiding Restraint. Using Filtered water without Chlorine, Fluorine, Nitrosamine, or toxic factors. Avoid extreme low frequency sound and magnetic fields. Obtain 800 mg+ or Between 7 and 4 mg of Choline each day. Coat communications and electrical infrastructure in environment and dwellings. Improve atmospheric population or particulate factors. 154 percent increase in curcumin availability was observed in a study at about 2 hours after administration while in the first half hour piperine/chavacine together or piperine results in 2000 percent increase in absorption of curcumin. Curcumin formulations as curcumin phytosome formulation was 790 percent higher than curcumin, while mix of curcumin volatile oils increase availability by 130 percent, although

was strongly effective in cervical tissue, preventing infections otherwise, inflammation otherwise, but specifically useful in preventing and alleviating proliferation and migration of oncology cultures while also introducing apoptosis, all with increasing effectiveness in correlation with increasing dosage. Oxid Med Cell Longev. 2021 Mar 16. 2021. Article 6692628.

iNOS is independently prognostic in intrahepatic cholangiocarcinoma. Information Journal of Oncology Management and Research. 8th Month, 26th Day, 2019. Volume 2019. Number 11. Pages 8005 to 8022.

Sound inhibiting structures, windows and other capabilities may also be essential to prevent extreme low frequency or extreme high frequency influences from causing iNOS.

Electromagnetic Energy, Wireless, Sound, and atmospheric particulate as well as environmental particulate safety modalities including care facilities, care rooms, buildings, Safe rooms in homes, safe rooms in buildings, all can provide essential improved ability to stabilize health and behavior.

Technology and communications devices which do not use VPN, do not use firewalls with explicit securing of all applications and communications, which are uncovered by EMF inhibiting devices and which use unnecessary protocols, all can add to EMF exposure levels.

Automobiles, other transportation or other influence emitting loud noises can contributed to both mechanical and iNOS as well as other levels impairment that are cumulative in this regard.

Wireless communications in most nay regard contributed to iNOS and phospholipase D, particularly

<p>civilizations. These present a diverse and encompassing deterioration of nutritional density in the food pyramid produced by artificial electromagnetic energy of diverse modalities and characteristics. Information. Biomed Res Int. Volume 2016. Article 1830262. 2016. PMID 26981524. Information. "Effect of RF Electromagnetic Field on Cucumber and Tomato Plants." Sultan Qaboos University. PC 123, P.B. 33. Online Information. www.researchgate.net/publication/311256455 Information. 978-1-5090-0996-1/16 IEEE. 2016.</p>	<p>formulation of curcumin with hydrophilic carrier, cellulosic derivatives and natural antioxidants resulted in a 4590 percent increased absorption of curcumin.</p> <p>Ethyl acetate extract from <i>Asparagus cochinchinensis</i> inhibit COX, iNOS, inflammatory Cytokines, as well as modulates Map Kinase Pathways, regulates cellular cycle and produces antioxidant activity. Information. Molecular Medicine Reports. Volume 15. Issue 4. April, 2017.</p> <p>Apigenin inhibits the STAT1/COX-2/iNOS signaling pathway and is therapeutic for multiple myeloma, causing apoptosis, cellular cycle pause, autophagy and ferroptosis in NCI – H929 cellular entities. Phytomedicine. 2021. Volume 80. Page 153371.</p>	<p>luminal expression of Phospholipase D and iNOS in which the role of physiology in protecting emerging, development and developed nuances of physiology, endothelium and other areas, producing conditions which physiology did not intend to occur and may not be equipped to manage efficiently, producing potential pervasive nuances of pathology.</p> <p>Combination therapy using iNOS inhibition along with inhibitors of epithelial to mesenchymal transition factors Snail, Slug, Zeb1 and Twist1 results in enhanced effectiveness of iNOS inhibition in triple negative oncology of the breast. A study found an integral and consistent correlation between iNOS expression, prognosis, and outcomes in triple negative breast oncology1. Page 25. FebruaryPMID 25849745.</p> <p>Ubiquitination of P53 by HPV E6 protein linked to AP protein to produces a E6/AP/P53 complex, results in proteasome deterioration of P53 to produce enhanced risk in HPV conditions. P53 can often be a focus for pathogens and pathogenic factors because P53 promotes apoptosis or senescence that can disrupt pathogenic processes. Cervical carcinoma resultant of HPV can be regressed and prevented using the small molecule E6 inhibitor NUTLIN as well as NSC 652287 5,5'-(2,5-furandiyl)bis-2-thiophenemethanol, both inhibit MDM2 enabled ubiquitination of P53, rescuing deterioration of P53 that is required for many diseases, HPV oncology enabled by E6/AP/P53 complex. Cat. No. 2443. Products. Tocris.com. Pubchem CID 374536. PLoS Comput Biol. Volume 10 Article e1003991 Open Virol J. Volume 5. Pages 80 to 95. iNOS levels are correlated with high risk HPV viral vectors and prognosis. PMID 22420338. Carrageenan is effective as therapy for HPV viral disease while</p>
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Curcumin also promotes cytotoxicity to cervical oncology linked to HPV viral conditions, particularly through inhibition of Inducible Nitric Oxide Synthase. Sex Transm Dis. Volume 48. Number 7. Pages 458 to 465. Pages 458 to 465. July 1, PMID 33433173. Mol ‘Cellular’ Biochem. Volume 325. Number 1 and Number 2. Beginning with Page 107. 5th Month, PMID 19191001083. Numbers 3 and 4. Pages 311 to 318. March and April,

HPV viral conditions present the ability for a discernibly linked pathology emerged from the Viral vector. HIV is highly correlated with iNOS expression and both expression of iNOS and uncoupling of iNOS, presented in studies, is required for continuous exhibition of HIV pathology. The HIV protease inhibitor Saquinavir fused to Nitric Oxide which is the product of coupled nitric oxide production, results in powerful cytotoxicity toward melanoma. LNAME was also powerful in its ability to deteriorate melanoma. LNAME is an inhibitor of iNOS and nitric oxide is the produce of coupled or unimpaired nitric oxide synthase catalysis. Clearly, inhibition of iNOS should be integral nuances of therapy in viral conditions including HPV, HIV, and even respiratory viral vectors such as that which is involved din the epidemiological events od 2020 and 2021. Of the diverse viral vectors reviewed by this compendium of research, iNOS was required to be modulated in some way by each of these viral vectorsOncol Rep. Volume 28. Number 2. Pages 682 to 688. 8th Month, 20212. PMID 22665020Med Res Rev. Volume 40. Number 1. Pages 158 to 189. Jan 2020. PMID 31192483.

Inhibitors of Phospholipase D are also recommended because the combination of iNOS and Phospholipase D enables viral vectors being impaired by the

reactive molecular species in the plasma membrane interstitial space to escape in in endosomes to become exhibited in the cytoplasm. Protection from wireless fields, communications fields, electricity fields, and atmospheric particular are also recommended during the therapeutic period. HPV is known to be upregulated by HPV 16 E7, promoting resistance of HPV to rapamycin that is correlated with pRb status.

iNOS and HPV are features of environmental wireless communications, electricity fields that are unshielded, and atmospheric particulate, while diseases linked to HPV or other viral vectors are enhanced because strong electromagnetic fields enabled iNOS and phospholipase D express in the epithelium and endothelium of lumina which are enclosures deep in tissues that are intended to be shielded from electromagnetic energy by the architecture of physiology. Phospholipase D upregulates phosphatidylcholine specific phospholipase C which deteriorates the most abundant phospholipid in cellular membranes and physiology, as well as deteriorates the pulmonary surfactant dipalmitoyl phosphatidylcholine which is essential to easing surface tension in the alveolar lumina of pulmonary tissue to enable improved exchange of Oxygen and exchange of CO₂. BMC Oncology. Volume 18. Number 1. Pages 485. 5th Month, 27th Day, 2018. Some pathogens emit their own phospholipase D which then perform catabolism of dipalmitoylphosphatidylcholine.

A new way of producing iNOS inhibitors is presented in this study of anchored plasticity inhibitor design. Nature Chemical Biology. Volume 4. Pages 700 to 707. Pages October 12,

viral vector linked to the epidemiological patterns of 2021

and 2022 imparts at least some of its pathology through iNOS expression. It should be presented also that viral vectors typically utilize iNOS to deactivate the immunological system and impart increasing levels of pathology similar to most if not all diseases when these become most detrimental to outcomes. Melatonin and curcumin inhibit iNOS similarly to curcumin and some studies present melatonin as well as nitric oxide instrumentation to replace and enhance that which would be provided by functional iNOS, eNOS or nNOS, is a significant therapy to improve outcomes from the viral vector linked to the epidemiological patterns of 2020 and

FASEB J. Volume 12Pages 685 to 693. June, 1998. PMID 9619447.

Life Sci. Volume 250. Number 117583. 6th month, 2020. PMID32217117.

Information. Food Sci Nutr. Volume 8 Number 10. Pages 5215 to 5227. September, PMID 33133525.

Curcumin inhibits Herpes, HIV , influenza a, HPV, hepatitis and other viruses, for a number of reason, although inhibition of iNOS is a central factor. Plant Medicine Research. “Curcumin from turmeric inhibits zika, HIV, Herpes, and other viruses”

Curcumin impairs replication of HIV1 and HIV2 and prevents HIV traversal of the genital mucosal epithelial barrier. Turmeric curcumin inhibits entry of all Hepatitis C variants into human hepatic cellular entities. Basant Polyherbal cream with curcumin, reetha, amla, aloe vera, as well as curcumin alone produce increased rates of clearance of HPV cervical affliction with basant polyherbal cream producing 83 percent clearance compare to placebo 73 percent and curcumin individually producing 81 percent clearance compared to 73 percent placebo. Enveloped viruses generally are inhibited by curcumin and this disruption included disruption and

leakage of liposomes used to encapsulate viruses, confirming that curcumin disrupts the escape of viral vectors from the plasma interstitial space that is enabled by iNOS and phospholipase D as well as phosphatidylcholine specific phospholipase C in which viruses escape into the intracellular spaces in endosomes produced from phospholipase D budding and iNOS production of escape conditions. These also explain similar process which might be occurring in the lumina or lumen. Plaque formation from enveloped viruses was inhibited by curcumin also. Influenza, vaccinia, herpes, hepatitis c, coxsackievirus B3, and other viruses were assayed among enveloped viruses with a therapeutics response that inhibited PI3K, PKB, MAPK, hemagglutinin, nf kb, UPS ubiquitinases and other factors. HIV1 integrase, Hepatitis B virus PGC1 alpha, Influenza virus pathology aggregately, and Influenza viral haemagglutination activity, all are inhibited by curcumin in particular.

Virology. Volume 373. Volume 2. Pages 239 to 247 Food Chem. Volume 119. Pages 1346 to 1351. FEBS Lett. Volume 584. Volume 11. Pages 2485 to 2490. Information. Biochem Pharmacol. Volume 49. Pages 1165 to 1170. April, 1995. FEBS J. Volume 280. Number 22. Pages 5829 to 5840. November, 2013. J Natl Med Associ. Volume 88. Number 6. Pages 333. 6th Month, 1996. Information. Food Funct. Pages 3412 to 3419. November, 2015. Information. PLoS One. Volume 8, Number 5. Pages e62482. May, Asian Pac J Cancer Prev. Volume 14. Number 10. Pages 5753 to 5759. Information. Gut. Volume 63. Number 7. Pages 1137 to 1149. July, Information. PLoS One. Volume 10. Number 4. Pages e0124903. April, 2015. Information. Volume 10. Number 6. Pages 27539. 6th month, Antiviral

Res. Volume 142. Pages 148 to 157. 6th month, 2017.

Glycolic acid is used as the basis to which other factors are integrated in production of viral adjuvants, particularly then the viral particles exhibit glycoprotein envelopes. Glycolic centered nanoparticle encapsulated inactivated porcine reproductive virus and porcine respiratory virus produces an effective comprehensive protective response in porcine populations. Glycolic Acid inactivates reptilian venom activation of PI metalloproteinase, and prevents BaP1 from causing enzymatic, hemorrhagic and edema conditions. Acquired epidermodysplasia verruciformis or EV and AEV linked to HPV viral conditions can be improved by Topical Glycolic Acid, Cidifor, HPV 9 valent vaccine, Acitretin, topical imiquimod, topical retinoids, cellular immunity improvement or changing transplant medication to mycophenolate mofetil. This presents clearly that glycolic acid, or methylthioglycolic acid may participate in viral protein proteolysis, tissue resection and desquamation, and in generally xenobiotic response as strong antihistamine. These present some of the ways in which withholding of dimethylacetothetin, sulfur deficiency and inactivation of the thiol homocysteine methyltransferase has been detrimental. Flat warts may be cleared using Glycolic Acid Lotion, as verified by clinical studies of 100 percent clearance among all patients. J Clin Aesthet Dermatol. Volume 4 Pages 62 to 64. September, PMCID PMC3175803 Toxicon. Volume 71. Pages 41 to 48. September, 2013. International journal of Nanomedicine 1. Pages 670 to 694. January, 2014. Vaccines (Basel). Volume 9. Number 4. Pages 310. 4th Month, 2021. PMID 33805880.

Int J Dermatol. 8th Month, 2021. PMID 34403500.Medscape. Article 750286.

Curcumin exhibits antibacterial activity toward staphylococcus versions, pseudomonas and streptococcus versions. Front Microbiol Volume 10. Number 912. 2019. PMID 31130924.

Catechin and Curcumin integrate with S protein and host receptor ACE2, both essential aspects of pathology and cellular access for the virus linked to the epidemiological events of 2020 and 2021, as well as integrates with the S Protein after it complexes with ACE2, the fusion point for the viral vector's access to the cellular environment. At least one study recommends catechin and curcumin as a therapeutic strategy for the viral vector linked to the epidemiological events of 2020 and 2021. Volume 11. Article 2043. 2021.

Curcumin prevents pneumonia from advancing to levels which are detrimental to vital being as well as downregulate Acute Respiratory Distress Syndrome by inhibiting the Nf kB Inflammasome. Information. Heliyon Volume 7. E06350. 2021.

Favipiravir exhibits improved outcomes among those receiving care for the viral vector linked to the epidemiological patterns of 2020 and 2021. Transfer of the HSV Thymidine Kinase protein into HPV16 cellular entities sensitizes these cellular entities and produces 85 to 90 percent level of apoptosis when exposed to Gancyclovir or Acyclovir. Hum Gen Ther. Volume 14. Number 1. Pages 45 to 47. January, 2003.

Probiotic usage increases chances of HPV clearance by about 50 percent. These clearly present inflammasome, iNOS, trimethylaminenoxide, phospholipase D, phosphatidylcholine specific phospholipase C, inhibited PEMT and other factors are important in enabling HPV viral conditions to emerge, persist, progress, impart

detriment and elude physiological and pharmacological capabilitiesEur J Cancer Prev. Volume 22. Number 1. Pages 46 to 51. January, 2013. ‘’ A study of emerging oncological conditions in the cervix implemented a vaccine that can be instrumented after affliction by HPV, resulting in as much as 36 percent of those exhibiting emerging HPV oncology in the cervix becoming cleared of such affliction and cleared HPV Viral affliction. The therapy is known as Tapkinogen or Sovacivec. Gynecologic Oncology. 4th month, 4th Day, 2019.

HSPe7 immunotherapy remarkably reduces warts in HPV afflictionDiseases of the Colon and Rectum. Volume 45.

Photodynamic Therapy using theta aminoleulinic acid was utilized to eradicated pathogenic oncological lesions in 83 percent of participants, cleared HPV affliction in 80 percent of patients whom were exhibiting oncology, regressed 57 percent of patients with oncologyVolume 93. Number 5. Pages 1269 to 1275. October, 2017.

Berberine completed inhibits 6 tested HIV1 isolates. Berberine in Obatoclax both individually inhibit the viral vector linked to the epidemiological events of 2020 and 2021 at low molecular levels. Berberine suppresses HPV Viral vectors, and causes apoptosis and abated development in cervical oncology resultant of HPV. The literature is considering presentation of berberine is a broadly active antiviral. The mechanism of action for berberine activity is presented as inhibition of AP!, but berberine is active in inhibiting a number of essential inflammatory cytokines required in diverse pathology. However, to summarize, most of the factors presented in therapeutic lists within the translationalwellness.com material produce beneficial affect to oncology, disease and other detrimental outcomes when these

		<p>factors are prevented or alleviated. Information. AMB Express. Volume 10. Number 1. Page 164. September 8, 2020. Information. "Berberine and Obatoclox." Viruses. Volume 13. 2020. Information. FASEB J. Volume 35. Number 4. Article e21360. 2021. PMID 33749932. Information. "Berberine modulates AP1". Molecular Oncology. Volume 10. Number 39.</p> <p>Lidocaine is an inhibitor of iNOS and inhibits Cationic Amino Acid Transport 2. Information. Anesthesia and Analgesia. Volume 102. Number 6. Pages 1939 to 1744. 2006.</p>
Uncoupled Nitric Oxide Synthase	<p>Uncoupled NOS is managed with L-Arginine, Ca²⁺(sometimes atypically), Iron, Tetrahydrobiopterin, Superoxide Dismutase, Catalase, N-Acetyl L Cysteine, Vitamin C. Peroxiredoxin, Reduced glutathione, glutathione. Sulfur such as methylsulfonylmethane. . Information <u>Nutrients</u>. 2017 Mar; 9. 3. 290.</p>	<p>Sapropterin, Calcium, Iron.</p> <p>Uncoupling of Nitric Oxide Synthase occurs when substrate for Nitric Oxide Synthase is depleted. The literature observes that Ca²⁺ substrate for iNOS may not cause uncoupling because Ca²⁺ depletion by iNOS deprives eNOS and nNOS Ca²⁺ which deactivates eNOS and nNOS. eNOS and nNOS do not have calmodulin and its four loci of Ca²⁺ sequestration already integrated into their structure. iNOS does have Calmodulin integrated into its structure and thus continues to depleted Ca²⁺ constitutively with exhibition of iNOS. Calmodulin activation and sequestration of Ca²⁺ occurs before or independent of integration of Calmodulin into eNOS or nNOS.</p> <p>eNOS is essential to Caveolae function because it is essential, when functional, to the dilatation of the Caveolae and enabling the signal transduction, secretion and signal reception center of the cellular entity to be open for interactions with physiology.</p> <p>Uncoupling of iNOS is required for some viral pathology and function, such as HIV which requires iNOS</p>

		<p>and requires uncoupling of iNOS to exhibit pathology.</p> <p>Uncoupled iNOS or uncoupled NOS generally, results in production of reactive oxygen species and reactive molecular species cascade, beginning with Superoxide, followed by superoxide dismutase production of H₂O₂ from superoxide, followed by production catalase production of water from H₂O₂, but includes intermittent production of Nitric Oxide as uncoupled status modulates to coupled status and back to uncoupled status. This cycling produces peroxynitrite while hypochlorite can also be produced and while these can culminate in production of trimethylaminenoxide, comprising a metabolic syndrome and the molecular elements of an infarct.</p> <p>Homocysteine and peroxynitrite are strong activators of biological molecules and homocysteine deactivates catalase as well as a diverse group of other molecules essential for biological function.</p> <p>L – arginine is a primary factor in uncoupling although other substrate and cofactors for iNOS in particular can promote uncoupling when inadequate. iNOS uncoupling and other areas in this information discuss iNOS and uncoupling in comparative contexts with more information.</p>
TrimethylaminenoxideTrimethylamine-N-Oxide	<p>Grapeseed Extract, Olive Oil, 33DMB, Pro/Pre/Post Biotic, Broad- Spectrum Antibiotic, all manage Trimethylamine-N-Oxide while management of Trimethylamine-N-Oxide is the only way known to improve Carotid Intima Media Thickness other than mechanical intervention.</p>	<p>3,3 DMB</p> <p>Trimethylaminenoxide is produced by uncoupled nitric oxide cascade or resultant of digestive pathway processing of trimethylamine as well as resultant of trimethylamine transiting the digestive pathway membranes to the hepatic organ where flavin monooxygenases metabolize trimethylamine to trimethylaminenoxide.</p> <p>Trimethylaminenoxide is the most pervasive and precise cause of stroke. Tissue plasminogen</p>

activator, or other clotbusting thrombolytics, manage exhibition of a thrombus, but both trimethylaminenoxide and homocysteine coordinate to produce pathogen coagulation, although creactive protein and other inflammatory processes perform un such capacity also. Upregulated choline kinase provides phosphocholine as a feed forward supplier of material for pathology and phosphocholine can activate platelets as well produce low level persistent activation of inflammatory components of immunological function.

Trimethylaminenoxide also is a primary factor in deterioration of carotid intima media plasticity and in order to improve carotid intima media plasticity, it is essential to manage trimethylaminenoxide.

Trimethylaminenoxide can coordinate with homocysteine to produce resiliency to some adverse health events.

Trimethylaminenoxide transits the digestive pathway membranes in a manner that is enabled by tn timer alpha which causes relaxation of tight junction proteins that would typically otherwise diminish digestive membrane permeability.

Trimethylamine lyase is an enzyme exhibited by less than optimal digestive pathway microflora to produce trimethylaminenoxide.

Periodic implementation of a laxative and excluding meat, chicken, eggs or fish from nutrition as well as fasting can all enhance the chronic or increasing levels of trimethylaminenoxide. Post/pre/anti biotics all can improve digestive pathway microflora. Grapeseed oil, olive oil, balsamic vinegar, probiotic foods, and living foods, all can be improved trimethylaminenoxide characteristics.

IDO or Indoleamine 2,3 Dioxygenase	Red Sage or Danshen, while other natural inhibitors abound. Tanshinone is an inhibitor of IDO.	IDO Inhibitors such as Indoximod, Epacadostat and Navoximod are inhibitors of IDO.
Methylglyoxal and Oxalate	Water, N Acetyl L Cysteine, L-Arginine, and Magnesium as well as Potassium, typically as Chloride version of the Metals, can be utilized to manage. Kidney Stuff by Golden Standards. Magnesium Citrate. Calcium Citrate, Pyridoxine, Vitamin E therapy for Methylglyoxal. Sulforaphane, L-arginine, Pyridoxamine, gallic factors, phenolic factors, and aminoguanidine for Methylglyoxal. Hyperoxaluria changes more than 500 metabolic factors toward oncology or other pathology. Methylglyoxal is considered among the best indicators of septicemia or septic shock, although iNOS is considered to be the manifestation of septicemic shock or septic shock.	Improvement Renal Function and Hepatic Function. DCR-PHXC or Nedosiran for Oxalate, which performs inhibition of Lactate Dehydrogenase A and can alleviate metabolic acidosis/alkalosis while also diminish depletion supply of NAD+ to PARP for PARP signaling through preventing pyruvate sequestration to lactate anion because this Lactate Dehydrogenase activity translates NADH to NAD+. Aldose Reductase inhibitors of Methylglyoxal, such as Alrestatin. Epalrestat. Fidarestat. Imirestat. Lidorestat. Minalrestat. Ponalrestat. Ranirestat.
D Lactate/Lactic Acid and L Lactate/Lactic Acid	Managemanage Methylglyoxal, Homocysteine, Oxalate, S-Adenosyl Homocysteine, Trimethylamine-N-Oxide, Uncoupled NOS, and iNOS, including managing Choline availability to prevent these from becoming factors.	Ringer's Solution and Methylene Blue, other factors in this list including Carbicarb. L lactate and D Lactate inhibition Histone Deacetylases HDACs and produce histone H3 and H4 hyper acetylation while also increasing LIG4 APTS and NBS1 expression in a way that enhances both DNA Pcs activity and in a way which enhances the levels of DNA repair. Inhibition of HCAR1 expression on the cellular surface diminishes this enhanced level of DNA repair, particularly when this inhibition of HCAR1 surface expression and activation occurs resultant of pertussis toxin exposure which is the causal pathogen in exhibition of

		<p>whooping cough. Importantly, this DNA repair enhancement by D Lactate and L Lactate promotes resistance in oncology of the cervix of the uterine organ. Information. Cellular Commun Signal. Volume 13. Number 36. July 25, 2015. Ringer's Solution and Methylene Blue, other factors in this list</p>
Genetic Anomaly and Gene Impairment	<p>Ribose Supplement, RNA Nucleotide Supplement, Supplemental DNA Nucleotides, supplementation of the substrate and product of the Gene. NAD⁺, NADH, Pyruvate, Uridine, NADH precursors (Vitamin B3, Nicotinic Acid/Niacin[causes flushing and not effective in all tissues], Nicotinamide/Niacinamide[widely effective but inhibits sirtuin activity], Nicotinamide Riboside[optimal and manages insulin resistance/sensitivity], Nicotinamide Mononucleotide[optimal, available in all tissues, activates SIRT1, does not inhibit sirtuins, has highest bioavailability in circulatory pathways and increases NAD in the intracellular environment the most among these other factors]). CD38 is major depletion pathway for NAD and CD38 is major factor in depletion of NAD resultant of and causal for senescence and detrimental factors associated with aging. Taxifolin, dihydroquercetin, Apigenin, Luteolin, which are supplements, as well as Callistephin found in deep/dark blue</p>	<p>CRISPR Perfect Gene Repair fused to Protein Transduction domains to assure 100 percent saturation of tissues and cellular entities during genomic repair. DNA repair occurs more than 1 million times each day in every cellular entity. DNA repair causes PARP to attach at loci of DNA impairment and persistently signal by catabolizing the ribose from NAD⁺ to distribute the ribose to molecules near PARP, producing a gradient upon which substrate for DNA repair is recruited to the locus of DNA impairment. DNA and deoxyribonucleotides are among the factors recruited to the locus of DNA impairment. The persistent PARP signaling occurs until DNA repair has been completed, while adequate NAD⁺ enables the polymerase PARP to detach from the locus of DNA repair, resulting in optimal Homologous Repair that is considered to be free of errors. Inadequate NAD⁺ and inadequate Deoxyribonucleotides produces error prone, paused, and ablated DNA repair, deteriorating the genome. However, the depletion of NAD⁺ causes available NADH to be translated into NAD⁺ often after enzymes enabled bidirectional exchange of NADH/NAD⁺ become excluded from metabolic pathways, deactivated or diminished in function. PEMT inhibition upregulates P53 and P53 inhibits entry of Glucose into Glycolysis, downregulating glycolysis to cause diminished availability of Pyruvate for translation into lactate in a way that enables NADH to become NAD⁺. Diminished entry of Glucose into the pentose phosphate pathway occurs in tandem with inhibited Glycolysis, resulting in depletion of NADPH by more than</p>

nutritional factor or foods such as blueberries, wine grapes, and pomegranates, and as well as Kuromaninis found in black currant, red raspberries, Peruvian corn and lychees. Flavonoid and Anthocyanin rich foods are recommended in this context such as black rice, blood oranges, red onions, red cabbage, acacia, black plums, etc. however, experimentally, the most rapid mechanism and most assured way to change NADH to NAD⁺ ratio in which NAD⁺ is required to be very high compared to NADH to potentiate release of H⁺ is to manipulate the ratio of pyruvate to lactate. Lactate Dehydrogenase translates pyruvate and NADH to lactate and NAD⁺. The cellular processes the mine for energy factors often mine for and release the hydridic energy factors which PEMT integrates into physiological structure when it integrates the three methyl groups first in enriched phosphatidylethanolamine to produce phosphatidylmonomethylethanolamine, then integration of another CH₃ into phosphatidylethanolamine to produce phosphatidyl dimethylethanolamine, and then into phosphatidyl dimethylethanolamine to produce enriched versions of phosphatidylcholine. Each methyl group,

60 percent and diminishing pentose phosphate pathway production of five carbon sugars used in Nucleotide synthesis. These inadequacies impair availability of NADH. NAD⁺, Nucleotides and Pyruvate, all in a way that promotes crisis in the diverse shared pathways for pyruvate, such that PARP signaling occurs persistently and produces a metabolic syndrome known as parthanatos in which already differentiate cellular entities incur apoptosis and emerging pluripotent or multipotent stem cellular entities exhibit and impaired ability to experience apoptosis. These are the canonical conditions for exhibition of disease, metabolic syndrome, DNA impairment and oncology.

PARS signaling occurs similarly to PARP signaling, such that areas where protein transactivation or translation of DNA into proteins is occur exhibits increased levels and increased instances in which levels of substrate utilized in polymerase activities are elevated. This produces an enhanced possibility for substrate such as NAD⁺ to already be exhibited for DNA repair in area where PARS is active or where PARS has been active. Importantly, these suggest that areas where DNA is being decoded have enhanced possibility for more efficient DNA repair. Importantly, also, noncoding DNA may have increased levels of changes in a way that allows such change to more adequately track human experience such as the exhibition of high levels of viral DNA within Human DNA or the exhibition of genetic change resultant of environmental or other conditions. These changes provide the possibility that when a particular, no longer extant factor or environmental condition emerges, noncoding DNA may be able to interact with factors produced by such conditions, resulting in activation of metabolic, genetic, or other pathways that link in

	<p>CH₃, has three hydrogen atoms, one of which is considered to be functional hydride, and the action of PEMT results in de novo synthesis of choline within phosphatidylcholine which has particular resonant characteristics that require a distant unattached molecule to balance its polarity, resulting in interesting quirks of physics that occur between its structure and other molecules including the hydridic effect that exudes negative polarity to produce background alkalinity or pH between 7.2 and 7.6 which is essential to biological potentials, function, and capacitant fields constituting conscious cognitive function.</p>	<p>immunological, metabolic or other function to produce an Immunological or stabilizing response. CRISPR Perfect Gene Repair fused to Protein Transduction domains to assure 100 percent saturation of tissues and cellular entities during genomic repair.</p>
<p>Cascading effects of inhibited PEMT, depletion of large ratio of NAD⁺ compared to levels of NADH.</p>	<p>Ribose Supplement, RNA Nucleotide Supplement, Supplemental DNA Nucleotides, supplementation of the substrate and product of the Gene. NAD⁺, NADH, Pyruvate, Uridine, NADH precursors (Vitamin B3, Nicotinic Acid/Niacin[causes flushing and not effective in all tissues], Nicotinamide/Niacinamide[widely effective but inhibits sirtuin activity], Nicotinamide Riboside[optimal and manages insulin resistance/sensitivity], Nicotinamide Mononucleotide[optimal, available in all tissues, activates SIRT1, does not inhibit sirtuins, has</p>	<p>NAD⁺ and to NADH ratio can be affected increased homocysteine and upregulation of BAX and BAK which inhibit oxidative phosphorylation, cause aggregation of NADH, such that NADH escapes the mitochondria when the mitochondrial membrane permeability transition pore is activate as well as along with release of cytochrome C and other apoptotic factors. Information. eLife. Volume 2. Article e00772. 2013. PMID 23991283.</p> <p>Some major pathways for Hydride attrition occur through inhibition of oxidative phosphorylation which causes accumulation of NADH and escape of NADH from mitochondria, inhibition of Pentose Phosphate Pathway by inhibited PEMT/upregulated P53/NAD⁺ depletion which inhibits nucleotide synthesis and 60 percent of NADPH production, inhibition of glycolysis</p>

highest bioavailability in circulatory pathways and increases NAD in the intracellular environment the most among these other factors]). CD38 is major depletion pathway for NAD and CD38 is major factor in depletion of NAD resultant of and causal for senescence and detrimental factors associated with aging. Taxifolin, dihydroquercetin, Apigenin, Luteolin, which are supplements, as well as Callistephin found in deep/dark blue nutritional factor or foods such as blueberries, wine grapes, and pomegranates, and as well as Kuromaninis found in black currant, red raspberries, Peruvian corn and lychees. Flavonoid and Anthocyanin rich foods are recommended in this context such as black rice, blood oranges, red onions, red cabbage, acacia, black plums, etc. however, experimentally, the most rapid mechanism and most assured way to change NADH to NAD⁺ ratio in which NAD⁺ is required to be very high compared to NADH to potentiate release of H⁺ is to manipulate the ratio of pyruvate to lactate. Lactate Dehydrogenase translates pyruvate and NADH to lactate and NAD⁺. The cellular processes the mine for energy factors often mine for and release the hydridic energy factors which PEMT integrates

by P53 and NAD⁺ depletion which diminishes glycolytic throughput to inhibit krebs cycle and storage of choline as Acetylcholine resultant of inhibit Acetyl – CoA levels, this results in the 1 million + instances of DNA repair to have inadequate nucleotides to prolong PARP signaling and continue depletion of NAD⁺ causing available NADH and pyruvate to be used by lactate dehydrogenase to produce more NAD⁺ in a way that also increases Lactate production.

NAD⁺ to NADH ratios are considered to be between 3 to 1 and 10 to 1, while free NAD⁺ compared to NADH is presented as being 700 to 1, which is relevant to the ability of dimethylthetin to reduce homocysteine levels at about 700 times the potency factors typically used to deplete homocysteine.

Free NAD⁺, by the literature, is regarded as NADH in a transitional phase in which Hydride has been freed to exhibit 2 eV⁻, resulting in fluorescent energy or light emittance or energy emittance into areas of physiology, while interactions with atoms, molecules, electrons and reintegration of H⁻ into NAD⁺ or integration of H⁻ into redox factors such as FAD constitute the completion of such a biological circuit. The circuit perspective present here is contrived and the literature does not typically describe it in this way, although certainly is relevant to regard redox potentials in such a way. This negative polarity from released H⁻ as well as negative polarity imparted to physiology by tissue integrated hydride, produces a background pH that is between 7.2 and 7.6, and which is essential to be approximated to optimally sustain physiology, biology and cognitive function. Redox occurs in this polarity synapse, between H⁺ and the opposite polarity produced by H⁻.

into physiological structure when it integrates the three methyl groups first in enriched phosphatidylethanolamine to produce phosphatidylmonomethylethanolamine, then integration of another CH₃ into phosphatidylethanolamine to produce phosphatidyl dimethylethanolamine, and then another CH₃ into phosphatidyl dimethylethanolamine to produce enriched versions of phosphatidylcholine. Each methyl group, CH₃, has three hydrogen atoms, one of which is considered to be functional hydride, and the action of PEMT results in de novo synthesis of choline within phosphatidylcholine which has particular resonant characteristics that require a distant unattached molecule to balance its polarity, resulting in interesting quirks of physics that occur between its structure and other molecules including the hydridic effect that exudes negative polarity to produce background alkalinity or pH between 7.2 and 7.6 which is essential to biological potentials, function, and capacitant fields constituting conscious cognitive function. AMPK can improve energetics at the cellular level, such as when induced by energy level deterioration, phenformin, AMP

The NAD molecule exhibits two nucleosides linked by pyrophosphate such that each nucleoside exhibits a ribose ring but with each nucleoside differing with one exhibiting adenine linked to the first carbon atom and the other nucleoside exhibiting nicotinamide at the first carbon. NAD accepts or abdicates two hydrogen factors, one being Hydride H⁻ and the other being H⁺ or a protonating H⁺. A hydride donator reduces NAD⁺ to NADH while releasing H⁺, releasing the donator, and allowing NAD⁺ to adsorb H⁻ in a way that involves a carbocation rearrangement known as hydride shift in which the Hydride is transferred to the Nicotinamide ring. Hydride shift and Methyl Group shift occur typically in biology as the Hydride within Methyl groups and Hydride move to innermost aspects of a molecule when such structure is capable of such rearrangements. Hydride electron pairs abdicate an electron which is transferred to the positively polarized nitrogen of the nicotinamide ring and the other hydrogen moves to the C4 Carbon atom on the opposite aspect of this same nicotinamide ring, producing NADH that when compared to NAD⁺ as NAD⁺/NADH, results in a midpoint potential near -0.32 volts. -0.32 volts represents a strong reducing factor. The rapid cycling between NADH and NAD⁺ protects NAD from being catabolized except when NADH is directed toward NAD⁺ by lactate dehydrogenase to supply PARP with NAD⁺ for Genomic Repair Signaling, resulting abdication of the ribose and production of Nicotinamide which can be managed by methylation using nicotinamide methyltransferase although the literature suggests nicotinamide can also reenter NAD biosynthetic pathways or be directed toward other pathways. Nicotinamide methyltransferases are among detoxification methyltransferases and use Hydridic

mimetic AICAR, and Salicylate, but AMPK can participate in diseases that commandeer energy dysbiosis as well as inhibit such diseases, reliant upon context.

Ubiquinol can transfer Hydride to Complex I, inverting the Electron Transport Pathway to produce NADH instead of Complex I producing NAD⁺ by freeing Hydride and H⁺. However, this activity by ubiquinol is known to produce high levels of reactive oxygen species and is a feature of Macrophage activation responsively to Bacterial xenobiotic infiltration, while also being participative in adaptation of the Carotid Corpus to changes hematopoietic or Cardiovascular conditions, as well as response of the electron transport pathway to changes in energy availability. The Carotid Corpus is chemoreceptor performs at the adventitia of the bifurcation of the common carotid artery, while its function include monitoring of the pH pCO₂ and P0₂ levels of hematopoietic fluid, potentiating changes to cardiovascular and respiratory function. These such that the sympathetic, parasympathetic, carbonate buffering and even processes that manage bradykinin and other factors can be interactive with this juncture of the Carotid systems. Responses to

character exhibiting CH₃ derived from S Adenosyl Methionine produced when ATP of S Adenosyl moieties are integrated into methionine, resulting in ionization of the Sulfur within S Adenosyl Methionine.

The recent literature offers an eloquent and advanced elucidation of this metabolic context. Homocysteine is not precisely characterized as a monomethylated cysteine because it exhibits a methylene bridge not a completely assembled CH₃, although there are disjoint hydrogens to comprise CH₃ in homocysteine. Homocysteine differs from cysteine in that it exhibits a methylene bridge which cysteine does not exhibit, although homocysteine is nonproteinogenic alpha amino acid. The literature characterizes homocysteine by its synthesis as a result of abdication of the concluding c moiety methyl group of methionine.

The literature now regards methionine synthetase as s - adenosyl methionine synthase, while a particular study observes that when s adenosyl methionine is synthesized exclusive of methionine synthetase or exclusive of s adenosyl methionine synthase, the CH₃ or methyl group can be donated or abdicated without the requirement of a methylpherase or methyltransferase, presenting the possibility of transfer, abdication and donation of hydridic character suggesting that hydridic hydrogen protonation can occur in physiology without a catalytic methylation transfer enzyme or at least without the consensus catalytic methylation transfer enzyme known as s adenosyl methionine synthase, therefore confirming that methionine polymers can perform as enzymes such that hydridic character from methyl groups and particularly the addition of the methyl group into homocysteine to produce s adenosyl groups in methionine may bring this molecule to life Information. Information. Synthetic Polymeric Variant in

Acidemia, Hyercapnea, or hypoxia, each result in autonomic activity that can change pulse, respiration, pressure characteristics, all through a complement of chemoreceptors that include also another Carotid Corpus mechanism in the Aortic Arch. The Carotid Sinus nerve is also interested in this instance because it can participated in autonomic, sympathetic and parasympathetic signaling, but like sinus nerves in other areas of physiology, can be susceptible to reactive oxygen species and inducible nitric oxide synthase, explaining why inhibition of iNOS, and protection of Carotid pathways and cardiac nerve pathways from electricity and wireless exposures can be beneficial to health outcomes. Inversion of Complex I is also interesting because increasing levels of NAD⁺ are typically beneficial until extreme levels of imbalances in NAD⁺/NADH are achieved, and models of nonmammalian span of vital being exhibit increased duration of vital being when experimental inversion of Complex I is produced to increase synthesis of NADH. NAD⁺ excess produces a gradient upon which NADH abdication of Hydride is favored, clearing presenting that inversion of this pathway alleviates a condition in which inadequate NADH is available to quench extreme levels NAD⁺

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The euphemism monocysteine utilized in this compendium of research to decrease the detrimental effect of merely presenting the word homocysteine is a euphemism and is misleading, such that monocysteine should always be regarded as a euphemism for the physiologically precise word homocysteine.

The defining CH₃ that is added to methionine to produce methionine from homocysteine occurs in the synthesis of methionine from homocysteine by BHMT, BHMT2, Methionine Synthase, as well as thetin homocysteine methylpherase and even the synthesis of S adenosyl methionine in its complete structure by INMT which interconverts S Adenosyl Homocysteine directly into s adenosyl methionine, while also S adenosyl homocysteine hydrolase bidirectionally produces homocysteine and s adenosyl homocysteine according nad⁺/nadh balance, although TTMT production of trimethylsulfonium and 5 methyltetrahydrofolate enables both thetin homocysteine methylpherase with trimethylsulfonium substrate and enables methionine synthase with 5 methyltetrahydrofolate. BHMT uses trimethylglycine while BHMT2 utilizes S methylmethionine sulfonium to produce Methionine from Homocysteine. Dimethylthetin and trimethylsulfonium are known to decrease homocysteine linearly in a graph of such depletion, although the transsulfuration pathway also is known to irreversibly deplete homocysteine toward Cystathionione, Cystine, glutathionine, cystine and HS molecules.

Serine and homocysteine are used by cystathionine beta synthase to produce H₂O and cystathionine while cystathionine gamma lyase utilizes H₂O and Cystathionine to produce Cysteine and alpha ketobutyrate, while also alpha ketobutyrate can be directed toward propionyl CoA using CoA SH and

with hydride derived from NADH.

NAD⁺ which produces CO₂ and NADH as output product, characterizing the nearest phases of transsulfuration pathway structural depletion of homocysteine.

Thus, the methylene bridge enables cysteine to escape as homocysteine into recycling pathways for homocysteine. Generally, the transfer of a thiol or sulfur group from homocysteine to cysteine occurs in the transsulfuration pathway, in a way that occurs without exhibition of the methylene bridge in the thiolated cysteine, keeping the cysteine out of homocysteine recycling pathways. Nicotinamide phosphoribosyl transferase metabolism of 5-phospho- α -D-ribose 1-diphosphate, H⁺ and nicotinamide metabolism towards beta-nicotinamide D-ribonucleotide and diphosphate can relieve nicotinamide methyltransferase production of cysteines with methylene bridge moieties that includes homocysteine exhibition, although it is not clear if this contributes to excessive NAD⁺/NADH ratios instead of exhibiting increases in homocysteine, or if both of these increase to produce homocysteine and NAD⁺ levels that are both integral to some pathologies.

However, managing NAD⁺/NADH ratios are recommended. Methylene bridges are located between two strong drawing molecular groups such as nitrogen ions in phosphatidylethanolamine which acquire the three lone pairs of three different CH₃ groups to produce phosphatidylcholine.

The CH₃ ions are very unstable because they do not have the eight electrons required to satisfy the octet axiom of reactivity. CH₃ can exhibit a vacant p orbital, while its structure exhibits oppositely oriented chiral Hydrogens and a nonchiral Hydrogen, one of which is, resultant chemical and structural characteristics, is considered to be Hydride, particularly because it

exhibits the effective oxidation characteristics of hydride.

Carbon typically exhibits 6 valence electrons, and is characterized as an electron deficient electrophile, such that in carbocation the hybridization of carbon is typically sp^2 in trigonal planar structure. A SN_1 or reactions producing multiple separate products can typically involve carbocation and the hybridization of Carbon in CH_3 in such reactions can look like Carbon cations with hydrogens sharing the three SP^2 orbitals from the top while an axial view can present an empty p orbital. Tertiary carbocations have Cationic, presumably hydridic influenced, center with Carbon at the center of three other atoms, while secondary carbocation can exhibit Cationic, presumably hydridic influenced, center with the carbon atom exhibited between two other atoms.

Primary carbocation exhibits an axial, offset Cationic, presumably hydridic influence center that is toward one of the two atoms that flank the Carbon atom at the center of the structure. Methyl carbocation considers CH_3 as the cationic, presumably hydridic influence, center. CH_3 , at least, typical has the Cationic center offset by atoms that are indirectly attached or possibly attached within another aspect of the same macromolecule using an opposite polarization.

Additional characterization of carbocations include allylic carbocations that occur when a Cationic Center is bordered by a carbon to carbon double adhesion or linkage, vinylic carbocation when this carbon to carbon double adhesion or linkage has the Cationic Carbon exhibit sp with linear geometry, while aryl carbocation consists of the Cationic Carbon participating in a benzene ring, while benzylic carbocation does not require the cationic carbon to be structurally attached to the benzene ring. Carbocation can also include Cationic Carbons which are near or depicted visually as hovering over

tetrameters with a +1 or pentameters with a +2.

Methyl shift involves shifting of hydrogen from the first carbon to the second carbon in molecular structure. The literature does not specifically link this movement to the exhibition of a methylene bridge, but molecule used as reference exhibit highly unstable methyl groups that are methylated, exhibit a methylene moiety and include transfer of the hydrogen to this methylene moiety to exhibit CH₃ in place of methylene. Methyl shift involves a similar movement of Hydrogen from the 1st Carbon to the second Carbon in molecular structure, exhibiting movement of a hydrogen from a methyl group with 3 CH₃, and resulting movement of the hydrogen across a hydrogen adhesion to the second carbon in the structure.

Compared to hydride shift or primary carbocation in which only two CH₃ are exhibited along with a hydrogen connected to the originating carbon, the secondary carbocation exhibited in methyl shift exhibits 3 CH₃ moieties connected to the originating Carbon. The result of the secondary carbon is a CH₃ and two Hydrogens connected to the Carbon that receives the shifted Hydrogen. The result of primary carbocation, in contrast, results in three hydrogens attached to the carbon that receives the shifted hydrogen.

Secondary carbocations are typically more stable than primary carbocations, and phenyl carbocations can occur to enhance stability even more substantially, while generally, resonances or intermolecular influences are considered to be typically more stabilizing for carbocations. The delocalization of the Cationic polarization enhances stability, a benefit for resonance such as in CH₃ and Choline once the nitrogen of phosphatidylethanolamine has received 3 CH₃ to become choline within phosphatidylcholine.

Tricyclo Propane carbocation, illustratively, is considered to be a most stable carbocation. It is also important to know that the electron transport pathway of oxidative phosphorylation exhibits freeing of Hydride from NADH to produce 2 eV- of energy as freed fluorescent influence that supplies energy to the electron transport pathway which equalizes the utilization of energy through process democratization among each of its phases, utilizing about 58 percent of such energy to produce ATP, such that ATP receives about 42 percent of such freed energy by integrating such energy into the oxonium exhibited between its phosphate groups.

The attachment of ATP to Methionine results integration of this hydridic source of energy into S adenosyl methionine, resulting in carbocation rearrangement that Ionizes the Sulfur of Methionine, changing sulfur to a Cationic or positively polarized Sulfur. Information. Carbocation. IT JEE Study Material. JEE. byjus.com website. Information. "Selenium." Chem Soc Rev. Volume 42. Number 23. Pages 8870 to 8894. Some of the literature presents phosphatidylcholine and phosphatidylethanolamine with the same structure near the nitrogen, with three open locations at the nitrogen or sometimes with three hydrogenase linked to the nitrogen, while in those depictions of the nitrogen in choline and the nitrogen in phosphatidylethanolamine the oxygen between the second carbon from the nitrogen and the other aspects of the these two molecules is presented as negative polarized or an oxygen anion.

These suggest that either CH₃ are not structurally attached to the Nitrogen, resulting in no change to the polarization of the Oxygen, or, this suggests that when 3 CH₃ molecules are linked to the Nitrogen, a carbocation or shift occurs at hydride, methyl or other levels, resulting in change in polarization of this linking oxygen.

Palladium exhibits deep latitudes of atoms with electron holes that allow it to accept and integrate hydride at levels that are massively more extensions than its own structure and mass, exhibiting one of the most capable systems of storing hydridic molecules or massively exhibit storage of electromagnetic current potential. However, these are exceed by permanent magnet systems which use magnets to generate a turbine that produces massive levels of electromagnetic energy without any fuel, without any by products or waste and which exhibit easily counteracted magnetic energy using EMF protection as covering or enclosures. These have the potential of produce minuscule or 100s of megawatts of energy for every application, with unheard of size to current ratios, including the ability power water from air systems that generate water freely from the atmosphere at hundreds of thousands of liter each day.

NAD⁺ and NADH redox interactions can recharacterize the hydridic microenvironment with near physics biophysics bending rapidity, likened to a ‘New York Minute’ and exhibiting enough rapidity to potentiate geodesics link to interactions or communication that transcend distance, location, space and time. Amon the other physics bending phenomenon that are typical of biology including hydrolysis of the water molecular at 98.7 degrees Fahrenheit, just in time movement of electrons to areas requiring particular biochemical characteristics, through space jumps in which electrons jump or move between atoms, resonance in which atoms in molecules behave as if they are integrated into other distance molecules, defined space in which a unit of bioplasm or nanoplasma with empirical characteristics behaves as any atom potentiated from the atom level or quantumly level makeup such define space, transcending or

tunneling of electrons through potentials or barriers imposed by circumstance and spooky action at a distance in which conditions between quantumly entangled material now changes the outcomes of those material which these were previously entangled, all described the nuances of biology that interactively improve those factors utilized in biology and managed interactively the conditions in which biological systems are exhibited. This include molecules like hormones which have aromatic polycyclic hydrocarbons that exhibit resonance and remote control of surrounding biological material to orchestrate metabolism, biology, biosynthesis metabolic homeostasis.

The literature observes NAD⁺ synthesis from tryptophan or aspartic acid, quinolinic acid, nicotinic acid mononucleotide, or obtainment of NAD⁺ from Niacin which is also mentioned in the literature, but Hydride load is often regarded in the literature as occurring through redox interactions, with the literature even suggesting that NADH or Hydride can be obtained by cellular entities from encompassing tissues.

Explanation of Hydride fracking or obtainment from tissues and methyl groups includes explanation of Hydride absorption from the tissues in the physiological environment. However, the Carbocation rearrangements by methyl groups and hydride known as methyl group transfer and hydride transfer, both supply an integrally useful perspective. Interactions between hydride carriers and methyl group carriers occur along with potential for hydride transfer and methyl group transfer as well as electron rearrangements, these allow hydride to be transferred between molecules followed by exhibition of interactions that release, free or separate aspects of the molecules involved in such transfer or rearrangements.

Another explanation includes the example of choline which uses the polarity of an unattached molecule to balance its structure and provide stabilization, producing interactions at a distance that can allow hydride to influence the microenvironment.

Free hydride is considered to be a rare occurrence, are not invoked for homogenous solutions, require extreme conditions to occur, and hydride typically occurs as molecules that have hydrogenic centers with such hydrogenic centers exhibiting hydridic characteristics.

Hydridic electron affinity of about 72.77 kJ/mol produces exothermic interactions to perform as Lewis Base, a strong Lewis Base. Hydride, likewise, is a strong reducing factor. Information. J Chem Educ. Volume 98. Number 12. Pages 3999 to 4008. 2021.

NADP⁺ can be produced from NAD⁺ by NAD⁺ kinase which performs phosphorylation of NAD⁺ typically using ATP, which links this process to oxidative phosphorylation or electron transport pathway production of ATP in aspects of cellular respiration.

Recycling of nicotinamide can occur using NAMPT, which produces NMN that then is used to produce NAD⁺. Nicotinic Acid, Nicotinamide and nicotinamide riboside, obtained from Vitamin B3 or niacin, representing environmental absorption through digestive pathways provides a correlated to absorption of NAD⁺ from tissues in the physiological environment. Some pathogens and viruses can only salvage NAD⁺ from the host or must absorb NAD⁺ from the host with not salvage pathways or capability of de novo synthesis of NAD⁺.

Redox factors that integrate with NADH are numerous, and typically exhibit the Rossman Fold which is a typical structural motif for NAD⁺ and NADH redox factors.

Methyl transferases, thus, perform a more complex role in physiology, enabling the distribution of Hydride and Methyl groups to molecules that carry methyl groups, promoting a hydride to hydrogen ration of 1 to 2, as well as enabling hydride shift and methyl group shift that performs a deeper integration of hydride into the methyl carriers' structure and into promoter regions of genome which acquire methylation. These, perhaps, explain why PEMT seems to be prefer or be designed to provide a stabilizing benefit that is strongest when PEMT is actively processing S-Adenosyl methionine and homocysteine, enabling a systemic circulation of methyl groups, hydride and a hydride to hydrogen ration of 1 to 2. Certainly, the accumulation of S – Adenosyl methionine with inhibited PEMT potentiates direction of methyl groups toward methylation factors that produce upregulated homocysteine.

The example of s-adenosyl methionine seems to be relevant because PEMT derives CH₃ from s-adenosyl methionine and the obtainment of CH₃ brings along Hydride that must be retrieved from its relocation in carbocation rearrangements typically occurring in hydride exhibiting molecules. Sometimes such rearrangements involve movement of electrons to produce a hydrogen center of hydridic nature or hydridic character and can involve comparative locations and polarities to produce a hydridic characteristics in a hydrogen center.

The data suggests that methyl groups, also known as one carbon units, can become loaded into carriers in obscure ways to protect the methyl group and protect the

hydride such that carbocation rearrangements, methyl group shift and hydride shift, are among the features of such obscuring processes. Methyl group loaders are included in a pathway of interconnected processing but the actual loading of one carbon units or methyl groups occurs through translation of homocysteine into methionine and s-adenosyl methionine which compares to depletion of s-adenosyl homocysteine through the transsulfuration pathway in a way that produces cystathionine, cysteine and other factors.

Methyl group or one carbon loaders that produce methionine and S-adenosyl methionine include Betaine homocysteine methyl transferase I, Betaine homocysteine methyltransferase II, Thetin homocysteine methylpherase, thiopurine Thioether s-methyltransferase, Methionine Synthase, Trimethylsulfonium Tetrahydrofolate Methyltransferase, S-adenosylmethionine synthetase, MARS1/MARS2 Methionyl – tRNA - Methionyl Ligase, S-adenosyl homocysteine hydrolase, Indolethylamine N - Methyltransferase/Thioether S – Methyltransferase. Among other factor listed in the linked visualization as well as others not depicted in the visualization these perform as methyl group or once carbon unit integrators or encoders that load hydride into methyl group and once carbon carriers.

Unlockers or decoders of encoded or integrated hydride either translate S-adenosyl homocysteine into methionine, transfers methyl groups from S-adenosyl methionine to toxic, xenobiotic, genomic, or other factors, as well as may support detoxify histamine accumulation during allergic or xenobiotic response. These include Guanidinoacetate N – Methyltransferase, Glycine N-Methyltransferase, inverted

Cystathionine Gamma Lyase, inverted transsulfuration pathway including the aspartate pathway, COMT, PEMT, Histamine N – Methyltransferase, Phenylethanolamine N – Methyltransferase, Norepinephrine N – Methyltransferase, Guanidino N – Methyltransferase, Glycine N – Methyltransferase, Methyl transferases such as histone methyltransferase and others.

The importance of hydride circulation is linked to promoting the 7.2 to 7.6 background pH required for l conscious cognitive function and biological function. Also, hydride shift protects the hydride from being utilized by pathogens and pathogenic processes.

A closer review of fracking operations is presented in a particular study. The study observes methyltransferases to nitrogen, carbon, sulfur nucleophiles and oxygen, as well as other atoms, using S-adenosyl – L – methionine, adomet. The unlocking or decoding pattern is presented in the study as a biomolecular nucleophilic substitution Sn2 reaction. Methyltransferases exhibiting rossmanlike folds exhibited a second TyIM1 active locus which assisted in the Carbon-Oxygen exhibition of a hydrogen to link to the Methyl Group Adomet, and this assistance was essential for methyltransferase catalysis. Similarly, methionine exhibits a nonpolarized sulfur while s-adenosyl methionine exhibits a positively polarized sulfur, while the study observes that Sulfonium Chalcogen adhesion forces were required along with Carbon-Oxygen hydrogen integration to enable catalysis. These suggest that hydridic shift may be occurring in the s-adenosyl methionine molecules near or at the positively polarized Sulfur.

The literature describes other instances of nucleophilic catalytic interactions imposed upon the methyl group of same, particularly including alkenes, as producing a carbocation rearrangement that must occur before subsequent catalytic phases. These suggest that at least hydridic shift rearrangements are exhibited in s-adenosyl methionine which protect hydride.

Another study clearly present a model for methyltransferase activity in which hydride shift is required to free a methyl group from S-adenosyl methionine and reconstitute the complete one carbon and methyl group molecular structure. RlmN and Cfr perform as methyl synthase because they must enabled hydride shift to complete the methyl group structure and constitute the hydridic character of the hydrogenic centers.

Another study observes that synthesis of S-adenosyl-L-methionine emerges from neighboring group reactions that produces five major ions, m/z 250 produced from methionine loss, m/z 102 and 298 produced from segmentation of gamma C-S adhesion of methionine, m/z 136 and 264 which are produced from abdication of protonated adenine and neural adenine. Although the mechanisms presented in the production of the major m/z 250 metabolite S-adenosyl-L-methionine are theoretical, 1,2 hydride shift seems to be the preferred mechanisms according to calculations. Information. ISBN 1464923485. Information. PROC Natl Acad Sci. U S A. Volume 108. Number 10. Pages 3930 to 3934. March 8, Information. Biorg Chem. Volume 39. Number 5 and Number 6. Pages 161 to 170. December, 21762947.

Foundationally, methionine synthesis by methionine synthase transfers a methyl group from N5-methyl tetrahydrofolate to

homocysteine, producing methionine through a methyl cob(iii)alamin intermediate, with the cobalamin performing as a shuttle for the methyl group transfer. The transfer involves shift between Co(i) and Co(iii) redox statuses although the formal process for the methyl group transfer is known as a carbocation. Information. ISBN 978-0-08-045047-6.

Information. “Characterization of Methyltransferase Carbon-Oxygen Hydrogen Bonding and Sulfur-Oxygen Chalcogen ‘Adhesion’ with the Sulfonium of S-adenosyl-L-methionine.” A dissertation submitted for the partial fulfillment of the requirements for the degree of Doctor of Philosophy (Biological Chemistry) in the University of Michigan. 2018.

The relevance of fracking processes occurring as carbocation rearrangement of hydride electrons, hydride, or methyl groups or one carbon units seems to be explained by the concept of hydrogenic center with hydric character. This concept enables the energetic, fluorescent, and anionic character of hydride to migrate within a molecule through rearrangements that can be molecular, atom level or even only involve electron migration. This migration is important when it is reiterated that hydride is the energy that fuels stars of the Universes and hydride is eluted pervasively in the environment hydride conjugates, particular sulfur which is eluted by an encompassing array of environmental atoms or molecules. The eluting of hydride by pervasive hydride conjugates or molecular hydrides occurs because hydride is not exhibited as a free molecule except in extreme conditions and because sulfur enables thetin – homocysteine methylpherase to become activated by dissolving the intramolecular linkages produced in thetin – homocysteine methylpherase when sulfur is

inadequate. These disulfide linkages produce a gel phase which has an interesting phonetic homologue in social systems that seems to be being eluted in the social transformation of physiology to systems that occurs in western civilization.

Thetin – homocysteine methyltransferase is an organic phase to inorganic phase separator and sequestration factor which elutes useful molecules from abiotic phases and transfers these to biotic phases, being useful in the environment as an elution catalyst for hydrides, sulfur and molecules essential to biological function, The produce of thetin – homocysteine methyltransferase metabolism, methylthioglycolic acid, was utilized as a derivatization catalyst that can be imputed into any environmental, molecular, biotic, abiotic or other context to elute useful molecules that were then utilized to produce pervasive drugs and therapies in the 1900s and 2000s.

Thus, hydride provides energy that enables biological activity to occur both with gradients when only being utilized within redox interactions and enabling biological activity against gradients which the literature regards as the role for chemical energy as ATP. However, hydride does not disappear during synthesis of ATP during oxidative phosphorylation or the electron transport pathway, such that, instead, hydride becomes obscured between the phosphate groups of the phosphate groups. Some of the literature presents this integration of hydride between the phosphate groups as being integrated into the oxonium factors of ATP's structure. Regardless, the literature refers to oxonium character, and hydronium character in the same manner which it refers to hydridic character. The definition of oxonium in the pubchem databank is presented as an oxygen hydride, an onium cation

and the conjugate acid of water. Oxonium. Pubchem. National Library of Medicine. National Institutes of Health.

Homocysteine is presented as a sulfur exhibiting amino acid that is nonproteinogenic which is causal of redox imbalance, oxidative distress, activation of immunological pathways, activation of platelets, causal of increased free fibrin and interaction with fibronectin that causes deposits of fibronectin in cardiac tissue to promote cardiac tissue remodeling. Homocysteine also causes oxidation of proteins, nucleic acids, and proteins as well as lipoperoxidation products or lipoxidation products.

Homocysteine causes pages and pages of detrimental factors that are known to be integral to almost all if not all pathology, while homocysteine can cause such factors directly, indirectly and independently of disease. These diseases include cardiovascular disease, neurological conditions, psychiatric conditions, chronic kidney disease, bone tissue conditions, gastrointestinal disorders congenital anomalies, oncology, and other factors or conditions.

Importantly, the thiolation of proteins, enzymes, atoms, and molecules by Homocysteine deactivates biological activity potentiated by such molecules and promotes deterioration of coordinate systemic function and causes cellular entities to exhibit self harm by upregulating BAX, upregulating P53, inhibiting PEMT, causing cytochrome C release, activating caspases and initiating opening or activation o the mitochondrial permeability transition pore, all of which are features of apoptosis or cellular deterioration pathways.

Homocysteinate, as an alpha amino acid anion produced by deprotonation of the carboxy group. It is a conjugate base of

homocysteine not mentioned in the literature but is presented here to include it among homocysteine metabolites s-adenosyl homocysteine, homocysteine, homocysteine thiolactone and homocysteic acid. Homocysteinate can be utilized as a food flavoring.

Information. Int J Mol Sci 10. Pages 1733. October 20, Information. Homocysteinate. Pubchem. National Library of Medicine. National Institutes of Health. Information. Bioorganic Chemistry. Volume 2. Issue 2. January, 1973. Pages 179 to 190.

The literature presents important distinctions between the major Sulfur containing amino acids and the ICD10 and ICD9 exhibit specific disease categories for anomalies sulfur exhibiting amino acids. Methionine, cysteine, taurine and homocysteine are regarded as the primary sulfur exhibiting amino acids all of which exhibit the less electronegative sulfur which is the same group as oxygen in the periodic table, but, of course, sulfur is, again, less electronegative than oxygen.

Methionine is required for the synthesis of 99.5 percent or more of all eukaryotic or, in particular, mammalian proteins, such that methionine is required for the priming sequences which initiated gene transcription completion and mRNA translation. Methionine is typically hidden in the hydrophobic core of molecules and proteins, exhibiting similar propensities as methyl groups, one carbon unites and hydrides to become obscured from cursory metabolic and molecular interactions, unless specifically fracked or eluted from the hydrophobic core. This exclusion from oxidative damage or impairment exhibited by methionine may be essential to sustained physiological processes, particularly during diminished physiological

environmental characteristics linked to pathogen challenges or disease.

Cysteine has a propensity to become involved in disulfide linkages and cysteine is an important factor in protein folding and protein structure. Methionine enters metabolism typically through S adenosylation to produce S-adenosyl methionine, the substrate for PEMT among many methyltransferases. Methylation reactions comprise the major pathway of S-adenosyl methionine utilization although methionine is an important factor in the Carbonate buffering system and along cysteine can become changed or utilized to assist in carbonate buffering, particularly during metabolic syndrome. S-adenosyl methionine may become S-adenosyl homocysteine and S-Adenosyl homocysteine can become homocysteine as homocysteine potentially becoming cysteine and glutathione in the transsulfuration pathway or homocysteine becoming recycled into methionine by one carbon unit loaders, methyl group loaders or hydride loaders. Transamination pathways may also deplete methionine, producing toxic products. Transamination is one of the directions which pyruvate may be directed toward exhibiting amino acids as potentiators of Pyruvate direction toward alanine which also produces alpha keto acid. Methionine is presented as an amino acid that is potentiated toward glutamate by alpha ketoglutarate which also produces alpha keto acid along with glutamate. Glutamate then donates its amino group to oxaloacetate to potentiate glutamate and oxaloacetate direction toward alpha ketoglutarate and aspartate. These transamination catalytic reactions are bidirectional. Oxaloacetate can, also, be shunted into the Krebs cycle to jumpstart Krebs cycle activity. Cysteine can become taurine as well as glutathione, although the literature

suggest cysteine can become cystine, H₂S and HS as well.

Information. The Journal of Nutrition. Volume 136. Issue 6. Pages 1636S to 1640S.

Hydride or hydridic character is maintained in ATP after the electron transport pathway frees hydride from NADH to produce NAD⁺, after which 58 percent of the energy from Hydride oxidation from NADH is utilized to support the electron transport pathway while 42 percent is maintained between the phosphate groups of ATP within the oxonium which is hydride.

Thus, the fluorescent potential and eV- potential of hydride is included as ATP such that attachment of ATP to a molecule potentiates acquisition of this hydridic character and fluorescent potential or eV- potentials by molecules having ATP attached. The hydridic potential can produce carbocation rearrangements but does not seem to always require such rearrangement or even require hydride electron migration, particularly when ATP is merely attached to molecules.

However, a closer analysis of PEMT and loaders of methyl groups, one carbon units or hydride reveals that Carbocation rearrangements occur in these processes. Trimethylglycine, for instance, exhibits 3 CH₃s and the Nitrogen to which these CH₃s are attached is negatively polarized. Methionine exhibits CH₃ but the sulfur does not have polarization, while S-Adenosylation of methionine polarizes the Sulfur to a positive status similarly to the positive polarization of choline and trimethylglycine.

Clearly, ATP, CH₃, One Carbon Units, and Hydride provide biologically active molecules with hydridic character or enhance the strength of the hydridic character in molecules to activate these

molecules and enable molecules to participate in biological activity, although ATP in particular seems to enable activity that is anathema to or opposite to the gradients in physiology enabled by redox or enabled by the difference between the polarity of molecules with hydrogen centers with hydridic character which comprises the background pH of 7.2 to 7.6 compared to the H⁺ or circulating anions.

ATP seems to be required for methyl transferases to comprise whole methyl groups for transfer from s-adenosyl methionine to methyl group acceptors. The obtainment of methyl groups in this regard along with exit of the adenosine does not leave a methionine as was exhibited before the Adenosylation occurred, but produces homocysteine. The Adenosine, ATP and Methyl group has been removed from Homocysteine resulting in an unstable reactive molecule that has had its hydrogenic center of hydridic character diminished.

The literature observes a foundational nuance of homocysteine ability to impart detriment as being its diverse nuances of reactivity, its sulfhydryl group and its ability for autoreactivity or ability to react with itself and other homocysteine molecules to produce detrimental products. This seem to be centered around the inherent nature of disulfide interactivity which includes production of intramolecular links, intramolecular links and links with thiols in essential nuances of biological fluids and tissues. Homocysteine, thus, seems to perform as a void, emptied by methyltransferase activity, and the void produces strong and diverse interactivity. Although the hydride being asymmetrically fracked from homocysteine by adenosylation followed by demethylation seems to

present a reasonable context for reactivity, the clearest context for reactivity is the sulfur and the sulfhydryl as H_2S and HS which are strongly interactive in physiology and which are strongly combustible when exposed to oxygen, such that interactions can be occurring in physiology resultant of Sulfur interaction with Oxygen, but also may be occurring because sulfur is a substitute in many metabolic reactions for Oxygen except the Sulfur is more electronegative than oxygen.

The electronegativity of sulfur is higher or stronger than for oxygen, these suggest that sulfur may be fitting itself into interactions in place of oxygen and causing such interactions become aberrant because of its stronger electronegativity. Because sulfur is deficient in humans typically enough to deactivate the α -ketoglutarate-dependent homocysteine methyltransferase, the characteristics of sulfur in homocysteine, including the exhibition of HS conjugated within the extremity of homocysteine but also because there may be other electron or proton or structural modifications made to sulfur within homocysteine resultant of the way in which CH_3 was removed in a context that requires ATP. There may be modifications occurring in contexts of carbocations that are not widely known or may not have been characteristics plainly enough to be presented here.

Sulfhydryls are presented in the literature as susceptibilities to toxicity while in other areas of the literature depletion of sulfhydryl is linked to hepatic toxicity. Disulfide linkages between sulfides, including sulfhydryl presents the possibility of intramolecular conjugates attrition of those molecules involved in such interactions because of the intermolecular reactions that deactivate molecules and because these may enable such molecular complexes to be removed by

proteolysis or autophagy. One study observes changes in GSSG structures which indicated disulfide intramolecular and intramolecular linkages as increasing along with hepatic toxicity to acetaminophen.

Information. Annu Rev Med. Volume 60. Pages 39 to 54
Information. Biochem Pharmacol. Volume 61. Number 2. Pages 245 to 252. January 15,11163339.

Cytoskeletal sulfhydryl content also changes when exposed to toxins.

Information. J Toxicol Environment health. Volume 31. Number 1. Pages 71 to 91. September, 1900. PMID 2120461.

The literature observes that homocysteine linked to albumin first displaces the cysteine residue in position 34 of Albumin. The calculated favorable interactive process includes sulfhydryl group reaction with the sulfur of the position 34 cysteine residue, synthesis of the disulfide that includes homocysteine and cysteine along with the free albumin thiolate anion, followed by albumin thiolate anion searching and finding the homocysteine cysteine mixed disulfide that exists on the homocysteine sulfur, all resulting in albumin linked to homocysteine as well as exhibition of cysteine thiolate anion.

Homocysteine becomes circulated and displaces the levels of albumin that would otherwise be attached to albumin and thiols generally. Experimental analysis observes a near 50 percent displacement of cysteine and cysteine glycine when experimental elevated homocysteine conditions were produced

eNOS can be activated by homocysteine to detoxify homocysteine, but this can result not only in exhibiti9on of Nitric Oxide, but can also produce S Nitroso Homocysteine which can produce

vasodilation and inhibit platelet aggregation, which is the opposite of the effects that homocysteine has, but hydrogen peroxide is not supported in this configuration, presumably because homocysteine deactivates catalase to prevent it from producing water from H₂O₂ and, interestingly, homocysteine diminishes expression and secretion of Superoxide Dismutase, while taurine the fourth primary sulfur exhibiting amino acid factor reestablishes superoxide dismutase expression and function in contexts of upregulated homocysteine.

Three studies suggest that homocysteine suppresses transactivation of superoxide dismutase in the extracellular environment, although when bona fide genetic impairment such as methylation and cystathionine deficiency were exhibited, homocysteine levels increased extracellular superoxide dismutase. These are the first indication that methylation pathways do more than effect genetic signaling changes but perform as genetic sensors that can change gene expression. Homocysteine is known to affect release of thymocytes from the thymus, affect cellular survival, affect reproductive tissue development, and promote deactivation of tissue development such as in exhibition of menopause. This instances observes that it is genetic status in methylation pathways that is activating or deactivating Superoxide Dismutase levels, although a simple explanation could be the dilatation status of the caveolae which is modulated by eNOS catalytic activity status at least. The study members in the gene impairment groups were also more than likely managing their genetic impairments or even may have been receiving therapy for hyperhomocysteinuria.

The study exhibiting inhibition of extracellular superoxide dismutase, however, clearly found upregulation

of foam cellular entities, cholesterol aggregation, H2O2 aggregation and oxidized low density lipoprotein exhibition, along with interestingly specific hypermethylation of superoxide dismutase promoter regions, upregulation of DNMT1 methyltransferase, exhibit no affect to DNMT3, integration efficiency of methyl CpG, integration efficiency of meCP2, along with upregulated monocyte exhibition of acetylation at H3 and H4.

Homocysteine above 100 um/L was found inhibit niR 143 expression, although Homocysteine should be managed to between 3.7 and 7 or 6 um/L, although 3.7 um/L or lower can be increasingly optimal, with 10 um/L used as therapeutic gateway threshold that requires continued therapeutic management to lower levels. Focused therapeutic intervention of homocysteic acid, homocysteine thiolactone, s adenosyl homocysteine or homocysteine may be produce increasingly beneficial effect. Such downregulation of miR143 produced upregulation in DNMT3a de novo methylation while an inhibitor of miR143 similarly inhibited miR143 activity and upregulated de novo methylation, such that changes to superoxide dismutase exhibition may be resultant of epigenetic changes produced by DNMT3a.

Information. Molecular Medicine Reports. Volume 13. Issue 1. Pages 483 to 490. January, 2016.

Information. J Exp Biol. Volume 211. Part 6. March, PMID 18310117. Information. Kidney International. Volume 63. Supplement 84. Pages S137 to S140. May,

Homocysteine enhances proteolysis and impairs coagulation in the fibrinolytic system to intensify haemostatic capacity in manner that enables proteolytic deterioration of the abdominal aortic wall.

Homocysteine enhanced Metalloproteinase2 levels but also induced tissue plasminogen

activator and also produced an increase in plasminogen in a way that was increased in sections of thrombus compared to tissue otherwise. Metalloproteinase2 and tissue factor were not decreased in the wall adjacent to thick thrombus segments compared to tissue otherwise not affected. Interestingly, homocysteine decreased MMP2 and tissue factor activity in thick sections of intraluminal thrombus or ILT. The serine protease inhibitor plasminogen activator inhibitor1, a primary inhibitor of tissue plasminogen activator, as well as tissue factor, and metalloproteinase2 were upregulated in control tissues when exposed to homocysteine. Information. Biomed Res Int. Volume 2018. Article 3205324. December 12, 2018. PMID 30643799. Tissue factor is known to stimulate thrombosis. Homocysteine promotes deterioration of subendothelial arterial elastic structures by upregulating elastolytic metalloproteinase 2 and metalloproteinase 9, as well as tissue kallikrein which potentiates these same metalloproteinases, although homocysteine downregulates metalloproteinase 3, metalloproteinase 7 and urokinase plasminogen activator. Information. Biochem Biophys Res Commun. Volume 316. Number 1. Pages 170 to 176. March 26, 2004. PMID 15003526. Homocysteine promotes modifications of tau protein including phosphorylation, truncation and oligomerization. Major Tau phosphorylating kinase GSK3B and CDK5 both were upregulated while a major Tau phosphatase which removes phosphate groups from Tau was deactivated by Homocysteine. Homocysteine caspase activation in the neuroblastoma cellular entities used in the study, and the as well as upregulated aggregate tau levels, enhanced levels of phosphorylated tau, increase segmentation of tau at

the catalytic site by which tau activates caspases to initiate apoptosis, and, importantly, increased the insoluble fraction of tau. Homocysteine increases tau oligomer complex 1 TOC1 resultant of increased tau oligomers along with increased toc1 immunoglobulin toward both soluble and insoluble tau. Small nonhuman mammals exhibited increased TOC1 positive oligomeric tau responsively to homocysteine increases, particularly in the brain, which were alleviated by s-adenosyl methionine to level in controls, clearly presented that homocysteine upregulates tau, tau pathogenic fraction of metabolites as well as immunological activation toward tau, in a way that was clearly resolvable by reducing homocysteine and enabling PEMT function. Information. Int J Mol Sci3. Pages 891. March, 2018. PMID PMC58877752

Methylation is considered to be key factor in Alzheimer's and conditions similar to Alzheimer's such as Huntington's Disease and Parkinson's Disease. Information. Cellular Communication and Signaling. Volume 19. Number 1. December, 2021.

Homocysteine has been found to exhibit a causal causal link in movement disorders such Huntington's Disease, Parkinson's Disease of idiopathic origin, and primary dystonia, with Ldopa instrumentation along with Ldopa metabolism by Catechol-O-methyltransferase to produce o-methylation of Ldopa to produce dementia, nonmotor symptoms and cardiovascular conditions which are primary paths toward outcomes of the most risk. Huntington protein changes cystathionine beta synthase activity resulting increase in homocysteine and this homocysteine is integral to a diverse group of ,movement, motor function, behavioral and cognitive anomalies. Cardiovascular and cerebrovascular disease emerge as risks in these contexts, along with

ganglia disorders, neurotoxicity, neurotransmitter imbalance in motor circuits, vascular infarcts, cognitive dysfunction and other impairment. Curr Vasc Pharmacol. Volume 4. Number 3. Pages 237 to 243. July, PMID 16842141. The pattern of movement disorder homocysteine confirms the patterns observed otherwise in which therapeutics can pervasively produce upregulated homocysteine unless focused on homocysteine enabling pathways. Homocysteine is increased in diseases of impaired movement such as Huntington Disease, although therapeutics such as Ldopa instrumentation can additionally increase homocysteine compared to study participants exhibiting Huntington's disease without any therapy being instrumented. Information. Mov Disord2. Pages 226 to 228. February, 2004. PMID 15978683.

Transcriptomics and metabolomics integrated analysis and studies clearly derive dysregulation of phosphatidylcholine metabolism in Huntington's disease peripheral hematopoietic samples. :Information. Metabolomics. Volume 12, Number 137. 2016. PMID 27524956.

Hydrogen sulfide is presented as potential therapeutic to alleviate neurodegenerative diseases caused by oxidative stress such as Huntington's Disease, Alzheimer's Disease, Parkinson's Disease, and Amyotrophic Lateral Sclerosis. Information. Int J Med Sci. Volume 16. Number 10. Pages 1386 to 1396. September 20, 2019. PMID 31692944.

Homocysteine causes downregulation of the ubiquitin proteasomal systems, exhibiting a pivotal influences over phenotype and metabolic status of cellular entities, suggest the upregulation of proteasome activity in oncology occurs to counteract BAX, proapoptotic potential, and involves upregulation of choline kinase, phosphocholine, potentially aSMase/nSMase, sphingosine

kinase 1 in non neurons and sphingosine kinase in neurons, S1P, S1P receptors, G Protein Coupled Receptors, GSK3B and other factors. Information. International Journal of Molecular Sciences. Volume 19. Number 3. Page 891. March, 2018.

Homocysteine causes downregulation of the ubiquitin proteasomal systems, exhibiting a pivotal influences over phenotype and metabolic status of cellular entities, suggest the upregulation of proteasome activity in oncology occurs to counteract BAX, proapoptotic potential, and involves upregulation of choline kinase, phosphocholine, potentially aSMase/nSMase, sphingosine kinase 1 in non neurons and sphingosine kinase in neurons, S1P, S1P receptors, G Protein Coupled Receptors, GSK3B and other factors. Information. International Journal of Molecular Sciences. Volume 19. Page 891. March, 2018. The literature observes that in endothelial cellular entities of the aorta, as presented on nonhuman mammalian bovine study participants, autophagy is required for apoptosis produced by upregulated homocysteine and amino acid deprivation, explaining why oncology can typically be reliant upon proteolysis upregulation. The same study observed that upregulated lactate dehydrogenase release accompanied caspase segmentation, autophagy and apoptosis, such that clearly, parthanatos enablement of apoptosis among differentiated cellular entities and parthanatos enabled diminished potential for apoptosis among newly exhibited cellular entities were integral features of this syndrome. A SLC7A11 inhibitor disrupted caspase segmentation and diminished apoptosis as well as diminished lactate dehydrogenase release in a way that clearly suggest that caspase activity, autophagy, lactate dehydrogenase, homocysteine, PARP signaling,

NAD⁺ depletion, and upregulated DNA impairment are features of apoptosis in this context. Correlatively, another study that in small nonhuman mammals and mammals. Inhibiting the SLC7A11 cystine/glutamate transporter can be therapeutic in preventing apoptosis as well as disrupting a precursor metabolic syndrome to more pathogenic and complex contexts of oncology. SASP is an efficient inhibitor of SLC7A11.

Homocysteine, in Human neurons as well as small nonhuman mammal neurons, activates mTORC1, resulting in inhibition of autophagy which produces impaired proteins synthesis and folding.

Homocysteine activates a complex of leucyl-tRNA-synthetase to folliculin, activating mTOR by regulating mTOR linkage to lysosomal membranes.

Homocysteine upregulation and cystathionine beta synthase deficiency causes chronic and acute impaired clearance of proteins as well as produces accumulation of atypical proteins such as Beta amyloid and phosphorylated tau protein.³¹ Number 2. Beginning on Page 598. February, 2017. PMID 28148781. Information. Heliyon. Volume 6. Number 1. Pages e03315. January, 2020.

Methionyl – tRNA synthetase MetRS produces homocysteine thiolactone to prevent tRNA^{Met} being conjugated to Homocysteine. Homocysteine enters the first phase of protein biosynthesis because it is structurally similar to Methionine and methionine is required or 99.5 percent or more of transcriptional products because of its role in the priming sequence that initiates proteins synthesis.

Because stars utilized hydride as a source of energy, there seems to be a clear homologue between hydridic activity and the production hydridic voids when methyl groups are removed from s-adenosyl methionine to produce s adenosyl homocysteine, resulting also in metabolism toward homocysteine.

The + polarity in NAD^+ is similar to the removal of the + polarity that is exhibited upon the sulfur of S-adenosyl methionine before methyl groups and hydride becomes removed. These voids, then, are maintained in homocysteine, causing homocysteine to become a ubiquitous deactivator of biological systems, pathways, structure and activity. This pattern is interesting because in a star, depleted for or deprived of hydride, it's possible that atoms, molecules or even more complex molecules may emerge that have similar hydridic voids. Such hydridic void exhibiting atoms or molecules may be able to absorb, or frack the hydride from atoms or molecules which may continue to exhibit hydride. Although homocysteine has a structure similar to methionine and homocysteine can masquerade as methionine in carbonate buffering systems, biosynthesis, metabolism, structural functions and energy metabolism for the incipient phases of these processes before imposing a detrimental influence in these pathway, it is also possible homocysteine begins to deteriorate biological function in a pattern homologous to how material in a black hole may be performing to continue deteriorating the star's ability to emit light. Some of the recent research suggests that stars transiting areas where black holes interact with them, exhibit peculiar characteristics which suggest that the event horizon is not gravitational insurmountable. This example suggests that the light of the universes integrated into Human metabolism may function similar to how hydride is utilized in stars of the universes, such that molecules with void may coordinate the disorder, deterioration and diminished emittance of energy or light observed during biological deterioration and observed when star transitions to a black hole.

The hydride transfer complex HTC has been presented in the literature. HTC transfers reducing equivalents from NADH to NADP+. Malate dehydrogenase 1, cytosolic pyruvate carboxylase, as well as malic enzyme 1 perform assembly of HTC. HTC is presented as a reprogramming factor for f NAD metabolisms which promotes oncology, enables escape from senescence, is exhibited in cytosol of oncology cellular entities, is repressed in senescent cellular entities but activated when P53 is inactivated to constitute aerobic glycolysis, are expressed in correlation to prostate oncology in particular although inactivation causes senescence in oncology cellular entities to be reconstituted. HTC expression in extracellular environment is adequate to promote escape from senescence including rescuing cellular entities from inhibition of complex I inhibitors, as well as participate in oncology promotion by RAS cause oncology changes in primary cellular entities. Information. Molecular Cellular. Volume 81. Issue 18. Paes 3848 to 3865. Supplement e19. September, 2021.

Homocysteine upregulates mRNA synthesis EF1 alpha, EF1 Beta and EF1 data. J biol Chem. Volume 273. Number 31. Pages 19840 to 19846. July 31, 1998. PMID 9677419. Cyclohexamide prevented such upregulation of EF1.

Folate receptors are upregulated by elevated homocysteine, and are a feature of pathogenic oncology and can occur in typical cellular entities. Folate receptor synthesis upregulation can occur in pathogenic conditions although in some oncology, such as cervical oncology, such upregulation is not regressed once folate adequacy is achieved. Such folate receptor upregulation occurs without required upregulation of folate receptor mRNA or upregulation or an increase in hnRNP E1.

Homocysteine upregulation was coordinated with Folate Receptor upregulation. Folate receptor inhibition may be therapeutic in cervical or other oncology to counteract persistently upregulated and overly persistent Folate Receptors. Impaired ubiquitinase activity, modulated proteolysis as well as changed autophagy may also explain overly persistent folate receptor activity. J Clin Invest. Volume 113. Number 2. Pages 285 to 301. Jan, 2004. PMID 14722620.

The literature presents that In Vivo delivery of CRISPR/CAS9 focused on excision of HPV E6 and E7 genetic structures using pegylated liposomes results in complete regression and disappearance of neoplasms and results in 100 percent survival of experimental study participants or subjects. The gene editing was largely linked to NHEJ compared to high fidelity homologous repair, but this could be a result of large gene editing payload, although the neoplasms disappeared through apoptosis suggesting that cellular and humoral immunity as well as cellular decision mechanisms were extremely efficient at removing neoplasm or oncology once HPV E6 and HPV E7 genetic sequences were excised. Protein transduction therapy is field that has emerged and mature in developing protein domains that enable large payload biologically active molecules to be moved into the intracellular environment where intracellular chaperones reshape the payload into biologically active quaternary structure to resume shape, twist, writhe and reactivity characteristics. Linking CRISPR/CAS9 to transduction domains should allow the removal and deactivation of every known pathogen from human biology and should be able to cause the disappearance of every known oncology by excising or repairing the required genetic factors, typically numbering more than 1000, in oncology. Protein

transduction domains enable large molecules to permeate cellular physiology with 100 percent coverage and the efficiency of a water molecule's permeability of cellular physiology. Information. Mol Ther. Volume 27. Number 12. Pages 2091 to 2099. December 4, 2019. PMID 31537455.

Information. Gene Therapy. Volume 8. Numbers 1, 2, 3 and 4. 2001. March, 2001.

CRISPR/CAS9 has already completed eradicated HIV in small nonhuman mammals in 2013. Information. Nature Communications. Volume 10. Article 2753. 2019.

A most important observation in this context seems to be that in the context of translation wellness achieve by translational medicine, CRISPR/CAS9 along with protein transduction domains, potentiates rapid translation or reducing of discoveries and insight in biology and therapies, as well as disease and regenerative medicine into practice using personalized groups of interventions. Simply, not only is it possible to devise a combination therapeutic or groups of therapeutics for an individual, but using proteomic decision which CRISPR has already demonstrated, each cellular entity in physiology can have an individual care plan.

A review of the literature suggests that PEMT depletion of homocysteine may not be the intended role of PEMT which seems to be focused on biosynthetic and antihistamine factor enablement. PEMT produces homocysteine similar to other molecules which manage toxicity, xenobiotics, allergic interactions and other detrimental factors. PEMT's activity inhibits itself. Particularly, methionine synthetase, in the biosynthetic phase of methylation that follows one carbon unit processing by methionine synthase, attaches Adenosyl and ATP to

methionine, directed the integrated Methionine into toxicity management and biosynthetic pathways.

L homocysteine utilized along with trimethylsulfonium, dimethylthetin or other substrate such as dimethylsulfonioacetate by the catalysis of thetin-homocysteine methyltransferase(methylpherase or transmethylase) exhibits HS as an unpolarized sulfone while S-adenosyl methionine exhibits a polarized sulfone and while methionine and homocysteine both exhibit no such polarization of the sulfone. The polarizes sulfone specifies S-Adenosyl Methionine for the methyltransferase fraction used to excise methyl groups.

Importantly, the products of thetin-homocysteine methyltransferase activity include methylthioglycolic acid or methylsulfanylacetate along with an unremarkable version of methionine. Thetin homocysteine methylpherase, thus, seems like it may be the preferred mechanism of depleting homocysteine with a throughput of 2000 moles of flux per mole of Enzyme.

The distribution of detoxification methods seems to be ameliorated because of sulfur inadequacy which causes thetin – homocysteine methylpherase to exhibit intramolecular disulfide linkages that deactivate the enzyme and produce a gel phase.

The literature observes that Iron Sulfides or Fe-S as well as diverse sulfur molecule including Iron Sulfides not only are commonly exhibited in redox interactions but have dynamics similar to methyl groups, one carbon units and hydride, with Iron Centers or Iron Sulfides that are participating in both redox and reducing hydrogenic centers as well as reducing other molecules, but Iron Sulfides are components of the electron transport pathway. These expand the

possibility that sulfur inadequacy is a much more pervasive detrimental factor in all disease and aging.

Cursory analysis suggests that homocysteine may be of such detrimental nature because there may be inadequate thiols or sulfones to reduce or interact with the sulfur in homocysteine to produce disulfide linkages. Similarly, homocysteine may enhance already exhibited sulfur inadequacy to enhance the impairment of the thiol homocysteine methyltransferase.

Most importantly, the substitution of oxygen for sulfur in the storied large oxygenic event that archeologists have presented as occurring to diminish the percentage of sulfur in atmosphere and biology enabled by the similar periodic table position of sulfur and oxygen, along with sulfur inadequacy in practical context, would cause a remarkable redistribution of the percentage of processing of homocysteine that would occur by the thiol homocysteine methyltransferase compared to the processing of homocysteine by PEMT in biosynthetic pathways, resulting in increasing size of organisms as an anatomical factor but also increasing the pathogenic differentiation of organisms regardless of size. Regardless, the role of PEMT2 as an inhibitor of size increase and stabilizer of tissues and organs as well as structure and metabolism, compared to PEMT1 which is essential during gestation also because PEMT2 is exhibited near or after birth, presents the comparative activity of the thiol-homocysteine and transsulfuration pathway as comparative contexts in which the production of methylthioglycolic acid is a desquamation factor or dedifferentiation factor while the transsulfuration pathway depletes homocysteine but is also biosynthetic in how it produces cystathionine, cysteine, and glutathione among other factors that can assist in redox, reactive

		<p>molecular species management and production of other molecules used in metabolism. Information. Acta Biochim Pol. Volume 53. Number 4. Pages 685 to 691. 2006. PMID 17143336. ISBN 978-1-78984-276-0.</p> <p>These describe how hydride, the molecule or factor that fuels the stars of the universes, is integrated into human physiology and fuels or catalyzes the synthesis of fuel, gradients, redox transactions, and fracking processes which produce optimal conditions in physiology.</p>
<p>(PEMT inhibition Cascade Rescue) Choline Kinase Alpha Inhibitor (omit version beta because it affects development)(version alpha upregulation is required at birth and during gestation for pulmonary development adjustment to environment)</p>	<p>Pregnenolone, is the original physiological inhibitor of choline kinase alpha that focuses priority on activation of PEMT. Adenosine. DMAE. Piperazine, Cyclophane, Quinacrine, Stearoyl-CoA.</p> <p>Nonephemeral upregulation of choline kinase alpha is causally linked to pervasive oncology and disease.</p> <p>Because upregulation of choline kinase alpha is required in all of the pathogenic configurations of diverse nuances of oncology, choline kinase as a therapy should have already been able to abrogate oncology as a vector disease and substantial human detriment. Choline kinase developers have been in development since 1999 or earlier. Oncology Res. Volume 59. Number 13. Pages 3112 to 3118. July 1, 1999.</p>	<p>Inhibitors of Choline Kinase. Purinyl – 6 – Histamine. N – Methylmaleimide. ICL-CCIC-0019 inhibits choline Kinase. TCD-717. Carbocyanine dye, JAS239. Near Infrared Fluorescent Carbocyanine. Cidifovir or HPMPC.</p> <p>The literature observes that AP1 produces upregulation of choline kinase alpha. This pivotal linkage suggest that the AP1 moiety accompanying cFos has an integration locus in the Choline Kinase Promoter beginning at position negative 875 for 11 or 12 sequences in the negative direction. Information. Biochim Biophys Acta. Volume 1171. Number 9. Pages 1148 to 1155. 2007. PMID 17728180.</p> <p>Olive oil is known to cause apoptosis among in oncology cellular entities. However, instrumentation of choline prevents such apoptosis occurring, Upregulation of phosphatidylcholine occurs during extra virgin olive oil instrumentations and includes exhibition of polyphenols. Choline instrumentation prevents upregulation of choline kinase, and when choline kinase is not upregulated, the ability of extra virgin olive oil to disrupted the CDP choline pathway becomes likewise ameliorated. Olive oil also causes groups with the highest levels and</p>

most extended duration of instrumentation to have the lowest incidence of oncology. Information. Choline kinase is somewhat or literally required to be upregulate in much if not all oncology. Oleocanthal is observed to activate cMet pathway to cause apoptosis, although extra virgin olive oil activates the cFOS pathway which seems to cause an oncology prevention role compared to the other moiety of AP1 which is a survival signaling pathway that activates choline kinase alpha to be beneficial in the acute phase but can become pathogenic in extended duration by promoting survival even when conditions do not require such enhanced resilience. However, it is well known that homocysteine increases cause apoptosis enhancing changes in cellular entities and survival signaling is exhibited to counter act these by increasing BCL2 comparatively Bax or Bak. Choline instrumentation provides methyl groups, while olive oil instrumentation provides lecithin as phosphatidylcholine to supply membranes with phosphatidylcholine that downregulates requirement of choline kinase alpha and CDP Choline pathway synthesis of phosphatidylcholine. Similarly, the methyl groups enable activation of PEMT which produces DHA enriched phosphatidylcholine and DHA as well as other omega-3 and polyunsaturated fatty acids, all of which are cytotoxic specifically to oncology exhibiting cellular entities. Inhibition of choline kinase selectively causes apoptosis in oncology cellular entities. World J Gastroenterol. Volume 15. Number 15. Pages 1809 to 1815. 2009. PMID 19370776. Information. Curr Pharm Des. Volume 17. Number 8. Pages 805 to 812. 2011. PMID 21443483. Information. Curr Cancer Drug Targets. Volume 8. Number 8. Pages 709 to 719. 2008. PMID 19075594. Information. Olives and Olive Oil in Health and Disease Prevention. Chapter 55. Volume 2021. Pages 661 to 669. 2021. Information. Nutrients Volume 14. Number 4. Pages 908.

2022. Information. Clin Oncology Res. Volume 19. Number 9. Pages 2310 to 2318. 2013. Information. Phytother Res. Volume 34. Number 11. Pages 2820 to 2834. 2020. PMID 32449241. Information. Curr Oncology Drug Targets. Volume 8. Number 8. Pages 709 to 719. 2008. PMID 19075594. Information. Curr Pharm Des. Volume 17. Number 8. Pages 805 to 812. 2011. PMID 21443483.

Imperatively the effects of homocysteine are major enablers of the hidden nuances of agrin signaling including upregulation of fibronectin deposits in scar tissue, cardiac tissue and other tissue, as well as deterioration of matrix, apoptosis that depletes cellular entity density per micrometer to cause iNOS and persistent upregulation of mitogenic signaling. The literature observes that Agrin is essential for survival of monocytes or monocytic cellular entities. Information. PMID 22517892.

Diabetic neuropathy exhibits decrease in the Agrin fraction of complete heparan sulfate proteoglycan levels, clearly linking persistent and advanced pathology, again, with deterioration of the structural nuances of biology, regenerative downregulation and ameliorated repair capabilities. Information. Exp nephrol. Volume 9. Number 3. Pages 214 to 222. 2001. PMID 11340306.

An object of the preceding paragraphs involving EVOO or extra virgin olive oil supplementation was not only to confirm the role of olive oil in ameliorating oncology, ameliorating risk for oncology and link diverse patterns in disease with inadequacies of EVOO metabolites. Such object, instead, included establishing a priority of assuring the foundational material exhibited in cellular structure as a nuances of sustained health and diminished risk. The causal link between EVOO and Agrin availability as a beneficial nuance of EVOO therapy provide some such perspective.

However, a study of cardiovascular disease clearly links ceramide increases causally to exhibition of cardiovascular disease, which confirms the well known role of ceramides in cellular membranes as a foundational structural signal for apoptosis of cellular entities. Most importantly, EVOO supplementation, or extra virgin olive oil supplementation, was found to diminish the risk of cardiovascular disease by diminishing ceramide levels. Correlatively, EVOO or extra virgin olive oil, causes expression of Agrin, diminishes ceramide levels, causes apoptosis of cellular entities exhibiting oncology in a way that is prevented by the addition of choline to nutritional regimen.

These clearly present that the foundational nuance of pathology are deterioration of the production, availability and structural integration lecithin or phosphatidylcholine, choline, PEMT enzyme production of enriched phosphatidylcholine, all of which results in supplanting of these for upregulation of the CDP Choline pathway which integrates recycled choline into unenriched phosphatidylcholine as a nuance of inflammatory response or response to methyl group inadequacy. These also clearly present that the complexity in managing disease as well as the risk and potential for unsuccessful cytotoxic therapy in oncology therapy has the essential component of inadequate supply of material to constitute cellular structure. This inadequacy diminishes Agrin availability or diminishes extracellular matrix, each in a way that ameliorates agrin signaling, causes impaired PEMT function, upregulates choline kinase, and requires survival signaling to prevent complete deterioration of anatomical structure.

iNOS expression, S1P synthesis from upregulated ceramide, S1P receptor activation including G Protein Coupled Receptor Activation, ATPase, GTPase,

GSK3B activation, and S1P Lyase along with upregulated Proteolysis, increase in BCL2 to counteract BAX/BAK, all are nuances of such inflammatory pathways, which culminated in diminishing plasticity by omitting PEMT production of adequate fraction of phosphatidylcholine. The advancing nuances of change linked to this syndrome include the eventual dissociation of the mitochondria from the endoplasmic reticulum, preventing supply of Ca²⁺, Phosphatidylserine, and Phosphatidylethanolamine from the endoplasmic reticulum to the mitochondria, such that merely diminished PEMT becomes obliteration of PEMT2 function in the mitochondria. Information. Choline kinase modulation is emerging as a therapeutic area to be used as antimicrobial vectors ex vivo and in vivo. Biomed Research International. Volume 2020. Article ID 1823485. 7th month, 10th day, 2020. Inhibition of choline kinase is beneficial in pervasive oncology and disease because upregulation of choline kinase is integral, required and sometimes constitutive of pervasive disease, particularly oncology. Highly pathogenic and TRAIL resistant oncology of ovarian tissues are resensitized to therapy and diminished in pathogenic character by choline kinase alpha inhibition. Information. Journal of Experimental & Clinical Oncology Research. Volume 40. Article Number 5. 2021. "Plasma Ceramides in the Mediterranean Diet." Circulation. Volume 135. Issue 21. May 23, 2017.

This important conclusion presents a very useful context to understand the effects of iNOS, uncouple NOS, phospholipase D expression and phosphatidylcholine specific phospholipase c, all of which are upregulated responsively to environmental influences including electrical fields, wireless fields, environmental particulate and atmospheric particulate. Each of these participate in producing the deterioration and catabolism of

		<p>membrane phospholipids, particularly phosphatidylcholine, similar to the dysbiosis which occurs when choline, phosphatidylcholine, and lecithin are inadequate in nutrition and not produced de novo by the function of PEMT in physiology.</p> <p>Many inhibitors of choline kinase are suggested in the patent application 20130281445. US Patent and Trademark Office.</p>
(PEMT inhibition Cascade Rescue) Sphingosine Kinase modulators or Inhibitors (inhibit only version 1 if viral vectors or viral risk exhibited, although this can escape other tissues that require therapy but exhibit Sphk1 compared to Sphk2)	The spice Zingiberaceae, Caoguo, tsao ko or Amonum tsaoko Crevost et Lemaire. Ellagic Acid.	Sphingosine Kinase inhibitors. Opaganib inhibits Sphk2. ABC294640 inhibits sphk2. SKI-II inhibits both Sphk1 and Sphk2. N, N-dimethyl-D-erythro-sphingosine (DMS) and L-threo-dihydro-sphingosine (safingol) inhibit Sphk1 and Sphk2 but the emerging literature on specificity must be reviewed.
(PEMT inhibition Cascade Rescue) Sphingosine 1 Phosphate Receptor modulators or Inhibitors. S1P is the major effector of inflammation cascade and differentiation cascade at catabolic levels, being shaped into an effect by genetic, metabolic and transcriptional programs. It becomes pathogenic mostly because upregulated choline kinase and upregulated nSMase/aSMase both generate inflammatory phosphocholine and S1P is upregulated and activates programs in the inflammatory metabolic/proliferation/mitotic/differentiation context.	<p>Harmaline inhibits Sphk1. Luteolin inhibits Sphk2 and possibly Sphk1. N,N-dimethylsphingosine inhibits both sphk1 and sphk2, and natural occurs in physiology but toxicity and utilization parameters were not available in the literature at this instance.</p> <p>G Protein coupled Receptors exhibit individual polypeptides with seven spans of membranes as membrane integral proteins. G proteins are comprise heterodimers with an alpha subunit that integrates with guanine nucleotides, with G proteins tightly integrating with GDP guanine diphosphate in the basal status, such that G Protein Coupled Receptors translated GDP to GTP in a way that activates the G</p>	<p>Inhibitors or modulators of Sphingosine Kinase receptor. Fingolimod and Siponimod inhibit S1P receptors. (review literature for emerging specificity info.) . G Protein inhibitors, ATPase inhibitors, GTPase inhibitors. GPCR or G Protein Coupled Receptors can inhibit or activate coughing reflexes. Prostaglandin E2 and Bradykinin can enhance airway sensory nerves such as by EP3 receptor and B2 receptor activation, subsequently including TRPV1 and TRPA1 receptors. Inhibition of coughing reflexes can occur through B2 adrenoceptor Cannabinoid, although B2 adrenoceptor activation subsequently activates CAMP reliant Protein Kinase A activity with recent conclusions suggesting that Protein Kinase G is involved in BKCa channel hyperpolarization to inhibit coughing. Information. Curr Opin Pharmacol. Volume 11. Number 3. Pages 248 to 253. 6th Month, 2011.</p> <p>iNOS is considered to be in the pathway of G Protein Coupled Receptor activities while G</p>

	<p>Protein to enable G Protein activation cascades. Intrinsic Hydrolyzing of integrated GTP to GDP results in inhibition of coughing but microbial vectors, microbial toxins and xenobiotics can cause covalent modification of G proteins. Cholera or whooping cough is an example for pertussis toxin modification of G Proteins. Information. Annu Rev Physiol. Volume 58. Pages 143 to 170. 1996.</p>	<p>Proteins are known also to regulate both Phospholipase d1 and d2, in a way that involves phospholipase D recycling of desensitized G Protein Receptors including opioid, formyl and dopamine receptors. Thus, these describe the role EMF exposure in changes to behavior, involvement in addiction and compulsion, as well as in producing other influences described in these or other analyses. Information. Membranes (Basel). Volume 4. Number 3. Pages 302 to 304. July, 2014.</p> <p>GPCRs activate both phospholipase D and Phospholipase C while Phospholipase D activates iNOS and DPPC surfactant depleting phosphatidylcholine specific phospholipase C. Information. Naunyn-Schmiedeberg's Archives of Pharmacology. Volume 392. Part 2. 8th Month, 2019.</p>
(PEMT inhibition Cascade Rescue) Glycogen Synthesis Kinase Beta	<p>Putative Factors that inhibit GSK3. Lithium. Copper, Zinc. Melatonin. Curcumin. Butyrate. Quercetin. Apigenin. Luteolin. Astaxanthin. Cinnamon. Berberine. Angelica Sinensis. Resveratrol. 154 percent increase in curcumin availability was observed in a study at about 2 hours after administration while in the first half hour piperine/chavacine together or piperine results in 2000 percent increase in absorption of curcumin. Curcumin formulations as curcumin phytosome formulation was 790 percent higher than curcumin, while mix of curcumin volatile oils increase availability by 130 percent, although formulation of curcumin with hydrophilic carrier, cellulosic derivatives</p>	<p>Pharmacological Factors that inhibit GSK3. SSRIs. Trichostatin. Ketamine. Valproic Acid. Intranasal Insulin. Glycogen synthase kinase 3B, along with iNOS and Phospholipase D, are required for upregulated PSA Antigen and are essential for oncology of the prostate although inhibition of PSA antigen levels only alleviates PSA antigen reliant prostate oncology although inhibition of PSA antigen in PSA antigen independent oncology does produce a therapeutic benefit. PSA antigen is a factor in breast oncology also. Natural Cytotoxic T Cellular entities, when activated and exhibiting cytotoxic activity, inhibit by GSK3B by phosphorylating GSK3B. The phosphorylation of GSK3B skews its phosphorylated status by impairing the ability of GSK3B to autophosphorylate itself response to particular interactions along with phosphorylation by other factors and along with the ability of GSK3B dephosphorylate and phosphorylate other factors such as P53. P53 and GSK3B interactions are considered master regulators of</p>

and natural antioxidants resulted in a 4590 percent increased absorption of curcumin.

Naturopathic Factors that inhibit GSK3. BDNF. HDAC Inhibitors. Growth Factors. P90 RSK. HSP70. PI3K. AKT. PKC. PKA.

Toxic factors from environment that may be inhibiting GSK3 include beryllium, tungstate and mercury.

cellular proliferation and differentiation. GSK3B ability to phosphorylate itself represents its roles as master differentiator with the ability since intricate changes in the metabolic microenvironment and cascade these changes to factors such as WNT and B-catenin. Microenvironmental differences in metabolism and status are sensed by WNT, B-catenin and GSK3B, enabling pervasive Nano level differentiation programs to become effected. However, GSK can be hyperactivated by S1P receptor pathway activation, along with or independent of PEMT inhibition, P53 upregulation, choline kinase upregulation, nSMase/aSMase upregulation, Sphingosine Kinase upregulation, S1P receptor activation or status of S1P lyase's ability to deplete S1P to downregulate S1P receptor activation in a way that produces enhances ethanolamine phosphate and in a way that produces the Docosahexaenoic Acid,. DHA, precursor hexadecenal, a known pheromone that that strongly activates even insect attraction to human physiology. S1P lyase upregulation, however, is known to be a pathway of oncology, pathogen and therapeutic resistance, such that inhibition of S1Pn lyase can rescue resistant therapies or resistant pathology. Information. J Immunol. Volume 174 Pharmacological Factors that inhibit GSK3. SSRIs. Trichostatin. Ketamine. Valproic Acid. Intranasal Insulin. Pages 4551 to 4558. April 15, 2005. Acute Amyloid Leukemia exhibits strongly impaired Natural Cytotoxic T Cellular activity and inhibition of GSK3B is increased in Natural Cytotoxic T Cellular entities during canonical AML conditions. Inhibition of GSK3B reactivates or enhances Natural Cytotoxic T Cellular entity activity in patients experiencing AML. Information. Nat Commun. Volume 7. 11154. April 4, 2016.

<p>(PEMT inhibition Cascade Rescue) Bag1. (Although the literature may present difficult to understand information, the conclusion seems to be that Bag3 upregulates potential for Macroautophagy while Bag1 upregulates potential for Proteolysis, each competitively with each other. Proteolysis upregulation can produce a cycle in which upregulated choline kinase and aSMase/nSMase produces phosphocholine to supply proteolysis with ATP and choline, followed by utilization of ATP by proteolysis, freeing of choline and entry of choline again to be recycled by Choline Kinase alpha in particular, nSMase, aSMase, Sphingosine Kinase, S1P and then inflammation and resistance cycles occurring through S1P receptors, G Proteins, G Protein coupled receptors, ATPase, GTPase, GSK3, PDK, and S1P Lyase. Although nSMase and aSMase produces ceramide to potentiate apoptosis, it can be depleted by Sphingosine Kinase and other factors.).)</p>	<p>Melatonin may increase Bag1. Bag1 is considered to be a steroid activated inhibitor of apoptosis that impose inhibition of apoptosis using a highly conserved 100 amino acid concluding moiety that upregulates proteolysis. BAG1 may be emerging as a strong early prognostic indicator of breast oncology. Information. British Journal of Oncology. Volume 87. Page 834 to 839. 2002. It is important to remember that BAX1 is an apoptosis potentiator while BCL2 depotentiates apoptosis such that BAG proteins being BCL2 associated proteins, are features upregulated BCL2 typically and are linked to enhanced cellular survival. Bax1 upregulation, however, results in upregulation of survival pathways such that a enhanced BAX availability can be countered by upregulation of choline kinase, CDP – choline pathway, nSMase/aSMase, sphingosine kinase, S1P, S1P receptor activation including G protein activation, ATPase activity, GTPase activity and GSK upregulation. Melatonin may increase Bag1.</p>	<p>THIO-2 inhibits Bag1 and has been integrated into therapies which have been considered for approval such as 4'-Thio-2'-Deoxycytidine.</p> <p>Thioflavin-s inhibits interaction between Bag1 and Bcl2.</p> <p>Lithium and Valproate increase Bag1.</p> <p>Imatinib inhibits interaction between BAG1 and BCR-ABL.</p>
<p>(PEMT inhibition Cascade Rescue) Bag3(upregulated to improve toxicity resistance and downregulated for cytotoxic therapy)</p>	<p>BAG3 is considered to be the only BAG isoform which is induced by stress. BAG3 enables chaperone selective autophagy such as interaction with HSP70</p>	<p>Bag3 Immunoglobulin is available for Anti Bag3 therapy.</p> <p>Upregulators of bag 3 include 3 (4 methylphenylsulfonyl) 2 propenenitrile. Anthra (1,9 cd)pyrazol-6(2H) one. (6 (4 (2 piperidin 1 ylethoxy)phenyl)) 3</p>

and through the ATPase domain of HSP70, suggesting the G protein and S1P receptor are activators of HSP70 and BAG3 interaction with HSP70. Some of the literature observes that hyperphosphorylation is feature of oncology and pervasive pathology, suggesting ingestion of milk, particularly A1 or nonA2 milk, the activity of Casein Kinase, upregulation of Choline kinase to produce phosphocholine, upregulation of aSMase/nSMase to produce phosphocholine, sphingosine Kinase, S1P and S1P receptors are integral factors in oncology and disease, particularly when including activating S1P receptors, G Protein Couple receptors as S1P receptors, and GSK3B.

BAG3 integrates with factors other than Hsp70 through its WW domain and proline enhanced PXXP repeat. Some of the literature suggests that Bag3 is active in cardiac tissue but other literature suggest a wider tissue role for Bag3 particular in oncological conditions. BAG3 is able to stimulate apoptosis, development, cytoskeletal organization, as well as autophagy, particularly in skeletal muscle and cardiomyocytes. Bag3 can sustain oncological mobility, resistance to therapy and cellular survival using autophagy. However,

pyridin 4 ylpyrazolo(1,5 a)pyrimidine. 4 (5 benzo(1,3)dioxol 5 yl 4 pyridin 2 yl 1H imidazol 2 yl)benzamide.

Downregulators of Bag3 include ABT 737, , Elesclomol, Vinclozolin, Diethylnitrosamine, Dactinomycine, 3 (4 methylphenylsulfonyl) 2 propenenitrile, Estrogen, Anthra(1,9 cd)pyrazol 6 (2H) one.

autophagy has distinct features from proteolysis in oncology and inhibition of proteolysis through 20S proteasome inhibition disrupts canonical oncology although autophagy in some contexts can be an escape mechanism for oncology deprived of proteolysis.

Information. Cellular 'Deterioration' Dis. Volume 2. Number 4. Page e141. April 7

Bag proteins compete with HIP for integration at the Hsc70/Hsp70 ATPase domain. Integration here promotes substrate release. Bag1, Bag2 and Bag3 integrate with Hsc70 ATPase as a feature of mammalian cellular function and this integration at Hsc70 ATPase represses HIP activity. HIP1 availability is regulated by htt Huntington Protein's polyglutamine length and HIP1 integrates with HIPPI in a way that potentiates recruitment of procaspase 8 into a HIPPI/HIP1/procaspase 8 complex to invoke an apoptotic cascade. Thus, BAG3, BAG1 and BAG2 depotentiate procaspase 8 cascade by competitively inhibiting HIP1 when BAG3, BAG1 or BAG2 integrates with the Hsc70 ATPase.

Information. Nature Cellular Biology. Volume 4. Pages 95 to 101. 2002.

	<p>Cantharidin inhibits Bag 3. Bag3 is upregulated by Phenethyl Isothiocyanate and 2'-Hydroxycinnamaldehyde.</p> <p>Upregulators of Bag3 include Lipopolysaccharide, Acetaminophen, Motexofin gadolinium, Zinc Acetate, and Valproic Acid. Atmospheric particulate from transportation increases Bag3.</p> <p>Downregulators of Bag3 include, Staurosporine.</p> <p>Pancreastatin from the bulb of <i>Hymenocallis littoralis</i> transactivates Bax.</p>	
(PEMT inhibition Cascade Rescue) Bax	<p>BAX upregulation enhances the potential for Beclin 1 to interact with BAX, Bak and Caspase-3, while inhibition of PEMT resulting in upregulation of P53 and upregulation of homocysteine results in increased caspase-3, cytochrome C release, and upregulated Bax. A study observes that L-cystathionine inhibits Bax upregulation enablement of apoptosis, mitochondrial superoxide anion upregulation, mitochondrial potential deterioration, mitochondrial permeability membrane pore opening, cytochrome C release, caspase 3, caspase 9, as well as inhibits BAX upregulation. The same</p>	<p>Promacta, also eltrombopag, is known to inhibit Bax. Downregulating homocysteine can inhibit BAX. BAX Inhibitor 1 is an inhibitor of Bax. BAM7. BAI1, BTSA1, Kaempferol-3-O-rutinoside.</p> <p>BAX is upregulated by Nodakenatin and Phytohemagglutinin. BAX and BAK are sufficient to invoke apoptosis pathways among the other proapoptotic BCL2 group of factors. Information. "BAX Selective Inhibitors." Selleckchem.com/subunits</p> <p>BAX Inhibitor 1 is highly conserved among Eukaryotes as an inhibitor of BAX and repressor of apoptosis. Information. Information. Front Plant Sci January, 2018.</p>

	<p>study clearly identified homocysteine as an efficient imposer of apoptosis, particularly in HUVEC endothelial cellular entities.</p> <p>Information. Oxide Med 'Cell' Longev. Volume 2019. Issues 1253289. November 25, 2019. eCollection 2019. PMID 31885769.</p> <p>Resveratrol upregulates Bax.</p> <p>Dimethoxycircumin upregulates Bax.</p> <p>Pancreastatin from the bulb of Hymenocallis littoralis transactivates Bax.</p>	
(PEMT inhibition Cascade Rescue) Bcl2	<p>BCL2 isoforms each inhibit the exhibition of apoptosis or prevent apoptosis. BCL2 is located at the outer mitochondrial membrane among other locations. The BCL2 group of proteins includes BCL-XL, MCL-1, BCL-1, BFL-1/A1, BCL-B, Bax, BAK, Bok, BCL2L12, BCL2L13, BCL-G, BFK and BID. Among this BAX, BAK, and BID enhances potential for apoptosis while the others typically inhibit or prevent apoptosis. Typically exhibiting four BH motifs of between 20 and 10 amino acids. BH3 motifs are required for BCL2 factors the impose apoptosis while there are factors that exhibit only BH3 sequences relegating their influence to enhancement of apoptosis without inherent potential to enhance survival, including the BCL2</p>	<p>Navitoclax inhibits BCL2.</p> <p>Oncology cellular entities exhibit overexpression of BCL2. The dissociation of mitochondria from the endoplasmic reticulum typically can involve the endoplasmic reticulum unfolded protein response along with depletion of mitochondrial potential and ablation of mitochondrial apoptosis pathways by depletion of mitochondrial phosphatidylserine, phosphatidylethanolamine, phosphatidylinositol and Ca²⁺. BCL2 exhibition helps produce interactions between the dissociated endoplasmic reticulum and mitochondria which sustains their impaired direct exchange of factors and which impairs exhibition of apoptosis. Information. Biomed Res Int. Volume 2014. Pages 234370. 2014.</p> <p>BCL2 enhances the segmentation of XBP1 when overexpressed, while IRE1alpha promoted by BAX and BAK both can also enhancing splicing of XBP1, suggesting that the unfolded proteins response control by PERK, ATF6 and IRE1alpha translocated to the endoplasmic reticulum can be regulated by BCL2, BAX and BAK as a stress response to accumulation</p>

<p>homologues BID, BAD, BIK, BIM, BMF, HRK, NOXA and PUMA. BCL2 homologues BCL2 factors and homologues are diverse in sequence structure except for BH domains and except for BID which is similar to BCL2.</p> <p>BH3 only exhibiting homologues or factors typically integrate with either BAX or BAK as well occupy and competitively inhibit antiapoptotic sequences or locations in antiapoptotic BCL2 factors. Information. Cold Spring Harbor Perspect Biol. Volume 5. Number 2. Pages a008722. February 1, 2013.</p> <p>Monk Fruit Sweetener derivatives derived from <i>Siraitia grosvenorii</i> inhibit BCL2 and upregulates Bax. Dimethoxycircumin inhibits BCL2. Pancreastatin from the bulb of <i>Hymenocallis littoralis</i> decreases BCL2.</p> <p>Depolarization of the Mitochondria results PINK1 translocation to the outer mitochondrial membrane where it requires parkin and PARKIN polyubiquitinates outer membrane molecules that are part of the mitophagy cascade, resulting in P62 and NBR1 targeting translocation of depolarized mitochondria to autophagosomes using</p>	<p>of unfolded or misfolded proteins accumulating in the endoplasmic reticulum. Information. Biochem Biophys Res. Commun. Volume 466. Number 1. Pages 40 to 41. October 9, 2015.</p> <p>BCL2 regulates IP3 receptors, Endoplasmic Reticulum storage, molecular chaperones BIP and HSP70, akt, membrane tethering and sorting proteins, PACS2, DRP1 and mitochondrial fission proteins among others. Mitochondrial Associated Membranes consists of factors linking the endoplasmic reticulum to the mitochondria and BCL2 is among these structural linking proteins. Information. Acta Biochimica et Biophysica Sinica. Volume 50. Issue 6. June 2018. Pages 618 to 619. 6th Month, 2018.</p> <p>IL6 inhibits or prevents hyperoxia lung injury, suggesting its applicability in preventing viral vectors of alveolar impairment from the viral vector involved in the epidemic of 2020 and 2021. IL6 induces BCL2 expression in vitro and in vivo, disrupting proapoptotic and antiapoptotic signaling. H2O2 increases and correlated impairment of mitochondrial potential was also inhibited by interleukin 6. Interleukin 6 may be in important prevention mechanism for acute pulmonary distress syndrome occurring with SARS or Covid Pathology. The detailed nuances of alveolar capillary deterioration and protein leakage were prevented by enhanced BCL2 expression, presented a clear duality in the role of BCL2 since BCL2 is linked with oncology similarly to upregulation of choline kinase, aSMase/nSMase, sphingosine kinase, S1P, S1P receptors, G Protein Coupled Receptors, S1P receptors and other inflammation pathways. Survival signaling can be an important mechanism preventing the deterioration of essential biological systems and lungs can be specific targets of these factors during</p>
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ATG8/LC3II for recycling into submitochondria and refusion of submitochondria with adequate polarity into existing stable mitochondria. BCL2 proteins regulate mitochondrial outer membrane permeabilization both during apoptosis and through influencing exhibition of macroautophagy through Beclin1. BCL2 and MCL1 both antagonize mitophagy when BECLIN1 is exhibited. Suppression of mitophagy occurs resultant of PARKIN being forced to translocate to depolarized mitochondria through activity of BH3 only proteins or BH3 only mimetics. Information. Mol Cellular. Volume 55. Number 3. Pages 451 to 466. 8th Month, 7th Day, 2014.

Antiapoptotic BCL2 proteins are exhibited on the outer mitochondrial membrane and inhibit activation of proapoptotic proteins such as Bax and Bak. BH3 only protein inhibit antiapoptotic proteins or activate BAX/BAK, producing opposite outcomes. These interactions occur on the mitochondrial membrane surface and regulate transmittal of apoptotic signals to the mitochondria followed by determination of the release of caspase factors into the cytosol. Information. 1813.

distress and immediately after birth to maintain biological function.

Survival of experimental nonhuman organisms were prolonged when IL6 was used ameliorate pulmonary mitochondrial impairment. BCL2 inhibition deteriorated resiliency to pulmonary pathology, hyperoxia, alveolar pathology and mitochondrial potential deterioration. BCL2 preventing dissociation of BAK from MFN2 and prevented interaction between BAK and MFN1. Information. American Journal of Respiratory Cellular and Molecular Biology. Volume 41. Issue 4. December, 18

Small experimental organisms present through experimental studies that Macrophage MFN2 is required for the adaptation of mitochondria to respiration distress, which is mitochondrial respiration or oxidative phosphorylation fracking of hydride from NADH resulting in integration 42 percent of 2 eV- in between the phosphate groups of ATP while 58 percent of 2 eV- is utilized by the process of such integration of the 42 percent into ATP. MFN2 is also required the production of reactive oxygen species during inflammatory pathway activation and is required for inflammatory Nitric Oxide, presumably through iNOS, and is required for inflammatory cytokine synthesis. MFN2 inadequacy promotes autophagy dysfunction, apoptosis, phagocytosis and antigen processing. This suggests that MFN2 was redirected toward deactivation and occupation of BAK.

Another study observes that MFN2 and MFN1 integrate with BCL-XL, MFN2 and MFN1 both do not integrate with BAX unless apoptosis is being exhibited during which both MFN1 and MFN2 integrate with BAX, while MFN1 associated with oligomerized BAK but MFN2 did not associated with oligomerized BAK. suggest that

Number 4. Pages 532 to 539. April,

The initiation of mitochondrial apoptosis programs have started, or become activated, when the mitochondrial outer membrane permeabilization phase emerges and soluble factors begin to be released such as cytochrome C and complexed SMAC/DIABLO factors, each of which cooperate with caspase cascade that involve activation of cysteine, histidine, aspartic acid sequences in caspase substrates by cysteine proteases. BCL2, BCLXL, BCLW and MCL1 exhibit all four BH domains and are generally active at the outer mitochondrial membrane to prevent apoptosis induced by the BCL2 protein factors which invoke apoptosis. Bax, Bak and Bok each exhibit only the BH1, BH2 and BH3 domains and are presented as activators of apoptosis and only activate apoptosis although this does not preclude posttranslational modifications that affect the efficiency by which apoptosis is potentiated by BH3 only factors. The functional areas of the BCL2 group of proteins are the BH domains and a tail at the concluding locus of the C moiety. Bax and Bok oligomerize at the mitochondrial membrane to open pores in the mitochondria while BID, BIM and

MFN1 and MFN2 function differently in immunological monocytes when compared to other cellular entities. These also suggest that BCL2 promotes cellular survival by promoting a specific configuration of MFN1 and MFN2 interactions between endoplasmic reticulum and mitochondrial at the mitochondrial membrane and this configuration prevents apoptosis by disrupting the exchange of essential factors enabling mitochondrial apoptosis pathways from being exhibited. This condition is essential during short duration conditions linked with ephemeral pathology, but become integral factors pathologies the emerge with extended duration of pathology. Particularly MFN2 inhibits mitophagy by promoting coupling of the endoplasmic reticulum to the mitochondria enabling exchange of essential molecules that enable mitochondrial resiliency. Optimal mitochondrial potential depotentiates exhibition of mitophagy such that PINK1 accumulates in the inner mitochondrial membrane and cannot recruit mitophagy enabling ubiquitylation factors. Occupation of MFN2 by ancillary factors competes with mitochondrial stability and promotes dissociation of Mitochondria from the endoplasmic reticulum, thereby impairing mitochondrial potential and promoting mitophagy by enabling PINK1 accumulation in the outer mitochondrial membrane which where it can recruit ubiquitylation factors.

Information. Autophagy. Volume 14. Number 9. Pages 1658 to 1660. 2018. PMID 30081712.
Information. J Cellular Sci. Volume 133. Number 20. October 26, 2020.
Information. Cellular Rep. Volume 32Page 108079. 8th Month, 25th Day, 2020. PMID 32846136.

BCL2 proteins operate as constitutive factors within the mitochondrial associated membrane which is the area between the

BAD activate BAX and BAK as well as inhibited or repress BCL2 and BCLXL. Functionally, the literature considers BID, BIM and PUMA as BAX and BAK activators but their interaction with BCL2 antiapoptotic proteins is a regulatory one in which BCL2 inhibits the availability of BID, BIM and PUMA to prevent activation of BAX and BAK unless an elevated level of BID, BIM and BAD occurs to surmount available antiapoptotic BCL2 levels and produce free BID, BIM and PUMA that are able to interact with BAX and BAK. Similarly, elevated levels of BCL2 antiapoptotic proteins are required in order to surmount inhibition by BID, BIM and PUMA and result in BCL2 antiapoptotic phenotype exhibition.

Antiapoptotic BCL2 factors with multiple BH domains typically occur on the outer mitochondrial membrane or the endoplasmic reticulum, while BAK is also located at the outer mitochondrial membrane and while BAX is typically observed in the cytosol and becomes recruited to the outer mitochondrial membrane during apoptotic signaling while also BAX can be found in some levels within the mitochondria but this exhibition may

mitochondria and endoplasmic reticulum and factors exhibited between the mitochondria and the endoplasmic reticulum when the mitochondria is in communication with and interactive with the endoplasmic reticulum. Hundreds of mitochondria can be active in an individual cellular entity at any particular instances. Information. Act Biochimica et Biophysica Sinica. Volume 50. Issue 6. Pages 618 to 619. 6th Month, 2018.

BOK upregulates uridine monophosphate synthetase UMPS, increases uridine synthesis, increases sensitivity to apoptosis pathway invoked by 5 fluorouracil, in a way that that is prevented when BOK is inhibited and in a way that that cellular entities exhibiting pathology such as oncology can downregulate BOK to produce resistance to therapy such as 5 fluorouracil. Information. Proc Natl Acad Sci. U S A. Volume 116. Number 31. Pages 15469 to 15474. July 30, 2019. PMID PMC6681708.

BCL2 opposes the SARS M Protein ability introducing mitochondrial apoptosis in pulmonary epithelial cellular entities. SARS M Protein stabilizes BOK to prevent its ubiquitylation and it is this ubiquitylation that activates BOK by causing translocation of BOK to the mitochondria. M Protein endodomain was essential to its interaction with BOK while ablation of BOK experimentally diminished M Protein ability to introduce apoptosis. M Protein could experimentally produce apoptosis through BOK without BAX or BAK availability. BOK apoptotic activity resultant of M Protein required the BH2 domain. Experimental organisms had alveolar capillary permeability, apoptosis and pulmonary edema alleviated by inhibition of BOK. Information. Cellular Deterioration and Differentiation PMID 35022571.

	<p>be a function of competing transitions and fluctuations toward apoptosis and away from apoptotic phenotype. Mitochondrial BAX can be metabolic cascade that has not deterministically committed to apoptosis or antiapoptotic activity. Information. Biochemica et Biophysica Acta (BBA) Molecular 'cellular' research. Volume 1813. Issue 4. April 2011. Pages 532 to 539. April, 2011. PMID 21056595.</p>	
<p>(PEMT inhibition Cascade Rescue) PD – L1 (inhibiting choline kinase alpha and PD – L1 can impair each other's effects, in experimental contexts).</p>	<p>Kaempferol (KO) and kaempferol 7-O-rhamnoside (KR) both, as well as their source, geranii herba, inhibit PD – L1. Curcumin and apigenin together, Flavonoids such as Apigenin, Golden thread (A formosanus Hayata) extract AFE, and resveratrol in specific application, each inhibit PD-L1 or PDL1 or PD – L1. 154 percent increase in curcumin availability was observed in a study at about 2 hours after administration while in the first half hour piperine/chavacine together or piperine results in 2000 percent increase in absorption of curcumin. Curcumin formulations as curcumin phytosome formulation was 790 percent higher than curcumin, while mix of curcumin volatile oils increase availability by 130 percent, although formulation of curcumin with hydrophilic carrier,</p>	<p>Tecentriq, Opdivo, libtavo, Bavencio, and And Imfinzi, each are known to inhibit PD – L1. Keytruda, performs inhibition of PD – L1. Numerous other PD – L1 inhibitors with specific application are available or emerging.</p> <p>A brief presentation of the adaptive immunological synapse includes the upregulation of inflammatory signaling typically along with inhibited PEMT in an impaired cellular entity, resulting in a diminished volume of protein translation by polymerase PARS but an increase level of expression of inflammatory genetic sequences including upregulation of Major Histocompatibility Complexes. MHC molecules are produced and wander through the cytoplasm integrating with molecules in what systems like a random way because MHC integrates with such an inclusive array of molecules. MHC integrated molecules are escorted to the plasma membrane extracellular interface, or surface, where the MHC/molecule complexes are presented in CD4+ and CD8+ receptors typically in exhibited within lipid rafts and typically most densely represented in the Caveolae. This suggests why dilation of the</p>

<p>cellulosic derivatives and natural antioxidants resulted in a 4590 percent increased absorption of curcumin. PD1 exhibits dual roles in oncology, inhibiting hyperactive or detrimental immunological patterns while also producing immune system tolerance.</p> <p>PDL1 inhibits immunological response and its foundational role is to inhibit the implementation of immunological response programs in immunological cellular entities or immunological monocytes such as t cellular entities, natural cytotoxic T cellular entities, B lymphocytes, macrophages, dendritic cellular entities and monocytes in a more general context.</p> <p>During chronic microbial affliction, PDL1 can become expressed in exhausted TCD8 cellular entities resultant of demethylation of relevant promoter loci while the FOXO1 transcription factor integrates with PDL1 to promote increase expression of PDL1. AP1 expression is increased by oncological cellular entity leakage and AP1 increases the expression of AP1.</p> <p>Some epithelial cellular entities and some activate T and B Cellular entities exhibit PDL1 also.</p>	<p>caveolae by Nitric Oxide Synthases may be integral to optimal cellular health.</p> <p>The plasma membrane exhibits an outer interface and inner interface, as well as an interstitial space exhibiting toxic reactive molecular species including Nitric Oxide. The plasma membrane interstitial space is efficient at detrimental effecting biologically active molecules and xenobiotics such as pathogens, such that phospholipase D and iNOS expression are used by pathogens to produces endosomes within pathogens, viruses, bacteria or microbial toxins, proteins and other molecules can escape the toxic plasma membrane interstitial space and enter the cytoplasm. However, the outer plasma membrane and inner plasma membrane have essential characteristics that enable cellular entities to participate in adaptive immunity through enabling a cellular immune response. This characteristic includes microbial permeability, at least in some manner including proteins, toxins or other factors, into the toxic interstitial space where these microbial factors can be deteriorated or impaired, become dysfunctional, but also be able to incur MHC molecules which integrate with these proteins, and move these pathogenic or xenobiotic factors to lipid rafts where they are presented in CD4+ and CD8+ receptors in lipid rafts.</p> <p>CD4+ receptors are integrated with by monitoring T Cellular entities which copy the escorted protein sequences in opposite order, then perform a V(D)J recombination DNA repair which integrates this opposite order amino acid sequence into the DNA of the T Cellular entity. The T Cellular entity then migrates to the Thymus where it proliferates, is chaperoned by nurse cellular entities, tested multiple times to prune those cellular entities which are too autoreactive or which</p>
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AKT, including the PTEN/AKT pathway can result in upregulation of PDL1. The MAPK pathways is linked with downregulation of PDL1. JAK/STAT upregulates expression of PDL1. NFkB enhances expression of PDL1. Hedgehog protein HH can cause expression of PDL1.

PD1/PDL1 pathway as well as PD1/PDL2 pathway performs and assistive role in oncology because their T Cellular Activation Roles, Proliferation enablement roles, both are most typically accompanied by enablement of cytotoxic secretion and all of these are commandeered by the neoplasm microenvironment to produce immunological tolerance in the neoplasm microenvironment.

Inhibitors of PD1/PDL1 including the monoclonal immunoglobulin Nivolumab, Pembrolizumab, JQ1, Atezolizumab, Avelumab, and Cemiplimab, Information. Am J 'oncology' Res. Volume 10. Number 3. Pages 727 to 742. 2020. PMID 32266087.

The more simplistic literature observes that PD-1 is a CD28 superfamily receptor that produces destabilizing

are too sensitive to endogenous physiological sequences, resulting highly specific T Cellular entities that that can then be directed toward free and cellular presented sequences correlated to the inverted sequences of nucleotides.

Bone marrow derived CD4-CD8- double negative progenitor development into competent T cellular entities occurs in the Thymus where a series of differentiation and selection phases, including presentation of self antigen by thymus endothelium and the influences of fibroblasts, macrophages, dendritic and other epithelial cellular entities results in depletion of 95% of developing thymocytes particularly by the F4/80+ macrophages. Thymic macrophages seem to emerge from hematopoietic cellular entity progenitors while tissue resident macrophages seem to be derived from lineages of yolk sack progenitor cellular entities. The development includes TCR B gene locus rearrangement to produce a preTCR complex, followed by rapid division into CD4+CD8+ DP thymocytes which have a rearranged TCR alpha gene sequence, such that when a thymocyte produces an alpha gene sequence, the preTCR becomes mature TCRalphabeta gene sequences, followed by production of CD4+/CD8+TRCAlphabeta^{low} thymocyte phenotype/genotype that allows interactivity with self.

Thymocytes with intermediate interactive affinity to self continue to differentiate into CD4+ or CD8+TCRalphabeta^{HIGH} single positive thymocytes, followed by exit from the thymic cortex into the thymic medulla to complete maturation phases, although those thymocytes sensitive to the TCRalphabeta strong signal are caused to incur apoptosis.

Thymocytes with higher specific affinities for particular ligands can be diverted into

intracellular signals when it is activated by PDL1 or PDL2. The upregulation of PD1 clearly is presented as adaptive mechanism, and the literature presents it as a feature of autoimmunological prevention in physiology. Its clear that PD-1 and PD-L1 as well as PD-L2 would be useful in managing the massive apoptosis that may be introduced resultant of choline inadequacy, PEMT inhibition, pathogenic influence, or cytotoxic therapy as well as upregulation immunological activity link to the deteriorating thymus, a in interstitial feature of which includes upregulated autoimmunological activity as the thymus deteriorates to loosely aggregated fatty tissue resultant of choline inadequacy and diminished levels of FOX factors which have been reintroduced into physiology to cause complete structural regeneration of the thymus. Information. Curr Top Microbiol Immunol. Volume 350. Pages 17 to 37. 2011. PMID 21061197.

A review of the clinical literature presents that management of homocysteine using B vitamins results in downregulation of PD-1. The mystery of PD-1 and PD-L1/PD-L2 upregulation in disease, thus, seems to clearly be linked to inadequate prioritization of

CD+CD25FoxP3+tT_{reg} silencing excessive peripheral immune responses to produce peripheral tolerance, thereby diminishing potential for autoimmunity. Approximately 90 percent of nonsilenced DP thymocytes express TCRs that are not able interact with MHC complexes load with nuances of endogenous or self molecule. Information. "Adenosine in the Thymus." From PharmacolDecember, 2017.

	<p>homocysteine, choline adequacy, fundamental lipid chemistry physics, methyl groups, and PEMT function. Essentially, withholding dimethylacetothetine from clinical application since 1878 is a primary cause of PD1, PDL1 and PDL2 correlated pathology. Information. Int J Mol Sci. Volume 20. Number 15. Page 3763. 8th Month, 2019. PMID 31374832.</p>	
<p>(PEMT inhibition Cascade Rescue) PTEN inducible Kinase1 PINK1 (in the inner mitochondrial membrane when the mitochondria has optimal unimpaired polarity) (In the outer Mitochondrial Membrane when the Mitochondria is has impaired polarity) PINK1 recruits Parkin in the outer mitochondrial membrane when polarity of mitochondria is impaired, activating autophagy sequestration ubiquitylation activity that deteriorates mitochondria to submitochondria, of which those with impaired polarity are deteriorated while those with optimal polarity are integrated into other or new mitochondria.</p>	<p>Inhibition of the competing USP30 protease enables upregulation of PINK1/Parkin activity, enhancing deterioration, cleaning of impaired Mitochondria. Betulinic Acid upregulates PINK1, enhancing autophagic mitophagy in polarity compromised/impaired mitochondria by recruiting Parkin to the outer membrane of impaired mitochondria, recruiting Mfn2, constituting a basic ubiquitylation sequestration that activates Autophagy as Mitophagy in which impaired mitochondria become deteriorated to many submitochondria which are integrated into new or other mitochondria if polarity is optimal but deteriorated additionally if the submitochondria is impaired. Betulinic acid is effective in treating diverse conditions of cellular proliferation and migration, including resistant versions of conditions resilient to diverse therapies.</p>	<p>Betulinic acid, particular its derivative B5G1, is known clinically as the FDA approved drug 3-o-(3',3'-dimethylsuccinyl) betulinic acid or Bevirimat already approved for retroviral therapy, although here it can be used to upregulate PTEN, protecting P53 from MDM2 ubiquitylation, as well as upregulating PINK1 to enable more complete sequestration of Parkin to PINK1 in the outer mitochondrial membrane of impaired mitochondria for recycling of impaired mitochondria. The Macrolide antibiotic Bafilomycin A1 or BAF A1 potentiates apoptosis when instrumented along with Betulinic Acid. MDIVI-1 instrumentation along with inhibition of PINK1 pathway results in similar effect as B5G1 derivative of Betulinic Acid. Quizartinib Inhibits FLT3 more effectively than CEP-701, MLN-518, PKC-412, Sunitinib and Sorafenib, performing as an inhibitor of the PINK1/Parkin pathway.</p>

	MDIVI-1 also upregulates PINK1. A natural inhibitor of FLT3 is Isoliquiritigenin ISL derived from Licorice.	
PARP, Poly (ADP - Ribose) Polymerase Management (persistently signals by catabolizing NAD ⁺ at locus of DNA repair until repair has been affected, dispensing ribose to local substrate causing gradients upon which substrate for DNA repair is recruited. DNA repair happens about 1 million times each day in each cellular entity while inhibition of PEMT impairs Pentose Phosphate Pathway synthesis of Nucleotides used in DNA Repair.) PARP use in gene transcription can have similar paradox. Causes depletion of NAD ⁺ , upregulation of Homocysteine through Nicotinamide Methyltransferase detox of NAD ⁺ remnants, inhibits Glucose 6 Phosphate Dehydrogenase, causes parthanatos in which differentiated cellular entities deteriorate and new stem cellular entities are unable to experience apoptosis.	Strong natural incubation inhibitors of PARP include myricetin's 93% potency, quercetin's 80%. Also, gossypetin's 73%, delphinidin's 62%, quercetin's 62%, fisetin's 60%, all as medium to strong inhibitors while 12 percent or lower levels of inhibition were observed with Baicalein, naringin, (+)-catechin, phenol, catechol, resorcinol, hydroquinone, methyl quercetins. Ribose Supplement, NAD ⁺ Supplement, NAD ⁺ upregulating supplement because the literature observes synthetic NAD ⁺ as being too large for metabolic effectiveness, and Ribonucleotide supplement RNA, Deoxyribonucleotide supplement DNA, management of homocysteine all help alleviate the conditions caused by PARP/PARP signaling. Flavanoids Trizetin, Myricetin, Gossypetin, Delphinidin, Quercetin, Rutin, Fisetin, are efficient inhibitors of PARP while Baicalein, Naringin, (+)-catechin, each performed low inhibition of PARP(PARP1 in particular). Niagen is a supplement which increases NAD ⁺ using precursors instead of	Olaparib Mitophagy/Autophagy inhibitor, Talazoparib, Niraparib, Veliparib, Senaparib, Rucaparib, Rucaparib Phosphate, Niraparib tosylate, Pamiraparib, Rucaparib, Talazoparib tosylate, Dehydrocorydaline chloride, 3 - Aminobenzamide, all are inhibitors of PARP.

directly supplying
NAD⁺. Ribose
Supplement, RNA
Nucleotide Supplement,
Supplemental DNA
Nucleotides,
supplementation of the
substrate and product of
the Gene. NAD⁺ ,
NADH, Pyruvate,
Uridine, NADH
precursors (Vitamin B3,
Nicotinic
Acid/Niacin[causes
flushing and not
effective in all tissues],
Nicotinamide/Niacinami
de[widely effective but
inhibits sirtuin activity],
Nicotinamide
Riboside[optimal and
manages insulin
resistance/sensitivity],
Nicotinamide
Mononucleotide[optimal
, available in all tissues,
activates SIRT1, does
not inhibit sirtuins, has
highest bioavailability in
circulatory pathways
and increases NAD in
the intracellular
environment the most
among these other
factors]). CD38 is major
depletion pathway for
NAD and CD38 is
major factor in depletion
of NAD resultant of and
causal for senescence
and detrimental factors
associated with aging.
Taxifolin,
dihydroquercetin,
Apigenin, Luteolin,
which are supplements,
as well as Callistephin
found in deep/dark blue
nutritional factor or
foods such as
blueberries, wine
grapes, and
pomegranates, and as
well as Kuromaninis
found in black currant,
red raspberries, Peruvian

corn and lychees.
Flavonoid and Anthocyanin rich foods are recommended in this context such as black rice, blood oranges, red onions, red cabbage, acacia, black plums, etc. however, experimentally, the most rapid mechanism and most assured way to change NADH to NAD⁺ ratio in which NAD⁺ is required to be very high compared to NADH to potentiate release of H⁺ is to manipulate the ratio of pyruvate to lactate. Lactate Dehydrogenase translates pyruvate and NADH to lactate and NAD⁺. The cellular processes the mine for energy factors often mine for and release the hydridic energy factors which PEMT integrates into physiological structure when it integrates the three methyl groups first in enriched phosphatidylethanolamine to produce phosphatidylmonomethylethanolamine, then into phosphatidylethanolamine to produce phosphatidymethylethanolamine, and then into phosphatidylmethylethanolamine to produce enriched versions of phosphatidylcholine. Each methyl group, CH₃, has three hydrogen atoms, one of which is considered to be functional hydride, and the action of PEMT results in de novo synthesis of choline within phosphatidylcholine which has particular

	resonant characteristics that require a distant unattached molecule to balance its polarity, resulting in interesting quirks of physics that occur between its structure and other molecules including the hydridic effect that exudes negative polarity to produce background alkalinity or pH between 7.2 and 7.6 which is essential to biological potentials, function, and capacitant fields constituting conscious cognitive function.	
Polo like kinase 1, Plk1, enables progression into Mitotic M Phase and is essential to stabilization of structure and genome through the mitotic phases. Plk1 is regulated by PTEN through integration with Plk1 and regulation of which factors can integrate with Plk1 as well as regulation of how Plk1 obscures and obscures its own catalytic loci. Plk1 phosphorylates Cdc25 which then dephosphorylates CDK1/Cyclin B complex which potentiates exhibition of M Phase or Mitotic phase.	The natural factor 3 {[(1R,9S) 3 (naphthalen 2 yl) 6 oxo 7,11-diazatricyclo[7.3.1.0 ^{2,7}] trideca 2,4 dien 11 yl)methyl}benzonitrile is known to inhibit PLK1. Purpurogallin PPG is known to inhibit Plk1 and Plk2 but not Plk3, as derivative from nutgall and oak bark. Aristolactam AIIIa inhibits Plk1. Thymoquinone inhibits Plk1.	There are 35 or more approved Plk1 inhibitors with more than 500 prospective inhibitors in development at one time in recent contexts. Volasertib, PCM – 075, Onvansertib, and Rigosertib, each are Plk1 inhibitors.
Upregulation of Cytochrome P450 Axial segmentation system, P450 scc, enabling cholesterol to become pregnenolone, inhibiting choline kinase, upregulation production Testosterone, Estradiol and Estrone, interactively. This is typically optimal or beneficial.	Forskolin. However, pregnenolone is an embryonic plasticity potentiator which inhibits upregulation of the CDP – Choline pathway and specifically upregulates the CDP – Ethanolamine pathway that leads to PEMT function. Pregnenolone may be therapeutic in diverse contexts.	Forskolin. Forskolin. However, pregnenolone is an embryonic plasticity potentiator which inhibits upregulation of the CDP – Choline pathway and specifically upregulates the CDP – Ethanolamine pathway that leads to PEMT function. Pregnenolone may be therapeutic in diverse contexts.
Phosphatidylserine, provides substrate for synthesis of the Phosphatidylethanolamine in the Mitochondria by Phosphatidylserine Decarboxylase	Phosphatidylserine, provides substrate for synthesis of the Phosphatidylethanolamine in the Mitochondria by Phosphatidylserine Decarboxylase	Phosphatidylserine, provides substrate for synthesis of the Phosphatidylethanolamine in the Mitochondria by Phosphatidylserine Decarboxylase

Phosphatidylethanolamine, provides substrate providers subtracted for PEMT1, PEMT2 and PEMT3 catalysis in the Endoplasmic Reticulum, Mitochondria, and Golgi Apparatus.	Phosphatidylethanolamine, ethanolamine, Phosphoethanolamine, CTP – Ethanolamine, phosphatidylserine	Phosphatidylethanolamine, ethanolamine, Phosphoethanolamine, CTP – Ethanolamine, phosphatidylserine
Ca ²⁺ supplement, helps assist to alleviate depletion of Ca ²⁺ caused by numerous stimuli of iNOS and eNOS as well as nNOS. Reenables exchange of Ca ²⁺ and therefor also exchange of Phosphatidylethanolamine and Phosphatidylserine between Endoplasmic Reticulum and Mitochondria, alleviate primary reason for cellular entities resistant to apoptosis/deterioration pathways and cellular entities overly sensitive to apoptosis/deterioration pathways.	Calcium as Ca ²⁺ , Vitamin K2 and Menaquinone – 4 each assist in promoting availability of Ca ²⁺ . FIPI prevents Ca ²⁺ depletion that occurs along with phospholipase D activation.	Calcium as Ca ²⁺ , Vitamin K2 and Menaquinone – 4 each assist in promoting availability of Ca ²⁺ .
Gray Hair. A solution to less than optimal nuances of aging.	Catalase reverses graying of hair, depleting H ₂ O ₂ . Increased levels of homocysteine deactivate Catalase. Managing homocysteine is recommended. Taking a complete regiment of Superoxide Dismutase, Catalase, N – Acetyl L Cysteine, Vitamin C, Vitamin E. Folate. Grape Seed Extract/Oil. Olive Oil.	Enlyte/EnlyteRX. 3,3 DMB. Catalase. TMA Lyase inhibitor.
Phosphatidylethanolamine/Phosphatidylcholine (nonmembrane, thereby referring to phosphatidylethanolamine supplementation or availability) (within membranes, the phosphatidylethanolamine to phosphatidylcholine ratio potentiates apoptosis when phosphatidylethanolamine is increased, resilience and dysbiosis when unenriched phosphatidylcholine is increased compared to phosphatidylcholine, and optimal status when ratio of ether linked fatty acids is increased in the phosphatidylcholine fraction of membranes phospholipids) Ratio. Phosphatidylethanolamine to phosphatidylcholine Ratio. Typically, ratio of available substrate for PEMT catalysis compared to produce of PEMT catalysis, suggesting that when Phosphatidylethanolamine is low, the fraction of membrane phosphatidylcholine produced in the unenriched or unenhanced CDP – Choline pathway, instead of by PEMT, is upregulated, promoting inflammation and dysbiosis. Indicates a dysbiosis in autophagy, mitophagy, and proteolytic as well as sequestrome catalysis link to all manner of disease involving cellular proliferation. Phosphatidylethanolamine is known to inhibit the effects of aging and stress upon physiology in a	Inhibitors of PEMT, Inhibition of PEMT, inhibition of the CDP Ethanolamine pathway, inhibition of Phosphatidylserine Decarboxylase pathway, inadequate phosphatidylethanolamine, inadequate phosphatidylserine, unfolded protein response, inhibition of choline kinase alpha, impaired mitochondria, impaired PEMT, Ca ²⁺ depletion, all can produce decreases in phosphatidylethanolamine compared to phosphatidylcholine.	Inhibiting inhibitors of PEMT, activation of the CDP Ethanolamine pathway, phosphatidylserine, phosphatidylethanolamine, mitochondrial stabilization, Ca ²⁺ availability, stabilizing Mfn1, stabilizing Mfn2, all assist in increasing phosphatidylethanolamine availability to the mitochondria.

<p>hormesis pattern, perhaps because its affect to aging is in alleviating the structural basis of changes that are precursors or empirical factors in aging. Inhibition of GSK3beta seems to inhibit a core molecular and metabolic cause of aging which occurs after the factors managed by phosphatidylethanolamine. The reduction of homocysteine by factors such as danshen or phosphatidylethanolamine is known to be accompanied by stimulated sense of hunger which is not precise but response to unfamiliar decreases in homocysteine, which is linked to hunger psychology because a typical meal can supply substrate for synthesis of homocysteine or factors which require homocysteine producing enzymes to exhibit catalysis. Phosphatidylethanolamine along with a GSK3beta inhibitor such as epigallocatechin results in decreased homocysteine but also results in an increase in glycogen synthase activity, thereby freeing amyloid B from storage fibrils, artificially suppressing an artificial hunger pathway by freeing energy substrate through glycogen cycling. Ordinarily, epigallocatechin would cause a similar effect, but Glycogen would be cycled from and into amyloid fibrils if homocysteine were increased because homocysteine inhibits PEMT, upregulates P53, inhibits endocytosis of sugar and greatly diminishes glucose entry into Glycolysis, thereby producing a trap that causes products of gluconeogenesis and ephemeral glucose endocytosis to be limited to glycogen cycling into amyloid fibrils and out of amyloid fibrils, constituting a canonical condition for adipose cellular entities to emerge, be sustained and grow. Instead, depleting homocysteine produces a status that potentiates gluconeogenesis if glucose is not being obtained at increased levels, while phosphatidylethanolamine enables PEMT which health diminish this hunger by inhibiting inflammation and promoting exhibition of oxidative phosphorylation, all while glycogen synthase activity is upregulated because epigallocatechin inhibits GSK3beta to prevent GSK3beta inhibitory phosphorylation of Glycogen Synthase. Essentially, Phosphatidylethanolamine replaces extended duration of being without reducing glucose obtainment that at least achieves extended vital being observed in a comparative model of glucose obtainment reduction. Phosphatidylethanolamine also prevents toxicity from amyloid beta.</p>		
<p>Bax/Bcl2 ratio. Bax to Bcl2 Ratio. Indicates how upregulated the stress upon cellular entities is promoting apoptosis, thereby requiring an inflammatory survival signaling response linked to pervasive disease. Inhibited PEMT activity and</p>	<p>Inhibition of PEMT, inhibitors of PEMT, inadequate Phosphatidylethanolamine, depletion of Ca²⁺</p>	<p>Inhibition of inhibitors of PEMT, phosphatidylethanolamine, phosphatidylserine, GSK3B inhibitors, S1P receptor inhibitors, Sphingosine Kinase a inhibitors, G</p>

<p>elevated homocysteine produce an inherent increase in Bax. Ki67 may also be coupled with the Bax to BCL2 ratio for optimal diagnostics regarding the effect of oncology therapeutics.</p>	<p>although depletion of Ca²⁺ impairs mitochondrial apoptosis pathways also, inadequate phosphatidylserine, impaired mitochondria, GSK3b, and Homocysteine, all increase the Bax ratio compared to Bcl2. S1P production is upregulated to preventing completion of the apoptosis cascade in cellular entities resulting in activation of G Protein survival signaling, S1P receptor activation and Pyruvate Phosphate Dehydrogenase Kinase phosphorylation which enables this enzyme to inhibit production of Acetyl – CoA by Pyruvate Phosphate Dehydrogenase in a way that redirects pyruvate toward lactate dehydrogenase and generation of additional NAD⁺ that can be consumed by PARP signaling.</p>	<p>Protein and G Protein linked GTPase/ATPase inhibitors, management of Homocysteine, assurance of PEMT, all can enable decrease in Bax Ratio. Supplementing Ca²⁺ can also be assisted by EMF protection as it blankets, clothing, covering, enclosures or devices as in supplying calcium to relieve muscle cramping in during exercise, such that the reason factors such as coconut water help to prevent cramps is that the phospholipids in coconut water exhibit Ca²⁺ encircling the lead groups within phospholipid structure. Energy fields and Electricity, as well as wireless communications cause iNOS which depletes Ca²⁺, particular effecting well lit, outdoor and broadcasted athletic events. Muscle, voluntary or involuntary, dysbiosis can involve external energy, wireless, communications, extreme, and not so extreme fields disrupting or inhibiting endogenous neurological, ionic and muscular rhythms.</p>
<p>Inhibition of the 20S Proteasome by particular versions of proteasome inhibitors in broadly specifically deteriorating of oncology exhibiting cellular entities. The 26S, 20S and 19S proteasomes are pervasively exhibited in tissues whereas the Immunoproteasome is exhibited in immunological monocytes and lymphocytes, and whereas the thymoproteasome exhibited in the thymus cortical epithelium, all as interesting factors in affecting the proliferation of these varied cellular entities. The 26S Proteasome is 31 subunit proteolysis performing molecular machine that exhibits either one 19S particle at one extremity or exhibits 19S proteasome particles at both extremities. The 19S regulatory complexes participate in sequestrome activities by assisting the movement of ubiquitylated proteins and material to the 20S proteasome particle. Eukaryotic cellular entities utilize these proteolysis particles to deteriorate proteins sequestered from the cytosol and nucleus in particular. The 26S Proteasome</p>	<p>Natural inhibitors of Proteasomes including lactacystin, green tea polyphenols, traditional medicinal triterpenes, Clastol Lactacystin beta Lactone, EGCG, Apigenin, Quercetin, Genistein, Curcumin, Genistein, daidzein, pigenin, chrysin, luteolin, myricetin, kaempferol, quercetin glucosides, hesperetin, naringenin, narirutin, eriodictyol, neohesperetin, catechin, ECG, EGC, EC, Cyanidin, Malvidin. 154 percent increase in</p>	<p>Bortezomib is an inhibitor of the 20S Proteasome and is indicated for myeloma but is toxic and potentiates toxicities and complications. Carfilzomib exhibits improved sustainability but also can be toxic and is indicated for myeloma also, similarly to its analogue, oprozomib. Ixazomib is orally administered compared to these others, but has a low duration of exhibition also. The natural Salinosporamide A, marizomib, inhibits proteasome with short duration of exhibition but is able to transit the membranes separate the brain from circulatory pathways. Proteasome inhibitors typically, but not always, inhibit the chTL B5 subunit because such inhibition causes a most substantial</p>

<p>requires ATP but the 20S proteasome utilizes along already unfolded peptides. Thus the exhibition of ATP within the 26S proteasome suggests that the supply of phosphocholine by choline kinase and by nSMase/aSMase as well as the catalysis of the 26S proteasome may be biosynthetic while the 20S proteasome may be a deterioration pathway. 26S ATPases as well as PAN perform unfolding of proteins, allowing unfolded 26S products and PAN products to enter 20S proteasome subparticle processing. 26S proteasomes prefers ubiquitinated proteins.</p>	<p>curcumin availability was observed in a study at about 2 hours after administration while in the first half hour piperine/chavacine together or piperine results in 2000 percent increase in absorption of curcumin.</p>	<p>impairment of proteolysis, although B5 inhibitors have, also, some affinity for B1 and B2 subunits.</p>
	<p>Curcumin formulations as curcumin phytosome formulation was 790 percent higher than curcumin, while mix of curcumin volatile oils increase availability by 130 percent, although formulation of curcumin with hydrophilic carrier, cellulosic derivatives and natural antioxidants resulted in a 4590 percent increased absorption of curcumin.</p>	<p>IPS1 -001 selective for B1i subunit and ONX0914 selective for B5i subunit, are both immunoproteasome inhibitors. IPS1 – 100 produces aggregated ubiquitylated sequestrome products as well as causes caspase pathway activation linked to apoptosis useful hematological proliferation conditions. ONX0914 decreases MHC antigen presentation at the cellular surface and reduces cytokine secretion without substantial toxicity, with high selectivity for the B5i subunit. KZR – 616 is also known to inhibit immunoproteasome activity.</p>
	<p>Celastrol is a natural proteasome inhibitor derived from <i>Tripterygium wilfordii</i> and <i>Tripterygium regelii</i>.</p>	<p>The small molecule inhibitor MG132 inhibits the 26S proteasome and prevents Androgen Receptor deterioration, including preventing galeterone inhibition of androgen receptor</p>
<p>Epoxomicin is derived from an Actinomycetes lineage resultant of its potent antineoplastic characteristics. Epigallocatechin gallate, Epigallocatechin – 3 – Gallate or EGCG, is an antioxidant derived from Tea, particularly Green Tea. glidobactin C is a general proteasome inhibitor. There are literature which specifically present natural proteasome inhibitors as a starting point for drug discovery.</p>	<p>Epoxomicin is derived from an Actinomycetes lineage resultant of its potent antineoplastic characteristics. Epigallocatechin gallate, Epigallocatechin – 3 – Gallate or EGCG, is an antioxidant derived from Tea, particularly Green Tea. glidobactin C is a general proteasome inhibitor. There are literature which specifically present natural proteasome inhibitors as a starting point for drug discovery.</p>	<p>Capzimin inhibits RPN11 with effectiveness among several versions of oncology, having activity that diminishes the deubiquitinase activity of RPN11 as a metalloisopeptidase in the lid of the 19S particle, such that inhibition activity prevents segmentation of polyubiquitin linkages from substrates that are to be processed by the proteasome.</p>
	<p>Inhibition of the 26S proteasome by bortezomib results in</p>	<p>Capzimin is effective in resistant oncology, particularly bortezomib resistance, while hindering cellular proliferation as well as causing the unfolded protein response such that inhibition of choline kinase alpha can upregulated CHOP and strongly potentiate apoptosis in this therapeutic context. The literature observes a requirement for more effective therapies in particular instances of pulmonary conditions of atypical proliferation, including PD L1 enabled pembrolizumab, particularly because of what was</p>

	<p>decreased Androgen Receptor Activity along with introduction of apoptosis in Androgen Receptor reliant oncology of the prostate, although inhibition of the Androgen also inhibits proliferation of prostate oncology that is independent of Estrogen Receptor activity. Akt and E3 ligase MDM2, which also promote deterioration of the genomic stability assuring factor P53, both complex with Androgen Receptor, promoting the translocation of Androgen Receptor to the 26S proteasome for proteolytic deterioration.</p>	<p>considered to be complexities in achieving penetration percentages required for therapy, although transduction therapies should be pervasively able to alleviate such complexities.</p> <p>Marizomib (NPI-0052, Salinosporamide A) has general activity inhibiting Proteasome. However, neoplasm, myelomas, lymphomas, leukemias, all have range of susceptibilities, potential for resistance and rescue from resistance produced by Marizomib, Bortezomib, Ixazomib, Carfilzomib, Oprozomib, Delanzomib, including proteasome and immunoproteasome inhibition. YU101 is an epoxyketone derived from Epoxomicin. Oxathiazolones inhibit mycobacterial proteasomes but also inhibit Human B5i and B5 proteasomal loci. YU – 102 inhibits caspase activity sites as does NC – 001. ML604440 is potentially affective to BLi locus.</p>
<p>Immunoproteasome. All cellular entities can express the immunoproteasome when stimulated by interferon-γ(IFNγ) TNFα or TNF alpha. However only immunological cellular entities can express Immunoproteasome constitutively, according to the literature. These provide better insight into immunological disease and inflammatory disease, particularly with regard to how immunological cellular entities and cellular entities affected by T cellular entities, B cellular entities, TNF alpha and Interferon γ or IFNγ. These factors, themselves, as well as affective to peripheral monocytes, tissues and structures, can activate immunoproteasome, enhancing the effect of proteolysis or even affecting the ability for ubiquitylation and sequestrome function. These describe why conditions, disease or pathogens to chronically hyperactivate immunological function result in wasting disease or deterioration of muscle or other tissue, as well as how these mimic the conditions required for atypical cellular proliferation.</p>	<p>LU-005i immunoproteasome selective inhibitor affect to human peripheral blood mononuclear cells (PBMCs) observed through ability to manage dextran sulfate sodium (DSS)-induced colitis model was determined by measuring weight loss and colon length. The result was that LU-005i is the first human immunoproteasome selective inhibitor of all three proteolytically active immunoproteasome subunits, exhibiting the ability to inhibit cytokine secretion from PBMCs and splenocytes, ability to impair differentiation of naïve T helper cells to T helper 17 and ability to</p>	<p>Bortezomib is mistaken for Immunoproteasome inhibition because its toxicity pattern includes inhibitions of a neuronal specific serine protease, such that serine proteases enabled by PEMT's PMME product are not able to reduce all proteins to their most simple structures without intramolecular serine linkages in process useful in pioneering regeneration, repair, regeneration, and optimal developmental programs known as embryonic plasticity. Oprozomib (ONX 0912) has immunoproteasome inhibition activity for subunit B5i, including pathogenic, expansion competent, B Cellular entity proliferation. Lactacystin inhibits proteasome. Z – LLL – VS also inhibits proteasome.</p>

	<p>inhibit DSS enablement of colitis. The studies confirm that exclusive inhibition of LMP7 is not necessary for immunoproteasome inhibition therapeutic effectiveness.</p> <p>Thiasyrbactins (NAM) have affinity for and affective inhibition activity toward the trypsin (B-L, Beta-2) locus of proteasome as well as even more focused effect toward T – L locus of the immunoproteasome.</p>	
<p>Thymoproteasome is considered to be proteasome active specifically in thymus or Cortical Thymic epithelial Cellular entities, such that expression of Subunit B5i shared with immunoproteasome and expression of thymoproteasome specific Subunit B5T both occur in thymoproteasome. B5T generates peptide sequences that promote preferred selection of thymocyte replicas, such that MHC complexes integrated with these and presented in CD8+/CD4+ receptors encompassed by lipid rafts. The presentation of these performsperform as both antigen presentation and monitoring complexes being monitored by immunological cellular entities. The way this functions is presented as being proteasome deterioration of proteins brought to by sequestrome comprised of multiple systems of tagging, aggregation and deterioration at the proteasome, resulting deteriorated proteins becoming proteasomal output products that MHC complexes monitor by attaching to these products, followed by movement of these MHC complexes to the plasma membrane where they are presented in CD4+ and CD8+ receptors encompassed by lipid rafts that are often aggregated in the caveolae. Dilation of the caveolae can become very much essential to efficient completion of some of these immunological activities. Ca2+, nNOS/eNOS function, particularly escaping of iNOS depletion of Ca2+, can be essential in such regard. The exhibition of these B5T sequences prevent thymocytes being culled by Thymus T Cellular entity copy competency assay programs. GMO, synthetic laboratory, laboratory hydrogenated, and other less than optimal molecular structures can become incompletely processed by proteasomes or inhibit proteasome, resultant in MHC presentation of inflammatory, incompletely processed, or</p>		<p>Conjugation of the epoxomicin IlelleThrLeu peptide targeting motif to the vinyl sulphone (Boronic ester) central catalytic loci, produces a potential ability to inhibit the thymoproteasome's specific β5t activity, thereby diminishing the ability of cellular entities to escape pruning by thymic competency assurance programs. Bortezomib inhibits the B5i shared immunoproteasome/thymoproteasome Subunit.</p>

<p>detrimental molecules that promote detrimental changes, while inability to process molecules can cause these to accumulate and integrate into structures, tissues or other molecules. Ag priming, lymphopenic conditions and Interleukin – 4, all generate CD8+ memory lymphocyte phenotype in thymus with increased potential for negative selection because of reactivity to self, which are conditions or outcomes which compete with B5T presentation that signals competent immunological phenotype. However, B5T presentation can obscure incompetent or self-reactive to self lymphocytes, suggesting that genetic anomaly, overproduction of B5T, transport of B5T, thymoproteasome or its products can be factors in atypically proliferation of monocytes, lymphocytes, and conditions such as lymphoma, leukemia and even myeloma.</p>		
<p>Autophagosomes emerge at Endoplasmic Reticulum domains enriched in Phosphatidylinositol synthase</p>	<p>Phosphatidylinositol.</p>	<p>Phosphatidylinositol.</p>
<p>Autophagosomes emerge at Endoplasmic Reticulum domains enriched in CEPT1 Choline/Ethanolamine Kinase Enzyme which produces enriched Phosphoethanolamine before PEMT Catalysis uses its products to produce enriched Phosphatidylcholine and before CEPT1 produces variably optimal configured phosphocholine. CEPT1 selects or prefers substrate with these factors when producing optimal phosphoethanolamine. 1,2-diacyl-sn-glycerol => Phosphatidylethanolamine, 1,2-di-(9Z-octadecenoyl)-sn-glycerol => phosphoethanolamine, 1-hexadecanoyl-2-(9Z-octadecenoyl)-sn-glycerol => phosphoethanolamine, 1,2-di-(9Z-hexadecenoyl)-sn-glycerol => phosphoethanolamine, each are used to produce phosphoethanolamine. Thus, supplementing with DHA, Octadecanoic Acid, Hexadecanoic Acid, glycerol and diacylglycerol all may be particularly beneficial.</p>	<p>Thus, supplementing with DHA, Octadecanoic Acid, Hexadecanoic Acid, glycerol, and diacylglycerol all may be particularly beneficial. DHA inhibits monocyte activation and prevents such activation by palmitic acid and TLRImmuno2.</p>	<p>Thus, supplementing with DHA, Octadecanoic Acid, Hexadecanoic Acid, glycerol, and diacylglycerol all may be particularly beneficial. DHA inhibits monocyte activation and prevents such activation by palmitic acid and TLR2.</p>
<p>Phospholipase D inhibits Macroautophagy but assists in fusion of mitochondria during mitophagy, suggesting that if cause Mitophagy instead of Macroautophagy, enhancing the possibility of deterioration of Mitochondria which are impaired, representing an oncology or pathogenic escape mechanisms, but also assist in mitochondrial fusion which an either cause impairment of stable mitochondria by impaired mitochondria</p>	<p>Phospholipase D inhibits Macroautophagy but assists in fusion of mitochondria during mitophagy, suggesting that if cause Mitophagy instead of Macroautophagy, enhancing the possibility of deterioration of Mitochondria which are impaired, representing</p>	<p>Halopemide, FIPI (5-fluoro-2-indoyly des-chlorohalopemide), triazaspirone, Ethanol inhibits Phospholipase D activity. Inhibitors of Protein Kinase C diminish Phospholipase D activity. N – nonerythroid spectrins such as Fodrin. Inhibition of PEMT results in impaired synthesis of enriched antiinflammatory phosphatidylcholine and PMME, PDME as antihistamine intermediates, but in prostate oncology and other oncology of</p>

an oncology or pathogenic escape mechanisms, but also assist in mitochondrial fusion which can either cause impairment of stable mitochondria by impaired mitochondria. lauroylphosphatidylethanolamine, N – myristoylphosphatidylethanolamine, Products of Humane N – Acylphosphatidylethanolamine Phospholipase D such as N – Acylphosphatidylethanolamines which have uniquely spaced polymers enabling close integration with phospholipids, particularly inhibiting phospholipase D utilization of phosphatidylcholine to free choline and free phosphoric acid lead groups. Fatty Acid N – acylethanolamides are lipid mediators that are enable foundational biological function in all organisms, performing highly conserved biological functions in immunity, stress management, energy balance, G – Protein Agonist activity, Cannabinoid Agonist activity, and nuclear receptor activity such as PPAR – alpha activity. Fatty Acid Ethanolamides are only several enzyme interactions away precursors that exist in interstellar space, and include a group of powerful antihistamines essential to conscious cognitive function and biological function including ethanolamine,

anatomy near the lower digestive pathway, less levels of inhibit PEMT and less levels of homocysteine seem to be required. These contexts are clearly linked to Superoxide, H₂O₂, Peroxynitrite, hypochlorite, trimethylamine-n-oxide and trimethylamine as well as TNF alpha as cascading syndrome resultant of eating meat, chicken, eggs, fish or other phosphatidylcholine dense or Carnitine dense nutritional factors without an antibiotic, prebiotic, postbiotic, macrobiotic or bioactive food/supplement. 3,3 DMB, Olive Oil, Grapeseed oil, Balsamic Vinegar, and other factors can be therapeutic also. Trimethylamine-n-oxide has to be managed because it is among the best indicators of susceptibility to a sudden adverse health event, detrimental behavior or perioperative complications, while also it must be managed to improve carotid intima media plasticity that is linked to these and other disease outcomes. Studies clearly link iNOS as a causal or integral factor in pervasive advanced levels of disease or risk. iNOS upregulation, Phospholipase D, both are required for Androgen enabled PSA increases in prostate disease and atypical proliferation of prostate. However, although Androgen independent prostate oncology exists, this condition is typically at least minimally responsive to Androgen inhibition therapy, suggesting that iNOS and Phospholipase D, as well as PEMT inhibition and Homocysteine continue to be integral foundational nuances of this pathology and almost every pathology that potentially affects vital being. However, a pivotal study observes that inhibition of Phospholipase D enables or causes sensitivity to Ionizing Radiotherapy in oncology of the breast. This pivotal study provides an even more meaningful explanation of the 50 or 60 hz baseline electromagnetic field in civilization produces detriment,

phosphoethanolamine, CTP – Ethanolamine, phosphatidylethanolamine, PEMT function which produces PMME, PDME and phosphatidylcholine. Inhibition of Plantae Phospholipase D by N – Acetyethanolamines suggest Human inhibition also. Inhibition of Phosphotyrosine kinase by any manner and inhibition of Phospholipase D by FIPI results in diminished phospholipase C – γ 1 activity, and Phospholipase C – γ 1 is essential to cellular migration linked to pathology and oncology in particular.

The Best way to understand this stuff is to consider that lipids comprise 87 percent or more of cellular membranes. That's right, cholesterol. If you cholesterol is not being shuttled by Star proteins into important areas like Mitochondria, or is not adequately exhibited in membranes, particularly through catalysis of PEMT1 and PEMT2 to exhibit hydride/hydrogen in ratio of 1 to 2 and to produce antiinflammatory influence or other anatomically essential properties, then the complete foundation for physiology starts changing toward control by the environment, systems, cues to finding adequate nutrients, and

comprising iNOS expression in pervasive aspects of endothelium which depletes calcium and increases susceptibility to Superoxide cascade known as uncoupling of nitric oxide synthases, causing a swelling of pervasive endothelial pathways and airway epithelium in similar regard, while also explaining why a hallmark of revealing indicators for prostate oncology includes luminal expression of Phospholipase D. This luminal expression of Phospholipase D and epithelial expression of iNOS, explains why prostate disease and increased PSA levels are considered uniquely increased in incidence among males populations of the wester world. Honokiol, derived from Magnolia Tree group of Plantae inhibits Phospholipase D experimentally. Rapamycin inhibits Proteolysis typically exhibited in pervasive oncology, including atypical proliferation of prostate tissues. Information [PMCID PMC5785744](#). Information [10.1038/emm.2013.75](#). Phospholipase D is another example of the immunological system becoming transformed to be utilized to sustain biological system resiliency because of unmanaged, nonephemeral detrimental influence that potentiates massive apoptosis and deterioration of physiology. iNOS similarly becomes utilized to increase the turgor of cellular entities, strengthening the ability of cellular entities to maintain physiology resultant of changes in gravity, massive apoptosis from choline deficiency linked to PEMT inhibition and homocysteine upregulation. Phospholipase D is important because it buds off aspects of membranes to produce vacuoles through which viruses or microbes can escape the toxic interstitial plasma membrane space and which can increase the physical structures required for Ionized particle must traverse before encountering an atom and displacing an electron, proton or

	<p>commandeering of these to the priorities of systems, other interests, individuals, objectives, quotas and revenue goals, other organism's influence, the biome or other factors.</p>	<p>neutron which constitutes impairment produced by Ionizing radiation. Radiation can also cause thermodynamic impairment, but certainly iNOS expression, DNA impairment, and other changes can occur without displacing atomic structure. iNOS is expressed by Astronauts returning to earth resultant of gravitational disparities and a requirement of assistance increasing turgor of cellular entities to sustain physiology and movement. Phospholipase is involved in producing clathrin coated vesicles, endocrine secretion vesicles, free phosphatidic Acid and free choline lead groups, and can be activated by Sphingosine-1-phosphate to regulated Interleukin – 8 or other factors, thereby is involved in canonical inflammation cascade produced by PEMT inhibition and homocysteine upregulation. Information PMID 12039947. Information 10.1074/jbc.M111078200. https://www.nature.com/articles/em201375.</p>
Assurance of optimal mitochondrial steroidogenesis.	<p>Providing StARD7, preferably conjugated to enriched versions of phosphatidylcholine and assuring that cholesterol pocket is filled, may enable recovery of impaired mitochondria and impaired mitophagy. Providing pregnenolone and DHEA, also supplies molecules in this hormone assurance pathway. These hormone stabilizers are contraindicated only in one or a few studies in which therapeutics otherwise were also being utilized and these studies did not include StARD7. Pregnenolone and DHEA stabilize mitochondrial cycling and can cause cellular resiliency during</p>	<p>Urolithin A derived by microflora stimulates Mitophagy as well as activates and enhances Mitophagy or mitochondrial recycling. Other Urolithin A products produces and services exist.</p>

	<p>cytotoxic therapy in which it is in the intent to cause apoptosis or cellular deterioration to occur, requiring that it be carefully assured that use of Pregnenolone and DHEA, as well as Astaxanthin and Spirulina or antioxidants generally occurs when cytotoxic therapies are not being used although these factors can certainly rescue the collateral toxicity of cytotoxic therapies. Pomegranate Juice, strengthened, as well as products such as Mitopure by Timeline Nutrition, activates and enhances Mitophagy or mitochondrial recycling. Other Urolithin A produces and services exist. Other Mitochondrial therapies include Alpha Lipoic Acid, CoQ10, Carnitine, Citrulline, complete B Vitamins, Vitamin C, Vitamin K2, Vitamin K3, Vitamin E and Folic Acid, but these have not been presented as being selective in enhancing mitophagy. The literature variably asserts a role for STARD2 and STARD7 both in transport of phosphatidylcholine while STARD1 and STARD7 are generally considered to deliver phosphatidylcholine or cholesterol to the Mitochondria.</p>	
Hydrogen Assurance	Molecular Hydrogen as Magnesium 80 mg tablets, the product H2 by Dr. Mercola.	
Stem Cellular entity synthesis enhancement and resilience assurance.	Visiclear is one among other produces in this supplement category.	Suppressing the enzyme Dicer can enabled Muller Glial stem cellular entities to become uninhibited and

	<p>Spirulina, Astaxanthin and mix of nutraceuticals such as the follow. blueberry extract, carnosine, green tea extract and vitamin D3. Bilberry, Lycopene, Horsetail, Lutein, Vitamins, Selenium, Alpha Lipoic Acid, Zeaxanthin Isomers, Carnosine, Carnitine, and lycopene. This pathway can require many months of therapy. Crocus sativus L or saffron and its derivative crocin, are therapeutically active enhancers of neural stem cellular development and can be a substitute for Platelet derived growth factor AA, PDGF AA. This information is generally applicable, but also specifically applicable to the regeneration of vision tissues.</p>	<p>become expressed in way that enhances regeneration of vision structure and function. This information is generally applicable, but also specifically applicable to the regeneration of vision tissues.</p>
<p>Macroautophagy, the metabolic capability and pathogenically elusive large scale recycling and cleaning capability that is escaped by pathogenic proteolysis and escaped by directing autophagy directly to only mitophagy by phospholipase D.</p>	<p>Scarcity of energy, nutrients, and growth factors, as well as cellular stress are activators of Autophagy. Rapamycin or Sirolimus, Metformin (1,1 dimethylbiguanide), trehalose (d trehalose) and Resveratrol are activators of Macroautophagy. Also, Vitamin K2 Menaquinone, Vitamin D as well as EB 1089, and Caffeine stimulate Autophagy. Oppositely, Amentoflavone, Apigenin, Fisetin, Hesperetin, Luteolin aglycone/unglycosylated , and quercetin inhibit Macroautophagy. Ceramide, which is produced by pathways including</p>	<p>Torin 1, Lithium carbonate (di Lithium carbonate), Carbamazepine (5H dibenzo[b,f]azepine 5 carboxamide0, Sodium valproate (sodium 2 propylpenanoate).</p>

	<p>nSMase/aSMase, strongly potentiates autophagy suggesting that nSMase/aSMase produce phosphocholine to enhance survival signaling in parallel to ceramide production. Inhibition of nSMase/aSMase as well as Sphingosine Kinase which utilizes Ceramide to produce S1P for survival signaling, perform as inhibitors of Autophagy. C2 and C6 Ceramide are typically processed into longer Ceramides by Ceramide Synthase to initiate autophagy enabled by Ceramide.</p>	
<p>Renal regeneration. Diabetic pathology is typically regarded as being autoimmune, thereby having causality in diminished immune function linked with PEMT dysfunction and homocysteine, while type II diabetes is often considered to be gradual or linked to other causality. However, the compendium of research linked to this list observes that inhibition of PEMT causes expression of P53 which then inhibits glycolysis, pentose phosphate pathway, glut endocytosis of sugar, causing gluconeogenic sugar to be trapped in glycogen cycling and causing extracellular sugar to accumulate in circulatory pathways, thereby overworking Islet Beta cellular entities and causing oxidative distress that causes apoptosis as well as causing Islet Beta Cellular entities to differentiate into other cellular versions. The literature and popular consumer health resources suggests that diabetes type II is results from an accumulation of ceramides in cellular entities that causes cellular lysis then empties electrolyte, inflammation factors, ceramides and fat into circulation which then impairs pancreatic catalysis, structure and function. It is most likely that the Ceramide increases and lysis which results in emptying of adipose material are the result of glucose trapped in glycogen cycling to produce a adiposity phenotype along with the affects of PEMT inhibition which increases homocysteine in a way that increase Bax and potentiate massive apoptosis of among cellular entities.</p>	<p>Kidney stuff by Golden Standards is naturopathic manner of rescuing impaired renal tissues. Banaba Leaf which includes corosolic acid from Lagerstroemia speciosa, Yarrow flower Achillea millefolium Linn. At least 1 instance of Corosolic upregulation of Lactic Acid in diabetic pathology has been observed, clinicians should be aware of this risk. , Curcumin, Berberine, licorice or glycyrrhizin uralensis which is triterpenoid Saponin, nettle, stinging nettle, white mulberry which exhibits protection from nephrotoxicity from cisplatin therapy(pyroglutamante, O B D glucoside, quercetin, kaempferol, Rutinoside, Rutin, 2 phenylethyl D rutinoside), all are can assist in stabilizing</p>	<p>154 percent increase in curcumin availability was observed in a study at about 2 hours after administration while in the first half hour piperine/chavacine together or piperine results in 2000 percent increase in absorption of curcumin. Curcumin formulations as curcumin phytosome formulation was 790 percent higher than curcumin, while mix of curcumin volatile oils increase availability by 130 percent, although formulation of curcumin with hydrophilic carrier, cellulosic derivatives and natural antioxidants resulted in a 4590 percent increased absorption of curcumin. IGF 1 growth factor is known to enable regeneration of Islet B cellular entities which are primary producers of insulin.</p>

	<p>diabetic pathology according to studies. 154 percent increase in curcumin availability was observed in a study at about 2 hours after administration while in the first half hour piperine/chavacine together or piperine results in 2000 percent increase in absorption of curcumin. Curcumin formulations as curcumin phytosome formulation was 790 percent higher than curcumin, while mix of curcumin volatile oils increase availability by 130 percent, although formulation of curcumin with hydrophilic carrier, cellulosic derivatives and natural antioxidants resulted in a 4590 percent increased absorption of curcumin. Phosphatidylethanolamine is downregulated in some studies of renal pathology but is sequestered by some pathogenic factors, although Ophiobolin A sequestration of Phosphatidylethanolamine helps control and alleviate oncology by inducing autophagy and activating mitochondrial apoptosis pathways in oncology as it destabilizes oncology cellular membranes.</p>	
<p>Hepatic Regeneration. Growth factor EGFR and one carbon factor C MET both participate in hepatic regeneration with TGF Beta abated hepatic tissue proliferation but PEMT1 functions similarly to EGFR while PEMT2 often perform the role of TGF Beta also in such regard.</p>	<p>Source for regeneration of hepatic tissue present Artichoke extract, White Beans, Turmeric/Curcumin, Beets, Dandelion, Milk Thistle the classic hepatic detoxification factor, L Cysteine, Alfalfa, and Ginger as being a group of hepatic</p>	<p>154 percent increase in curcumin availability was observed in a study at about 2 hours after administration while in the first half hour piperine/chavacine together or piperine results in 2000 percent increase in absorption of curcumin. Curcumin formulations as curcumin phytosome formulation was 790 percent higher than curcumin, while</p>

regeneration factors. 154 percent increase in curcumin availability was observed in a study at about 2 hours after administration while in the first half hour piperine/chavacine together or piperine results in 2000 percent increase in absorption of curcumin. Curcumin formulations as curcumin phytosome formulation was 790 percent higher than curcumin, while mix of curcumin volatile oils increase availability by 130 percent, although formulation of curcumin with hydrophilic carrier, cellulosic derivatives and natural antioxidants resulted in a 4590 percent increased absorption of curcumin. LiverhealthFormula by purehealth research exhibits many of these hepatic regeneration factors. Obtaining and supplementing with whole organism glandular that includes hepatic tissue or which is comprised of hepatic tissue, is known to assist in repairing and regenerating tissues and matrix structure of tissues such as the hepatic organ. Selenium, Zinc and Tocotrienols ay also assist in hepatic regeneration. The full complement of the essential pathway Phosphatidylethanolamine, phosphatidylcholine, Methyltetrahydrofolate, Folate, Betaine, B12, B6, 6s 6578 Tetrahydrofolate, glutathione, Cysteine,

mix of curcumin volatile oils increase availability by 130 percent, although formulation of curcumin with hydrophilic carrier, cellulosic derivatives and natural antioxidants resulted in a 4590 percent increased absorption of curcumin.

	<p>Cystathionine, Selenium, methylsulfonylmethane, S-methylmethionine sulfonium, Zinc, CA2+, phosphatidylserine, phosphatidylinositol, Curcumin with piperine and berberine all provide strong protective and regenerative influence to hepatic tissues.</p>	
<p>IGF-1 Estrogen Receptor Alpha positivity and IRS1 Estrogen Receptor Alpha Negativity proliferation programs including IRS-1 accompaniment by 16 gene Serine324 phosphorylators of Progesterone Receptor along with PI3K/Akt/GSK3. Inhibiting IGF-1 can disrupt the inherent cascade of pathogenic potential resultant of upregulated Estrogen receptor alpha because it is IGF-1 integration with complexes of Estrogen Receptor Alpha and AP1 within PS2/TFF1 promoter regions that causes cyclic amplification of AP1 which is an inhibitor of PEMT catalytic transactivation.</p>	<p>Chronic Fatty Acid Supplementation can inhibit IRS1. Preventing exposure to electricity fields, wireless communication, atmospheric particulate, environmental particulate, extreme gravitational changes, viruses, bacteria and choline deficiency, all can prevent iNOS expression, as well as iNOS inhibitors such as Curcumin or others, can prevent or diminish PI3K, Akt Signaling. Management of GSK3 using natural or other factors presented in in this document can also provider benefit in disrupting this cascade.</p>	<p>IGF-1 and IRS-1 both can be inhibited by Olanzapine. Inhibitors of IRS-1, PI3K, Akt, and GSK3 can disrupt these pathogenic cascading conditions.</p>
<p>Sphingosine Kinase activates Akt and GSK3 among its activation cascade which includes S1P receptors, GTPase activation, ATPase activation, G protein activation, Akt enablement and GSK activation. Hypoxia Inducible Factor 1 alpha is activated by Akt and GSK3 in a way that removes the requirement for hypoxia to be exhibited and which enables persistent upregulation of Hypoxia Inducible Factor 1 alpha pathways to become activated, including activation of erythropoiesis which explains atypical proliferation of erythropoiesis and conditions of atypical proliferation of circulatory fluids. S1P is known to be active in atypical proliferation of vascular tissue, hepatic tissue, as well as smooth muscle cellular entities. The expression of VEGF is also induced by VEGF, while version S1P3 is known to induce</p>	<p>S1P3 is known to cause migration which can be inhibited to depotentiate expansion of pathology or enhance regenerative repair while SIP2 is known to inhibit expansion of pathology. NHOBTD inhibits expanding pathology from S1P including preventing VEGF activation by Hypoxia Inducible Factor as well as preventing Hypoxia Inducible Factor 1 expression.</p>	

migration of cellular entities while S1P2 is known to inhibit migration, although the S1P derivative NHOBTD inhibits Hypoxia Inducible Factor increases from S1P and prevents VEGF synthesis enabled by Hypoxia Inducible Factor 1.		
<p>Mcl-1 is a major resistance factor in therapies which endeavor to induce cytotoxicity at the cellular level for therapeutic benefit. BAG-3 is known to inhibit cytotoxicity by upregulated proteolysis in a way that enables atypical proliferation of cellular entities affected by pathology. BAG-3 enhancement of proteolysis protects Mcl-1 from deterioration, thereby providing a focused enhancement of Mcl-1 activity in sustaining resistance to cytotoxic therapeutics and evading inherent or endogenous pathways that would cause apoptosis and deterioration of cellular entities. Mcl-1 inhibits outer mitochondrial membrane permeabilization. Mcl-1 also integrates with Bcl-2 Bag factors to direct cellular phenotype away from apoptosis pathways. BLC – 2 proapoptotic and antiapoptotic effector variants each can exhibit the idiosyncratic BCL-2 domains 1, 2, 3 and 4. Some variants exhibit BH3 only, and are known as BH3 only variants, except for BID that is included among BH3 only variants and which exhibits all four of the BH domains. Antiapoptotic variants prevent the activation of BAX and BAK. BH3 only proteins are grouped into Activators and Sensitizers with Activators activating BAX and BAK and inhibiting antiapoptotic proteins. Sensitizers suppress antiapoptotic proteins and do not necessarily upregulated BAX or BAK. Anti-apoptotic proteins include A1, BCL-2, BCL-W, BCL-XL, and MCL-1 while having the BH4, then BH3, then BH1, followed by BH2 and then TM domains. Proapoptotic proteins include BAX and BAK while having the same order of BH domains, BH4, BH3, BH1, BH2 and TM. BH3-only proteins include Activators BID, BIM and PUMA, while BH3-only Sensitizers include BAD, BLK, BMF, HRK and NOXA, such that these exhibit BH3 only domains except for BID which has all 4 BH domains.</p>	<p>Cantharidin inhibits Bag 3. Bag3 is upregulated by Phenethyl Isothiocyanate and 2'-Hydroxycinnamaldehyde. Upregulators of Bag3 include Lipopolysaccharide, Acetaminophen, Motexofin gadolinium, Zinc Acetate, and Valporic Acid. Atmospheric particulate from transportation increases Bag3.</p> <p>Downregulators of Bag3 include, Staurosporine.</p> <p>Pancreastatin from the bulb of Hymenocallis littoralis transactivates Bax.</p> <p>Among sensitizers, BIK inhibits A1, BCL-2, BCL-W and BCL-xL. BAD inhibits BCL-2, BCL-W, and BCL-xL. BMF inhibits BCL-2, BCL-w, BCL-xL. HRK inhibits BCL-xL. NOXA inhibits MCL-1.</p> <p>Among Activators BID, BIM and PUMA each inhibit A1, BCL-2, BCL-W, BCL-xL, MCL-1. When A1, BCL-2, BCL-W, BCL-xL and MCL-1 are inhibited it prevents these proteins from performing their defacto activation of BAX and their activation of BAK.</p>	<p>Bag3 Immunoglobulin is available for Anti Bag3 therapy.</p> <p>Upregulators of bag 3 include 3 (4 methylphenylsulfonyl) 2 propenenitrile. Anthra (1,9 cd)pyrazol-6(2H) one. (6 (4 (2 piperidin 1 ylethoxy)phenyl)) 3 pyridin 4 ylpyrazolo(1,5 a)pyrimidine. 4 (5 benzo(1,3)dioxol 5 yl 4 pyridin 2 yl 1H imidazol 2 yl)benzamide.</p> <p>Downregulators of Bag3 include ABT 737, , Elesclomol, Vinclozolin, Diethylnitrosamine, Dactinomycine, 3 (4 methylphenylsulfonyl) 2 propenenitrile, Estrogen, Anthra(1,9 cd)pyrazol 6 (2H) one.</p> <p>Deubiquitinase USP13 is required for proliferation of HPV viral positive cervical oncology cellular entities and tissue, and USP13 promotes Mcl1 stabilization thereby promoting the antiapoptotic activity of Mcl1 to sustain HPV viral positive cervical oncology tissue. Inhibiting the deubiquitinate USP13 or inhibiting MCL1 or both can destabilize HPV enabled cervical oncology in particular. Information. Oncogene (oncology genetics). Volume 40. Pages 2112 to 2129. 2021. PMID 33627786. Spautin1 inhibits USP13 and small molecule analogues of Spautin1 are able to traverse the barrier between circulatory pathways and the brain. Information. Metabolites. Volume 11. Number 9. Pages 622. September 2021. PMID 34564439. Inhibiting USP13 deteriorates toxic lewy entity clumps found in Parkinson's disease. Information. "Inhibiting USP13 'deteriorates' toxic clumps seen in Parkinsons disease." News. DrugTargetREview.com November 18, 2018.</p>

		<p>USP13 inhibition can be accompanied by ABT 263 antagonist of BCL and BCLXL to inhibit antiapoptotic signaling in a general context. Inhibiting these three factors produces a generally inhibition of prosurvival antiapoptotic cellular signals. Information. Nat Commun. Volume 9. Pages 215. 2018. PMID 29335437. Curcumin inhibits deubiquitinases generally. AC17 analog of curcumin inhibits USP14 and UCHL5. Betulinic acid inhibits broad spectrum deubiquitinase activity. Gambogic acid causes accumulation of polyubiquitin. Through alpha, beta unsaturated ketones similarly to curcumin. Information. Pharmacology and Therapeutics. Volume 147. Pages 32 to 54. 3rd Month, 2015.</p> <p>bAP15 prevents deubiquitylation activity performed by USP14 and UCHL5. bAP15 and its piperidine ring differs from the deubiquitinase inhibitor VX1570 and its azepane ring. Curcumin D inhibits USP13. A useful number of USP deubiquitinase inhibitors are located in this study. Information. Int J Mol Sci. Volume 22. Number 9. Page 4546. 2021.</p>
<p>CRISPR GENE Therapy with transduction Domains for assured access to every cellular entity in physiology. This capability enables repair of genes representing escape mechanisms preventing cellular deterioration or escape mechanisms causing cellular deterioration. Similarly, this capability enables introduction of cellular senescence or preventing additional cellular division. These capabilities can be introduced by ascertaining specific molecules, genes, genetic sequences, impairments, structures or conditions in only those cellular entities require intervention, followed by introducing changes to those cellular entities. Essentially, these are pervasively constitutive of abatement of what is known as disease. The epidemiological patterns of 2020 and 2021 reveal how rapid these existing capabilities should have been enabled to come into practice and application. Many of these capabilities have utilized to experimentally eradicate viral, microbial and other disease already. Much of the technologies</p>	<p>CRISPR Perfect Gene Repair. Protein Transduction Therapy. Eradication of oncology affected cellular entities. Deactivation of stem cellular entities or other cellular entities affected by disease. Repair of genes that have become impaired or were impaired to cause both susceptibility and proximately cause escape into disease. Synthesis of complex proteomics with nutrients and therapies that ask cellular entities questions or assay cellular entities and then deliver specific</p>	<p>CRISPR Perfect Gene Repair. Protein Transduction Therapy. Eradication of oncology affected cellular entities. Deactivation of stem cellular entities or other cellular entities affected by disease. Repair of genes that have become impaired or were impaired to cause both susceptibility and proximately cause escape into disease. Synthesis of complex proteomics with nutrients and therapies that ask cellular entities questions or assay cellular entities and then deliver specific</p>

<p>have now been exhibited for nearly 1 decade, several decades or even earlier.</p>	<p>therapeutics required for any disease, any genetic impairment, or which walk the cellular entities along developmental pathways to become reprogrammed or developed into cellular entities, structures and tissues required for sustained or reconstituted vital being.</p>	<p>tissues required for sustained or reconstituted vital being.</p>
<p>Deubiquitinase inhibition of the Proteasomal sequestrome.</p> <p>Ubiquitylation activity, when exhibited in multiplicity, completes a linkage of ubiquitylation factors that performs as a signal for translocation to the 26s Proteasome for deterioration by the units and subparticles of 26S proteasome.</p> <p>Deubiquitinases are pervasively active in oncology, lymphoma, leukemia, autoimmune, Fanconi anemia and numerous other conditions.</p> <p>Deubiquitinases rescue molecules and enzymes from being deteriorated by proteasomal activity, resulting in accumulation of rescued molecules, proteins and enzymes comparatively to rapid and pervasive deterioration of other molecules, causing remarkable and deterministic changes in the balances of particular molecules that determine cellular status, progression, survival, apoptosis, migration, inflammation, etc.</p> <p>Systems models of cellular decisions, outcomes and activity pervasively exhibit persistent activation of molecules or complex as well as receptors that potentiate an outcome or change along competing processes that opposes that change such as deactivation, complex separation, proteolysis of complexes, or autophagy of activated structures or activated complexes.</p> <p>Proteolysis can causally and substantially changes these balances using ubiquitylation while deubiquitylation can selectively cause rapid, complete and substantially deterministic influence to cellular outcomes. Ubiquitylation can prime or activate some enzymes, such that ubiquitylation can be required to occur before an enzymes catalytic activity within a molecular pathway can be activated, before an enzyme can activate a molecular pathway or in conclusion of such pathway as well as possibly in deactivation or prevention of particular pathway.</p>	<p>Galeterone inhibits Androgen Receptor but its effect is mitigated when MDM2 or CHIP are also inhibited. Galeterone may be interestingly different from some other therapies because it seems to function by inhibiting deubiquitinase. Androgen Receptor reliant and Androgen Receptor Independent prostate oncology benefits substantially and minimally, respectively, from inhibition of Androgen Receptor.</p> <p>USP7 is inhibited by succinimide motifs producing dual hydrogen adhesion to the allosteric pocket of USP7, redesigned benzofuran amide scaffolds with simplified ether series inhibitors, as well as producing acyclic conformation control using amine placement, particularly ether linked amines with carbon linked morpholines motivated by free energy perturbation calculations.</p> <p>Information, 10.1021/acs.jmedchem.0c00245.</p>	<p>. Inhibition of USP12 and inhibition of USP46, result in enhanced Androgen Receptor deterioration. Experimental upregulation of USP7 promotes MCL1 enablement of stem cellular pathogenic transition toward atypically proliferation status. USP9 and USP13 stabilize MCL1 and the duration of MCL1 exhibition without proteolytic deterioration is correlative to risk of pathogenic transition, resilience and pathology in ovarian and pulmonary examples of atypical cellular proliferation. Ubiquitinases, including USP9 and USP13, when inhibited have a strong potential to inhibit atypical proliferation, open the door to diverse therapeutics through enhanced sensitivity, as well as directly therapeutically benefit diverse version of neoplasm. BH3 Mimetic Inhibitors are particularly enabled by USP13 inhibition. USP1 is inhibited by Pimozide, SJB2-043, and GW7647 while Pimozide inhibited ID1, effectively diminished leukemia in assay and also are linked to improvement in diverse oncology. USP7 inhibitors USP7-443, USP7-866, USP7-797, USP7-055, USP7-414, USP7-877, all were active in a diverse array of conditions benefiting from impaired P53 function including escape mechanisms enabling aerobic glycolysis. DC – U43 inhibits colony formation by H1975 cellular entities, suggesting effectiveness in inhibiting USP8 and its ability to sustain effects of EGFR and its linked pathways. Inhibition of USP8</p>

The best way to consider ubiquitinase activity is to consider that it rescues a molecule from progression toward participation in other pathways and most typically rescues progression toward deterioration or proteolysis by proteasome, immunoproteasome or thymoproteasome. The rescuing activity produces an upregulation in duration and volume (bioavailability) of a molecule thereby comparatively increasing such rescued molecule compared to molecule which compete with the rescued molecule for deterministic influence, receptor activation/deactivation. The result is that, in typical conditions of inhibited PEMT and upregulated homocysteine which increase BAX, increase choline kinase alpha/aSMase/nSMase upregulation of phosphocholine, thereby increasing the intensity of proteolysis, deubiquitinase can assert fairly intense deterministic influence to cellular status, proliferation, differentiation, survival and apoptosis.

ALG13, USP7, USP32, JOSD1, OTULINL, USP8, USP33, JOSD2, OTUB1, USP9X, USP34, UCHL1, OTUB2, USP9Y, USP35, BAP1, OTUD1, USP10, USP36, UCHL3, YOD1, USP11, USP37, UCHL5, OTUD3, USP12, USP38, BRCC3, OTUD4, USP13, USP39, COPS5, OTUD5, USP14, USP40, COPS6, OTUD6A, USP15, USP41, EIF3F, OTUD6B, USP16, USP42, EIF3H, OTUD7A, USP17L2, USP43, MPND, OTUD7B, USP18, USP44, MYSM1, TNFAIP3, USP19, USP45, PRPF8, OTULIN, USP20, USP46, PSMD7, VCIPI1, USP21, USP47, PSMD14, ZRANB1, USP22, USP48, STAMBP, USPL1, USP24, USP49, STAMBPL1, CYLD, USP25, USP50, MINDY1, USP1, USP26, USP51, MINDY2, USP2, USP27X, PAN2, MINDY3, USP3, USP28, USP53, MINDY4, USP4, USP29, USP54, MINDY4B, USP5, USP30, ATXN3, ZUP1, USP6, USP31, ATXN3L, SAGA

ALG13	USP7	USP32	JOSD1
OTULINL	USP8	USP33	JOSD2
OTUB1	USP9X	USP34	UCHL1
OTUB2	USP9Y	USP35	BAP1
OTUD1	USP10	USP36	UCHL3
YOD1	USP11	USP37	UCHL5
OTUD3	USP12	USP38	BRCC3

The deubiquitinase SAGA promotes class switch recombination genomic repair and DNA repair. USP9x can be essential to dendrite spine development and cortical dendrite density. The deubiquitinase YOD1 stimulates YAP/TAZ which regulate or control proliferation of cellular entities and size of organs, particularly being coactivated by LATS and particularly stabilizing ITCH to perform such control. These can powerful factors in control neoplasm and atypical proliferation. The deubiquitinate USP14 promotes oncology by rescuing Androgen Receptor from deterioration by the 26s Proteasome, although inhibition of 26s proteasome result in deterioration of prostate oncology cellular entities, again, minimally, and substantially, but certainly such cellular entities escaping 26S Proteasome inhibition exhibit some manner of resistant mechanisms. Androgen receptor transcriptional activity relies upon the 26s Proteasome's activation.

USP14 rescues Androgen Receptor from proteolytic deterioration. inhibition of USP10, USP12, USP14, USP26 and USP7 all are linked with promotion of Androgen Receptor to the 26S

suppresses non small cellular lung oncology that is resistant to Gefitinib. Inhibition of USP2 eliminates triple negative oncology of breast stem cellular reservoirs, inhibits BMIL, inhibits TWIST and downregulates epithelial to mesenchymal transition, all be resuming β -TrCP ubiquitylation of TWIST, such that Triple Negative oncology of breast also becomes much more sensitive to molecular therapeutics. USP1 deregulation, including upregulation by its cofactor UAF1, enables DNA impairment tolerance, correlated with Fanconi anemia, and is linked to pathogenic neoplasm, sarcoma, melanoma, gastric oncology, and atypical proliferation of the cervix, such that inhibition of USP1 can ameliorate much of these pathologies. USP2 and USP2a are linked to pathogenic oncology of prostate, as well as a diverse array of neoplasm and oncology including oral squamous cellular carcinoma, bladder oncology, particularly through upregulating fatty acid synthase, MDM2, MDMX, FASN, Aurora A, Cyclin A1, and cellular cycle activation enzymes. USP7 or HAUSP is linked causally to prostate oncology, multiple myeloma, ovarian oncology, and diverse other nuances of oncology including non small cellular pulmonary oncology, epithelial ovarian oncology, glioma, and is expressed in correlation with more detrimental prognosis, neoplasm size, neoplasm infiltration activity, and increased risk, all occurring through its regulation of P53, MDM2, PTEN and FOXO which are remarkable because together these potentiate exhibition of aerobic glycolysis, downregulation of PINK1, and exhibition of the pioneering original anatomical development factor FOXO, thereby removing genetic stability mechanisms, impairing apoptosis pathways and promoting developing differentiation in what may be a typical post gestational context of

OTUD4	USP13	USP39	COPS5
OTUD5	USP14	USP40	COPS6
OTUD6A	USP15	USP41	EIF3F
OTUD6B	USP16	USP42	EIF3H
OTUD7A	USP17L 2	USP43	MPND
OTUD7B	USP18	USP44	MYSM1
TNFAIP3	USP19	USP45	PRPF8
OTULIN	USP20	USP46	PSMD7
VCPIP1	USP21	USP47	PSMD14
ZRANB1	USP22	USP48	STAMB P
USPL1	USP24	USP49	STAMB PL1
CYLD	USP25	USP50	MINDY 1
USP1	USP26	USP51	MINDY 2
USP2	USP27X	PAN2	MINDY 3
USP3	USP28	USP53	MINDY 4
USP4	USP29	USP54	MINDY 4B
USP5	USP30	ATXN3	ZUP1
USP6	USP31	ATXN3 L	SAGA

Consult the literature and genomic data for more deubiquitinases. Ubiquitinases include these factors at this instance. ZUFSP family function as cysteine proteases, ovarian tumour proteases (OTUs), ubiquitin C-terminal hydrolases (UCHs), ubiquitin-specific proteases (USPs), the Machado-Josephin domain superfamily (MJD), the MINDY family, as well as JAB1/MPN/MOV34 metalloenzymes (JAMMs) are zinc-dependent metalloproteases.

proteasome for deterioration, while USP7 seems to specifically enable translocation of Androgen Receptor to glucocorticoid receptors in DNA and Androgen Receptor response elements which are essential to enabling changes which Androgen Receptors causes to metabolism.

USP7 has been coprecipitated or encountered with the Androgen Receptor within complexes attached to the Androgen Response Elements FKBP5, PSA and PDE9A, and is considered essential for Androgen Receptor response element activation within DNA.

USP10 inhibition experimentally impaired response of neoplasm to Androgen Receptor, and prevention expansion of neoplasm pathology. USP10 amplifies the Androgen Receptor activation potential. USP12 stabilizes androgen receptor to enable its transcriptional activity required for PSA upregulation. USP26 is a deubiquitinase and counteracts ubiquitylation of Androgen Receptor.

HBX - 19818 is a USP7 inhibitor. Inhibition of USP7 and PLK1 are catastrophically affective to small cellular oncology of pulmonary tissues that is

inhibited PEMT1, as well isolated and inhibited PEMT2. FOX factors enable original anatomical development, regeneration, repair, language acquisition and other essential developmental patterns while FOXO is rapidly deteriorated in the presence of growth factors but suppresses oncology and stabilizes conditions during stress, such that FOXO is experimentally linked with expanded or indefinite span of being. Ubiquitylation of RIG-I at Lysine-63 is essential to activate RIG-I and enable RIG-I to stimulate type I interferon pathway, such that USP3 deubiquitylation prevents this capability, thereby affecting interferon availability and presenting how USP3 can inhibit the RIG-I pathway that selectively causes apoptosis of cellular entities with latent viral vectors such as HIV. SARS virus linked to epidemiological patterns of 2020 and 2021 utilizes ORF9b to inhibit RIG-I-MAVS antiviral signaling by interrupting K63linked ubiquitylation of NEMO, although literature observes that RIG-I directly inhibits polymerase mediation of first phase of SARS genome translation after ascertaining that the SARS genome is an RNA virus by inspecting the 3' extremity. These suggest that providing all trans retinoic acid to assure RIG-I protein expression along with inhibition of USP3 might enable activation of highly conserved innate cellular immunity mechanisms that prevents SARS viral condition progression. Information 10.1038/s41590-021-00942-0 .

Heparin and Heparin Sulfate inhibit Agrin enabled acetylcholine receptor focal aggregation. The same study found that typically the effects of Agrin are reversible.

Triptchloride or Triptolide is an inhibitor of NF – kB. NFkB, SP1 and NFY, are activators of SND1. Information. Biochimie.

<p>resistant to paclitaxel. Spautin – 1 inhibits USP10 and inhibition of USP10 causes deterioration of atypical proliferating cellular entities, doing so by causing deterioration of AMPK, SYP and FLT3 among other factors. USP10 can be complementary to Crenolanib. Midostaurin or HBX19818 inhibit USP10. USP8 inhibition causes deterioration and impaired migration of HER2 positive gastric cellular proliferation, indicating that USP8 stabilizes HER2. RA9 inhibits USP8 and causes retreat of corticotroph cellular neoplasm with wild type USP8 and genetically impaired USP8 by differentially upregulating P27 compared to treated with pasireotide. Inhibition of USP2 by Z93 causes inhibition of SARS Papain Like Proteases. Inhibition of USP2 causes colorectal oncology proliferation to abate, also causing similar outcomes in mantle cellular lymphoma by enabling rapid deterioration of Cyclin D1. USP9X can inhibit or promote oncology, rescuing MCL1 to promote resistance to therapies and overly resilient stem cellular entities, enhancing multiple myeloma pathology, stabilizing CEP131 to potentiate oncology of breast tissue by enabling centrosome biogenesis regulation, also</p>	<p>Volume 95. Issue 4. April, 2013. Pages 735 to 742. 2012. Major enablers of pathogenic transition to mesenchymal cellular phenotype and pathogenic migration of tissue to distal locations include HaRas or Ha Ras, PI3K/AKT, ERK, Wnt/β-catenin, MTDH, SND1, NF-κB, c-Myc and FOXO1/FOXO3a. These, particularly SND1, are linked to diverse oncology. Information. Molecular Basis of Disease. Volume 286. Issue 22. Page 19982 to 19992. 2011. Int J Oncology. Volume 46. Number 2. Pages 265 to 473. 2015. PMC42772250. NFKB includes SP1 in its transcriptional activation pathways although other pathways also include SP1 in their transcriptional pathways. Upregulation of SP1 is linked, causally, with detrimental prognosis in triple negative oncology of the breast tissues, particularly those being managed by doxorubicin. Information. Scientific Reports. Volume 6. Article Number 31804. 2016. CRISPR removal or changing of the SP1 and NFKB loci in the HIV LTR sequences can may be useful to abrogate exhibition of viral activity and pathology. Information. Sci Rep. Volume 6. Pages 34532. 2016. SP1 inhibition by phloretin enables its suppression of Prostate Oncology. SP1 inhibition is accompanied by SP1 deterioration that occurs through SP1 inhibition of GSK3B, which suppresses the nucleolin enhancement of SP1 mRNA translation, promoting transcriptional inactivation of SP1. Interestingly, inhibition of SP1 results in inhibition of nucleolin, Sp3/4, survivin, VEGF. SP1 inhibition also upregulates apoptosis potentiating proteins as well as downregulates cellular cycle proteins. Phloretin, correlative, produces cellular growth inhibition along with causing apoptosis in PCA cellular versions. "Phloretin." Biomed Res Int. Volume 2020. Number13586784. 8th Month, 2020. PMID 32851058.</p>
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exhibiting oncology suppression by upregulating and stabilizing LATS kinase along with LATS inhibition of YAP/TAZ thereby characterizing enabling LATS to suppress oncology in pancreatic, breast and other neoplastic change. Genes have common microRNA and competitive interactions can cause one gene to sequester the micrRNA of another, such that USP3 3'UTR can be overexpressed and result in inhibition of oncology and other disease that is resultant of impaired or unexhibited USP3 function. USP3 enabled oncology and pathogenic migration can be inhibited by downregulation USP3 or downregulating SUZ12.

Inhibition of UCHL5 and USP14 results in proteasome inhibition and causes accumulation of proteasomal substrate, all of which destabilize oncology cellular stability and function. USP4 deubiquitylates pRb, and, thus, performs a dual role linked to phosphorylated or dephosphorylated pRb which oppositely regulates cellular cycle, such that in numerous oncology conditions USP4 is linked to detrimental or pathogenic circumstance while it can perform an essential role in enabling apoptosis in cellular entities of oncology of breast. More than 100

	<p>deubiquitinases are characterized in the literature. Information 10.1016/j.bbamcr.2004.10.003. Vialinin A, derived from particular mushrooms, inhibits USP4 and alleviates experimental hepatitis and leukemia.</p>	
<p>Molecular mass of Hyaluronic acid. Correlation of hyaluronic acid to increased pathology in atypical proliferation of breast tissue, thus seems reasonably causally linked to aerobic glycolysis and increased synthesis of Hyaluronan as method of diseased cellular entities becoming integrated into existing tissues or structures when hyaluronic acid is being processed into smaller oligosaccharides.. An explorative analysis of the multiplicity of Hyaluronic acid conclude that High Molecular mass Hyaluronan promotes cellular stability and structure, while inhibiting cellular release from tissues, inhibiting migration as well as preventing distal integration into tissues.</p> <p>These beneficial functions were linked to the enzymes CD44, LYVE-1, and STABILIN2. Free radicals and hyaluronidases exhibit activity during tissue remodeling, wound repair and inflammation, as well as enable cellular migration which can include acceptance of immunological monocytes. The inflammation phase is linked the activity of RHAMM and TLR2 as well as TLR4. This suggest that like most pathways, Hyaluronic Acid synthesis and consumption by processes can become commandeered by immune response or repair processes in a way that can increasingly potentiate commandeering by pathogenic atypical proliferation. The context presented here includes an upregulation of glycolysis that also includes dysregulation of glycolytic control, while other processes such as inhibition of PEMT and upregulated inflammation may continue to occur. These seem to be how Hyaluronic Acid becomes utilized for pathogenic atypical proliferation.</p> <p>The systematic, continued and constant exposure of Human populations to wireless, electrical, and environmental particulate influences, all seem to be processes by which inflammation transforms hyaluronic acid to be included in pathogenic processes, but certainly these suggest why PEMT inhibits glycolysis and Hyaluronic synthesis, since otherwise Hyaluronic acid might be overly produced as well as become integrated into pathogenic processes. Certainly, upregulation of</p>	<p>Oral and Topical instrumentation of Hyaluronan or Hyaluronic Acid are presented in beneficially literature. The hyaluronic acid inhibitor 4-methylumbelliferone inhibits proliferation in a diverse group of atypical proliferation conditions, including rescue of pathogenic and resistant versions of this diverse group of atypical proliferation conditions.</p> <p>7-Hydroxy-4-Methyl Coumarin HMC also inhibits Hyaluronan Synthase in a way that directly inhibits particular oncology versions, including prostate oncology. Sulfated Hyaluronic Acid inhibits prostate oncology.</p> <p>A resilient stromal cellular entity example of prostate inflammation was clearly managed and inhibited by Hyaluronic Acid supplementation. These present how important it is to consider how pathogenic processes escape PEMT inhibition enabled P53 expression's inhibitory cascade, resulting in emergence of a diverse array of pathways that</p>	<p>Oral and Topical instrumentation of Hyaluronan or Hyaluronic Acid are presented in beneficially literature. Inhibiting CD44 or Hyaluronan synthase 3, as well as inhibiting other Hyaluronan Synthases can provide therapeutic benefit. HPV viral conditions required Hyaluronic Acid integration with CD44 to enabled HPV16 E6 expression such that inhibiting Hyaluronic Acid expression with small interfering RNA results inability of HPV16 to express E6 protein, inability for Hyaluronic Acid to complex with CD44, as well as inhibition of the expression of the prosurvival proteins CIAP1 and cIAP2, impairing essential phase of pathology. However, high molecular mass hyaluronic acid promotes CD44 clustering while oligosaccharides of hyaluronic acid diminish clustering although high molecular mass hyaluronic acid prevent cellular adhesion and oligosaccharides of Hyaluronic Acid or low molecular mass hyaluronic acid promotes cellular adhesion. Information. J Biol Chem. Volume 287. Number 51. Pages 43094 to 43107. December, Inhibiting CD44 or Hyaluronan synthase 3, as well as inhibiting other Hyaluronan Synthases can provide therapeutic benefit.</p>

Krebs cycle being supplied Acetyl-CoA by upregulated glycolysis, while PEMT inhibition and P53 upregulation continue to occur, produces a specific potential that inflammation enzymes and processes might be produced supplantively for stabilizing enzymes which might otherwise be being produced. Similarly, upregulation survival signaling, proteolysis, S1P signaling, G – Proteins, ATPase, GTPase, GSK3B or other factors linked to inflammatory proliferation, all explain how hyaluronan might be increased pathogenically because it is occurring in context in which it would most typically be inhibited. Hyaluronic Acid produces an insulation layer that prevents the infiltration of leukocytes during inflammation.

would be inhibited such that this escaped pathways are exhibited among the inflammatory changes in the cellular environment and disease microenvironment. A spontaneous leisure or sports event using durable projectiles can be fun and beneficial, but this can change if such event emerges in a crowded and full parking lot and no one uses safety equipment. 1,25-Dihydroxyvitamin D3 (1,25D3) produces inhibition of Hyaluronic Acid Synthase 2 in a way that is beneficial in breast oncology cellular examples and in tissue, although high molecular mass Hyaluronic Acid, which is decreased by Hyaluronic Acid Synthase Activity, diminishes this beneficial effect. These demonstrate the essential nature of an encompassing inflammation environment in causing Hyaluronic Acid to become pathogenic. 4-Methylumbelliferone inhibits Hyaluronic Acid Synthase as well as inhibits Uridine Diphosphate, or UDP, in ways that occur across barriers protecting neurological centers, exhibited therapeutic effect in neurological centers and having beneficial effect to atypical proliferation which occurs to astrocytes, glial cellular entities and other tissues.

<p>Macrophage polarization as M1 compared to M2. M1 macrophages recruit other macrophages into its inflammatory M1 status that includes use of L – Arginine and Ca²⁺ to produce L - Citrulline and Nitric Oxide, although uncoupling of iNOS can produce superoxide, which becomes H₂O₂, Peroxynitrite, hypochlorite and trimethylamine-n-oxide, requiring management to prevent disabling of biological active molecules and production of an infarct. M2 macrophages perform along with resolvins and other inflammation resolution pathways to resolve inflammation, utilizing the Arginase pathway that produces L -Ornithine and Urea. Macrophages of one phenotype can recruit other phenotypes to change. This explains complexities in inflammation. Information Sci Rep 10, 16554 (2020).</p>	<p>Inhibiting STAT6, IRF4, JMJD3, PPARδ, PPARγ STAT6 can downregulated M2 macrophage polarization, as can inhibition of the Arginase pathway. M1 activation can be inhibited by inhibiting NF-κB, STAT1, STAT5, IRF3, RF5 but particularly by downregulating NF-κB and STAT1, although inhibiting iNOS can be useful in this regard. This explains how iNOS inhibition can be pervasively useful in disease management. However, these also explain that iNOS expression resultant of energy fields can cause a somewhat consistent inflammation status by recruiting Macrophages into M1 status, particularly benefiting diseases such as viral disease, HIV, SARs and other conditions.</p>	<p>M1 Macrophages involve TLR2, TLR4, CD80, CD86, iNOS, and MHC-II surface phenotypes releasing cytokines and chemokines which convert other macrophages into M1 status, producing NF κB, STAT1, STAT5, IRF4 and IRF5 that are detrimental to emerging neoplasm and microbes. M2 polarizations emerges to resolve inflammation, being invoked by IL4, IL13, IL10, IL33, and TGFB with IL5 and IL5 being converting pathways and IL25, IL33, both amplify macrophage activation through TH2 cytokine expression. M2 cellular surface indicators include IL10, TGFbeta, CCL1, CCL17, CCL18, CCL22, CCK24 which also recruit macrophages toward M2 phenotype. STAT6 is the major M2 polarization pathway for macrophages, while prevention of infection, tissue repair, immunomodulation and angiogenesis are all functions of M2 macrophages.</p>
<p>Trimethylamine compared to Trimethylamine-n-oxide. Choline, l-carnitine, and betaine can be metabolized by digestive pathway microflora into trimethylamine, followed by microbial TMA lyase production of trimethylamine-n-oxide or transit of trimethylamine through the inflammation affected, TNF alpha affected, relaxed tight junction proteins of the digestive pathway into circulation, where trimethylamine-n-oxide can be produced from trimethylamine.</p>	<p>Anaerococcus hydrogenalis, Clostridium asparagiforme, Clostridium hathewayi, Clostridium sporogenes, Edwardsiella tarda, Escherichia fergusonii, Proteus penneri, and Providencia rettgeri, all are particularly linked to trimethylamine production. Broad Spectrum antibiotic during an emergency, phenotype digestive pathway microflora during an emergence, or therapeutically otherwise, preceded by a laxative therapeutically other than an emergency although this can be an</p>	<p>3,3 DMB and a TMA lyase inhibitor can be strongly therapeutic. Information. 10.1155/2020/4634172</p>

	<p>emergency option, while also followed by a prebiotic, postbiotic, macrobiotic, all are indicated. Olive oil, Grapeseed oil, Balsamic Vinegar, boxed or filtered water, are all indicated. Management of the uncoupled NOS cascade can occur by using Superoxide Dismutase, Catalase, N – Acetyl L Cysteine, Peroxiredoxin, Glutathione, tetrahydrobiopterin, as well as vanadium and L arginine can be beneficial. Ornithine and Citrulline can repress macrophage polarization.</p>	
<p>NAD⁺ levels, typically compared to NADH.. Balance can include NAD⁺, NADH, NADP⁺ and NADPH. Nadmed diagnostic assay technology quantifies all four of these with diagnostic assay. Inhibition of PEMT and upregulation of P53 causes inhibited glycolysis which substantially decreases NADPH, glycolysis, pentose phosphate pathway, krebs cycle.</p> <p>Information. Nat Cell Biol. 2011 Mar; 13. 3. 310 to 316. The literature regards the free NAD⁺ to free NADH ratio near 700 as being typical because it favors redox interactions that enable H⁺ abdication from NADH to produce 2 eV⁻, fluorescence, and promotes a negative background pH that enables excitement in the natural gradient between H⁺ and H⁻. 2 eV⁻ and fluorescence interact with and effect factors in the field of influence, and can sometimes promote assembly of the 2 eV⁻ into NAD⁺ but can also be absorbed, utilized or become expended through particle, wave, atom and molecular interactions. Molecules, atoms, ions and other material are promoted out of the ground state such that electrons, in particular, leave the ground state into an excited state that can become so excited that its distance from the nucleus exceeds that distance required for an electron to be considered as integrated into the atom, resulting in an Ionization. The energy emitted in a redox transaction promotes different behavior in the affected biological environment, with enough energy being presented to enable an electron to change energy levels. These dynamics seems to elucidate efficiently the difference of biochemistry</p>	<p>NAD⁺ accepts two H⁺ cations from an ethanol molecule, detoxifying the molecule and producing NADH in a pathway that can bidirectionally use alcohol dehydrogenase, aldehyde dehydrogenase, inorganic phosphate exchange, that leads to NADH and pyruvate or which depleted NADH and pyruvate. Exposure of living cellular entities to Ultraviolet light results in Mitochondrial NADH emittance of Blue Fluorescence, occurring at near 340 nm varying at about 30 nm and emitted at near 460 nm varying by about 50 nm. The remote location of the phosphorylation in NADP results in NADPH having an absorption and emittance fluorescence of the nicotinamide ring that is similar to or the</p>	<p>Quenching occurs when an excited molecule does not return to a stable status through fluorescence, but does so through interaction with other molecules near or attached to it, as well as when an aspect of the molecule itself exhibits a change such as spin that results in return of the excited molecule to a stable status.</p> <p>Remarkable imbalance in NADP to NADPH or NAD to NADH can result in inversion of NNT Nicotinamide Nucleotide Transhydrogenase, which has catalysis of NADH and NADP⁺ bidirectional interaction with NADPH and NAD⁺, occurring in the mitochondria as this enzyme spans the inner mitochondrial membrane at the DII domain of its DI, DII and DIII domains. tetrapeptide SS-31 rescued patients with pressure overload that has inversion of NNT as a canonical nuance of such pathology. Information. Cellular Metabolism. 22. 472 to 484. September 1, 2015. Online Information. 10.1016/j.cmet.2015.07.008. NNT polymorphism is linked to inability to thrive at conclusion of gestion</p>

from chemistry and the biochemical or biomolecular nuance that might be considered foundational nuance of life, with life being the performance of redox interactions to produce the exhibition of biological activity particularly against natural gradients in one perspective, although it is known that the insertion of hydridic character into a molecule, often by carbocation rearrangements exhibited along with phenyl, methyl group or hydridic interactions as well as aryl or resonant interactions in which carbocations are exhibited near, but directly attached to, a four carbon ring or a five carbon ring.

It should be presented here that freeing H^+ from NADH or freeing hydride does not result in circulating H^- , unless extreme conditions exist hydride is integrated into other factors, but the 2 eV-, fluorescence and possible angular characteristics of hydride are exhibited separated from the molecule abdicating H^- when H^- becomes abdicated from a hydride carrier. Information. "Atoms and Light Energy." Imagine the Universe. Educators Corner. Goddard Nasa Flight Center. The National Aeronautics and Space Administration.

The ratio of aggregate NAD^+ to aggregate NADH is presented as being between 3 and 10, although the ratio of hydride to hydrogen in methyl groups is 1 to 2 or 1 in 3 such that circulating methylation factors promote this minimal ratio of unreleased hydridic potential although hydride carried in Iron Sulfides exhibit a different ratio. Some of the literature presents observed ratios of NAD^+ to NADH at as much as 2000 although substantial variance is typically linked with pathology or ephemeral biological contexts and circumstance. The environment is a substantial sources of hydride with methyl pathways being substantial capabilities in such regard. Methylthioglycolic acid elutes hydride and sulfur form the environment while other antihistamines in the CDP ethanolamine pathway and PEMT's production of PMME, PDME and enriched phosphatidylcholine exhibit inorganic to organic phase transfer which sequester biological materials from the environment and move these into physiology. Methylthioglycolic Acid, PMME, PDME and enriched phosphatidylcholine, including CDP – Ethanolamine pathway elements are scrubbing factors and can be utilized to deteriorate industrial wastes, pollution and toxic factors emitted into the environment.

Interestingly, these factors enable humans to be used by the environment as scrubbing factors that clean the environment, although this relies upon human physiology not being impaired by factors

same as NADH. Freely diffusing NADP/NAD levels determined by photoelectric moment confirmed by time anisotropy imaging analysis compared to enzyme integrated NADP/NAD levels is used to monitor fluorescence decay is correlated to enzyme integrated NADPH/NADH ratio.

This confirms that emitted 2 eV- from oxidized Hydride is a metabolic determinant factor exhibited between the levels of two different metabolic molecules reduced by hydride, clearly constituting a correlation, and which is described by the causal mechanistic link of Hydride oxidation, reduction, and redox. Essentially, the two primary factors to which hydride is reduced and from which hydride is oxidized in redox are in relationship with one another, and that relationship is determined by or correlatively exhibited in fluorescence emitted by the 2 eV- freed when hydride is oxidized. Reduction is not specifically included in this relationship because the energy freed from hydrides can be distributed in tissues, environment, and metabolic pathways, sometimes resulting in production of more tangible factors such as ATP or pyruvate, as well as included in

such that CRISPR, gene therapy, and proteomics, all may rescue such a diverse array of conditions correlated with inability to thrive.

Impaired redox balance in cardiomyopathy is known to cause enhanced acetylation within the Mitochondria contributing to pathogenic changes link to increasing levels of impairment. Managing redox balance and causes of impaired redox balance can be strategies for resumption of improved cardiac function. Information Circulation. 2016. 134. Pages 883 to 894. Information online 10.1161/circulationaha.116.022495.

LOXCAT, a fusion of lactate oxidase and catalase, can reconstitute $NADH/NAD^+$ ratio in the brain and in the heart. The study included small mammal models of nonhuman redox impairment but both factors are active in redox pathways in human physiology. Information. Nature Chemical Biology. 16. 225. Page 645. 2020. Online Information 10.1038/s41589-020-0485-1.

Rapamycin stabilizes the otherwise increasingly impaired $NAD^+/NADH$ redox balance in muscle cellular entities exhibited in culture. Rapamycin favors a more oxidized balance suggesting that it increases NAD^+ compared to NADH in a way that prevents attrition of NADH balance. Rapamycin also rescues PARP inhibitor resistance in BRCA1 deficient oncology and is used along with Olaparib in non small cellular pulmonary oncology or NSCLC.

Impaired levels of NAD^+ compared to levels of NADH, $NAD^+/NADH$ levels are linked to the Cisplatin toxicity and hepatic impairment suggesting clearly that assuring NAD^+ to NADH ratio, or adequate NAD^+ in this context, may be able to rescue toxicity, while presented

produced by humans that inhibit PEMT. Nature, for instance, can modulate human inclination for reproduction in ways that are correlated to natural events. These scrubbing factors if included plastic and drinking cups, typically deteriorate the structure of these factors, being considered to be caustic substances outside of physiology and increased volume as well as increased molarity. Methylation of some toxic metals can enhance toxicity, presented the important role of thetin - homocysteine methylperase and thioglycolic acid in managing metal toxicity. Although glycolic acid is considered to be incompatible with some active heavy metals, methylation of metals changes reactivity and incompatibility can be feature of separating metals from biological material or cleaning as well as dispersing factors in toxic conditions.

TIGAR ameliorates this by increasing glycolysis and increasing NADPH production, sometimes with pathogenic potential which compares to reenabled function of any pathway suppressed by P53 without resumption of PEMT catalysis which represents canonical pathway pervasive disease. NAD⁺ depletion inverts some central enzymes in glycolysis and gluconeogenesis, as well as impairs s – adenosyl homocysteine conversion to homocysteine by S – adenosyl homocysteine hydrolase, as well as enhances P53 inhibition of glycolysis and P53 inhibition of the pentose phosphate pathway by downregulating glucose – 6 – phosphate dehydrogenase. Inhibition of PEMT downregulates PEMT attachment of Methyl Groups, CH₃, to phosphatidylethanolamine, thereby enabling a universes level suggested optimal ratio of hydride to hydrogen, 1 to 2 or 1 in 3. Canonical pathway of pathogenic deterioration of NAD⁺ also includes PARP signaling which utilizes the ribose from NAD⁺ to attach to substrate at the loci of genomic impairment to cause gradients upon which nucleotides and other substrate for genomic repair is recruited. The PARP signaling occurs on million or more instances in each day in each cellular entity, requires adequate PEMT or choline to produce nucleotides, persists without cessation until adequate nucleotides and substrate are produced to conduct such repair, potentiates inclusion of nucleotides derived from exogenous sources when PEMT is not functional, performs less than optimal DNA repair when inadequate NAD⁺ is exhibited, produces Nicotinamide remnants during such persistent signaling that requires Nicotinamide Methyltransferases to manage in a way that results in increased levels of homocysteine, and performs optimal DNA repair when adequate NAD⁺ and Nucleotides are exhibit

processes that produce NADH or NADPH. Information Nat Commun. 2014 May 29, 2014. Volume 5. Number 3936.

EGCG or Epigallocatechin competitively inhibits NADPH from attaching to its enzymes. β-hydroxybutyrate vs acetoacetate, often titrated, can be used to manage NAD⁺/NADH ratio and is widely instrumented in this capacity.

Other modalities of NAD⁺ stabilization include these.

Acute provision of NAD⁺ through reversal of mitochondrial malate dehydrogenase. Acute provision of NAD⁺ through reversal of isocitrate dehydrogenase. Acute provision of NAD⁺ through residual complex I activity. Acute provision of NAD⁺ through reverse-operating malate-aspartate shuttle. Acute provision of NAD⁺ through NAD⁺ import across the inner mitochondrial membrane. Artificial means of manipulating matrix NAD⁺/NADH INCLUDING MitoLbNOX, LbNOX, LoxCAT, β-hydroxybutyrate using titration vs acetoacetate using titration. Acute provision of NAD⁺ through mitochondrial diaphorases with early

how even toxicity from therapeutics can be managed by assuring optimal PEMT function and function its linked pathways. Online information [10.1155/2016/4048390](https://doi.org/10.1155/2016/4048390).

IV Hydration therapy, IV NAD⁺ therapy, as well as NAD⁺/NADPH therapies with supplemental, oral, or IV capabilities are emerging in clinical and care contexts as well as in consumer contexts not requiring prescriptions. Although NAD⁺ and NADH are available supplementally, the literature suggest that these are difficult to absorb requiring precursor supplements in order to comprise an optimal therapeutic level when being instrumented orally.

to cause the polymerase to separate from the loci of DNA repair in enablement of optimal genetic repair machinery implementation.

It is known that mammalian mammary epithelial cellular entities have NADPH oxidase and cellular migration inhibited by wild type P53, while particular polymorphism of P53 reactivate NADPH Oxidase activity as well as enable cellular migration. Study observes higher, more acid intracellular pH, as well as higher ATP levels over the complete cellular cycle in typically proliferating cellular entities. Pathology statuses, such as oncology or atypical proliferation exhibited a NAD⁺ / NADH ratio and NAD⁺ /NADPH ratios which were experimentally 500 percent, 1000 percent, or more higher than typical. Information Metabolites. 2016. 6. 33. Information. Internet resource. 10.3390/metabo6040033. Small nonhuman mammalian NADPH/NADP⁺ ratio /in pancreatic islet beta cellular entities increased correlative to instrumentation of glucose, but did not result in an NADH/NAD⁺ ratio change, while fructose instrumentation abrogated the ability of glucose to increase NADPH/NADP⁺ levels, and while leucine instrumentation increased insulin but decreased cytosolic free NADH/NADP⁺, leucine and glutamine together changed both NAD and NADP ratios, clearly suggesting that possible pathways of pyruvate metabolism was a dominant factor in how redox flux with the factors is changed. Information Biochem J. 1987 January 1. 241. 1. 161 to 167.

The fluorescent moment of enzyme integrated NADPH or NADP⁺ was 2.7 nanoseconds when NAD Kinase was inhibited while overexpression of NAD Kinase increased free NADPH between 400 and 500 percent with an enzyme integration duration of 3.8 nanoseconds. NAD Kinase modulation did not experimentally affect NADH levels while NADPH has an increased enzyme integration duration than NADH, such that NADPH to NADH ratios inform the NADPH or NADP⁺ enzyme integration duration. These suggest that when NAD is exhibited as NAD⁺ or NADP⁺ or is integrated into a molecule or enzyme, the H or hydride has been abdicated into a molecule, into a metabolic pathway, into a biosynthetic pathway or other, in a way this correlated with a freed 2 eV-fluorescent moment or field that is included in the cellular level energetics, capacitance or cellular battery. This energetic level includes emitting of fluorescent energy or light. This might be regarded as hydridic character. Ether linked fatty acids integrated by PEMT into phosphatidylethanolamine

research in the 1930s by Straub and research in the mid 1940s and later by Ernster which are omitted from pubmed but included in Acta Chemica Scandinavica since the 1940s and included at www.actachemscand.org. It's a mystery why this research has been excluded from pubmed. Acute NADH provision to counteract acute reductive distress. Countering NAD⁺ inadequacy in disease, impairment, ischemia, reperfusion, neuronal impairment, neurological center deterioration, emergency conditions and other contexts.

Information.

[Experimental
Neurology](#).

Volume 327. May, 2020. 113218.

and phosphatidylcholine, as well as PMME and PDME, along with LPCAT Lands cycle shuffling of fatty acids along with lipids and lead groups that have hydride integrated into them, act as insulators because of the diminished conductance of alcohols, assisting in the exhibition of this foundational nuance of capacitance and biological compartmentalization. Information Free Radic Biol Med. 2016. November. 100. 53 to 65.

The capacitance of mitochondria can require 3 hours or more without sustaining nutrients to become diminished while massive levels of mitochondria in massive cellular bases within tissues can take days for such capacitance to become abrogated, while such capacitance can also be reconstituted, protected, prevented from being diminished and rescued. These might challenge heretofore exhibited opinion in this regard. Cytosolic NAD⁺ density is observed in some literature as about 100 uM, while Mitochondria exhibited about 250 uM, although NADH to NAD⁺ to NADH can be as high as 1 to 1000 in the cytosol with mitochondrial gradient of NADH to NAD⁺ at about 1 to 10.

Managed NAD⁺/NADH ratio is widely stabilizing and preventive in most every concluding phase of disease or metabolic syndrome, including Alzheimer's.

The Water molecule in its fundamental configuration is bipolar with oxygen exhibiting a partial negative polarity and Hydrogen exhibiting a partial positively polarity. Spontaneous production of hydride seems to be an important inadequately promoted participation of water in foundational nuances of biology. Proteins in solution are encompassed by a hydration shell, and this hydration shell can be different according to the shape, twist, writhe, molecular constitution, and distance which molecules exhibit from one another, such that a change in molarity of water might naturally occur along this continuum. However, generally, the hydration shell can range up to about 10 Angstrom in density. The hydration shell keeps particles dispersed in water.

The medical literature describes the process of molecular interactions as relying upon the hydration shell for molecular structure as well as an essential role for H⁻ and H⁺ in molecular interactions with H⁺ being utilized to quench negatively polarized atoms and H⁻ being utilized to quench positively polarized ions.

The H^+ produced from H_2O exhibits a proton or is, essentially, a proton and it is known as H^+ because the periodic table guides the observer to that conclusion. There does not have to be a hydroxide produced from each atom that becomes an H^+ , such that the dissociation of H to either hydroxide or H^+ does not have to occur from the same Hydrogen atom. Separately from the Hydrogen that becomes an H^+ , Hydrogen is dissociated and becomes complexed with oxygen and its 8 electrons and 6 electrons in the outer energy level to be $(HO)^-$ while no electrons are obtained from the H but a proton is obtained from H , resulting in Oxygen having an additional proton.

This causes Oxygen and Hydrogen as a proton exhibiting 9 protons and 8 electrons, which is utilized to calculate the -1 polarity of $(HO)^-$. This calculation ignores the fact that 2 open electron orbitals are unoccupied in the outer energy level. Thus, dissociation of the water molecule, following the literature, results in either an H^+ Cation or a hydroxide anion. Information. Hydrogen Ion. Britannica. Information. The Complete MCAT. Hydration, the Hydronium Ion. Information. PNAS. Volume 104. Number 52. Pages 20749 to 20752. December 26, 2007. These are at least conceptually relevant or similar, since some of the literature exhibits divergent information in this regard.

The interface or encapsulating solute for a solvent or solute is known as the solvation shell or solvation sheath, such as the hydration shell or hydration sphere exhibited about solvent and solutes in water solution. Some of the literature attribute thickness of the solvation shell to polarity of the atom adjacent to the shell or polarity of the molecular complex generally. This clearly suggests that hydration of the microenvironment can determine polarity characteristics of biologically active molecules.

The Hydration Shell lends characteristics to proteins that are essential in biochemistry, providing essential functional characteristics to proteins through protein hydration, amongst distinct characteristics of water that is within 1 nm of the protein. Flux through interaction or actual interface with the protein by an individual water molecule is presented as occurring in the less than nanosecond range. The flux duration of water molecules through the complete thickness of the hydration shell might be nearer to picosecond range or femtosecond range. Hydrophilic dynamics seems to potentiate diminishing of these attractive forces between the hydration shell and protein within the hydration shell.

Dehydrons are hydrogenic adhesions which are produced in hydrophobic intramolecular areas, similarly to how methyl group shift, hydride shift or hydridic donation of an electron, as well as methionine move to molecular centers. hydrogenic centers or hydrophobic areas in center of molecules. Dehydrons perform energetically favored and thermodynamically favored dehydration because they are protected from active catalytic involvement by water molecules. The result is a change to the surface tension around a protein or molecule, link to nonpolar axial groups that wrap polar pairs within protein structure, requiring protein associations to perform dehydration or requiring ligand integration as well as requiring activation to perform dehydration. Therapeutics have been developed which wrap and shield dehydrons from water molecule access and catalytic activation by water.

Protein hydration dynamics were analyzed in a particular study using acetylcholinesterase, subtilisin Carlsberg, ubiquitin and Lysozyme, resulting in the observation that water molecular reorientation dynamics are slowed by between 100 percent up to 300 percent when perturbation of the hydration shell occurs from proteins or molecules coming into close proximity with one another. The perturbation causality was not ascertained by the study. Information. J Phys Chem B. Volume 118. Number 28. Pages 7715 to 7719. July, 2014.

Another study observes intricate interactions between the hydration shell, protein interactions, as well as intramolecular interactions. Glass producing liquids were utilized in the study and such liquids are known to exhibit alpha fluctuations which affect large expanse motions of proteins and seem to be excluded from deterministic influence of hydration shells, at least for glass producing fluids. Beta fluctuations are reliant upon hydration and occur even in a rigid structural environment, not particularly of regard in biological environments. Presumptions for such study included the observation that myoglobin, the oxygen storage proteins, exhibit permeability of carbon monoxide through the interior of myoglobin even if myoglobin is exhibited in a durable nonfluidic structure. Published information about the Fe group in myoglobin was used to determine protein fluctuations and a fraction of gamma rays emitted by Fe in myoglobin occur without abdication of energy as well as are controlled by Fe nuclei. The data suggests that proteins have unique fluctuation frequencies and suggests that beta fluctuations are integral to internal molecular motion. Information. "The Role of the Hydration Shell of Proteins."

LANL Directed Research and Development Program. LANL.

These are interesting because they suggest that a change in ionization or change such as carbocation, shift of methyl groups and one carbon units, hydridic shift, or rearrangement of an electron from hydridic molecules, can result in a ionization or result change to ionization, producing a rearrangement that changes the characteristics of the hydration shell.

The ATP attached to methionine when adenosine is attached to methionine, results in a polarization of the sulfur, making it a + to suggest that one of it is the receiver in an ionization change.

The acquired shared electron activates the Methyl Group, and the interaction of PEMT in particular or interaction of other methyltransferases with the methyl group of S-adenosyl methionine is enabled by the ATP activation of the Methyl Group through polarization or ionization of the sulfur. The nitrogen and its three open locations within phosphatidylethanolamine which are target destinations for each methyl group freed from s adenosyl methionine, actually are represented as 6 open electrons on the accepting nitrogen because the Carbon adjacent to the accepting nitrogen donates an electron, resulting in 6 electrons in the outer valence level of the compound molecule produced by Carbon and Nitrogen. Each CH₃ group exhibited as CH₃ has each Hydrogen sharing an electron with a Carbon Electron, while two electrons are unpaired, resulting in a lone pair that causes the Carbon and H₃ compound molecule to also have an – polarity or negative polarity, indicating that donated electrons, in this instance donated protons as well which occurs when hydrogen is integrated into a molecule. CH₃ when integrating through the action of PEMT into the nitrogen of Phosphatidylethanolamine attaches the CH₃ to the open location of phosphatidylethanolamine and its negatively polarized Nitrogen. The three CH₃ molecules attached to the N- of phosphatidylethanolamine results in phosphatidylcholine which as positively polarized Nitrogen as N⁺. This N⁺ is correlated to the S⁺ of the s adenosyl methionine from which each CH₃ originated when PEMT frees them for transfer to the Nitrogen of Phosphatidylethanolamine. This + polarization results from abdication of hydridic character from S adenosyl methionine molecule that has caused the sulfur to become + ionized, followed by transfer of this hydridic character to the Nitrogen of Phosphatidylethanolamine 3 times to cause

Hydridic character to be exhibited in the destination product phosphatidylcholine. Because of carbocation arrangements, the hydridic character which is typically negative or - in raw hydride, is exhibited as + polarization of Sulfur and Nitrogen.

The abdication of a carbocation integrated hydride or methyl group as hydridic character rearrangement that can involve protons and electrons instead of a whole molecule, exhibited by S adenosyl methionine resultant of the action of a methyltransferase such as PEMT, seems to produce a particularly unstable and particularly toxic molecule known as S adenosyl homocysteine, such that even the activity of S adenosyl homocysteine hydrolase does not completely diminish this toxic characteristic. Correlatively, a particularly gifted group, individual or team, correlatively, can be particularly affected if such gifted characteristics become abdicated, particularly if such gifted entities are aware of such capabilities and utilizes such capabilities in benefit thereto. These present hydridic character as enable change to molecules that might use hydridic character or benefit in sustainment of biologically beneficial metabolic transactions.

A most important observation is that the polarization of the sulfur abdicates or shares an electron from sulfur and the abdication of the electron fits very nicely into PEMT affixing the Methyl Group with an additional electron to Nitrogen to fill open electron positions.

These suggest that hydride in molecules can cause an excited status without abdication, fracking or freeing of Hydride to produce 2 eV^- . Hydride can cause an excited status that polarizes environment and molecules without being freed and, of course, resultant of carbocation or rearrangements using electrons derived from hydride, hydride, once carbon units or methyl groups. Biological compartmentalization, then, produces a capacitor. These can emit fields or exchange ions with the external environment in which such a compartmentalized capacitor is exhibited.

Hydride freed from NADH along freed H^+ produces a natural gradient that is used for energy along with emittance of fluorescence and a freed field of energy that causes electron transport pathway metabolites and enzymes to enter an excited status that includes even distribution of about 58 percent of such freed energy among the phases of the electron transport pathway that can result in the ionization's required for energetics

used to perform these phases of the electron transport pathway, culminating in integration of about 42 percent of such energy freed as 2 eV- from NADH becoming integrated into the oxonium exhibition between the phosphate groups of ATP. Ether linked fatty acid fraction of phosphatidylethanolamine preferred by PEMT in selection of substrate for catalytic activity results in enhanced density of enriched ether linked phosphatidylcholine followed by distribution of these fatty acid species to diverse phospholipids through lands cycle processing which includes freeing of fatty acids by phospholipases and phosphodiesterases followed by reintegration of such fatty acids by LPCAT/MBOAT processes. Lysophosphatidylcholine is included in such processing to produce phosphatidylcholine in particular. The freed energy from NAD⁺/NADH redox is maintained through membranes with enhanced insulation resultant of ether linked fatty acids in phospholipid fraction of membrane lipids, membranes constitutively and superconductivity characteristics which occur when phospholipids cause an increase near to physiological thermodynamic thresholds of the superconductivity thresholds for material, atoms and fields exhibiting in the biological compartment. This produces a capacitance or energy trap that benefits from the caustic quaternary ammonium, inorganic to organic phases transfer, abiotic to biotic phase transfer, useful biological material eluting capabilities of methyl transfer and methyltransferase pathways, along with activation of magnetism enabled indefinitely sustainable permanent magnetic energy production link to indefinite spin characteristics of magnetism enabled electrons.

These present integral indefinitely sustainable physiological energy that is mimicked by civilization utilization of permanent magnet systems emerging in civilization which require no substrate, no fuel, and produce not output, pollution, or factors other than indefinitely sustainable current. These suggest that Humans may be able to produce hydride and current to resupply stars of the universe with energy to counteract an emerging opinion in research that the Universe's stars might not have an indefinite supply of energy, light and energetic fluorescence. These also escape Humanity from resources limitations in assuring light, power and energy. These also suggest that the universes sources of energy might be experiencing natural recycling processes that explain the reason for exhibition or which explain, at least, a explaining usefulness for the exhibition of magnetism.

The trapped energy in the intracellular environment results in exhibition of hydride intermetallic compounds of 3d metals that include rare earth elements. This results in increased exhibition of

principals of magnetism including RKKY interactions or Ruderman-Kittel-Kasuya-Yosida interaction which occur through nucleus magnetic moment coupling, which are essentially localized inner d shell or f shell electron spins that occur in metals. RKKY interactions occur through interactions produced by or produced through conductance electrons and are participative of magnetic interactions. Information. "Conductance Electrons." Physical Review. Volume 96. Number 99. 1954.

Field effects, valence instabilities, coexistence of superconductivity, magnetoeleastic properties, and magnetic order occur along with RKKY interactions in magnetism. 3d metals Mn, Fe, Ni and Co are known to participate in biologically active molecular and hydridic interactions, provide one context of what may be other molecules or factors that are sequestered into biological compartments, biotic phases and organic phases by inorganic to organic phase transfer, abiotic to biotic phase transfer and eluting activity perform b pathways involved in hydridic or hydride eluting, mining, transport and application. The hydrides RCo5, R2Co17, Nd2Fe14B, and RFe11T have emerged as extraordinarily applicable to high performance, highly efficient Permanent Magnet application for substrate bereft, clean, nonpolluting, indefinitely sustainable energy production that provides indefinite, modular and extraordinary energy to form factor size energy for all manner of application. Information. Magnetism of Hydrides in Handbook of Magnetic Materials. Volume 17. Pages 293 to 456. 2007.

Magnetism in this context explains how the background characteristics of the extracellular environment are changed by capacitant cellular entities and capacitant subcellular compartments. These suggest that intracellular and systemic energy production and recycling, as well as energy sustainment may be occurring as an aspect of physiology and that it is a deterioration of rare earth element, mineral and other nutrient obtainment that destabilizes the endogenous permanent magnet energy homeostasis, superconductivity which translates this source of energy through assurance of foundational nuances of cellular existential characteristics and tissue existential characteristics, into subcompartment, cellular compartment, and tissues, and anatomy level traps that sustain physiology. However, the civilization level and biological microenvironment level exhibition of magnetism as a manner of producing sustainable energy seems to emerge amid a context of universes levels fields which physiological compartmentalization seems to be trapping, even at conception, to produce physiological capacitance that is response to external influence and eventually develops to exhibit interactivity, more complexity and ability to be intermediated by conscious

cognitive cognition emerging from brainstem intermediation and into areas of the neurological context involved in enhanced conditioning and enhanced control.

Phospholipids are affected by nano level factors and exhibit nano level change along with imposing nano level or greater influence that it includes structure and thermodynamics, resulting in phases that emerge as structure moves through Boltzmann transition thresholds typical of phospholipid and lipid chemistry in producing anatomical and physiological structure. Phospholipids affect the bending, folding and shaping of cellular membranes, membranes otherwise and nuances of physiology otherwise. Information. "Lipophobic Interaction." Biochemistry. Volume 50. Number 32. Pages 6806 to 6814. 8th Month, 16th Day, 2011.

Another version or characteristic of carbocation arrangements include Saytzeff's or Zaitsev's axiom in which stability of an alkene is highly potentiated when the alkene is excrete from a molecular interaction involving carbocation of from a carbon atom with low number of hydrogens. These likely are reflected in sociopolitical ideology linked to the observer for which the reaction is named.

Another study observes that not only do carbocations prefer and increasingly potentiate achievement of stability but molecules such as iminodiazonium molecules, carbocations that have not achieved stability may have a propensity to achieve such stability, including exhibiting ephemeral excited status resultant of carbocation followed by resumption of stable status. This suggests that carbocations can result in abdication of a hydride in the intramolecular space that results in fluorescence and release of free energy. This suggests that systematic freeing of hydride through carbocations can result in controlled, focused and transport of energy emitting molecules, moieties, phenyl groups, methyl groups, alkyl groups or hydride. Logically this could be very much like a filament in a lighting apparatus or light bulb.

Information. "Carbocations." Journal of Chemical Sciences. Volume 115. Number 1. Pages 41 to 47. February, 2003. Information. Management of Carbocation. Pharmaguideline.com

Thus, cellular membranes and ether linked fatty acids enable enhanced and management of released 2 eV- from hydride, reintegration of hydride into NADH through activities of ret, distribution of energy freed from NADH as hydride, natural gradients produced when NADH releases Hydride as well as release H⁺, translation of free and fluorescent energy kinetic, thermodynamic, and excited status, all by trapping such vectors of energy to produce managed compartments. Cellular

<p>entities emerge in this context of manage contexts of polarity with stable potentials and ability to exhibit polarity changes resultant of activation through neuronal receptors as well as through other ligands, channels or receptors. The energetics are intensified through phospholipid ability to increase toward physiological temperatures the thermodynamic thresholds required for superconductive activity.</p> <p>It is known that phospholipids increase the temperature at which superconductivity occurs, enabling such superconductivity to occur nearer to physiologic thermodynamic levels. The ether linked fatty acid versions of phosphatidylethanolamine which are a preference for PEMT selectivity provide insulation result in ether link fatty acids become integrated in increased fractions of membrane phospholipids. NADH abdication of Hydride in oxidative phosphorylation electron transport pathway activity results in abdication of 2 eV- of energy as current, free energy of fluorescent energy that is used in an even distribution by phases of the electron transport pathway, resulting about 58 percent usage of such energy in pathway processing along with 42 percent being integrated into the oxonium exhibited between phosphate groups of ATP. However, released energy is efficiently trapped by the insulation provided by membranes, particularly ether linked fatty acids in phospholipids.</p>		
<p>A canonical molecular context of cellular cycle progression includes exhibition of cellular cycle typically and through primitive cues, until inflammation, Genetic Impairment, Injury, choline deficiency, inhibition of PEMT, homocysteine, or influence lymphocytes or inflammatory monocytes, as well as inflammatory cytokines emerges. This change can result in inhibition of PEMT or results in exhibition of P53 can downregulate P53, although P53 is upregulated when PEMT is inhibited. P53 introduces cellular cycle pause at G1 where cellular phenotype is already programmed to respond in different ways such as apoptosis, senescence or permanent nonreplicative status, or P53 activation of PERP, a PMP – 22 /gas3 group member, which causes apoptosis. However, P53 occurs along with other pathways, such as homocysteine increases which invoke pathways that potentiate caspase invoking of apoptosis, cytochrome C proton, pathways of deterioration, or other pathways that are a response to these including survival signaling pathways that include choline kinase alpha, proteolysis, S1P kinase that siphons off increasing ceramide that would others be an apoptotic signal, S1P receptors, GSK3B, G –</p>	<p>P27 (CDKN1B) inhibits CDKs Cyclin Dependent Kinases, similarly to P21, but in a different pattern. P27 is regarded along with P21 as inhibitors of G1 exit, but P21 is known to be upregulated by Progesterone – A/ P53 complexes while P21 that cannot integrate with CDKs results in impaired ability to inhibit Cyclin/CDK complexes. Information. J Steroid Biochem Mol Biol. 2019 Jan. 185. 163 to 171. Information J Clin Invest. 2012 Mar. 122. 3. 844 to 58. The deubiquitinase HAUSP is known to integrate with and</p>	<p>Pentagalloylglucose inhibits CD4 and CDK2, as well as upregulate P27kip and P21cip, to cause G1 phase pause in cellular entities of breast oncology. Information Biochem Pharmacol. 2003, Jun 1. 65. 11. 1777 to 1785.</p> <p>P21 is active toward Cyclin D complexed with CDK4/6 in a way that interacts with pRB while P27 interacts with Cylcin E complexed with CDK2 in a way that interacts with pRB, preventing exit from G1 that might otherwise occur resultant of pRb, a ubiquitous inhibitor of cellular cycle progression.</p> <p>E6 and E7 Viral proteins are known to enhance or enable the most pathogenic phenotype of HPV, HPV16 and HPV18, particularly because E6 and E7 transactivates E3 Ligase E6AP and transactivates Cullin2 ubiquitin ligase complex.</p>

<p>Proteins, ATPase, GTPase, etc. P53 can be inherently impaired in Li Fraumeni syndrome impairing at least one P53 gene, but which can be repaired by CRISPR Genetic Repair. TIGAR can be express to in some capacity alleviate inhibition points imposed by P53. However, P53 activates P21 CDKN1A at its genetic promoter region. Information. <i>Genes & Dev.</i> 2000. 14. 704 to 718. PTEN protects P53 from ubiquitylation by MDM2, while MDM2 promotes the ubiquitylation also of pRb which is a ubiquitous inhibitor of cellular cycle progression. Information <i>EMBO J.</i> 2005 Jan, 12. 24. 1. 160 to 169. Phosphorylation of pRb, which P21, p27 and Cyclin/CDKs utilize to enable cellular cycle progression, results in inhibition of pRb cellular cycle suppression pathways, while dephosphorylation results in resumption or continued exhibition of pRb cellular cycle suppression. pRb exhibits 16 locations of potential phosphorylation, but the phosphorylated activation has to occur in particular groups or patterns, while some phosphorylation loci or some enzymes participating in such activity potentiate inhibitory activity. Information <i>Curr Opin Genet Dev.</i> 1998 Feb. 8. 1. 21 to 7. Atypical proliferation conditions canonically exhibit upregulated Cyclin/CDKs, inhibited P53/impaired P53, as well as downregulated P21 and P27, such that exit from G1 phase typically exhibits heavily modified pRb. <i>Genes Dev.</i> 2012 Jun, 1; Volume 26. Number 11. 1128 to 1130.</p> <p>The hidden switch in this context seems to be that dephosphorylated pRb readily integrates with E2F, and it is this integration that suppresses advancement of the cellular cycle. Information. <i>Genes & Dev.</i> 1998. 12. 2245 to 2262. Growth of cellular entities occurs through G1 and G2 but not particularly during S phase. Pauses occur in G1/S transition and G2/M transition, in which P53 implements in checkpoint algorithms that produce an adjudicative interaction between cellular phenotype programming, P53 pathways, status of Genetic and other cellular factors, as well as apoptosis signaling and survival signaling, resulting cellular cycle progression, continued pause, apoptosis, senescence or other outcomes. <i>Methods Mol Biol.</i> 2013. 962. 49 to 61. The hypertrophic phases are G1 and G2, and these phases exhibit accumulation of nutrients and expanding size. P53 causes pause of cellular cycle in hypertrophic phases.</p>	<p>stabilize pRb, resulting in inhibition of cellular cycle progression. HAUSP is overexpressed in glioma and enhances pathology, particularly because glioma exhibits impaired function of HAUSP toward Rb, such that decrease in G1 phase paused cellular entities occurs with more cellular entities moving through G1 phase. P53 is independent of this Hausp and pRb complexity in glioma, but this context is also proteasome reliant, introducing ability to manage these in wider aspects of cellular metabolism presented here. Information <i>FEBS J.</i> 2014 Jul. 281. 13. 3061 to 3078.</p>	<p>HPV E5 protein enhances EFGR signaling by decreasing c – Cbl ubiquitination deterioration of EGFR. USP13, MCL1, and other deubiquitinases are involved in HPV pathology and can be utilized although CRISPR should be able to mitigate HPV, as can polymerase inhibitors such as Favipiravir, iNOS inhibitors such as curcumin, EMF protection, choline kinase inhibitors and BH3 mimetic inhibitors. Information 10.1038/s41388-021-01679-8.</p>
<p>HIPPO, LATS, ITCH, YAP/TAZ pathway factors for intricate cellular control. P53 is exhibited during choline inadequacy, inhibited PEMT and inadequate synthesis of enriched phosphatidylcholine. Cellular entities aggregate</p>	<p>Nitric Oxide Donors and Genetic transfer of iNOS, both resulted in upregulation of P21, causing particular</p>	<p>Cyclin e assists in cellular cycle enabled in all phase, cyclin e exhibits assistance in surmounting the G1/S transition, cyclin e assists beginning before G1/S through</p>

massively in G1/S, G2/M, which are hypertrophic phases. Cellular entities incur massive apoptosis and have to be rescued by inflammatory survival signaling and the immunological systems, as well as inherent cellular programs to direct cellular outcomes away from apoptosis. This amelioration of apoptosis pathways are incipient nuances of pathology. The cellular entities which persist are exhibited in a decreased cellular density per micrometer, such that cellular entities are able to become larger and resulting in expression of iNOS which improves the turgor of cellular entities to enable sustained ability to carry physiology. Growth of cellular entities occurs throughout interphase which includes G1, S and G2 phases. Choline deficiency is an inclusive concept that includes impaired phosphatidylcholine synthesis by PEMT1/PEMT2/PEMT3, impaired nutritional obtainment of choline, impaired transport of choline by transporters as well as genetic or other impairment of choline, methylation, homocysteine depletion/recycling, and impaired one carbon metabolism pathways. Information Nutrition Today. 11 12, 2018. Volume 53/. Issue 6. pages 240 to 253. Confluence is presented as the nature of cellular division in which cellular entities divide until, being experimentally observed in culture but also is known to occur in physiology. Essentially, cellular entities stop proliferating when they come into contact with other cellular entities in a way that occurs through inhibition of CDK enzymes by P27kip1, cyclin D1 downregulation and which has similarity to the way that iNOS production of Nitric oxide or Nitric oxide also causes cellular cycle pause/exit. Information. 'Cellular' Cycle. Volume 7. Number 14. Page 2038 to 2046. July 1, 2008. P27 inhibits a – Cdk1/2, downregulates cyclin D1, and diminishing phosphorylation of pocket protein of pRb, all before or in performance of cellular cycle exit resultant of confluence. Information. How do to a proper cellular culture quick check. Science lab. www.leica-microsystems.com/.

The most substantial observation with confluence is that, massive cellular apoptosis that occurs resultant of choline deficiency, inhibition of PEMT and upregulated homocysteine, each and all, both upregulation cellular proliferation and cause expression of iNOS. iNOS can cause exit of cellular cycle earlier than confluence because cellular entities exhibit a pause induced by P53, P21 and P27 as well as resultant of iNOS in concert with these if iNOS is producing Nitric Oxide, particularly because Nitric Oxide and hypertrophic growth phases occurring in G1 to G1/S phases expand cellular size to potentiate contact with

cellular entities to exit from cellular cycle. iNOS cause exit from the cellular cycle and inhibits proliferation, typical until iNOS depleted Ca²⁺, thereby inhibiting nNOS and eNOS, resulting impaired or uncoupled iNOS, nNOS and eNOS. These confirm that iNOS expression ameliorates basic foundational and molecular mechanisms of cellular proliferation, presumably by increasing turgor of cellular entities in hypertrophic phases. Information. BASIC RESEARCH STUDIES. VOLUME 31. ISSUE 6. P1214 to 1228, JUNE 01, 2000.

. CHREBP can activate cellular cycle in response to carbohydrate. Choline obtainment enables progression of cellular by massive cellular entity groups. Mimicking of choline availability by phospholipase catabolism of cellular membranes can enable cellular cycle progression. Information. Molecular 'Cellular'. Volume 71. Issue 4. 8th Month, 16th Day, 2018. Pages 581 to 591. Exit from cellular cycle occurs for a number of reason and can be enabled by P53, P21, P27, pRb, Genetic Impairment or other reasons, although Genetic Impairment can eventually become

G2/M transition, cyclin b, then assists from completion of G1/S transition to middle mitosis/meiosis potentially to kinetochore checkpoint band perhaps until cytokinesis has stabilized. Cellular populations paused in particular phases, particular G1, may escape when P53 levels cycle downward even when such cellular entities are considered to be senescent. CDK2 upregulation occurs when P21 is downregulated with diminished P53 availability, thereby enabling cellular cycle progression.

The G2 phase is important, as is the G2/M transition, because the hypertrophic phases of cellular cycle occur to obtain adequate nutrients before outside communication is abated before tunneling through the Mitotic/meiotic/cytokinesis occurs. Cellular entities, then, reestablish communication with local, tissue, organ, and systemic environment. G1 exhibits Gene assay checkpoint in late phase, G2 exhibits chromosome structural assay in late phase, M exhibits assay of kinetochore attachment to spindle fibers. iNOS enables proliferation of cellular entities in melanoma, although inhibition of iNOS causes G2M phase pause and introduces apoptosis in melanoma affected cellular entities. Information Oncology Research. 74. 4. January, 2014. Vascular cellular entities seem to proliferate during hypoxia as a signaling cascade along with HIF induced erythropoiesis, presumably because Hypoxia suggests that vascular impairment or injury has occurred, such that Nitric Oxide staves off such proliferation and erythropoiesis or vascular cellular proliferation seem reasonably correlated with an injury response. This suggests that diminished density of oxygen in particular areas or resultant of atmospheric particulate or pollution may cause increases in HIF expression and cause

extracellular environment. Nitric Oxide in the intracellular environment sequesters Ca^{2+} from the cytosol into the endoplasmic reticulum and prevents its entry from the extracellular environment to the intracellular environment. However, when iNOS is exhibited persistently it depletes Ca^{2+} , L-arginine, iron, Tetrahydrobiopterin, vanadium and other factors, causing it to pause, causing iNOS to produce superoxide O_2^- instead, as well as enabling PI3K signaling and other pathways to open pores in the endoplasmic reticulum to sequester and deplete Ca^{2+} from endoplasmic reticulum. The important assertion here is that iNOS competes with PI3K, in a way that SHIP1 or SHIP2 can assist iNOS in prevailing because SHIP1 and SHIP2 two sequester Phosphatidyl Inositol away from PI3K favored PI(3,4,5)P3 to SHIP favored and cholesterol transport favored PI(3,4,)P2 species, thereby enabling more Ca^{2+} to be available for iNOS function. Uncoupled of dysfunctional expressed iNOS depletes Ca^{2+} because iNOS has calmodulin constitutively integrated into its structure, such that the four hands of calmodulin that attach to Ca^{2+} molecules remove available Ca^{2+} regardless of if iNOS functions or is proteolyzed. This sequestration of Ca^{2+} continues even when there is already low or deficient levels of Ca^{2+} . This causes nNOS and eNOS operating in the communication center of the cellular entity known as the caveolae to discontinue operations because calmodulin is integrated with eNOS and nNOS during or after calmodulin is able to attach to Ca^{2+} . The depletion of Ca^{2+} by iNOS impairs exchange of Ca^{2+} between Endoplasmic Reticulum and Mitochondria required for PEMT2, Mitochondrial, Apoptosis, energy, capacitance each to function or become exhibited. Such that PEMT2 dysfunction, mitochondrial dysfunction and Ca^{2+} inadequacy, as well as impaired apoptosis, and pathology, particularly pathogenic cellular proliferation, are correlated. PEMT2 function is typically obliterated in oncology and disease, according to the literature. Thus, when iNOS has become uncoupled or dysfunction and is linked to pathology, inhibition of iNOS results in apoptosis or therapeutic effect that is remarkably effective and remarkably inclusive among disease. The reason it is effective is because it relieves the detrimental affect to Ca^{2+} , mitochondria and endoplasmic reticulum, enables cellular cycle to progression beyond G1 and G1/S, reaching G2/M where sizes are larger but there is also another exit point from the cellular cycle and potential for apoptosis resultant of P53, P27 and reconstituted pathways for apoptosis enabled by resumption of mitochondrial exchange of Ca^{2+} with endoplasmic reticulum.

ignored as an inhibitory factor.

Pulmonary arterial smooth muscle cellular entities respond to hypoxia by proliferating, except when iNOS gene expression is exhibited, presumably in coupled and not uncoupled function, resulting iNOS and Nitric Oxide enabled inhibition of cellular cycle by preventing Hypoxia from downregulating P27. iNOS expressing NOS assures P27 inhibition of cellular cycle. Confirmingly, Nitric Oxide inhibits cellular proliferation in experiments using vascular smooth muscle cellular entities. Information PMID 12899770. Information. Circulation. 2000. 101. 1982 to 1989.

Cellular entities in sparse conditions were activated compared to restrained proliferation by cellular entities in confluence when exposed to fibroblast growth factor which would ordinarily activate p42/p44, c-fos, Mapk MKP1, MKP2, cyclin D1, although distant early pathway factors Ras and Mek1 both were not affected by confluence in these experimental conditions. Sodium orthovanadate reactivated p42/p44 MAPK activity but Okadaic Acid did not. Enzyme analysis presented that lysates from confluent cellular entities

pathophysiological changes that emerge when Nitric Oxide production cannot become exhibited or when Nitric Oxide Synthases become uncoupled.

Nitric Oxide is generally beneficial in numerous conditions. Thus, obtaining nitric oxide directly or through nitric oxide donors can be therapeutics in numerous contexts. However uncoupled NOS of any version, as well as iNOS which is not ephemeral or has depleted its substrate Ca^{2+} in particular, becomes detrimental in numerous contexts. Nitric Oxide is expressed in tissues of astronauts returning from space in order to counteract affect of gravity upon physiology.

PBISe is a therapeutic that is 10 times more effective than its Sulfur exhibiting homologue PBIT in producing apoptosis in melanoma cellular entities. PBISe inhibits iNOS as well as inhibits Akt3 pathway, MAPK cascade. PBISe introduced iNOS inhibition, G2/M phase exit from cellular cycle, inhibition of cyclin d1, upregulation of P21, upregulation of P27, increased Caspase 3 pathway activity and increased PARP signaling. Information. Molecular Oncology Therapeutics. Volume 7. Issue 5. May, 2008.

iNOS is known to cause cellular exhibit at G1 by stimulating P21. The numerous studies suggest that iNOS can cause cellular cycle pause when it is producing Nitric Oxide instead of operating as uncoupled Nitric Oxide Synthase. The literature observes a pervasive role of iNOS in disease and cellular cycle advancement in atypical or pathogenic proliferation of cellular entities. The example of hypoxia here presents that hypoxia, which invokes Hif hypoxia inducible factor, has the potential to invert the effect of some enzymes causing an ameliorated effect of some pathways, but with iNOS expression

These suggest that Nitric Oxide causes cellular cycle to exit early, G1 or G1/S phase because Nitric Oxide is enhancing the turgor or cellular entities and improving contact with surrounding tissue or structure. Exit from cellular cycle caused by inhibition of iNOS, thus exhibits later at G2/M because iNOS may no longer be producing Nitric Oxide and may be operating in uncoupled modality as well as may have depleted intracellular Ca^{2+} and may have depleted store operated Ca^{2+} from the Endoplasmic Reticulum. This depletion of Ca^{2+} from the Endoplasmic Reticulum impairs exchange of Ca^{2+} between the Endoplasmic Reticulum and the Mitochondria through the mitochondrial associated membrane, resulting impaired ability of mitochondrial control of cellular outcomes, cellular metabolism, cellular cycle cellular survival/apoptosis decisions. Because mitochondria produce energy that coordinates activity, dysregulation of cellular energy and metabolisms, occur, resulting control cellular cycle, differentiation, survival, and apoptosis by circumstantial, environmental, energetic, and increasingly extracellular pathway. These also potentiate that control of cellular entities produced by shared capacitant fields link to consciousness and cognitive processes might also be diminished.

Cytotoxic therapy, surgery, or massive apoptosis among cellular entities naturally upregulates cellular proliferation until synthetic confluence when hypertrophic status or Nitric Oxide enhancement to size and turgor mimic confluence occurs or until true confluence occurs, but there are signals such as survival signaling, and other cues that cause cellular entities escape confluent boundary influence.

Typically diverse tissue SHIP2 and IPP4A in the nucleus as well as typically haemopoietic SHIP1 and IPP4B at the membrane are potent regulatory signals that inhibit proliferation and pathogenic mobility. Vaccines that prevent disease, prevent oncology and used as therapy after oncology therapy as well as which might be used as oncology therapy have been successful and are expanding in availability and usage. Vaccines including MRNA vaccines are emerging into wide usage while vaccines can benefit when immunological function is enhanced by assuring PEMT function as well as supplementing phosphatidylcholine.

PI(3,4,5)P3 or PtdIns(3,4,5)P3 accumulation along with AKT signaling, particular AKT2 also, can be highly pathogenic regarding proliferation and

dephosphorylated p42 Mapk compared to less dephosphorylating activity performed by nonconfluent sparse cellular entity lysate. Raf1 cascade eventually was able to reactivate p42/p44 activity even in confluent cellular entities. Mol Cell Biol. 1999 Apr. 19. 4. 2763 to 2772.

Nutrient restriction or inadequacy, such as fasting, initiates autophagy.

AMPK inhibits MTORc1, resulting in inhibition or diminished exhibition of autophagy.

Class III PI3K complexes phosphorylate phosphatidylinositol, typically at locations of the plasma membrane considered to represent canonical function of autophagy, macroautophagy, performing as locations of autophagosome emergence. Information Curr Biol. 2017. Apr 24. 27. 8. R318 to R326.

Cellular entities which have escaped contact inhibition or escaped confluence exhibit YAP/TAZ inability to regulate Myosin – II abrogating the action of F – actin stress fibers that are essential to autophagosome exhibition. This escape occurred with impaired autophagy, increased sensitivity to Hypoxia and increased sensitivity to glucose starvation,

Hif did not cause such inversion in a study, such that it is the exhibition of iNOS in a substrate deficient environment that causes it to become a participant in pathology. iNOS operations as uncoupled iNOS or operates deficiently when not having adequate Ca^{2+} , iron as Heme, tetrahydrobiopterin, L-Arginine and some other factors. These clearly indicate that when iNOS is producing Nitric Oxide, it enhances the turgor of cellular entities and results in exit from cellular cycle similar to the way that confluence promotes exit from the cellular cycle. These present why iNOS is expressed with choline deficiency that results in massive attrition of cellular entities per micrometer, as well as explains why astronauts experience iNOS expression when returning to Earth's gravitational fields to promote increase turgor of cellular entities to strengthen plasma membrane interstitial space and cytoplasmic turgor to assist in carrying physiology. Information. Circulation. 1997. 95. 2303 to 2311. The literature suggests that Nitric Oxide in the intracellular environment promotes Ca^{2+} sequestration for storage in the Endoplasmic Reticulum and prevents cellular uptake of Ca^{2+} , thereby causing Ca^{2+} to be supplied principally through pores that open into the extracellular environment through the plasma membrane where it integrates with the Endoplasmic reticulum, such that a strong axis of Ca^{2+} exchange between the extracellular environment, directly to the Endoplasmic reticulum and to the Mitochondria occurs in a way that causes tight control of PEMT1, PEMT2 and Mitochondria over cellular outcomes. This control of the mitochondria includes an essential availability of Ca^{2+} to implement mitochondrial apoptosis pathways. J Physiol. 2002. Feb 15. 539. 1. 77 to 91.

motility, such that disruption of these and disrupting the exhibition of these together can be strongly influential in inhibition cellular cycle progression, oncological phenotype, and pathogenic mobility phenotype.

Interactions among PTENT, SHIP1, SHIP2, IPP4A, IPP4B can be complex and confounding while stable determinant in this relationships can be accumulation of PI(3,4,5)P3 or PtdIns(3,4,5)P3 being most indicative of atypically proliferation and potential accumulation of the less pathogenic PI(3,4,)P2 or PtdIns(3,4)P2 potentially becoming transformed to enable proliferation in confounding conditions or extreme levels of accumulation.

The complexities and confounders in PTEN, SHIP, IPP4 and PI3K signaling clearly are encompassed by deterministic affect or interactions with Sphingosine Kinase, S1P receptor activation, GSK3B activity and PDK activity, which is additionally expressed as autophagy, compared to proteolysis and mitophagy.

Dysregulated PI3K/VSP34 activities, enhanced levels of PtdIns(3,4,5)P3, or even extreme levels of PtdIns(3,4)P2 or other phosphatidylinositols can compete with iNOS for substrate such as Ca²⁺, such that depletion of iNOS substrates iron, tetrahydrobiopterin, Ca²⁺ in particular can leave adequate amounts of L-arginine for competing L-arginase pathway activity that promote healing, growth, and collagen synthesis. Its possible for this arginase activity to occur while a highly pathogenic phenotype persists regarding cellular cycle, homocysteine levels, PEMT levels, iNOS function, uncoupled status of iNOS and PI3K dysregulation among excess phosphatidylinositol species. PI3K/VPS34 competes with iNOS when iNOS is operating in functional or coupled mode, with iNOS production of Nitric Oxide being prevalent because calmodulin is already integrated into iNOS and must be catalytically integrated with PI3K. However, substrate bereft iNOS can become uncoupled to produce Superoxide which is a macrophage DAMP pattern for activation and inflammation, causing a macrophage cascade and depleting local L – Arginine. Generally, however, uncoupled iNOS continues to function

Since SIP and Sphingosine Kinases as well as choline kinase alpha and nSMase/aSMase are major phosphorylation upregulators and inflammation pathways by cytokines and T Cellular activated pathways involve phosphorylation cascade, it can be generally regarded that disease and oncology

suggesting the proteolysis had emerged in canonical models of atypical proliferation or oncology presented here. Hypoxia is known to cause proliferation of cellular entities except when iNOS is functionally expressed to produce Nitric Oxide.

INPP4A phosphatidylinositol polyphosphatase completes the PI3K phosphorylation cascade pathway and shuttles between the cytosol and the nucleus, such that its translocation to the nucleus depotentiates oncology by introducing potential for massive apoptosis while its exclusion from the nucleus seems to strongly potentiate pathology such as oncology. No specific canonical system has been presented to explain INPP4A shuttling characteristics, although it is not difficult to consider that its utilization and occupation catalytically might be a natural inhibitor of nuclear translocation, as may its post translational modification characteristics.

IP3 3 – Kinases attach to F – Actin in F - Actin's proline rich sequence exhibiting within its N extremity comprised of 66 amino acids. The IP3 3 – Kinases rapidly phosphorylate Inositol 1,4,5 – triphosphate to Inositol 1,3,4,5 tetrakisphosphate in a

Lithium inhibits inositol monophosphatase IMPase activity causing free Inositol to become availability for IP3 catalysis, resulting in activation of mammalian autophagy, presenting an alternative to activation of autophagy by mTOR activation using rapamycin. Information. Autophagy. Volume 2. Number 2. Pages 132 to 136. 5th month and 6th Month, 2006. Lithium, Rapamycin and Inositol enhancing mood stabilization therapies can decrease fragments of proteins participating in Huntington's pathology and enhance autophagy for therapy of autosomal dominant versions of Huntington's pathology.

IP3 receptor 2 and IP3 Receptor 3, both distribution near the apical plasma membrane requires an in place F – Actin network at the subplasmalemmal region, such that depolymerization of F – Actin by factors such as latrunculin B impairs or decreased IP3 receptor 2 and IP3 Receptor 3 distribution, while other IP3 receptors were not affected. Latrunculin B prevents essential attachment of IP3 receptors 1 and 2 to F – Actin polymers integrated into the Cytoskeleton and diminishes Ca²⁺ ability to produce Cl- current spikes and also diminishing the local Ca²⁺ signal. Information. J Cellular Sci. Volume 118. Part 5. Pages 971 to 980. March 1, 2005.

A review of the literature suggests that Tetrahydrobiopterin and L – Arginine are essential for coupled function or Nitric Oxide production by Nitric Oxide synthases generally, with exclusion of Ca²⁺ which does not seem to cause uncoupling when depleted experimentally. However, NOS enzymes are fundamentally different with eNOS and nNOS catalytically integrating Calmodulin and its four Ca²⁺ molecular load potential while iNOS constitutively exhibits calmodulin in such regard. The data here suggests that Ca²⁺, L

have an almost requisite characteristics of dysregulation phosphorylation, kinase or phosphatase activity. Dephosphorylated pRb is a foundational signal for deactivation of pRb attachment to E2F resulting in return of cellular cycle to phase 0.

The literature observes that NOS catalysis only considers Tetrahydrobiopterin and L – Arginine to be essential for coupled catalysis. However, because iNOS has Calmodulin and its 4 Ca²⁺ loading potential already constitutively integrated, compared to nNOS and eNOS and PI3K/VPS34 which must catalytically integrate Calmodulin into its structure, it is likely that iNOS can become uncoupled when Ca²⁺ is depleted, when L - arginine is depleted or when tetrahydrobiopterin is depleted. Since Ca²⁺ load by calmodulin is an initiator for NOS activity, and iNOS is able to function when Ca²⁺ levels have a reached physiological scarcity while PI3K, eNOS and nNOS are limited by calmodulins own activation pathways that sense Ca²⁺ levels, it possible that iNOS can prevail competitively because it's the only functioning NOS and among few continued functioning utilizers or Calmodulin/Ca²⁺ complexes at low physiological Ca²⁺ levels. This lead out continued function of iNOS is important because it established an adhoc expression of the enzyme in a preferred sustainable context that continues when other competitive influence have become abated, thereby potentiating movement to uncoupled pathogenic function that is linked to cascade of physiological pathogenic events and changes. Moreover, the research as irrefutable established wifi and other microwave energy influences, along with synthetic electromagnetic fields otherwise and some natural electromagnetic spectra, as being pathogenic with regard to oxidative stress, sperm and testicular damage, neuropsychiatric effects including EEG changes, apoptosis, cellular DNA damage, endocrine changes, as well as calcium overload. Voltage Gated Calcium Channel activation is clearly involved in such pathogenic influence, according to the literature, along with voltage gated ion channels generally, calcium cyclotron resonance, as well as geomagnetic magnetoreception mechanisms. These pathways of impairment don't seem foundationally different than causal nuances presented in numerous contexts here, except for how such affects have been obfuscated and obscured through misinformation, preventing decades or centuries of opportunity to understand these and mitigate these to human benefit. This conclusion is supported most by the numerous sources of iNOS which is

way that requires Ca²⁺ and assistance of calcium integrated into calmodulin – reliant protein kinase II. Integration of IP3 3 – Kinase to F – Actin occurred near dendritic spines. Particularly in neurons of hippocampal region, IP3 3 – Kinase A was found near dendritic spines of pyramidal neurons colocalized to postsynaptically with Calcium integrated calmodulin reliant protein kinase II. Information. Mechanisms of Signal Transduction|. Volume 276. Issue 40. Pages 37537 to 37546. October, 2001.

PI(3,4)P2 strongly attaches to ORP1L on the surfaces of liposomes where ORP1L senses cholesterol levels and performs as a cholesterol shuttle, providing new information about why its SHIP1, SHIP2, IPPN4A, IPPN4B coordinate isolation of PI(3,4)P2 to produce shuttle tunnel with the nucleus and the plasma membrane or membrane as completion points.

PI(3,4,5)P3 exhibition coordinates Rho and Arf GTPase activity, which organize cortical cytoskeleton by causing the exhibition of dorsal ruffles. Pleckstrin homology domain effectors such as KT and Myosin – I motor protein reshape the

– Arginine and Tetrahydrobiopterin may be essential for coupled Nitric Oxide function and the reason for Ca²⁺ being instrumental seems to be involved with its Cationic Diatomic Metal classification that presents two open acceptance locations for electrons that exhibits a more substantial gradient between Hydride's net -1 status when compared with circulating H⁺ that exhibits only a + status. Ca²⁺ is also linked with membrane transport, enabling it to readily supply the intracellular environment with substrate except when Nitric Oxide Synthase is being produced to sequester Ca²⁺ to the endoplasmic reticulum and to the extracellular environment. Information. Nitric Oxide. Volume 89. 1 August 2019. Pages 14 to 21. Information. EMBO J. 2003, Feb 17. Volume 22. 4. 766 to 775. Information J Biol Chem. 1996. Sep 13. 271. 37. 22679 to 22686. Information. Biophysical Journal. Volume 113. Pages 1956 to 1967. November 7, 2017.

SHIP1 downregulates proliferation, cellular survival, and activation of haemopoietic cellular entities, translocating to cellular members to performance such influence after extracellular signals are incurred, as well as performing such influence by hydrolyzing Phosphatidylinositol PIP3 3,4,5 species to the strong signal PIP2 3,4 species which restricts signaling and substrate while excluding other PIP3/PIP2/PtdInsP2/P3 species. PtdIns(3,4)P2 or PIP2 3,4 species. PtdIns(3,4)P2, PIP2 3,4 species signals such that these more exclusively link INPP4A to SHIP2 in the nucleus and more exclusively link INPP4B with SHIP1 at the cellular membrane, thereby inhibiting proliferation, perform as inhibitors of proliferation by downregulated PI3K/Akt signaling. These factors, therefore, exhibit therapeutic effect in oncology of breast, ovary, pancreas, esophagus,

clearly a central pathway by which these detrimental effects seemed Information. Such effects include neurological, perceptive, physiological cellular cycle, and other changes, all inclusively linked to iNOS function, particularly uncoupled iNOS which is not widely understood because of its beneficial and detrimental contexts of influence. Oncology, genetic damage, neurological disease, reproductive disorders, immune dysfunction, kidney damage, as well as electromagnetic hypersensitivity, cognitive effects, and other effects that includes tolerance mechanisms described here that prevent comprehensive awareness of numerous physiological changes, even obvious physiological changes such as swelling of epithelial areas. These factors invariably involved effects to the intricate intracellular and extracellular polarity changes and gradients required for cellular cycle, cellular migration and sensing of environment as cues to proliferate, differentiate or exhibit apoptosis. The link of iNOS and other cytokines to inhibition of PEMT foundationally links EMF influence as a concerted actor, along with choline deficiency, inflammation, viral influence, bacteria, atmospheric pollution, toxins, gravitational changes, injury and disease in abrogating the major pathway of integrating Hydride into physiology. Thus, its detrimental effect to the very foundational basis of biology and to Human physiology has already been established. The utilization of electrical fields in medical application have established and implemented in care for many decades, while emerging control of cellular migration, proliferation, particular with regard to wound healing, all reveal continue potential benefit of shielded and directly focused application of EMF, but reveal how fields of exposure to populations are able to change characteristics of cellular function, proliferation and migration. Information. Environmental Research. Volume 164. July 2018. Pages 405 to 416. Information. Biomol Ther (Seoul). 2019. May. Volume 27. Number 3. Pages 265 to 275. Information Adv Wound Care. 2014, Feb 1; Volume 3. Number 2. Pages 184 to 201.

SHIP1 and SHP2, and phosphatidylinositol species used in by these are not presented as directly integrating with calmodulin. Thus, between PI3K, iNOS, nNOS and eNOS, there is competition for calmodulin and its payload of four Ca²⁺ molecules. Phospholipase C is a typical supplier of Phosphatidylinositol, linking phosphatidylinositol with inflammation cascade and choline deficiency. The constitutively integrated calmodulin of iNOS,

macropinosomes. Dorsal ruffles cannot become macropinocytic vesicles until structural deterioration of PI(3,4,5)P₃ occurs. SHIP to produces PI(3,4)P₂ from PI(3,4,5)P₃, causing TAPP1 to be recruited to the dorsal ruffle. TAPP1 recruits actin – integrating protein syntrophin to perform cytoskeletal rearrangement. SNX can be recruited by PI(3,4,5)P₃ to detach the nascent macropinosome from the plasma membrane. INPP4 then can complete the deterioration of PI(3,4)P₂, such that PI(3)P is then able to be released as PIs through the phosphatase activities of MTM6, MTM9, which are myotubularin – associated proteins that complete the closure of the membrane ruffles as conclusion of the pinocytic process. These Phosphatases are also involved in Clathrin – mediated endocytosis. Information. Frontiers in Oncology. Volume 10. Page 360. 2020.

INPP4A inhibits neuronal deterioration resultant of N – methyl – D – aspartate type glutamate receptors NMDAR activation. Impaired or inhibited INPP4A produces deuterium of the striatum at the input nucleus of the basal ganglia, which may be associated with substantia nigra that

melanoma and others. PTEN impaired, INPP4B impaired conditions are highly pathogenic and exhibits high motility by linking PIP3 3,4,5 species with Akt2 signaling. Phosphatases dephosphorylate proteins and enzymes. Disrupting or uncoupling PI(3,4,5)P₃ accumulation from enhanced AKT signaling can be highly disruptive of oncology and pathogenic phenotype.

Inhibition of both PTEN and IPP4B are pathogenic regarding proliferation, as is inhibition or impairment of IPP4B individually.

IPP4B overexpression is linked to AML but this link includes confounders such as activation of SGK3 that is activated in correlation to increased levels of PIK3ca.

Biochem Soc Trans. 2004 Nov. 32. 5. 785 to 793.

The literature observes that modulation of the PI3K/SHIP2/PTEN pathway is pathogenically commandeered by HPC genetic products and in NAFLD pathology, its mechanism includes disruption of cellular polarity characteristics. Information. World J Hepatol. 2017, Jan 8. Volume 9. Number 1. Pages 18 to 29.

SHIP2 integrates with RhoA in a way that cause front and rear polarization of pathogenic migration of oncology affected glioma cellular entities of U251 variety. Disrupting of the SHIP2/RhoA complex as well as the GTP availability that correlates positively with SHIP2/RhoA enablement of oncological mobility, preventing such mobility. Interestingly, inhibition of SHIP2 availability, as expected, impaired the translocation of PI(3,4)P₂ species to the nucleus. Mol Biol Cell. 2012, Jul 1. Volume 23. Number 13. Pages 2593 to 2604.

provides its an advantageous position in this context, potentiating that it prevail in competitive obtainment of Ca^{2+} , potentiate that it persist when these other factors have become dysfunction, and potentiate that its conversion into uncoupled status occur when these competing factors have abated catalytic activity. The literature does not acknowledge scarcity of Ca^{2+} as an uncoupling factor, but unlike all NOS enzymes that are susceptible to Tetrahydrobiopterin deficiency and L – Arginine deficiency as uncoupling initiators, the constitutive integration of calmodulin into Ca^{2+} suggest that it might be able to be uncoupled by Ca^{2+} inadequacy or that its Isolated function in low Ca^{2+} conditions may typically cause it to become an Isolated uncoupled factor among PI3K, iNOS, nNOS and eNOS. Thus, iNOS promote sequestration of Ca^{2+} to the Endoplasmic reticulum and exclusion of Ca^{2+} from being endocytosed when it is producing Nitric Oxide, increasing turgor and perhaps volume of the cytoplasm as well as supplying the plasma membrane with nitric oxide, potentiating more complete confluence characteristics, inhibiting cellular cycle. iNOS potentiates pause in G1, linked to P53 and P21 activity, when iNOS is producing Nitric Oxide. iNOS, thus, unrelieved and being expressed for duration potentiates exhibition of uncoupled iNOS, producing superoxide that activates macrophages and enabling cellular cycle to progress to go G2/M where P27 promotes cellular survival that can be diminished by inhibiting either iNOS or inhibiting P27. P53 promotes survival of specific phenotypes of cellular entities programmed for P53 resilience, modified by Tigar rescue of these from apoptosis and modified by selection of cellular entities specifically for apoptosis. P53 adjudicative processing then potentiates increase in P21 while P21 represses cyclin/CDK activation. Increased levels of cellular stress potentiates that inflammatory pathways become more and more determinant of cellular cycle status and progression, instead of p53 programming. Increase in cellular size, pRb is increasingly phosphorylated until it become prevalently phosphorylated by cyclin/CDKs, enabling transit through G1/S phase. P27 can also promote cellular cycle pause in either G1 or G2. pRb dissociates with E2F and other positive transcriptional activators of cellular cycle progression when it becomes prevalently phosphorylated.

Cellular expression of INPP4A and nuclear translocation of INPP4A is massive, massive apoptosis signal, and produces such an outcome in tissues exhibiting nuclear INPP4A. This is only 1

exhibits neuromelanin interactive with universes level fields and which has central role in motor and cognitive behaviors. Genetic depletion of INPP4A results in remarkably observable involuntary movement disorders. The link between INPP4A include cholesterol used in the production of dolichol or other precursors of neuromelanin synthesis. Neuromelanin accumulates in aging and in Parkinson's disease as integrated with Iron because it performs as an excellent metal chelator. Excess integration with Iron, maintains the Iron in dopamine neurons and norepinephrine neurons, such that high iron or high toxin levels cause a neurodegenerative process of neuronal lysing to occur, causing release of toxic neuromelanin that induces a cascading effect to which causes other neurons to deteriorate. Information. Nature. 465. 497 to 501. 2010. Information. Progress in Neurobiology. October, 2015.

A particular study suggests that PI3K's p110 subunit impairs iNOS promoter transcriptional activation by Lipopolysaccharide induced autocrine signaling pathways. Information. J Immunol. 1999, May 15. Volume

T cellular effector function regards PI3K catalysis or processing of PIP3 phosphatidylinositol (3, 4, 5) species as being essential. 5' and 3' Inositol Polyphosphatases SHIP1 and SHIP2 perform shunting of PIP3 3,4,5 species to rare but strong signal comprised of PIP2 3,4 species. INNPP4A and INPP4B both depleted these strong PIP2 3,4 species, such that along with SHIP1 and SHIP2 these strongly modulated PI3K signaling modalities that have affinity for PIP2 3,5 phosphatidylinositol species. These suggest that shuttling of INPP4A may be occurring through strong signaling of PIP2 3,5 species in a way that results in deterministic exhibition in the nucleus or the cytosol. SHIP2 phosphorylation status is correlated to its nuclear translocation or its near plasma membrane location, suggests that SHIP1 and SHIP2 invoke a strong phosphatidylinositol species signal that galvanizes substrate strongly to either the plasma membrane or the nucleus. Phosphatidylinositol species have 31 different species configurations and are anchored to the inner leaflet of the plasma membra in way that can receive signals from ligand activation on the outer leaflet of the plasma membrane including T Cellular activation or antigen presentation/registration at CD4+ and CD8+ receptors. Some of these species are essential in activating kinase phosphorylation cascades, post translational modification of enzymes such as phospholipases and inositol phospholipases, interactions with Doks and Gabs adapter/scaffolding proteins, Pleckstrin homology PH and C2 domains of proteins perform as phosphatidylinositol species parsers that review configuration characteristics of the Inositol Ring with the C1 alcohol linking the membrane integrated acyl species to the inositol ring, C2 alcohol being protected and deactivated by its proximity to the C2 alcohol, with a

among thousands of homologues or dualities in which systems of civilizations and outcomes correlates, mimic or provide insight into biology. Information. Biochimica et Biophysica Acta - Molecular Cell Research. Volume 1865. Issue 10. Pages 1501 to 1514. October, 2018.

Providing BAX, BID, INPP4A, SHIP2, Bcl-XS, FADD Fas-associated protein with deterioration domain, TRAIL, an inhibitor of iNOS, an inhibitor of BAG3, a proteasome inhibitor (thymoproteasome or immunoproteasome for specific effect), a choline kinase inhibitor, methylsulfonylmethane, s-methylmethionine sulfonium, phosphatidylcholine, 6s 5678 tetrahydrofolate, Methylselenol or Methylselenic Acid, trimethylglycine, folic acid, B12, B6, an inhibitor of phospholipase D, sphingosine kinase 1 inhibitor, Phosphatidylethanolamine, Phosphatidylserine, A PI3K inhibitor if Phosphatidylinositol is provided, as well as selective GSK3 modulation, PD – L1 therapy, DHA, diverse omega-3 fatty acids, dimethylacetothetin, along with Ca²⁺, Tetrahydrobiopterin, L – Arginine, Vanadium, nitric oxide donor, dense methyl group resources, potentially including N – Acetyl L Cysteine, Superoxide (Mg²⁺ and Mn²⁺ or other), Catalase, therapeutically in a strategy that might include introducing one factor after another in serialized and or accumulating way, as well as potentially in a specific order or pattern, while utilize very specific nuances of therapeutics otherwise, should be very effective, widely effective and completely effective when using the factors included in this document. These should be affective managing pervasive, if not every nuance of oncology, leukemia and lymphoma. Similarly, these can be particularly effective when integrated with CRISPR gene repair to eradicate microbial, retroviral, viral, genetic inclusion in genome as well as to kinetically interact with microbes and microbial genome. Information Hepat Oncol. Volume 2. Number 4. Pages 381 to 397. October, 2015.

Diverse autophagy pathway therapeutics and modulators can be found at this url

YAP/TAZ are unable to coregulate expression of Myosin II, such that f – actin stress fiber function is impaired, this results in impaired autophagosome emergence and function. The relationship between autophagy and cellular cycle is very complex. However, a particular study links abated cellular

162. Number 10. Pages 6184 to 6190.

SHIP1 downregulated proliferation in an experimental context but genetically bereft SHIP1 conditions exhibit increased proliferation, increased M1 polarization of macrophages toward inflammatory phenotype, enhanced infiltration of neutrophils and monocytes, hyperactivity among osteoclasts, and osteoporosis confirming the canonical pathology of persistent iNOS expression that was observe to be 10 times lower in this context. However, dysregulation and upregulation of PI3K activity from impaired SHIP1 activity also would enable depletion of Calcium because Ca²⁺ integrated Calmodulin is depleted by PI3K, seemingly competing with iNOS for Ca²⁺. Upregulation of Arginase and ornithine which polarizes macrophages toward M2 phenotype may be beneficial except when occurring in a pathogenic context. It is possible that uncoupling of iNOS or depleted iNOS substrate such as Ca²⁺, iron or tetrahydrobiopterin allows Arginase to utilize L – arginine and promote pathways such collagen synthesis and growth while a highly pathogenic isolated context exists as

C2 hydroxyl linked to the inositol ring to depotentiate phosphorylation of the phosphatidylinositol by other kinases, results in only 15 possible active phosphatidylinositol species that mammalian genome does not seem to be completely utilize as far as existing research has revealed. SHIP2 location at the plasma membrane controls or diminishes PIP3 3,4,5 species by shunting to PIP2 3,4 species, while in the nucleus SHIP2 controls or diminishes PIP2 4,5 species. Information Adv Biol Regul. 2013 Jan. 53. 1. 28 to 37.

Approved PI3K inhibitors for isoforms δ and/or γ include Duvelisib (δ and γ), Idelalisib (δ) and Copanlisib (α and δ) have demonstrated efficacy in the treatment of hematological malignancies, chronic lymphocytic leukemia CLL, follicular lymphoma (FL) while PI3K- δ also performs regulation of the immune system and protection from oncology development. PI3K α inhibitor Alpelisib with the antiestrogen Fulvestran are approved for HER2 oncology with polymorphism of PI3KCA. Information. Oncology Basel. 2021. Feb. 13. 4. 890.

PIPKI γ 5 production of PtdIns(4,5)P₂ directly regulates ATG – 14 / VPS34 complex assembly in a way that presents PtdIns(4,5)P₂ direct involvement with autophagosome membrane initiation. The BATS domain of ATG14 senses the curvature of PtdIns(3)P exhibiting membranes. VPS34 is also known as PI3K Class III which performs a regulatory role for autophagy and phagocytosis in pathogenic and typical cellular function such that class II and class III PI3Ks can enable proliferation by enriching PI(3)P on an endosomal membrane to cause SGK3 recruitment where PDK-1 and mTORC2 phosphorylate SGK3, resulting in mTORC1 activation. PDK is nuance of the Sphingosine

cycle progression without contact inhibition to impaired f-actin and cytoskeleton involved amelioration which affects cellular cycle. Decreased proliferation that occurs from contact inhibition also is linked by the same study to autophagy as is confirmed by contact inhibition cellular entities exhibiting cellular pause having increased sensitivity to hypoxia and glucose deprivation. Such sensitivity exhibited by contact inhibited cellular entities is also a feature of cellular entities exhibiting autophagy. Information. Nature Communications. Volume 9, Article number 2961. 2018 Information. Scientific Reports. Volume 6. Article number 33146. 2016.

Hedgehog HH cellular signaling affects proliferation by upregulating Cyclin E and Cyclin D, thereby dephosphorylating pRb in a way that dissociates pRb from E2F and other cellular progression factors to potentiate cellular cycle progression. Hedgehog is presented as canonically requiring cellular entity to cellular entity contact to be expressed, although the literature does not suggest that this contact must constitute confluence in order for Hedgehog signaling to occurs. Information. "Oncology Origination". Volume 3. Page 112. 2014. Online Information. 10.1038/oncis.2014.27.

Experimental contexts exhibit YAP suppression of Hedgehog when cellular to cellular contacts are not exhibited. Inhibition of YAP increases Hedgehog/GLI activity while overexpression of YAP inhibits Hedgehog signaling. Hedgehog causes expression of YAP posttranscriptionally such that YAP is autoregulator or feedback regulator of Hedgehog. Strong nuclear YAP availability with low Hedgehog activity is observed in pancreatic oncology. Proteases can invert the function of YAP and HIPPO to produce pathogenic change.

Hippo regulation, control and inhibition of YAP and TAZ activity is inactive during low cellular density, such YAP/TAZ enter the nucleus to perform transactivation of their respective genetic response elements. Hippo activation cascade is invoked during increased or high cellular density, causing YAP/TAZ to become phosphorylated by the proliferation inhibitor LATS Kinase, thereby deactivating YAP/TAZ by sequestering and excluding YAP/TAZ to the Cytoplasm. Confluence or contact inhibition describes Hippo activation and sequestration as well as exclusion of YAP and TAZ outside of the nucleus, while nuclear location of YAP and TAZ is linked to proliferation and oncology. Hedgehog signaling upregulates cellular

uncoupled or impaired iNOS expression along with PI3K depletion of Ca²⁺. The exhibition of osteoporosis clearly suggest that systemic gradients can emerge regarding Ca²⁺ because of iNOS and PI3K activity, resulting depletion of Ca²⁺ from bones. Biochem Soc Trans. 2004 Nov. 32. Pt 5. 785 to 793. Online Information 10.1042/BST0320785.

SHIP1 inadequacy results in bone mass deterioration. SHIP1 dephosphorylates GSK3B at pY216, suppressing its kinase activity, while stabilizing B – Catenin. Mesenchymal Stem cellular entities' potential to differentiate into Osteoblasts or Adipocytes become impaired toward adipocytes when SHP1 is inhibited. This suggests that adipocyte synthesis and accumulation is exhibited at a potential detriment to bone density. Information. Cellular Reports. Volume 16. Pages 769 to 780. July 19, 2016.

Macrophages and Neutrophils phagocytose material and xenobiotics, as well as refuse and waste from physiology, performing monitoring and sampling role in physiology that occurs with T cellular activation, but can be enhanced by adaptive immunity. Damage

Kinase, S1P, GSK3B, S1P receptor, PDK cascade. Information. Oncology (Basel). 2021 Feb. 13. 4. 890.

SHIP2 disrupts insulin/IGF-1 signaling that results from Akt. Information Mol Endocrinol. Volume 24. Number 10. Pages 1965 to 1977. October 1, 2010.

Bone derived mast cells upregulate SHIP but not SHIP2 and not PTEN when stimulated by Lipopolysaccharide, because of their haemopoietic classification, followed by a decreased expression of SHIP in subsequent exposure to Lipopolysaccharide as a nuance of tolerance although this upregulating of SHIP results of autocrine linked TGFbeta and neutralizing immunoglobulin toward TGFbeta that produces the observed tolerance mechanism. This context clearly presents the duality of iNOS expression and its competition with PI3K for calmodulin/Ca²⁺ complexes. Lipopolysaccharide is a potent activator of iNOS through Autocrine pathways presented in this study. It is not mentioned in this context but iNOS expression and function, as substrates decrease and Ca²⁺ in particular becomes depleted, may become uncoupled iNOS or substrate bereft abated iNOS function which defines beneficial compared to pathogenic context, respectively. PI3K/VPS34 catalysis can become dysregulated and enhanced by phosphatidylinositol availability particularly PI(3,4,5)P3 but possibly by other species. SHIP1 INPP4 or SHIP2 INPP4 sequesters phosphatidylinositol species toward PI(3,4)P2 which deprives PI3K/VPS34 of substrate and also causes INPP4 to shuttle to the membrane of haemopoietic cellular entities and causes INPP4 to shuttle to the nucleus of cellular entities generally, while the accumulating PI(3,4)P2 is able to integrate into surface enzymes in liposomes to

proliferation and typically does so only when confluence or cellular to cellular contact is exhibited, such that Hedgehog automatically causes expression of its inhibitor YAP although YAP expression otherwise is also able to downregulate Hedgehog activity. YAP downregulates GLI and thus also Hedgehog/GLI pathway factors. Information/. Nature. Volume 417. Pages 299 to 304. 2002. Information. Oncology Origination. Volume 3. Page 112. 2014. Online Information 10.1038/oncis.2014.27. Information J Cellular Sci. 2014, Feb 15. Volume 127. Number 4. Pages 709 to 717.

Hypersensitivity to contact inhibition is correlated with remarkably atypical increases in duration of vital being among particular experimental contexts of small nonhuman mammals. Oncology cellular entities exhibit programming during development in which inadequate contact potentiates subsequent potential for oncology and potential to escape contact inhibition, similarly to human dissociative disorders. Future Oncol. Volume 11. Number 24. December, 2015. Pages 3253 to 3260.

Inadequate cellular density is a strong signal for proliferation. Adequate cellular density is a strong inhibitor of cellular proliferation.

The deubiquitinase YOD1 deubiquitinates ITCH, followed by enhanced ITCH ubiquitination of LATS1 and LATS2, thereby resulting in increased levels of YAP/TAZ, potentiating nuclear location of YAP/TAZ to potentiate proliferation. Managing YOD1, ITCH and YAP/TAZ presents strong ability prevent and abated oncology. BMB Rep. Volume 50. Number 6. 2017. Pages 281 to 282. 6th Month.

Associated molecular patterns as DAMPs and Pathogen Associated Molecular Patterns as PAMPs describe activation receptors on the surface and interior of Macrophages in particular. DAMPs are patterns recognized such as ATP, Hypoxia, osmolality flux, pH or Heat Shock Proteins, while PAMPs include proteins, sugars, nucleic acids and sugars often highly conversed in correlation to apoptosis, necrosis, immunologic response, inflammation, pathogens, microbes, or immunoglobulin/C Proteins, comprising innate, adaptive, cellular, humoral, complements system immunological function. The literature suggest that Heal mode is the foundational status for Macrophages, although a switch can rapidly occur. Macrophages engulf or remove debris, deteriorating cellular entities, or other material, and macrophages deplete L-arginine in local areas of pathology, explaining why deterioration of extracellular matrix occurs in diverse disease and advanced chronic pathology. A mixture of compacted granularized matrix is considered an early phase of healing which seems to be impaired because PEMT catalysis is inhibited, hyaluronic acid production is thereby inhibited, and potentially because of persistent substrate

shuttle cholesterol into the nucleus and to the cellular membrane. PI3K clearly competes with iNOS for Ca²⁺, but PI3K does not exhibit integrated Calmodulin, suggesting that when iNOS is operating in functional mode, Nitric Oxide production can prevail over PI3K/VPS34 catalysis. A study observes that phosphorylation of calmodulin promotes PI3K activation by integrating with PI3K at SH2 domains. Since SIP and Sphingosine Kinases as well as choline kinase alpha and nSMase/aSMase are major phosphorylation upregulators and inflammation pathways by cytokines and T Cellular activated pathways involve phosphorylation cascade, it can be generally regarded that disease and oncology have an almost requisite characteristics of dysregulation phosphorylation, kinase or phosphatase activity. Unphosphorylated pRb is a foundational signal for deactivation of pRb attachment to E2F resulting in return of cellular cycle to phase 0. Information. Immunity. Volume 21. Issue 2. Pages 227 239. 8th Month, 2004.

PI3Ks (class I α , β , γ , δ ; class II α , β and class III) are expressed in platelets. Class II PI3K γ is not. Managing the lesser PI3K δ and major class I PI3K β factors with regard to causing or participating in thrombosis is presented in the literature. PI3Ks, thus, are linked to upregulated of choline kinase alpha which can constitutively activate platelets by overproducing phosphocholine. Information. Adv Biol Regul. 2016 May. Volume 61. Pages 33 to 41.

L-arginine through the Arginase pathway becomes Ornithine and Urea, such that Ornithine is essential in that ornithine is required for cellular proliferation and synthesis of extracellular matrix, both being essential nuances of

bereft iNOS expression or uncoupled iNOS expression that competes with arginase pathway use of L-arginine. J Innate Immun. 2014. 6. 716 to 726.

Filamins are homodimeric proteins which cross link F Actin, with extents of about 96 amino acids usually between 24 and 6 repeats, folded link immunoglobulin, that perform as hinges in actin networks. Filamin uses this agility to perform as agile scaffolding that can be utilized by intricate molecular cascades emitted from the membrane which produce changes to the cytoskeleton. Information. Volume 31. Issue 7. Pages 411 to 419. July, 2006.

SHIP1 and SHP2, regardless of exhibition of the SH2 domain, are not presented in the literature as directly integrating with calmodulin. These explain how they function as exclusion devices that sequester Phosphatidylinositol species from PI3K, iNOS, nNOS and eNOS, although phosphatidylinositol species can cooperatively attach to pleckstrin homology domains with calmodulin. These clearly present Phosphatidylinositol species, SHIP1 and SHIP2 as modifiers

anatomical, tissue and organ regeneration. Nitric Oxide Synthases perform catalysis of 2 L-arginine, 3 NADPH, 3 H⁺, and 4 O₂ into 2 Citrulline, 2 Nitric Oxide, 4 H₂O, 3 NADP⁺ while utilizing the cofactors electron transport cofactors NADPH to FAD to FMN to HEME to O₂, and in doing so binds to FAD Flavin adenine dinucleotide, flavin mononucleotide, heme, BH₄ tetrahydrobiopterin, and calmodulin (Ca²⁺ sequester upon its 4 Ca²⁺ acceptance loci) although iNOS has calmodulin integrated into its structure and while eNOS as well as nNOS integrate calmodulin through catalytic activity. The first phase of two phases in NOS catalysis includes production of N-hydroxy-L-arginine NOHLA from L-arginine, followed by production of L-citrulline from NOHLA. This presents how inhibition of NOS can occur between the first and second phase. The completed catalysis of NOS results five electron oxidation of a guanidino nitrogen of L – Arginine. The kind of Iron utilized in NOS catalysis is Heme Iron and it is derived from animal tissue in nutrition, but Iron is stored as Ferritin in physiology. This suggests that ingestion of animal tissue can promote NOS activity such that when iNOS is expressed, this can promote continued expression of iNOS, although it is possible that the complicated catalysis of iNOS might be even more complicated from exogenous sources of Heme – iron. Bioorg Med Chem. 2002 Sep. 10. 9. 3049 to 55. iNOS, eNOS and nNOS are integral Hydridic systems processing enzymes.

The literature performs that PI3K and SHIP1 oppositely regulate activation of macrophages through Toll Like Receptor 2 and Toll Receptor 4, TLR2 and TLR4, respectively. Information. J Immunol. April, 2010. 184. 5809 to 5818.

calmodulin sequestering pathways, particularly modifying relationships between PI3K, iNOS, nNOS and eNOS. Phospholipases, particularly, Phospholipase C is an initiator of phosphatidylinositol's processing cascade by production of PI3 from phosphatidylinositol-4,5 bisphosphate. ISBN 978-0-12-134836-6.

SHIP1 is required for efficient osteoblast development from Mesenchymal stem cellular entities and promotes osteogenic orientation by inhibiting PI3K/Akt/B-catenin pathway thereby inhibiting the MSC stemness factor Id2. Stem 'Cellular Entities' and Development. Volume 23. Number 19. 2014.

PI3K/Akt regulate cellular proliferation, doing so through FOXO and GSK3beta, such that GSK3beta phosphorylation by Akt, as well as FOXO phosphorylation by Akt, downregulates potential for cellular cycle progression through GSK3beta phosphorylated deactivation of Cyclin D1 as well as FOXO inhibition of Cyclin D1 and other cellular cycle progression factors, although SHIP1 sequesters phosphatidylinositol away from PI(3,4,5)P3 which deprives Akt to essential substrate to deactivate Akt.

SGK3-PROTAC1 downregulates the PX domain exhibiting PI3K/Akt pathway factors SGK3 by 50 percent in 2 hours as well as 80% in 8 hours, circumventing resistance to PI3K Class I inhibitors as well as resistance to Akt inhibitors. Information. ACS Chem. Biol. 2019. 14. 9. Pages 2024 to 2034.

Autophagy utilizes essential complexes ULK to exchange signals with TOR, Class III PI3K/Vps34 complexes which produce PI3P metabolites used in Autophagosome emergence and development, Atg9/mAtg9 transmembrane protein which shuttles substrate for membrane remodifications between loci for Autophagosome biogenesis, Ubiquitin like Atg12 and Atg8/LC3 which perform lipidation of Atg8/LC3 with phosphatidylethanolamine to enable Autophagosome development and closure. Information. Curr Biol. 2013, Jan 7. Volume 23. Number 1. Pages 33 to 45.

These suggest that PI3K signaling can become increased and persist because of impedance to autophagosome construction, completion and function. The exhibition of 50 to 60 hz foundational fields in western civilizations is correlated to somewhat unique exhibition of prostate oncology in western civilizations linked to both iNOS or uncoupled iNOS and phospholipase D expression in luminal tissues or membranes. Phospholipase D intercepts Macroautophagy and directs it toward Mitophagy, and this may explain such persistent inhibited completion of autophagosomes and autophagy, as well as pathogenic changes to mitochondrial stability, although iNOS uncoupling can explain such mitochondrial changes also. Particular version of autophagy are utilizes to manage pathogens and

PI3K/Akt cause osteoclasts to become activated and exhibit cellular cycle progression, at least in studies of macrophages. Among Osteoclasts, SHIP1 diminishes this proliferation potential in osteoclasts, such that SHIP1 bereft organisms exhibit large hyper resorptive osteoclasts, are osteoporotic, and become polykaryons with multinucleated potential that includes resilience to programmed apoptosis. These observations somewhat precisely parallel iNOS dynamics and the effect of iNOS to Ca²⁺ availability in the Mitochondria relationship to the Endoplasmic Reticulum. Cytokine exposure and Erk1/Erk2, or P42/P44, stabilization of PI3K/Akt/GSK3 β occurs, resulting in Cyclin D factors integrating as Cyclin D/Cdk4 complexes, enhancing phosphorylation of pRb pocket protein. CSF1R is colony stimulation factor for Myeloid, bone, cellular entities and is tyrosine receptor activated kinase, of which M – CSF as well as IL 34 are macrophage orientating version of CSF1R. CSF1R informs oocyte, trophoblast, myeloid lineage, osteoclast, monocyte, macrophage, microglia, as well as dermal Langerhans cellular entity maintenance or developmental orientation. Induced and

microbes as well as proteins and enzymes produced by these.

Myosin – II performs as structural filaments that constitute cross links within Actin fibers, such that Myosin – II exhibits two heavy links, two light links and two regulatory links, in which phosphorylation of light links regulate structure and self assembly into myosin filaments. Heavy links in these nonmuscular myosin – II exhibit actin integration activity and ATPase activity in coiled coil domains' lead domains. Heavy link isoforms NM IIA, NM IIB, NM IIC, each have specific kinetic characteristics. NM II regulates cellular protrusion, adhesion and polarity through interactions with and in it actin cross links and its contractile properties. The other Isoforms modulate these characteristics also. These suggest that inhibiting or upregulating NM II as well as NM IIB and NM IIC can control cellular adhesion, migration and polarity characteristics. Information. Nature Reviews Molecular Cell Biology. Volume 10. Pages 778 to 790. 2009.

Yap can be inhibited by Verteporfin which used to remove atypical vasculature in the eye associated with macular degeneration.

AICAR can inhibit YAP. Cytochalasin D inhibits YAP and inhibits f – actin integration with cofilin, while also causing decreased survivin and preventing YAP from reaching the nucleus. Lats – In – 1 inhibits YAP, Autophagy and Mitophagy by activating AMPK. Other YAP modulators are presented in many commercial and research sites.

YOD1 suppresses MAV viral response and NEDD4 cellular cycle enablement. Cell Physiol Biochem Volume 54. Pages 1 to 14. 2020. J

<p>Embryonic Pluripotent self – renewing stem cellular survival is essentially assisted by Basic Fibroblast Growth Factor, enabling survival, stemness and self-renewal, such that PI3K/AKT regulates viability and apoptosis, with inhibition of Akt by SK690693, AKT Inhibitor VIII or AKT Inhibitor IV, all caused exhibition of Caspase 9, Caspase 3, along with PARP segmentation linked to apoptosis. Akt activation which produces inhibition of GSK3B is essential to such maintenance and survival while either inhibition of Akt or upregulation of GSK3B was able to introduce Caspase-9, Caspase-3 and PARP segmentation. Assuring AKT function sustains inducible and embryonic pluripotent stem cellular renewal while inhibition of GSK3B promotes the same, such that inhibition of AKT or upregulation of GSK3B downregulates Inducible and embryonic pluripotent stem cellular renewal. Information. Scientific Reports. Volume 6. Article Number 35660. 2016. Information. Experimental & Molecular Medicine. Volume 52. Pages 1239 to 1254. 2020. Information. Academic Press 2010. ISBN 0123756715, 9780123756718.</p> <p>PTEN expression inhibits cellular cycle</p>	<p>Immunol. Volume 202. Number 10. Pages 2957 to 2970. May 15, 2019.</p> <p>YAP and TAZ are considered among the TEAD group of reprogramming factors that program cellular entities to have increased proliferation, metabolism, resiliency, and potential for oncology. This reprogramming is similar to how parthanatos resultant of PARP signaling that depletes NAD⁺ because of PEMT inhibition that upregulates P53 to inhibit Glycolysis and Pentose Phosphate Pathway supply of Nucleotides for one million or more instances of genetic repair that must occur in every cellular entity each day. Parthanatos causes already differentiated cellular entities to exhibit apoptosis while new cellular entities are reprogrammed toward immortal status with remarkably diminished ability to experience apoptosis. TEAD group of factors have multiple classes and multiple classes of therapeutics. Some of the literature observes that YAP/TAZ must integrate with TEADs. Theranostics. Volume 10. Number 8. Pages 3622 to 3635. 2020. Genes & Diseases. Volume 8. Issue 3. Pages 241 to 249. May, 2021.</p> <p>Ischemic wounds can be rapidly healed with enhanced characteristics using Reseveratrol loaded Matrix Scaffolds. Information. International Journal of Pharmaceutics. Volume 558. Number 10. Pages 177 to 186. March, 2019.</p> <p>Injection of matrix enhanced with sLeX supports tissue, organ, muscle and structural regeneration. Information. Eur Cell Mater. Volume 24. Pages 175 to 195. September, 2012.</p> <p>The extracellular matrix protein Agrin promotes regeneration of Cardiac organs and Cardiac tissue, particularly through disruption of the dystrophin – glycoprotein</p>
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progression in G1 by upregulation P27 and cyclin G2, particularly when PI3K/Akt has emerged as primary determinant in cellular cycle progression. Information. 10.11588/heidok.00012480. www.ub.uni-heidelberg.de/archiv/12480.

SHIP1 ability to inhibit PI3K products, Akt activation and cellular survival in haemopoietic cellular entity contrasts with SHIP2 activation by M – CSF through tyrosine phosphorylation, in macrophages, subsequent SHIP2 association with M – CSF Receptor, and SHIP2 association with the Actin – Integrating Protein Filamin along localization to the cellular membrane through proline rich domain dynamics. Information. J Immunol. December 1, 2004, Volume 173. Number 11. Pages 6820 to 6830.

A study observes lamin associated domains mediate interaction between nuclear lamins and chromatin, enabling spatial arrangement of genome, particularly including Lamin A/C associated domains become distributed according to histone H2B modifications on serine 112 which perform as epigenetic markings. O linked N acetylglucosamine is attached to serine 112 in such regard being

complex along with Yap pathway signaling and Erk pathway signaling. Agrin promotes mammalian heart regeneration In Vivo. Nature. Volume 547. Number 7662. Pages 179 to 184. July 13, 2017.

Progeria is dysfunction causing aging to occur rapidly and is caused by lamin A farnesylation and subsequent segmentation of the farnesylated moiety becoming impaired such that segmentation of the farnesylated domain does not occur. Lonafarnib prevents farnesylation of lamin A, and is therapeutic for Hutchinson – Gilford disease. Information. www.medpagetoday.com. Opinion. Progeria. April 24, 2018.

IKK γ /NEMO enables NF – κ B activation, while NEMO dysfunction is linked to progeria syndromes or rapid nuances of aging. GSK3B phosphorylates NEMO at serines 8, 17, 31 and 43 within its N extremity domain, resulting in a GSK3B/NEMO complex that can be impaired by genetic polymorphism of NEMO at such phosphorylation competent Serines. Genetic polymorphism of NEMO is linked to upregulated K63 polyubiquitination of NEMO, clearly linking upregulated proteolysis with impaired protein exhibition that can be linked also to the unfolded protein response and upregulated choline kinase. Polymorphism affected NEMO was found to exhibit increased IKK α and IKK β integration, generally presenting a dysregulated or destabilized NEMO. TNF α enablement of NF – κ B was impaired and NF – κ B signaling became dysregulated when GSK3B was not available to produce the NEMO GSK3B Complex. This NEMO dysregulation is linked to progerias or aging disorders. Information. Scientific Reports. Volume 6. Article Number 38553. 2016.

known as Glucosamine Associated Domains. Adipogenic metamorphosis of progenitor cellular entities exhibit a two phase process of Lamin associated domain synthesis using Lamin A and Lamin C, which results in a Lamin Associated Domain influenced change from progenitor cellular proliferation to cellular cycle pause. Cellular differentiation status produces both repressive and activating epigenetic markings within chromatin. Before differentiation, lamin associated domains of Lamin A and Lamin C are gene rich, while after differentiation such markings resemble repressive features shared with Lamin B1 lamin associated domains. Release of Lamin A/C associated domains is presented as being upregulated through glycolysis, suggesting that functional PEMT1/PEMT2 during optimal cellular plasticity or aerobic glycolysis which emerges as cellular function deteriorates during repression of glycolysis by P53 during periods of inhibited PEMT1/PEMT2, both can produce epigenetic markings that promote change of cellular entities toward exhibition of megabase, intergenic markings upon chromatin domains such that once

Hutchinson – Gilford progeria syndrome is causally linked to genetic impairment of lamin A that results in truncated version of lamin A known as progerin. Accumulation of progerin is an integral link to disease of rapid aging and aging in a general context. Interestingly, progerin accumulation causes impaired heterochromatic epigenetic H3K27me3, along with early senescence, although functional telomerase prevents pathology from these factors. Lamin associated polypeptide – alpha interacts with lamin A in a manner that is diminished once lamin A becomes progerin. Increased levels of LAP2alpha occupies lamin A as lamin A, thereby decreasing production and accumulation of progerin. This activity of LAP2alpha produces an inhibition of cellular proliferation changes that occur with progerin accumulation as well as inhibits attrition of H3K27me3 epigenetic factors, all of which can be therapeutic for progerias including Hutchinson – Gilford progeria syndrome. Information. eLife. Volume 4. Article e07759. 2015. Online Document 10.7554/eLife.07759.

Strategies for managing lamin A include autophagy activators, restoration of IGF – 1 and GH balance, inhibitors of notch or notch signaling pathways, scavenging of reactive oxygen species, activation of telomerase or activating telomere repair function, regulation of Rb protein to counteract lamin A/Rb protein complexes that cause rapid aging in disease as well as typical aging, autophagy activation, apoptosis inhibitors, lamin A/C management using Doxorubicin or Sangivamycin or Tunicamycin/thapsigargin or paclitaxel, cAMP activation, thyroid hormone supplementation, PI3K pathway inhibitors, repair of epigenetic indicators, farnesylation

differentiation occurs, these massive epigenetic markings are typically repressive. Lamin associated domains occur, but do not occur randomly and become redistributed as a cellular entity approaches differentiation status, while release of preadipogenic markings occurs during change toward adipogenic differentiation in a way that is correlate with Glucosamin Associated Domain increases, suggesting that glucosamine associated domains determines or affects patterns in lamin associated domains upon chromatin, but most importantly these suggest that epigenetic markings upon chromatin involve and affect lamin and occur in a pattern that is correlated to the differentiation vector decided upon by a cellular entities phenotype, location and environment. Most imperatively, these suggest that PEMT1/PEMT2 status is integrally involved in divergence of a cellular entity toward differentiation. These seem to clear up the mystery of PEMT1 and PEMT2 function, since PEMT2 does not correlate with regrowth or anabolic change, although providing the same function, clearing and antihistamine function as PEMT1, suggesting its known role in maintaining

inhibitors, kinetic interactions with lamin A polymorphism products, CRISPR Gene Repair. Information. Journal of Molecular Medicine. Volume 90. Pages 1361 to 1389. 2012.

LATS1 and LATS2 restrict YAP/TAZ activity, resulting in promotion of nephron progenitor cellular differentiation in the mammalian renal organ or Kidney. YAP/TAZ expression is also known to cause regeneration of stemness in already differentiated cellular entities which can be pathogenic with oncology but which can also have a different effect therapeutically or within typical contexts. Information. J AM Soc Nephrol. Volume 28. Number 3. Pages 852 to H61.

Lamin A/C, which performs as an intermediate filamin of the inner nuclear matrix, interacts with emerin, which is a protein that is impaired in versions of Muscular Dystrophy. Impaired emerin and therefore impaired interactions between emerin and lamin A/C are causally linked to diverse conditions of progeric nature, including Cardiomyopathy with arrhythmogenic features, Dunnigan lipodystrophy, mandibuloacral dysplasia, Hutchinson – Gilford progeria, Werner syndrome, and Emery – Dreifuss dystrophy. Although these factors interact within the nuclear matrix, these factors clearly have a deterioration effect to extracellular matrix. Information. ISBN 978-0-08-045046-9.

Lamin A levels in the nucleus are correlated with structural architecture stiffness. Information. Science. Volume 341. Pages 965 to 965. 2013.

Lamin proteins exhibit arms, in a cruciform shape, which integrate with other factors, molecules and proteins in the matrix, nuclear,

organs and tissues without overgrowth. PEMT1, however, seems to be correlated with development programs implementation along with its genetic stability, environment cleansing and antihistamine functions. Although its known that PEMT inhibition produces a proteomic and epigenetic switch toward P53 cascade, there does not seem to be voluminous information that link PEMT1 and PEMT2 to epigenetic change except for the obvious accumulation of glucose, more complex proteins, and toxins, and cytokines that occur merely as a result of inhibited PEMT function. Genome Research. Volume 25. Number 12. Volume 1825 to 1835. December, 2015. Internet Information 10.1101/gr.193748.115.

The literature observes that angiosarcoma may be the most rapid version of oncology and may have highly pathogenic pattern of emergence and advancement. The foundational science observes that luminal epithelia can often exhibit a subjacent connect tissue layer, such that the basement membrane is exhibited between these two layers and performs as an enhanced platform of epithelium attachment. Lumen typically regards

organelle, and extracellular matrix, such that these form sheets or leaflets as well as integrate cellular entities to one another including integrating cellular entities into extracellular matrix or connective tissue. Lamin assists in producing the glue that integrates humanity is a unitarian physiological compartment, and it is this structural nuance of physiology that changes individual cellular entities into a being that is differentiated from all else among the Universes. This essential role of structure is presented clearly in systems of civilization in which civilization's systems clearly were intended to perform incipiently and most importantly as the structural nuance of civilization, with characteristics remarkably similar to nuances of life. The structure of lamin is also interestingly similar to the shapes of the caduceus. The assured function of civilizations systems in assurance of vital being, thus, must be managed to consciously prioritize vital being because physiology exhibits functions such as immunological function which are not always beneficent from a structural or functional context. Conscious consideration of environment, health, nutrition and behavior are essential factors in managing the physiological compartment just as these are essential in managing nuances of civilization that mimic the physiological compartment. The 15 laminins exhibit a combination of three different subdomains, which include nidogen, heparin, integrins, dystroglycan, collagen, and syndecan, among others. The compendium of research linked to this document has found that reconstitution of biological activity in animate tissue involves a pattern of cellular entities being exposed to choline, calcium and other factors that proceeds as an opening of ion gates in the cellular membrane, such that once polarity levels and ionic distribution is constituted, the ion

the open area within cavernous areas of physiology, such that luminal epithelia typically regards epithelium in cavernous areas of physiology which is typically of enhanced protection from environmental influences because of its innermost proximate location compared other areas of physiology affected by the environment.

Thus, iNOS expression and Phospholipase D expression in the Lumen is interesting because these areas exhibit the most insulation, from a tissue layer perspective. With regard to Regarding environmental influences., The exhibition of luminal Phospholipase D expression and a wider context, iNOS, expression in lumen suggest that inflammatory or toxic factors are reaching luminal areas, subverting the protective insulation of tissues, dermis, biological fluids and anatomy. Angiosarcoma potential for migration and expansion is linked to impaired basement membrane, overexpression of metalloproteinase MMP1, MMP3,, uPA or others, as well as upregulated extracellular proteolysis, all of which can have deterioration affect to luminal function and structure. Prostate oncology is

gates rapidly close, producing a capacitant compartment separate from other nuances of physiology, representing emerging capacitant nuances of being. ISBN 978-0-12-809847-9.

Heparin can complex with “heparin integrating EGF like growth factor”, such that heparin integration with EGF is essential for endothelin ability to produce vasospasm. Information. Faseb J. Volume 20. Number 11. Pages 1936 to 1938. 2006. PMID 16877529.

Heparin is among the factors used in MATH+ and HAT protocols which have been able to produce remarkably more effective intervention and therapy for hospitalized populations presented with the viral condition linked to the epidemiological patterns of 2020 and 2021. Methylprednisolone, Ascorbic Acid, Thiamine, Heparin, and Vitamin D3, melatonin, zinc, magnesium, B complex vitamins, atervastin, famotidine as well as therapeutic plasma exchange are included in the MATH+ protocol. HAT therapy includes Hydrocortisone, intravenous Vitamin C, and Thiamine. Information. Orthomolecular Medicine News Service, Orthomolecular.org December 23, 2020. Information. “What Exactly does a HAT Trick have to do with Sepsis Therapy. Vitamins and Minerals. The nutrition.org website. September 17, 2019.

EGFR ligands include amphiregulin and “heparin integrating EFG like growth factor”, such that both are upregulated in peritoneal carcinomatosis when gastric tissue is expression CXCR4. Information. Clin Oncology Res. Volume 17. Number 11. Pages 3619 to 3630. 2011. PMID 21482691.

Heparin and Heparin Sulfate inhibit Agrin enabled acetylcholine receptor focal aggregation. The

considered to be highly correlated to Western civilization, which exhibits a 50 or 60 hz foundational field along with numerous contributors to electromagnetic fields. Pervasively, the literature which focus on angiosarcoma describe the pathologies in ways that clearly present the effect magnetic, electrical or spatial influences which produce anomalies that seem like geological or weathering resultant of mechanical or electromagnetic forces.

Information ISBN 978-0-323-37712-6.

Information ISBN 978-0-323-39255-6.

Information ISBN 978-0-323-44310-4.

Information ISBN 978-0-323-39255-6.

Information ISBN 978-0-323-44307-4.

Information ISBN 978-0-323-37675-4.

Information ISBN 9778-0-323-37715-7.

The statistical low prevalence of angiosarcoma is probably less important than its linkage to radiation therapy, chronic lymphoedema, and emergence as secondary pathologies in other oncology, as well as the prevalent literature describing the characteristics of the oncology. Information. Angiosarcoma. Volume 11. Issue 10. P983 to P991. October 01, 2010.

same study found that typically the effects of Agrin are reversible.

Tripchloride or Triptolide is an inhibitor of NF – kB.

However, Tripchloride performs some of these functions and is remarkably absent form utilization in therapies from viral conditions, to oncology, sepsis and in other diverse application. A useful example is the ability of Tripchloride to impose protective neurotrophic and neurotrophic effect in dopaminergic neurons, preventing neurotoxicity from MPP+ at remarkably low availability for Tripchloride. Tripchloride causes BDNF mRNA upregulation, inhibited D amphetamine enabled rotational behavioral challenge, nearly ¾ decrease in substantia nigra dopaminergic neuron deterioration, prevented the depletion of dopamine in striatum, suggesting that it is a model therapeutic for neuronal disease, parkinson's disease, myasthenic disease generally, and status in which neurons and neurological tissue are at risk. Information. Experimental Neurology. Volume 179. Number 1. Ages 28 to 37. February, 2003. PMID 1250 4865.

Deregulation of nutrient sensing, mitochondrial dysfunction, impaired intercellular communication as in caveolae dysfunction, FOXO3 anomaly, PI3K/AKT1 dysregulation, metabolic anomaly such as in proline metabolism impairment, Ehlers – Danlos Syndrome of Proteoglycan inadequacy, along with stem cellular entity depletion, cellular senescence, Telomere attrition, genomic stability, epigenetic changes and los of proteostasis, all have been presented in these compendium of indicators in some regard. Telomere attrition is most interesting because it was found to be a function of genetic anomaly, choline inadequacy, inhibition of

<p>Lipopolysaccharide stimulated iNOS expression is diminished or inhibited by also inhibiting Phospholipase D2. Phospholipase D1 inhibition does not enhance this vector of iNOS expression, although there are numerous other vectors of iNOS expression including NF κB. Phospholipase D expression resultant of Lipopolysaccharide exhibition could be inhibited, in the study, to also inhibit S6K1, STAT3 and P42/p44 MAP phosphorylation. Phospholipase D2 was essential to STAT3 integration into the promoter region of iNOS. Phospholipase D2 thus participates in at least some fraction of iNOS expression, producing a link between luminal expression of iNOS and luminal expression of Phospholipase D. Information. Cellular Signal. Volume 22. Number 4. April, 2010. Pages 619 to 628.</p> <p>Importantly, NF – κB is known to integrate into the promoter region of Phospholipase D1, clearly suggesting that iNOS expression can be activated by NF – κB along with activation of phospholipase D1, while other pathways of iNOS expression may also contributed to iNOS expression. These explain why Phospholipase D1, Phospholipase D2 and iNOS expression are</p>	<p>PEMT, and impaired function of Telomerase Reverse Transcriptase or Alternate Lengthener of Telomeres, both of which replenish telomeres. PARP1 depletion of NAD⁺ is another factor in detrimental nuances of aging. Progerias are nuances of aging and include these factors as well as telomere attrition, Cockayne syndrome, ataxia telangiectasia, NER Genomic repair impairment and other factors. Information. “Metabolic Control of ‘Span of being.’” ‘Cellular.’ Volume 166. Issue 4. 8th Month, 11th day, 2016. Pages 802 to 821.</p> <p>Fibroblasts in small nonhuman mammals exhibiting lamin A polymorphism experience inhibited proliferation and apoptosis after gestation but not before gestation concludes, in a way that clearly links such inhibited proliferation and apoptosis to impaired ability to produce functional extracellular matrix. Inhibited production of ornithine and depletion of l – arginine by iNOS and macrophage proliferation, particularly in correlation to electricity and wireless exposure but also resultant of iNOS linked to atmospheric pollution and choline inadequacy, all seem to converge at impaired production of extracellular matrix which is also inhibited by inhibition of PEMT. However, PEMT1 is exhibited before gestation concludes and PEMT2 function in the mitochondria emerges at the conclusion of gestation or after gestation. Typical extracellular matrix exhibition is an inhibitor of cellular proliferation. The pathophysiological context exhibits inhibited Wnt signaling that occurs resultant of diminished Lef1 transcriptional activity and nuclear localization, which is a feature of progeric cellular entities and which explains deterioration of vasculature and cardiac organ function through how these relate to extracellular matrix characteristics.</p>
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linked to luminal expression of Phospholipase D. Information. Int J 'oncology'. Volume 128. Pages 805 to 816. 2011.

Particularly, expression of phospholipase D in the lumen invokes, also, expression Phospholipase Cyl which specifically catabolizes phosphatidylcholine. Phosphatidylcholine exhibits three CH₃ molecules with ratio of one hydride to two hydrogens considered to be essential to hydric, redox, and background pH levels between 7.2 and 7.6. Also, these increase the level of catalysis required for PEMT1 and PEMT2, as well as PEMT3 to maintain anti-inflammatory fraction of Phosphatidylcholine. Since phosphatidylcholine is directed toward synthesis of gastric enzymes and gastric acids. This suggests that luminal expression of phospholipase D promotes deterioration of glandular function such as pancreatic production of digestive pathway acids. However, luminal expression of phospholipase D promotes depletion of the most abundant lipids, pH stabilization, and anti-inflammatory nuances of structure. PEMT1 and PEMT2 function, similarly, can be affected by

Information. Developmental 'Cellular'. Volume 19. Issue 3. Pages 413 to 425. September 14, 2010.

Lamin A is required for 3D or 3 Dimensional assembly of chromosomes as well as maintenance of the chromosome territories within the nucleus. Information. Front Mol Biosci. Volume 8. 705595. 2021. Internet Document 10.3389/fmolb.2021.705595. PMID 34513921.

Extracellular matrix exhibits Polysaccharides and fibrous proteins, exhibited as Basement Membrane subgroup and Interstitial/Stromal Extracellular Matrix, including collagen, laminin or lamins such as lamin A or lamin C, fibronectin and hyaluronan. Each of these are known to be inhibited by PEMT downregulation. Extracellular Matrix performs a role as organ, gland or tissue bony structure. Basement Membrane extracellular matrix is exhibited underneath epithelial and endothelial cellular entities while interstitial/stromal extracellular matrix occupies areas between cellular entities. Matrix exhibits assisting factors to perform adhesions to cellular entities such as Cd44, Rhamm, discoidin domain receptors, dystoglycans, integrins, syndecans. Extracellular matrix linkages to extracellular receptors produces a signaling cascade that can reach the nuclear compartment and produce an interactive group of changes that promote cellular adhesion and cellular stability. Information. Histol Histopathol. Volume 29. Number 9. Pages 1083 to 1092. September, 2014.

Acute changes to actomyosin stress and changes to extracellular matrix elasticity result in rapid changes to Lamin A which is accompanied by DNA impairment and changes to the cellular cycle. These vectors of

phospholipase D through depletion of phosphatidylcholine. STAR Protein function can be affected by decreased levels of phosphatidylcholine. These clearly suggest that environmental or other sources of luminal expression of phospholipase D and iNOS represent a most destabilizing context which physiology does not expect to occur, such that influences which permeate tissues or otherwise are able to stimulate these inflammatory pathways in luminal areas represent asymmetric vectors of disease which physiology does not seem to expect. These present risk to humans during before, during and after conception, during gestational development, at conclusion of gestation and through other phases of being.

Wireless, Communications, Wireless Communications, Electricity fields in particular along with other sources of iNOS and Phospholipase D, such as bacterial lipopolysaccharide, choline deficiency, inhibition of PEMT, atmospheric/environmental particulate, gravitational changes, extreme low frequency or high frequency sound, cause expression of phospholipase D, iNOS and phosphatidylcholine

change, as signals from extracellular matrix into the intracellular environments as well as the nucleus complex, are perhaps substantially undersconsidered as nuances of DNA impairment, persistent DNA impairment, changes in intracellular signaling pathways, and unexplained nuances which include the recruiting of physiology's own metabolic and molecular pathways to the detriment of physiology. DNA impairment is major source of cellular senescence, particularly in cardiomyocytes, preventing cellular division, regeneration of matrix, regeneration of tissues and ameliorating indefinite sustainability of function. Actomyosin perturbation participates in nuclear deterioration of progenitor or stem cellular entities, particularly affecting Embryonic cardiomyocytes, cardiac differentiated Induced Pluripotent Stem cellular entities, and other nonmuscle cellular entities. This deterioration includes impaired recruitment of genetic repair factors, increased genetic impairment, binucleation or micronucleation, telomere depletion, enhanced rapidness of matrix deterioration by matrix specific metalloprotease MMP2. These factors are remarkably consistent not with PEMT2 canonical function because PEMT2 does not inhibit glycolysis, Pentose phosphate pathway or Krebs cycle, although, inhibition of PEMT2 is linked to overgrowth or hyperactive development as well as disease, while PEMT1 function is typically regulated by PEMT2 function. These factors seems precisely homologous to inhibition of PEMT2 and PEMT1 or exhibition of any of these. Information. Dev 'Cellular'. Volume 49. Number 6. Pages 920 to 935. June 17, 2019.

Inducible nitric oxide synthase, iNOS, is stimulated by NF kB, while NF kB is produced resultant of TNF alpha, such that this

<p>specific phospholipase C which deteriorates the major surfactant DPPC and deteriorates the major cellular membrane phospholipid phosphatidylcholine, but also causes these to be expressed in the lumina in diverse aspects of physiology, including alveolar lumina in pulmonary organs. These fields cause areas of brain and neurological pathways that stimulate coughing to be hyperactivated, disrupt lumina and alveolar luminal function, cause inflammation, apoptosis, and exhibition of freakish changes to lattices and structure in the lumina that are presented in the literature with such disturbing graphic representations that it clearly illustrates what human populations are being subjected to when these conditions are allowed to occur. Information. Journal of Molecular Biology. Volume 67. Number 1. Pages 75 to 83. Information. 1531. Number 3. Pages 222 through 229. April 30, 2001. Information. Lancet Infect Dis. Volume 20. Number 10. Pages 1135 to 1140. October, 2020. Information. BMJ. Volume 58. Number 11. Pages 1211 to 1214. 2005.</p>	<p>pathway prevents apoptosis, particularly in hepatocytes, from occurring resultant of TNF alpha or Fas apoptosis pathways. NF kB involves IKKbeta which is knknown to invoke iNOS. Inhibition of iNOS in HELA cellular lineages is known to produce apoptosis in Hela cellular entities, such that sources of iNOS expression are known to dovetail or converge in cellular survival, cellular pathology and canonical oncology phenotypes. Information. Gastroenterology. Volume 120. Number 5. Pages 1251 to 1262. April, 2001.</p> <p>The role of iNOS as an NADPH oxidase is probably inadequately represented in pathology analyses. The literature, interestingly, suggests that Superoxide O₂⁻ is constitutively produced by the function of iNOS even before uncoupling or substrate inadequacy occurs. Depletion of L – arginine and perhaps Ca²⁺ although the experimental analysis suggests that Ca²⁺ depletion results in abated iNOS function without production of O₂⁻ specifically, results in increasing production of superoxide compared to production of Nitric Oxide, resulting in increased potential for peroxynitrite synthesis as OONO⁻. NADPH oxidases also perform oxidation of NADPH to produce superoxide oxide. However, these are Boltzmann dynamics that reflect a contextual observations, particularly because iNOS oxidation of NADPH releases Hydride while NADPH oxidases similarly release Hydride, such that 2eV- for ach oxidized hydrogen atom occurs, along with correlated energy release before the energy deteriorates spin of quantum material, through molecular interactions or recapture of Hydride by reduction of a redox factor. Information. PNAS. Volume 94. Number 13. Pages 6954 to 6958. June 24, 1997.</p>
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<p>Autophagy, Xenophagy for microbes or xenobiotics, Mitophagy and Macroautophagy, Chaperone mediate Autophagy. Autophagy when reconstituted in disease can cause beneficial change and become therapeutic. Sometimes, however, autophagy is an essential escape mechanism enabling therapeutics resistance and enabling immunological resistance. Microautophagy involves lysosomal update of enzymes or proteins directly. Chaperone – Mediated autophagy involves chaperones such as Hsc-70 that across lysosome membranes after integrating with LAMP-2A receptors. Autophagy performs quality control for intracellular enzymes, proteins and processes. Autophagy involves initiation, phagophore nucleation and elongation, autophagosome construction, fusion with lysosome, autophagolysosome synthesis, and deterioration of intravesicular factors. Unc – 51 like Kinase 1, ULK1, or ATG1, complexes are considered initiators of Autophagy. Oncology. Cancers. Volume 11. Number 8. Page 1209. 8th Month, 2019.</p>	<p>Xenophagy utilizes LC3/Atg8 complexes which are recruited to ubiquitylation receptors that recognize microbes. Some microbes utilize lysosomes and autophagosomes for protection, to escape the plasma membrane's toxic interstitial spaces, and for replication. Stimulators of xenophagy include rapamycin and nutrient deprivation. Inhibitors of Xenophagy include Wortmannin. Information. Methods. Volume 75. Pages 120 to 127. March, 2015.</p>	<p>Inhibitors of Xenophagy include chloroquine, bafilomycin, and 3-methyladenine (3-MA) [32, 56–60].</p> <p>Malvide – 3 – 0 – arabinoside chloride uses AMPK, mTOR inhibitor – 8 uses FKBP12 to inhibit mTOR resulting in activation of autophagy, 4,4' – dimethoxychalcone inhibits aging process by stimulating autophagy, Norswertianin stimulates autophagy oxidative stress reliant upon Akt/mTOR, MHY1485 inhibits autophagy by suppressing lysosome/autophagosome fusion and Rapamycin use mTORC1 to activate autophagy to modulate autophagy.</p>
<p>Diverse inhibitors, activators or modulators of autophagy versions can be found at this url.</p>	<p>Mycobacterium tuberculosis inhibits autophagosome maturation while Listeria escapes autophagosomes. P62, NBR1, Optineurin and NDP52 are Xenophagy sequestration factors that LC3 and Ubiquitin can integrate with for shuttling to autophagosomes. Information. ISBN 978-0-12-394796-3. ISBN 978-0-12-812146-7</p>	<p>Rapamycin – d3 inhibits mTOR to activate autophagy but also is an immunosuppressant.</p>
<p>Combination of both Autophagy stimulators and Autophagy inhibitors together is emerging as a strategy in oncology therapy. Information. Front Pharmacol. Volume 11. Page 408. 2020.</p>	<p>Boswellic Acid induces autophagy. Scientific Reports. Volume 6. Article number 33146. 2016.</p>	<p>AUTAC4 specifically stimulates Mitophagy which is utilize in mitochondrial recycling.</p> <p>SK1-I hydrochloride is an inhibitor of SPHK1, Sphingosine Kinase 1, thereby inhibits the major source of inflammatory signaling in mammalian cellular physiology in a way that enhances autophagy but also inhibits S1P production, inhibits S1P receptor activation, and inhibits G Protein inflammation cascades including GTPase and ATPase, although SPHK1 inhibitors also potentiate upregulation of ceramide which is a foundational apoptosis signal in cellular biology.</p> <p>Ca²⁺ permeable cation TRPM2 channel activator Adenosine 5' – diphosphoribose sodium enhances autophagy and is a NAD⁺ metabolite.</p> <p>.</p> <p>.</p>
<p>Autophagy, Xenophagy for microbes or xenobiotics, Mitophagy and Macroautophagy, Chaperone mediate Autophagy. Autophagy when reconstituted in disease can cause beneficial change and become therapeutic. Sometimes, however,</p>	<p>Xenophagy utilizes LC3/Atg8 complexes which are recruited to ubiquitylation receptors that recognize microbes.</p>	<p>Inhibitors of Xenophagy include chloroquine, bafilomycin, and 3-methyladenine (3-MA) [32, 56–60].</p>

<p>autophagy is an essential escape mechanism enabling therapeutics resistance and enabling immunological resistance. Microautophagy involves lysosomallysomal update of enzymes or proteins directly. Chaperone – Mediated autophagy involves chaperones such as Hsc-70 that across lysosome membranes after integrating with LAMP-2A receptors. Autophagy performs quality control for intracellular enzymes, proteins and processes. Autophagy involves initiation, phagophore nucleation and elongation, autophagosome construction, fusion with lysosome, autophagolysosome synthesis, and deterioration of intravesicular factors. Unc – 51 like Kinase 1, ULK1, or ATG1, complexes are considered initiators of Autophagy. Oncology. Cancers. Volume 11. Number 8. Page 1209. 8th Month, 2019.</p> <p>Diverse inhibitors, activators or modulators of autophagy versions can be found at this url.</p> <p>Combination of both Autophagy stimulators and Autophagy inhibitors together is emerging as a strategy in oncology therapy. Information. Front Pharmacol. Volume 11. Page 408. 2020.</p>	<p>Some microbes utilize lysosomes and autophagosomes for protection, to escape the plasma membrane's toxic interstitial spaces, and for replication. Stimulators of xenophagy include rapamycin and nutrient deprivation. Inhibitors of Xenophagy include Wortmannin. Information. Methods. Volume 75. Pages 120 to 127. March, 2015.</p> <p>Mycobacterium tuberculosis inhibits autophagosome maturation while Listeria escapes autophagosomes. P62, NBR1, Optineurin and NDP52 are Xenophagy sequestration factors that LC3 and Ubiquitin can integrate with for shuttling to autophagosomes. Information. ISBN 978-0-12-394796-3. ISBN 978-0-12-812146-7</p> <p>Boswellic Acid induces autophagy. Scientific Reports. Volume 6. Article number 33146. 2016.</p> <p>Deregulation of nutrient sensing, mitochondrial dysfunction, impaired intercellular communication as in caveolae dysfunction, FOXO3 anomaly, PI3K/AKT1 dysregulation, metabolic anomaly such as in proline metabolism impairment, Ehlers – Danlos Syndrome of Proteoglycan inadequacy, along with stem cellular entity</p>	<p>Malvide – 3 – 0 – arabinoside chloride uses AMPK, mTOR inhibitor – 8 uses FKBP12 to inhibit mTOR resulting in activation of autophagy, 4,4' – dimethoxychalcone inhibits aging process by stimulating autophagy, Norswertianin stimulates autophagy oxidative stress reliant upon Akt/mTOR, MHY1485 inhibits autophagy by suppressing lysosome/autophagosome fusion and Rapamycin use mTORC1 to activate autophagy to modulate autophagy.</p> <p>Rapamycin – d3 inhibits mTOR to activate autophagy but also is an immunosuppressent.</p> <p>AUTAC4 specifically stimulates Mitophagy which is utilize in mitochondrial recycling.</p> <p>SK1-l hydrochloride is an inhibitor of SPHK1, Sphingosine Kinase 1, thereby inhibits the major source of inflammatory signaling in mammalian cellular physiology in a way that enhances autophagy but also inhibits S1P production, inhibits S1P receptor activation, and inhibits G Protein inflammation cascades including GTPase and ATPase, although SPHK1 inhibitors also potentiate upregulation of ceramide which is a foundational apoptosis signal in cellular biology.</p> <p>Ca2+ permeable cation TRPM2 channel activator Adenosine 5' – diphosphoribose sodium enhances autophagy and is a NAD+ metabolite.</p> <p>Progeria is dysfunction causing aging to occur rapidly and is caused by lamin A farnesylation and subsequent segmentation of the farnesylated moiety becoming impaired such that segmentation of the farnesylated domain does not occur. Lonafarnib prevents farnesylation of lamin A, and is therapeutic for Hitchinson – Guilford disease. Information.</p>
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	<p>depletion, cellular senescence, Telomere attrition, genomic stability, epigenetic changes and loss of proteostasis, all have been presented in these compendium of indicators in some regard. Telomere attrition is most interesting because it was found to be a function of genetic anomaly, choline inadequacy, inhibition of PEMT, and impaired function of Telomerase Reverse Transcriptase or Alternate Lengthener of Telomeres, both of which replenish telomeres. PARP1 depletion of NAD⁺ is another factor in detrimental nuances of aging. Progerias are nuances of aging and include these factors as well as telomere attrition, Cockayne syndrome, ataxia telangiectasia, NER Genomic repair impairment and other factors. Information. "Metabolic Control of 'Span of being.'" 'Cellular.' Volume 166. Issue 4. 8th Month, 11th day, 2016. Pages 802 to 821.</p>	<p>www.medpagetoday.com. Opinion. Progeria. April 24, 2018.</p> <p>IKKγ/NEMO enables NF – κB activation, while NEMO dysfunction is linked to progeria syndromes or rapid nuances of aging. GSK3B phosphorylates NEMO at serines 8, 17, 31 and 43 within its N extremity domain, resulting in a GSK3B/NEMO complex that can be impaired by genetic polymorphism of NEMO at such phosphorylation competent Serines. Genetic polymorphism of NEMO is linked to upregulated K63 polyubiquitination of NEMO, clearly linking upregulated proteolysis with impaired protein exhibition that can be linked also to the unfolded protein response and upregulated choline kinase. Polymorphism affected NEMO was found to exhibit increased IKKα and IKKβ integration, generally presenting a dysregulated or destabilized NEMO. TNF α enablement of NF – κB was impaired and NF – κB signaling became dysregulated when GSK3B was not available to produce the NEMO GSK3B Complex. This NEMO dysregulation is linked to progerias or aging disorders. Information. Scientific Reports. Volume 6. Article Number 38553. 2016.</p> <p>\</p>
<p>Laminin structure and nucleus translocation minimization</p> <p>Autophagy, Xenophagy for microbes or xenobiotics, Mitophagy and Macroautophagy, Chaperone mediate Autophagy.</p>	<p>Fibroblasts in small nonhuman mammals exhibiting lamin A polymorphism experience inhibited proliferation and apoptosis after gestation but not before gestation concludes, in a way that clearly links such inhibited proliferation and apoptosis to impaired ability to produce functional</p>	<p>Hutchinson – Gilford progeria syndrome is causally linked to genetic impairment of lamin A that results in truncated version of lamin A known as progerin. Accumulation of progerin is an integral link to disease of rapid aging and aging in a general context. Interestingly, progerin accumulation causes impaired heterochromatic epigenetic H3K27me3, along with early senescence, although functional telomerase prevents pathology from</p>

<p>extracellular matrix. Inhibited production of ornithine and depletion of l – arginine by iNOS and macrophage proliferation, particularly in correlation to electricity and wireless exposure but also resultant of iNOS linked to atmospheric pollution and choline inadequacy, all seem to converge at impaired production of extracellular matrix which is also inhibited by inhibition of PEMT. However, PEMT1 is exhibited before gestation concludes and PEMT2 function in the mitochondria emerges at the conclusion of gestation or after gestation. Typical extracellular matrix exhibition is an inhibitor of cellular proliferation. The pathophysiological context exhibits inhibited Wnt signaling that occurs resultant of diminished Lef1 transcriptional activity and nuclear localization, which is a feature of progeric cellular entities and which explains deterioration of vasculature and cardiac organ function through how these relate to extracellular matrix characteristics. Information. Developmental ‘Cellular’. Volume 19. Issue 3. Pages 413 to 425. September 14, 2010.</p> <p>Lamin A is required for 3D or 3 Dimensional assembly of chromosomes as well as maintenance of the</p>	<p>these factors. Lamin associated polypeptide – alpha interacts with lamin A in a manner that is diminished once lamin a becomes progerin. Increased levels of LAP2alpha occupies lamin A as lamin A, thereby decreasing production and accumulation of progerin. This activity of LAP2alpha produces an inhibition of cellular proliferation changes that occur with progerin accumulation as well as inhibits attrition of H3K27me3 epigenetic factors, all of which can be therapeutic for progerias including Hutchinson – Gilford progeria syndrome. Information. eLife. Volume 4. Article e07759. 2015. Online Document 10.7554/eLife.07759.</p> <p>Strategies for managing lamin A include autophagy activators, restoration of IGF – 1 and GH balance, inhibitors of notch or notch signaling pathways, scavenging of reactive oxygen species, activation of telomerase or activating telomere repair function, regulation of Rb protein to counteract lamin A/Rb protein complexes that cause rapid aging in disease as well as typical aging, autophagy activation, apoptosis inhibitors, lamin A/C management using Doxorubicin or Sangivamycin or Tunicamycin/thapsigargin or paclitaxel, cAMP activation, thyroid hormone supplementation, PI3K pathway inhibitors, repair of epigenetic indicators, farnesylation inhibitors, kinetic interactions with lamin A polymorphism products, CRISPR Gene Repair. Information. Journal of Molecular Medicine. Volume 90. Pages 1361 to 1389. 2012.</p> <p>LATS1 and LATS2 restrict YAP/TAZ activity, resulting in promotion of nephron progenitor cellular differentiation in the mammalian renal organ or Kidney. YAP/TAZ expression is also known to cause regeneration of stemness in already differentiated cellular entities which can be pathogenic</p>
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chromosome territories within the nucleus. Information. Front Mol Biosci. Volume 8. 705595. 2021. Internet Document 10.3389/fmolb.2021.705595. PMID 34513921.

Extracellular matrix exhibits Polysaccharides and fibrous proteins, exhibited as Basement Membrane subgroup and Interstitial/Stromal Extracellular Matrix, including collagen, laminin or lamins such as lamin A or lamin C, fibronectin and hyaluronan. Each of these are known to be inhibited by PEMT downregulation. Extracellular Matrix performs a role as organ, gland or tissue bony structure. Basement Membrane extracellular matrix is exhibited underneath epithelial and endothelial cellular entities while interstitial/stromal extracellular matrix occupies areas between cellular entities. Matrix exhibits assisting factors to perform adhesions to cellular entities such as Cd44, Rhamm, discoidin domain receptors, dystoglycans, integrins, syndecans. Extracellular matrix linkages to extracellular receptors produces a signaling cascade that can reach the nuclear compartment and produce an interactive group of changes that promote cellular adhesion and cellular stability. Information. Histol Histopathol.

with oncology but which can also have a different effect therapeutically or within typical contexts. Information. J AM Soc Nephrol. Volume 28. Number 3. Pages 852 to H61.

Lamin A/C, which performs as an intermediate filamin of the inner nuclear matrix, interacts with emerin, which is a protein that is impaired in versions of Muscular Dystrophy. Impaired emerin and therefore impaired interactions between emerin and lamin A/C are causally linked to diverse conditions of progeric nature, including Cardiomyopathy with arrhythmogenic features, Dunnigan lipodystrophy, mandibuloacral dysplasia, Hutchinson – Gilford progeria, Werner syndrome, and Emery – Dreifuss dystrophy. Although these factors interact within the nuclear matrix, these factors clearly have a deterioration effect to extracellular matrix. Information. ISBN 978-0-08-045046-9.

Lamin A levels in the nucleus are correlated with structural architecture stiffness. Information. Science. Volume 341. Pages 965 to 965. 2013.

	Volume 29. Number 9. Pages 1083 to 1092. September, 2014.	
<p>CRISPR GENE Therapy with transduction Domains for assured access to every cellular entity in physiology. This capability enables repair of genes representing escape mechanisms preventing cellular deterioration or escape mechanisms causing cellular deterioration. Similarly, this capability enables introduction of cellular senescence or preventing additional cellular division. These capabilities can be introduced by ascertaining specific molecules, genes, genetic sequences, impairments, structures or conditions in only those cellular entities require intervention, followed by introducing changes to those cellular entities. Essentially, these are pervasively constitutive of abatement of what is known as disease. The epidemiological patterns of 2020 and 2021 reveal how rapid these existing capabilities should have been enabled to come into practice and application. Many of these capabilities have utilized to experimentally eradicated viral, microbial and other disease already. Much of the technologies have now been exhibited for nearly 1 decade, several decades or even earlier.</p>	<p>CRISPR Perfect Gene Repair. Protein Transduction Therapy. Eradication of oncology affected cellular entities. Deactivation of stem cellular entities or other cellular entities affected by disease. Repair of genes that have become impaired or were impaired to cause both susceptibility and proximately cause escape into disease. Synthesis of complex proteomics with nutrients and therapies that ask cellular entities questions or assay cellular entities and then deliver specific therapeutics required for any disease, any genetic impairment, or which walk the cellular entities along developmental pathways to become reprogrammed or developed into cellular entities, structures and tissues required for sustained or reconstituted vital being.</p>	<p>CRISPR Perfect Gene Repair. Protein Transduction Therapy. Eradication of oncology affected cellular entities. Deactivation of stem cellular entities or other cellular entities affected by disease. Repair of genes that have become impaired or were impaired to cause both susceptibility and proximately cause escape into disease. Synthesis of complex proteomics with nutrients and therapies that ask cellular entities questions or assay cellular entities and then deliver specific therapeutics required for any disease, any genetic impairment, or which walk the cellular entities along developmental pathways to become reprogrammed or developed into cellular entities, structures and tissues required for sustained or reconstituted vital being.</p>
<p>The literature, data and science strongly present the ability to regenerate aspects of anatomy, stably and predictably. The mechanisms include reseeded, supplementally enabling, or therapeutically enabling regeneration matrix in organs, tissues and limbs. Similarly, utilizing and managing stem cellular entities, or assuring optimal health of mesenchymal and pluripotent stem cellular centers such as marrow. Imperatively, enabling the function of PEMT is known to promote regeneration of anatomy because inhibition of PEMT inhibits Acetyl – CoA, Hyaluronic, Lamin production, synthesis of matrix, synthesis of myelin basic protein, synthesis of ornithine, and antihistamine functions required for regenerative/pioneering anatomical development, etc. The factors which inhibit PEMT, along with increasing genetic polymorphism, produce conditions required for impaired spontaneous regeneration of anatomy.</p>	<p>Regeneration of limbs has been documented in developing populations using silver ions and in adults using extracellular matrix. Developing populations exhibit increased levels of PEMT like the Axolotl which regenerates large aspects of its anatomy. PEMT function also enables production of matrix proteins and hyaluronic acid. www.worldhealth.net/forum/topic/435/ limb</p>	<p>Fluids from aloe vera are utilized to assist in resuscitation and sustainment. Bioscaffolds and biomimetics are both as well as adaptive alloys or resorptive alloys such as magnesium are used in regenerative and reconstructive interventions. Soluble macromolecules and friction reducing polymers including slow mitotic self-renewing cellular populations to prevent contribution of surgical procedures to inflammatory processes in oncology and other interventions. Graphs, transplants, and regeneration or pioneering generation of transplant organs, tissues, or graphs have become widely applicable in</p>

The classic reference in decellularization studies that remove cellular entities from matrix of organs and are able to reseed organs with stem cellular entities in a way that causes regeneration of organs, particularly pulmonary and cardiac organs, such that these organs exhibits spontaneous rhythms outside of a functioning anatomy. The strongest conclusion is that there is required role for continued inflammatory processes and inhibited PEMT in order to stifle regenerative pathways in physiology. iNOS is among these factors, particularly through its stimulation by unshielded electricity, wireless communications, and environmental particulate. The role of biotech innovation here is clearly differential.

regeneration.

Regenerative health.

Bioregenerative sleeves are being utilized to cover and maintain areas exhibiting regeneration using matrix, stem cellular entities, tissue transplant, transplants, graphs and other factors. Porcine bladder matrix, peptides, collagen gel, and other factors are being utilized to regenerate vision tissue, limbs and other areas of anatomy. Decellularization of removed and xenogeneic organs followed by reseed using direct parenchymal injection, continuous perfusion, multistep infusion, etc.

www.nextbigfuture.com/2011/06/tissue-regeneration-with-extracellular.html.

Annual Review of Biomedical Engineering, Volume 13. July 15, 2011. Chlorella and Astaxanthin have been found to enable regeneration of large aspects of anatomy in aquatic organism and at least repair and regeneration of Human vision tissues and lense over extended duration supplementation.

Although a specially processed version of these may be required for phenylketonuria, Chlorella and Astaxanthin promote tissue regeneration and repair, including reactive molecule species management and

surgical and therapeutic procedures otherwise. Regenerative and mechanical organ function assistance capabilities have been utilized to sustain function until transplantation as well as support sustained function while regeneration of anatomical structure and function is performed. These factors have important roles in preventing pervasive nuances of abated being.

These capabilities involve intricate analysis of the deterioration processes involved in organ, tissue, and anatomical system dysbiosis. Human lymphatic centers and porcine bladder cellular entities have been utilized to perform organogenesis, stimulating the generation of organs and tissues for human physiology.

Muscle regeneration capabilities have been implemented to produce functional muscle and connective tissue. Information.

www.upmc.com/services/regenerative-medicine.

GSK3B Inhibitors have been known to promote regeneration of enamel and regrowth of removed dental structures. Tideglusib and sponges are can be utilized to promote regeneration and repair of dental structures.

Mammalian tissues can reattain developmental epigenetic phenotype and genetic expression, resulting regeneration of vision tissues and abatement of deteriorating vision, glaucoma and macular deterioration. Particularly when the genes Oct4 (also known as Pou5f1), Sox2 and Klf4 genes (OSK) are expressed. Transcriptomes reemerge which cause axonal regeneration and synthesis to occur. Expression of DNA demethylases TET1 and TET2 were essential to the function of these reactivating genes.

Information.

www.eye.hms.harvard.edu/publicati

collagen synthesis in vivo and in vitro. Oxid Med Cellular Longev. Volume 2020. Article 4946902. 2020.

Chlorella and spirulina as aquatic factors exhibit increased levels of DHA and EPA omega 3 Fatty acids which are among preferred fractions of fatty acid used in substrate and products of PEMT which prefers newly synthesized, unglycosylated, low glycosylated phosphatidylethanolamine with fatty acids that are ether linked, DHA, omega-3, oleoylate, palmitate first fatty acid in fatty acid beta oxidation, extended length arachidonate, insulation enabling. While the products of PEMT includes these factors also, such as PMME, PDME and phosphatidylcholine, PEMT integrates hydride/hydrogen ratio 1/2 methyl groups sequentially in three steps to the open locations of the nitrogen in phosphatidylethanolamine to produce enriched PMME, PDME and phosphatidylcholine. DHA and EPA are highly toxic to oncology cellular entities. The link between DHA and EPA having positive correlation with prostate oncology seems to result from foods with high DHA and EPA content such as fish oils, or other oils, as well as fish, each having a correlation with

[ons/reprogramming-recover-youthful-epigenetic-information-and-restore-vision/](#).

Injection of exosomes can also occur in this regard to assist in communications with the cellular entity. Life Sci. Volume 15. Number 249: Page 117447. May, 2020.

Information. Proc Natl Acad Sci U S A. 2015 Nov 24. Volume 112. Number 47. Pages 14452 to 14459.

Melatonin invokes miR-143-3p/Yap/Ctnnd1 pathway, resulting cardiomyocyte proliferation to repair infarct in small nonhuman mammalian organisms. Information. Acta Pharmacologica Sinica. Volume 42. ?Number 6. Pages 921 through 931. 6th Month, 2021. PMID 32839503.

Sequestration of laminin products to the nucleus is correlated to cellular and tissue stiffness, and advanced levels of stiffness in experimental models require decellularization of extracellular matrix to enable regeneration, particularly in experimental small nonhuman mammalian neonatal cardiac organs and tissue. Information. Acta Biomater. Volume 113. Pages 380 through 392. September 1, 2020. PMID 32590172.

A single instrumentation of Agrin to matrix infrastructure can promote cardiomyocyte development and guide pluripotent stem cellular migration and differentiation upon extracellular matrix structure, particular in mammalia. The literature observes a controlling role for extracellular matrix and changes to the characteristics of matrix in enabling cardiomyocyte proliferation to regenerate the infarct affected cardiac organ and affect the emerging as well as regenerating mammalian cardiac organ. PMID 28581497. Information. Nature. Volume 547.

trimethylamine-n-oxide levels resultant of less than optimal digestive pathway microflora.

Dimethylsulfoxide is known to promote tissue regeneration including when mixed with matrix or whole organism glandular supplement. This can also include topical instrumentation. Although cardiac care instances recommend 1 gram or more of DHA sometimes, more than 300 or more than 500 milligrams a day of DHA supplementation can result in inhibition PEMT by affecting gradients participating in PEMT catalytic throughput, as well as producing interactions within PEMT's sensing pathways. Information. J Clin Med. 2016 Feb. Volume 5. Number 2. Page 15. Information. Online Information 10.1007/s10555-018-9744-y. PMID 29971573.

The dystrophin glycoprotein complex is released by the extracellular matrix protein agrin, resulting in inhibition of Yap, constituting a central mechanism of enabling cardiac organ and tissue regeneration. Information. Dev 'Cellular'. Volume 42. Number 1. Pages 7 through 8. July 10, 2017. PMID 28697334.

Issue 7662. Pages 179 through 184. July 13, 2017. PMID 28581497. Information. Front Bioeng Biotechnol. Volume 8. Number 447. May 13, 2020. PMID 3247860.

The role of the Land's cycle should be strongly presented here because LPCAT/MBOAT/Acytransferases, generally, perform roles of reintegrating free fatty acid into phospholipids, particularly in membranes, thus playing in important roles in the progression of the structural crystallization phases incurred by membrane phospholipids and lipids otherwise, producing the characteristics superstructural characteristics of any tissue, organ or organism. The Lands cycle involves both phospholipases and diesterases involved in detrimental cascades, although particular noninflammatory phospholipases including versions of iPLA2 occur without requirement of detrimental cascades. Essentially, the lands cycle is comprised of freeing fatty acids and phospholipids from cellular membranes where these interact with the environment allowing DHA, EPA, Oleoyle, Palmitate, Ether Linked, Extended Length Arachidonate, all to become involved microenvironment metabolism, become changed into advanced versions of these including neuroprotectins, protectins, resolvins and maresins, all can be produced resultant of advanced processing of Omega-3 fatty acids and ablate cascading pathology, such as abatement of detrimental versions of prostaglandin, eicosanoid, thromboxane, eicosatriene, and leukotriene pathology cascades as well as many other detrimental factors including lipoxygenases. However, not all prostaglandins enable cascading detriment, and prostanoids, poxytrins, elovanoids, (R) - Resolvins, (S) - Resolvins, are all considered linked to specialized

Pro - Resolvin Mediators which resolve pathogenic cascades. Versions of these mediators include sulfido - peptide conjugated mediators which, like other versions of these mediators, methylthioglycolic acid and other factors, acquire specialized characteristics in the acute phase or cascading pathology microenvironment, derived from the molecular, ionic, atom and quantum characteristics of such microenvironment, very much being adaptive in ways that are similar to molecular therapeutics. These mediators are searched for, screened, tested and used to produce therapeutics.

Macrophages produce PCTR or Protectin Conjugates in Tissue Regeneration, Resolving Conjugates in Tissue Regeneration or RCTR, (MCTR), Maresin Conjugates in Tissue Regeneration, MCTR including glutathione, and DHA exhibition in MCTR1 as well as cysteinylglycyl MCTR2, and DHA exhibition in MCTR2 along with Cysteinyl Hydroxy DHA in MCTR3, such that MCTR is processed by Leukotriene C4 or glutathione S -Transferase Mu 4 (GSTM4), resulting in MCTR1 which then is processed by gamma - glutamyltransferase to become MCTR2, followed by dipeptidase catalysis which produces MCTR3. Sulfido - conjugates emerge similarly from catalysis or interactions incurred by resolvins RCT1, RCT2, RCT3, protectins and 17 Series Resolvins such as RCT3 to resolve pathology cascades in lymphatic tissue.

Macrophages have type 1 and type 2 versions which are determined by the balance between arginase activity compared to nitric oxide activity, both of which can be affected by availability of substrate including changes balances of L - ornithine, L- citrulline and L - Arginine , although Iron, Tetrahydrobiopterin, Vanadium, and

Ca²⁺ are also essential factors in this regard, along with NADPH, as maintainers of iNOS catalyzes in coupled phases, preventing uncoupling of nitric oxide synthase. G4 Quadraplexes, however, have emerged as factors that depleted L – Arginine toward L – Citrulline competitively with iNOS in a way that can cause iNOS to be uncoupled without promoting arginase activity, resulting in an M1 inflammatory phenotype macrophages, citrulline upregulation and locking of the cellular entity into M1 status because citrulline is efficiently recycled into L – arginine.

Macrophages produce resolvins although Macrophages can also stimulate express iNOS and stimulate iNOS expression in tissues. PCTR1, PCTR2, PCTR3, as well as RCT1, RCT2 and RCT3 can incur similar processing as MCTR1, MCTR2 and MCTR3. These provide a strong homologue to the derivatization process through which molecular therapeutics have been produced using methylthioglycolic acid, although these also present how natural processes which perform inorganic to organic phase transfer and which sequester biologically beneficial molecules from flux, racemic or abiotic phases into biological phases such as within foundational compartmentalization factors of biology or cellular entities. These are also important in understanding how and why phosphatidylethanolamine, PDME, PDME and PC produced by PEMT, along with Methylthioglycolic Acid produced by Thetin - Homocysteine methylperase, as well as Trimethylsulfonium, tetrahydrofolate, and other linked pathways, are not passive in the environment but constitute caustic and active substants that produce a place in the environment for biological systems in a way that is demonstrated by the utilization of these factors as potent industrial

waste, petroleum, chemical, and other cleaning factors utilized in some of the most challenging environmental waste or hazardous material management processes.

The literature observes Lipoxin A4, AT-LXA4, RvD1, AT-RvD1 and RVD3 to have receptors ALX/FPR2 and GPR32.

RvD1, AT-RvD1, RvD3, LXA4, AT-LXA4, RvD5 have receptors DRV1(GPR32).

RvD2 has receptor DRV2(GPR18).

RvE1 and RvE2 exhibit receptors ERV1(ChemR23) and BLT1.

Protectins such as PD1 have receptor GPR37.

DPA resolvins, however, including RvD5n-3PDa, exhibit GPR101 as a receptor.

Maresin 1, similarly, has receptors RORalpha and LGR6.

15 - lipoxygenases enable production of lipoxins, resolvins and protectins, while although Leukotrienes including LTB4 and Prostaglandins, including PGE2 and PGD2 perform as syndrome cascade enablers in acute phase while also participating in resolution of detrimental cascade and acute phase by inducing 15 - lipoxygenases to counteract the neutrophil migration which these also enable.

Specifically, Arachidonic Acid, activated in the acute phase, is programmed for SPM enabled acute phase resolution by being able to transition from acute phase activity to SPM enablement by moving from leukotriene production to lipoxin production. DHA is a signal that activates autacoid protectins, resolvins and maresins by excluding granulocytes and recruiting nonacute, and anti-inflammatory monocytes. Mast cellular entities and Macrophages engulf debris, pathogens, neutrophils, and other factors via draining lymphatic capability. Factors such as aspirin assist because these are able enhance conversion of omega-3 EPA and DHA into 18R and 17R Oxygenation products which are

	<p>precursors to Resolvin D series and Resolvin E Series.</p> <p>Polymorphonucleus neutrophils remove infiltrating pathogens during ephemeral duration of exhibition, particulate enabled by Macrophage resolution enabled SPM mediator activity, although in the extended duration exhibition of these neutrophils exhibit excessive tissue infiltration that can be diminished by hyaluronan, hyaluronic acid, although inhibition of PEMT and inhibition of glycolysis also diminishes synthesis of hyaluronan, and 16S as well as 17S Oxygenation products which are precursors for Resolvin E series and Resolvin D series. SPMS RvD3, RvD4 and RvD6 are known focus on leukocytes, activating extracellular signaling interactively with neutrophils and myocytes to enhance phagocytosis, perform antimicrobial activity and improve host defense.</p> <p>Inhibition of proteolysis potentiates macroautophagy. However, exhibition of phospholipase D from particular pathology but also resultant of electrical fields and wireless communications fields, causes Macroautophagy to become repress, resulting in exhibition of Mitophagy. The exhibition of autophagy, however, can be inadequate, producing a condition in which proteolysis is inhibited and Macroautophagy is inhibited, focusing on exorbitant mitochondrial processing through mitophagy. Depletion of Ca^{2+} by iNOS impairs Mitochondrial membrane association with endoplasmic reticulum while detrimental conditions, inadequate nutrition, iNOS and the 12, 20 or 50 or more factors. This results in the mitochondrial associated membrane being separated from the endoplasmic reticulum, such that exchange of Ca^{2+}, Phosphatidylserine, Phosphatidylethanolamine and group of other molecules and</p>
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		chemical message molecules between the Endoplasmic Reticulum and Mitochondria, all becoming deteriorated. The canonical conditions of pathology in such regard include substantial increasing impairment of PEMT2 along with potential impairment of PEMT1.
Homocysteine should be managed to between 3.7 and 7 or 6 um/L, although 3.7 um/L or lower can be increasingly optimal, with 10 um/L used as therapeutic gateway threshold that requires continued therapeutic management to lower levels. Focused therapeutic intervention of homocysteic acid, homocysteine thiolactone, s adenosyl homocysteine or homocysteine may be produce increasingly beneficial effect.	<p>A practicing health provider presents a study of 10,000 along with a control of about 10,000. presents that an average of about between 7 and 6 um/L per liter or lower, results in 500 to 1 decrease in abate being from all causes over a period of 1 decade.</p> <p>Strenuous and extreme duration exercise is known to increase homocysteine levels. Short duration moderate exercise using resistance lowers homocysteine. Walking, marching or lightly running in place for up to 200 seconds, followed by rest, then followed by angled presses against a wall or push ups for 3 groups of 10 seconds, along with squats or sitting and rising from a chair for 3 groups of 10 seconds, also using hands to provide resistant to move head and neck in each direction for 10 seconds, all provide a foundational basis for lowering homocysteine. It is important to always pause all activity before fatigue occurs. Performing a running motion with each individual leg, lifting knee high, touching the ground as the leg moves</p>	<p>If a study presents guidance or care intervention does not focus on managing homocysteine to below 6 or 7 um/L with 3.7 um/L or lower as a therapeutic objective and 10 um/L as a gating mechanism for continued therapeutic management to lower levels, then it can be obfuscating or incomplete. Therapeutics, prescription or pharmaceutical in particular, can increase homocysteine, inhibit PEMT and upregulated iNOS, suggesting that brief duration, focused use of such therapeutics along with homocysteine lowering combination therapy are essential modalities of instrumentation. Extended duration therapy must also include homocysteine lowering combination therapy, as well as a focus on understanding and alleviating incipient, empirical causality.</p>

	<p>behind physiology and then curling heel to the gluteus maximus, with 3 groups of 10 seconds for each leg, concludes a foundation for exercise. Walking and other activity can be added, as long as the recommendations provided here are always conducted safely, with supporting structures, and with a pause every time fatigue is potentiate to stabilizing energy levels, breathing, cardiac rhythm and cognition.</p>	
<p>Prebiotic, Postbiotic, Probiotic management of trimethylamine-n-oxide, leaky digestive pathway, and impaired absorption of choline which accompanies obtainment of choline, carnitine, ornithine and amino acid dense sources such as meat, chicken, eggs, fish and other factors in choline dense foods that mitigate beneficial effects of nutritional choline obtainment.</p>	<p>Product Biocomplete 3, Vital Reds, Total Restore and Primal Plants are four products which satisfy many of the Prebiotic, Postbiotic and Probiotic, and other therapeutic natural factor supplements requirement concluded by the study link to this compendium of research. The Vitamin Supplement MyKind Men's Multi or MyKind Women's Multi by M GardenOfLife presents organic NonGMO supplements that encompassing enough to compliment Biocomplete 3, Vital Reds, Total Restore and Primal Plants to nearly satisfy the supplemental requirements for therapeutics in this study. These five supplements offer a very strongly capable solution to digestive pathway and therapeutics presented in this study. Although focused management of Homocysteine is also required and specific areas of each indicator</p>	

have to be analyzed for omitted areas or areas in which the supplemental levels in these produces may be inadequate.

Strenuous and extreme duration exercise is known to increase homocysteine levels. Short duration moderate exercise using resistance lowers homocysteine. Walking, marching or lightly running in place for up to 200 seconds, followed by rest, then followed by angled presses against a wall or push ups for 3 groups of 10 seconds, along with squats or sitting and rising from a chair for 3 groups of 10 seconds, also using hands to provide resistant to move head and neck in each direction for 10 seconds, all provide a foundational basis for lowering homocysteine. It is important to always pause all activity before fatigue occurs. Performing a running motion with each individual leg, lifting knee high, touching the ground as the leg moves behind physiology and then curling heel to the gluteus maximus, with 3 groups of 10 seconds for each leg, concludes a foundation for exercise. Walking and other activity can be added, as long as the recommendations provided here are always conducted safely, with supporting structures, and with a pause every time fatigue is potentiate to stabilizing energy levels,

	breathing, cardiac rhythm and cognition.	
<p>Cholesterol, a better perspective. Steroidogenesis, or, the production of hormones and steroids including Testosterone, Estradiol, Estrone, Estriol, Estetrol, androgens, estrogens, androstenedione, and other, all are derived from aggregation of cholesterol at membranes where phospholipids such as phosphatidylcholine exhibit START Domains. START Domains exhibiting star proteins with pockets. STARD7 is known to provide shielded transport phosphatidylcholine from membranes to the mitochondria, where cholesterol is delivered from the pocket protein to the mitochondria for processing by cytochrome P450 scc. Cytochrome P450 scc produces pregnenolone from cholesterol. Pregnenolone is a choline kinase alpha inhibitor that diminishes other pathways of phosphatidylcholine synthesis to assure that PEMT specifically produces phosphatidylcholine that is enriched with omega-3, oleoylate, palmitate first fatty acid in fatty acid beta oxidation, extended length arachidonate, insulation promoting, CH3 integrated, hydride enriched, Hydride to Hydrogenation 1/2, DHA enriched. Subsequently, estradiol even activates estrogen receptor alpha and estrogen receptor beta, producing estrogen receptor alpha activation and AP1 activation that is regulated by Estrogen Receptor Beta to diminish AP1 inhibition of PEMT, to prevent overactivation of Estrogen Receptor Alpha, and, however, enable both Estrogen Receptor alpha activated and Estrogen Receptor beta activated to integrate with 13 perfect estrogen response element sequent of the estrogen response element within genome. Estrogen Receptor alpha and Estrogen Receptor beta differ by a particular number of sequences but both use AF-1 and AF-2 domains to activate estrogen response element. PEMT1, PEMT2 and PEMT3 are aspects of the Estrogen Response element. Information. STARD7I has a specific mitochondrial affinity domain while STARD7II does not and is mostly distributed in the cytoplasm at any cellular density compared to STARD7I which is mostly located at the outer mitochondrial at low cellular density as well as is located mostly in the cytoplasm when cellular density is increased. The difference between high density cellular material and low density cellular material indicates status of confluence factors and mitogenic factors. SHIP1 translocates to the nucleus and sequesters phosphatidylinositol into version PtdIns(3,4)P2, excluding PI3K catalysis from occurring in different version of Phosphatidylinositol as well as sequesters IPP4A to the nucleus. SHIP1 similarly sequesters phosphatidylinositol to version</p>	<p>Cholesterol must be managed if the source is produced through atypical unnatural hydrolyzed or hydrogenated products produce in a lab, by toxins or by detrimental pathogens as well as pathogenic processes. However, beneficial and considered to be detrimental cholesterol have roles in physiology. The hepatic organ and its tissues become dysfunction if provide only 'good' fats, resulting in compartmentalization of the hepatic organ to produce increasingly isolated function in different aspects of the organ's tissues. VLDL is considered to be detrimental, or at least LDL is considered to be detrimental. However, VLDL is secreted by functional PEMT processes and dysfunctional PEMT, at least dysfunctional PEMT2 is required in pervasive disease, aging and in enablement of disease, aging and detrimental behavior. LDL is utilized to transport triglycerides to areas where these can be used for energy or stored as fat. However, VLDL, in particular, distributes cholesterol to areas such as lymphatic centers and disease of lymphatic centers can be enabled by impaired VLDL transport and impaired access to adequate supply or</p>	<p>Cholesterol is presented as factor for therapeutic prevention, although cholesterol can comprise 87 percent of cellular membrane structure. A known practitioner, Coldwell, presents that cholesterol comprises 87 percent of membranes and that VLDL is secreted as a transport for cholesterol which is upregulated to coat, cover and protect the vascular epithelial interface to counteract the striations in such epithelium from foods which exhibit striates. Typical table salt can be among such striates, suggesting that natural, organic Pink Himalayan Sea Salt utilization can alleviate some of such striates. Hydride integrated into cellular membranes by PEMT and in redox transactions, are the most efficient sources of energy, particularly from fats or cholesterol such as phosphatidylcholine. Phosphatidylcholine decorated with low levels of glycosylation or unglycosylated versions, as well as with DHA, oleoylated, palmitate, extended length arachidonate, and omega-3, provides a good source of anti-inflammatory factors that accompanies the sequestration or fracking of hydride from membranes, phosphatidylcholines, etc. Neurons of cognitive centers prefer fats and cholesterol factors as sources of energy compared to glucose. Essentially, diabetes type 3 is emerging as a factor that is coupled with Alzheimer's disease although this type of insulin resistance is now known to be resultant of the typical pattern of mitochondrial dysfunction linked to PEMT2 inhibition, PEMT1 dysfunction, and upregulation of GSK3B that is known to affect function Glycogen Synthase Kinase function. P53 inhibition of insulin receptor, inhibition of GLUT transport of glucose from extracellular space into intracellular environment, and inhibition of</p>

PtdIns(3,4)P2 excluding PI3K catalysis using other versions of phosphatidylinositol. Both SHIP1 and SHIP2 catalysis in this required uses versions of phosphatidylinositol PtdIns(3,4)P2 that can sequester cholesterol from liposomes, suggesting that this tunneling of PtdIns(3,4)P2 to SHIP1 and SHIP2 also functions to enable transport of cholesterol to the plasma membrane and nucleus. STARD2 is another protein that enable transport of phosphatidylcholine between membranes of the cellular environment. Stard7 is overly expressed in trophoblastic neoplasms and Stard2 is over expressed in steatosis. The mitochondrial associated membrane links the mitochondria to the endoplasmic reticulum where exchange of numerous factors occurs including lipids such as phosphatidylserine, phosphatidylethanolamine, and including Ca²⁺, and Ceramide. The enzymes used in this capacity include PSS1, PSS2, PSD, PEMT2, Cytochrome P450, SMase, CerS, DES, and others. Information. Information. J Biol Chem. Volume 285. Number 10. Pages 7385 through 7365. March 5, 2010. Information. AM J Physiol Gastrointest Liver Physiol. Volume 313. Number 1. Pages G50 through G61. July 1, 2017. Information. "Overexpression of Integrin beta1 inhibits proliferation of hepatocellular carcinoma .. through preventing Skp2 – reliant deterioration of p27 which would occur through PI3K signaling." J 'cellular' biochem. Volume 102. Number 3. Pages 704 through 718. October 15, Cholesterol, a better perspective. Information. www.maybemito.com

resupply by VLDL. Constant inflammation or autoinflammatory processes can involve activation of B – Cellular entities by T – Cellular entities, depleting resources, energy and stem cellular availability in lymphatic centers, adaptive immunological centers or other centers. Natural fats, Omega-3 at least in 1 to 4 ratio with omega-6, omega-9, omega-12 can be essential in to optimal health, while laboratory or synthetic fats that are nonorganic and gmo, all increasingly potentiate confusion of physiology. Essentially, hydride to hydrogen at a ratio of 1 to 2 is a proportion derived from methyl groups even in interstellar space, such that exhibition of these phosphatidylcholine the most abundant phospholipid sustains a proportion that links humans to a homologous field extending to celestial and universes levels. Structurally imposed nuances of this hydridic to hydrogenic ratio is important in maintaining the pH of 7.2 to 7.6 essential conscious function and background biological pH use as a gradient upon which hydric redox interaction can efficiently occur. Each redox interaction exhibits 2 eV- of energy as blue light, producing a foundational hydride enabled energy field which excites animate

Glycolysis, downregulation of Pentose Phosphate pathway, resulting in glycogen cycling in which intracellular glucose is directed toward glycogen storage amyloid fibrils and retrieved directly from glycogen amyloid fibrils, provides a context of understanding the effects GSK3B inhibition of Glycogen Synthase Kinase.

Lamin and integrin a6 signaling increases AIF apoptosis inducing factor levels in cisplatin therapy, particularly alleviating neoplasm of gonad haploid reproductive cellular entities. Information. Life Science Alliance. Volume 3. Number 7. July, 2020.

Glycogen synthase converts glucosyl domains of uridine diphosphate glucose into glucose, which is then integrated into glycogen using an alpha(1-->4) glycosidic adhesion. Glycogenin initiates the de novo synthesis of glycogen to provide the oligosaccharide primer, followed by performance of glycogen synthase catalysis. Glycogen synthase 1 within muscle and tissues generally, along with hepatic Glycogen Synthase 2, both are version of Glycogen Synthase, such that extended duration adaptation to hypoxia causes expression of Glycogen Synthase 1 causes accumulation of glycogen linked impaired cardiac organs and impaired neuronal center function particularly during ischemic events.

Glucose is a multibranched glucose polymer that is used to store glucose. Increased levels of glycogen are correlate with increase levels of amyloid fibrils and tau fibrils linked to Alzheimer's disease. Insulin resistance within neurons in particular is considered to be a factor in diabetes emerging in the literature as type 3 diabetes that is linked to Alzheimer's

material sequestered into the biotic compartment, resulting in the thing humans associated with the function of life. The structural translation of hydride into ATP through hydridic transfer from NADH to NAD⁺ when the electron transport or oxidative phosphorylation pathway makes such exchange to utilized 58 percent of such energy to produce ATP while integrating about 42 percent of hydride between the phosphate groups of ATP, represents among the most efficient ways of seeding metabolism with substrate because this pathway prioritizes maintain of physiological structure and the biotic compartment, as well as enables stable source of ATP to be directed as required into GTP, UDP, pyruvate. Glucose, 5 carbon sugars, nucleotides, NADPH, RNA, DNA,

krebs cycle metabolism, acetyl – CoA, amines, or other factors. Much of the nutritional, medicinal, supplemental and other guidance merely supports the observation that managing input of energy and factors through less empirical contexts or pathways requires understanding, therapeutics or acknowledgement of a priority for the most

disease. Information. Neuron. Volume 96. Issue 1. Pages 115 through 129. September 27, 2017. Information. Rejuvenation Research. Volume 11. Number 2. Pages 365 through 369. May, 2008. Information. ACS Omega. Volume 6. Article 21960. 2021.

Amylase frees glucose or carbohydrate from Starch fibers and glycogen branches.

P53 inhibition of Glucose endocytosis also occurs resultant of constriction of the caveolae which occurs with inhibited PEMT and with iNOS depletion of Ca²⁺ that deprives eNOS or nNOS of Ca²⁺. eNOS, at least, is active in the caveolae and promotes dilation of the caveolae when functional.

Accumulation of sugar in circulation resultant of impaired endocytosis of glucose results in activation of insulin release at hyperactive levels in the pancreatic Beta Cellular entities as well as the same cellular entities in aspects of Renal and Hepatic tissue. Pancreatic release of Glucagon occurs when levels of circulating glucose become too low. Glucagon activates Glycogenolysis while Insulin inhibits Glycogenolysis. Glycogen polymers and inorganic Phosphate are processed bidirectionally into Glycogen polymers diminished by 1 molecule of glucose and one molecule of glucose 1 phosphate by glycogen phosphorylase by substitution of a phosphoryl group within the glycogen branch being segmented by replacement with alpha (1→4) linkage. Phosphoglucomutase processes glucose-1-phosphate into glucose-6-phosphate. Glucose are released through phosphorolysis from glycogen branches until four residues before an alpha(1→6) branch that is hydrolyzed by an alpha(1→6) glucosidase, such that the concluding glucose in an individual branch is removed in a

empirical pathways of deriving essential physiologically requisite factors. Protecting these empirical pathways from deterioration by electricity, wireless communications, environmental toxins or particulate, satellite influence, electromagnetic energy and other factors is a priority because these can affect PEMT, and enabled expression of iNOS, as well as luminal iNOS and phospholipase D.

The literature suggests that sources of Casein A2 are better for health than Casein A1. Casein Kinase 2 is an acid amino acid metabolizing enzyme. Here, the information becomes very interesting.

Lutheran Blood Group and Basal cellular adhesion molecule B-CAM antigens are exhibited upon glycoproteins Lu and Lu(v13). These are two among many of the Ig supergroup Lu and Lu(v13) differ by exhibition of a cytoplasmic tail. LU and B-Cam glycoproteins are laminin alpha5 receptors, and are found on red blood cellular entities as well epithelial cellular entities in particular diverse tissue types. Sick Cell Condition affected red blood cellular entities adhere to laminin alpha6 with enhanced affinity particularly when cAMP

way removes the branch and such that the other 3 of the glucose moieties are moved to another branch by Glycogen Debranching enzyme.

Myocytes, muscle cellular entities, utilize glucose-6-phosphate derived from glycogenolysis for energy while hepatocytes utilize glycogenolysis for secretion into circulation. GLUT2 enables effusion of Glucose after glucose-6-phosphatase enables freeing of the phosphate group from Glucose-6-phosphate.

Glucose-6-phosphate activates glycogen synthase while phosphorylation deactivates glucose-6-phosphate such as Protein Kinase A phosphorylation at Site 1a or 1b, AMPK phosphorylation of site 2 Serine 7, Casein Kinase 2 phosphorylation of site 2a or Serine 10, GSK3 phosphorylation of site 2a, 3a, 3b, 3c 3d or 4 which are serines 641, 645, 649, 653 or 727.

Casein 2 can participate in cellular proliferation or cellular growth as well as prevention of apoptosis. Casein 2 phosphorylates sites adjacent to activation sites of caspases during the G2 to 1 and G2 to M phase transitions. Casein Kinase also phosphorylates MAPK and P53. Casein Kinase 2 is upregulated in oncology of prostate, breast, lung, and colon.

Casein Kinase 2 inhibition mildly inhibits dendritic oncology cellular entities as well as substantially inhibits polymorphonuclear myeloid derived suppressor cellular oncology and substantially inhibits macrophages associated with oncology. Casein Kinase 2 inhibition downregulated of CCAAT-enhancer integrating protein – alphas well as upregulation of antineoplasm activity of anti-CTLA-4 antibody.

is upregulated resultant of Epinephrine or other mechanisms, although the epinephrine mechanism is reliant upon protein kinase A and Lu/B-Cam. Increased levels of phosphorylation are observed in Madin-Darby Kidney, Erythroleukemia K 562 and sickle cellular red cellular entities in a way that can be amplified by phosphorylation of Lu glycoprotein on serine 621 by protein kinase A, on serine 598 by Casein Kinase II, and on serine 596 by GSK3B, while alanine substitutions on serine 596 and serine 598 abrogate amplifying phosphorylation by GSK3B and Casein Kinase II. K562 cellular entity adhesion to laminin under flow conditions were not decreased by alanine substitutions. Epinephrin was observed as a strong upregulator of Lu glycoprotein phosphorylation that results in adhesion to laminin. Managing the phosphorylation status of Lu gp by alanine substations or by inhibiting milk ingestion, GSK3B, Casein Kinase 2, or protein kinase A, all may be therapeutic for sickle cellular conditions. Information. J Biol Chem. Volume 290. Number 34. Pages 30055 to 30062. 8th Month, 2005.

Casein Kinase 2 is a kinase and promotes

Information. Oncology Research. Volume 18. Number 19. Pages 5644 through 5655.

Inhibition of casein kinase 2 also inhibits NF kB, such that inhibition of head and neck squamous cellular carcinoma also occurs. Information. Oncology Research. Volume 66. Number 13. Pages 6722 through 6731. July 1, 2006. PMID 16818647.

Emodin inhibits Casein Kinase 2 and results in sensitization of oncology cellular entities to Fas Ligand, TNF apoptosis inducing ligand (TRAIL) apoptosis pathways. This effect was observed in HELA and Hepatocellular carcinoma cellular entities. Casein Kinase 2 inhibition enhanced Natural cytotoxic T-cellular entity activity. 4,5,6,7-tetrabromobenzotriazole also exhibit the therapeutic effect observed with emodin instrumentation. Clinical Exp Immunol. Volume 152. Number 2. Pages 336 through 344. May, 2008.

Casein is phosphoprotein often found in milk, with sheep and buffalo having higher casein content than other versions, although some of the literature suggests that bovine milk exhibits A1 casein which is less beneficial than A2 casein of goat, sheep and ox. This is known to be only a distinction when the cows are A1 genetic variety instead of A2 which makes milk most similar to cows, goat, sheep and oxen.

Glycogen Synthase Kinase 3 is activated by exhibition of Glucose 6 Phosphate thereby competing with glucose 6 phosphate dehydrogenase.

Casein Kinase II and GSK3B both phosphorylate PTEN and have a role in leukemia such that inhibition of these may benefit leukemia therapy. Casein Kinase II also phosphorylates Ikaros. Phosphorylation of PTEN promotes

phosphorylation cascades or participates in phosphorylation cascades. It is linked to growth, proliferation and survival. Having two catalytic subunits alpha and alpha as well as to regulatory Beta subunits. Its expression is typically upregulated in oncology such as acute myeloid leukemia. MRP1 transport activity is increased by its phosphorylation by casein kinase 2 at threonine 249 increases doxorubicin efflux performed by ABCC1/MRP1 complexes. Adult AML disease typically exhibits upregulated casein kinase II while increased expression is correlated with lower disease levels and improved outcomes and while lower expression is linked to higher disease, but selective inhibition of casein kinase II by apigenin or tetrabromobenzotriazole prefers acting upon cellular entities with higher casein kinase 2alpha expression. Casein Kinase 2 is also known to destabilize activation of B2-Adrenergic Receptors. Information. Advances in Oncology Research. Volume 125. Pages 171 through 196. 2015.

Casein exhibits 13 or more isoforms of which A1 and A2 are most studied because A1 and A2 differ by a proline juncture compared to histidine juncture, and A2 is noninflammatory

PI3K signaling pathway. Phosphorylation of PTEN results in downregulation of its catalytic activity and increased susceptibility to USP7 deubiquitylation and export from the nucleus, preventing PTEN from protecting P53 from deubiquitylation by MDM2. PTEN downregulation also diminishes PINK1 levels such that recycling of impaired mitochondria is downregulated, potentiating canonical conditions for oncology in which impaired mitochondria persist without recycling in a way that can cause mitochondrial apoptosis pathways from being effectively implemented. Information. The context of PTEN inhibition exploration also revealed that free fatty acids, supplemental or resultant of inflammation, activates the rapamycin (mTOR)/S6K pathway such that S6K phosphorylates PTEN at serine 380 in a way that decreased acetylation of P53, decreased transcription of P53, reduced P53 enabled transcription of glutathione peroxidase to promote Reactive Oxygen Species accumulation and impairment of endothelium. Hight Fat atherogenic nutritional regimens promoted this inflammatory context. DHA also inhibited PTEN but DHA also downregulates NF kB and like the Fish Oil supplementation regimen also inhibits PI3K and AKT suggesting that DHA maintains its antiinflammation pattern although an enhanced version of DHA docosaheaxaenoic acid monoacylglyceride (MAG-DHA) decreases PI3K, decreases AKT, inhibits COX2 and enhances PTEN expression, producing an improved beneficial pattern in cellular proliferation stability that enables mitochondria to be recycled to promote exhibition of potent mitochondrial apoptosis pathways. The downregulation of PTEN, however, linked to reactive oxygen species upregulation is observed in neuroprotection and innervation of Spinal Cord interneurons and

<p>compared to A1. Until about the last 10,000 years, A2 was the only version of bovine casein, followed by a spontaneous polymorphism cause the substation resulting in North American, European and western civilizations located in nonwestern locations to produce A1 more than likely resultant of selective adaptation promoted by industrialization, volume and focus revenue obtainment priorities in such civilizations. A2 alleviates inflammatory digestive pathway syndromes linked to milk metabolism while nutritional regimen and vitamin D obtainment through sunlight as well as through nutrition, environment and selenium levels in the environment all may have influenced selection of bovine lineages most useful in particular locations. Samoa, Iceland, Africa and some Asian contexts exhibit A2 bovine milk, although true bovine milk is regarded as milk from Sheep, Goat and Water Buffalo.</p> <p>The activity of Casein Kinase II including its metabolisms of Casein A1 and possibly other Casein versions, including other acid amino acids, provides and explanation of how and why amino acid defined and choline inadequate nutritional regimens consistently</p>	<p>innervation of Motor Neurons to improve recovery after impairment or injury to spinal cord, suggesting that PTEN inhibition is intended to promote proliferation in regeneration and repair while upregulation of PTEN may function to enable stability. Information. Journal of Neuroscience. Volume 35. Issue 37. September 16, 2015. Information. Antioxid Redox Signal. Volume 20. Number 9. Pages 1382 through 1895. March 20, 2014. Information. Recent Patents on Anti Oncology Drug Discovery. Volume 8. Issue 3. 2013. Breast 'atypical proliferation' Res Treat. Volume 118. Number 1. Pages 213 through 228. November, 2009. Information. Advances in Biological Regulation. Volume 65. Pages 16 through 25. 8th Month 2017.</p> <p>Metabolism of Casein A1 results in release of beta-casomorphin 7 which has affinity for opioid receptors which transit the inflamed digestive pathway into circulation with detrimental effects. BCM7 can be toxic to the permeable digestive pathway of developing humans in the early years and moments of being. DPP4 or dipeptidyl peptidase 4, is the only enzymatic pathway for deteriorating BCM7 with conditions such as SIDS, type 1 diabetes, cardiac disease, food allergies, delayed development and autism all being linked to BCM7 and ability to metabolize BCM7 efficiently. Eczema and Asthma both are potentiated from A1 Casein. A2 exhibits proline in the 67th position of casein while A1 exhibits other factors such as histidine. The literature observes a remarkable increase histamine release with A1 casein. A very early study clearly links tissues and circulating histamine levels with levels of histidine. Information. Br J Pharmacol. Volume 34. Number 3. Pages 551 through 563. November, 1968.</p>
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produce oncology in experimental conditions. However, amino acid supplementation also directs available pyruvate toward alanine and production of alpha keto acid. Information. Ann Neurosci. Volume 22. Number 4. Pages 239 through 243. October, 2015.

Regarding cellular metabolism, medium chain triglycerides are also a source of energy and directly move to the mitochondria to prevent lipid peroxidation but, however, the literature does not present that an improvement in endoplasmic reticulum to mitochondria exchange of phosphatidylethanolamine, phosphatidylserine and Ca^{2+} also occurs, suggesting that an overly resilient but dissociated and dysfunctional mitochondria might result of medium chain triglyceride instrumentation without assuring improvement of the mitochondrial associated membrane characteristics.

Casein can be removed from milk such as skim milk. However, the literature does not suggest that Casein can be inactivated similarly to how lectins can be deactivated by pressure cooking. Similarly, meat, chicken, eggs and fish can be pressure cooked or granularized to improve digestion and increased possibility of

A review of the literature presents that a phospholipase D version which activates phospholipase C versions specific to phosphatidylcholine catabolism, exhibits histidines at locations 29, 125, 133 and 158, and these histidines are able to be substituted, particularly indicating that high use of histidine in histamine production from inflammation, xenobiotic, or allergic response may result in histidine decreases and potentiate epigenetic changes to gene transcription products. However, the phospholipase D version is known to cause expression of Phospholipase C version specific to phosphatidylcholine catabolism and also specific to reproductive activation of Ca^{2+} signal changes that enable oocyte activation by spermatozoa. Substitution of depleted histidine can occur in this version of phospholipase c, such that proline is exhibited instead. Histidine to proline substitution in phospholipase C impaired reproductive capabilities. Histidine to asparagine, likewise, promoted deterioration of phospholipase D and activation of phospholipase C while Cysteine substitutions did not abrogate catalytic function of phospholipase D in a way that suggests Cysteine is able integrate with inorganic Zn^{2+} similar to Histidine to enable phospholipase D catalysis. The data suggests that histamine release increases or depletion of histidine can result in proline substitutions, luminal expression of phospholipase D and phospholipase c versions preferring phosphatidylcholine can also additionally promote depletion of histidine, these can result in conditional promotion of substitutions. Milk production in at least some mammals occurs in the alveolus unit of the mammary gland which exhibits a single layer of epithelial secretory cellular entities that encompass the central storage are known as the lumen. These clearly suggest that iNOS and

removing remnants from the digestive pathway.	<p>phospholipase D pathway expression in luminal tissues explains western civilization organisms that produce milk having increased A1 beta casein which exhibits histidine at position 209 instead of proline at 209. L-arginine, Arginase activity, L-ornithine, glutamate gamma semialdehyde and NADPH are in the pathway of proline production in the prospectively presented pathway of lactation synthesis of proline. iNOS, iNOS resultant of phospholipase D, and inhibition of glycolysis and pentose phosphate pathway by P53, all deteriorate NADPH, L-Arginine, Ornithine, and Glutamate availability as precursors. This suggest that inflammation, exposure to wireless communication, electricity fields, artificial electromagnetic fields including artificial light, distress, atmospheric or environmental particulate. It is likely that change in solar electromagnetic fields, universes level electromagnetic fields, nutritional scarcity, geological, or other event may have coincided with exhibition of A1 beta casein in milk. Increase in amino acid availability potentiates an increased level of transcription of variants having that amino acid as an alternative version of a protein. The quantum nature of electromagnetic influence, potentiates that civilizations having higher levels of electromagnetic energy exposure may also have such exposure affect future and antecedent eras, explaining why earlier populations seem to have been affected by the quantum aspects of electromagnetic energy. Information. Biochem J. Volume 391. Part 2. October 15, 2005. PMC1276926. Information. Human Reproduction. Volume 26. Issue 12. December 2011. Pages 3372 through 3387. Information. Fertility and Sterility. Volume 98. Number 2. Paes 423 through 431. May, 2012. Information. www.uoguelph.ca/foodscience/book</p>
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		<p>-page/milk-biosynthesis. Information. Journal of Dairy Science. Volume 80. Number 12. Pages 3241 through 3248. January, 1998.</p>
Fructose ParadoxUnderstanding mechanisms of systemic imposition of human outcomes	<p>Civilizations exhibit the potential that individual, organizations or systems obtain benefit from exhibition of detrimental human outcomes. Civilizations that do not freely provide assure obtained of shelter, economic resources to reobtain the level of freely accessible resources in nature that are no longer available because these have been sequestered away by civilization for obtainment only at a cost, water, nutritional substance, factors that alleviate homocysteine, factors providing hydride/hydrogen at adequate ratios, choline and phosphatidylcholine, nutrients otherwise, housing, and satisfaction of the human, social, behavioral and physiological condition, exhibit an enhanced ability to obtain benefit from exhibition of human outcomes and become causal to production of detrimental outcomes in a ways that enable economic, social, behavioral, or other benefit to be obtained from detrimental outcomes.</p> <p>The canonical operation of this system includes omitting incipient and empirical causality such</p>	<p>The potential to obtain benefit from detrimental physiological, behavioral, or social outcomes, explains why pervasive therapeutics do not resolve causality or provide solutions at empirical levels, often merely moving outcomes into other areas of disease, behavior, or conditions from which even more economic or other benefit can be obtained. Therapeutics, intervention, and solutions often involve deprivation of vital being which when not utilized has been shown to increase lifespan as well as increase yearly survival rates between 20000 and 1 million or more with correlative benefit among global populations. Therapeutics, intervention and solutions can also include excision or surgical removal of an outcome without resolving incipient or empirical causality. Therapeutics, interventions or solutions can involve deprivation of Liberty while not acknowledging participation of systems in producing detrimental outcomes. The canonical example are social constructs preventing the utilization of reproductive rights for maternal carriers in which restricting of individual assertion of self-determination by a maternal carrier has been periodically moved to the social and political agenda as essential to human rights assurance to gestational populations while exhibition of abated vital being as a sanction is maintained and, while, essentially, all of the reasons for which a maternal carrier might consider discontinuation of a gestational nuance are omitted from acknowledgement, understanding, consideration and the social/political agenda. Instead of providing clear understanding of the systemically imposed influences,</p>

as massive import and distribution of artifacts specifically produced for humans to impart detriment to each other, allowed exhibition of destituteness and homelessness, omitting of PEMT and homocysteine as therapeutic and diagnostic priorities, gender bias, age bias, social bias, ethnic bias, and other bias, all in a way that systemically identifies an advanced outcome that emerges in these contexts as a symptom or anomaly at physiological, social or behavioral levels in a way that allows such outcomes to continue to occur, allowing benefit to be obtained from such outcomes, and causing the causal factors to such outcome to persist in a way that new or other existing detrimental outcomes from which benefit might be obtained are potentiated.

Thus, as movement or redirection between social, economic, physiological, behavioral, systemic contexts and outcomes occurs, increasingly diminished possible constructive alternative individual management of these causal factors occur, resulting in substantially detrimental behaviors, substantially advanced diseases or pathologies, and increasingly pathogenic outcomes or events linked to aging, stigmatization or abated

factors and conditions involved assertion of reproductive rights, civilizations can try to assert the rights of gestational developing humans from ideology without also applying reason, such that civilizations are able to impose enhanced burden upon humans, more assuredly produce detrimental human outcomes for which benefit might be obtain, and prevent acknowledging the myriad linked and homologous human condition factors exhibited within civilizations that systems of civilizations are integrally causal to. Essentially, this context is similar to a person deciding not to eat food because one meal among countless different meals, at one place, at one instance with a few among billions or trillions of recipes might have been disfavorable to them, all exhibiting how reproductive rights assertion can be window into the wider interdependent inadequacies of systems that reflected in emergency room outcomes, deprivation of liberty, deprivation of vital being, disease, and behavior.

Reproductive rights, although assurance of vital being in this context is as commendable as in any other context, are convenient context to impose falsely ideal nuances of ideology without acknowledging causal factors that are largely being imposed by systems endeavoring to impose control in these contexts. Family medical leave, adequate family medical leave, assured healthcare, requiring that the cause of detrimental health and behavior be alleviated at metabolic, economic, social and behavioral levels, bias at gender, social or other levels, as well as exhibition of systemic nuances of deprivation in these contexts, all are omitted to apply falsely binary, unobjective, and cascadingly detrimental solution that has been already observed to cause substantial decreases in abated being. Additionally, therapeutics, interventions and

being occur. All such outcomes are allowed to occur while benefit is allowed to be obtained from exhibition of such occurrence. Thus, often social constructs involved in affecting such outcomes can often proportion to have the priority of assuring life but are conceptually and in implementation typically a political interaction that is determining which individual, organization or system is to benefit most from imposing such social construct, depriving liberty or vital being as a sanction linked to such social construct, all while omitting the ways in which systems are integrally participating in producing or causal to producing such outcomes.

The literature links fructose with diabetes and unhealthy anatomical mass, particularly because fructose may be directed toward triglyceride synthesis and storage. The recommendations in the popular information recommend against eating fruit unless it is out of season, although pervasively the phytonutrients are considered to be essential. Some information recommends fruit ingestion only when it is in season.

Nutritional obtainment of phospholipids is known to prevent

solutions can cause iNOS, upregulation of homocysteine, PEMT inhibition, economic distress and other conditions that not only omit incipient causality, not only move outcomes into other areas of detriment, but increase risk factors associated with detrimental physiological outcomes otherwise, detrimental behavior, deterioration of socioeconomic status and decreased span of vital being.

This context presents that endeavoring to favor maternal carrier or gestationally developing humans in social constructs regarding reproductive rights are typical in producing falsely objective and falsely ideal outcomes unless all of the reasons for which a maternal carrier might choose to discontinue a gestational circumstance are analyzed, understood, acknowledged, and systemically alleviated, managed, prevented and mitigated are included as priorities, first. Mitigating reproductive rights without such encompassing analysis, understanding and action, merely allows linked factors among all human populations to persist as unacknowledged and unmanaged factors, while also causing assurance of gestational being by mitigating maternal carrier reproductive rights assurance to merely become a way of assuring that more humans survive gestation to have their lives and maternal carriers' lives used as substrate to assure sustainment of systems that allow and obtained benefit from exhibition of detrimental human outcomes. These suggest that the priority of sustaining all gestational nuances of being, which is empirically a commendable priority, is not derived from priority of humanity or priority of vital being but is derived from the economic potential which might be realized from allowing these nuances of vital being to emerge comparative to gestational carriers which may have

fructose from causing dislipidemia. Fructose enabled increases in lipogenic enzymes such as SREBP1, ChREBP, and other enzymes enabling increased production of hepatic triglyceride, diglycerides, ceramides and oleates, all were inhibited by nutritional phospholipid obtainment. The literature observes also that PEMT and GNMT in experimental small nonhuman mammals are inhibited after feeding, increasing S-Adenosyl Methionine, inhibiting phosphatidylcholine synthesis, although ablated expression of FGF15/19 and ablated expression of SHP both prevented downregulation of PEMT and GNMT. Ablated expression of FGF19 diminishes bile acid synthesis in a way that counteracts loss of phosphatidylcholine when producing bile acid enabled by MDR2 to prevent an aggregate loss of phosphatidylcholine when obtaining a low choline nutritional regimen, while FGF15 is known to enable regeneration and repair of hepatic tissues as well as enabled glycogen storage and glucose tolerance. FGF19 also interacts with the Farnesoid X Receptor FXR, integrating with FGFR/B-Klotho complex to inhibit Cyp7a1. FGF is a fibroblast growth factor.

already been allowed to deteriorate, such that newly emerged humans might also be allowed deteriorate in a systemic pattern from which economic or other benefit might be obtained.

These are important because imposing any inhibition of liberty in the reproductive context, favoring gestational development human rights over maternal carrier's rights, or favoring maternal carriers' rights over gestational rights, are an endeavor obfuscate and obscure all of the conditions and outcomes occurring in civilizations and outside of civilizations which nature has not ever intended to occur, resulting in human behavior which no anatomical nuance of human physiology has been developed, architected, exhibited or emerged to exhibit, which is detriment by humans toward other humans. The exhibition of social constructs and sanctions in this context is similar to exhibition of any other social construct or sanction which is not specifically educational, rehabilitative and stability oriented. That context is the promote and sustain exhibition of outcomes consistent with bias toward particular anatomical, genetic, ethnic, social, socioeconomic, gender or other groups, such that outcomes not intended to occur through anatomically exhibited propensity, outcomes which do not seem to be potentiated otherwise, those outcomes which are detrimental, all can be acculturated, caused to occur by sequestration away from human those factors freely available in nature, allowed exhibition of PEMT inhibition, impose influences that diminish PEMT, imposed conditions that enable elevated homocysteine, imposed conditions that allow homocysteine to persist, deterioration of physiological basis of conscious biological function, allowed deterioration of rewards systems, enabled deterioration of

Similarly, SHP or small heterodimer partner is nuclear receptor group member that generally inhibits nuclear receptor activity by interacting with nuclear receptors to produce dysfunctional heterodimers, particularly because it does not have a nuclear integration domain. SHP integrates with retinoid receptors, thyroid hormone receptors and when co-crystallized with EID1, enables EDI1 to fill in the Helix alpha1 of ligand integration domains such as that exhibited by AF-2 of estrogen receptor or other glucocorticosteroid receptors.

Estrogen receptor alpha exhibition enables flow mediate dilation of vasculature, such that inhibition of estrogen receptor alpha prevents flow mediate dilation of vascular but can be reconstituted by reactive oxygen species prevention or scavenging therapy. Information. eLife. Volume 10. Article 368695. 2021.

Prelamin A accumulation results in mitochondrial dysfunction, endoplasmic reticulum distressed, upregulated DHA phosphatidylcholine, upregulated PEMT, decreased stearoyl coenzyme A desaturase 1 metabolism, increased monounsaturated fatty acids compared to

conditioning and control, imposition of influences that diminish the capacitance nuances of cognition emitted from functional mitochondria with adequately dense PEMT2 function as well a PEMT1 correlated capacitance from hydride to hydridic optional ratios in other organelles, all among the distribution of detrimental artifacts specifically produced to cause detriment to humans.

The intent, from a systemic utility, seems to be to sustain systems by acculturation exhibition of detrimental outcomes, ignore empirical causality, then require that humans acknowledge and acquiesce to being attributed causality for such outcomes, with often complete unawareness of systems promote changes from quantum, genetic, to physiological, metabolic and cognitive, as well as behavioral levels that potentiate exhibition of such outcomes, resulting in humans coming to believe that such outcomes are endogenously originated, required to occur and only occur resultant of conscious control. Pervasively, benefit may be being obtained by individuals, organizations and systems from exhibition such detrimental outcomes including exhibition quotas, revenue objectives, and other objectives that require exhibition of such outcomes.

Populations required to acquiesce to being attributed causality for outcomes which do not occur in nature, then internalize these correlations and potentially come to believe that this outcomes are endogenously originated. Thus, when places which manage, intervene and particularly benefit from detrimental human outcomes in any way become underutilized or empty, a new social construct can be produced to victimize scapegoated populations, a new therapy can be produced which is distant from empirical causality, and

optimal polyunsaturated fatty acids, as well as decreased triglyceride availability, suggesting that PEMT2 function in the mitochondria was impaired, PEMT1 might be upregulated as observed but certainly that communication between the endoplasmic reticulum and mitochondria had become disrupted. CTP Phosphocholine cytidylyltransferase and Prelamin A perform remodeling of the Endoplasmic reticulum and nuclear complex.

Leaves and Peelings of fruits and vegetables exhibit folate and phosphatidylcholine as well as other nutrients that can ameliorate this fructose paradox and prevent upregulated triglycerides from contributing to enhance triglyceride storage and factors in adiposity. emergence of every civilization in the wilderness exhibits that every human endeavor and every enterprise had the priority of or derived benefit from making itself obsolete, since sustainment of stability, security, health, and vital being were immitigably unobfuscatable priorities. However, when nature and water were sequestered away from being freely accessible and usable by humanity, the requirement of economic resources for the social condition to be obtained including before satisfaction of

the electricity production as well as the wireless coverage or wireless power levels can be enhanced. These produce scope creeping dynamics in which every nuance of human activity potentially becomes systemically commandeered to fill places that manage detrimental human outcomes such that communities, groups, and home become revenue generating opportunity boxes in locations, people, molecular pathways, all systemically become utilized in a way that directs human outcomes into those outcomes that may be most benefit from, such as the sometimes 90 million dollars per instance when abated being is implemented as a sanction, or until only a limited number of outcomes can be participated.

These explain why an aromatase inhibitor or any factor that inhibits production of Estradiol thereby inhibiting PEMT, can be allowed to be instrumented. Eventually, any inhibitor of PEMT will cause outcomes that increasingly potentiate abated vital being, but instrumentation of an inhibitor of Estradiol production can provide a benefit today, regardless of such a therapy abrogating the basis of conscious biological function. The same occurs among populations unable to or unwilling to understand and change the fact that the cause of disease, aging and detrimental behavior are being openly omitted from priority in a way that cause massive levels of abated vital being and disease. Populations, being caused to live from pay period to period, or not allow to have sustainable stability resultant of fees required for land or real estate, as well as not being assured shelter, homes, human condition, social condition, behavioral condition or physiological condition freely without required economic ability, all are conditioned to focus on what is required to sustain vital being today.

water, nutrition, shelter, homes, stability, health and other factors emerged.

These enable civilization's own inadequate understanding, priorities, opinion and development to impose upon humanity those outcomes that allow benefit to be obtained from detrimental outcomes as well as impose upon humanity those outcomes which confirmed the biases and opinion of the civilization.

Homocysteine, inhibited PEMT, impaired Methionine synthase, Betaine homocysteine methyltransferase, Betaine homocysteine methyltransferase II(2), Thetin-Homocysteine methyltransferase, Indolethylamine N – Methyltransferase, Thioether S – Methyltransferase, S – Adenosyl homocysteine Hydrolase, or cystathionine β -synthase (CBS) and cystathionine γ -lyase (CSE), Thiopurine/Thioether S – Methyltransferase, Trimethylsulfonium Tetrahydrofolate N Methyltransferase, S-adenosyl Methionine Synthetase, MARS1/MARS2 Methionyl – tRNA – Methionyl Ligase function, all can be major pathways for increased exhibition of homocysteine as well as inhibited PEMT.

Eventually, these factors result in environmental fields that diminish and crowd out the capacitant fields emitted by mitochondria and endoplasmic reticulum, along with influences and conditions that diminish PEMT, resulting abrogation of vital being. These are allowed to occur and even imposed using these systematic ways scapegoating particular populations, until all among an age cohort become included in one of the scapegoated populations known as the aged. Every pharmacological therapeutic organization knows that expending 50 percent or more of a products investment on advertising is essential to a product's success because inhibited PEMT and increased homocysteine produce a susceptibility that in nature would lead to discovery of resources to alleviate homocysteine but in civilization those cues have been commandeered as shapes, colors, smells, tastes, associations and inclinations to cause consumer behavior or exhibit outcomes form which benefit might be obtained even when such benefit requires exhibition of detrimental human outcomes. Resultantly, every genetic expression, epigenetic factor, metabolic pathway, cue that would lead to resolution of elevated homocysteine, factor assuring PEMT function, becomes commandeered to lead to an outcome or inclination from which benefit may be obtained leading to a permanent status from which benefit may be obtained such as deprivation of being or deprivation of liberty, volatility from which benefit may be obtained such as chronic conditions, addiction, compulsion, or overuse of emergency services, or extinguishing of the basis of capacitant being.

Political interactions pervasively exclude mitigation of these dynamics and focus on which individual, group, system or entity

The best way to understand these factors is consider that every system of the universe has a potential to prioritize itself over its incipient purpose, incipient utility and over humanity. This is natural factor in organisms as organisms emerge and then acquire ability for sustaining their own being. Humans are differentiated because reliance of humans upon others during incipient nuance of being becomes participation in reciprocal caring for one another during developmental and socialization processes, typically. Human systems seem to have been intended to function in similar ways except the inherent propensity to prioritize themselves can emerge as hidden interactions among systems which allow or enable benefit to be obtained from human outcomes. Systems have to be managed to prioritize vital being and humanity, along with program management KPIs, monitors and measure that assure such prioritization is occurring. These also include exhibition of a priority each decade for systems to resolve causality, alleviate and produce mechanisms of preventing the factors and outcomes which such systems incur during activity or operations. Continued exhibition of benefit

is to benefit most from the assured revenue or benefit obtained from allowing these factors to persist without empirical resolution focus on public visibility into these dynamics. Since 1878 when dimethylthetin could be shown to deplete homocysteine at 700 times the potency of therapeutics even used today or since 1841 when glycollate was presented in the literature, the option to empirically resolve a most empirical factor in disease and detrimental behavior was excluded in favor of utilizing the metabolic product of dimethylthetin and homocysteine being metabolized by thetin-homocysteine methylpherase, known as methylthioglycolic acid, to produce diverse array of derivatives which could be presented to human populations instead of utilizing dimethylacetothetin. Since homocysteine below 6 or 7 um/L per liter, in a particular study showing a 500 to 1 decrease in abated being over a decade period among a population of 10,000 and control of nearly 10,000, was observationally observed to result in massive decreases in detrimental human outcomes, these clearly presented that a human rights quagmire had emerged. However, Homocysteine should be managed to between 3.7 and 7 or 6 um/L, although 3.7 um/L or lower can be increasingly optimal, with 10 um/L used as therapeutic gateway threshold that requires continued therapeutic management to lower levels. Focused therapeutic intervention of homocysteic acid, homocysteine thiolactone, s adenosyl homocysteine or homocysteine may be produce increasingly beneficial effect. Even today, the literature among systems of civilizations cautiously offer 10 um/L as threshold for homocysteine, mostly because, like ascertainment of optimal choline levels or choline obtainment levels, confounding factors such as

from the same outcomes for decades clearly suggest an impetus to not resolve causality and not prevent detrimental human outcomes. Each decade, every system should be required demonstrate learning regarding empirical and incipient causalities, while reengineering of policies, procedures, presumptions and capabilities to improve human outcomes as well as more empirically resolve causalities while clearly identifying detrimental aspects of the status quo to produce clear KPIs and monitors to assure alleviation of these also. This includes implementation of Clinical Information Technology systems with analytics to produce correlations, linkages, mechanistic links and analyze outcomes. Many clinical informatics enable correlations and required causal factors to be ascertained, such that mapping of empirical causal factors can be performed.

It is clear that the continued exhibition of detrimental outcomes may be unnecessary and may be the result of this paradox of required exhibition economic sustenance in order to obtain human, social, and other requirements, amid the context of sequestration away from humanity of those factors available in nature that would such

trimethylamine-n-oxide and the fact that a choline adequate cohort has not been known to occur in a human populations because these would result in an indefinite span of being, the statistical data cannot report on what is not observed, but requires inferences possible with machine learning, data science, and artificial intelligence.

However, the literature, data and practice outcomes information have increasingly suggest that Homocysteine should be managed to between 3.7 and 7 or 6 um/L, although 3.7 um/L or lower can be increasingly optimal, with 10 um/L used as therapeutic gateway threshold that requires continued therapeutic management to lower levels. Focused therapeutic intervention of homocysteic acid, homocysteine thiolactone, s adenosyl homocysteine or homocysteine may be produce increasingly beneficial effect.

The major affect of the largest geopolitical conflicts of the 1900s included a distribution of detrimental artifacts among the world's populations, an acculturation of alternative concept of social constructs as sanctions compared application of PEMT and homocysteine management known of since the 1870s and or even 1840s, and exhibition of a somewhat impossibility for western civilizations to implement comprehensive social welfare assurances, although such assurances were likely the incipient objective of civilizations, because doing so disrupt sociopolitical alignment exhibited in the détente era. Economic opportunity was not an incipient objective of any civilization, while assurance of human, social, behavioral physiological requirements were a central impetus in such regard, such that the now exhibited requirement for economic sustenance in achieving almost any human, social,

sustainment, along with the imposition of those factors, influences, and outcomes potentiate outcomes consistent with the bias, objectives, quotas, revenue objectives, inaccurate opinion and interests of controlling interests of the status quo. While workers, researchers and practitioners in systems may be incentivized to ascertain and alleviate empirical causalities, the executive roles and controlling interests such as Boards of stewardship, directorship or leadership intermediate these activities with decisions that maximize value to organization and those with interests such systems and organizations. Organizations release the derivatized patterns, influences and solutions upon civilizations, such that these interact with one another upon the context of humanity and systems affecting humanity. These patterns and derivatized solutions.

The practical implementation of these dynamics explain much of civilization's outcomes.

It is both fortunate and the result of enduring human qualities that systems and workers in this context have enabled systems to function in this regard with any semblance of the original priorities for the synthesis of modern

behavioral, and physiological requirement may have a resulted in the inability for any organization that derives economic benefit for itself and for those whom perform for such organizations, exhibit an inherent inability to acknowledge, prevent, and alleviate the empirical and incipient cause of hardly anything. This paradox seems to be integral factor in almost every detrimental human outcome.

The dynamics suggest that because the utilization of abated being causes an increase of between 20,000 and 1,000,000 or more instances each year, with similar increases in civilizations all around the world, and populations of civilizations are not aware of this or have decided to utilize political and electoral process to persist this level of detriment toward their own populations as well as global populations, systems may have come to elute detrimental interactions, geopolitical circumstance and allowed exhibition of detriment within such civilization as natural response to exhibition of apathy toward humanity and toward vital being. What is certain, however, is that because certain economic, organizational and systems providing services exhibited remarkable decrease in accidents and detrimental outcomes experienced by workers, it is clear that systems and organizations of civilizations have an interested in abating the utilization of abated being as a sanction.

Eventually, if such dynamics are not changed, every human will not know of context in which iNOS, atmospheric particulate and benefit from detrimental human outcomes has existed. Systems workers, correlatively, must be more adequately compensated, benefited and protected in order to assure that populations are shielded also from these dynamics of systems. More

civilization. Using an apportioned ratio that represents the levels of vital being that occurs compared to detrimental behavioral, physiological or accidents in one modern civilization, a trend of decreasing abated vital being resultant of imposition of moratorium preventing usage of abated being as a sanction demonstrated 20,000 actual prevented instances of abated being that was trending toward 1,000,000 prevent instances of abated vital being each year. This translates to 500,000,000 detrimental outcomes of diverse nature in order to correlated to 1,000,000 instances of abated vital being.

This suggests that exhibition of abated vital being as a sanction in one civilization alone is causally linked to exhibition of 500,000,000 or more instances of detrimental outcomes, explaining how changes to global levels of abated being, detrimental outcomes and geopolitical conflict all are causally linked to imposition of abated being as a sanction. This suggests that system workers of civilization perform an incalculably valuable service diffusing massive detrimental potential before even performing any activity or roles. It is perhaps the fact that such workers are the most educated,

works, more safety technologies, robotics to decrease risk, and technologies to improve understanding, as well as mechanisms of translating understanding into policies, procedures and public information will be essential.

Social constructs and decisions will also likely have to demonstrate the applied policy analysis performed, decisions made, why such decisions have been made, and how these relate to human priorities, safety, health, and sustainability of environment.

Important integrative mechanisms must be implemented that assure individual and organizational solvency in a way the relieves the change in priorities and behavior that is derived from a requirement to exhibit solvency or a profitability. Its clear that systems of civilizations commandeer human activity in a way that benefits civilizations, often enabling achievement but too often resulting in less than optimal outcomes. These conditions require that civilizations begin to continuously assure that human requirement are available to all through direct compensation, through direct subsidization of organizations providing opportunity and providing encompassing benefits structure such as health, vital being, injury, impairment, employment, and leave benefits or assurance that demonstrate immitigable human priority. Benefits in this regard should be aggregately provided for each individual directly according to ability and should be offset by organizations providing economic opportunity according to the ability of such organization to both assure stability of opportunity provided to workers and the ability to maintain any solvency requirement that exceeds such worker focused solvency priorities.

informed, capable and compassionate cohorts which have ever performed in such capacity in the span of the human experience.

Attributing causality to humans for outcomes prevents prioritizing understanding of required incipient and empirical causal factors, such that, instead, continued focus on those factors that are incipient, empirical and causal, as well as required, should be immediate objectives or the objective of iterative improvements.

Once nature becomes commandeered by civilizations systems and is sequestered away from humanity to only be obtainable according economic ability, even including water, clean air, freedom from massive toxicity, or nutritional quality, civilization becomes the integral shaper of pervasive human outcomes. The priorities of nature and its inherent priority for humanity and vital being humanity, become subjugated to the whim of civilization's systems and the inherent propensity of systems to prioritize themselves over humanity. Workers and humans in system become required to understand the mechanisms through which human deprioritization and diffuse these in diverse ways. The challenge to humans working within

The literature clearly links prelamins A, homocysteine, progerin and impaired catalysis of PEMT2 with early developmental metabolism, abated vital being in early nuances of being similarly to progerias, senescence of cellular entities similar to progerias, and changed shape of nuclear or other structures linked to phospholipid distribution anomalies, and therefore progerias exhibit the same integral mechanisms through which aging otherwise occurs including PEMT2 impairment, changed lipid characteristics, homocysteine and disruption of essential metabolic and structural nuances of development. Information. Medicine. Volume 99. Number 7. Article e19022. February, 2020. Information. Molecular and Cellular Biochemistry. Volume 387. Issue 1 and Issue 2. February, 2014. Information. Biochimica et Biophysica Acta. Molecular 'Cellular' research. Volume 1773. Issue 5. Pages 661 through 674. May, Information. Journal of Cellular Science. Volume 124. Part 24. Pages 4253 through 4266. December 01, 2011. Information. The Journal of Endocrinology and Metabolism. Volume 100. Issue 7. Pages E964 through E973. Information. Physiol Res. Volume 70. Number 4. Pages 533 through 542. 8th Month, 2021. Information. Signal Transduction and Targeted Therapy. Volume 6. Number 108. 2021.

	<p>systems in assuring human priority is most eloquently described by the requirement for economic ability in order to obtain water.</p> <p>Organizations are becoming more focused on providing customers what they think they want, but also what the analytics indicate are true values in consumer activity, along with benefiting customers in asymmetric ways such as integrating knowledge and understating into products or services, integrating value that manages detrimental influences, managing the environment and supplying an improved environment, all in ways that customer's may not be consciously aware of but can be packaged into value proposition of solutions, products and services.</p> <p>These potentiate acceptance of fewer product iterations, fewer social constructs and fewer decisions, along with these being more intricately analyzed, explained, presented and focused on value that humans can translated into quality of life, duration of being, health, stability and sustainability..</p>	
Fructose Paradox	<p>The literature links fructose with diabetes and unhealthy anatomical mass, particularly because fructose may be directed toward triglyceride synthesis and storage.</p>	<p>A better perspective is to consider, completely, how humans utilize fruit in natural conditions. Natural conditions exhibit that fruit being incurred in nature may have been removed or extricated from plants already. Otherwise, fruit may be obtained from plants. Also, before</p>

The recommendations in the popular information recommend against eating fruit unless it is out of season, although pervasively the phytonutrients are considered to be essential. Some information recommends fruit ingestion only when it is in season.

Nutritional obtainment of phospholipids is known to prevent fructose from causing dislipidemia. Fructose enabled increases in lipogenic enzymes such as SREBP1, ChREBP, and other enzymes enabling increased production of hepatic triglyceride, diglycerides, ceramides and oleates, all were inhibited by nutritional phospholipid obtainment. The literature observes also that PEMT and GNMT in experimental small nonhuman mammals are inhibited after feeding, increasing S-Adenosyl Methionine, inhibiting phosphatidylcholine synthesis, although ablated expression of FGF15/19 and ablated expression of SHP both prevented downregulation of PEMT and GNMT. Ablated expression of FGF19 diminishes bile acid synthesis in a way that counteracts loss of phosphatidylcholine when producing bile acid enabled by MDR2 to prevent an aggregate loss of phosphatidylcholine

horticulture and agriculture, and when movement to food sources may have been the status quo, populations or groups might have moved or migrated to incur food sources. Incurrence of a food source could then result in maintaining the location of the food source until the food source had been practically depleted. Also, capabilities for removal of peelings were not readily available. This suggest that whole fruit and vegetables and food sources such as edible covering of foods of plantae, animalia, fungi, archaea, etc may have been ingested unless such outer covering ingestion is impractical or unsafe. Whole organism ingestion can assure that adequate phosphatidyl and concluding phase differentiation phenotype proteins, amino acids, enzymes and molecules are obtained, assisting in managing developmental nutrient rich aspects of fruits, vegetables, legumes, and other foods. Juicing using whole organisms is the best way to circumvent difficulty in including outer coverings of natural foods in meal preparation.

Aryl hydrocarbon receptor translocates to the nucleus resultant of resultant of insulin/PKB complex signaling immediately after feeding, causing PEMT and GnmT expression, presenting an axis of PEMT expression that is not clearly presented here as involving Estrogen Receptor Alpha or Beta although SHP is known to affect AF-2 domain of estrogen receptors loci of integration with genetic sequences. However, since AF-2 of estrogen receptor beta exhibits a sequence difference compared to estrogen receptor alpha, its possible that SHP can enabled a differential effect between estrogen receptors. FGF19 and SHP become activated in later in feeding or after feeding, diminishing PEMT and GNMT activity. Experimental obesity in small nonhuman mammals exhibits

when obtaining a low choline nutritional regimen, while FGF15 is known to enable regeneration and repair of hepatic tissues as well as enabled glycogen storage and glucose tolerance. FGF19 also interacts with the Farnesoid X Receptor FXR, integrating with FGFR/B-Klotho complex to inhibit Cyp7a1. FGF is a fibroblast growth factor.

Similarly, SHP or small heterodimer partner is nuclear receptor group member that generally inhibits nuclear receptor activity by interacting with nuclear receptors to produce dysfunctional heterodimers, particularly because it does not have a nuclear integration domain. SHP integrates with retinoid receptors, thyroid hormone receptors and when co-crystallized with EID1, enables EDI1 to fill in the Helix alpha1 of ligand integration domains such as that exhibited by AF-2 of estrogen receptor or other glucocorticosteroid receptors.

increased phosphatidylcholine synthesis and enhanced steatosis responsively to aryl hydrocarbon receptor AhR expression. SHP inhibits this association between phosphatidylcholine produced by aryl hydrocarbon receptor in obese small nonhuman mammals and potential for steatosis. This potential for steatosis also can be prevented by inhibition of PEMT. PEMT, aryl hydrocarbon receptor and phosphatidylcholine levels are elevated in simple steatosis, while advanced steatosis as steatohepatitis fibrosis exhibit remarkably decreased phosphatidylcholine. Aryl Hydrocarbon Receptor activation is known to upregulate choline kinase, sphingomyelin, and fatty Acid- carnitine conjugate as well as lysophosphatidylcholine. Inhibition of SCD1 in this context is known to prevent production of oleic acid from stearic acid, palmitoleic acid from palmitic acid. Aryl hydrocarbon receptor activity, thus, promotes enhanced exhibition of saturated fatty acids and diminishes the availability of unsaturated fatty acid.

Folic Acid also inhibits the potential for nonalcoholic fatty liver disease through activation of AMPK and LKB1 along with inhibited phosphorylation of acetyl coenzyme A carboxylase ACC.

Information. Biochemica et Biophysica Acta, BBA, - Lipids and Lipid Metabolism. Volume 1004. Issue 2. Pages 274 through 277. 8th month, 8th Day, 1989. Information. Xenobiotica. Volume 49. Number 5. Pages 591 through 601. May, 2019. Nutr Res Pract. Volume 14. Number 4. Pages 309 through 321. 8th Month, 2020. Information. Nature Communications. Volume 9. Article Number 540. 2018. Information. The Journal of Nutrition. Volume 141. Issue 11. Pages 2003 through 2009. November, 2011.

Casein Kinase II upregulated in oncology as a metabolizer of acid amino acids and hyperactivated by Casein A1 milk to cause histamine cascade and enabled cellular survival, differentiation, mitosis and meiosis. Prefers phosphoproteins and performs phosphorylation cascade. Upregulated in diverse oncology. Consider to be the cause digestive pathway inflammation, endometriosis, and other inflammation that occurs in digestive pathway areas. Casein Kinase upregulation by Estrogen Receptor Alpha along with Estrogen Receptor Alpha upregulation of AP1 to inhibit catalytic activity of PEMT enzymes, represents both canonical susceptibility exhibit in most if not all oncology, but also represents the canonical behaviors at the cellular that constitute oncology.

Diminished activity of estrogen receptor beta, compared to estrogen receptor alpha also diminishes the ability of estrogen receptor to downregulate AP1 and downregulate Estrogen Receptor Alpha activity. The uneven activation of estrogen receptor alpha by conditions, glucocorticosteroid receptors without the perfect 13 sequence human estrogen response element activation sequence, as well as hormones or glucocorticosteroids derived from therapy, recreational usage and potential phytohormones, thus, can participate in canonical oncology signaling. PEMT inhibition, is not only integral to oncology, but is an integral aspect of pervasive detrimental behavior, disease, and detrimental physiological outcomes.

Both Casein Kinase II alpha subunits participate in increased Androgen Receptor Singling observed in prostate oncology. Information. PMID 24418032.

A diverse group of oncology of breast health statuses including triple negative oncology of breast exhibit remarkable upregulation of Casein Kinase II (2). Information Casein kinase has emerged as factor in diverse and increasingly substantially array of oncology, disease, inflammation and dysbiosis. Casein kinase may be involved in the link between increased acidity, and choline deficient amino acid defined nutritional regimens and potential for oncology. The activity of Casein Kinase is a causalmechanistic causal link to the cellular behaviors which constitute oncology. Information. Breast 'oncology' Res. Volume 17. Number 19. 2015. PMID 25837326.

CDK11P110 inhibition is expressed in all known oncology versions known to be tested to this instance, while CDK11P58 linked to cellular cycle pause and apoptosis. Particularly in oncology of breast, inhibition of CDK11P110 cause remarkable deterioration of oncological activity including stimulating

Prostate oncology exhibits independent activation of PI3K and Casein Kinase II to activate NF-kB through EGFR and through HER-2 signaling. Information. Prostate. Volume 65. Number 4. Pages 306 through 315. December 1, 2005. PMID 16015604.

The exhibition of casein kinase upregulation even in HR+ oncology of breast, presents a clear somewhat encompassing coverage for casein kinase in breast oncology and diverse aspects of other oncology, with a clear, consensus, causalmechanistic group of causal changes that not only are linked to oncology but constitute oncology. Information. Oncotarget. Volume 5. Number 15. 8th Month, 2014. PMC4171645.

Dysregulated expression of estrogen receptor alpha is strongly linked with disease and oncology, since uneven activation of estrogens receptors, particularly diminished comparative expression of Estrogen Receptor Alpha which downregulates Estrogen receptor alpha and which decreases expression of AP1. Estrogen receptor beta decreases Estrogen Receptor alpha and decreases expression of AP1, preventing AP1 from inhibiting the activity of PEMT. The upregulation of Estrogen Receptor Alpha without equal upregulation of Estrogen Receptor Alpha, causes increased AP1 expression which diminishes PEMT transcript catalytic activity through AP1 inhibition of PEMT catalysis. However, it is the pivotal activation of Casein Kinase 2 by estrogen receptor alpha which transforms mere susceptibility resultant of PEMT inhibition to exhibit also causalmechanistic catalytic changes by Casein Kinase that constitute most of the constitutive behaviors known as oncology at the cellular level. Information. Cellular SignalingSignalling. Volume 28.

<p>exhibition of apoptosis as well as causing cellular cycle pause. A participative or integral role for CDK11P110 in osteosarcoma as well as liposarcoma, such that diseases of mesenchymal cellular entities are linked to CDK11P110 upregulation. The therapeutic affect of CDK11P110 is interested because casein kinase II phosphorylates CDK11 and RNA Polymerase II Carboxyl – concluding extremity domain. This present a clearly discernible causal link between PEMT status, Estrogen receptor alpha status, Estrogen receptor beta status, AP1 status, casein Kinase II status, CDK11 catalysis and RNA Polymerase II activity. Information. J Biol Chem. Volume 278. Pages 2265 through 2270. 2003. PMID 12429741. Information. Scientific Reports. Volume 5. Article 10433. 2015.</p> <p>Emodin or 4,5,6,7-tetrabromobenzotriazole enhances natural cytotoxic T cellular entity activity toward hepatocellular carcinoma and HeLa cellular entities and enhanced cytotoxic activity when TRAIL or Fas agonistic immunoglobulin was instrumented. Information. Clinical and Experimental Immunology. The Journal of Translational Immunology. Volume</p>	<p>Number 6. Pages 675 through 687. 6th Month, 2016.</p> <p>Silmitasertib CX-4945 selectively inhibits casein Kinase II (2). Information. Casein-kinase.html Selleckchem.com website. Information. www.eurekaselect.com website. CX4945. Information. Current Pharmaceutical Design. Volume 23. Issue 1. 2017.</p> <p>DMAT (2-dimethylamino-4,5,6,7-tetrabromo-1H-benzimidazole) is strongly selective inhibitor of casein kinase 2. Information. PMID 18588507. Biochem J. Volume 415. Number 3. Pages 353 through 365. November 1, 2008.</p> <p>Casein kinase inhibition disrupts myeloid cellular entity differentiation in oncology exhibiting cellular entities. Information. Oncology Res. Volume 78. Number 19. Pages 5644 through 5655. October 1, 2018. PMID 30139814.</p> <p>Inhibition of casein kinase alpha by D4476 activates cytotoxic activity from 5-fluoracil particularly in microsatellite instable colorectal oncology cellular entities. Autophagy flux inhibition occurred correlative to cytotoxic activity of 5-fluoracil enhanced by D4476. Information. Arch Immunol Ther Exp. Volume 69. Number 1. Page 26. 2021. PMID 34536148.</p> <p>Umbralisib inhibition of PI3K-delta and inhibition of casein kinase 1-epsilon CK1-epsilon is approved for therapeutic intervention of marginal zone lymphoma when the therapeutic experience already includes at least one anti CD20 therapeutic. Umbralisib is also indicated for adult therapeutic contexts of relapsed or refractory follicular lymphoma with therapeutics experience including at least three systemic lines of therapy. Umbralisib. United States Food and</p>
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	152. Issue 2. May, 2008. Pages 336 through 344.	Drug and Administration. Online Information.
CDK11P110 upregulation of proliferation compared to CDK11P58 inhibition of proliferation.	<p>CDK11P110 inhibition is expressed in all known oncology versions known to be tested to this instance, while CDK11P58 linked to cellular cycle pause and apoptosis. Particularly in oncology of breast, inhibition of CDK11P110 cause remarkable deterioration of oncological activity including stimulating exhibition of apoptosis as well as causing cellular cycle pause. A participative or integral role for CDK11P110 in osteosarcoma as well as liposarcoma, such that diseases of mesenchymal cellular entities are linked to CDK11P110 upregulation.</p> <p>The therapeutic affect of CDK11P110 is interested because casein kinase II phosphorylates CDK11 and RNA Polymerase II Carboxyl – concluding extremity domain. This present a clearly discernible causal link between PEMT status, Estrogen receptor alpha status, Estrogen receptor beta status, AP1 status, casein Kinase II status, CDK11 catalysis and RNA Polymerase II activity. Information. J Biol Chem. Volume 278. Pages 2265 through 2270. 2003. PMID 12429741.</p> <p>Information. Scientific Reports. Volume 5. Article 10433. 2015.</p>	

<p>CDK11P110 upregulation of proliferation compared to CDK11P58 inhibition of proliferation.</p>	<p>CDK11P110 inhibition is expressed in all known oncology versions known to be tested to this instance, while CDK11P58 linked to cellular cycle pause and apoptosis. Particularly in oncology of breast, inhibition of CDK11P110 cause remarkable deterioration of oncological activity including stimulating exhibition of apoptosis as well as causing cellular cycle pause. A participative or integral role for CDK11P110 in osteosarcoma as well as liposarcoma, such that diseases of mesenchymal cellular entities are linked to CDK11P110 upregulation. The therapeutic effect of CDK11P110 is interested because casein kinase II phosphorylates CDK11 and RNA Polymerase II Carboxyl – concluding extremity domain. This presents a clearly discernible causal link between PEMT status, Estrogen receptor alpha status, Estrogen receptor beta status, AP1 status, casein Kinase II status, CDK11 catalysis and RNA Polymerase II activity. Information. J Biol Chem. Volume 278. Pages 2265 through 2270. 2003. PMID 12429741. Information. Scientific Reports. Volume 5. Article 10433. 2015.</p>	<p>CDK11P110 is essential oncology of breast proliferation and growth. Information. Scientific Reports. Volume 5. Article 10433. 2015.</p> <p>Inhibition of CDK11P110 remarkably downregulated breast oncology cellular entity proliferation, growth and migration, typically producing G1 phase pause.</p> <p>OTS964 is a potent inhibitor of CDK11B which is also known as CDK11P110, while also inhibiting TOPK, in a way that produces some level hematopoietic toxicity. Information. Genetic Databanks. CDK11B/CDK11P110. Information. Sci Transl Med. Volume 6. Number 259. Article 259ra145. October 22, 2014. PMID 25338756.</p> <p>CDK11B or CDK11P110 is inhibited by pazopanib, AST-487, Linifanib, barasertib-hQPA, crizotinib, foretinib, BMS-345541, tozasertib, BMS-387032, AT-7519. Information. Cyclin dependent kinase 11B. IUPHAR/BPS guide to Pharmacology.</p> <p>CDK11 inhibition produces remarkable deterioration of BRAF melanoma cellular entities and NRAS melanoma cellular entities. Information. Pharmaceuticals Volume 12. Number 2. Page 50. 2019.</p>
<p>Progerin is a factor in deterioration of cellular biology link to numerous pathologies and changes linked to aging, as well as being a causal factors in diseases which cause early or rapid exhibition of nuances of aging.</p>	<p>Inhibiting farnesylation of Laminin diminishes that pathology of Hutchinson-Gilford syndrome and</p>	<p>Progerin is a permanently farnesylated version of laminin that accumulates in correlation with aging now known to affect how lamin is processed in the cytosol,</p>

Laminin sequestration to the nucleus is correlated with stiffness of tissues. Some experimental models of regeneration require decellularization and reseeded of extracellular matrix with stem cellular entities to regenerate plasticity. Laminin causes expression of two mitochondrial proteins during neurite growth in the emerging and developing physiology. *Int j Dev Neurosci*. Volume 14. Number 3. Pages 365 through 374. 6th Month 1996.

Intercellular adhesion molecules or iCAMs are molecules that adhere adjacent cellular entities. Lamins and other matrix proteins participate in stabilizing cellular entities but laminin performs in this general cellular, extracellular matrix, connective tissue role that, in a way, becomes, connect or promotes synthesis, exhibition and stability of factors that hold physiology together. Laminin has two arms along a central structure that is a cruciform or is similar to a structure that has welcoming arms that are utilized to bring together physiologically distinct factors. Connexons are proteins that permeate the plasma membranes of cellular entities to link or sew together different cellular entities, such that these become tunnels that allow cellular entities share molecular and metabolic phenotype, producing stability through these gap junction proteins by allowing tissues to function together in response to circumstance and stimuli. These allow synchronized response to stimuli such as neurotransmitter and are an important way in which cellular level oncological change is propagated to adjacent cellular entities sometimes without requiring other cellular entities to actually exhibit such oncological changes themselves. www.lisbdnet.com

diminishes a potent effector of the detrimental nuances of aging, while also improving the morphology of keratinocytes in a way that can be monitored for effectiveness of farnesylation of laminin. Both FTI-276 or pravastatin along zoledronate produce inhibition of farnesylation in this regard. *Information. Nucleus*. Volume 1. Number 5. Pages 432 to 439. September through October, 2010.

JH4, a lamin integration inhibitor, diminishes progeria syndrome and Hutchinson-Gilford syndrome in a way that preventing suppressed cellular proliferation, prevented reshaping of the nucleus and enhances span of vital being. Progerin SLC-D011, increases experimental organisms duration of being 5 times longer than lonafarnib, a farnesyl transferase inhibitor. Progerin also enhances, substantially, histological and physiological nuances of small experimental organisms. Human experiments produced extension of vital being by 2 years using a progerin therapeutic while other studies exhibit only 1 instance of abated being among therapeutic populations compared to other therapies which have much higher instance of adverse outcomes. *Information.*

affecting how it accumulates within and affects the nucleus as well as affecting how laminin is presented in the extracellular environment to produce extracellular matrix. *Information. Exp 'Cellular' Res*. Volume 318. Number 1. Pages 1 through 7. January 1, 2012.

Prelamin A, lamin A, Progerin and lamin B staining are different between control and Progerin Syndrome. Progerin was reduced in staining assay using Farnesyl Transferase Inhibitors and combination pravastatin and zoledronic acid. Farnesyl transferase inhibitors, Farnesyl transferase inhibitors along with Geranylgeranyltransferase inhibitor, and a farnesyl transferase inhibitor along with pervastatin and zoledronic acid, all improved distribution characteristics of Lamin A. Progerin staining was improved by rapamycin, a farnesyl transferase inhibitor, pervastatin and zoledronic acid. The most effective inhibitors of progerin also produce increases of laminin A and act through inhibition of the mevalonate pathway, while rapamycin and N Acetyl L Cysteine affect the MTOR pathway in a way that decreases progerin without also increasing prelamin A. *Information. Biogerontology*. Volume 20. Pages 337 through 358. 2019.

Progerin syndrome nucleus shape impairment and pathogenically early concluding differentiation induced by progerin, both were remarkably ameliorated in human fibroblasts as well as mesenchymal pluripotent stem cellular entities by metformin. The mechanisms of action included decrease of SRSF1 integration into Genetic sequences that promote lamin A expression, such that progerin synthesis from impaired farnesylation decreases and such that SRSF1 cannot perform as ASF1 or SF2 in influencing mRNA splicing decisions in which eIF4E enhances initiation of translation of mRNA

Communications
Biology. Volume 4.
Article number 5. 2021.

Zovinsky is included as a therapy for laminopathies and may be utilized in treatment of progeria disease. Progeria is a factor in aging and it is not clear if Zovinsky will be utilized to treat the detrimental aspects of aging. Information. Information. "FDA approves First Treatment for Hutchinson-Gilford Progeria Syndrome and Some Progeroid Laminopathies." FDA News Release. United States Food and Drug Administration. November 20, 2020.

Lonafarnib is utilized for Alzheimer's. Alzheimer's Drug Discovery foundation.

Antroguinol, tipifarnib, lonafarnib, L744832, L778123, BMS-214662, FTI-277, FTI-276 LB-42708, moverastin, manumycin A, FTI-2153, PD169541, ABT-100, all are inhibitors of farnesylation transferase. Numerous oncology conditions, viral conditions, and other pathologies can be improved by preventing or diminishing prenylation which includes modification of isoprenoid lipids through farnesylation and geranylgeranylation. Information. Medchemcomm. Volume 8. Number 5.

already attached to ribosomal molecular machines to also suppress 4E-BP along with promotion of deterioration of nonsense mRNA. SRSF1 is an alternative splicing enabler that allows the 5' prime splice to be connected to the 3' splice as well as recruits U1 snRNP to the 5' prime splice in a way that promotes proximal intron usage instead of distal intron site usage particularly by occupying exon splice enhancer sequences. The Serine/Arginine-rich splicing factor 1, SRSF1, thus, can be affected by depletion of arginine by iNOS. SRSF1 competes with tat protein of the immunodeficiency viral protein genome for access to TAR transactivating response element protein preventing requisite changes to the active RNAPII complex required for viral transcription and function, such that through alternate splicing linkages of viral introns and dissociation of viral exons, SRSF1 inhibits B, C and D subtypes of the immunodeficiency viral vector by about 10,000 percent or by 100 hundreds of times. Information. Oncotarget. Volume 6. Number 23. Pages 19362 through 19363. 8th Month, 2015. PMID 26305984. Information. npj Aging and Mechanisms of Disease. Volume 2. Article Number 16026. 2016.

Farnesylation is substantially upregulated in Alzheimer's disease, particularly. Hras is particularly upregulated in Alzheimer's Disease. Acta Neuropathologica communications. Volume 9. Number 129. 2021.

Sulforaphane, isoprenylcysteine carboxyl methyltransferase inhibitors, and statins have exhibited ability to inhibit progerin through mechanisms that include enhanced autophagy. Information. Aging 'Cellular entities'. Anatomical Society. Volume 14. Number 1. Pages 78 through 91. February, 2015.

	<p>Pages 841 through 854. May 1, 2017.</p> <p>Laminopathies, including progerin accumulation as well as farnesylation precursors derived from mevalonate pathway are known to be integral factors in detrimental aspects of typical nuances of aging as well as enhanced or rapid aging exhibiting in progerias or dystrophies. Prevastatin inhibits the HMB-CoA reductase pathway and FPP Synthetase enzymes of the mevalonate pathway. Temsirolimus and everolimus produce diverse improvements in HGPS contexts of aging. Information. Biogerontology. Volume 20. Pages 337 to 358. 2019.</p>	<p>Flavopiridol is a potent inhibitor of CDKS or cyclin kinases and may have inhibitory activity for Casein Kinase II. Information. www.scbt.com</p>
G Quadraplexes or G4 Quadruplexes	<p>U07.1 G Quadraplexes are guanine enhanced nucleic acid structures, which perform as scaffolds for integration of ligands and may be exhibited in primates as well as viruses. G quadraplexes modulate viral transcription, replication, and translation. G4 activating molecules are presented as inhibitors of viral transcription, replication and translation and reverse transcription, which are broadly inclusive of viruses, but also includes modulation of the lytic balance of viral activity versus latent activity of viruses. A synthetic system of controlled biocatalytic</p>	<p>Macrophages have type 1 and type 2 versions which are determined by the balance between arginase activity compared to nitric oxide activity, both of which can be affected by availability of substrate including changes balances of L - ornithine, L- citrulline and L - Arginine , although Iron, Tetrahydrobiopterin, Vanadium, and Ca²⁺ are also essential factors in this regard, along with NADPH, as maintainers of iNOS catalyzes in coupled phases, preventing uncoupling of nitric oxide synthase. G4 Quadraplexes, however, have emerged as factors that depleted L – Arginine toward L – Citrulline competitively with iNOS in a way that can cause iNOS to be uncoupled without promoting arginase activity, resulting in an M1 inflammatory phenotype macrophages, citrulline upregulation and locking of the cellular entity into M1 status</p>

cascade engineering reveals that G4 Quadruplexes promote directing or catalysis of L arginine to L Citrulline suggesting that G 4 Complexes resolve the acute phase which is typically link to virulent activity as well as promote production of inflammatory macrophages, suggesting that G4 quadruplexes can produce an inflammatory antiviral response. This G4 complex, thus, may present how HIV modulates between lytic viral activity when iNOS is not uncoupled compared to latent viral activity when iNOS becomes uncoupled. Diminished G4 activity, therefore, would occur during diminished L arginine availability which is circumstance the modulates HIV from lytic activity to latent activity. G quadruplex activators for Human G4 complex may be potent capabilities especially if these can circumvent G4 deactivation by L-arginine depletion to continue the inflammatory antiviral response during latent viral activity. Molecules that inhibit the G4 complex in microbes may be able to produce a novel level of therapeutic affect at the source of viral affliction, within microbes themselves. Specificity Protein 1 can be included in G4 complexes and is included in all primate

because citrulline is efficiently recycled into L – arginine.

The role of the Land's cycle should be strongly presented here because LPCAT/MBOAT/Acytransferases, generally, perform roles of reintegrating free fatty acid into phospholipids, particularly in membranes, thus playing in important roles in the progression of the structural crystallization phases incurred by membrane phospholipids and lipids otherwise, producing the characteristics superstructural characteristics of any tissue, organ or organism. The Lands cycle involves both phospholipases and diesterases involved in detrimental cascades, although particular noninflammatory phospholipases including versions of iPLA2 occur without requirement of detrimental cascades. Essentially, the lands cycle is comprised of freeing fatty acids and phospholipids from cellular membranes where these interact with the environment allowing DHA, EPA, Oleoyle, Palmitate, Ether Linked, Extended Length Arachidonate, all to become involved microenvironment metabolism, become changed into advanced versions of these including neuroprotectins, protectins, resolvins and maresins, all can be produced resultant of advanced processing of Omega-3 fatty acids and ablate cascading pathology, such as abatement of detrimental versions of prostaglandin, eicosanoid, thromboxane, eicosatriene, and leukotriene pathology cascades as well as many other detrimental factors including lipoxygenases. However, not all prostaglandins enable cascading detriment, and prostanoids, poxytrins, elovanoids, (R) - Resolvins, (S) - Resolvins, are all considered linked to specialized Pro - Resolvin Mediators which resolve pathogenic cascades. Versions of these mediators include sulfido - peptide conjugated mediators which, like other versions of these mediators, methylthioglycolic acid and other

lentiviral LTR regions exhibit promoter or integration regions for SP1 and NFkB which is comprised of guanine enhanced consensus sequences in the integration regions for both SP1 and NFkB. This consensus region is compatible with G4 Complex's when multiple integration loci for such transcription factors become juxtaposed in a particular location. G4 complexes may have broad utility in viral activity and structure, as well as have been linked to HCMV, HBV, HOV and perhaps viruses generally, particularly because iNOS is pervasively essential to viral activity. HIV genome exhibits two RNA molecules linked together in a parallel direction at a dimerization initiation locus, while short RNA oligonucleotides with the SP1 integration loci also can dimerize with characteristics observed among intermolecular G quadruplex construction. Nef protein of HIV also exhibits guanine enhanced sequences that fold into G4 structures, suggesting that Nef performs sequestration and deterioration of intracellular CD4 while also causing MHC I complexes on the cellular surface to become sequestered and deteriorated. This results in an impaired ability to sense HIV proteins and DNA, impaired ability

factors, acquire specialized characteristics in the acute phase or cascading pathology microenvironment, derived from the molecular, ionic, atom and quantum characteristics of such microenvironment, very much being adaptive in ways that are similar to molecular therapeutics. These mediators are searched for, screened, tested and used to produce therapeutics.

Macrophages produce PCTR or Protectin Conjugates in Tissue Regeneration, Resolving Conjugates in Tissue Regeneration or RCTR, (MCTR), Maresin Conjugates in Tissue Regeneration, MCTR including glutathione, and DHA exhibition in MCTR1 as well as cysteinylglycyl MCTR2, and DHA exhibition in MCTR2 along with Cysteinyl Hydroxy DHA in MCTR3, such that MCTR is processed by Leukotriene C4 or glutathione S -Transferase Mu 4 (GSTM4), resulting in MCTR1 which then is processed by gamma - glutamyltransferase to become MCTR2, followed by dipeptidase catalysis which produces MCTR3. Sulfido - conjugates emerge similarly from catalysis or interactions incurred by resolvins RCT1, RCT2, RCT3, protectins and 17 Series Resolvins such as RCT3 to resolve pathology cascades in lymphatic tissue.

Macrophages have type 1 and type 2 versions which are determined by the balance between arginase activity compared to nitric oxide activity, both of which can be affected by availability of substrate including changes in balances of L - ornithine, L- citrulline and L - Arginine, although Iron, Tetrahydrobiopterin, Vanadium, and Ca²⁺ are also essential factors in this regard, along with NADPH, as maintainers of iNOS catalyzes in coupled phases, preventing uncoupling of nitric oxide synthase. G4 Quadruplexes, however, have emerged as factors that depleted L - Arginine toward L - Citrulline competitively with iNOS in a way that can cause iNOS to be

of MHC I to integrate with HIV biological machinery, as well as impaired ability to present HIV on the extracellular surface for T lymphocyte and Natural Cytotoxic Cellular entity cellular immunology processes. The result is only CD8+ exhibition that attracts lymphocytes and promotes cytokine expression to attach lymphocytes but no ability to present HIV in CD4 receptors which enable the viral proteins to be presented, integrated with by lymphocytes, performance of V(D)J recombination to integrate the viral sequences into the genome of lymphocytes followed by movement of the lymphocytes to the thymus for replication and pruning. Lymphocytes are lured and then cause to incur apoptosis but the ability to actually produce and complete the immunological synapse is impaired. TMPyP4 and BRACO-19 are examples of G4 interactive molecules and are examples of factors that make latent HIV susceptible clearance. Cellular entities that are lymphocytes and which have CD4+ diminished become impaired in the ability to exhibit 'Helper' function or search and identify function to find diseased cellular entities, while it is not clear that CD4+ downregulation

uncoupled without promoting arginase activity, resulting in an M1 inflammatory phenotype macrophages, citrulline upregulation and locking of the cellular entity into M1 status because citrulline is efficiently recycled into L – arginine.

Macrophages produce resolvins although Macrophages can also stimulate express iNOS and stimulate iNOS expression in tissues. PCTR1, PCTR2, PCTR3, as well as RCT1, RCT2 and RCT3 can incur similar processing as MCTR1, MCTR2 and MCTR3. These provide a strong homologue to the derivatization process through which molecular therapeutics have been produced using methylthioglycolic acid, although these also present how natural processes which perform inorganic to organic phase transfer and which sequester biologically beneficial molecules from flux, racemic or abiotic phases into biological phases such as within foundational compartmentalization factors of biology or cellular entities. These are also important in understanding how and why phosphatidylethanolamine, PDME, PDME and PC produced by PEMT, along with Methylthioglycolic Acid produced by Thetin - Homocysteine methylpherase, as well as Trimethylsulfonium, tetrahydrofolate, and other linked pathways, are not passive in the environment but constitute caustic and active substances that produce a place in the environment for biological systems in a way that is demonstrated by the utilization of these factors as potent industrial waste, petroleum, chemical, and other cleaning factors utilized in some of the most challenging environmental waste or hazardous material management processes.

The literature observes Lipoxin A4, AT-LXA4, RVd1, AT-RvD1 and RVD3 to have receptors ALX/FPR2 and GPR32. RvD1, AT-RvD1, RvD3, LXA4, AT-LXA4, RvD5 have receptors DRV1(GPR32).

automatically causes CD8+ oversufficiency which programs cellular entities to become cytotoxic cellular entities. This clearly suggests that viral downregulation of CD4+ impairs the antigen presentation, recognition, monitoring, and search aspects of adaptive immunology. The literature suggests that MHC I is linked to CD8+ specifically, such that the study presented here must meant to present that MHC1 inhibition impairs CD8+ function and such that CD4+ clearance occurs also but not as a result of MHC1 clearance. This suggests that Both CD4+ and CD8+ function would be impaired, presumably including continued exhibition of dysfunctional CD8+ along with clearance of CD4+ from the extracellular surface. Viruses may use G4 complexes to enhance genetic variability of pathogens, resulting in escape from immunological capabilities, such that G4 density in viral genes and structure used for cellular entry and genetic inclusion are linked to enhanced ability for genetic variability among viruses. Introducing apoptosis in Latent HIV is demonstrated in a study that focuses on modulating G4 ligands. G4 Ligand stabilization was found to prevent SP1 integration into the

RvD2 has receptor DRV2(GPR18). RvE1 and RvE2 exhibit receptors ERV1(ChemR23) and BLT1. Protectins such as PD1 have receptor GPR37. DPA resolvins, however, including RvD5n-3PDa, exhibit GPR101 as a receptor. Maresin 1, similarly, has receptors RORalpha and LGR6.

15 - lipoxygenases enable production of lipoxins, resolvins and protectins, while although Leukotrienes including LTB4 and Prostaglandins, including PGE2 and PGD2 perform as syndrome cascade enablers in acute phase while also participating in resolution of detrimental cascade and acute phase by inducing 15 - lipoxygenases to counteract the neutrophil migration which these also enable.

Specifically, Arachidonic Acid, activated in the acute phase, is programmed for SPM enabled acute phase resolution by being able to transition from acute phase activity to SPM enablement by moving from leukotriene production to lipoxin production. DHA is a signal that activates autacoid protectins, resolvins and maresins by excluding granulocytes and recruiting nonacute, and anti-inflammatory monocytes. Mast cellular entities and Macrophages engulf debris, pathogens, neutrophils, and other factors via draining lymphatic capability. Factors such as aspirin assist because these are able enhance conversion of omega-3 EPA and DHA into 18R and 17R Oxygenation products which are precursors to Resolvin D series and Resolvin E Series.

Polymorphonucleus neutrophils remove infiltrating pathogens during ephemeral duration of exhibition, particulate enabled by Macrophage resolution enabled SPM mediator activity, although in the extended duration exhibition of these neutrophils exhibit excessive tissue infiltration that can be diminished by hyaluronan, hyaluronic acid, although inhibition of PEMT and inhibition of glycolysis also diminishes synthesis

	<p>HIV1 promoter was prevented by TYMPyP4 but required coupling with Latency reversal factors to cause Apoptosis in vivo. BRACO19 was included with TMPyP4 as producing apoptosis when coupled with latency reversal capabilities. It is interesting to consider that inhibitors of iNOS and inhibitors of uncoupled NOS might be broadly affective in this context, as well as SP1 and the diverse factors presented in this compendium of analysis and research. The literature presents that SP1 performs as a switch that enables lytic transcription when integrated into the HIV1 promoter, while preventing SP1 integration into the HIV1 promoter causes latency. It is not clear if the means latent transcription or latency or inactivity. This same mechanism was observed for Epstein Bar EBV, HSV-1, and KSHV viral conditions. TRIM22 is known to cause silencing of HIV transcriptions by preventing integration of SP1 into the HIV1 promoter. B20. Information. 'Unlocking G Quadruplexes as Antiviral Targets.' Pharmacol. Rev. Volume 73. Pages 897 to 923. July 2021. Information. Small. e2104420. January 17, 2022. PMID 35037383.</p>	<p>of hyaluronan, and 16S as well as 17S Oxygenation products which are precursors for Resolvin E series and Resolvin D series. SPMS RvD3, RvD4 and RvD6 are known focus on leukocytes, activating extracellular signaling interactively with neutrophils and myocytes to enhance phagocytosis, perform antimicrobial activity and improve host defense.</p>
Folate Receptor Status	Folate receptor is upregulated during	Continued upregulation of folate receptors can occur during folate

	<p>folate inadequacy and by Vitamin D supplementation. Information. Proc Natl Acad Sci U S A. Volume 116. Number 35. Pages 17531 to 17540. PMID 31405972. Folate receptor is upregulated by homocysteine using stimulation of hnRNP E1. J Clin Invest. Volume 113. Number 2. Pages 285 to 301. January 15, 2004. PMID 14722620.</p>	<p>adequacy or in a way that causes a reliance, artificial or contrived reliance, upon folate, such that folate endocytosis is hyperactivated and upregulated even during inadequacy as well as even when typical levels of folate are exhibited. this enhance the biosynthetic pathways with which folate is linked, particularly upregulation one carbon pathways in a way that cause accumulation of folate and exacerbate the folate trap. Folate assists in enhancing the exhibition of choline in physiology through assisting recycling of homocysteine into methionine and then s adenosylmethionine. However, folate cannot replace deficient choline, Vintafolide, farletuzumab, as well as IMGN853 are existing or emerging modalities for downregulating folate receptor. upregulated folate can mimic choline availability, particularly when a reprogramming of the cellular entity has occurred to cause persistent upregulated folate endocytosis. Information. Ther Adv Med Oncol. Volume 7. Number 4. Pages 206 to 218. July, 2015. PMID 26136852. Folinic acid can circumvent occluded folate receptors and move across the circulatory membrane that separates brain from circulation, such that folate is absorbed through other mechanisms. This may be beneficial in autism. Folinic acid, or leucovorin, mimics the therapeutic effect of 5 FU in alleviating oncology of breast tissue. Information. Statpearls Website. Folinic Acid. Latest update July 16, 2021.</p>
Nanog	<p>Importantly, Bryostatins 1 is able to introduce apoptosis in breast tissue affected by highly pathogenic oncology, regardless of the status of P53 and regardless of the status of Protein Kinase C. These conditions also</p>	<p>A review of the literature presents voluminous information that confirms the role of Nanog, Sox, OCT4 and UTRF1 in this parthanatos context. PARP1 is A regulator of SOX2. FGF/ERK cause PARP to polyADPriboseylate itself, producing a PARP/SOX2 enhancement cascade. PARP1/SOX2 integration excludes</p>

potentiate activation of Nanog. A review of the commercial literature presents that Bryostatin 1 performs as protein kinase c agonist that causes nanomolar level expression of protein kinase c which results in protein kinase suppression. The activity towards protein kinase c and Nanog by Bryostatin is market as antiNonog immunoglobulin. Information. Anti Nanog Immunoglobulin (IE6C4). SC 293121. Santa Cruze Biotechnolog website, SCBT.com

Information. 'P53 and Protein Kinase C.' Int J Mol Med. Volume 1. Number 6. Pages 915 to 923. 6th Month, 1998. PMID 9852625.

Information.

"Regulation of P53." J Cancer Res Clin Oncol. Volume 123. Number 7. Pages 365 to 369. 1997. PMID 926587.

Information. EMBO J. Volume 21. Number 12. Pages 300 to 3008. 6th Month 17th Day, 2002. PMID 12065413.

Information.

Carcinogenesis. Volume 34 Number 7. Pages 1497 to 14507. July, 2013. PMID 23536578.

Pivotaly, a review of the integration loci for Nanog in the HPV LCR revealed that Nanog integration Loci occurred only in the in those HPV types that were causal of oncology. Information. Viruses. Volume 13. Number 8. Page 1482.

OXT4/SOX2 integration. This suggests that FGF or FGF2/ERK signaling enhance SOX2/PARP1 complex synthesis. SOX2/PARP1 complexes exhibition resultant of FGF2 or FGF balances stemness compared to differentiation. LIF and BMP4 maintain stemness even through cellular entity propagation without exhibition of differentiation. SOX2 integrates, along with OCT4 cooperation, into OCT/SOX enhancing genetic loci and promoters. OCT/SOX promoters occur in FGF4 and Nanog transcriptional regions or promoters, as well as in UTF1, SOX2 and OCT4 promoter regions. OCT4/SOX2/Nanog complexes both repress and activate transcription to maintain stemness. This nuance describes how PARP upregulation and signaling promote stemness but also results in upregulation of homocysteine that produces canonical apoptosis pathways that cause completely differentiate cellular entities to exhibit parthanatos modality of apoptosis as well as result in a persistent stemness in emerging pluripotent stem cellular entities that involves also upregulation of homocysteine such that in response, a cellular entity must exhibit strong pluripotency or escape apoptosis by upregulating BCL2 and inflammation pathways for survival such as proteolysis, S1P, S1P receptor upregulation, G Protein upregulation, all of which are linked to GSK3B upregulation. Upregulation of choline kinase and upregulation nSMase/aSMase produces phosphocholine which assist in survival pathway activation. Eventually, these conditions produce a stochastic potential for dissociation of the mitochondrial associated membrane which isolates Mitochondrial PEMT2 from endoplasmic reticulum supply of Ca²⁺, Phosphatidylserine and Phosphatidylethanolamine, although PI3K also sequesters Ca²⁺ away from the Mitochondria, resulting the

July 28, 2021. PMID 34452350.

The analysis becomes more interesting. Nanog, along with OCT4 and SOX2, coordinate to maintain embryonic stem cellular entities in an undifferentiated status, such that Nanog mRNA are only found the epiblast, but minimal levels are ascertainable in reproductive cellular entities, although fibroblasts involved in extracellular matrix, connective tissue and wound healing maintenance, repair and regeneration also exhibit low levels of Oct4, SOX2 and Nanog mRNA. Fibroblast growth factor 2 FGF2 is essential in stimulating Nanog expression. Breast, ovarian, cervical and renal oncology are typically inclusive of Nanog expression. Parthanatos is a version of cellular deterioration or a version of apoptosis which occurs when P53 becomes automatically upregulated, followed by P53 inhibition of pentose phosphate pathway to reduce availability of pyruvate while also inhibiting the pentose phosphate pathway at the same juncture which glycolysis is inhibited, glucose 6 phosphate dehydrogenase. The inhibition of the Pentose Phosphate pathway causes about 60 percent or more decrease in NADPH as well as impairs throughput of

characteristics obliteration of PEMT that accompanies, typically, oncology and disease. Information. Proc Natl Acad Sci U S A. Volume 109. Number 10. Pages 3772 to 3777. March, 2012. PMID 22362888.

The context presents that resultant of FGF/ERK signaling, PARP PolyADPRibosylates itself and this self PolyADPRibosylation enhances its completeness or duration of interaction with SOX2, such that SOX2 is less availability to complex with FGF4.

Nanog is correlated with pathogenic oncological transitions that enable movement or migration.

TWIST1/BMI1 activation by Nanog may be an essential activator of pathogenic oncology migration. Another pivotal observation in the literature is a comprehensive confirmation of the observations made in this compendium of research. NANOG transcription activation occurs when P53 mechanisms of control are suppressed, while P53 is known to be upregulated when PEMT expression and catalysis is impaired or inhibited. These explain, clearly, why inhibition of PEMT causes upregulation of PEMT and when P53s suppression of a number of pathways, such as pentose phosphate pathway and glycolysis, becomes surmounted, a condition known as aerobic glycolysis occurs in which differentiation and proliferation of a cellular entity becomes commandeered by pathogens or pathogenic conditions to produce disease. Essential to emergence, persistence, progression, imparting of detriment, eluding of immunology and eluding of therapeutics by such disease is the dissociation of the endoplasmic reticulum from the mitochondria, preventing the ability to exhibit mitochondrial rescues pathways including programmed exhibition of cellular deterioration. Information. Oncology Biol Ther. Volume 17. Number 1. Pages 1 to 10. January 2016. PMCID PMC4848008.

five carbon sugar synthesis that is linked to production of nucleotides. Resultantly, the 1 million or more instances of DNA repair that can occur in any or all cellular entities each day exhibits inadequacies of nucleotides and substrate to conduct genomic repair. This cause PARP to attach loci of genetic impairment and signal persistently until enough nucleotides and other substrate are recruited to the same locus by PARP catabolism of NAD⁺ to remove the ribose, followed by attachment of the ribose to local molecules. This attachment of ribose causes a gradient upon which molecules for repair are recruited and produces nicotinamide products that require nicotinamide methyltransferase to attach detoxify by attaching methyl groups to the nicotinamide, resulting in production also of homocysteine at increased levels. The pauses and incomplete performance of genetic repair results in incomplete repair and exhibition of nonhomologous repair because adequate levels of NAD⁺ are required to enable PARP to disassociate from the repair locus to allow Homologous Repair or error free repair to occur. The required levels of NAD⁺ become so substantial that pyruvate is directed away from

the diverse other pathways for energy synthesis and metabolism, as well as away from protein synthesis, toward, instead, lactate because NADH and Pyruvate are used by lactate dehydrogenase to produce NAD⁺ and: Lactate anion. The levels of genetic repair required, thus, can cause NADH and pyruvate to become commandeered. This process causes inhibition of glucose 6 phosphate at even more substantial levels because NAD⁺ is required by glucose 6 phosphate. This also causes levels of s adenosyl homocysteine, a primary inhibitor of PEMT, to increase because s adenosyl homocysteine hydrolase that produces homocysteine from s adenosyl homocysteine also requires NAD⁺. Homocysteine generally inhibits PEMT and increase BAX which causes cellular entities to spontaneously exhibit apoptosis although when the mitochondrial membrane is separated from the endoplasmic reticulum, the mitochondrial membrane chaperoned apoptosis pathways cannot be completed. Resultantly, a version of apoptosis occurs which is presented as parthanatos. Parthanatos is characterized by deterioration or apoptosis of already completely differentiated cellular

entities along with exhibition of pluripotent cellular entities that emerge in a somewhat immortal phenotype, which eventually cause change of tissue toward oncological susceptibilities although these may enable resilience in the immediate context. A review of the literature presents voluminous information that confirms the role of Nanog, Sox, OCT4 and UTF1 in this parthanatos context. PARP1 is A regulator of SOX2. FGF/ERK cause PARP to polyADPriboseylate itself, producing a PARP/SOX2 enhancement cascade. PARP1/SOX2 integration excludes OCT4/SOX2 integration. This suggests that FGF or FGF2/ERK signaling enhance SOX2/PARP1 complex synthesis. SOX2/PARP1 complexes exhibition resultant of FGF2 or FGF balances stemness compared to differentiation. LIF and BMP4 maintain stemness even through cellular entity propagation without exhibition of differentiation. SOX2 integrates, along with OCT4 cooperation, into OCT/SOX enhancing genetic loci and promoters. OCT/SOX promoters occur in FGF4 and Nanog transcriptional regions or promoters, as well as in UTF1, SOX2 and OCT4 promoter regions.

OCT4/SOX2/Nanog complexes both repress and activate transcription to maintain stemness. This nuance describes how PARP upregulation and signaling promote stemness but also results in upregulation of homocysteine that produces canonical apoptosis pathways that cause completely differentiated cellular entities to exhibit parthanatos modality of apoptosis as well as result in a persistent stemness in emerging pluripotent stem cellular entities that involves also upregulation of homocysteine such that in response, a cellular entity must exhibit strong pluripotency or escape apoptosis by upregulating BCL2 and inflammation pathways for survival such as proteolysis, S1P, S1P receptor upregulation, G Protein upregulation, all of which are linked to GSK3B upregulation. Upregulation of choline kinase and upregulation nSMase/aSMase produces phosphocholine which assist in survival pathway activation. Eventually, these conditions produce a stochastic potential for dissociation of the mitochondrial associated membrane which isolates Mitochondrial PEMT2 from endoplasmic reticulum supply of Ca²⁺, Phosphatidylserine and Phosphatidylethanolamine, although PI3K also sequesters Ca²⁺ away

from the Mitochondria, resulting the characteristics obliteration of PEMT that accompanies, typically, oncology and disease. Information. Proc Natl Acad Sci U S A. Volume 109. Number 10. Pages 3772 to 3777. March, 2012. PMID 22362888.

The context presents that resultant of FGF/ERK signaling, PARP PolyADPRibosylates itself and this self PolyADPRibosylation enhances its completeness or duration of interaction with SOX2, such that SOX2 is less availability to complex with FGF4. Nanog is correlated with pathogenic oncological transitions that enable movement or migration. TWIST1/BMI1 activation by Nanog may be an essential activator of pathogenic oncology migration. Another pivotal observation in the literature is a comprehensive confirmation of the observations made in this compendium of research. NANOG transcription activation occurs when P53 mechanisms of control are suppressed, while P53 is known to be upregulated when PEMT expression and catalysis is impaired or inhibited. These explain, clearly, why inhibition of PEMT causes upregulation of PEMT and when P53s suppression of a number of pathways, such as

	<p>pentose phosphate pathway and glycolysis, becomes surmounted, a condition known as aerobic glycolysis occurs in which differentiation and proliferation of a cellular entity becomes commandeered by pathogens or pathogenic conditions to produce disease. Essential to emergence, persistence, progression, imparting of detriment, eluding of immunology and eluding of therapeutics by such disease is the dissociation of the endoplasmic reticulum from the mitochondria, preventing the ability to exhibit mitochondrial rescues pathways including programmed exhibition of cellular deterioration.</p> <p>Information. Oncology Biol Ther. Volume 17. Number 1. Pages 1 to 10. January 2016. PMID PMC4848008.</p>	
<p>Agrin extracellular matrix protein status and Interleukin 11 status</p>	<p>Agrin performs as a mechanosensitive sensor in the extracellular matrix and connective tissue, such that its sensing of the stiffness or flexibility of matrix cause signal transduction into the cellular entities, fibroblasts and other cellular versions in the area adjacent to matrix. This mechanosensing activity is described in other factors in this list also. Importantly, Agrin changes both the characteristics of cellular entities interfacing extracellular matrix and extracellular matrix, although an</p>	<p>HIPPO enablers and HIPPO signaling cascade inhibits the YAP/TAZ transcription factor cascade. The reason that these seem new may be that the literature does not adequately link these factors to metabolic pathways. Essentially, HIPPO diminishes pathways that upregulated choline kinase alpha, S1P, Phosphocholine exhibition, G Protein Coupled Receptor activation, GSK3B, BCL2 and Proteolysis. These are all survival pathways and are exhibited during distress, challenge, xenobiotic exposure, toxicity, or response to an immunological challenge. As well as inhibition of PEMT and upregulation of homocysteine.</p> <p>Also, YAP/TAZ respond to mechanical input and translate these into biological responses or signal</p>

important impedance to its ability to enable tissue, mitotic, extracellular matrix and connect tissue stability continues to be constituted by choline inadequacy and inhibition of PEMT, both of which challenge the existential aspects of cellular entities and impair the ability of fibroblasts to produce laminin, hyaluronan and collagen, as well as agrin, all of which become exhibited in typical or optimal connective tissue and extracellular matrix.

This list and these analysis presented in other clinical indicator analyses that extracellular matrix may be a hidden, underconsidered factor in oncology because a deteriorated matrix or impaired plasticity in the extracellular matrix might result in signaling cascade to change cellular entity levels and characteristics near the matrix, affecting both mitotic activity persistently during such deteriorated statuses but also causing a complicated pattern of mitotic activity during cytotoxic therapy for oncology or other disease. Certainly, even this complexity might be enhanced when there is inadequate lipids and cholesterol available to maintain existing cellular membranes as well as enable cellular division from already deficient cellular entity membranes required to

transduction activity, extended to the nucleus where changes to gene expression occur resultant of mechanical interactions, changed, distress, pressure, stiffness, flexibility or changes to plasticity. Agrin activates Lrp4 lipoprotein linked receptor 4, thereby activating Muscle specific tyrosine kinase MuSK, resulting in complex at the neuromuscular junction. Agrin is essential to pioneering development and regeneration of organs and tissues regarded as unable to be regenerated. Moreover, Agrin is linked to organization of tissues, cellular entities and neurons in regeneration of cardiac organ and may be involved in a similar role in much of mammalian organisms. Information. Oncology. Number 10. Volume 2. Page 45. 2018. PUBMED 29415512. Information. NPJ Regenerative Medicine. Number 3. Article Number 6. Volume 2018.

The literature observes that TGF4 is involved in fibrotic response to impairment or injury, resulting in myofibroblast differentiation using already exhibited fibroblasts at the locus of injury and includes differentiation of endothelial cellular entities at the locus of injury. Excessive matrix production occurs in this context along with secretion of matrix cross link producing enzymes. This promotes scarring and enables production and exhibition of stiff, collagen dense material into the extracellular matrix. This suggests that without the assistance of Interleukin 11, scar tissue pervasively results in deterioration of matrix plasticity, changing signals transduced from the matrix to cellular entities and tissues including affecting agrin signaling. This demonstrate how less than optimal regeneration, scar tissue causes accumulating and persist signaling that changes biomechanical processes including mitosis, mesenchymal transitions, and other changes.

produced new cellular entities that are adequate for physiological structure as well as adequate to diminish the natural exhibition of mitotic signals that occurs once cellular entities are encountered on all adjacent interfaces by either matrix or other cellular entities. It is also likely that extracellular matrix is aware of or senses aspects of its structure that are not covered by cellular entities, resulting in upregulation of mitotic signaling.

However, generally Agrin was incipiently encountered in a type of aquatic organism known as a ray, being found in the electricity generating organs of aquatic organisms generally. Agrin causes the focal aggregation of acetylcholine receptor exhibiting cellular entities. This suggests that agrin participates in transduction of signal and transduction of current in physiology. Agrin was found not only stimulate aggregation of acetylcholine receptor, but also causes patching of laminin to heparan sulfate proteoglycan, colocalizing these patched conjugates with Acetylcholine receptors. However, laminin patching to HSPG continues to occur when inhibition of acetylcholine receptor aggregation occurs. Information. J Cellular Biol. Volume 111. Number 3. Pages 1161

Interleukin 11 promotes dissociation of matrix proteins at the locus of injury or prevents these from occurring, which presents that context of Agrin which performs such dissociation among particular proteins, links other proteins, produces an ordered organized response to injury, prioritizes repair processes, aggregates acetylcholine receptors, organizations the neuromuscular junction and enables exhibition of the regenerative blastema. Information. Sci Adv. Volume 7. Number 37. Article eabg6497. September, 2021. PMID 34516847. Information. Circulation. Volume 142. Number 9. Pages 868 to 881. 6th Month, 2020.

The produced Matrix Electrolyte Powder, thus, promotes healthy matrix by providing electrolytes that manage the integration affinity of matrix proteins including Agrin. Its exhibition of Potassium suggests that it also manages the stability G4 quadruplexes. Information. Matrix Electrolyte Powder. Biopure.

The relevant article presents that Wnt, G Protein Coupled Receptors and EGF are all able to commandeer YAP/TAZ to enable survival promotion pathways to become exhibited.

Hematopoietic or blood stem cells develop in particular areas or notches known as hematopoietic niches. Agrin is essential as a signal that controls survival and differentiation of hematopoietic stem cellular entities. This is pivotal, since it explains instances of spontaneous blood diseases and oncology found near and incident to injury or impairment. Multipotent nonhematopoietic stem cellular entities express agrin, along with expression of agrin by differentiated osteoblasts lining the endosteal bone surface. Lin Sca1+ CKit+ (LSK) cellular entities express alpha dystroglycan receptor for agrin. Lin CKit+ cellular entities are deficient when nonhematopoietic stem

to 1170. September 1, 1990. PMID 2167896.

An example of therapeutics and pathogenic factors linked to multiple instances of DNA in one cellular entity is not presented here, is presented in the compendium of research linked to this analysis, and is augmented by the literature's presentation that there are typically nearly 1000 mitochondria in each cellular entity and more than 60% of these mitochondria must be impaired to exhibit mitochondrial syndrome, suggesting that a similar level of impaired mitochondria must be exhibited to result in deterioration of apoptosis pathways. Many of the apoptosis pathways known of a this instance require functional mitochondria. Impaired mitochondrial DNA is presented as a causal factor in deteriorated mitochondrial function with an individual cellular entity exhibiting heterogenous DNA in its mitochondria in which some mitochondria exhibit impaired DNA and other Mitochondria exhibiting unimpaired DNA, being known as heteroplasmic. Homoplasmic cellular entities exhibit impaired mitochondrial DNA in all of the available mitochondria. Information. "Mitochondrial Base Editing." Nature Communications.

cellular entities when agrin is deficient. Agrin transcriptional ablation produced CD34+CD135+LSK cellular entities that exhibited apoptosis and impaired hematopoiesis. Agrin sufficient stroma repaired these characteristics.

These suggest that Hematopoietic disease as well as oncology may be linked to impaired matrix, Agrin inadequacy, inflammatory signaling by Agrin, as well as impaired nonhematopoietic stem cellular status. Most importantly, agrin is emerging as systemic organizer of development at the tissue, organ, connective tissue and even hematopoietic level. Processes that diminish Agrin availability and cause inflammatory signaling by Agrin, may be linked to a remarkable percentage of diseases accordingly, such that managing agrin and factors that affect agrin may be enable therapeutic management of a diverse and expansive array of the factors that are essential to disease. Information. Blood. Volume 118. Number 10. September, 2011.

The literature presents that agrin is essential for epicardial epithelial

Heparin and Heparin Sulfate inhibit Agrin enabled acetylcholine receptor focal aggregation. The same study found that typically the effects of Agrin are reversible.

Polyanion such as dextran sulfate diminish which exhibit negatively polarized factors inhibit Agrin integration into ligands by about 60% in culture.

A study of diminished Ca²⁺ availability which is an integral and ubiquitous factor in disease found that inadequate Ca²⁺ results a 30% decreases in Agrin integration activity, presumably including diminished Agrin integration into Ligands. Pivotaly, these explain why persistent iNOS signaling and

Volume 13. Number 1. 2022.

Agrin invokes or enhances the YAP, promoting YAP activation of Integrin Focal Adhesion catalysis as well as upregulating Lrp4/MuSK receptor enabled signaling cascades. Together, these downregulate LATS1 and LATS2. Some of the literature suggests that Agrin promotes or enables oncology but as the information here will present, Agrin upregulates cellular division in response to matrix deterioration, and although this enhances proliferation of pathways and cellular entities that sustain matrix regeneration and repair, the signaling pathway cascade invoked by agrin can be commandeered by pathogenic process. Information. Cellular Rep. Volume 18. Number 10. Pages 2464 to 2479. March, 2017. PMID 28273460.

Agrin is a 210kDA basal lamina linked heparan sulfate proteoglycan and Agrin is not only activator of YAP but also enables YAP/TAZ. J Int Med Res. Volume 49. Number 5. Article 03000605211009722. May, 2021. PMID 34018826.

Although the literature links Agrin to angiogenesis in oncology and pioneering as well as regenerative

persistent depletion of Ca²⁺ otherwise by PI3K results in such diverse pathology, producing impairment of Agrin ligand integration which impairs epithelial cellular entity transition to mesenchymal phenotype to enable regeneration of internal areas of tissues and organs. Pivotaly, also, these explain the deterioration of organs in physiology that typically occurs with aging as iNOS signaling and PI3K signaling can occur during choline deficiency, viral exposure, electricity exposure, wireless communications exposure, and during inhibition of PEMT. Information. The Journal of Neuroscience. Volume 10. Number 11. Pages 3576 to 3582. November, 1990.

Mini Agrin exhibits reduced ligand activation and in models of regenerative therapy including repair of muscular dystrophy, congenital muscular dystrophy, Mini agrin along with apoptosis inhibition therapeutically, together resulted in improved regenerative therapy. Muscular dystrophy was able to be improved reliably and therapeutically by inhibiting BCL2 or BCL2 levels compared to BAX or BAK levels, which are foundational factors in apoptosis potential, along with instrumentation of Mini agrin. Mini agrin along with Omigapil also produced amelioration and improvement of muscular dystrophy through regeneration of tissues and function. Increase in muscle force levels, diminished fibrosis, prevention of tissue atrophy, each were characterizations of improvement using apoptosis inhibition along with mini agrin. These clearly demonstrate that deterioration of matrix is an important and increasingly ubiquitous factor in detrimental aspects of aging and diseases otherwise. Information. EMBO Mol Med. Number 3. Pages 465 to 479. 2011.

development, the literature clearly presents that Agrin is dispensable not essential without alternative capabilities in each of these. There are other mechanisms by which each of these can occur. Information. Cardiovascular Med. Volume 8. Number 810477. January, 2022. PMID 35174224.

The literature presents that Agrin is essential in the transition of epicardial cellular entities into the mesenchymal phenotype required for migration from the cardiac epithelium to internal areas of the cardiac organ to support growth and regeneration. Basement Membrane Proteoglycan Agrin exhibition in particular was correlated with areas of Matrix at which enhanced or particular propensity for epicardial cellular entities to migrate to internal areas of the cardiac organ to promote regeneration and repair. Inhibition of Agrin availability resulted in downregulation of transition of epicardiocytes to mesenchymal phenotype while, interestingly, such inhibition of Agrin availability also downregulated Wilm's Neoplasm 1 protein. This transition was experimental explored, revealing that Agrin cause epicardial cellular entities to downregulate B Catenin and upregulate pFAK at

Agrin promotes exhibition pathway activation by YAP/TAZ. YAP/TAZ are linked to oncology but are also enable remarkable levels of organ and tissue regeneration in those tissues and organs regarded as having low or no capacity for regeneration otherwise. Information. Nature Reviews Molecular 'Cellular' Biology. Volume 20. Pages 211 to 226. 2019. PMID 30546055.

Downregulationg CRAD in soft substrates produces uipregulated YAP sequestration to the cytoplasm, such that cytoplasmic YAP represses NANOG and OCT4 activity. Repression of NANOG and OCT4, thus, in soft tissue, or with soft substrate, promotes stemness and mesenchymal transitions linked to movement of cellular entities. CRAD levels are correlated with phases of oncology, at least in this model of colorectal disease. The histone deacetylase inhibitor Trichostatin A as well as H3K27me3 methyltransferase EZH2 inhibition using EPZ 6438 rescues NANOG and OCT4 upregulation imposed by CRAD upregulation as well as caused by YAP translocation to the Nucleus, which contrasts with repression imposed upon Nanog and OCT4 when YAP is sequestered to the cytoplasm by decreased CRAD. This paragraph requires summarization because of the ambiguity of the article in which it is presented which oppositely presents linkages between CRAD, YAP, NANOG and OCT4 in different paragraphs.

The summarization of these in the article suggests that CRAD decrease, results in YAP sequestration to the cytoplasm, which results in colorectal cellular stemness upregulation resultant of upregulated NANOG and upregulated OCT4 expression

<p>focal adhesion loci, stimulates dystroglycan aggregation along with causing dystroglycan accumulation in the golgi apparatus.</p>	<p>Importantly, this clarification of NANOG and OCT4 promoting of stemness is important because ERK complexes with FGF to promote PARP1 signaling and PARP1 complexes with SOX2 to exclude SOX2 from integrating into the NANOG/OCT4 complex. The integration of SOX2 enhances the transcriptional activation activity enabled by NANOG/OCT4 complexes. Information. “Substrate Rigidity.” Cellular Reports. Volume 38. Issue 7. February 2022. PMID 35172140.</p>
<p>Agrin availability inhibition produced dispersal of dystroglycan, causing basement membrane deterioration as well as causing inhibition of mesenchymal transitions by epicardiacocytes, suggesting that that cardiac tissues have a mechanisms of inherent regeneration, agrin activates this regeneration, and the oscillating mechanisms which controls this regeneration is the extracellular matrix and proteins within the extracellular matrix. This counteracts or debunks the widely held opinion that cardiac tissue does not generate. This compendium of research also revealed that the diminished exhibition of cardiac tissue mitosis and exhibition of multiple copies of DNA was the result of a pathogenic mechanisms in which mitosis may be becoming averted after DNA replication occurs. Information. Development. Volume 148. Issue 9. Article dev197525. May, 2021.</p>	<p>Agrin/LRP4/MuSK complexes perform tyrosine phosphorylation of Rapsyn, enabling Rapsyn self phosphorylation and enabling E3 Ligase ubiquitinase activity performed by Rapsyn, all of which is interesting because small mammal models of Rapsyn ablation prevents development after birth while impaired function of Rapsyn in hominids links Acetylcholine receptors to the cellular cytoskeleton as well as enables E3 Ligase activity. Impairment of Rapsyn catalysis is linked to myasthenic syndrome of congenital. Information. eLife. Volume 2019. Volume 8. Article 349180. September, 2019.</p>
<p>Oral oncology pathogenic activity can involving agrin enabled enhancement or activation, while inhibition of agrin inhibits expansion, growth, colony</p>	<p>Agrin acetylcholine receptor aggregation occurs at the neuromuscular junction.</p> <p>Agrin enables limbal Stem cellular entity proliferation and promotes corneal wound healing. This is enabled by HIPPO pathway signaling including YAP. Limbal stem cellular proliferation was linked to P63a and not particularly Keratin 12. Agrin upregulation of wound healing and upregulation of corneal epithelium occurred in vivo. YAP1 translocation to the nucleus was enabled by dephosphorylation of YAP1 by Agrin resulting also in expression of Cyclin D1. Invest Ophthalmol Vis Sci. Volume 761. Number 5. Page 7. May 2020. PMID 32392315.</p>

proliferation and neoplasm exhibition by causing diminished phosphorylation of FAK, Cyclin D1 and ERK. Information. British journal of oncology. Volume 118. Pages 1628 to 1638. 2018.

The literature observes that Agrin causes dystrophin and Glycoprotein to dissociate, disrupting exhibition of complexes between these two factors, resulting in stimulation of YAP and ERK signaling upregulation. This promotes substantial regeneration of mammalian cardiac organs and tissues even after only 1 instrumentation of agrin. Information. Nature. Volume 547. Number 7662. Pages 179 to 184. July, 2017. PMID 28581497.

The data to support the missing link in oncology as being inadequate choline, a focus of cellular resources to support membrane phospholipid availability, and agrin signaling aberrance that increases with scarring, persistent impairment and unresolved disease, along with the spectra of plasticity impairments that emerge when PEMT is inhibited, is expanding and becoming more specifically indicative of why therapeutics are not 100 percent effective.

HIPPO typically inhibits YAP and TAZ. AGRIN, HIPPO, YAP and TAZ are linked to a network of nucleus exhibited biomechanical signals that perform as semaphore for movement, migration, mitosis, meiosis, and other activity. STK4 and LATS1 as well as LATS2 performs along with adapter proteins such as Salvador and MOB, all with a homeostasis promoting role that inhibits potential for oncology. LATS1 and LATS2 activation results in LATS1 and LAT2 phosphorylation of YAP/TAZ at their HXRXXS moieties. This results in sequestration of YAP/TAZ to the cytoplasm. YAP/TAZ interact with TEF at its TEA domain, known as TEAD transcription factors. TEAD activation by YAP/TAZ produces a cascade of hundreds of survival enhancing transcriptional factors.

The literature observes that API produces upregulation of choline kinase alpha. This pivotal linkage suggest that the API moiety accompanying cFos has an integration locus in the Choline Kinase Promoter beginning at position negative 875 for 11 or 12 sequences in the negative direction. Information. Biochim Biophys Acta. Volume 1171. Number 9. Pages 1148 to 1155. 2007. PMID 17728180.

Olive oil is known to cause apoptosis among in oncology cellular entities. However, instrumentation of choline prevents such apoptosis occurring, Upregulation of phosphatidylcholine occurs during extra virgin olive oil instrumentations and includes exhibition of polyphenols. Choline instrumentation prevents upregulation of choline kinase, and when choline kinase is not upregulated, the ability of extra virgin olive oil to disrupted the CDP choline pathway becomes likewise

Correlatively, the literature observes that not only is agrin and interleukin 11 linked to dissociation of particular proteins, linkages of particular proteins and inhibition of fibrosis essential in maintaining plasticity, but Agrin/metalloproteinase 12 axis produces a mechanically competent microenvironment in the performance of wound healing. Information. Nat Commun. Volume 12. Number 6349. 2021. PMID 34732729.

Agrin is enriched in the incipient phase of wound healing, while in particular tissues it is known that PEMT is diminished in the incipient phases of wound healing, performing in the role of mechanoactivity programming for keratinocytes by causing keratinocytes to enhance traction stress, stiffness, and the ability to respond to bulk substrate rigidity with exhibition of fluidic velocity fields. Agrin enhances actomyosin linkages, responsively to geometric stress and responsively to force, correlative to exhibition of injury, producing changes to cytoskeletal structure. Metalloproteinase 12 is an essential element of this mechanobiological programming performed by Agrin.

Moreover, olive oil is emerging as a therapeutic vector for oncology, and the

ameliorated. Olive oil also causes groups with the highest levels and most extended duration of instrumentation to have the lowest incidence of oncology. Information. Choline kinase is somewhat or literally required to be upregulated in much if not all oncology. Oleocanthal is observed to activate cMet pathway to cause apoptosis, although extra virgin olive oil activates the cFOS pathway which seems to cause an oncology prevention role compared to the other moiety of AP1 which is a survival signaling pathway that activates choline kinase alpha to be beneficial in the acute phase but can become pathogenic in extended duration by promoting survival even when conditions do not require such enhanced resilience. However, it is well known that homocysteine increases cause apoptosis enhancing changes in cellular entities and survival signaling is exhibited to counteract these by increasing BCL2 comparatively Bax or Bak. Choline instrumentation provides methyl groups, while olive oil instrumentation provides lecithin as phosphatidylcholine to supply membranes with phosphatidylcholine that downregulates requirement of choline kinase alpha and CDP Choline pathway synthesis of phosphatidylcholine. Similarly, the methyl groups enable activation of PEMT which produces DHA enriched phosphatidylcholine and DHA as well as other omega-3 and polyunsaturated fatty acids, all of which are cytotoxic specifically to oncology exhibiting cellular entities. Inhibition of choline kinase selectively causes apoptosis in oncology cellular entities. World J Gastroenterol. Volume 15. Number 15. Pages 1809 to 1815. 2009. PMID 19370776. Information. Curr Pharm Des. Volume 17. Number 8. Pages 805 to 812. 2011. PMID 21443483. Information. Curr Cancer Drug Targets. Volume 8. Number 8. Pages 709 to 719. 2008. PMID 19075594. Information.

<p>literature is very responsive in linking polyphenols as well as oleocanthal as causal factors in this efficacy in oncological therapy. However, although it is intuitive in constructing structures that foundational nuances of structure guide physiological development in a more comprehensive context, the importance of foundational dynamics in matrix interactivity with confluence characteristics of cellular entities are not always including in diagnostic and therapeutic analysis..</p> <p>Correlatively, studies indicate that the effectiveness of olive oil as an oncological therapy that causes apoptosis specifically in cellular entities exhibiting oncology pathology becomes diminished when choline supplementation as added to therapy or in vitro. Similarly, the literature observes that lysosomal membranes are susceptible and diminished in stability when oncology is exhibited in cellular entities. These susceptibilities emerge because oncology is comprised, pervasively, of incipient inhibition of PEMT and obliteration of PEMT2 function because the mitochondria becomes estranged from the endoplasmic reticulum. Although P53 is upregulated to introduce stability when PEMT is</p>	<p>Olives and Olive Oil in Health and Disease Prevention. Chapter 55. Volume 2021. Pages 661 to 669. 2021. Information. Nutrients Volume 14. Number 4. Pages 908. 2022. Information. Clin Oncology Res. Volume 19. Number 9. Pages 2310 to 2318. 2013. Information. Phytother Res. Volume 34. Number 11. Pages 2820 to 2834. 2020. PMID 32449241. Information. Curr Oncology Drug Targets. Volume 8. Number 8. Pages 709 to 719. 2008. PMID 19075594. Information. Curr Pharm Des. Volume 17. Number 8. Pages 805 to 812. 2011. PMID 21443483.</p> <p>Imperatively the effects of homocysteine are major enablers of the hidden nuances of agrin signaling including upregulation of fibronectin deposits in scar tissue, cardiac tissue and other tissue, as well as deterioration of matrix, apoptosis that depletes cellular entity density per micrometer to cause iNOS and persistent upregulation of mitogenic signaling.</p> <p>The literature observes that Agrin is essential for survival of monocytes or monocytic cellular entities. Information. PMID 22517892.</p> <p>Diabetic neuropathy exhibits decrease in the Agrin fraction of complete heparan sulfate proteoglycan levels, clearly linking persistent and advanced pathology, again, with deterioration of the structural nuances of biology, regenerative downregulation and ameliorated repair capabilities. Information. Exp nephrol. Volume 9. Number 3. Pages 214 to 222. 2001. PMID 11340306.</p> <p>An object of the preceding paragraphs involving EVOO or extra virgin olive oil supplementation was not only to confirm the role of olive oil in ameliorating oncology, ameliorating risk for oncology and link diverse patterns in disease with inadequacies of EVOO metabolites. Such object, instead, included</p>
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inhibited, abrogation of PEMT contribution to cellular membrane phosphatidylcholine results in upregulation of the CDP Choline pathway.

Choline kinase alpha, the first enzyme in the CDP choline pathway becomes upregulated and produces increased levels of phosphocholine that is used as substrate for pervasive disease and is directed toward increased levels of proteolysis which requires ATP and which recycles the choline to be directed again to choline kinase attachment of ATP to choline. This process is important because Phosphocholine is directed toward subsequent nuances of the CDP choline pathway to produce unenriched plasticity impairing versions of phosphatidylcholine without the focused antiinflammatory and plasticity enhancing fraction of phosphatidylcholine produced by PEMT.

The siphoning off of phosphatidylcholine toward proteolysis and toward survival signaling through S1P, S1P lyase, G protein coupled receptors, GSK3B, S1P receptors and other mechanisms, explains why cellular entities, although having focus on the CDP Choline pathway, continue to exhibit deficient membranes

establishing a priority of assuring the foundational material exhibited in cellular structure as a nuances of sustained health and diminished risk. The causal link between EVOO and Agrin availability as a beneficial nuance of EVOO therapy provide some such perspective. However, a study of cardiovascular disease clearly links ceramide increases causally to exhibition of cardiovascular disease, which confirms the well known role of ceramides in cellular membranes as a foundational structural signal for apoptosis of cellular entities. Most importantly, EVOO supplementation, or extra virgin olive oil supplementation, was found to diminish the risk of cardiovascular disease by diminishing ceramide levels. Correlatively, EVOO or extra virgin olive oil, causes expression of Agrin, diminishes ceramide levels, causes apoptosis of cellular entities exhibiting oncology in a way that is prevented by the addition of choline to nutritional regimen.

These clearly present that the foundational nuance of pathology are deterioration of the production, availability and structural integration lecithin or phosphatidylcholine, choline, PEMT enzyme production of enriched phosphatidylcholine, all of which results in supplanting of these for upregulation of the CDP Choline pathway which integrates recycled choline into unenriched phosphatidylcholine as a nuance of inflammatory response or response to methyl group inadequacy. These also clearly present that the complexity in managing disease as well as the risk and potential for unsuccessful cytotoxic therapy in oncology therapy has the essential component of inadequate supply of material to constitute cellular structure. This inadequacy diminishes Agrin availability or diminishes extracellular matrix, each in a way that ameliorates agrin

such as in impaired lysosomal function. Lysosomal impairment also results in impaired exhibition of autophagy which would causes pervasive oncology to be abated, although S1P Lyase and particular ubiquitinases can causes an escape from such autophagy in resistant oncology.

Thus, the reason that olive oil is effective in therapy is that it supplies a larger fraction of lecithin than choline alone, directly supplying phosphatidylcholine to cellular membranes, which causes choline kinases upregulation to be diminished and choline kinase upregulation no longer is efficient at supplying phosphocholine that is a ubiquitous enabler of disease, pathogens, c reactive protein activation, low level inflammation, complements immune system activation, platelet activation and supplier of energy for proteolysis.

Foundationally, olive oil supplies choline to help alleviate inadequate cholesterol and lipids required to sustain cellular membranes. Pervasively, therapeutics and care have prioritized avoiding cholesterol and avoid lipid obtainment, thus exacerbating a core cause of pathology.

Clinicians using cytotoxic therapy often reach a paradox in which the therapy is

signaling, causes impaired PEMT function, upregulates choline kinase, and requires survival signaling to prevent complete deterioration of anatomical structure.

iNOS expression, S1P synthesis from upregulated ceramide, S1P receptor activation including G Protein Coupled Receptor Activation, ATPase, GTPase, GSK3B activation, and S1P Lyase along with upregulated Proteolysis, increase in BCL2 to counteract BAX/BAK, all are nuances of such inflammatory pathways, which culminated in diminishing plasticity by omitting PEMT production of adequate fraction of phosphatidylcholine. The advancing nuances of change linked to this syndrome include the eventual dissociation of the mitochondria from the endoplasmic reticulum, preventing supply of Ca²⁺, Phosphatidylserine, and Phosphatidylethanolamine from the endoplasmic reticulum to the mitochondria, such that merely diminished PEMT becomes obliteration of PEMT2 function in the mitochondria. Information. "Plasma Ceramides in the Mediterranean Diet." Circulation. Volume 135. Issue 21. May 23, 2017.

This important conclusion presents a very useful context to understand the effects of iNOS, uncouple NOS, phospholipase D expression and phosphatidylcholine specific phospholipase c, all of which are upregulated responsively to environmental influences including electrical fields, wireless fields, environmental particulate and atmospheric particulate. Each of these participate in producing the deterioration and catabolism of membrane phospholipids, particularly phosphatidylcholine, similar to the dysbiosis which occurs when choline, phosphatidylcholine, and lecithin

causing what P53 is designed to prevent, which is the deterioration of biological structure to the point at which physiology deteriorates. Choline supplementation, thus, can adversely affect cytoxic therapy by causing cellular entities to become more resilient, and although the literature observes choline obtainment being correlated with oncology in some contexts before therapy, typically and studies observed by this analysis, choline obtainment is linked to improved prognosis and improved outcomes.

The complexity here is definitely that logically, therapeutically and analytically, uncoupling Argin and Matrix status from nuances of confluence and from foundational inadequacies of cellular membranes, produces oscillating mechanisms that can sense therapies being applied and result in sustained exhibition of disease. This particularly so because it is the objective of physiology to prevent itself from being deteriorated.

Agrin and Matrix supplementation, along with supply of choline and supply of lipids and cholesterol utilized in cellular membranes, thus, should either by foundational therapies or may have to be delayed until after

are inadequate in nutrition and not produced de novo by the function of PEMT in physiology.

S Methyl methionine sulfonium chloride has been utilized to promote regenerative repair. S methylmethionine sulfonium is an exogenously originated substrate for BHMT2 which has no known substrate in physiology otherwise. However, having a strong aroma, the literature observes that filtering s methyl methionine sulfonium for activate metabolites provides 2S,4S phenylthiazolidine 4 Carboxylic Acid, as well as 2R,4S phenylthiazolidine 4 Carboxylic Acid, as well as 2S,4R 2phenylthiazolidine 1,3 Carboxylic Acid, and as well as 2S,4R 2 phenylthiazolidine 1,3 Carboxylic Acid, as well as 2S,4S 2 phenylthiazolidine 1,3 Carboxylic Acid, as well as as well as 2R,4R 2 phenylthiazolidine 1,3 Carboxylic Acid, as well as 2R,4S 2 phenylthiazolidine 1,3 Carboxylic Acid. These phenylthiaolidines enhanced proliferation, survival during ultraviolet light exposure, collagen type I and MMP mRNA, presenting mechanisms of regenerative repair and depletion of scar tissue. Information. "Derivatives." Biomolecules and Therapeutics. Volume 26. Number 3. November, 2017.

S-methyl methionine sulfonium also exhibits antineoplastic activity in hepatic oncology. Information. Int J Environ. Res Public health. Volume 18. Number 18. Page 9726. 2021. PMID 34574650.

cytotoxic therapy in particular contexts. However, regenerative therapy that includes Agrin, Matrix, Cellular Membrane assuring might be used to rescue therapeutics.

The empirical nature of these analysis suggests that these empirical nuance of disease such be priorities, although it is known that cytotoxic therapies can be pervasively exhibited is incipient and front line interventions. Agrin can promote enhanced levels of oncology promoting pathways even when it is functional and typical because it senses inadequacies associated with disease and tries to resolve them.

Olive oil, likewise, promotes apoptosis, but choline supplementation alleviates the core reason why cellular entities exhibit disease. Clinicians should explore gradual change of practice to include alleviating foundational nuances of disease incipiently to avoid this paradox.

Olive oil suppresses the CELF1/AIF1 pathway, clearly suggesting that it alleviates homocysteine which is a primary effector of AIF1 signaling and Bax upregulation. Information. J Cellular Mol Med. Volume 22. Number 3. Pages 1562 to 1573. March, 2018. PMID 29105957. Information. World J Gastroenterol. Volume

15. Number 15. Pages 1809 to 1815. April, 2009. PMID 19370776.

Also, olive oil resulted in exhibition of apoptosis in oncology exhibiting cellular entities in 30 minutes instead of 16 or more hours during which typical programmed apoptosis typically occurs. This suggests that that olive oil invoked foundational mechanical membrane dynamics in order to produce apoptosis. Such apoptosis thus, can emerge in a way that is not sensitive to the roles that cellular entities perform to maintain organs, tissues and physiology. These explain why oncology occurs. Simply, physiology becomes focused on preventing massive deterioration of physiology resultant of foundational membrane mechanics that would occur because of lipid, cholesterol, phosphatidylcholine inadequacy. Information. "Can Olive Oil Cure 'Oncology'." Drweill.com Website. 6th Month, 2015.

These apoptosis prevention pathways transform immunological and xenobiotic, as well as allergy and injury response to be utilized to prevent physiology from deterioration in a rapid, uncontrolled manner. The complexity in managing oncology reflects how much complexity must

be imposed to produce a system of resiliency. One might correlate this to what it requires to maintain hundreds of houses or structures with only adequate enough material to optimally support one house. Many material, factors and contexts become applied in roles, contexts, interactions, structural nuance which are very disparate from how such factors were intended to be utilized or disparate from how these factors are otherwise optimally utilized.

Correlatively, removing a card from a house of cards slowly can have a different effect than remove such a card rapidly and haphazardly. A physiology that does not have its foundational nuance of structure assure, can likewise be susceptible to detrimental physiological and behavioral outcomes.

However, with regard to aging, the regenerative factors presented here that manage extracellular matrix, scaring, wound healing and connective tissue, as well as foundational nuances of cellular membrane physiology, may be powerfully transformation capabilities that are simple, manageable and accessible to diverse aspects of populations.

Latency linked to or causing disease. Particularly caused by microbes including viral vectors, but leading to or other disease. A strongest simplistic link in this regard is that SP1 can result in prolonged expression of AP1 because prevents AP1 enabled cellular senescence that can occur through telomere attrition. This prolonged exhibition of AP1, although SP1 can also downregulate the intensity of inflammation, results also in prolonged inhibition of PEMT along with prolonged upregulation of P53, enhancing the cause of pervasive disease but also making P53 and its pathways a target for pathogenic processes, being subverted and becoming commandeered by pathogens. Both SP1 and AP1 inhibit PEMT. SP1 transactivation of PD1 and PDL1 to obscure cellular entities from immunological function, SP1 downregulation of functionality and availability of CD4+ and CD8+ at the cellular surface for discovery by immunological synapse, SP1 diminishing of pathogenicity to obscure cellular entities from immunological monitoring, SP1 upregulation of Telomerase to counteract AP1 downregulation of telomerase that would otherwise deplete telomeres and cause afflicted cellular entities to fuse chromosomes thereby invoking senescence and prevent such cellular lineages from continuing to exhibit mitosis, as well as the exhibition of SP1 within G quadruplexes as a major actor in G quadruplex destabilization, all seem to be major activities of latency enablement. The inhibition of PEMT is also an important influence which SP1 exhibits although SP1 is participative in numerous pathways including hormone synthesis cycles, EGFR signaling and other pathways.

The central causal factors of the latency contexts analyzed in this instance, emerge from the destabilization of G4 or G Quadruplexes. These models are applicable for viral vectors including HIV, Hepatitis C, CMV, HPV, HSV and numerous others vectors. G4 quadruplexes are ringed structures, or circular folds in guanine dense regions of genome, typically at telomeres. The four ringed structures can exhibit Coding DNA typically including SP1 and TNF. Destabilization of G4 quadruplexes occur when cations such as K+ are not exhibited in the central location of the four rings, as well as by coding operations by polymerases. SP1 in particular upregulates telomerase reverse transcriptase which repairs telomeres and protects telomeres generally. Thus destabilization of G4 that includes activation of SP1 protects regions of DNA were SP1 may be activated in G4 quadruplexes. This is important because AP1 expression inhibits telomerase and can limit viral inflammatory transcription, potentiating senescence of cellular entities when cellular division depletes telomeres. SP1, however, upregulates Telomerase, inhibits viral transcriptive processes, prolongs potential for AP1 expressio which also inhibits PEMT, while SP1 also upregulates PD1/PDL1 to obscure

Activin A and Activin B are in the group of TGF beta factors. Thus, Activation of Activin A and Activin B performs activation of PI3K and Smad Pathways that compete for shared consensus sequences within the SP1 integration loci generally, but particularly occurring in the EGFR proximal promoter. Prevailing of Smad in this competition results in SP1 EGFR activity that involves crotonylation, while prevailing of PI3K results in EGFR expression. Experimental inhibition of INHIBIN A in oral oncology resulted in repression of EGFR, repression of Activin A Smad activation, downregulation of phosphorylated AKT at serine 473, and downregulation of AKT phosphorylated at Serine 473, as well as repression of SP1. This confirms that Inhibin inhibits Activin A. EGFR2, HER2 or ERGG2, in contrast to other EGFR versions that variably integrate with ligands in the extracellular exposed aspects of the transmembrane protein which can be as many as 11 extents of extracellular exposed protein, does not integrate with any ligands, having particular nuance of pathology. This explains why HER1 and HER2 are distinctively represented in the literature. The extracellular domains can be activated by ligands to produce autophosphorylation of tyrosine kinases in the intracellular environment, activating PI3K/AKT, Ras/MEK/ERK, PLCy/PKC which activates protein kinase C and catabolism of phosphatidylcholine specifically from cellular membranes requiring more activity by PEMT and CDP Choline Pathways, JAK/STAT, all of which are linked to pathways of survival, inflammation, cellular proliferation described in this document in numerous instances, including mesenchymal transitions.

Although PEMT2 ablation has about 100 percent of oncology, including oncology of the breast, EGFR upregulation is found in between 30 and 15 percent of oncology of breast, while perusing

diseased cellular entities from being encountered by T Lymphocytes, as well s diminishes function and cellular surface exhibition of CD4+ and CD8+ receptors essential for adaptive immunological synapse. Although SP1 participates EGFR receptor signaling activation, this involves feedback mechanisms that result when reproductive tissues deteriorate or are removed, disrupted feedback suppression signaling while Estradiol and inhibin B subunits both dimmish EGFR signaling, but require extended duration exhibition, a clear juxtaposition to the use of estrogen inhibitors in oncological therapy. Follistatin, Activin and non beta subunits of inhibin, expect reproductive tissues as feedback inhibitors, such that when these are not exhibited, while Estradiol and Inhibin Beta subunits repression GnRH along with deterioration or removal of gonad or reproductive tissue enhances GnRH/FSH/EGFR signaling axis. EGFR signaling is produced in pathways that include SP1, Estrogen derived from Fat is typically Estrone while Estradiol, which evenly activates Estrogen Receptor Alpha and Estrogen Receptor beta as well as diminishes AP1 to prevent it from causing hyperactivation of Estrogen Receptor Alpha along AP1 inhibition of PEMT, typically is produced

the axis to include homocysteine, hnRNP E1 inhibition, viral or other disease, GSK3B upregulation and ARIH1 upregulation, as well as SP1 upregulation, can be pervasively linked to oncology while in reproductive tissues, these factors can be linked to expansive if not all disease. This is because genetic anomaly in these pathways and invoking of other translational wellness indicators can often involve invoking of SP1. SP1 seems to be invoked as protective mechanisms, as might many of the indicators in such regard, when compared to AP1, but can be commandeered by pathology, pathogens or circumstance. Downregulation of BRCA1 and upregulation of tissue transglutaminase both can upregulate EGFR metabolically, although other analyses here, at the translational wellness site, and among particular specific no longer publicly published translational wellness information, clearly observe numerous PI3K, mitogenic, developmental and other signaling that upregulate SP1, Smad, TGFb group of factors, etc. These include environmental factors. Regardless of how naturopathic therapies make sense and might be preferred, nutraceutical and pharmacological capabilities may be essential in counteracting what may be pharmaceutical levels of influence produced by the environment. Importantly, pharmaceuticals may have to utilize pathways different from the cytochrome p450 pathways now utilize, by using cleaner, greener and more specifically and tangentially active factors. Information. "Role of HER2, EGFR, and other Receptor Tyrosine Kinases in Oncology." Oncology Migration Rev. Volume 35. Number 4. Pages 575 to 588. December 2016. PMCID PMC5215954. hnRNP E1 is relevant for HPV, HIV, and PV(myelitis). An expansive array of viral, bacterial and fungal vectors are inhibited by SP1 inhibition using Durhamycin A and the aureolic acid group of antineoplasm compounds. Durhamycin A performs as in

from reproductive tissues but is also an aspect of healthy testosterone and estrone processing in steroidogenic pathways. Information. Proc Natl Acad Sci U S A. Volume 111. Number 47. Pages 16778 to 16783. November 25, 2014. Information. J Endocrinol. Volume 210. Number 1. Page 71 to 79. July, 2011. PMID 21490134.

SP1, SP1 coactivator crotonylation writer P300 as CBP/p300, as well as Smad protein, all were recruited to the EGFR promoter after Activin A instrumentation.

Eradicating HIV and other latent viral factors. These suggest that Istodax, Ingenol Mebutate, Panobinostat and Bryostatin, together may have broad enough coverage to cause HIV latency clearance.

HDAC Inhibitors and Histone Methylation Inhibitors may be effective.

However, stabilization of stabilization of the G4 quadruplex using TMPyP4, BRACO-19 and TRIM22 along with these may be especially effective.

Protein Kinase C inhibitors generally may be effective.

Inhibitors of SP1 may be particularly useful, including TRIM22 but also including pharmacological inhibitors as well as curcumin or berberine.

The utilization of CRISPR Genome editing capabilities to eliminate viral and latent

inhibitor of HIV TAT transactivation protein which must be phosphorylated to enable replication and exhibition of pathogenic phases of HIV affliction. Durhamycin is another member of the aureolic acid antibiotics and it exhibits tetrasaccharide as well as disaccharide moieties linked to aglycone, all three being antimicrobial aureolic compounds. Information. Applied Microbiology and Biotechnology. Volume 73. Number 1. Pages 1 to 14. December, 2006. Information. J Nat Prod. Volume 65. Number 8. Pages 1091 to 1095. 8th Month, 2002. PMID 12193009.

Mithramycin can be encapsulated into polymeric micelles at the nano level and used for therapeutic intervention of sarcomas.

Metathramycin, mithramycin, each are among a expanding list of antimicrobial and therapeutic aureolic acid metabolites. cMyc and SP1 are factors that inhibited by Aureolic Acid metabolites including chromomycin inhibition of SP1 among others, as well as durhamycin abrogation of TAT transactivation and phosphorylation, along with chromomycin inhibition of SP1 to disrupt its integration into HIV long terminal repeat regions essential to provirus synthesis and likely causing destabilization of the HIV proviral G4 quadruplex.

Information. "Screening." Drug Development Research. Volume 73. Number 7. November 2012.

Information. "Aureolic Acid." Applied Microbiology and Biotechnology. Volume 73. Number 1. Pages 1 to 14. December 2006.

"Polymeric Micelles." Journal of Clinical Medicine. Volume 10. Number 7. Pages 1358. March 2021.

Durhamycin, Mithramycin and Chromomycin, add a multiple level therapeutic that can be added to more completely abrogate diverse aspects of HIV and other viral pathology.

hnRNP E1 is also downregulated by the ubiquitinase ARIH1, and experimentally inhibition of hnRNP E1 by ARIH1 results in

viral affliction, as well as repair genetic disease and alleviate accumulated genetic impairment, particularly when utilized with transduction domains that enable therapeutics to permeate all cellular types with the efficiency of a water molecule, may be indispensable in this regard and there should be galvanization of populations to have such therapies rapidly developed.

Viral Specific Factors that destabilize Replication, Transactivating Protein Phosphorylation and SP1, including three levels of HIV function. Durhamycin, Mithramycin and Chromomycin, add a multiple level therapeutic that can be added to more completely abrogate diverse aspects of HIV and other viral pathology.

Peels of citrus fruit can inhibit SP1.

mesenchymal transitions as well as enhanced pathology in breast tissues affected by oncology. Inhibition of the Ubiquitinase ARIH1 can delay oncology pathology and prevent mesenchymal transitions. ARIH1 protects cellular entities from exhibition of toxicity enabled impairment of the genome. ARIH1 signaling causes PDL1 to become deteriorated. A study observes that ARIH1 overexpression can produce therapeutic apoptosis in neoplasms, except when immunocompromised statuses are exhibited, presumably because deterioration of PDL1 prevents obscuring of cellular entities exhibiting oncology such that removing the obscuring influence of PDL1 requires immunological competent signaling to result in apoptosis. The EGFR/GSK3alpha/ARIH1 signaling pathway produces ubiquitylation of PDL1.

GSK3alpha and GSK3B are considered to be affected by the same inhibitor patterns. GSK3B can be inhibited by curcumin or turmeric.

Indirubin, Tideglusib, Indirubin 3' Oxime, Alterpaullone, Resibufogenin, 5 Bromoindole, 1 Slskrnpsullonr, BIO, Bikinin, Bio Acetoxime, BRD0705, 9 ing 41m and MAZ51 are inhibitors of GSK3, GSK3alpha and GSK3beta.

Information. GSK3. The Selleckchem.com website.

Ubiquitylation includes a proteasomal system and can function even when selective autophagy is not available.

However, ARIH1 upregulation also produces inhibition of hnRNP E1. Homocysteine can inhibit hnRNP E1 as well.

EGFR is upregulated in oncology of the cervix and oncology of the breast, including enabling escape of HIV, HPV and PV(myelitis) from protective effects of hnRNP E1 on pathogenic mRNA elongation and translation.

Upregulated EGFR, upregulated GSK3B, upregulated ARIH1 and Homocysteine all may be therapeutically inhibited to improve outcomes in oncology of breast and

oncology of cervix, including HPV enabled oncology as well as HIV, and PV(Myelitis). It is very likely that much more expansive oncology and viral conditions are therapeutically affected by managing these factors. Certainly, this explains gender differences in outcomes with viral conditions. Inhibitors of hnRNP E1 otherwise may also be inhibited as required specifically in oncology of reproductive tissues. Importantly, inhibition of SP1 disables much of this system at multiple levels for use therapeutically in oncology or reproductive issues.

Research was found which clearly observes that destabilized G4 can be used to compete with iNOS for L Arginine, diminishing :L Arginine availability for M2 macrophage polarization, Arginase activity, ornithine synthesis, and iNOS while causing L – Arginine to become directed toward M1 macrophage polarization, L – Citrulline, recycling into L – arginine and strong persistent exhibition of M1 inflammation phenotype for macrophages. L – Citrulline can promote M1 inflammation phenotype as priming or prophylaxis context in general immunology when supplemented or obtained in food such as watermelon.

iNOS expression is fundamentally beneficial and benefits immunology as well as cellular stability and cellular proliferation management, although extended duration or persistent exhibition of iNOS deteriorates its substrate, destabilizes mitochondrial associate membrane which has plumbing infrastructure that links mitochondria to the endoplasmic reticulum, depletes L – arginine and Ca2+ away from mitochondria, nNOS as well as away from eNOS, followed by impairing how P53, P21 and P27 inhibition of cellular cycle. G4 sequestration of L – Arginine may diminish the beneficial effect of iNOS and, however, diminish the detrimental

		<p>effect of iNOS that occurs with persist iNOS signaling.</p> <p>SP1 enabling prolonged expression of AP1 because of tERT replaces telomeres to counteract AP1 inhibition of tERT that would promote senescence, may a feature of parthanatos because parthanatos promotes deterioration of already differentiated cellular entities. It should be presented also that PARP1 signaling which persists because of inadequate NAD⁺ and inadequate nucleotide precursor synthesis in the hexose monophosphate shunt resultant of PEMT inhibition and P53 upregulation, as well as by competition among the fates of pyruvate because glycolysis is also inhibited by P53, can result in recruitment of substrate for DNA repair that can come to include recycled nucleotides that are exogenously originated, resulted in changes to characteristics of nucleotides in genome at quantum and biophysics level, including exhibition of quantum entanglement with exogenous artifacts and systems.</p>
<p>Double Thymidine block/pause. Strengthening and enforcing the pause exhibited by cellular entities before entry into DNA replication phases of the cellular cycle. The double thymidine block/pause involves invoking cellular cycle pause for cellular entities in G1 before DNA replication or in S Phases, followed by capture in these same phases of cellular entities that were already in mid G1 or mid S phases during the first pause/block. The literature does not mention thymidine kinase in the similar behavior of choline deficient cellular entities, although this phenomenon is likely to be are least relevant.</p>	<p>Thymidine exhibition pausing of cellular cycle at G1/S phase presents how kinase or phosphorylation cascade can regulate or dysregulate cellular cycle. Kinases, such as Casein Kinase promote phosphorylation cascades, although some phosphorylation can upregulated catalytic activity, downregulate catalytic activity as well as facilitate entry or exit of factors from metabolic pathway. A particular study observes that Nocodazole, inhibits microtubule synthesis to cause synchronization of cellular entities at G2/M, while hydroxyurea inhibits</p>	<p>Hexoses can escape P53 inhibition of Glut1, Glut3, Glut4 sugar endocytosis through import by other receptors. Information. "Mannose." Biol Chem. Volume 390. Number 1. Page 41 to 48. January, 2009.</p>

dNTP synthesis which results in synchronization of cellular entities in early S phase, while also thymidine causes cellular cycle pause at G1/S phase. These phases are transition points between phases of the cellular/mitotic/meiotic cycle. Thymidine inhibits DNA synthesis, while thymidine is exhibited in telomeric repeats, as well as is exhibited in G quadruplexes, while G4 or G Quadruplexes are exhibited in Telomeric regions.

Deoxythymidine, the contextually equivalent in deoxyribonucleic acid polymers of DNA is reduced by thymidine kinase to produce thymidine monophosphate. This may be one of many mechanisms by which PEMT inhibition, P53 upregulation, NAD⁺ depletion, resultant inhibition of glucose 6 phosphate dehydrogenase and resultant inhibition of the pentose phosphate pathway, inhibition of hexose monophosphate shunt and inhibition of glycolysis, culminate in diminishment of nucleotide synthesis, particularly by downregulation availability of hexose monophosphate shunt pathway metabolites to also downregulate thymidine kinase reducing of deoxythymidine to deoxythymidine monophosphate which, although deoxythymidine is a constitutive nucleotide,

	<p>is relevant because thymidine kinase contributes to NADPH availability.</p> <p>Accumulation of deoxythymidine, thus, results in inhibition of DNA Synthesis.</p> <p>Estradiol enhances catalytic activity of Deoxythymidine Kinase to promote enhanced DNA synthesis.</p> <p>Information. “Double Thymidine Block.” Bio Protoc. Volume 8. Number 17. Article e2994. September 5, 2018.</p>	
<p>Estrogen therapy as Estradiol to cause vascular repair and regeneration, using AF1 to promote rapidness of repair particular during emergency intervention and using AF2 produce core atheropreventative and vascular repair components, while however, modulating AF2 to prevent uterotrophic effects when necessary. The level of AF2 exhibition, thus, can be modulated to manage regenerative compared to hypertrophic effects as well as manage gender specific exhibition of ancillary changes which may sometimes be less than beneficial.</p>	<p>Estradiol evenly activates Estrogen receptors Alpha and Beta, which evenly produces also inhibition of AP1 by estrogen receptor alpha, upregulation of AP1 by estrogen receptor alpha, and resultant even effect when AP1 inhibits synthesis and activity of PEMT.</p> <p>However, Estradiol is derived from testosterone most directly and can be derived from estrone less directly, while aromatase is involved in both of these ways of producing estradiol although adipose tissue can also produce estrone.</p> <p>Estetrol, estriol and estrone each, according to the literature block estradiol’s effect at estrogen response elements in DNA as well as produce a somewhat focused effect at estrogen receptor beta catalysis by activating estrogen receptor beta. Estrogen receptor beta downregulates estrogen</p>	<p>However, AF2 is essential to exhibition of atheroprotective effects of Estradiol including C3 activation, thymidine Kinase depletion of thymidine to prevent downregulation of DNA synthesis, although AF2 also upregulates uterotrophy. The research also suggests that AF1 functions to enable rapid exhibition of the repair and reepithelization of vasculature.</p> <p>Correlatively, AF2 removal and LDL synthesis inhibition prevents atheroprotection of Estradiol. This confirms a popular assertion by a member of the medical community who suggests that cholesterol levels are typically upregulated for a reason, and that reason is that the production of salt or possibly other factors, includes exhibition of striates. Such striates cause tears, scratches and bleeding of the vasculature, resulting in upregulation of VLDL or LDL to coat the insides of vasculature. The analysis of cholesterol using assay or testing, thus, is misleading because it is the oxidation of cholesterol that that typically promotes risk. Cholesterol as class of molecules constitutes nearly 87 percent of cellular structure. These analysis found that it is inadequate replenishment of foundational aspects of cellular structure that is the incipient cause of pervasive disease and is the reason that oncology therapies are not pervasive</p>

receptor alpha and downregulates AP1, thereby counteracting estrogen receptor alpha and counteracting estrogen receptor alpha upregulation of AP1. Estrone, estetrol and estriol open the PEMT pathway by removing AP1 as an inhibitor, prevents AP1 from causing cellular senescence result of upregulation of AP1 or disease, but also enables unutilized estrogen receptor alpha to accumulate although subsequent expression of estrogen receptor alpha is diminished.

Thus, as the mysteries of hormonal pathways emerge with information useful in therapy, but also useful in understanding Humanity, it becomes reasonable to search for obvious characteristics of biological molecules and function, similar to the early nuances of this compendium research which result homologues to lipid chemistry and discoveries in lipid chemistry with biology, behavior, disease, therapy and Human events. Estetrol exhibits an Aryl Hexameter, two Hexameter, and a Pentameter, that exhibits the typical

successful, particular because the oncology phenotype pervasive emerges from impairment of the foundational nuances of biological compartmentalization and are pervasively acce control by nuances of cellular structure and physiology. Choline, chocolate, phosphatidylcholine, and even ceramides, along other phospholipids, have somehow escaped foundational nuances of health and medicine, and these factors are highly influential of oncological statuses at the cellular level. The study observed included also panama red apples with cholate and olive oil.

Information. "Polyphenols, Olive." Acta Biomed. Volume 92. Number 6. Article e2021307. 2022.
Information. "Chocolate with Olive Oil." European Society of Cardiology. 8th month, 29th Day. 2017.

A review of the estrogen response element reveals that the integration domain for Estrogen Response elements are 13 sequences in in length with a three sequence variable aspect between the 5 prime direction Arginine and the 3 prime direction Threonine. Imperfect or half sequence integration of ligands changes the level of transcription and can be assisted by flanking ligand attachments that improve transcription or enable transcription. Although these seem interesting, the AF1 domain of Estrogen receptor alpha exhibits a 6 sequence extent at locations 54 to 49 which is homologous to 10 to 5 sequence extent in estrogen receptor beta.

The literature is not widely specific regarding these sequences, and a particular study potentially offers this sequence as Glutamine before position 203 and Alanine following the 203 positions. Glycine then occurs before position 204 and Alanine occurs after position 204. The sequences conclude with Arginine before position 211 and with Glutamine occurring after position 211. The sequence gaps seem to occur because of the

Estrogenic and hormone structure that exhibits resonant interactions with distant molecules to exhibit control influence to development of surrounding physiology. However, Estetrol, the most reproductively focused and most reproductively competent of the Estrogens, according to the literature exhibits 7 hydroxyls that are able to interact with, accept integration or influence, as well as emit influence, with external environment. Early aspects of this compendium of research observed that the structure of estrogens were current competent and pattern of atoms exhibited in the structure consistent with propagation of fields of influence using the patterns of similar atoms and transfer of these to other patterns and atoms. Among the estrogens, this hydroxyl density is most dense within estetrol suggesting that physiology and developing physiology may utilize such

quaternary molecular structure which is three dimensional with different extents of amino acids or of polymers being projected to be adjacent to one another or near one another regardless of how distant they may be in the linear polymer structure. These are regarded here as shape, twist, and writhe of biologically active molecules. However, in the perfect estrogen response element literature, there does not seem to be homology to the findings in this particular study which define the six sequence homology between AF1 in estrogen receptor alpha and estrogen receptor beta. There is not, however, also exhibited an indication of if this six sequence homology is including in the 13 sequence estrogen response element in any regard. Information. "Designer Monotransregulators Provide a Basis for a Transcriptional Therapy for De Novo Endocrine Resistant 'Oncology of Breast Tissue'" Mol Med. Volume 16. Numbers 1 and 2. Pages 10 to 18. January and February 2010. Information. "Modulation of Estrogen Response element 'Enabled' 'Genetic' Expression and cellular Proliferation with Polar Directions by Designer Transcription Regulators." PLoS One. Volume 10. Number 8. Article e0136423. 2015.

It is important here to present that the products of PEMT catalysis, PMME, PDME and enriched phosphatidylcholine that includes fatty acids of extended length arachidonate, omega 3, docosahexaenoic acid, palmitate first fatty acid in fatty acid beta oxidation, oleoylate, and ether linked varieties, which perform as inorganic to organic phase separators and sequesters of biological beneficial factors from inorganic phases and transfer of these to the organic phase. Similarly, these factors

diversity to orchestrate emergence of physiology.

Thus, when the Estetrol structure is superimposed upon models and illustrations of physiology, the structure mimicked, was consistent with, and elutes the same offset of cardiac organ with nuances of entry and exit, along with offset hepatic organ in the opposite direction, suggesting the estriol may be participative in determining the characteristics of the splanchnic system of web organs that physiology otherwise seems to be centered upon. The major openings of the Cardiac organ have a similar difference in size and location to the Hexameter in the uppermost region of the estetrol structure as well as the smaller size of the Cardiac organ exhibited where the upper, right pentameter exists in the estetrol molecules. Estradiol exhibits in typical presented structure, 2 less hydroxyls than

perform as and stimulate production of serine proteases, tissue plasminogen activators, and other factors that produce clean, empirical, embryonic plasticity potentials essential for original pioneering anatomical development, optimal repair and regenerative capabilities. These factors can melt plastic and are used to clean massively toxic industrial wastes and pollutants. Thus, the homocysteine management and phosphatidylcholine synthesis pathways produce a caustic group of phospholipids and quaternary ammonium factors that sequester and produce a space in the biome for the emergence, development and persistence of biological systems. These are the nuances of cellular biology that are sustained by a prioritization of cellular structure, function, and metabolism. Diindolylmethane may be specifically assistive in depleting estrogen through methylation, although methylation of management and homocysteine, as well as much of this document involvements management of homocysteine, management of methylation, and other capabilities.

Estetrol. Estrone exhibits 3 hydroxyls, which is 2 less than Estradiol and which is four less than Estetrol. Estriol exhibits 6 hydroxyls and is linked to reproduction similarly to Estetrol.

Thus, hydroxyl structure somewhat characterizes estrogens uniquely, and the interactivity of these with the environment in ways essential to protect the emerging gestational human physiology also are obviously correlated with a reason for such control and hydroxyl exhibition in structure. Estetrol and Estriol both are presented in the literature as factors that protect the conceived of and developing Human from even the biological systems of the maternal carrier. This information was obtained by observing one source of information and its presentation of the structure of varied Estrogen factors. Some other sources present structure of each estrane variedly and

differently, although many such sources may be making specific comparative analytical elucidations for which such structure has been particularly illustrated. The clinical literature observes that endometrial hyperplasia is contrived from overproliferation or thickening of the uterus, typically uterine epithelium, but complex proliferation linked with oncology is potentiated in this context. The typical metabolic scenario presented by the literature includes unbalanced hormone exhibition, such as in upregulated estrogen along with inadequate levels of progesterone because progesterone stimulates menstruation or removal of the thickened uterine tissues or thickened uterine epithelium. Information. "What to Know About Endometrial Hyperplasia." Reference. The WebMD website. November 17, 2021.

These analyses suggest some guiding and more complicated factors in such regard, such as the requirement of AF2 for hyperplasia to occur resultant of Estradiol catalytic activity. Similarly, the anatomy level exhibition of tissue thickening when PEMT is downregulated and the CDP Choline pathway is upregulated, presents an another more intricate scenario, although Estradiol is known to upregulated Estrogen Receptor Alpha and Beta, and upregulation of estrogen receptor alpha also upregulate AP1 which inhibits PEMT, suggesting the thickening of the uterus is expected in this canonical signaling pathway. Generally, all pathology may be usefully or accurately described in a context of inhibited PEMT and upregulate choline kinase alpha, along with upregulation of the CDP choline pathway.

The biological assay databases and patent

literature present this scenario as exhibited in pervasive oncology and applicability of this therapeutic paradigm includes such substantial aspects of disease and oncology, that it is challenging to find any such disease that is not therapeutically including in this context. The interesting and obvious distinction here is that, like an allergic reaction, choline kinase alpha inhibition acts without requiring removal of epithelium, generally, suggesting that this disparity may be clinically and observationally relevant during therapy.

The literature continues to present interesting nuances that may be discussed. Some of the literature presents testosterone has having a CH₃ attached at the juncture of the uppermost Purinyl groups Hexameter and Pentameter rings, while Estrogen is presented as having a methyl group,

CH₃, at such juncture in each of its 2 Purinyl groups. Another source presents testosterone as having similar methylated structure but presents progesterone as having similar methylation structure with another methyl group attached as a hydroxylation along with a Oxygen Double Adhesion at the same Hydroxyl and Hydrogen integration into the rightmost apical carbon of the Pentameter ring of the upper most Purinyl moiety.

However, most intricately, all of the estrogens presented here, in at least some of the literature, exhibit opposite chirality hydroxyls, representing an internal molecular system that is able to produce systemic function from the opposing chirality of hydroxyls and those factors which such hydroxyls interact with physically or which the aryl Hexameter rings interact with across distance without physical interactions.

Moreover, the interesting omitting of methyl groups from the structure of estrogen is more than remarkable, but is, instead, a curiosity or even a mystery. As complex as estrogen function may be, without physical interactions and with physical interactions, these are mundane aspect of methyl group metabolism and integrate estrogen and hormone activity into typical, but complex aspect of methyl group and hydride metabolism.

These even potentiate reducing estrogen metabolism to choline, methyl group, hydride and phosphatidylcholine pathway metabolism. The changes in methyl group metabolism with enzymes, foundational aspects of cellular existential characteristics, DNA methylation, acetylation, and characteristics of homocysteine interaction with pervasive biologically active molecules, is encompassingly determinant of

cellular proliferation, cellular metabolism, apoptosis exhibition and ability to impose mitochondrial developmental and control programs within cellular lineages.

Information. The methylated structure of Estrogen and Hormones are presented at the growyourownhrt website, or Grow your own HRT website. “Grow Your Own HRT.” Growyourownhrt.com

Observationally, upregulation of hormone expression would sequester CH₃ resources in competition with PEMT and other depletory of Homocysteine, explaining what estrogen therapy in oncology regards as a mysterious upregulation of Estrogen in oncology being linked to disease. Instead of being a mystery, the upregulation of estrogen is a primary cause of upregulated Homocysteine and downregulate of PEMT. The known

causal link between homocysteine and oncology is tangentially invoked by upregulation of estrogen.

The activity of PEMT, P53, choline kinase alpha and Homocysteine are a bit complex, but these analyses have explored these intricately.

PEMT downregulation, P53 upregulation, and Homocysteine promote massive apoptosis through a variety of mechanisms, including BAX and BAK but also with mitochondrial signaling. Cellular entities respond by upregulation BCL2 and other pro survival variants of Bcl2 along with upregulation proteolysis, the unfolded protein response, upregulation of choline kinase alpha which produces feed forward energy as phosphocholine to sustain disease patterns, sustain proteolysis, upregulate S1P, upregulate G protein couple receptors used in survival signaling, involve GSK3B, and

otherwise counteract massive apoptosis that would otherwise cause physiology to disintegrate. Choline deficiency, resultant of inadequate expression of PEMT which supplies antiinflammation competent and enriched phosphatidylcholine through de novo synthesis of phosphatidylcholine, thus become diminished, impairing the existential aspects of cellular function, and structure, being replaced by upregulated CDP Choline pathway activity which produces more species of phosphatidylcholine that is not enriched, producing inflammatory changes that might characterized exactly as an allergic reaction or uterotrophic development or uterine epithelial hyperproliferation. The most important and less than intuitive factor in this context is that CDP Choline pathway upregulation uses already existing

choline, it is not a de novo synthesis pathway for choline, but uses choline, attaches atp to choline through choline kinase alpha catalysis, and then supplies this energy molecule to any relevant process, disease, pathway, or pathogen, including hyperproliferation of proteolysis. Choline is continuously obtained from phospholipases that are catabolizing choline from membrane phospholipids, resulting in an unsustainable cycle of choline distribution that it is typically inadequately obtained nutritionally, is catabolize in particular biological processes, but integrally absent in cellular membrane to challenge the very existing of the foundational aspects of the biological compartment required for biology and mammalian physiology.

Thus, with pervasive guidance therapeutically that suggest avoiding cholesterol which

comprises about 87 percent of cellular membranes, along with inadequate obtainment of choline or phosphatidylcholine at between 7 and 4 milligrams per kg of anatomical mass, as well as without about 1325 mg of choline or phosphatidylcholine each day, the foundational aspects of anatomy are increasingly challenged existentially, NOS2 is utilized to increase the turgor of cellular entities paused in hypertrophic phases in order to sustain physiology which has come to exhibit inadequate cellular entity density per micrometer of tissue, and all the nuance of oncology, disease, impairment, chronic disease, deterioration of neurological centers required for social behavior, all are not allowed to occur, but are nurtured into the diverse diminished outcomes exhibited among Human populations. NOS2 expression, uncoupled expression of NOS2, PEMT level

of expression and obliteration of PEMT2 catalysis typically occurring through dissociation of the mitochondrial associated membrane which when optimal links hundreds of intracellular mitochondria with the endoplasmic reticulum, along with Homocysteine, are pervasively correlates in level of disease, prognosis, duration of vital being, risk for adverse outcomes, detrimental behavior, behavioral health conditions, the difference in typical span of being between genders, and, importantly, have been found in particular studies when homozygously impaired as PEMT upon both strands of DNA, produce maternal carrier populations that have been obliterated with deprivation of liberty, unsustained vital being, dependency, compulsion and atypically expansive requirement for health services resources or biomedical capabilities in order

	to sustain vital being. These people survive like warriors in a complicated context which reveals the path to resolving human detrimental outcomes.	
Ret+ protein tyrosine kinase compared to Ret inversion of electron transport pathway of oxidative phosphorylation.	Another most imperative paradox to present is the integral role of methyl groups and sulphones in detoxification of estrogens and hormones as well as managing these to levels below the 50 th percentile that is sometimes used to represent indicative pathology. Sulphur or sulphones such as Methylsulfonyl methane are important because these provide sulfur to increase exhibition of thiols or sulfur in circulating hematopoietic fluid which is essentially blood, while this increase in sulfur is used to detoxify hormones, particularly estrogen, as well as while the increase in sulfur allows it Sulphur to participate in reactive oxygen species deactivation, perform as a less volatile substitute in	

some oxygenic interactions, but particularly performing interaction with the sulfides within thetin homocysteine methylperase to prevent intramolecular disulfide links within thetin homocysteine methylperase. Intramolecular links within thetin homocysteine methylperase occur when inadequate sulfur is exhibited in blood and in the microenvironment, such that this enzymes role as one of the most abundant anatomical enzymes becomes ablated and the enzyme becomes packed and stored away in tissues in a gelatinous phase that is bereft of catalytic activity. GSK3B inhibitors which are invoked in S1P receptor pathways and methylsulfonylmethane both are used in oncology therapy. Methylsulfonylmethane is used in breast oncology and prostate oncology, bridging the disparity between estrones and androgens. Androgen

upregulation is a causal factor in prostate oncology while oncology that does not exhibit upregulation of androgens are known to respond anyway to androgen inhibition therapy. GSK3B inhibition surmounts and prevents endothelial to mesenchymal transitions and as well as surmounts and prevents chemoresistance in oncology of the breast while GSK3B is known inhibitor of upregulated androgens exhibited in prostate oncology. Moreover, the patterned characteristics of oncology generally and somewhat inclusively involve increased catalytic activity in particular cellular lineages which are redirected as bona fide catalytic activity, mitosis or differentiation, although kinase activity upregulation is typically linked to each of these. Catalytic activity can be upregulated by nutritional obtainment, such as chREBP activation which can power through choline

deficiency enabled cellular cycle pause, although catabolism of cellular membranes by phospholipase can mimic choline availability in this regard, and although ankyrin repeats in molecules are also able to deactivate P53 impose cellular cycle pause, such that also in this regard postprandial activation of protein kinase C may also upregulate kinase phosphorylation cascades. Although T lymphocyte activation of cellular entities can involve such phosphorylation cascade, another Kinase, the protein tyrosine kinase ret⁺, is also known to be involved phosphorylation cascades that lead to increased catalytic activity redirected as differentiation, mitosis, or bona fide catalytic activity. There should not be a confusing of ret⁺ with RET that is the inverse of NADH release in the electron transport pathway in which Hydride is released to sue 58 percent of hydridic energy in loadbalanced energy utilization in a

democratized process during oxidative phosphorylation, while 42 percent of hydridic derived energy is integrated between the phosphate groups of the ATP which is the product of oxidative phosphorylation. RET involves reintegration of hydride into NADH at the incipient phases of oxidative phosphorylation instead of hydride being abdicated from NADH to produce free energy as florescent energy that molecules, particles and processes can absorb, interact with, entered excited status as result of, or experience spin as a result of. However, differently, ret+ is a transmembrane protein tyrosine kinase that is able to receive mitogenic signals from the extracellular environment resulting in division, mitosis, intracellular phosphorylation cascade, or other change representing redirectable catalytic potential. Ret+ upregulation has been observed

in particular oncology and its catalytic activity has been observed as a differential factor in some such oncology, including NSCLC, while ret+ genetic polymorphism has been linked with EGFR polymorphism, MET amplifications, both without requirement of combustive use of detrimental tobacco products. Importantly, in order of therapeutic efficacy, Cabozantinib, vandetanib, Lenvatinib, selpercatinib, and pralsetinib have been tested or indicated for management of ret+. “Ret Inhibitors.” ‘Oncologies’(Basel) . Volume 13. Number 17. Pages 4415. 9th Month, 2021. Information. “GSK3B.” Breast ‘Oncology’ Research. Volume 21. Number 1. Page 37. 3rd Month, 7th Day, 2019. Information. Int J Mol Med. Volume 28. Number 1. Pages 95 to 100. 7TH Month, 2011. Information. PLoS. ONE. Volume 7. Number 4. Article

e33361. 4th Month,
2nd Day, 2012.
Information.
“GSK.” ‘Oncology’
Letters. Volume
380. Number 2.
Pages 384 to 392.
Pages 384 to 392.
October 1, 2016.
The literature
presents
configuration of
propanoic acid,
hexanoic acid and
cyclohexane
carboxylic acid, as
natural compounds,
that exceeded the
integration free
energies of the
selpercatanib and
vandetanib.
Information. “Ret
Tyrosine Kinase.”
RSC Adv. Volume
12. Number 2.
Pages 1194 to 1207.
December 22, 2021.

Managing homocysteine

1. Homocysteine
 1. Bystolic or Nebivolol. Saline. NMDA Receptor inhibitors
 2. Phosphatidylcholine, Choline, Alpha-GPC, Choline Kinase alpha inhibitor Pregnenolone, DHEA, S - Methylmethionine sulfonium, Methylsulfonylmethane, A complete mineral supplements, minerals from pink Himalayan sea salt, a complete natural vitamin supplement with B12/B6/thiamine/pantothenic acid/K2/Biotin, Riboflavin, other vitamins. Glutathione. Catalase. Selenium. Sulfobetaine. Superoxide Dismutase. N Acetyl L Cysteine. Peroxiredoxin-6. Cysteine. Histidine. Cystathionine.
2. Transsulfuration Pathway Depletion of Homocysteine. zzz
 1. This suggestssuggest that sulfur should be added to B6, Methionine, NAD⁺, Serine, Danshen/Red Sage/Salvia M, Propionate, Succinate.
 2. Metabolites Cystathionine, Cysteine, Alpha-Ketobutyrate, CoA, Glutathione, and simple Sulfates such as H₂S or HS, and Cystine.
3. Managing Homocysteic Acid, Derivative of Homocysteine
 1. Saline along with Alkalinization Therapy.
 2. Vitamin K1 and Vitamin K2 as Menaquinone-4.
 3. NMDA Receptor inhibitors
4. Managing Homocysteine Thiolactone, Derivative of Homocysteine
 1. However, PON1 by a number of factors.

2. PON1 Translocation through SREBP2 and SP1 integration at the PON1 promoter occurs resultant of Statin, Quercetin and Glucose.
3. PON1 activation through the aryl hydrocarbon receptor occurs resultant of Quercetin, Resveratrol and Aspirin utilization.
4. Berberine, however, induces PON1 through the JNK-c-JUN signaling pathway. Resveratrol is a phytoalexin. trans 3,4,5,4'-tetramethoxystilbene
5. Pomegranate juice polyphenolics stimulate PON1 expression through the PPAR γ -PKA-cAMP signaling pathway.
6. Unknown mechanisms of action enable PON1 upregulation resultant of utilizing Curcumin, Betanin, Isothiocyanates, Licorice Polyphenolics, and olive oil.
5. BHMT Pathway for decreasing Homocysteine through recycling into Methionine
 1. Glutathione. Trimethylglycine. 6s 5678 Tetrahydrofolate, Zinc. N Acetyl-L Cysteine, Peroxiredoxin.
6. BHMT2 Pathway Homocysteine through recycling into Methionine
 1. Glutathione. S-Methylmethionine (S – Methylmethionine Sulfonium). 6s 5678 Tetrahydrofolate, Zinc. N Acetyl-L Cysteine, Peroxiredoxin.
7. Thetin-Homocysteine Methyltransferase Pathways decreasing Homocysteine through recycling into Methionine
 1. Dimethylthetin, Trimethylsulfonium, dimethylsulfonioacetate, ethylmethylthetin, dimethyl-alpha-propiothetin, dimethyl-beta-propiothetin, ethyl methyl-beta-propiothetin, dimethyl-gamma-butyrothetin, methionine, methylsulfonium, trimethylsulfonium, ethyldimethylsulfonium, butyldimethylsulfonium.
8. Thiopurine/Thioether S – Methyltransferase
 1. S-Adenosyl homocysteine, H⁺, and 6 methylthiopurine.
 2. 6 – methyl thioguanine, H⁺ and S -adenosyl L homocysteine.
 3. S -adenosyl L homocysteine and a thiopurine s – methylether
9. Methionine Synthase
 1. 5, Methyltetrahydrofolate, Vitamin B12 Methylcobalamin
10. Trimethylsulfonium Tetrahydrofolate N Methyltransferase
 1. Trimethylsulfonium and 6s 5678 Tetrahydrofolate bidirectionally potentiates dimethylsulfide and 5 methyltetrahydrofolate
11. S-adenosyl Methionine Synthetase
 1. Methionine, Water and ATP, potentiate phosphate, diphosphate and S-Adenosyl Methionine.
12. MARS1/MARS2 Methionyl – tRNA – Methionyl Ligase
 1. Methionine is important because it is a starting factor or primer in synthesis of more than 99.5 percent of gene transcription products. MARS1, for instance, as Methionine tRNA Ligase catalyzes synthesis of AMP, diphosphate, L-methionyl tRNA^{Met} from ATP, L – methionine and tRNA^{Met}. MARS1 occurs in the Nucleus of Homo Sapiens and MARS2 occurs in the mitochondria, performing a role in enabling incipient nuances of synthesis of RNA in Ribosomal Molecular Machines.
13. S-adenosyl Homocysteine Hydrolase
 1. NAD⁺ availability, compared to NADH, potentiates production of Homocysteine from S-Adenosyl Homocysteine.
14. INMT, Indoethylamine N – Methyltransferase, Thioether S - Methyltransferase
 1. Dimethyl Sulfide, Trimethylsulfonium, a primary methylated amine, a secondary methylated amine. 2-methylthioethanol, Dimethyl Selenide, Dimethyl Telluride, Diethylsulfide, Tryptamine, Diethylsulfide, all along with H⁺. Increased levels of S-Adenosyl Methionine can naturally potentiate this enzyme toward S-Adenosyl Methionine, but the trimethylated versions of these substrate are exclusive in catalyzing activity toward S –Adenosyl Methionine. Trimethylsulfonium, Trimethylselenonium, Trimethyltellurium , and possibly Trimethylglycine, although Trimethylglycine can be used by BHMT to produce Methionine and Dimethylglycine. Trimethylsulfonium produces linear graphs of the depletion of S-Adenosyl Homocysteine because it is used by TTMT toward 6s 5678 Tetrahydrofolate/Dimethylsulfide, used toward Thioglycolic Acid/Methionine by Thetin - Homocysteine Methyltransferase , and used toward S-Adenosyl Methionine/Dimethyl Sulfide.