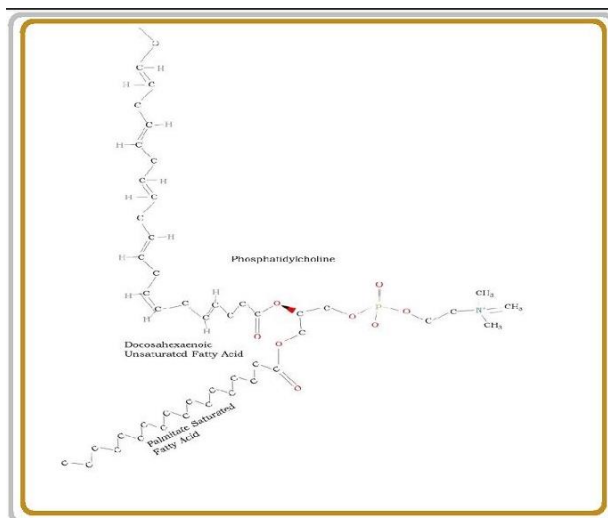


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Interestingly, it is telomere attrition, detrimentally when telomeres are depleted, beneficially when telomerase stabilizes telomeres and G Quadruplexes exhibited within telomeric regions, typically when AP1 downregulation of telomerase causes depletion of telomeres to introduce senescence fusion of chromosomes in inflamed or diseased cellular entities, detrimentally when SP1 upregulates telomerase to counteract AP1 downregulation of telomerase in a manner that results in continued mitosis of cellular lineages with upregulated inflammatory AP1 activity, as well as including methyl group attrition. Methyl group attrition is constituted of myriad detrimental factors, toxicity management factors, including therapeutic or drug toxicity management factors, that either deplete methyl groups, typically producing homocysteine is a direct catalytic byproduct, or which either inhibit PEMT or compete for methyl groups essential to the function of PEMT. The canonical example of the methyl group attritive, telomeric status commandeering, pathological cellular entity, are constitutively causal and participative in all pathology, although the extended pathogenic example includes destabilization of G quadruplexes, expression of NOS2 inducible version, G quadruplexes sequestration of L arginine away for NOS2, NOS1 and NOS3, Uncoupling of NOS2, NOS1 and NOS3 because only L arginine depletion is required for uncoupling of NOS1, NOS2 and NOS3, impressment of cellular entities to function as inflammatory M2 polarized macrophages, production of L Citrulline that is a substrate for synthesis of more L Arginine consistent with the M2 macrophage impressment cycle, release of SP1 from G quadruplexes at greater levels than typically exhibited to promote continued mitosis of AP1 upregulated cellular entities, diminishes expression and cellular surface exhibition of both CD4+ and CD8+ to impair the adaptive immunological synapse, finally including SP1 enabled upregulation of PD1 and PDL1 which obscure diseased or inflamed cellular entities from immunological monitoring and intervention.

These reprogram foundational cellular structural metabolism by inhibiting PEMT, diminished enriched anti-inflammatory phospholipid synthesis and diverse anti-inflammatory fatty acid species diversity within cellular membranes, upregulates P53 to impair Rubisco, pentose phosphate pathway, glycolysis and hexose monophosphate shunt essential for NADPH, Nucleotide synthesis, biosynthesis and supply of Ribulose to the Krebs Cycle, along with upregulation of choline kinase to supply pervasive pathogens and pathology with energy rich phosphocholine or atp choline, supplying the inflammatory/xenobiotic/allergy linked cdp choline pathway that uses choline freed by inflammatory phospholipase catabolism of cellular membranes to produce phosphocholine. This inflammatory production of choline can occur in the lumen, such as alveolar lumen, or any other most intermetal open area of microbiological environment, where, along with phospholipase D and NOS2 inducible, these can produce freakish biological monstrosities that are link to electromagnetic fields and inflammatory processes, particularly including mere upregulated catabolism of cellular membranes to challenge existential aspects of cellular structure and where phospholipase C gamma in particularly is known to catabolize the major pulmonary alveolar surfactant dipalmitoyl phosphatidylcholine. The upregulation of the CDP Choline pathway does not produced choline de novo, while such de novo synthesis of choline is a feature of PEMT that follows newly produced phosphatidylethanolamine in the CDP ethanolamine pathway and which follows decarboxylation of phosphatidylserine produced from phosphatidylcholine and such de novo synthesis of choline which is a feature of inversion of the choline oxidation pathway which instead of going in the direction of choline to betaine aldehyde to betaine, instead, correlated to characteristics of NAD⁺/NADH balance and thermodynamic characteristics, enable betaine to become betaine aldehyde and enables betaine aldehyde to become choline.

Upregulation of the CDP Choline Pathway always include direct supply of ATP as phosphocholine to upregulate proteolysis which prevents cellular entities from exhibiting apoptosis and makes cellular entities resistant to therapies as well as signaling that controls these in a coordinated manner, resulting in the ability inhibit the proteasome particles to impose massive therapeutic effect, although some cellular dynamics such as particular Bag proteins and Bcl2 upregulation can promote resilience that requires inhibition of autophagy also. CDP choline pathway upregulation also upregulates Ceramide synthesis that would promote foundational levels of cellular structure toward apoptosis, as existential mechanisms, but these can be siphoned off toward Sphingosine Kinase(version I in anatomical regions and version II in n Neurological centers) S1P synthesis, thereby activating diverse survival and inflammatory signaling pathways including G Protein Coupled Receptor Activation, S1P receptor activation widely, PDK phosphorylation, GSK3B activation, and other inflammatory survival pathway signaling, including S1P lyase which, although depleting of S1P toward Phosphoethanolamine shunt into the CDP ethanolamine pathway and toward hexadecenal which is major pheromone and precursor to docosahexaenoic acid, is a major resistance pathway for oncology variants.

Ceramide, which is produced by pathways including 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.

CDP Choline pathway upregulation of the Unfolded Protein Response, emerges at choline kinase alpha but includes other endoplasmic reticulum and Golgi apparatus components to determine cellular outcome, and is an important context for choline kinase inhibitor influencing of CHOP to determine cellular outcomes. AP1 and SP1 upregulated particular enzymes in the CDP Choline pathway, such as Citidylcholine Phosphotransferase, halfway through the CDP choline pathway, while aSMase/aSMase also upregulate phosphocholine synthesis from phosphocholine/phosphatidylserine/ceramide interaction junctures.

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 an adiposity phenotype along with the effects of PEMT inhibition which increases homocysteine in a way that increase Bax and potentiate massive apoptosis of among cellular entities. This massive potential for apoptosis results in upregulation of homocysteine and may be constituted of upregulation of homocysteine which upregulates Bax, Bak, cytochrome c release, inhibition of glucose absorption, trapping of intracellular glucose from absorption or gluconeogenesis into glycogen cycling as storage and release, DAPK, caspase activity, all which promote massive apoptosis on a lattice determined by P53, P21, PD7 and Thymidine Kinase status, although, all of which require selection for reprogramming of cellular entities toward survival signaling, Bcl2 upregulation, proteolytic prowess, and resistant phenotypes that counteract the control of mitochondrial development, proliferation and metabolic programs.

Importantly, Choline, Phosphatidylcholine, Trimethylglycine, Folate, trimethylsulfonium, B12, B6 and methyl carriers contribute to activation of Glucose 6 Phosphate dehydrogenase activity which upregulates lactone synthesis from glucose, resulting in a potential metabolic changes that spurns substantial increases in lactones, general metabolisms and possible excretion of lactones along with fatty acids produced from enhanced glycolytic, and pentose phosphate metabolism, explaining why even hypodermic instrumentation of phosphatidylcholine promotes leanness and promotes increases in cellular density per micrometer, while also enabling enhance cellular membrane density.

These phenotypes promote parthanatos in which PARP signaling occurs persistently, depletes NAD⁺, causes NADH to be metabolized NAD⁺ through lactate dehydrogenase metabolism of pyruvate into lactate anion. These metabolic factors enhance inadequacy and competition among pyruvate pathways or pyruvate metabolic fate which are already diminished by P53 inhibition of glucose 6 phosphate dehydrogenase and NAD⁺ decrease compared to NADH which causes inhibition of glucose 6 phosphate, resulting inhibition pentose phosphate pathway, inhibition of hexose monophosphate shunt pathway and inhibition of glycolysis. However, it is the inhibition of Nucleotide synthesis canonically regarded as occurring because of hexose monophosphate shunt or pentose phosphate pathway production of 5 carbon sugars, but now can be specifically characterized as occurring resultant of downregulated hexose monophosphate shunt synthesis of monophosphate used by thymidine kinase to reduce deoxythymidine to deoxythymidine monophosphate such that deoxythymidine accumulates to resulting in coordinate pause of cellular entities in phases preceding DNA replication, G1/S while Deoxythymidine performs as a deoxyribonucleotide specifically.

Thymidine availability impedes mitosis at G1 phase preventing completion of S Phase and increases nucleotide Synthesis because Deoxythymidine is a Deoxyribonucleotide or the T in the DNA structural code. 60% of NADPH, used in nucleotide synthesis among other biosynthetic pathways, is derived from the Hexose monophosphate shunt when it is unimpeded by P53, thymidine kinase reducing of Thymidine versions using hexose monophosphate results also in synthesis of NADPH. G1 and S are hypertrophic phases.

Downregulation of the hexose monophosphate shunt by P53 and diminished NAD⁺ decreases phosphorylation of glucose, while the supply of Glucose is inhibited by P53's inhibition of Glut 1, Glut3 and Glut4 endocytosis of Glucose. However, other hexose's can be imported using other receptors. Activated insulin receptor along with phosphofructokinase, as well as Hexokinase and Glucokinase can attach ATP to Hexose Sugars, these modulate the molecular translation of energy even after Glucose 6 Phosphate Hydrogenase has regulated entry of the Glucose Hexose molecule into glycolysis, pentose phosphate pathway and hexose monophosphate shunt pathways. Gluconeogenesis may also contribute glucose for regulation by glucose 6 phosphate dehydrogenase as well as contribute glucose to glycogen cycling into amyloid fibrils and to be freed as glucose from amyloid fibrils. Information. "NADPH." Themedicalbiochemistrypage.org. Information. Thymidine Kinase. EC 2.7.1.21. "Deoxyriboside Control and Synchronization of Mitosis." Nature. Volume 682 to 683. Volume 194. 1962. Information. "Immunity." Basic Biology and Clinical Assessment. Volume 2005. Pages 350 to 360. Information.

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 at least relevant.

PARP persists in signaling because it depletes NAD⁺, sequesters the ribose of NAD⁺ to distributed this in the local microenvironment to produce gradients upon which Nucleotides and substrate for repair, including more NAD⁺, may be recruiting to the locations within genome where DNA repair is occurring. This activity can occur until the requirements are quenched and also occurs in more than 1 million instances each day in every cellular entity, typically. Inhibition of the hexose monophosphate shunt and inhibition of nucleotide synthesis as result, as well as 60 percent or more decrease in NAD⁺ as a result, along with inhibition of NAD⁺ availability resultant of diminished glycolysis and as well as diminished pyruvate synthesis through glycolysis, all contributed to duration of PARP signaling being increased, exacerbating substrate availability. PARP produces nicotinamide byproducts that must be detoxified by nicotinamide methyltransferase which compete for CH₃ within s adenosyl methionine, diminished the function of PEMT, enhance methyl group attrition, as well s produce homocysteine as a byproduct. Also, inadequate NAD⁺ prevents PARP from dissociating from the locus of repair, potentiates apoptosis that is counteracted by enabling stem cellular entities to be resistant to apoptosis, causes already differentiated cellular entities to experience parthanotos version of apoptosis and impose less optimal versions of nonhomologous DNA repair, while delays in DNA repair also promote errors and inadequacies in repair.

Homocysteine emerges as the central indicator of systemic efficiency, sustainability, and homeostasis. The homocysteine performance in this regard requires that attrition of cellular membrane existential characteristics and cellular entity density per micrometer of tissue. Particularly because inhibition of PEMT, increased levels of homocysteine, and the massive apoptosis that results among cellular entities and tissues as well as among structure otherwise, result in upregulation of NOS2 inducible, which is typically beneficial when expressed ephemerally, but becomes utilized in extended duration to counteract microbial factors and to increase the turgor of cellular entities in a way that helps physiological structure from collapsing. NOS2 is expressed by Astronauts' physiology when returning from space travel, NOS2 depletes Ca²⁺, causes openings in the endoplasmic reticulum toward intracellular and extracellular interfaces, causes mitochondrial dysfunction by depleting Ca²⁺, sequesters extracellular L arginine and Ca²⁺ to promote collapses of the sarcolemma. The depletion of L arginine impairs synthesis of myelin by causing inadequacy of L arginine used in synthesis of myelin basic protein. NOS2 inducible causes cellular entities to exhibit an amoeba shape. NOS2 inducible thus causes systemic gradients in physiology to be reprogrammed to deplete Ca²⁺ from bones, exhibit increases in circulating calcium, promoting calcification of soft tissues, and requiring vitamin K2 at higher levels to manage systemic levels of Calcium. Chemokine and cytokine function, as well as migration patterns of stem cellular entities, all become changed by these strong gradients of Ca²⁺, including bone health, marrow health, and function of Agrin in determining monocyte, circulating, blood, tissue and structure stem cells which become affecting where these emerge, developed, are released, transit routes and at distal locations.

These result in changes to supply of stem cells, both systemically and at local locations.

The canonical progression of these factors includes eventual dissociation of the mitochondrial associated membrane, in which the linkages or pipping that connect 100s of mitochondria to

endoplasmic reticulum is impaired or dissociated. The disconnect of the mitochondria from the endoplasmic reticulum constitutes disease and is integral to oncology along with factors presented otherwise in this example. The mitochondria, thus, dissociated from the endoplasmic reticulum becomes unable to receive phosphatidylserine, phosphatidylethanolamine, Ca^{2+} , phosphatidylinositol, and the function of PEMT becomes increasingly abrogated. Some examples of PEMT2 function observe that PEMT2, considered to be a mitochondrial enzyme, may actually be exhibited in the mitochondrial associated membrane, such that dissociation results PEMT2 function becoming abrogated. The clinical data observes that in oncology or disease that becomes detrimental vital being, the function of PEMT2 is quite literally obliterated, typically. NOS2 inducible, prevents Mitochondria from exiting cellular entities and being shared among multiple cellular entities.

These intricate junctures of function in this platform and its interact stack of factors, are important to conclude because the function of PEMT integrates CH_3 and its hydridic contingent into phospholipids by attaching deriving CH_3 from S adenosyl methionine that has been derived from attaching s adenosyl and ATP to methionine, resulting in a carbocation as a hydridic shift or methyl group shift, in which the hydride of the s adenosyl moiety shifts into the intramolecular structure of methionine, while methionine itself also exhibits a shift to the intramolecular aspects of biologically active molecules. This carbocation results in an ionization of the Sulfur within methionine.

PEMT removes the CH_3 that has assumed hydridic character, then attaches the CH_3 to the open location of the ethanolamine in phosphatidylethanolamine using adhesion that includes the lone pair of the CH_3 becoming shared by the Nitrogen of the phosphatidylethanolamine molecule. This first attachment of PEMT produces PMME, the second attachment of CH_3 produces PDME, and the third attachment of CH_3 produces enriched versions of phosphatidylcholine, also resulting in de novo synthesis of choline as enriched phosphatidylcholine. PMME, PDME, and phosphatidylcholine are regarded as antihistamines that are strong enough to melt plastics, including plastics integrated into tissues, as well as are used to clean up toxic industrial wastes, representing a caustic agent that clears a way in the biome for the development of biology and exhibition of Life. Particularly, PMME, PDME, and Phosphatidylcholine, along with other homocysteine management pathway, comprise inorganic to organic phase separators and transfer agents, which separate biotic phases from abiotic phases, as well as sequester biologically useful factors from inorganic phases and moves these into organic phases.

Oxytocin is a neuropeptide, derived from the hypothalamus, and is an integral factor in relationships, reproduction, birthing processes as well as interactions and statuses of those involved reproduction after birth of developing Human. Its competition with synthetic peptides, social processes, and cognitive influences, may be integral to outcomes and risk involved in reproduction. An imperative observation in this context is that vast aspects of synthetic therapeutics and environmental particulate, as well as electromagnetic influence increase levels of homocysteine and result in inhibition of PEMT, thereby, also, affecting synthesis of oxytocin.

Hydride is integrated into the oxonium exhibited between the phosphate groups of ATP, such that ATP integration into the Adenosyl group which is attached to methionine by methionine

synthetase, experiences a carbocation rearrangement and that rearrangement results in a hydridic center moving to an innermost location of the S adenosyl methionine molecule, while methionine is hydrophobic and moves to the innermost aspects of macromolecule, typically. Methyl groups, as CH₃ with at least one of the Hydrogens constituting a hydridic center, also experience carbocation rearrangements, constituting methyl group shift compared to hydride which experiences hydride shift. This hydridic center is known as hydridic character. The carbocation rearrangement results in ionization of the Sulfur of methionine produces a sulfur Cation. PEMT excises, removes, and causes methionine to abdicate CH₃, this void of the structural methyl Group, structural Hydride, and structural hydric center, causes a particularly volatile methionine homologue known as homocysteine. This molecule, homocysteine, can be recycled by numerous pathways, catabolized by proteolytic processes, or eliminated through excretory pathways. However, increased levels of homocysteine occur because such a diverse array of toxicity management, xenobiotic management, and detrimental molecule management pathways use S adenosyl methionine to perform methyltransferase activity to transfer CH₃ to deactivate, signal molecules for removal. Certainly, in a comparative model of behavior and civilizations systems, homocysteine is competent inferential duality for which homologues in behavioral outcomes may be relevant.

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 Polymer Chemistry. Online ISSN 1759-9962.

The euphemism monocysteine utilized in this compendium of research to decrease the detrimental effect of merely presenting the Hcy or eHcy. eHcy, Hcy, and monocysteine should be considered as methylene bridge cysteine which is a molecule exhibiting methylene cysteine bridge.

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 the methionine methyltransferase 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 to NAD⁺/NADH balance, although TTMT production of trimethylsulfonium and 5-methyltetrahydrofolate enables both the methionine methyltransferase 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 cystathionine, cystine, glutathione, 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 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-alpha-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 sp² in trigonal planar structure. A SN₁ or reactions producing multiple separate products can typically involve carbocation and the hybridization of carbon in CH₃ in such reactions can look like carbon cations with hydrogens sharing the three sp² 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 CH₃ as the cationic, presumably hydridic influence, center. CH₃, 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.

Phosphatidylserine, phosphatidylethanolamine, methyl hydride shift carbocation of s-adenosylmethionine to ionize its sulfur to a cation, transfer of s adenosyl methionine or other thetin/thetine methyl/hydride, phosphatidylmonomethylethanolamine, phosphatidylmethylethanolamine, phosphatidylcholine enriched with docosaheptaenoic acid, palmitoylate first fatty acid in fatty acid beta oxidation, oleoylate, extended length arachidonic acid, omega 3 fatty acids, ether linked fatty acids. NAD⁺ and thermodynamic enabled inversion of choline oxidation, resulting in n,n,n glycine Betaine or trimethylglycine, betaine aldehyde, and Choline. n,n,n glycine betaine or trimethylglycine, B6 Vitamins, methionine via BHMT. S methylmethionine sulfonium exogenously originated substrate, B6 and Methionine via BHMT2. B12 methylcobalamin and Methionine Synthase MET of homo sapiens, along with folate as 5 methyltetrahydrofolate resulting in methionine and reconstituted methylcobalamin.

Methionine, possibly a unique fraction of available methionine derived from recycling of monomethylated cysteine, methionine synthetase now known s adenosyl methionine synthetase integration of ATP into Methionine to causes the canonical activation of the methylene bridge in methionine in which a strong electron withdrawing Group attaches. Integrates or interacts with a methylene bridge to cause the methylene bridge to sequester hydride to complete its trio of hydrogen that seems to include only two hydrogens in its inactive status, but is either aromatically, virtually, or otherwise stabilized by potentials such as current in the environment as 2 eV⁻, eV⁻, fluorescent energy, or hydride in adjacent structures. The attachment of atp by methionine synthetase, or s-adenosyl methionine synthase as it is now known, sequesters an electron from the sulphone hydridic character, resulting in cationic or positive polarity. The specific literature characterizes Hydrogen with 1 negatively polarized electron as e⁻ and 1 proton,

molecular hydrogen as two of these, and hydride as H^- that is constituted of 1 Proton and 2 negatively polarized electrons as $2e^-$. Hydride is, materially, electron reduced hydrogen, because education is constituted of receiving and oxidation involves abdication or release of material. Activation of the methylene bridge by strong electron withdrawers materializes the hydride from its performance as an aether or its aether characteristics that speckle or weave hydride in areas, space, atmosphere, material, tissues and physiology. The materialization of hydride as an emitted, abdicated, transferred, materialized from environment, delivered by molecular process, or otherwise exhibited electron is similar to how an electron has superposition multiplicity until a photon causes electrons as material orbiting orbitals in energy levels with only positional probability to collapse into an exhibition of its particle characteristics, although it's other multiplicities such as wave function, energy function, and others are not abdicated.

Ionization of sulfur enhances the enzyme s - adenosyl methionine function as an enzyme. Phosphatidylethanolamine methyltransferase I of endoplasmic reticula, phosphatidylethanolamine methyltransferase III, and Phosphatidylethanolamine methyltransferase II of the mitochondrial associated membrane shared between hundreds of mitochondria and the endoplasmic reticula transfer CH_3 which is a hydride primed or hydride integrated methylene bridge that pump primes or jump starts the hydridic current pipeline and trains strong electron withdrawing groups to exhibit homologous current sequestration by getting such activities started. Specifically, the lone pair electron configuration of CH_3 and CH_3 itself is removed from s -adenosyl methionine to the Nitrogen of Phosphatidylethanolamine in three successive transactions without changing the polarization or ionization of such Nitrogen and without changing the oxonium of the phosphate group, particularly because methylene divinyl patterns in fatty acids that are attached to the sn-1 location through methylene bridge and an oxygen, all result in efficient current management throughout the molecular structure. Methionine abdicated hydridic character transferred by PEMT must also be considered because methionine function enabled by methylene bridge activity is involved in tertiary and quaternary structure of more than one-third of all known proteins including tens of thousands of proteins which exhibit 10 or methionine methylene bridge intramolecular links, including many which exhibit hydridic and methylene character as much as or prevalent to exhibition hydrophobic character typical of methionine.

CH_3 and its constitutive hydride, and also phenyl moieties and tricyclopropane propane exhibit carbocation rearrangements that move hydridic centers and distribute hydridic character in ways that include resonant influence that stabilizes carbocation experiencing molecules. Electron Transport pathway of oxidative phosphorylation freeing of hydride from NADH as $2e^-$ and as fluorescent influence along with utilization of as much as about 58 percent of such energy to fund evenly distributed utilization by the different phases of the electron transport pathway, resulting in integration of about 42 percent of such energy into the oxonium exhibited between the phosphate groups of ATP or Adenosine Triphosphate. The attachment of ATP to Methionine, enhances the hydridic character of methionine which exhibits a methyl group with likely experienced carbocation rearrangements, hydride shift and methyl group shift which are integral stability enhancers for carbocation experiencing molecules.

Dimethylsulfide and 6s 5678 methyltetrahydrofolate being used by TTMT or trimethylsulfonium tetrahydrofolate n methyltransferase to produce trimethylsulfonium and 5 methylene tetrahydrofolate to provide methylene for carbocation potential, 5 methyl tetrahydrofolate for both tetrahydrobiopterin synthesis and methionine synthesis, as well as supply trimethylsulphonium substrate for thetin methyltransferase function along with sulfur to free the intermolecular deactivating disulfide linkages in thetin Methyltransferase enzymes that causes this most abundant enzyme to enter a gel phase, while trimethylsulfonium, dimethylthetin, and several other substrates causes 700 times more potent metabolic recycling methylene bridge cysteines into s-methylthioglycolic acid desquamation factor used to produce vast therapeutics through derivatization as well as producing methionine.

Amide translation into Nicotinic Acid Adenine Dinucleotide, adenylation of Nicotinic Acid Adenine Dinucleotide, followed by ATP and Mg^{2+} enablement of synthesis of NAD^+ and AMP from the adenylated Nicotinic Acid Adenine Dinucleotide, although complete B vitamins including niacin or niacinamide perform as substrate for NAD^+ synthesis, Glucose 1,6 Phosphate to Pyruvate produces NAD^+ in the glycolysis pathway although PEMT function may be required to enhance this regeneration, while nicotinamide phosphoribosyl transferase metabolism of 5 phospho alpha D ribose 1diphosphate, H^+ and nicotinamide metabolism towards beta nicotinamide D ribonucleotide and diphosphate to relieve nicotinamide methyltransferase production of cysteines with methylene bridge moieties, while melatonin assist recycling of and+ through biorhythms and NAD^+ precursors may be optimal because synthetic NAD^+ may be inadequately absorbed and NAD^+ to $NADH$ ratio can have different ranges in cytosol, mitochondria, and in duration of fluorescent moment.

Indolethylamine methyltransferase production of S – Adenosyl methionine and a tertiary amine from a methylated tertiary amine, H^+ and S adenosyl L homocysteine. Serine and B6 usage by cystathionine beta synthase to produce cystathionine, along with use of cystathionine by cystathionine gamma lyase to produce cysteine and alpha ketobutyrate, while alpha ketobutyrate is directed toward propionyl CoA using CoA SH and NAD^+ , characterizing the nearest phases of transsulfuration pathway which is activated generally when a thiol is removed from methyltransferase catalytic products and transferred to cysteine which does not have a methylene bridge because a methylene bridge enables escape of cysteines from the transsulfuration pathway into pathways which recycle cysteine exhibiting molecules otherwise into methionine.

Sulfur and Methyl Group supplementation to metabolize hormone and glucocorticoid factors, along sustainment of methyltransferases that integrate CH_3 into phospholipids instead of freeing CH_3 from management of homeostasis, resulting in integration of Hydride into cellular membranes, increase density of phospholipids in cellular membranes, increase number of cellular entities per micrometer of tissue, and enable systemic pH of near between 7.2 and 7.6 that is involved in assuring consciousness, cognitive function, and vital being. Water or H_2O is essential because it assures that intramolecular and intermolecular interactions occur with intended and optimal throughput, velocity and consistency, as well as enables particular molecular phenomenon, including hydridic, hydrogen, hydrophobic, hydrophilic, and particularly including methionine and methionine carbocation occur in physiology. Clean, filtrated and sometimes supplemented water, can substantially enable physiology while betaine

and other factors are known to stabilize the quaternary structure of biologically active molecules by performing as osmotic assurers of the shape, twist and writhe that typifies the interaction of biologically active molecules with living structures, tissues, glands, organs and anatomy.

Assuring exhibition of DHA enriched phosphatidylcholine, through synthesis within biological systems and otherwise, substantially assures these factors and pathways.

Particularly, efficient and agile management of sulfur carrying amino acids that have methylene bridges toward recycling into methylated versions with subsequent adenylation, carbocation, and Ionization of its sulfur, and alternative transsulfuration in which methylene bridges are changed towards cystathionine, alpha ketobutyrate, cysteine and glutathione, although methylene bridges in cysteines qualify cysteines for inclusion in methylene bridge sulfur carrying amino acid metabolism. Methylene bridges promote strong energy potentials used in biology such as participating in hydride, methyl, phenyl shifts, such as in S-adenosyl methionine in which, instead of freeing 2eV- and fluorescent influence when hydride is oxidized or freed from NAD+ or NADPH, shift of hydridic character occurs in carbocation rearranges in a controlled way preventing abdication of the hydride while using the 2eV-, ionizing the sulfur, and exciting the microenvironment which includes excitement of the outer incomplete energy levels and orbitals that are shared by all atoms of the universe, or metabolism, which is an antonym for nanoplasma or the empirical representation of any material or group of atoms in a defined space.

Methylene bridges are if such structural eluding if biological activity and energetic sequestration that if methylene bridge cysteines are not reduced by methyl groups which donate hydridic character to or reduce methylene bridge cysteines, then these oxidized or unmethylated cysteine bridge cysteine may attach to or sequester hydridic character in biologically active or living molecules in a manner that is integral to all diminished Human outcomes in correlation to $\mu\text{m/L}$. Asymptomatic 15 $\mu\text{m/L}$, symptomatic 10 $\mu\text{m/L}$, are admission heuristics for interventional alleviation of unmethylated or oxidized methylene bridge cysteines, while therapeutics and proactive care objectives are 7, 6, and toward 3.7 $\mu\text{m/L}$.

L-arginine is essential to alleviate diminished hexose sugar endocytosis if PEMT and Choline de novo exhibition emerges by enabling vasorelaxation vascular repair, distribution of insulin from Islets of the hepatic, renal, pancreatic axis to other areas of anatomy, while diversity of hexose sugar versions such as mannose and active hexose correlate compound as well as assured PEMT function, all current or surmount as well as assure Pentose phosphate, hexose monophosphate, glycolysis pathway mining of hydride from sugars, hexoses, and from the oxonium between the phosphate groups of ATP where hydridic character is packed when the electron transport pathway of oxidative phosphorylation frees hydride from NADH or NADPH resulting in freeing of 2 eV- of fluorescent influence of which about 58 percent is utilized about equally among the phases of the pathway, such that about 42 percent of the freed 2 eV- per unit of oxidized hydride is packed or integrated into the oxonium integrated between the phosphate groups of ATP.

Such hydridic character packed into ATP can be donated to molecule during molecular interactions, across space, and resonantly resulting in a hydride, methyl, or phenyl carbocation or shift, as with ATP integration into methionine, donating hydridic character, moving the newly donated hydridic center and possibly shifting more distantly an already existing hydridic center,

exciting or ionizing one of the molecular centers differentials such as the ionization of sulfur in methionine resultant of ATP integration into Methionine to produce the ATP adenylation methylation reduced methylene bridge cysteine known as s methyl methionine.

Methyltransferase or methylpherase freeing of CH₃ or methyl groups from s methyl methionine oxidizes the CH₃ from a carbocation strengthened or rearranged hydridic center distribution about the methylene bridge, resulting in a enhanced, freed, center of biophysics that is participative in the caustic quaternary ammonium structures that strongly sequester space in the biome while also eluting from abiotic/inorganic phases those factors useful for biology for transfer into biotic/organic phases. This sequestration potential of methylene bridge cysteines of space in the universe in which biology life and Humanity emerges, persons and advances, must be made by reducing activity, structural deteriorating, recycling or otherwise directing of these methylene bridge cysteines toward application to prevent potential massive deactivation of hydridic centers in biologically active or living molecules that is integral to all diminished outcomes.

Indefinite sustainability of physiology, thus, is able to be correlated with level of PEMT function and exclusion of increased um/L of unmethylated methylene bridge cysteines excepted rapid flux, and beneficial anabolic application. Correlatively, experimentally confirmed ability to regenerate major functional nuances of anatomy to exhibit spontaneous functional biological rhythms including regeneration of essential splanchnic system anatomical elements outside of anatomy along with exhibition of spontaneous physiological rhythms in these anatomical elements outside of the encompassing anatomical compartment without requirement of anatomical support, thus clearly presents prevention of prolonged, intensive, or chronic nonephemeral nonresolution cytokines and prevention of increased um/L of unmethylated methylene bridge cysteines, each of which are typically inversely correlated with PEMT production of enriched phosphatidylcholine, from occurring. PEMT function assures optimal cellular entity density per micrometer of tissue, adequacy of cholesterol which can comprise 85 percent or more cellular membrane structure, fundamentally changes presumptive nuances of conventional health assay and therapy.

Resolvins, neuroprostanes, freed fatty acid, docosahexaenoic acid, macrophage M2 polarization toward orbiting production by arginase, other resolution phase cytokines or factors, derivatives of these, and numerous other capabilities are concluding, stabilizing and resolution phase factors. Cysteine as well as methionine are carbonate buffering system participants, while DHA diminishes strongly exhibited methylene bridge anabolic building phase activity including diminishing of methylene bridge deactivation of trypsins that would otherwise dissolve serine intramolecular linkages in a way that promotes clean environmental plasticity compared to anabolic differentiation, although methylene bridges benefit from sequestration of magnetic metal molecules used to produce permanent magnet indefinite clean energy without fuel or byproducts. Methylene bridges participate in these microenvironment to Universes level fields by attaching to these permanent magnet competent metals, drawing current flowing through such fields or sequestering current actively from such fields.

The matrix protein agrin emerges at conception and enables exhibition of capacitance fields that that develop into consciousness, coordinates pervasive anatomical development, aggregates acetylcholine receptors to produce innervation, galvanize regenerative repair, enables stable and functional hematopoietic stem cellular and tissue stem cellular development, as well as monitors extracellular matrix plasticity to respond with mitotic signaling and secretory signaling which enables laminin, other matrix protein, and other connective tissue protein synthesis. Correlatively is coordinated the build phases of which methylene bridge proteins are integral to, including trypsin resistant, serine protease resistant methylene bridge NH₂- structures in cysteines.

The exhibition of methylene bridges in these contexts sequester capacitance or current from intramolecular or extra molecular environment, to Universes level magnetic and electromagnetic fields, and apply these toward construction from foundational physiological compartments to the anatomical compartments themselves, while capture of hydride oxidation freed 2 eV- by membranes in correlation to insulating ether linked fatty acid availability in cellular membranes, magnetic field interactions used in permanent magnet sustainable energy dynamics, and membrane phospholipids which increase superconductor temperature thresholds of efficiency toward the physiological temperature range, while also physiological pressurization and thermodynamics enable fundamental interactions, such as hydrolysis of the water molecule, nearer to physiological environmental parameters, all present methylene bridge and methylene bridge cysteines as an oscillating mechanism that informs status of indefinitely sustainable physiological energetics.

Organisms and mammalian tissue have extraordinary regenerative potential. Bereft of scarring, regenerative, repair, sustainability, resilient to diminished outcomes physiological capabilities are positively correlative with PEMT level of function, substrate access, and copy number of PEMT genomic sequences, all in a way that is correlated with management of methylene bridge cysteines toward either methylation and subsequent adenylation, or toward transsulfuration, or both although proteolysis, autophagy and ubiquitylation processes can each also diminish how unmethylated and unadenylated methylene bridge cysteines integrally and essentially participate in nonoptimal, diminished outcomes. Particular interleukins and particular metalloproteinase enzymes participate in regenerative repair, as does agrin and laminin processing that enhances the structure of connect tissue and extracellular matrix.

Correlatively, biophysics phenomena in which any defined space in biology may behave as any material essential to sustain metabolism long as that defined space has enough electrons, protons and atoms to transitively approximate the nanoplasmic empirical representation of such essential metabolic material, through space jumps in which electrons of unattached or transitively attached atoms move just in time to enable essential metabolic interactions, tunneling of electrons through impeding limitations to potentials and through the nucleus, and resonant or aromatic sharing of electrons and hydridic character without being attached, all are mechanisms of physiological resilience and stability which benefit from environmental, nutritional, hydridic, methylation, cholinergic adequacy, and phospholipid stability.

However, availability of sulfur or thiols without methylene bridges supplies sulfur to integrate with intramolecular sulfide of Thetin unmethylated bridged cysteine methylpherase, thereby

linking sulfur adequacy with preventing deactivation of this beneficial enzyme because intramolecular disulfide bridges occur in this most physiologically abundant enzyme during sulfur inadequacy. SP1 genomic sequence copy number increases in the folds of G quadruplexes and are counteracted by G quadruplex Stabilization as well as is counteracted by diminishing SP1 activity, thereby preventing SP1 increase of telomerase to diminish telomerase replacing of telomeric repeats when they are removed by DNA Replication primer activity during each cellular division.

Telomerase and Alternate Replacement of Telomerase enzymes both are beneficial in PEMT functional, unimproved cellular entities, cellular lineages, and tissues. G quadruplex stabilization and counteracting of SP1 also prevent SP1 diminishing of immunological CD4+ availability and diminishing of CD8+ availability, as well as prevents SP1 enabled increase of PD1 AND PDL1 receptors which all perform obscuring of cellular entities, impaired and unimproved, from immunological Synapse monitoring, counteraction, removal, or introduction of senescence. AP1, when increased, just as SP1 is a deactivator of PEMT when increased, is a nonresolution cytokine.

AP1, constitutively, includes telomeric attrition because it diminishes the activity of telomerase in a way that decreases the number cellular divisions that a divergent cellular lineage incurs before chromosomes fuse to disable additional proliferation. Counteracting SP1 and assuring stabilization of g quadruplexes prevents SP1 increase in telomerase and Prevents SP1 enabled obscuring of impaired cellular entities or impaired tissues from immunological control, as well as correlatively. SP1 deactivation of AP1 enabled rapid telomeric attrition toward senescent impedance to mitosis along with obscuring of cellular entities from immunological control by SP1 which allow proliferation of impaired or commandeered cellular entities, cellular lineages and tissues, are all counteracted by counteracting SP1 and assuring stabilization of G quadruplexes.

Assuring stabilization of G quadruplexes and counteracting increases in SP1, prevents prolonged mitotic lineages and proliferation of impaired cellular entities, impaired cellular lineages, and impaired tissue proliferation, all of which are integral to latent diminished outcomes or latent conditions. Counteracting PEMT and stabilizing G quadruplexes particularly allow immunological control and allow AP1 to increase rapid exhibition of senescent attrition of telomeres, preventing prolonged impaired proliferation and eventual dissociation of the hundreds of mitochondria in each cellular entity from endoplasmic reticula which disrupts the supply of phosphatidylserine, phosphatidylethanolamine, Ca²⁺, phosphatidylinositol and other factors from endoplasmic reticula to mitochondria through the mitochondrial associated membrane.

The enzyme version PEMT2 IS a transmembrane protein woven through the mitochondrial associated membrane and exhibited near conclusion of gestational development to control cellular, tissue and anatomical development. Assuring optimal function of PEMT prevents canonical and noncanonical modalities of diminished outcomes and diminished conditions by assuring mitochondrial potential, mitochondrial capacitance, and control by the mitochondria

over cellular outcomes using mitochondrial guided programs and mitochondrial involvement in signaling.

Methyl Groups are known to attach themselves to the leading edges of expanding structural lattices in biology, changing the vibrational, rotational and thermodynamic characteristics while abating expansion and anabolic aspects of structure, sometimes reaching one to one ratios with atoms at the expanding aspect of biological structural lattices.

The exhibition of Methyl Groups in membrane phospholipids including phosphatidylcholine as well as the reducing of structural potentiating methylene bridge cysteines by methyl groups strongly explain how and why physiological proliferation and deproliferation are linked to methyl group availability and methylene bridge cysteine availability, such that the watchful presence of Methyl Groups, PEMT and particularly mitochondrial PEMT2 that emerges near transition from gestation, are important control mechanisms that sustain regressive repair and regulate species specific size. Metabolism and structural characteristics.

The solvation or hydration shell constitutes a differentiated, molecule specific encapsulating H₂O sheath that is distinct in molecular, ionizing, and Michaelis as well as velocity of interactions and movement when compared bulk water beyond the 2 angstrom base shell and particularly beyond the 15 angstrom extended hydration shell. Intramolecular characteristics and catalytic activity, as well intermolecular characteristics and catalytic activity, including compound molecules and closely linked molecules with overlapping hydration shells are all shaped by the Hydration shell dynamics which can promote not only planar behavior of the solvation shell but also can cause ligand or biological molecule catalytic interfaces to more precisely mimic experimental pharmacologically derived estimates of ligand behavior, particularly when between 70 and 10 water molecules comprise the solvation sheath within a subdomain of a macromolecule or when between 10 and 70 water molecules comprise the Hydration shell of a molecule. But inclusive of folds and overlaps that can occur between subdomains of compound molecules or such overlaps that can occur between closely linked molecules.

This essential revealing perspective explains why small molecule therapeutics have become a priority in nutrition and therapeutics, although protein transduction therapy has already used purified transduction domains to insert large biologically active domains into each cellular entity in physiology with the efficiency of a water molecule. Distinct water network motion characteristics are observed up to 20 angstroms away from the molecular surface, suggesting that solvation shell chaperoning begins 20 angstroms away from the molecular surface.

Actively managing methylene bridge cysteines prevents the potential of methylene bridge cysteines to occupy fibronectin, preventing also increases in free fibrin, as well as preventing deposit of occupied fibronectin in tissue such as cardiac tissue.

The active management of methylene bridge cysteines, therefore, prevents fibronectin from increasing the occurrences of Fibronectin connection between cytoskeleton and the extracellular matrix where fibronectin has the potential to increase signaling which promotes tissue remodeling, changes to extracellular matrix, and promote fibronectin polymer assembly. This connection imposes the effect of methylene bridges, changing both the characteristics of

signaling at molecular and electromagnetic levels, but most importantly disrupting the characteristics of the foundational energies that link particles, waves, energy, superposition, angular characteristics and linkages between different instances of space and time, practically represented as changes to spatial interactions involving carbocation, hydridic characters, remote carbocation/hydridic/aromatic balancing interactions, and foundational remote programming of the environment by hormones, agrin, RNA, DNA and fields that orchestrate physiology.

Methyl bridges must be encapsulated because they are foundational biological polymerizing linkages that enable fatty acids sequences, DNA and RNA sequences, and enable alkanes that promote alkalinity necessary for mundane amino acids, such as s adenosyl methionine to become enzymes and exhibit carbocation rearrangements in which hydridic energy is free at intramolecular levels without releasing the hydride that is separated from its original incipient location, similar to a how a filament in a light bulb is excited by current to emit electromagnetic energy. Methylene bridge cysteines, when activated by biosynthetic groups that are strong electron withdrawing groups such as those with nitro, nitrile, carbonyl or others, resulting in methylene bridge sequestration of e⁻, current, or the additional e⁻ integrated into hydrogen to produce hydride H with 2e⁻ electrons and typically 1 proton, which is woven into the biome and universes somewhat pervasively and which almost every if not every atom foundationally uses as a superclass from which such nonhydrogen atoms are derived as subclasses, at least from a logical perspective. Methylene bridge activation sequesters the e⁻ that it is already attached to remotely in the aether of the Universes, galvanizing metabolism, flow of electrons, through space jumps of electrons, enabling molecules and atoms to perform as essential molecules even when the electron configuration of atom is not precisely what is require or when nanoplast is not precisely configured as required, producing tunneling of electron through impedance and limitations, as well as producing biosynthetic activity and even movement or chemotaxis that results in deliver of e⁻. Hormones and glucocorticosteroids exhibit biologically relevant symmetry and offset from symmetry while exhibiting hydridic patterns that weave hydridic fields into the aromatic hexameter that emits resonant or remote control of diverse aspects of molecular activity and metabolism, including exhibiting remote carbocation interactions and shift of hydridic character. These analyses often present that every outer incomplete energy level or every outer energy level outside of a completed energy level might be considered as a shared energy level among all atoms of the universes, practically explaining these phenomena although these also explain quantum entanglement as a result of enhanced direct particle exchange between material of the universe along with encompassing influences of the Universes.

However, methylene bridges produced by many methyltransferases result in in freed unmethylated methylene cysteine bridge factors that must be encapsulated to prevent being utilized in dysregulated polymerization. Transsulfuration translates methylene cysteine bridges into cysteine without methylene bridges. Numerous pathways recycle methylene cysteine bridge into methionine that is used in synthesis of 99.5 percent of genetic transaction products because methionine is a foundational enabler of DNA and RNA polymerase primer sequences required for attachment of polymerases to begin synthesis at least at the T-RNA level within ribosomal molecular machines. Methionine can then become adenosylated to produce s – adenosyl methionine, resulting in the hydride within the oxonium between the phosphate groups of methionine becoming rearranged or experience carbocation that results in ionization of the Sulfur

of Methionine, although it is the methylation that results in production of methionine that encapsulates or reduces the methylene bridge of methylene bridge cysteine, according to the literature. Importantly, methylene bridge is the gateway through which Hydride in the aether of the Universe becomes systematically integrated into physiology to become molecular energy, become translated into chemical energy such as pyruvate produced in glycolysis, become mined by the electron transport pathway of oxidative phosphorylation to become ATP for instance, derivation of pyruvate from Glucose, freeing of molecular and chemical energy as 2 eV-, reintegration of eV- through RET, integration of Hydride into physiology such as through PEMT packing of hydride within CH₃ into the three open location of the nitrogen within phosphatidylethanolamine to produce Choline lead group, synthesis phosphatidylcholine, packing 3 CH₃ molecules into the nitrogen which balances the methylene bridge at the SN1 fatty acid moiety, balances the fatty acid alkane in the SN-1 position, balances the oxonium in the phosphate group between the lead group and the fatty acyl integration loci. Importantly, methylene bridges are included as activated bridges in CH₃ such that the nitrile group performs as an electron withdrawing group and the 3 CH₃s seem to be constituted activated methylene bridges with hydride already sequestered and integrated since the third Hydrogen in CH₃ is considered to be hydride, according to the literature.

Relevantly, the Warburg Effect in which upregulation of Glycolysis as aerobic glycolysis to produce the between 29 and 32 molecules of energy instead the about 6 or more during anaerobic glycolysis provides useful example. Inhibition of aerobic glycolysis occurs because of P53 and decreased NAD⁺/NADH ratio, both linked to inhibition of PEMT1/PEMT2/PEMT3 but particularly decreased levels of mitochondrial PEMT2 during most diminished phases of almost every diminished health status. P53 pathways and the CDP - Choline pathway is upregulated automatically when PEMT is inhibited.

Supplementation of phosphatidylcholine, experimentally, causes a decrease or cooling of thermodynamics in the microenvironment compared to production of phosphatidylcholine in the cdp-choline pathway because the cdp-choline produces phosphocholine that is used by numerous pathways as an energy molecule including pathways used by pathogens and pathways used in pathology, particularly platelet activation and complements immune system activation which phosphocholine can perform constitutively, presenting why activity by PEMT as a producer of resolution phase fatty acids integrated in newly produce choline as phosphatidylcholine is beneficial in the about 30% fraction of phosphatidylcholine or higher which PEMT produces compared to about 70 percent or lower synthesis of phosphatidylcholine by the cdp-choline pathway using already non de novo choline typically obtained from nutrition or from phospholipase/phosphodiesterase lipase catalytic activate at cellular membranes. It is now known that riboflavin is an enabler of cholesterol and fatty acid synthesis and in many ways enable hormone pathways that produce PEMT, such that it is now essential that all B vitamins be included as a foundational of any nutritional or therapeutic regimen. Particularly, the inhibition of PEMT prevents the packing of Hydride into and locking of Hydride into the electron withdrawing nitrile group that balances one of the methylene bridges maintained from its phosphatidylethanolamine while removing the other methylene bridge. This important pathway of ethanolamine as an essential nutrition which supplies substrate to the cdp-ethanolamine pathway, resulting in production of phosphatidylethanolamine, produces an encapsulation of two

methylene bridges that are encapsulated in phospholipid as substrate for PEMT catalysis and which can be redirected toward attachment catalysts for autophagy or toward pathways in which methylene bridges can be revealed and implemented by pathology promoting conditions or activity. The redirection of recycled methylene bridges reinserted into the cdp-ethanolamine pathway such as by ceramide to S1P to S1P lyase activity, or obtainment of ethanolamine nutritionally or supplementally or otherwise, during inhibition of PEMT, promotes conditions of intracellular clearing through autophagy compared to the cdp-choline pathway which enhances proteolysis by producing phosphocholine in two loci, choline kinase and aSMase/nSMase, that supply phosphocholine for enhanced autophagy, increase influence ubiquitinase pathways which are numerous, and reprogram cellular phenotype toward dysregulate metabolisms focused on sustaining membrane resiliency during inadequate levels of choline and inadequate levels resolution phosphatidylcholine. BAG3 promotes autophagy while BAG1 promotes proteolysis, although some conditions can become resistant and persist when BAG1 or the 26S, 20S or 19S proteasomes, immunoproteasome or other proteasomes are therapeutically disruptive, often requiring therapeutic decreasing of GSK3B, GPCR receptors, other S1P receptors, BCL2, or S1P Lyase which although depletes S1P can be a resistance pathway.

Methylene bridges and methylene bridge cysteines sequester eV^- , e^- , hydride or the extra e^- in H_{2e-1p} from its pervasive integration in the biome and aether of the universes particularly when methylene bridges are activated by strong electron withdrawing groups that typically are biosynthetic but can have effect otherwise. Such sequestration of hydride results in a multiplicity in which polymerization at the foundations of biology occur, alkane polymers occur which have alkalinity that transforms amino acids into enzymes or produces higher amino acid catalytic activity, resulting in translation of hydridic character or current between ATP, Glucose, Pyruvate, 5 Carbon Pentoses, 1 Carbon metabolism toward methionine, polymerization of Flavin/Ribitol as well as structure of Riboflavin and in both FADH and FADH₂, polymerization which produces NAD (NADP⁺, NADPH, NAD⁺ and NADH), DNA and RNA polymerization observable between phosphate group and pentoses although CH₂ in DNA and RNA within phosphodiesterase complexes which have negative polarization are counteracted by histones, methylation, polyamines and Mg²⁺, although the atypical D chirality of DNA prevents a diverse array of structural interactions possible from L chiral or achiral molecules. Importantly, methylene bridges are integral, alkaline influencing polymerization enablers such that when dysregulated, methylene bridges can be sequestered by differentiation and polymerization processing, dysregulate molecular and chemical energy, integrated into physiology to disrupt molecular and electromagnetic signaling as well as change the energies utilized to stabilize particle, atom, adhesion, spatial and angular characteristics that are essential to the characteristics of interactions in tissue, between monocytes, in basement membranes of tissue, and between monocytes and structure. Methylene bridge activation sequesters metabolism, chemotaxis, metabolic pathways, molecular interactions, movement of molecules, current, electrons in through space jumps, just in time through space jumps, tunneling through potential limitation, and reorganization of atoms and particles in define space to exhibit essential molecules, all to result in delivery of electrons to strong electron withdrawing groups which have sequestered such methylene bridge.

Methylene bridge cysteine is an amino acid exhibiting sulfur which is also a methylene cysteine bridge that, when untransformed into cysteine or unrecycled into methionine through methylation as well as potentially adenosylated to become s-adenosyl methionine, can have its methylene bridges become dysregulated and commandeered into pathology promoting differentiation, energetics, and polymerization, such that when methylene bridge cysteine is below 6 or 7 micromoles per liter, a 99.95 or more percent decrease in risk of the most substantial adverse physiological and behavior outcomes over a decade of observation among a population of about 10,000 compared to control populations of about 10,000. The enzyme PEMT packs methylene bridge cysteine that has sequestered hydride as a third Hydrogen, CH₃ or methyl groups, into the three possible orbitals exhibited in the nitrogen of phosphatidylethanolamine, resulting in phosphatidylcholine, while removing one methylene bridge from phosphatidylethanolamine and producing enriched fatty acids with numerous methylene bridges including a double adhesion divinyl methylene spacer at the SN-1 location, although SN-2 and SN-1 can both exhibit enriched resolution phase fatty acids, which all, along with oxonium in the phosphate group of phosphatidylcholine, provide unique, fatty acid alkene extents, packed hydride, and hydric oxonium to balance or stabilize the nitrogen cation. Resultantly, a highly stable most abundant phospholipid that is packed with energy is produced that is a foundational factor in development, regeneration and repair. Diminished PEMT catalysis results in remarkably diminished decrease of pyruvate because diminished PEMT results in P53 imposition of diminished Glycolysis and imposition of diminished pentose phosphate or hexose monophosphate shunt activity, although upregulation of the cdp-choline pathway also occurs when PEMT is diminished in activity. The cdp-choline pathway produces phosphatidylcholine that is not particularly enriched and uses recycled choline which has methylene bridges already integrated into the nitrogen of choline. Thus, when PEMT is diminished anaerobic glycolysis or diminished throughput in energy metabolisms occurs because the protective influence of a resilient mitochondria and its programming as well as optimal resolution phase fatty acid PMME, PDME and phosphatidylcholine, sequential metabolites produced by each addition of CH₃ to the nitrogen of phosphatidylethanolamine may not be adequately available. Aerobic glycolysis, which is considered to be involved in pervasive pathology, occurs when P53 suppression pathways become surmounted or dysregulated, resulting in increase in energy pathway throughput without the protective saving of energy into hydride packed phosphatidylcholine, balancing of energy throughput with savings, redirection of current or hydride toward structural resiliency, and the without the angular, hydridic, and other 'special' effects that occur in physiology to produce the typical, or not so typical, resiliency of biological systems. Aerobic glycolysis is known as the 'warburg effect.'

Hormones exhibit Hydrogen in patterns that weave hydridic fields from the upper right offset pentameter into the midline upper hexameter and then into the lower midline hexameter, followed by delivery into the lower leftmost aromatic resonant hexameter through a methylene bridge within a a divinyl methylene bridge, resultant in hydridic character in such lowermost resonant aromatic hexameter which is utilized in aromatic, resonant, through space interactions that program spatial aspects of biology through remote, spatial, angular, hydridic and ionic influences that enable development and sustainment of biology. Hormones, at these core pentameter and hexameter elements, exhibit close or strongly similar symmetry as the internal,

wet or splanchnic organ systems. Hormones and glucocorticosteroids, thus, do more than merely incompletely or completely integrate into and transactivate the perfect 13 sequence DNA estrogen response element which includes PEMT and number of transcription sequences otherwise essential for pioneering, regenerative and repair development, such hormones and glucocorticosteroids program development through remote hydridic shift, remote hydridic character, angular influence, spatial influence, ionic influence and in other ways. Conditions involving dysregulated or impaired hormones can typically involve the methylene bridges and programming influences in such regard as well as involve upregulated exhibition of dysregulated methylene bridges, as well as dysregulation of methylene bridge cysteines. Importantly, methylation is an important primary regulatory, deactivator and encapsulated of methylene bridges and methylene bridge cysteines as well as impedes the leading edge of polymerization structures, while also is utilized to detoxify hormones and glucocorticosteroids similar to sulphones or thiol molecules also detoxify or deactivate hormones through the COMT pathway, cytochrome P450 pathway, and in other sulfur pathways inclusive of the transsulfuration that structurally deteriorates methylene bridge cysteine to cysteine. Recycling pathways for methylene bridge cysteine and s-adenosyl methylene bridge cysteine are major pathways of stabilizing. The major pathway for methylene bridge cysteine and dysregulated unencapsulated methylene bridges are in their ability to sequester current from any location in which hydride has been woven into the aether of physiology, the biome and the universes, particularly because these disrupted signaling, metabolisms, tissue structure, extracellular matrix structure and signaling, agrin structure and signaling, as well as disrupt the energies that constitute the foundational aspects of matter and material of the Universes.

PEMT production of PMME, PDME and enriched phosphatidylcholine, as well as pathways that manage methylene bridge cysteines such as thetin methylene bridge cysteine methyltransferases, along with supply of biologically essential nutrient ethanolamine through nutrition and recycling with its constitutive encapsulated methylene bridges, followed by supply of phosphatidylethanolamine at the conclusion of the cdp-ethanolamine pathway as substrate for PEMT production of enriched PMME, PDME and phosphatidylcholine, all constituted cuastic quaternary ammonium, inorganic phase to organic phase transfer agents, all of which can be utilized to structurally deteriorate pervasive carcinogens in the natural universes as well as are used to clean up toxic industrial wastes. These pathways are the foundations of physiology and are required to be impaired, deteriorated or disrupted in pervasive if not all disease. Lecithin or mixed phosphatidylcholine/Choline suspension, has been known of since the middle aspects of the 1700s. Methylene cysteine bridge has been known of and represented in the clinical literature, since 1810. Dimethylthetin has been known of since 1878 as potent depletion capability for methylene cysteine bridge, nearly 700 times more potent than pervasive pharmacological therapies or therapies otherwise. Danshen, salvia m., or red sage, which depletes homocysteine through the transsulfuration pathway is an ancient therapeutic which predates modern medicine, modern science and predates, according to some perspectives, exhibition of prevailing philosophies which prioritize human vital being and the philosophical nuances of care, by Humans, for one another in such a capacity.

Wholistically, biomedically, pharmaceutically or adjuvantly otherwise assuring that ICD-10 code E72.11, or elevated methylene bridge cysteine, is prevented or alleviated with an asymptomatic inpatient therapy at 15 $\mu\text{M/L}$, symptomatic inpatient therapy or asymptomatic outpatient therapy

at 10 $\mu\text{m/L}$, Outpatient, out of office location therapy, or office location therapy above 6 or 7 $\mu\text{m/L}$ and with an objective of between 6 or 7 to 3.7 $\mu\text{m/L}$ is essential to both Human health and Human behavior.

It should be presented here that the one instance of adverse outcome among the population of about 10,000 over a decade of observation, may have been the result statistical nuance in which a perfect 100 percent was not possible, although the study presents that only 1 instances of the most adverse of outcomes occurred among populations with methylene bridge cysteine below 6 or 7 $\mu\text{m/L}$ while in the population of about 10,000 with methylene bridge cysteine above 6 or 7 $\mu\text{m/L}$ there were 500 of such adverse outcomes in the same duration of observation.

Importantly, alkanes inherently cause amino acids to exhibit catalytic activity and methylene bridges sequester current for application in development, structural synthesis, biosynthesis and metabolism, all from the hydride, current or eV^- exhibited in the aether of the biome and the Universes.

The conclusion in this context is that when PEMT is optimal, Methylene Bridges are managed in pathways differently from when PEMT is functionally, mostly such that PEMT protects the environment for polymerization and biosynthesis to occur mostly by removing the limitations on glycolysis throughput and availability of chemical or molecular energy. Diminished PEMT function results in limitation of energy pathways to prevent excess molecular, chemical and polymerization phases of methylene bridge energy regulation, mostly through P53 pathways that diminish proliferation and energy metabolism and particularly because the storage of Hydride packed into phosphatidylcholine is also diminished. P53 limitations on metabolism, thus, when surmounted or diminished while PEMT and particularly PEMT2 is diminished in activity, s in conditions of dysregulated and upregulated molecular and chemical energy availability as well as results in increased availability of methylene bridges for redirection toward polymerization, although the most essential factor in this context is the diminished storage of Hydride and methylene bridges in the process that produces newly synthesized choline and phosphatidylcholine.

Thus, the diminished storage of methylene bridges, encapsulation of methylene bridges, and packing of hydride into cellular and physiological structure, along with dysregulated energy, is known as aerobic glycolysis which is only meaningful when compared anaerobic glycolysis because aerobic glycolysis can occur beneficially when PEMT is fully functional and fully catalytically bioavailable. PEMT removes a methylene bridge from phosphatidylethanolamine packs methylene bridges within CH_3 into the open locations within the nitrogen of phosphatidylethanolamine in a way that also packs hydride as the third hydrogen sequestered upon activation of the methylene bridge within CH_3 into the nitrogen, and balances the methylene bridge in the lead group, the oxonium in the phosphate group in the lead group connector, as well as balances the methylene bridges in the fatty acids linked by the methylene spacer in the connect to the SN-1 fatty acid location, although SN-2 location can also host fatty acids. The balancing of hydride in the positivity polarized nitrogen of phosphatidylcholine contrasts the obscured negative or alkaline polarity of the third hydrogen in each CH_3 , the oxonium in the phosphate group and the alkaline polarity of alkane linkages in fatty acids at the SN-1 positions and at possibly also at the SN-2 positions.

Experimental observation of poly ethyl acrylate has observed that it differs from poly methyl acrylate in that poly methyl acrylate has one less methylene bridge and this one less methylene bridge in poly methyl acrylate is accompanied by a methyl group which results in poly methyl acrylate being unable to promote or being enabled to actively diminish fibronectin polymer assembly.

The potential of fibronectin polymer assembly when poly ethyl acrylate and its methylene cysteine bridges are inadequately managed contrasts with the prevention of fibronectin assembly by methylation in a similar molecule poly methyl acrylate.

Functional assay of diverse fibrillation integration molecules including those involved in conditions involving fibril polymerization reveals that methylated methylene bridges are unable to promote or participate on fibronectin polymer assembly.

Precisely, the lead group of ethyl acrylate exhibits an increased number of methylene bridges and this increased number of methylene bridge moieties increases the motion of the lead group, producing a less dense and less stable hydration or solvation shell.

However, it is known that increased numbers of methylene bridges sequester more current and are more powerful invoking influences to anabolic structural processes and metabolic processes, explaining why preventing dysregulation of structural anabolic or anabolic conditions can include also prevention of the monopolization of energy by such conditions.

The contrasting role of ethyl factors in promoting polymerization compared to methyl diminishing or concluding polymerization, suggests that the enzyme PEMT, particularly mitochondrial PEMT2 which emerges near, in synchronization with, or subsequent to conclusion of gestational development, manages the reducing potential and polymerization potential of the antihistamine phosphatidylethanolamine by sequentially methylation phosphatidylethanolamine in three phases which shuttles phosphatidylethanolamine through three functional derivatives as phosphatidylmonomethylethanolamine, then phosphatidylmethylethanolamine and then enriched fractions of phosphatidylcholine. This phased promotion moves phosphatidylethanolamine into derivatives exhibiting acquired ligand and enzyme functionality that promotes embryonic plasticity, pioneering anatomical regeneration, serine protease, molecular simplification, environment cleaning, directed and explicit development programs, and stabilization of the solvation shell. Essentially, phosphatidylethanolamine provides shielded transport of methylene bridge juncture, two adjacent methylene bridge junctures, which benefit from the ethanolamine lead group mobility and diminished solvation shell stability by accessing current while the phosphatidylethanolamine structure prevents methylene bridges from performing in extensive structural polymerization but allows phosphatidylethanolamine to produce point reducing interactions constitutive of antihistamine function.

Phosphatidylethanolamine is a source of methylene bridges for glycosylphosphatidylinositol anchored proteins which invoke autophagy by performance as attachment loci for emergence of autophagosomes which essential for cellular sustainment, preventing increased comparative proteolysis, controls proliferation and controls metabolic commandeered changes linked to uncontrolled proliferation. Contextually, adjacent methylene bridges or multiple methylene

bridges 77in phosphorylated ethanolamine's explains why increase in S1P lyase, which results in the depletion of the S1P pathways typically linked diminished outcomes but also results in hexadecenal and ethanolamine phosphate, culminates of resistant conditions, particularly because methylene bridges are recycled when ethanolamine phosphate produced during S1P lyase pathway catalysis is reinserted into the cdp - ethanolamine pathway.

Thus, ethanolamine as an essential exogenously obtained nutritional, metabolic and structural factor, as de novo ethanolamine as well as recycled ethanolamine, presents its exhibition of methylene bridge moieties in multiplicity in such capacity and presents dualities potentiated in correlation to management of methylene bridge availability and methylene bridge structural access.

Glycosylation of phosphatidylethanolamine tails diminishes its selection by PEMT, particular introducing preference specificity for lightly glycosylated or unglycosylated phosphatidylmethylethanolamine by PEMT in the third methylation sequence performed by PEMT which results in synthesis of enriched phosphatidylcholine. Such third sequential methylation is delayed by what the literature presents as slower catalytic kinetics, although, presumably, the slower kinetics have reason to be increasingly selective when producing the stable phosphatidylcholine compared to production of PMME and PMME which seem to be intended as caustic, volatile advocates of biotic phase exclusivity, serine protease and tissue plasminogen activation, inorganic to organic phase transfer of biologically useful factors, and generally enhanced plasticity. PEMT selectivity at PDME before exit of ethanolamine into the choline lead group phospholipid fraction, suggests that recycling is occurring to produce phosphatidylethanolamine, phosphatidylethanolamine is being produced from phosphatidylserine, or accumulation of glycophosphatidylinositol because of impaired completion of risk averting autophagosomes and impaired risk averting autophagy, all may be potentially occurring, although inadequate obtainment of ethanolamine may be integral to such context. Methylene bridge availability and management is an integral multiplicity in assurance of optimal health status.

Ethanolamine, like phosphinic acid, CH₃, hydride, precursors to RNA, precursors to DNA, and other essential biological factors, has been incurred in interstellar space, and ethanolamine is an integral component of neurological membranes in a way that sequestration of current in fields that extend to universes level and participation in fields that are boundless temporally, each are presented as mundane nuances of physiological function.

Correlatively, methylene bridges are linked to agrin acetylcholine receptor aggregation during pioneering, reparative, and regenerative anatomical development as well as during physiological development programs through ethanolamine and in other ways, while preventing methylene bridge cysteine attachment to fibronectin and preventing depositing of the resulting complex to tissue along with preventing polymerization of the resulting complex upon extracellular matrix also prevents aberrant agrin signaling between extracellular matrix and fibroblasts, thereby alleviating potential for dysregulated mitogenic signaling, preventing confluent stability as an aspect of mitogenic signaling, as well as alleviating potential for the major pathway for organ deterioration which is granularization of extracellular matrix.

Agrin insertion is known to cause regeneration of organs and reestablishment of plasticity in extracellular matrix as well as enables regenerative reestablishment of plasticity in connective tissue.

Resolution phase phospholipases in particular, but also nonresolution phase phospholipases and phosphodiesterases free fatty acid by lysing membrane phospholipases during choline inadequacy or challenges to biological systems, such that LPCAT and MBOAT acyltransferases reintegrate free fatty acid into lysophospholipids to resynthesize phospholipids with shuffled fatty acid signatures. Phospholipid plasmalogens, such as phosphatidylmonomethylethanolamine plasmalogens are similarly freed by lipase and diesterases and reintegrated by lysoplasmalogenases, while lipase and diesterase activity also can lyse the fatty acids and lead groups of phospholipids including phosphatidylcholine and Phosphatidylethanolamine in particular as the leading phospholipids by content in cellular membranes.

Phosphatidylethanolamine is diminished in adipose dysregulation while phosphatidylcholine, presumably in unenriched cdp – choline pathway fraction, is upregulated in susceptibility to adipose dysregulation. Since glycerol and glycosyl moieties differ both only an oxygen molecule in the literature, the selective preference of PEMT for lightly glycosylated or unglycosylated participates on triage of glycerylphosphatidylethanolamine tower autophagy autophagosomes compared lightly glycosylated or unglycosylated phosphatidylethanolamine being preferred substrate in PEMT synthesis of enriched fraction phosphatidylcholine, resulting in methylation of methylene bridges molecules, resulting in removal of one of the two methylene bridges of phosphatidylethanolamine, but also allowing phosphatidylcholine to perform as a more stable membrane structure to trap eV- freed by oxidation of Hydride from NADH or other redox factors. This context explains the usefulness of phosphatidylethanolamine being typically presented on the inner leaflet of membranes where it's enhanced lead group range of motion allows current to be accessed by its methylene bridges, gathering the fluorescent 2 eV- emitted when hydride is freed during hydridic redox transactions. Including the exhibition of phosphatidylethanolamine in the inner leaflet of the inner mitochondrial membrane, revealed is the modality of capacitance, both in emitting of capacitance and gathering of capacitance, that contributes consciousness and cognitive function. Logically, the movement of hydride in metabolic processes, or current, such as in methyl group or hydride transfer, represents a structural movement current which is somewhat homologous to freeing of hydride as 2 eV- and fluorescent influence, particularly when considering the capture of current by methylene bridge complexes.

The three methylations of the nitrogen in phosphatidylethanolamine by PEMT effectively diminish lead group flexibility and transform current transfer characteristics of phosphatidylethanolamine to exhibit the hydride packed Nitrogen lead group Choline which is linked by one methylene bridge to the insulating ether linked fatty acids comprising enriched phosphatidylcholine.

The one methylene bridge of phosphatidylcholine compares to the two methylene bridges of phosphatidylethanolamine, while both of these molecules maintain the hydride packed oxonium in the unlinked oxygen of the phosphate group which links the methylene bridge to the fatty acid, glycerol or glycosyl tails. PEMT may prefer unglycosylated tails because it's processing my

require or prefer selective configuration of the fatty acids linked methylene bridge and phosphate group, particularly in the third methylation in which phosphatidylethanolamine is exited into the phosphatidylcholine fraction.

Energies are ubiquitously involved in how atoms and material are exhibited in multiplicity as structures. Correlatively, methylene bridges effect, affect, or change these energies involved in metabolism and structure.

Availability, control, management, and directing of methylene bridges, including methylene bridge cysteines, are foundational determinants of health status.

Importantly, it has been derived a most essential empirical observation, which is that eHcy may merely be a most obvious example of the multiplicity exhibited by methylene bridges which attach to and promote structural development and structural polymerization as well as which sequester current emitted from oxidation of Hydride or emitted current from carbocation rearrangements in molecules with hydridic character.

PEMT translates two methylene bridges protected within phosphatidylethanolamine between the Ethanolamine lead group and the oxonium exhibiting phosphate group, to exhibit only one methylene bridge while attaching three CH₃ molecules to the open locations upon the ethanolamine lead group to produce choline in place of ethanolamine. The potential of methylene bridges to attach to structures and polymerize is diminished as a result and the packing of hydride into ethanolamine counteracts the methylene bridges sequestration and counteracts the susceptibility of methylene bridges to being commandeered, allowing inner membrane phosphatidylethanolamine to capture current and PEMT to package ethanolamine lead groups by packing hydride into the lead group and attach ether linked fatty enriched fatty acids to the tails as insulation.

Clinical canonical methylene bridge cysteine $\mu\text{M/L}$ at 15 without or regardless of exhibition of symptoms, 10 with symptoms, otherwise above 6 or 7, but increasingly with levels further above 3.7, are thresholds for asymptomatic inpatient admittance, symptomatic inpatient admittance if not already admitted, therapeutic intervention on any setting, and focused monitoring without regard to admittance status, respectively. Correlatively, when any methylene bridge molecule is not encapsulated or education through methylation or not both methylated and adenylated/adenosylated, or when unencapsulated methylene bridges are not either stabilized, being recycled, being applied in beneficial biosynthetic virtual pipelines, or are not being deteriorated into nonmethylene bridge molecules by transsulfuration, proteolysis, autophagy, ubiquitylation, or otherwise, such methylene bridges may be commandeered by unbeneficial pathways such as fibronectin occupation of methylene cysteine bridges to increase free fibrin and deposit methylene bridge fibronectin complexes in tissue such as methylene bridge cysteine fibronectin complexes deposited into cardiac tissue to cause tissue remodeling. Including Ethyl molecules promote methylene bridge activity, particularly polymerization promotion, while methylation stabilizes methylene bridges.

Unmanaged methylene bridges may attach to structure, biologically active molecules, structure, promote polymerization, sequester current in these contexts, display signaling, disrupt hydridic

character and carbocation rearrangements to hydridic character, and since energies are involved in structural adhesions in much if not all nuances material if the universe, the essential presumptive nuances of physics, biology and biophysics may be destabilized by accumulated, unmanaged and inadequately available methylene bridges.

Ethanolamine, de novo as nutritionally obtained ethanolamine, but recycled in pathways that can result in glyceryl and glucosyl phosphatidylethanolamine excluded by PEMT on its substrate selection, thus, represents this important Duality of methylene used in defense of a space in which biology may flourish along with its ability to sequester current and useful biological factors which can be overly exhibited or commandeered by less than biologically beneficial conditions.

A review of the lengthy list of required effects of Hcy or eHcy, active in or required for most if not all manifestation of diminished health status, pervasively reveal patterns of methylene bridge dysregulation, causing methylene bridge to emerge as a new most empirical specific encompassing empirical parameter in health and behavior. Particularly because methylene bridges affect the solvation shell or hydration which guides intramolecular and intermolecular interactions, as well as determines hydrodynamic characteristics at least up to 20 angstroms from the molecular surface. Methylene bridge proactive management may replace much if interventional care in developed civilizations, allowing Care infrastructure to be sustained as is with inpatient, outpatient, Office, mobile or home nuances of care to enable vibrant industry to be sustained and grow with a correlated beneficent effect to health and behavior. This contrasts, clearly, delaying assay and care total methylene bridge mismanagement has been allowed to deteriorate physiology onto emergent or substantial pathology.

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. Ethanolamine attaches the fatty acids to cdp – ethanolamine using diacylglycerol or allocated acylglycerol as linkages while this catalytic interaction prefers sn-1,2 diradylglycerol as substrate, result in in major output as phosphatidylethanolamine and some fraction glycerophosphatidylchol7ine. Oxidative phosphorylation, cellular respiration Complex III enabling, essential phosphatidylserine decarboxylase translation of phosphatidylserine to phosphatidylethanolamine occurs at the inner mitochondrial membrane such that sn-1,2 diacylglycerol phosphatidylserine and sn-1,2 diacylglycerol are Selectively preferred as substrate by both phosphatidylserine decarboxylase 1 and phosphatidylserine decarboxylase 2.

These conclusions present how interconnected systems and incentives that promote information sharing and divulgence of information, may be among the most powerful developments among the Universes, particularly if excluding the first instance in which organisms exhibited the inclination to beneficently care for one another. Such inclination, in objective opinion, changed everything that has since emerged and has changed everything that may ever be.

Methylene bridges are practically CH3 methyl groups without the third Hydrogen which is considered to be hydride. Methylene bridges are susceptible to strong withdrawers of electrons

and are affected in such regard strongly enough to cause deprotonation, such that assimilation of electrons from flowing 7.2 current, intramolecular current, hydride in intramolecular locations or hydride in unattached structures can be withdrawn to become localized to the electron withdrawing complexes, even when this results in deprotonation, even when this results in a carbocation arrangement or shift in hydridic character, as well as when this results in a withdrawing of an electron across space between unattached carbocation participants.

5,10 methylene tetrahydrofolate is processed by the enzyme MTHFR to produce 5 methylene tetrahydrofolate which methionine synthase uses to produce methionine. which contributes Methylene bridges, Methylene Spacers, Methanediyl group, or Methano factors, all used to indicate methylene bridges, exhibit CH₂ with individual linkages of the carbon to other factors, such that when located between strong electron withdrawing groups such as Nitro NO₂ linked to nitric oxide biological benefit, Carbon double linked to Oxygen as Carbonyl, and nitril composed of an axial group linked to a Carbon that is triple linked to a nitrogen, exposure to strong bases can result in highly biosynthetic products such as enclaves and carnations, explaining why 7.2 to 7.6 alkaline environmental pH is linked to homeostasis as well as explaining how methylene bridges must be encapsulated or counteracted in the homeostasis alkaline environment.

Because methylene bridges are homologous to CH₃ without the third Hydrogen which completes the electron configuration for packed Hydride in which triplets of Hydrogen are known to attach to structures in triplets at one dimensional valley structures in which one of the hydrides is structurally dissociated. This dissociation across spanning across obscuring structures and unidimensional structural valleys are obvious similarities to both carbocation or hydride shift including methyl shift and other carbocation as well as methane bridges or methylene spacers which participate in carbocation.

Conditions of structure, energetics or metabolism, including those otherwise involving migration of. circulation of, or circulating monocytes, from a review of the literature, clearly seem to pervasively involve or are empirically differentially characterized by attrition, upregulation, diversion, inadequacy, impedance or increased volume of substrate flow through the CDP - ethanolamine pathway to PEMT and the Lands cycle.

Ethanolamine exhibits two methylene bridges, to which ethanolamine kinase activity contributes a phosphate group to produce phosphoethanolamine, followed by ethanolamine phosphate citidylyltransferase attachment or polymerization of the existing phosphate group in phosphoethanolamine using another phosphate group, a hydroxyl attached pentameter and a hydroxyl linked hexameter. Ethanolamine phosphotransferase then attaches glycerol molecules to the methylene bridges through an oxygen intermediary. A review of lipid chemistry structural phase progression literature, research and application, clearly links the characteristics of these processes not only with phosphatidylethanolamine movement of current or energy from the inner leaflet of membranes to outer leaflet of membranes such as when PEMT packs Hydride as CH₃ around or into a strong electron withdrawing biosynthetic nitril adjacent to a methylene bridge such as enriched phosphatidylcholine, but also presented are the microstructural, superstructure, phase development progression that differentiates organism structure, function and energetics.

The one hydrogen difference between glycerol and glycol factors suggest that PEMT prefers the extra hydrogen of glycerol to spread the energetics of hydride across the molecule including the fatty acid that is attached to the one methylene bridge, presumptively explaining why phospholipids are characterized by sn-1 fatty acid species and sn-2 fatty acid species because these determine nonresolution/resolution phase interactivity, bending, folding, insulation, shape, twist and writhe of areas between phospholipids, of membranes and plasticity characteristics in general.

Glycosylated tails are less preferred than glycerol tails in PEMT selection of its substrate fraction of available phosphatidylethanolamine while supply of phosphatidylethanolamine by serine decarboxylase and ethanolamine phosphotransferase are presented by the literature as scrutinizing only the sn-1 loci for diradyl glycerol during production of phosphatidylethanolamine as substrate for PEMT. The cdp – choline pathway uses already existing choline processed in the same pathway transactions as ethanolamine, to result in phosphatidylcholine. with diminished focus on enriched diversity of fatty acids. Phosphatidylcholine can be recycled generally through phosphatidylserine decarboxylase, including direction toward trypsin synthesis by MDR2, lipase activity, diesterase activity or direction toward ceramide and toward the sphingolipid signaling pathways which should be managed because are cellular existential challenge response pathways linked to diminished health status response. Sphingolipid signaling pathways should be managed because these can emerge as causal factors of diminished health status if exhibited for extended duration.

Methylene bridge participation has probably eluded requisite centrality because of the Duality of methylene bridges as active structural polymerization promoters and passive susceptibility to strong electron drawing factors, clearly leading analysts toward the electron drawing groups in analytic research to obscure methylene bridges while also being obscured in the effect of methylene bridges to spatial energetics as well obscuring effect to structural energetics or metabolism. The affection of structure to space, factors and monocytes circulating in physiology, as well as the affection emitted by circulating monocytes, enzymes such as carbocated methionine of structure and other circulating material, does not seem to be intuitively represented in experimentation, research, Health, Nutrition, diagnostics, development, therapeutics development, although at interactivity levels, S.O.A.P. objective assessment processes seem to represent homologues hydridic interactivity.

Ethanolamine, phosphoethanolamine, Citidylethanolamine, diradyl or glycerol phosphatidylethanolamine, diradyl or glycerol phosphatidylmonomethylethanolamine, diradyl or glycerol phosphatidylmethylethanolamine, (palmitate first fatty acid in fatty acid beta oxidation, oleoylate, extended length omega-6 arachidonic acid, Docosaheptaenoic acid, omega-3, ether linked, diverse fatty acid) enriched glycerol phosphatidylcholine followed nonresolution/resolution phase lipase/diesterase freeing of fatty acids which are applied in immunology or shuffled while being reintegrated into phospholipids such as enhanced diversity fatty acid phospholipids and enhanced diversity phosphatidylcholine by

LPCAT/MBOAT/Lysoplasmalogenase catalysis, provides a central perspective of ethanolamine shuttling of methylene bridges.

Although the genetic conditions can have enhanced sequelae, these and other extended differential characteristics pervasively involved methylene bridge and methylene bridge cysteine escape from transsulfuration, recycling, proteolysis, serine proteolysis, tissue plasminogen activator activity, autophagy, ubiquitylation or other excretion and recycling pathways. AP1 which diminishes the cdp-choline pathway at CTP--choline citidylytransferase while both diminishing PEMT and diminishing telomerase replacement of telomeres during each mitotic cellular cycle, compared to SP1 performance of these same changes although SP1 upregulates telomerase instead of diminishing telomerase, provides of differentiating influence in the developmental sequelae following pipelining of ethanolamine to diverse shuffled phosphatidylcholine. The small amount of cdp-choline pathway substrates produced by cdp-ethanolamine pathway enzymes and the small amount of cdp-ethanolamine pathway substrates produced by the cdp-choline pathway enzymes are more than interesting, particularly because through phosphatidylcholine conversion to phosphatidylserine, phosphatidylserine conversion to phosphatidylethanolamine and phosphatidylcholine direction through ceramide, sphingolipid synthesis, and then to hexadecenal and ethanolamine phosphate, as well as PEMT de novo synthesis of choline as enriched phosphatidylcholine, the CDP-Ethanolamine pathway and the CDP-Choline pathway pipeline substrate to one another.

These revealing observations directed by perspectives of methylene physiological effects open the field of convergent contexts for nutrition medicine, research, diagnostics, and proactive health assurance at the foundational aspects of material of the universe, surmounting the divide between physics and biophysics in biomedical discovery.

Intriguing is the way in which methylene bridge multiplicity enables or participates in which any defined space can have its constituent material behave as other material such as atoms of one nature perform as other atom level configurations. Methylene bridges have an omitted third Hydrogen, relatively, hydride, such that electron withdrawing groups attaching to methylene bridges cause a sequestration of 2 eV- or cause sequestration of current, either in through space jumps, electron tunneling across atom or biological structure, constitutively from freed fluorescent hydridic energy, or by causing a physiological pathway or pipeline to be invoked that culminates in delivery of current, electrons as 2 eV-, four essential energy immersion such as nitril groups packed with methyl groups which have hydride. Correlatively, methylene bridge as order takers and delivery invokes of hydride may result in synthesis or delivery of ATP or Pyruvate. Inherently, hydride negative polarity or alkalinity promotes natural gradient in the H⁺ prevalent solution, + environment or unpolarized environment, while its order taking and delivery of current or energy molecules as well as fluorescent enables physiological activity to occur against natural gradients, allowing organism to increasingly use conscious priorities on shaping of physiological outcomes and behavior. Methylene bridges exist in space and although essential biological factors are also found in space suggesting that there integral processing requires a spontaneous event. Order taking by the methylene bridge and its sequestration of current and interaction with the solvation or hydride shell is more than adequate cause spontaneous or more accurately, designed programmatic integration of these foundational

components of biology. Thus, in any defined space the components can be galvanized to perform actions enabling or sustaining biology, at least one modality in such regard is the sequestration hydridic character or current by methylene enabled changes.

Polyunsaturated fatty acids Docosahexaenoic acid and Eicosapentaenoic acid exhibit methylene bridges and phosphorylate delta carbons of tryptophan 448 and 553 of PDK1 which along with AKT phosphorylation at tryptophan 424 by these PUFA fatty acids, results in PDK1 translocation to the cytoplasmic membrane, depletion of the Pyruvate Dehydrogenase downregulator known as PDK1, enhances glucose depletion correlative to acylation of AKT and acylation of PDK1, while also enhancing insulin resistance because the physiological effect of insulin receptor downregulation by P53 is circumvented by these conditions to enhance glucose removal and processing. A study observes that polyunsaturated fatty acid, PUFAs, counteract the potential for upregulation of glycolysis when glycolysis should typically be downregulated responsively to diminished PEMT activity and responsively to upregulated P53. This potential increase in glycolysis amid PEMT downregulation and amid P53 upregulation is a canonical integral factor in diminished health status, and is counteracted by Docosahexaenoic acid and Eicosapentaenoic acid, in a way that results in beneficial tissue remodeling which. DHA and EPA enabled tissue remodeling, in this regard, follows reintroduction of diminished glycolysis that is coupled with reintroduction of Krebs Cycle upregulation compared to glycolysis.

DHA and EPA PUFA methylene bridge associated reprogramming of energy metabolism destabilizes the “warburg effect” in which upregulation of glycolysis occurs in detrimental contexts, with particular including of glycolysis upregulation occurring amid PEMT downregulation or P53 upregulation, and presents how methylene bridges perform or enable enzyme activity that can be empirically described as current flow, flow of ambient current as well as molecules, metabolites and structural activity that constitutes migration of electrons or flow current. Any way that any organism, function, device, machine, building, activity, function, Pipeline, or wire in nature or civilization uses to obtain or sequester, logistically supply, deliver, elute, or transmit energy, power or current can reasonably be represented by methylene bridge enablement in physiology.

The “warburg effect” can be simply reprinted as an increase in glycolysis without the assistance of PEMT packing of hydride into biosynthetic electron withdrawing nitril lead groups of newly produced, unglycosylated or lightly glycosylated, glycerol, ether linked, Omega-3, DHA, EPA or otherwise enriched phosphatidylethanolamine metabolites including likewise enriched PMME, PDME and phosphatidylcholine. Phospholipase and diesterase are increased in detrimental conditions including when PEMT is downregulated, freeing choline, other lead groups such as ethanolamine, phosphatides, and fatty acid from cellular membranes along freeing Ca^{2+} encircling lead groups of phospholipids to sustain Ca^{2+} reliant versions of lipases and diesterases, although versions of these include those which function independently of Ca^{2+} . These can result in release of unencapsulated methylene bridges, although phospholipase or phosphodiesterase activity can generally be correlated with increase in methylene bridge cysteines. Diminished PEMT results in diminished migration of phosphatidylethanolamine from the inner leaflet of membranes to the outer leaflet or outer membrane as phosphatidylmonomethylethanolamine then phosphatidyl dimethylethanolamine, then

phosphatidylcholine, resulting also in diminished hydride packing at the Plasma membrane, diminished support of hydridic field attenuation into the extracellular space which decreases support of the near 7.2 to 7.6 background pH and diminishing inherent enablement of polarity gradients, solvation, solvation shell, intramolecular dynamics. intermolecular dynamics, and water dynamics up to 20 angstrom or more from molecular surfaces. The result of impaired PEMT packing of hydride, in this regard, may be a foundational destabilization of existential nuances of the foundation biological compartment, including upregulation of the cdp- choline pathway to counteract massive programmed deterioration of cellular compartments, increase in proteolysis over autophagy, increase in ceramide from diminished directing of phosphatidylcholine toward phosphatidylethanolamine resultant of PEMT inhibition that produces accumulation of phosphatidylethanolamine, increase in sphingosine 1 phosphate from ceramide because tissue stability requires that the cellular deterioration signal ceramide be redirected toward S1P massive pathways of survival signaling, all of which are lessened in availability by PEMT function or PEMT metabolites.

G protein coupled receptors, S1P receptors, including GSK3B, PDK, cellular survival Bcl2, and proteolytic enhancing BAG1 which links chaperone complexes to the 26s proteasome using ubiquitylation pathway signal attachments that are diverse and can be commandeered by detrimental conditions or axial pathways, all are S1P effected. BAG1 compares to BAG3 in that BAG3 invokes, preferentially, autophagy, which although endosomes can be used by microbes to escape the toxic plasma membrane interstitial space, is nonetheless vacuous to intracellular substrates moved into autophagosomes resulting in clearing of diverse material from the intracellular environment.

Excess unencapsulated methylene bridges, impaired PEMT encapsulation of methylation bridges, and commandeered these by detrimental conditions or factors are integral to spatial and interactive nuances of dismissed health statuses which become obscured by unintuitive characteristics of quantum, physics, biophysics which can escape ascertainment because methylene bridges can be passively or actively applied in the physiological context. The click information suggest that since diversity in hexose sugars circumvented GLUT 1, GLUT 3, GLUT 4 and glucose- 6 -phosphate dehydrogenase downregulation by P53 by circumventing impedance to the hexose glucose being endocytosed and shuttled into glycolysis. Particular using other hexoses and other hexose transporters to supply the pentose phosphate pathway with substrate and potentiate controversial supply of the latter aspects of glycolysis near the Krebs Cycle with Ribulose through Rubisco glycerol carboxylation and supply of the Krebs Cycle with glycolate produced through Rubisco glycosyl oxygenation.

The hexose monophosphate or pentose phosphate supplies pentose sugars uses in structure and polymerization of DNA and RNA, while monophosphate integration can divert pentoses away from Nucleotide synthesis, some reduction of pentoses result in a monophosphate reduction that essentially results in a Nucleotide or nucleotide precursor. Thymidine kinase produces thymidine monophosphate from atp and deoxythymidine and it polymerizes thymidine into nucleotide sequencing in a way that results in integration of thymidine monophosphate into genomic polymers, constituting an essential Nucleotide synthesis mechanism use in Pharmacology and able to be modulated to affect cellular cycle by causing Nucleotide imbalance and inadequacy.

The junctures at which PEMT inhibition affects glycolysis such as at GLUT endocytosis of the hexose glucose, glucose - 6 - phosphate dehydrogenase processing of glucose – 6 – phosphate, insulin receptor inhibition, or other, affect availability of glucose into the hexose monophosphate shunt also while diverse other hexoses may be able to be circumvent these to assist in enabling continued supply of glycerol factors, glycolate, and pentose sugars, as well as nadph. The literature does not delineate if the utility of P53 in imposing these regulatory influences when PEMT is diminished includes pentose phosphate pathway as happenstance, if the such inclusion of the pentose phosphate pathway is intended to exclude the specific hexose glucose from both glycolysis and hexose monophosphate pathway processing, although the analysis here clearly produces a Referential context in which glycolytic activity uninhibited by P53 causes deterioration of cellular structural and deteriorates esoteric and unintuitive aspects of biological systems.

A review of the structure of trimethylamine reveals that it accumulates in less than beneficial digestive pathway microflora proliferation, transiting the leaky gut typically resultant of the alpha relaxation of tight junction proteins of digestive enzyme, such that in hepatic tissue its one oxygen among the three methyl groups attached to its cationic nitrogen becomes reduced by flavin monooxygenase to produce a negatively polarized exposed oxonium, resulting in an unusually accessible juncture that fills the canonical methylene bridge omitted hydride. The commandeered methylene bridge can rapidly include tmao, explaining why tmao has a priority in being prevented in proactive and interventional care, and explaining why tmao is the among the most indicative biomarkers of susceptibility to sudden adverse health events, sudden adverse behavior, perioperative complications, diminished outcomes linked to diminished carotid plasticity, and other diminished outcomes. Interestingly, Areas of physiology near hepatic processing of tma into tmao, are the only areas, wet or splanchnic system of organs, in which proliferation conditions occur without irrefutable dismissed levels of PEMT activity. The effect of tmao, thus, may include the increased priority ascertaining PEMT2 impairment instead of aggregate PEMT2 impairment, tma, tmao, and the different metabolic methylene bridge cysteine compartments as s adenosyl, thiolactone, 'eic' acid version, and constitutive methylene bridge cysteine.

Active hexose correlated compound's name suggests that it activates the hexose monophosphate shunt, although the data suggests AHCC may also be an inhibitor of choline kinase alpha and may affect other metabolic enzymes. These suggest chemical energy may be dispensable in the 29 to 32 molecules of ATP exhibited when oxidative phosphorylation electron transport, glycolysis and Krebs cycle are all fully supplying products and substrate to one another except when PEMT is fully functional to move this nearer to 29 and 32, while the 6 molecules of ATP generated during P53 downregulation of glycolytic pathways resultant of diminished PEMT function prevents energy metabolism from excessively fracking or mining hydride packed into phosphatidylcholine without replenishment of enriched phosphatidylcholine on particular.

The linkage of nitril packed Hydride in membrane phosphatidylcholine, being fired lipase and diesterase for access in nad⁺/nadh and nadp⁺/nadph redox transactions such as the electron transport pathway freeing of hydride from nadh to emit 2 eV⁻ with about 58 percent as fluorescent energy loadbalanced across each phase of oxidative phosphorylation electron

transport and with about 42 percent integrated into the oxonium exhibited between the phosphate groups of ATP, Product of oxidative phosphorylation also known as cellular respiration, as well as entry of ATP into metabolism in Diverse contexts, including its integration into the glucose – 6 – phosphate dehydrogenase processing of glucose – 6 – phosphate to support translation of the hexose glucose into the hexose monophosphate shunt or into glycolytic synthesis of Pyruvate followed by either NADH enabled translation of pyruvate into lactate, the NAD⁺ and CoA enablement of Pyruvate translation into Acetyl – COA which can be shuttled by oxaloacetate into enabled shuttling of Pyruvate into the Krebs Cycle as Citrate and CoA. CO₂ is supplied toward fatty acid synthesis during Acetyl-CoA production, Acetyl - CoA can be directed toward Acetyl - choline storage if excess choline. Alanine, phosphoenolpyruvate, oxaloacetate, and acetaldehyde each are major pathways of pyruvate processing.

These nonintuitive nuances of hydridic migration are simpler to understand if free current, flow of current throw structure, movement of molecules, and changes biological structure are considered as aspects of current. Thus, glycolytic upregulation without replenishment causes a shift in the preemptive spatial, fluidic, structural, and current aspects of physiology that capture and apply the hydridic field in concerted way known as cellular physiology.

Prolonged dysregulation toward assured management of methylene bridge dynamics can result in impairment of PEMT1, PEMT2, or PEMT3 function, particularly including PEMT2 which emerges near conclusion of gestational status as a regulator of development, growth, and as regulator of mitochondrial potential, plasticity, and control of developmental programs and cellular developmental programs. PEMT2 level of impairment is typically strongly correlate level of condition impairment and outcomes.

EPA and DHA exhibit fatty acid Configuration that resemble connected V or connect W letters, as well as resemble the keys of a piano with darker keys as carbons. Cis linkages between hydrocarbons in fatty acid extents invert the V to cause a bend. Compared to Trans linkages which produce linear, but flexible, extents of hydrocarbons, EPA and DHA exhibit two or more double carbon linkages in their fatty acid extents with a methylene bridge between these double adhered carbons, known as a divinyl methylene pattern or an interrupted methylene bridge pattern. The essential omega-6 and omega-3 fatty acids each exhibit this divinylmethane or methylene-interrupted pattern.

Divinylmethane patterns results in a interrupted methylene, as in EPA and DHA, at the sn-1 position of phospholipids, interacting with the methylene bridge in the sn-1 linkage to oxygen, the oxonium exhibiting phosphate group and the lead group to which these are attached such as the hydride packed lead group Choline or the unpacked strong electron withdrawing nitrile Ethanolamine. The literature does not openly express the obvious, which is that the Trans extents of fatty acids can typically exhibit characteristics of methylene bridges, suggesting their integral participation in biosynthesis and presenting simpler examples of how hydridic character and polarity are distributed across large aspects of biologically active molecules.

Regardless, the conceptual nuance of biosynthesis thus integrates conceptual nuances of creative forces of the universe and the fulfillment systems which are responsive to best fit, utility and satisfaction or fulfillment processes that satisfy creative influences.

An increase in the pentose phosphate pathway or increase in the hexose monophosphate shunt would be linked to increase in glycerol synthesis or glycolate synthesis or both, such that when this produces upregulated glycolate then the PEMT pathway selection of glycerol phosphatidylethanolamine may be diminished. Nucleotide synthesis is upregulated with hexose monophosphate pathway upregulation, being enhanced by general ribulose activity linked to 60 percent increase in nadph synthesis during hexose monophosphate activity, but reasonably link to increased glycosyl fraction of the hexose monophosphate shunt going to the biosynthetic Krebs cycle compared to shunting of glycerol factors into glycolysis near the Krebs Cycle interface with glycolysis. The glycerol selectivity by ethanolamine phosphotransferase and PEMT suggest glycosylated phospholipid tail upregulation diminishes these enzymic factors in favor of a de facto increase in the cdp-ethanolamine pathway, explaining why the cdp-choline, in some organisms, is known as the nucleotide biosynthesis pathway.

Imbalances or inadequacy, correlative diminished glycolysis and dismissed hexose monophosphate shunt catalysis, thus, emerge when PEMT is diminished, suggesting that Replication and its fraction of requisite replication competent nucleotides are diminished by PEMT to prevent Replication in conditions where PEMT is diminished used in function. However, DNA repair occurs in more than 1 million instances each day within each cellular entity, such that downregulation of glucose shuttling through glucose – 6 – phosphate dehydrogenase becomes a genomic Replication inhibitor and invite genomic repair, unless PEMT resumes its activity. P53 is repressed as in the “warburg effect”, or diversity in hexoses is exhibited to circumvent P53 imposed downregulation of GLUT. Glycolysis and pentose phosphate pathway. Arellano study observes that nucleotide imbalance or inadequacy invokes replication nonresolution cytokines such as ATR, differently from other known cytokines, to enable cellular cycle progression during nucleotide inadequacy and allow cellular entities to escape excessive growth as hypertrophy or differentiation during nucleotide inadequacy. Representing small cellular compared to large cellular difference in cellular phenotype. It is known that an ankyrin repeats repress P53, carbohydrate circumvent P53 through ChREBP activity, phospholipase and diesterase free phospholipid and Choline from cellular membranes to mimic available choline to surmount P53 downregulation of nervous pathways, and high powered phosphorylation such as cases kinase, T-Lymphocyte activation of Ligands, as well as immunological response, all can upregulate pathways repressed by PEMT including expanding the group of specific cellular entities allowed through P53 to P21, to P27 and pRb phosphorylated status as a Regulator in this regard, as gated pathways applying coordinated cyclin function to complete the cellular cycle. Thymidylate performs an essential role in nucleotide adequacy while nucleotide adequacy likewise is essential to cellular entities escaping G phase and S phase to progress to subsequent mitotic or meiotic phases.

ATR escapes cellular entities from the beneficial effects of nucleotide inadequacy and the detrimental effects of cellular hypertrophy, which may be circumstantial beneficial, although, like cytokine increases over extended periods otherwise, can enable exhibition of diminished health status and risk if exhibited for extended duration. PEMT, thus, seems to expect diverse hexoses to be available during its diminished function enabling substrate to be shunted into glycolysis as well as allowing Rubisco supply of glycerol substrate to be shunted into glycolysis near the Krebs Cycle and enabling glycolate substrate to be inserted into the Krebs Cycle.

PEMT might have no specific regard for D chiral Glucose except that when Glucose - 6 - Phosphate Dehydrogenase produces glucono lactone from Glucose phosphate nadp⁺ is used as a cofactor and becomes nadph which decreases the essential increased levels of nadp⁺ when nadp⁺ is compared to nadph, a disparity that is a foundational enabler of activity, gradients, transactions and flow of hydridic current in physiology and on biology generally. P53 seems to prevent both production of nadp⁺ and Glucose phosphate during gluconeogenesis as well as prevents production of glucono lactone and nadph because PEMT diminished function impairs the flow of hydridic current through structure as a priority. Structure is essential in trapping current and controlled directing of current, enabling cellular capacitance and potentials, post synaptic neuron polarization baselines, hydridic effect, and galvanizing of concerted tissue capacitance linked to consciousness and cognition. The spooky spatial aspects of these influences are another dimension of why trapping and recycling current is a priority, such that shuttling current through structure enables the foundational biological compartment to exist and function in ways that are increasingly both spookily biological and strangely biological.

The literature is in consensus observation that aggregate methylene bridge cysteine diminishes PENT, although vague in clear presentation of if s adenosyl methylene bridges only, instead both s adenosyl and s adenosyl bereft methylene, are mechanistic downregulators of PEMT. However, s – adeonsyl methylene bridge cysteine is a downregulator of PEMT and diminished performance of translation of s – adenosyl methylene bridge cysteine into methylene bridge cysteine by the hydrolase SAH decreases cellular division and causes hypomethylation of Genome. What is clear is that is that SAHH is redox or nad⁺/nadh ratio regulated, and the um/L linked to detrimental changes is 0.012 for s adenosyl methylene bridge cysteine compared to 6 or 7 um/L for methylene bridge cysteine, suggesting a potency variation, although there are pathways for specific detox of each of these varieties of methylene bridges and there is likely attenuation between these methylene bridge fractions in diverse metabolic conditions.

The activation potential for SAHH has been solved, in a study, as NAD⁺ increased comparatively to nadh, suggesting it is inherently and strongly potentiated toward translating s-adenosyl methylene bridge cysteines by release of the adenosyl moiety, producing nadh from nad⁺. However, the same study observes that SAHH then proceeds to synthesized adenosine which is a downregulator of choline kinase alpha attachment of ATP to Fee choline at the incipient phase of the cdp-choline pathway, resulting interestingly, in production of nad⁺ from nadh. Thus, s- adenosyl methylene bridge cysteine is trapped by diminished nad⁺ and produced when nadh levels are increased, while P53 reinforces the glycolytic translation of glucose into pyruvate that already potentially occurs when nad⁺ is diminished in availability from its typically strong prevalence over nadh. These are clearly two among other mechanisms that manage structural molecular to phases of energy or flow of current. Pathways of methylene cysteine bridge processing, deterioration, or recycling which do not produce adenosine have an increased potential of upregulating the cdp-choline pathway because it relieves deterioration of PEMT diminished packing of Hydride by using already produce choline lead groups packed with hydride and CH₃ to produced phosphatidylcholine using non newly produced choline lead groups. Phosphatidylcholine and phospholipid structure is produced from recycled structure when PEMT is not adequately synthesized choline lead groups. Particularly, redirecting current from escape, depletion and attenuation toward, instead, reintegration into membranes and

structure. The priority seems to be structural translation, sustainment and cycling if current, particularly hydridic aether.

Redirecting of s-adenosyl methylene bridge cysteine toward thioether methyl transferase results in production s adenosyl methionine while each catalytic action by thio ether methyl transferase results in polymerization selenium, tellurium. sulphonium, other amine, or other factors, while trimethylsulphonium is then used as a substrate for thetin methylene bridge cysteine transpherase production of the desquamation depolymerization factor used pervasively in therapeutics production in the 1900s and 1900s, while dimethyl thetin performance as an alternate for trimethylsulphonium has been presented in the literature since 1878, methylene bridge cysteine has been presented in the literature since 1810, and lecithin as mixed choline and phosphatidylcholine was characterized in the literature in the middle to later aspects of the 1700s.

Some of the literature limits thioether s methyltransferase to bidirectional translation of dimethylsulfide and s-adenosyl methylene bridge cysteine into trimethylsulphonium and s-adenosyl methionine. Trimethylsulphonium tetrahydrofolate produces, bidirectionally from trimethylsulphonium and tetrahydrofolate, the products dimethylsulfide and 5-methylenetetrahydrofolate substrate for one carbon MTFHR/methionine synthase /methionine synthetase pathway processing if methylene bridge cysteines into methionine and s-adenosyl methionine.

Methylene bridge Management pathways and factors, such as methylthioglycolic acid, elute or derive molecules that affect methylene bridge polymerization, energy sequestration, and ability to be commandeered to change current and structure as well as affect how accumulation of methylene bridges potentiate typical and atypical development, differentiation and bending of spooky aspects of biology toward anomaly of seemingly idiopathic origin. Derivatization occurs in this regard in almost any environment in the universe because methylene bridge factors and foundational aspects of biology with which it interacts are found in the biome and in space, exhibiting how these molecules are active caustic pathways that sequester a space in the biome for biological systems, transfer useful products from abiotic phase into biotic phases and increasingly derivatives factors in the biotic phase in service to physiology and in service to the foundational biological compartment which are cellular entities.

The human inclination to derivatize important contexts to empiricism implores what may have been futile endeavor, in the more than two centuries since methylene bridge cysteine was first characterized, to simply explain the methylene bridge multiplicity. The methylene bridge sequestration by biosynthetic strong electron withdrawing groups sequester hydride and galvanized molecules, structure, metabolism and development of biological systems as a result. Much of human activity and behavior seems to be likewise sequestered to such priority or shaped in ways that indicative of such priority. However, the application of methylene by biosynthetic strong electron withdrawers, through polymerization potential, transforms current into structure, explaining why and how mitochondria, sometimes hundreds in an individual cellular entity, effect and regulate developmental programs, particular through PEMT and particularly through

mitochondrial PEMT2 activity which typically emerges near the transition from gestational phase.

ATR be a therapeutic locus of susceptibility in diminished health status as well as may be a locus of support in supporting resumption or stabilization of homeostasis.

Systems modeling perspectives observe correlation between hypomethylation and s-adenosyl methylene bridge accumulation, particularly linked to diminished unimpeded flow of methyl resources and, particularly, with diminished PEMT throughput. Increased methyl group resources without methyl resource metabolic processing and increased cysteine bridge exhibition without metabolic processes at result in accumulation, aggregation and sedimentation, with methylene bridge accumulation potentially resulting in. Both, deposits without requirement of being integrated into complexes, but also potentiating integration into complexes that activate methylene bridges to transfer hydride for translation into structure polymerization activity. Slowing metabolism may cause methyl and cysteine bridge requirements to decrease, resultant in methylene bridge accumulation within junctures of metabolic pathways, particular areas of physiology, as structural polymerization, or as mechanisms enabling less beneficial process to commandeer such dysregulated methyl resources.

The literature, in some instances, describes methylene as a carbon atom with hydrogen adhesions at each extremity, while methylene bridging occurs when a methyl group described as CH₃, contrasting methyl groups presented as CH₃ elsewhere, is attached to methylene. The methylene bridging process results, then in sequential methylene molecules bridged together, resulting, also, in the sequential adhered methylene constituting fatty acids, particularly sn-1 fatty acids of phospholipids which can cis, trans and divinyl methylene bridges with two sequential double adhesions.

Methylene bridges are alkanes, the simplest of the carbon adhesions with single adhesions typically, promoting alkalinity, explaining some of the hydridic effect in which background pH is alkaline in the physiological environment, resulting in change to the environment encapsulating phospholipids, fatty acids, methylene bridges, and cellular membranes. Methylene is described as a colorless gas that integrates with atmospheric hydrogen to produce methane while methylene can be rapidly, also, oxidized into Carbon monoxide and water, although being soluble water. These characteristics confirm. the aether characteristics of hydride because the two hydrogen atoms of methylene are linked logically to hydride in many statuses or versions in the environment, because the literature observes trios of hydride participating when hydride attaches to surfaces, because activation of methylene bridges sequesters electrons from hydride through .metabolic, molecular, current or other vectors, and because methyl groups as exist and are known to attach to the leading edges of structural lattices to diminish or control expanding structure.

The hormones and glucocorticosteroids such as estradiol are interesting because the offset pentameter and offset hexameter are presented with the pentameter offset in the direction of the cardiac tissue offset with, also, the hexameter being offset in the direction of the hepatic organ offset, presenting the two uppermost organs offset from symmetry in correlation to hormone shared structure.

Because hormones exhibit aromatic resonant hexameters they interact with other molecules through space without being connected, carbocation's or hydridic character can also be shared across discontinuous structure, interesting observations can be presented.

Another analysis in the compendium of research linked to this analysis presented that the hydroxyl and chiral hydrogen of hormone including estradiol exhibit a pattern that integrates the uppermost cardiac pentameter into the adjacent hexameter, subsequently to a hexameter connected to the first hexameter, and then is interrupted by divinyl methylene spacers in the lower offset hexameter in in both directions the shared methylene bridge between the lower center hexameter and the offset lower hexameter. Hydroxide is exhibited linked to the carbon after the lower divinyl methylene spacer in the leftmost, lower most, offset hexameter. This configuration suggests that the hydrogen configuration is intended or performs weaving of the hydrogen field or hydridic potential into the lowermost offset hexameter while a methylene bridge linked to two divinyl methylene spacers, comprising one divinyl methylene sequence, performs as strong electron withdrawing circuit and the hydroxide in the leftmost. Lowermost hexameter primes this circuit. This configuration enables remote hormone methylene bridges to be activated integrally or remotely as well as enables hormone to exhibit remote carbocation, emit current, galvanize metabolism, polymerization and development, as well as participate in these physiological phenomena. Hormones weave in hydridic fields as control interfaces and PEMT performs as a satellite pin by integrating and stabilizing hydrogen and hydridic current by integrating CH₃ as Carbon, 2 hydrogen s and 1 hydride, all onto the ethanolamine lead group of phosphatidylethanolamine where the two methylene spacers become 1 methylene spacer that secures the satchel hydride, resulting in production choline as phosphatidylcholine which has three CH₃ moieties. The fatty acids in the sn-1 position are on the opposite extremity of the methylene spacer compared to the location of choline, performing along with other atoms as a stabilizing counterion or contributing stabilizing polarity, although the oxonium of the phosphate group may also contribute in such capacities.

Studies observe that amino acids perform as strong catalysts when exposed to the alkaline microenvironment, such as that promoted by methylene, methylene ridges, fatty acids and phospholipids. These gradients, thus, not only emerge in the physiological background, but emerge between the interacting molecules at the foundations of biology. Thus, metabolism seems to be self-starting, at least in this regard.

Methylene blue is a salt applied as a dye or colorant as well as is used as a therapy for methemoglobinemia which occurs when the typical trace amounts of the ferrous 2+ oxidation status of Iron in Haemoglobin/Hemoglobin become metabolized into ferric 3+ oxidation status version of Iron, which becomes clinically substantial by convention at 30% of aggregate haemoglobin/hemoglobin content either systemically or in the microenvironment. Oxygen therapy and Methylene Blue are used to counteract methemoglobinemia and counteracts the symptoms of impaired oxygen transport, blueish Green sweating, blueish/greenish/chocolate colored hematopoietic fluid/blood and excretory material, all linked to impaired metabolism, toxicity, or impaired access to essential blood gases, although these might include systemic organ destabilization syndromes. Discomfort in cerebellar area, nausea, impaired muscle coordination, cyanosis of bluish epidermal characteristics, seizures and arrhythmias are

correlated with methemoglobinemia, while other causal factors can include foods, chemicals, additives, particulate and other factors such as dapsone, benzocaine, nitrates, although the most indicative diagnostic is inadequate oxygen without responsiveness to oxygen therapy.

Hyperbaric Oxygen, vitamin C, exchange transfusion, oxygen therapy and methylene blue are typical therapies. Accompanying conditions can decrease the clinical symptom threshold to as low as 5% of aggregate Ferric 3+ Iron in hemoglobin compared to other oxidation status of Iron in hemoglobin, such that therapies such as trimethoprim, sulfonamides, dapsone, articaine, benzocaine, prilocaine and lidocaine, or particulate colorants such as anilines, rasburicase, chlorates, bromates, nitrites, umbellulone, and netoclopramide, particularly nitrates, may be primary or complicating factors in methemoglobinemia. Thiazine dye is among the heterocyclic compounds derivatized as pharmacological factors along with thiazolidinones in early therapeutics development linked to derivatization of methylthioglycolic acid at the beginning of the 1900s. These suggest that electron metabolism was integrally considered in therapeutics and methylene bridge management is confirmed as an essential, empirical indicator or factor in health, disease and therapy. Benzocaine used for gums and dental structural relief as well as lozenges for esophageal comfort are causally correlated with risk for methemoglobinemia.

Information. ISBN. (78-0-08-044705-6.

Information. United States Food and Drug Administration. Bulletin. April 7, 20011.

Information. United States Food and Drug Administration. Bulletin. May 23, 2018.

Nitrates in drinking water and therapeutics, thus, are substantial vectors of risk before or near 6 months after gestation because these are correlated with increases in risk or increased levels of Ferric 3+ Iron in hemoglobin.

The literature observes that Adult hemoglobin in early development is correlated with improved outcomes and lower risk for adverse biological outcomes, particularly lowering risk for sudden adverse health events. However, SCD and Beta-thalassemia are conditions that can be improved by reconstituted or preventing deterioration of prevalent gestational hemoglobin even as development progresses after conclusion of gestation. The literature observes that between 6 and 12 months after gestation, Adult Hemoglobin typically comes to constitute near 99 percent of hemoglobin with only 1 percent of hemoglobin being constituted of the gestational or second emerged gestational version known as hbf. hbf or gestational Hemoglobin exhibits 2 globin domains according to the literature, such that during first few weeks after conception the conception Hemoglobin can have about 20 times less stable tetra/dimer affinities than adult hemoglobin, presumably with gestational hemoglobin hbf being somewhere along this continuum while the dimerization/monomer stability in this same paradigm can be 500 times less stable for conception hemoglobin compared to adult hemoglobin with gestational hemoglobin being somewhere along such continuum. However, conception Hemoglobin, particularly if comprised of zeta subunits readily are exchanged for other subunits phenotypes, particularly having affinity for exchange of zeta subunits for beta subunits, resulting in stronger versions known as gestational or hbf or adult hba versions of hemoglobin. Dimer stability or structural affinity strength is observed in one study to flow from less stable to more stable as portland-2 zeta2/beta2, portland-1 zeta2/gamma2 equal to gower-1 zeta2/epsilon2, gower-2

alpha2/epsilon2, hbf1, hbf as alpha2/gamma2, hba2 as alpha2/delta2, thereby presenting a continuum of increasingly stable monomer to monomer interactions within the hemoglobin structure.

Omega-3 fatty acids EPA and DHA are both potent diminishing therapies to decrease discomfort or adverse health events resultant of SCD and similar diseases.

Although the subunits are presented in the literature are being expressed differently during phases of development, competition among subunits favoring stability seems to prioritize availability of more stable subunits and more stable hemoglobin phenotypes. Liganded hemoglobin can have 20 times more stable structure than unliganded hemoglobin, while liganded typically indicates CO integration or Oxygen integration and while about 20 to 25 percent of hemoglobin can typically be performing transport of CO metabolites or molecules. Oxygen integrated gestational hemoglobin hbf is presented as being 70 times less catalytically potentiated to abdicate dissociated dimers when compared to adult hemoglobin hba, thereby explaining gestational hemoglobin resistance to malaria toxicity along with explanation of gestational hemoglobin resistance to aggregation of SCD hemoglobin, particularly potentiated through the enhanced stability of the gamma subunit as observed is experimental hybridization. Acetylated gestational hemoglobin, which represents as much as between 20 and 10 percent of aggregate gestational hemoglobin produces, is presented in the literature as resulting in decreased stability of gestational hemoglobin. However, experimental data suggest that oxygen prefers integrated into deoxygenated hemoglobin subunits without preference between beta or alpha subunits, while organic phosphate level increase is correlated with preference for oxygen integration into the alpha subunit of deoxygenated hemoglobin while indicators of changed affinity suggest also that beta subunit variance in oxygen integration in the presence of inorganic phosphate seem to be the result of competitive subunit exchange dynamics. These explain changes to hemoglobin and oxygenation of hemoglobin when upregulation of phosphocholine and the cdp-choline occur, as well as potentially suggest differences in hemoglobin activity when increased levels of electron transport pathway production of ATP occur.

Hbf to hba shift occurs in reasonable correlation to the GABA switch in which GABA changes from being an excitatory modulator of neuronal polarity to become a downregulator or neuronal excitatory polarity upon stimulation by neurotransmitters. The GABA switch occurs in reasonable correlation of the NKCC1 to KCC2 switch in which KCC2 becomes increased compared to NKCC1. Inhibition of KCC2 experimentally prevents increase in KCC2.

Impoverished environments delays inversion of GABA signaling and presumably delays KCC2 expression. Selective therapeutic downregulation of NKCC1 rescues down syndrome symptoms, mimicking the same effect produced by phosphatidylcholine supplementation. Cl⁻ are substantially diminished after the NKCC1 to KCC2 transition. IGF-1, exhibited in breast milk, mimics environmental enrichment in causing earlier transition of the NKCC1 to KCC2, and, correlatively, the transition of GABA from being excitatory in its modulation of neuronal activation to becoming, instead, a downregulation modulator of neuronal level of activation. Gaba is instrumented for neurons during development, priming the environment for synapse developing through signal amplification, followed by downregulating such signals after the NKCC1 to KCC2 Gaba switch, presumably enable patterning of synapse and neurological

characteristics to environment during development compared to reliance upon the enhanced neuronal density and structure after development in a way that requires downregulation of signal sampling by neuronal infrastructure.

Information. *Ann Nutr Metabo*. Volume 65. Number 4. Pages 317 to 323. 2014.

Information. *PLoS ONE*. Volume 6. Number 8. Article e23020. 2011.

However, separation of early developing mammals from maternal hosts during early development delays the NKCC1 to KCC2 GABA switch. However, these phenomena are closely correlated with the inversion of the polarity direction exhibited by neurons which are stimulate by the neurotransmitter Cl⁻ and potentially other neurotransmitters also. The compendium of research linked to this analysis observes that breast feeding introduces diverse advantages, social outcomes improvement, duration of being, social function, levels of susceptibility to disease, potential for genetic conditions, conditioning, recall of conditioned association, blocking as an aspect of conditioning, memory, prevention of neuronal deterioration, and other effects, and previously this context was oversimplified by suggesting that the NKCC1 to KCC2 switch was delayed by breast feeding when, instead, either breast feeding delays the NKCC1 to KCC2 GABA switch or it is possible that choline adequacy is distinct mechanism that enables patterning by GABA by sustaining PEMT activity to enable Oxytocin expression to chaperone and invoke the NKCC1 to KCC2 GABA switch. A useful example includes the observation that PEMT function enables decreased P53 which enables more complete throughput through glycolysis, such that increased levels of Acetyl-CoA can be used to for choline acetyl transferase production of acetylcholine that performs as a neurotransmitter and storage factor for choline, while inhibition of PEMT diminishes both acetylcholine and Cl⁻.

Oxytocin, which is enabled by the function of PEMT, performs as an emotional, social, and psychosocial patterning and linkage factor that enables mammals and other organisms to establish social, emotional, reproductive, and other correlated linkages. Oxytocin Receptor coordinates the NKCC1, KCC2, GABA switch through Oxytocin Receptor which, when activated, upregulates KCC2, thereby promoting GABA excitatory modulation becoming inverted to become a downregulator of neurotransmitter signal intensity. These factors are all in reasonable correlation with hbf hemoglobin transition to hba hemoglobin. Certainly, choline adequacy, particularly from breast milk, and methyl group availability, as well as tight regulation of methylene cysteine bridge activity, all are linked to oxytocin upregulation, and NKCC1 to KCC2 GABA switch, in conditions of optimal development, all of which compares to choline inadequacy which may stimulate the same transitions for different reasons linked to deteriorated availability acetylcholine resultant of diminished availability of breast milk factors IGF-1, Choline, methyl groups and other factors.

Information. *Blood*. Volume 117. Number 15. Pages 3945 to 3953. 4th Month, 14th Day, 2011.

Information. *Cold Spring Harb Perspect Med*. Volume 3. Number 1. Article a011643. 1st month, 2013.

Information. *Pediatr Res*. Volume 76. Number 5. Pages 477 to 482. 11th Month, 2014.

Information. Arch Pediatr Adolesc Med. Volume 158. Number 4. Pages 366 to 371. 2004.

Information. "Cellular" Reports. Volume 15. Pages 96 to 103. 2016.

Information. Front Mol Neurosci. Volume 15. Article 893111. 7th month, 8th Day, 2022.

Information. Biochem Biophys Res Commun. Volume 493. Number 3. Pages 1243 to 1249. 11th Month, 25th Day, 2017.

Information. Neuron. Volume 15. Pages 1287 to 1298. 1995.

Information. IGF-1. Neuropharmacology. Volume 113. 2nd month, 29th Day, 2016.

Information. Chem. Volume 6. Number 8. Pages 2073 to 2096. 8th Month, 6th Day, 2020.

Information. Cold Spring Harb Perspect Med. Volume 3. Number 1. Article a011643. 1st month, 2013.

Information. "What is the Difference Between HbF and HbA Hemoglobin." Doctor.ndtv.com.

Each of the two subunits of hemoglobin exhibit two additional subunits which, themselves, exhibit a heme or iron moiety utilized to host integrated oxygen, nitric oxide, CO, etc. These explain why L-arginine is a substantial and remarkable therapy for an extensive array of disorders and diseases. Sulfohemoglobinemia and Carboxyhemoglobinemia are linked to reactions to therapy, toxicity and systemic anatomical dysfunction, while also Heinz anomalies can also arise in this context in correlation in different factors in the physiological environment. Thiolated or S-Nitrosothiols are powerful vasodilators similarly to L-arginine and L-citrulline.

Information. Proc Natl Acad Sci U S A. Volume 75. Number 11. Pages 5462 to 5465. November, 1978.

Information. Protein Sci. Volume 16. Number 8. Pages 1641 to 58. 8th Month, 2007.

Information. ISBN 978-3-030-41768-0.

The link between hemoglobin F and SCD, beta-thalassemia, other similar disorders is very interesting because a systems view reveals that although increased level of gestational hemoglobin may be therapeutic, the context of diminishing expression of gestational hemoglobin is canonically a decrease in the number of cellular entities or cellular phenotypes that continue to express hemoglobin F or hbf. Thus, an estimated 7 percent or less of erythrocytes expressing hemoglobin F is complicated by diminished expression of hemoglobin in these versions of cellular entities. Studies observe correlations between the number of cellular entities expression hemoglobin F and the levels of systemic hemoglobin F. Hydroxyurea and Erythropoietic therapy together have substantial ability to stabilize diseases such as SCD.

Genetic disease should become obsolete with full implementation of CRISPR gene editing. However, genetic assay occurring before such implementation should include understanding what the substrate and cofactors are for any impaired enzyme and understanding of what the products are for such an impaired enzyme along with supplementation of either products or both products and substrates. Recombinant versions of such impaired enzymes should also be

available, while the industry should be rapidly moving to provide all of these possible solutions for most, if not all, impaired enzymes and deficiencies.

Information. N Engl J Med. Volume 328. Number 2. Pages 73 to 80. January, 1993.

Information. Blood. Volume 46. Number 5. Pages 671 to 682. November, 1975.

The P50 or partial pressure at which hemoglobin F is 50 percent oxygenated is about 19 mmHg. The P50 of hemoglobin A is about 26.9 mmHG. Hemoglobin F, thus, is more easily, more completely and more rapidly integrated with Oxygen compared to Hemoglobin A.

Information. ISBN 978-0-323-39006-4.

Thiamin pyrophosphate is remarkably interesting because it presents how patterns of atoms in a molecule can comprise a field that traverses molecular structure, particularly when these atoms are indicative of strong electron withdrawing groups, and particularly when methylene bridges and hydridic character effects promote signing and packing of current within the molecular structure. Thiamin pyrophosphate exhibits a methyl group CH₃ joined to the upper left most corner of the leftmost cyclic hexameter hydrocarbon ring where the carbon linked to CH₃ is connected to right or clockwise toward an apical Nitrogen by a single adhesion Nitrogen that is has a double adhesion to a carbon shared with another hexameter along a shared wall comprised by another clockwise single adhesion to carbon. Followed by separation of the hexameters in clockwise direction through a double carbon to carbon adhesion, followed, then, by a clockwise single carbon to nitrogen adhesion, then concluding with double adhesion between this nitrogen and the carbon to which the methyl group is linked. It is clear that the Nitrogens are asymmetric to the hexameter but somewhat symmetric to the methyl group with double adhesion in the clockwise direction for each nitrogen, both strong electron withdrawers, and both clearly drawing current away from and then toward the Methyl Group which has an activated methylene bridge that has galvanized, sequestered or continues to galvanize delivery off or continuously sequesters current, eV-, e-, e- integrated into hydrogen as Hydride or H (e²⁻) (p1).

The leftmost hexameter shares a single adhesion carbon linkage as a cyclic hexameter hydrocarbon wall with the centermost cyclic hydrocarbon hexameter which, clockwise, exhibits the uppermost shared carbon of such shared wall participating in an adhesion with NH₂ in the apical uppermost position within the ring although the literature does not present the NH₂ with a line linking this NH₂ to the uppermost carbon in a shared double Carbon to N⁺ adhesion, but presents this as an NH₂ linked to the N⁺ either instead of the carbon, or integrated into such uppermost carbon of the shared wall with the third cyclic hydrocarbon or as NH₂ linked to the carbon but unable to be represented because of the font size in required to represent NH₂ in the visualization. The shared wall seems interestingly vague although the structure of the middle hexameter certainly follows, in other respects, the same characteristics otherwise of the leftmost hexameter, suggesting the visualization, although consistently represented in thiamine within the same visualization otherwise, might not be suggested any particular anomaly. Regardless, the the shared wall between the middle and rightmost hydrocarbon rings exhibits a double adhesion with a lowermost N⁺, followed by a subapical or lowermost CH₂ in clockwise direction, followed by a single adhesion to a carbon, followed by the carbon to carbon single adhesion shared with the leftmost hydrocarbon hexameter ring.

These clearly present that a true methylene bridge is linked to an N^+ cation in a way that it may be performing as an electron withdrawing moiety along with linkage to NH_2 or Carbon/ NH_2 moiety in the counterclockwise direction from the N^+ through a double adhesion, all which suggest that current is being drawn in some direction, possibly counter clockwise toward the N^+ and possible from the methyl group in the rightmost aspects of thiamin pyrophosphate. The rightmost hydrocarbon pentameter ring exhibits linkage to the uppermost NH_2 /Carbon of the shared single adhesion to the N^+ in the shared wall with the middle hexameter, such that the uppermost atoms or atom group in the shared wall is linked to the apical nonionized Sulfur of the rightmost pentameter, followed by linkage to the a Carbon through a single adhesion, followed by linkage to another single adhesion to carbon, followed by, along the lowermost unattached wall, a single adhesion linkage in the clockwise direction between carbon and nitrogen. This suggest that current is being directed from left to right, although it is clear that each field comprised of atoms in the thiamin pyrophosphate molecule may be distinct and may be merely converging into and out of the thiamin pyrophosphate molecules to comprise converging, interactive but otherwise distinct systems of current and affect to material, particles, quantum, atom, angular, spatial and other nuances of systems from the microenvironment to systemic and universes level, representing trillions or more of interactive systems and participation in fields that extend to universes levels. The rightmost carbon to carbon wall of the rightmost pentameter exhibits a CH_3 adhered to the lowermost carbon, while the upper carbon of this rightmost pentameter wall exhibits a single adhesion to a methylene bridge as CH_2 , followed by a single adhesion between this CH_2 and another methylene bridge, followed by a single adhesion of such second methylene bridge CH_2 to an oxygen of a phosphate group followed by attachment of this first phosphate group to another phosphate group. Although the lowermost oxygens of the phosphate group are attached to their own hydrogen, and although the unencapsulated oxygen of the concluding phosphate groups is represented as hydridic oxonium, convention considers each of the oxygen atoms before the phosphate group possibly, but at least between the phosphate groups, all as hydridic oxonium, at least according to some of the literature.

The electron transport pathway of oxidative phosphorylation or cellular respiration pathway, frees hydride to emit eV- of current or fluorescent energy that activates the subsequent aspects of the oxidative phosphorylation pathway by loadbalancing such component in democratized loadbalancing way, resulting in utilization of about 58 percent of such energy for pathway catalysis and packing of about 42 percent of such current or energy as hydride packed into the oxonium exhibited between phosphate groups of ATP or adenosine triphosphate. The structure of thiamine suggests that Phosphate groups and oxonium within such groups can perform as batteries or energy storage modules which molecular structure can withdraw, utilize, replenish, move into different areas of the molecule, shift through carbocations, focus into intramolecular, extramolecular or intermolecular domains, all in a way that can be affected by or effect ionic, hydridic, spatial, molecular, quantum, angular, or other characteristics involved in biology. These provide a very important view of intangible aspects of molecular interactions that may have been inadequately considered in earlier eras, particularly in eras when adequate nutrition may have been excluded as substantial causes of diminished Human outcomes. Thiazoles are linked to thiazolidinones which have storied experience in therapeutics development since the 1910s or earlier. Information. "Thiamine1." Basicmedicalkey.com

Erythrocyte production begins the encompassing material of the newly produced conception compartmental structure, followed by migration to the hepatic tissues, followed by migration to the inner aspects of bones, all of which are known to involve agrin, particularly involving agrin activity within the spongy aspects of bone structure. Where a central macrophage chaperones development of erythrocytes through development, excretion of the nucleus as a pyrenocyte, release of the reticulocyte, and flipping of phosphatidylserine onto the outer membrane of the pyrenocyte as an 'eat me' signal that stimulates macrophage engulfment by the central chaperone macrophage or by other macrophages or immunological cascade.

SCD, according to the literature, exhibits substantially changed, as in decreased or ameliorated otherwise, levels of polyunsaturated fatty acid species at the sn-2 location of all 31 of the phospholipids included in the study, although changes in these factors are pervasively known to ameliorate structural phase development progressions that determine biological structure while the replacement of these optimal unsaturated fatty acids with either saturated fatty acids or monounsaturated species are known to be linked to diverse pathology. The unconfirmed literature, such as in the already produced but at this instance unascertainable literature, clearly has presented a therapeutic influence of phosphatidylcholine, particularly enriched phosphatidylcholine, to SCD disease.

The data regarding Vitamin D supplementation and Folate supplementation were not remarkable, such that the literature linking choline and phosphatidylcholine supplementation to digestive pathway production of trimethylamine-n-oxide sometimes imprecisely concludes that choline and phosphatidylcholine nutrition or supplementation increases atherosclerotic risk, risks that can include diminished outcomes in SCD and similar disease. However, these studies are counteracted and mitigated by the fact that complete supplementation with choline, phosphatidylcholine, folate, all B Vitamins, glycine betaine, s methyl methionine sulfonium, 65 5678 tetrahydrofolate, sulfur such as methylsulfonylmethane, along with detoxification of dimethylglycine because dimethylglycine downregulates methylene bridge cysteine recycling into methionine performed by both BHMT/BHMT2. Glutathione and tetrahydrofolate as well as Zinc are presented in the popular literature as depletion factors for dimethylglycine, although dimethylglycine dehydrogenase produces sarcosine, formaldehyde and a reduced electron receptor from dimethylglycine, water and an electron acceptor, presenting, again, how metabolism pervasively is centered on movement of current into particular statuses such as storage, molecular energy, chemical energy, structure or eV-. FAD is the preferred electron acceptor while dimethylglycine dehydrogenase also produces glycine from sarcosine, participates in serine metabolism and participates in threonine metabolism. Electron transferring flavoprotein hETF regenerates oxidized FAD by accepting the electron from reduced FAD or FADH₂, while subsequently transferring hETF electrons to membrane anchored ETF-ubiquinone oxidoreductase ETF-QO which directs the electrons toward the mitochondrial electron transport pathway of oxidative phosphorylation or cellular respiration. Sarcosine Dehydrogenase similarly catalyzes linkage to the electron transport pathway by electron-transferring protein ETF through catalysis homologous to dimethylglycine dehydrogenase, such as sarcosine, electron acceptor and H₂O being bidirectionally converted to glycine, formaldehyde and a reduced electron acceptor. Sarcosine and dimethylglycine catalysis is bidirectional. Importantly, choline is oxidized bidirectionally according to thermodynamics and NAD⁺/NADH balance to

trimethylglycine or n,n,n, glycine betaine through the intermediary betaine aldehyde, followed by production of methionine from methylene bridge cysteine and n,n,n glycine betaine within a context that is enabled to be more efficient by a complete B vitamin supplement.

Dimethylglycine, again, downregulates both BHMT and BHMT2 requiring detoxification of dimethylglycine or use of other methylene bridge cysteine depletion pathways. The literature presents an unfortunate correlation of choline and dimethylglycine with increased Superoxide and H₂O₂, in a way that was experimentally lowered by diminishing the activity of complex II and Complex III of the electron transport pathway, although, quite plainly, the electron transport pathway already produces 4 molecules of H⁺ immediately after complex II by activity of complex III along with already producing 2 molecules of H⁺ immediately after complex III within complex IV, such that choline may merely be upregulating dimethylglycine through oxidation of choline into n,n,n trimethylglycine while also choline availability may be relieving inhibition of PEMT and relieving the regulatory influence of P53 upon energy metabolism output.

Information. Febs Letters. Volume 590. Issue 23. December, 2016.

It is important to present that choline oxidation throughput is affected by PEMT availability because of P53 is upregulated when PEMT is diminished in function, the cdp-choline pathway is upregulated also to attach atp to choline when PEMT is diminished in function, and when P53 is upregulated, the levels of pyruvate are substantially diminished also, downregulating the directing of pyruvate to Acetyl-CoA, while availability of Acetyl-CoA for choline acetyltransferase also becomes downregulate to diminish storage of choline as acetylcholine in a way that increase free choline for cdp-choline pathway catalysis, choline oxidation pathway, ore utilization be nonoptimal processes, conditions, contexts, and pathology.

BMT2 is expressed in very low levels in diverse tissues and substantially in hepatic, renal, proximal tubular, adipocytes of breast, visceral adipocytes, subcutaneous adipocytes and hepatocytes, according to the Protein Atlas Online. BHMT is expressed substantially in renal, proximal tubular and hepatocytes, again, according to the Protein Atlas Online, proteineatlas.org.

Information. The FEBS Journal. Volume 283. Issue 19. Pages 3587 to 3603. October, 2016.

Information. J Lipid Res. Volume 38. Number 12. Page 2516 to 2528. December, 1997.

Information. ISBN 978-0-12-820155-8.

Experimental comparative analysis of cellular proliferation at the microbial level revealed that when choline is the primary source of carbon in high salt conditions, decreasing choline prevented growth, while when excessive salt conditions exhibited glucose as the primary source of carbon, then depletion of glycine betaine resulted in diminished growth in a way that could not be mimicked by decreasing choline. These present a practical application of osmoprotection, such that choline direction to the cdp-choline pathway and methyl group availability become primary focus in enabling adequate membrane phospholipids when choline is available without adequate glucose as a source of carbon, compared to glucose availability, when prominent supplier of carbon without adequate choline, being prevented from supplying adequate material for cellular growth when glycine betaine is removed from recycling pathways. These clearly

present that metabolic pathways are foundationally focused on supplying methionine, s-adenosyl methionine and methyl groups from membrane phospholipid synthesis and maintenance as an existentially priority, with PEMT de novo synthesis of choline being a primary source for optimal enriched phosphatidylcholine, storage of methyl groups, storage of energy resources, membrane stability and tissue stability. Although the microbe in this study is associated with nosocomial disease and resistant microbial conditions, diminished availability to both choline however, Phage therapy which applies the diverse phages available in nature, physiology and the biome to engulf and eradicate microbes therapeutically is emerging as a widely inclusive therapy. CRISPR directed toward bacteria either as excision vectors or antisense RNA/DNA impedance, also may be beneficial in this context, including also being potential effective for diverse microbial conditions.

Information. J Bacteriol. Volume 194. Number 7. Pages 4718 to 4726. 6th month, 29th Day, 2012.

The literature clearly observes that the correlation between choline and trimethylamine-n-oxide as well as phosphatidylcholine and trimethylamine-n-oxide, supplementally, is systemically complex, such that when control groups were compared to phosphatidylcholine supplementation groups and separate choline supplementation groups, it was phosphatidyl choline which exhibited the least atherosclerotic lesions and structural remodeling compared to increased levels of atherosclerotic indicators both controls and choline supplementation groups. The most interesting perspective was that plasma trimethylamine-n-oxide was 200 percent higher in the phosphatidylcholine supplemented group compared with control groups and compared with choline supplementation groups. Plasma trimethylamine-n-oxide typical results from cascade of superoxide to H₂O₂, Peroxynitrite, Hypochlorite then trimethylamine-n-oxide prevented by vitamin c, vitamin a, tetrahydrobiopterin, N acetyl L cysteine and L arginine, while also trimethylamine-n-oxide results from nutritional obtainment of meat, chicken, eggs, fish or choline/phosphatidylcholine dense nutritional factors otherwise, along with suboptimal digestive pathway microflora, microflora exhibiting tma lyase activity, trimethylamine or trimethylamine-n-oxide constitutively, butyryl factors of particular version, all accompanied by tnfr alpha enabled relaxation of tight junction proteins in the digestive pathway which allows transit of trimethylamine into the splanchnic system areas where Flavin monooxygenases reduce trimethylamine to trimethylamine-n-oxide, resulting addition of an electron to the outermost oxygen to produce an oxonium in trimethylamine-n-oxide that is similar in structure to the molecule choline. Trimethylamine-n-oxide is known to interact with methylene bridge cysteine by producing a resilience to both methylene bridge cysteine correlated pathology as well as resilience to trimethylamine-n-oxide correlated pathology. Trimethylamine-n-oxide is known as being among the most, if not the most, causally linked indicator of susceptibility to adverse health events, perioperative complication and sudden adverse health events, including sudden adverse behavior.

Information. J Nutr Biochem. Volume 92. Article Number 108617. 6th Month, 2021.

Importantly, SCD disease can exhibit duration of RBC circulation before by splenic recycling or hemolysis at 20 days instead of 120 days. This seems to be an aspect of pathology although aggregation of SCD specific erythrocytes may be more resilient than 20 days. A separate study

clearly observes that flippase/floppase/scramblase enzymes may be flipping phosphatidylserine to the outer leaflet of plasma membrane of the nucleus which is typically excreted from erythrocytes may be exhibiting phosphatidylserine on its outer leaflet to early before being excreted from the erythrocyte, although its possible that the nucleus may be unable to be excreted from the erythrocyte, each of which might result in autoimmunological activity towards the erythrocytes. However, it is known that phase progression of phospholipids and curvature of membranes are all affected by ameliorated levels of each phospholipid or fatty acyl species associated with each phospholipid, such that changed levels of phosphatidylserine, phosphatidylethanolamine and phosphatidylcholine in the outer and inner membrane leaflets of membranes are known to ameliorate structure and curvature of membranes. LPCAT/MBOAT shuffling and distribution of fatty acids evenly or more diversely among aspects of cellular membranes, when impaired, may also result in some aspects of membranes having impaired curvature while others may have enhanced or typical curvature. Methylene bridge cysteine levels, trimethylamine-n-oxide and other nonresolution cytokines, choline and phosphatidylcholine inadequacy and including diminished PEMT function, all have the potential contribute to, exacerbate or even invoke such syndromes to pathology, such that managing this group of factors is pervasively beneficial therapeutically to and preventative of diverse pathology.

Information. J Clin Invest. Volume 71. Number 6. Pages 1570 to 1580. 6th Month, 1983.

SCD and beta-thalassemia are both conditions that are correlated with impaired pentose phosphate hexose monophosphate shunt pathway, Heinz anomalies, membrane lipid peroxidation, and conclusively, splenic accumulation of methylene diphosphonate, presented clearly the pathways that are impaired when PEMT is inhibited, interactive pathways involving methylene bridge sequestration of current into chemical/molecular/structural aspects energy, and the factors that occur when methylene cysteine bridge cycling and regulation become dysregulated. Interestingly, increase in glucose 6 phosphate dehydrogenase was also observed, suggesting that the “warburg effect” is activate in SCD, resulting in dysregulation of glycolysis and pentose phosphate pathway as upregulation occurs when PEMT is diminished, occurs when P53 is upregulated, and occurs among diminished PEMT balancing of energy production and mining by packing hydride into newly synthesized enriched phosphatidylcholine. Methylene blue instrumentation increased pentose phosphate hexose monophosphate shunt pathway activity in this context.

Am J Hematol. Volume 15. Number 1. Pages 1 to 13. 8th month, 1983.

Information. Int J Nucl Med Biol. Volume 10. Number 4. Pages 269 to 270. 1983.

Osteoclast differentiation and fusion are essential to recycling of bone structure, such that phosphatidylethanolamine mobilization is essential to osteoclast differentiation and function, presenting among diverse other data that phosphatidylethanolamine and its cysteine bridges are integral to diverse aspects of physiology, physiology and health. Dysregulation of phosphatidylethanolamine or ethanolamine may be substantially different than dysregulation of methylene bridge cysteine.

Methemoglobin Reductase or Ferricytochrome-b5 reductase utilizes NADH, NADH exhibiting factors, or factors that delivery Hydride or the extra electron integrated into hydride to the FAD domain of Ferricytochrome-b5 as a cofactor to metabolize Ferric 3+ Iron to Ferrous 2+ Iron, although the enzyme exhibits an integrated FAD domain which is integrally involved in using NADH to perform such catalytic conversion of Fe 3+ to Fe 2+. Canonical cytochrome-b5 reductase catalysis includes NADH, H⁺ and ferricytochrome-B5 as substrate for cytochrome-b5 reductase to produce NAD⁺, 2 molecules of ferrocycytochrome-b5 as products. NADH-Cytochrome -b5 reductase is found in pervasive cellular versions although a soluble version that is truncated to include only the larger domain or encoded as an alternative transcript is exhibited in erythrocytes. FADH distributes the Hydride or hydridic character from NADH and distributes it to the small molecules of cytochrome-b5. Movement of hydride or Electrons or current is a revealed integral priority in many more complex interactions among small and large molecules, as well as complex and simple molecular, metabolic or chemical reactions, including sometimes complete molecular pathways. Cytochrome-b5 reductase also performs elongation and desaturation of fatty acids, participates in metabolism or detoxification of therapeutics and participates in cholesterol biosynthesis.

Information. Crit Rev Biotechnol. Volume 34. Number 2. Pages 134 to 43. 6th Month, 2014.

Methylene Blue instrumentation along with Phototherapy causes massive deterioration and apoptosis among breast oncology tissue bases with considerably less effect to typical cellular entities in tissues. Methylene blue is a therapy for Alzheimer's disease, is a therapy for behavioral health and psychological/psychiatric conditions, increases flow of blood to the brain, supports mitochondrial function, and performs as an electron pathway factor that reduces superoxide to water, circumventing the role of superoxide dismutase production of H₂O₂ from Superoxide as well as circumventing the role of catalase in producing water from H₂O₂, such that the effects of trimethylamine-n-oxide, peroxyxynitrite and methylene bridge cysteine in deactivating superoxide dismutase and deactivating catalase becomes instantly alleviated by methylene blue instrumentation. Methylene blue is a cationic thiazine dye. Thus, the effects of methylene blue and photodynamic therapy were not aggregately that of canonical apoptosis pathways and included upregulation of autophagy which typically results in apoptosis among nonresistant oncology cellular entities which rely upon proteolysis for oncological phenotype while autophagy typically is only linked to oncology when a resistant phenotype has emerged. Thus, methylene blue uses irradiation from phototherapy to invoke distress signaling and shuttles the cellular phenotype through the Bag1/Proteolysis compared to Bag3/autophagy decision paradigm which involves BAX, BAK, BCL2, Cytochrome C, Mitochondrial Status and influences of methylene bridge cysteine, all of which are foundational survival/apoptosis signaling axes. The inclusion of methylene blue suggests that even these axes are reduced methylene bridge metabolism and introduction of regulatory influence through stabilization of the methylene bridge hydridic sequestration pathways to prevent dysregulation among chemical, molecular, metabolic, structural, and storage phases of hydride sequestered by methylene bridge into cellular dynamics and structural phase progression. Cytochrome C release is remarkable event in apoptosis pathways and cytochrome c is an aspect of the electron transport pathway of

oxidative phosphorylation cellular respiration. Interestingly, methylene blue circumvents complex I and circumvents complex III of the electron transport pathway and then donates electrons to cytochrome c, which is very interesting because release of cytochrome c is an important pathway of invoking cellular apoptosis or cellular fragmentation. Not only does donation of electrons enhance cytochrome c oxidase activity, but conditions and therapies that modulate complex I and complex III can be diminished by Methylene Blue. Cytochrome C oxidase is a major loci of oxygen utilization in the mitochondria and cytochrome C oxidase performs the scission of the oxygen/Oxygen adhesion while resultantly producing binuclear loci comprised of heme a 3 and CuB presumably enabled by a covalent cross link between tyrosine 280 and one of the histidine ligands. Again, proton donation for the binuclear heme a 3 and CuB active loci seems to be obtained from tyrosine 280 cross linked to the histidine ligands of CuB. Since mitochondria proliferate, depopulate, attach to mitochondria, expand to hundreds within one cellular entity, migrate between cellular entities, exhibit DNA, control cellular proliferation and exhibit capacitance including constituting strong aspect of capacitance comprising consciousness and cognitive function, its difficult to exclude mitochondria from the category of living things or living entities. Methylene Blue inhibits MAO-A, delays cellular aging, disrupts aging, improves dementia, ameliorates huntington's disease, disrupts alzheimer's, delays aging effects to skin, improves memory and cognition, increases mitochondrial NAD⁺ synthesis, increases ATP synthesis, increase brain cellular duration of being, all according popular and clinical information. Although methylene blue integrates with DNA to enable photosensitive segmentation of the foundational DNA helical structure in a way that clearly links methylene blue to the methylene bridges that comprise the linkage between molecules of DNA. Methylene blue structure exhibits three linear cyclic aromatic hexameters linked to one another with by a double adhesion carbon bridge between the hexameter exhibiting Nitrogen and Sulfur at opposite unconnected apices in the central hexameter, such that each of the complete carbon hexameters also exhibit nitrogens oriented toward the direction of the sulfur in the central hexameter, and such that a resonant unconnected balancing CL⁻ ion balances the sulfur as an S⁺ cation of the central aromatic hexameter, and such that each of these axial complete carbon aromatic cyclic hexameters have two methyl groups attached to the nitrogen that is oriented in the direction of S⁺ of the central hexameter. Thus, the linear tri polycyclic aromatic hydrocarbon links two molecules of choline, except that the choline's nitrogens are not ionized, but seem to have been replaced by the ionization of the sulfur that is ionized in the central hexameter of the of the linking structure as well as is balanced by CL⁻ paired with the S⁺, although the canonically, the CL⁻ may be considered to be balancing the whole structure of the methylene blue molecule. Some of the literature presents the complete carbon hexameter that shares a single adhesion carbon to carbon wall or bridge with the central hexameter in a way that includes a double adhesion to the nitrogen, cationic ionic character of such nitrogen and with the CL⁻ depicted as balancing this axial Nitrogen Cation, along with the sulfur presented in the central hexameter not having an ionic characteristic or not being signed. A Diverse group of oncology including multiple drug resistant lineages of oncology were all extremely susceptible to deterioration at very low dosage exposure to b-nor-methylene colchinoid PT-100, such that the exhibition of oncology cellular entity deterioration did not require apoptosis, at least not canonical nuances of apoptosis. The groups of susceptible conditions including oncology, lymphoma, leukemia, carcinoma, Nalm6, Melanoma, MCF7, acute myeloid leukemia and BJAB. PT-100 was

particularly effective, among other colchicinoids, while colchicinoids are typically derivatives of *colchicum autumnale* L which is known commonly as autumn crocus, meadow saffron or other names not mentioned here because of their lewdness, but is not a true crocuses plant although *colchicum autumnale* L is an autumn flowering plant in the colchicaceae.

Information. "B-nor-methylene colchicinoid PT-100" ACS Omega. Volume 7. Number 3. Pages 2591 to 2603. 1st Month, 11th Day, 2022.

Information. Environmental Nanotechnology, Monitoring and Management. Volume 7. Pages 110 to 120. May, 2017.

BMC Oncology. Volume 17. Number 1. Page 194. 3rd Month, 15th Day, 2017.

Information. "Methylene Blue." Thefuelstop, Website, fuelstop.com.

Information. Antioxidants (Basel). Volume 10. Number 2. Page 305. 2nd Month, 16th Day, 2021.

Information. "O-O." Proceedings of the National Academy of Sciences. Volume 105. Number 31. Pages 10733 to 10737. September 2008.

5-HT₃ receptors are postsynaptically activated by serotonin or 5-hydroxytryptamine, the neurotransmitter, resulting in opening of the 5-HT₃ receptor channel to allow a typically excitatory response. Sodium and potassium ions are typically allowed inward in a pattern of rapid activation and desensitization. 5-HT₃ receptors are at least ionotropic and is included in the Cys-Loop Supergroup that includes GABA_A in two variants, Nicotinic Acetylcholine receptors and Receptors activated by Zinc. Arylguanidines, as dihydroquinazolines including A6CDQ, are examples of how addition of a methylene bridge to arylguanidine structure performs somewhat pervasively in a 5-HT₃ receptor agonist, regardless of the function of the arylguanidine to which the methylene bridge is attached. These suggest clearly that methylene bridge accumulation results in receptor activation, enhancing the ability of methylene bridges to sequester systemic current mobility, although in this instance the example of how including methylene bridges into molecular structure can activate molecules and proteins in active status for receptor, ligand, and current activation. Methyl group introduction ameliorates the agonist function of the methylene bridge enabled arylguanidine.

Information. "Methylene Bridge to 5-HT₃." ACS Chem Neurosci. Volume 10. Number 3. Pages 1380 to 1389. 3rd Month, 20th Day, 2019.

Phenylene as cycloparaphenylene bridged in multiplicity using methylene bridges produce nonalternate aromatic belts when using nickel intermediated aryl-aryl linkages of triflate functionalized pillar arene, all of which causes methylene bridge enabled coplanarizing of paraphenylene, decrease in energy gap, increased pi conjugation, and high internal strain energies near 110.2 kcal mol⁻¹.

Information. J Am Chem Soc. Volume 142. Number 29. Pages 12850 to 12856. 2020.

Studies of nonhuman organisms indicates that increased levels of phosphatidylethanolamine are correlated to increased levels of methylene bridge cysteine when the control groups were obtaining phospholipid supplements otherwise, and a control group from nonsupplementation

was omitted from the study. There were no substantial differences between the phosphatidylethanolamine among all groups in the study, including the phospholipid supplemented control group, suggesting that not all instances of increased methylene bridge are the same, such that increased levels along with correlative increased cycling or metabolic throughput may reflect exhibition of beneficial processes and conditions.

Information. British Journal of Nutrition. Volume 94. Issue 5. Pages 684 to 690. November, 2005.

Neurodegenerative disorders, refractory discomfort linked to oncology therapy, all are presented in professional service contexts as being alleviated or prevented by methylene blue. Androgen reliant and androgen unreliable cellular proliferation disease are each responsive to methylene blue. N-Oleoyl-phosphatidylethanolamine and EGCG are both utilized to diminish the level of hunger in dieting populations, although studies observed that after about 4 weeks, such effects become diminished, although NOE which has ethanolamine instead of phosphatidylethanolamine, produces a similar effect. NOPE1, NOPE2, 1-palmitoyl-2-oleoyl-phosphatidylethanolamine, NAPE, anandamide and other n-acyl ethanolamines including PEA, OEA, NAE, AEA, DHEA/synaptamide/n-docosahexaenylethanolamine, n-acetyethanolamine, n-palmitoylethanolamine, n-oleoylethanolamine, n-arachidonylethanolamine, all are molecules used in therapy for a diverse group of conditions, diseases and behaviors.

Information. "anandamide." Progress in Lipids Research. Volume 2022. Article 101194. 9th month, 20th day, 2022.

Hepatoma assay, in a study, observed a 47 percent increased in phosphatidylethanolamine compared to phosphatidylcholine in the inner mitochondrial membrane while a this same ratio increased y 117 percent in the outer mitochondrial membrane.

Information. Oncology Letters. Volume 11. Issue 2. Pages 133 to 139. December, 1980.

Phosphatidylcholine can move from the inner to the outer mitochondrial membrane as well as move from the outer mitochondrial membrane to the inner mitochondrial membrane.

Nat Commun. Volume 10. Number 1432. 2019.

Phosphatidylethanolamine integrates with, and links into the membrane, Calcium transporting transmembrane proteins.

ISBN 978-0-12-800047-2.

Phosphatidylethanolamine is essential to stabilizing Translocase of Outer Membrane enzyme which is essential to its stabilization and when phosphatidylethanolamine is inadequately available, impaired production of mitochondria results through Translocase of Outer Membrane inefficient integration with precursor proteins.

J Biol Chem. Volume 288. Number 23. Pages 16451 to 16459. 6th Month, 2013.

Information. Minerva Gastroenterol Dietol. Volume 57. Number 3. Pages 323 to 331. 9th Month, 2011.

Information. "Methylene Blue." Kotsanis Institute Website. Kotsanisinstitute.com

Glycosylphosphatidylinositols are fusion, linking or integrating phospholipids that that exhibited mannoses that are modified by ethanolamine phosphate such that ethanolamine phosphate is linked by mannose 1, mannose 2 is remove typically when the glycosylphosphatidylinositol integration with ethanolamine phosphate is transient, the enzyme PIGG which transfers ethanolamine phosphate to mannose 2 causes glycosylphosphatidylinositol deficiencies when PIGG is dysfunctional, PIGG dysfunction causes neuronal dysfunction, and preferential mannose 2 ethanolamine phosphate bridges are found in CD59, Ect-5 prime-nucleotidase, and Netrin G2.

Information. EMBO Rep. Volume 23. Number 7. Pages e54352. 7th Month, 4th Day, 2022.

Ethanolamine Plasmalogen decreases with age, decreases correlative to level of dementia and alzheimer's disease, and diminished levels of plasmalogen ethanolamine in every version of dementia as well as level of dementia observed in a particular study. Clearly, the role of phosphatidylethanolamine in increasing methylene bridge cysteine can be clearly linked to PEMT function which produces methylene bridge cysteine as a product used for self-regulation that relies upon recycling pathways and transsulfuration pathways for sustainment of PEMT function by either removing the downregulating methylene bridge cysteine or by translated methylene bridge to methionine and then s-adenosyl methionine. S-adenosyl methionine and methylene bridge cysteine are both substrates for PEMT function. The obscure literature presented in other aspects of the compendium of research linked to this document presents the possibility, from observed fractional studies of traced metabolites in these pathways, that PEMT may utilize a fraction of its own metabolites, substrates and products in its catalytic activity, although the literature does not clearly confirm such dynamics even in the works where such fraction specificity is eluted in analyses. The literature, also, it should be mentioned, does not presence a consensus convention on if the PEMT2 only or most exhibits catalysis at the mitochondrial associated membrane between endoplasmic reticulum and the outer mitochondrial membrane. Certainly, PEMT amplifies biosynthesis by producing methylene bridge cysteine and by upregulating protective mechanisms that enhance or assure biological development and biosynthesis. The packing of hydride into the lead group of phosphatidylethanolamine to produce enriched phosphatidylcholine, resulting in packing of methyl groups into membranes and structure, certainly constitutes methylene bridge diversion and methylene bridge deactivation, such that methyl groups are known to deactivate methylene bridge cysteine along with abated growth of the leading edges of expanding lattices in membranes and structure.

Information. J Lipid Res. Volume 48. Number 11. Pages 2485 to 2498. 11th Month, 2007.

A particular study observes that although Alzheimer's disease exhibits laterality and ipsilateral specificity of plasmalogen phosphatidylethanolamine deficiency, local to each area or lesions which constitute causality for alzheimer's disease, the caudate nucleus was not likewise strongly affected by phosphatidylethanolamine plasmalogen inadequacy for huntington's disease and the substantia nigra was not as likewise strongly deficient of phosphatidylethanolamine plasmalogen in parkinson's disease. Membrane instability resultant of phosphatidylethanolamine plasmalogen inadequacy seems to be an integrally causal factor in alzheimer's disease.

Information. Brain Res. Volume 698. Number 1 and Number 2. Pages 223 to 226. 11th Month, 1995.

Ethanolamine has been found in the center of the milky way galaxy.

Information. "Ethanolamine." Proceedings of the National Academy of Science. 2021. Volume 118. Number 22. Page e2101314118. 5th Month, 24th Day, 2021.

Ethanolamine Plasmalogen decreases tnf-alpha in lower digestive pathway, preventing the nonresolution phase relaxation of tight junction proteins that is linked to leaky gut syndrome and is linked to trimethylamine reduction to trimethylamine-n-oxide which occurs in hepatic tissues after trimethylamine transits tnf-alpha phenotype digestive pathway membranes.

Information. ACS Omega. Volume 6. Number 4. Pages 3140 to 3148. 2021.

Ethanolamine plasmalogen enhances the efficiency and capacity of phagocytosis while inadequacy produces impaired ability to phagocytose experimental particles, linking upregulated proteolysis linked to pervasive disease and aberrant proliferation to inadequate availability of newly obtained phosphatidylethanolamine. Similarly, digestive pathway cellular entity proliferation increases responsively to ethanolamine plasmalogen availability. These suggest that not only does methylene bridge cysteine of different versions exhibit the potential to attach to structure or affect spatial aspects of biology by sequestering away electrons, hydride or eV- in a way that can deteriorate or destabilize the energies that enable quantum an atom level structures to persist, but the experimental literature and data also both suggest that structural aspects of biology can exhibit deterioration if recycling of methylene bridges is not adequately supported by nutritional, supplemental or therapeutic obtainment of carbon, methylene bridges, ethanolamine and methyl groups.

Information. Frontiers in Immunology. Volume 9. 2018.

Ethanolamine changes the characteristics of digestive pathway microflora beneficially to diminish nonresolution cytokine activity. DHA and EPA Ethanolamine Plasmalogen decreases in atherosclerotic lesions by 98 percent and serum LDL-c by 73 percent, while increasing CYP7A1 expression through diminished activation of FXR receptor. Experimental niclosamide ethanolamine, or NEN, which is an anthelmintic therapeutic, instrumentation produces glycemic blood control, uncouples mammalian, mitochondria, prevents steatosis resultant of a high fat diet, all of which represent useful diabetic therapies although uncoupling of mitochondria can result in energy inefficiency and can increase lipid oxidation. Oral nen instrumentation in experimental contexts results increased energy utilization and increases lipid metabolism.

Information. Nat Med. Volume 20. Number 11. Pages 1263 to 1269. November, 2014.

Information. Journal of Functional Foods. Volume 66. Number 3. Article 103824. 3rd Month, 2020.

Information. British Journal of Nutrition. Volume 120. Issue 3. Pages 241 to 249. 8th Month, 14th Day, 2018.

Information. *In Vitro 'Cellular' Dev Biol Anim.* Volume 52. Number 5. Pages 562 to 567. May, 2016.

Carbonaceous meteorites have been found to exhibit amino acids, nucleobases and sugars, such that organic material density seems to be correlated with lower levels of ^{13}C or carbon 13, explaining why carbon 13 levels have been diminished in meteorites exhibiting organic material.

Information. *Science Advances.* Volume 7. Issue 18. 4th month, 28th Day, 2021.

Thus, the list of diagnostic, quantum, molecular, metabolic, genetic, systemic, and environmental factors along with natural, pharmacological and wholistic ways of managing these nuances of factors in Human outcomes with which this analytical artifact is associated are a useful representation of data science. The most incipient findings may represent in nonlinear and disjoint derived curves or graphs. However, integration of each of these curves or graphs, although the data points or scatter graph may be difficult to integrate into one column or shard, produce an increasingly competent or increased uniform curve or Graph. Thus, subsequent findings analyzed with a social constructivist integration on these perspectives can result in derivation of increasingly competent findings and increasingly uniform integrated curves and graphs. The objectives of data Science, thus, seems to be movement toward the most uniform graphs and curves in which the relationships between the variables of a derivative function are increasingly stable and such that highly predictable derivation of the value of any such variable can occur from the ascertainment of the known value or characteristic of any of such variables otherwise.

The culmination of data science objectives seems to be the generation of increasingly linear graphs, curves, training data, tensors, correlates, causal links, Mechanistic links, dualities or inferential correlations, multiplicities, information tuples, shards, data columns, columns, natural language representation of these as stories. The object of data Science also seems to include ascertainment, with increasing certainty, of relationships between variables, with increasing certainty enabling derivation of the values or characteristics of any variable using the values or characteristics any other of these variables or using variables otherwise. Resultantly might be achieved structured logical, mathematical, clinical or other simple or complex analyses, explorative or presumption questions which can be presented as proofs that, also, with increasing certainty confirm such relationships, values and characteristics.

The interactions of analytical processes as observations in polynomial time and the nonpolynomial time interactions which are known to reshape, both, outcomes in antecedent eras and outcomes which are to emerge, to exhibit relationships, values and characteristics increasingly produce by the analytical processes themselves, should be, can be, and optimally should be directed to bend future outcomes and future potentialities to Human benefit in the eras of immediacy and toward increasing benefit of humanity wholistically, extended to include also Humanity in antecedent or future eras, although Humanity, now, obviously performs as a priority pathway for Humanity to be benefited in the future.

Because a most unique benefit provided to the universe is experience or exhibition of livingness among inanimateness otherwise among the known Universes, thus both enable vicarious

exhibition of vital being on behalf of the known Universe, but also potentially being vibrant confirmation through the Human experience that Universe, itself may, too, be living. Data Science, thus continually finds and presents confirmation of Human livingness, information which can be applied to sustain the Human Experience, confirmation to the Universes that through the Human Experience it vicariously or integrally too, lives, while also enabling integrated of Universes level integral share priorities in sustaining Humanity among the Universes.

Thus, an objective derivatization of pathology and disease emerges, because management of methylene bridges is beneficial in discomfort, genetic disease, adverse physiological outcomes, adverse health events and adverse behavior. Pathology seems to be the systematic deterioration of packing of chemical, molecular, hydridic and constitutive current into phospholipid lead groups that occurs because of anaphylactic response. The anaphylactic response occurs resultant of inadequate obtainment of ethanolamine, overuse of recycled methylene bridges, methylene bridge cysteine attachment to, destabilization of, or angular influence to structure of quantum and structural aspects of Atoms, redirection of ethanolamine toward autophagy instead of to phosphatidylethanolamine as substrate for PEMT, redirection of phosphatidylethanolamine toward autophagy but exhibition of Bag1 prevalently compared to Bag3 to result instead in upregulated proteolysis, resistant phenotypes that prevent autophagy from clearing dysregulate proteins and allergens or nonresolution cytokines, upregulation of the cdp-choline pathway both resultant of inhibition of PEMT and resultant of environmental/electromagnetic/atmospheric/xenobiotic/artificial and other pollution/particulate including cytokines that inhibit PEMT such as cytokines invoked by pathogens or particulate, commandeering of methylene cysteine bridges by pathology promoting factors and upregulation of phosphocholine as a source of energy for managing anaphylactic activators, redirection of ethanolamines including ethanolaminephosphate/cdp-ethanolamine/phosphatidylethanolamine/PMME/PDME toward antihistamine activity, diminished storage of Hydride and methylene bridges as phosphatidylcholine because of diminished PEMT activity resulting in structural depletion that causes cellular entities to exhibit unfolded protein response along with upregulated proteolysis and cdp-choline pathway that reprograms cellular entities toward upregulated BCL2/Survivin/GPCR/S1P/S1PR/GSK3B signaling that hastens and extends differentiation, redirection of s-adenosyl methionine toward diverse methyltransferase that attach methyl groups to allergens/toxins/hormones/cytokines/xenobiotics/particulate causing increasing levels of methylene bridge cysteine while also depriving PEMT of substrate, genetic conditions which pervasively produce methylene bridge cysteine and cause nonresolution cytokines that affect PEMT, nonresolution phase transcription of genome that results from all of these factors to deprive typical and optimal transcription from occurring, deficiency in aminos/nutrients/phytonutrients/enzymes/substrate/products that would supplementally alleviate genetic and epigenic inadequacy.

Disruption of PEMT activity also changes or diminishes the supply of phospholipids enriched in DHA, EPA, Oleoylate, Palmitate first fatty acid in fatty acid beta oxidation, extended length arachidonic acid, omega-3, diversity and ether linked for membrane insulation, both through

diminished PEMT and exacerbated by inadequate nutritional obtainment of such factors. The diminished production of enriched phosphatidylcholine through diminished PEMT activity causes diminished ability of lipid cycle resolution phase phospholipases and phosphodiesterases to free phosphatidylethanolamine plasmalogen, phosphatidylcholine plasmalogen, phospholipid plasmalogen otherwise for mixing and shuffling, followed by reattachment to phospholipids or reattachment to lysophosphatidylcholine by LPCAT, MBOAT and other fatty acyl transferases. The synthesis of a diverse group of resolution phase factors and resolution phase cytokines occurs from freed membrane fatty acids using both resolution phase and nonresolution phase phospholipase and phosphodiesterase including resolvins, neuroprostanes and others. Methylene bridge cysteine also disrupts systemic molecular, cytokine, chemokine and other signal transduction, including causing disrupted neuronal signaling or changed activation of neuronal receptors, including changing of import agrin signaling in diverse areas including bone structure and extracellular matrix. These factors promote accumulation of methylene cysteine bridges and redirection of encapsulated methylene bridges toward nonresolution cytokine signaling which disrupts lipid chemistry and phase progression of lipids and phospholipids that produces microstructure, intermediate structure and super structure, all of which are involved in producing hydridic, angular, spatial, ionic, quantum and atom levels influences that are essential to typical or optimal physiological, metabolic, molecular and systemic function.

Methylene bridge cysteine also causes promotion of BAX, BAK, cytochrome c release and other factors that increasingly potentiate programmed cellular deterioration, particularly through the action of P53 that is upregulated when diminished PEMT function occurs. Resultantly of methylene bridge cysteine upregulated availability is a, likewise, upregulation of BCL2, choline kinase alpha, CDP-choline pathway, proteolysis, GCPR, and other survival signaling and differentiation pathways, all in a way that directs adaptive, complements, cellular, humoral and other aspects of highly conserved immunology becoming directed toward prevention cellular deterioration and tissue deterioration, including NOS2 which is required to enhance the turgor or strength of individual cellular entities to counteract depletion of the number of cellular entities per micrometer as well as counteract the increased percentage of cellular entities which become paused in hypertrophic enlarged phases that is also an adaption to cellular entity density depletion. Enlarged or hypertrophic phases can fill up space produced by massive cellular entity deterioration in a way that is comparative optimal tissues which exhibit increased number of cellular entities per micrometer along with decreased diameter of each cellular entity.

Among original inhibitors of choline kinase include adenosine, pregnenolone and the 1940s and 1950s discovery of purinyl-L-histamine which has escaped, along with dimethylthetin first characterized in 1878 at the latest and along with glycollate first described in the 1840s at the latest, application to cause what could have been a remarkable improvement of outcomes at massive population levels.

These describe why antihistamine therapy derived in the 1910s became diminished as a focus in therapy in correlation to emerging of diverse sources of industrial, atmospheric, electromagnetic, audio, magnetic and other pollution/particulate, such that cumulatively the factors presented here cause dysregulation of methylene bridges and methylene bridge cysteine in a way that promotes

existential challenges among cellular entities individual and among tissues bases. Managing one factor among these, as an example, merely allow the other factors emerge or continue to downregulate PEMT, dysregulate methylene bridge cysteine, deplete ethanolamines, deteriorate atomic structure, deteriorate energies that stabilize subatomic or disrupt quantum/angular/spatial level characteristics require for the spooky special effects typical of biological function, and deteriorate of essential aspects of prolonged biological and physiological function, particularly aspects of sustainability essential in shaping emergence, phases, and outcomes regarding health conditions, pathology and aging. Importantly, therapies pervasively, but not comprehensively, increase methylene bridge cysteine, increase cytokines that inhibit PEMT or compete with PEMT for methyl groups at cytochrome, COMT, s-adenosyl methionine, and PEMT catalysis. The data, to this instance, suggests an essential inclusion of wholistic factors influences, conditions, and status that stack up on, dysregulated, exacerbate, change, or improve methylene bridge and methylene bridge cysteine. Wholism, including the way in which natural systems, artificial systems and civilization's systems affect methylene bridge availability and methylene bridge cysteine availability. Methylene bridge availability, regulated availability of methylene bridge cysteine and regulation of methylene bridge cysteine levels, all emerge as convergent single focus loci to which all layers of natural, artificial and civilizations' can be diagnostically, causally, therapeutically and behaviorally correlated.

These factors are useful in understanding how a continuous exhibition of cellular survival status or existential challenge results in eventual deterioration of mitochondrial potentials, separation of the typical hundreds of mitochondria from attachment to the endoplasmic reticulum through the mitochondrial associated membrane where PEMT2 may be transmembrane protein between these through the mitochondrial associated membrane or where PEMT2 may be integrated into either of these outer membranes, or where PEMT2 may be in other areas of mitochondrial membranes. The literature suggests that PEMT2 may be an integral mitochondrial associated membrane and the literature observes that supply of phosphatidylserine, Ca^{2+} , Phosphatidylethanolamine, phosphatidylinositol or other essential factors occurs, in particular, from the endoplasmic reticulum, through the mitochondrial associated membrane and to mitochondria. NOS2 expression, impaired PEMT function, particularly impaired PEMT2 function, trimethylamine-n-oxide, and upregulated methylene bridge cysteine, all are integral to adverse physiological, cognitive, neuronal and behavioral outcomes. However, it is the continuous expression of the transcriptional activator AP1 which would ordinarily deplete its own cytokine resources and downregulate telomerase TERT function to result in increasingly limited division or proliferation of cellular entities affected by disease or nonresolution cytokine proliferation through fusion of chromosomes and exhibition of senescence. It is the specificity protein transaction factor SP1, which is included g quadruplexes within telomeric regions that is upregulated by destabilization of g quadruplexes which enables access to SP1 and other cytokines by transcription polymerases, along with SP1 downregulation of Telomerase to prevent telomeric attrition that would clean G quadruplexes from telomeric regions, which counteracts or surmounts the way in which AP1 prevents extended division or mitosis among lineages of diseased cellular entities from occurring. SP1 copy number increase because of SP1 in telomeric regions and g quadruplexes, along with destabilization of g quadruplexes to allow transcription polymerase access to what can be increasing number of quadruplexes that may

harbor SP1 or other cytokines. SP1 and AP1, both inhibitors of PEMT, both upregulate the CDP-Choline Pathway, both being opposite regulators of telomerase, each have roles in typical and optimal physiology as well as may be commandeered by pathology and may be commandeered by pathology causing vectors.

Although AP1 enhances nonresolution cytokine signaling is constitutive of nonresolution cytokine signaling, it inherently limits cellular entities affected by or emitting nonresolution signaling, suggesting that it may be an effector of senescence, at least among diseased or impaired cellular entities, such that SP1 upregulation diminishes this ability to limit disease progression that eventually can result in intricate and comprehensive impairment of PEMT2 that is link to disease. The factors presented here are all able to be prevent, alleviated or have their detrimental effects removed. Inhibition of AP1, as an example, causes disease phenotypes at the cellular level to be become removed and cellular entities then can come to exhibit near embryonic, tissue competent phenotypes.

Diseases are pervasively or inclusively the cumulative hyperactivation of anaphylaxes pathways by multiple cumulative factors to cause hydride/e- sequestered by methylene bridges into molecular, chemical, quantum, angular, polymerization potential, and into membrane structure as enriched omega-3 EPA/DHA diverse resolution-phase-enhancing ether linked phosphatidylcholine all to become changed. Dysregulation of methylene bridge cysteine emerges to above 6 or 7 $\mu\text{m/L}$, causing aberrant polymerization, differentiation and development, while also making these available for pathology promoting processes to disrupt signaling, quantum/atom/particle level electromagnetic/angular influence, spatial aspects of physiology interactions and enable sequestration of energy from all manner of biologically active molecules or structure, and disrupt spatial aspects of biology, . Methylene bridge translation of current, eV- , e- or hydride into molecular, metabolic, chemical, polymerization and structure as de novo synthesized phosphatidylcholine by PEMT, all become changed through particularly diminished catalysis by PEMT which causes upregulation of anaphylaxes pathways and is complicated by trimethylamine-n-oxide. Thus, diseases result in deterioration of cellular membranes and tissue, existentially, resultant in survival focused changes to cellular membranes, tissues and physiology, resulting in cytokine expression which counteracts massive apoptosis among cellular entities, decrease of cellular entity density per micrometer, decreased availability of membrane phospholipids to abate growth of membrane structural leaflets, and decreased diversity of fatty acids for shuffling in the Lands cycle by LPCAT/MBOAT and other fatty acyltransferases. Because natural, artificial, endogenous, exogenous, xenobiotics, particulate and even therapeutics all contribute to inhibition of PEMT and competition for PEMT substrate, the result is an cumulative and cooperative deteriorate of enriched phosphatidylcholine, upregulation cdp-choline pathway, upregulation of nonresolution cytokine, increase in feed forward supply of substrate for nonresolution cytokines, deterioration of cellular membranes, deterioration of tissue, and impaired capture of freed eV- to impair cognition and consciousness, as well as phased increased diminished of factors contriving angular/quantum/atom/spatial/spooky and hydridic effects essential to maintain ph between near 7.2 and 7.6 which enable consciousness, cognition and biological stability. These are all aspects of anaphylaxes pathways.

CYP3A4, among other cytochromes and cytochrome P450 isozymes, are known to process a diverse array of therapeutics and pharmacological agents into derivatives or metabolites. Processing pathways can involve obtainment of an inactive version of a therapeutic, such as a methylated version, followed by processing by CYP3A4 to produce a demethylated or desmethyl version, constituting an activating change, producing an active therapeutic. The desmethylated version, thus, also, must be detoxified or cleared from physiology through processing typically and somewhat pervasively involving attachment of a methyl group to cause entry into methylation pathways. Particularly, COMT which competes with PEMT, as well as a large group of other transmethylases which also compete with PEMT for s-adenosyl methionine as substructure, are involved in processing of therapeutics.

cMAX is an indicator or statistics regarding the levels, density, or availability which one dose of a therapeutic achieve in the physiological environment before diminishing without being followed with another dosage. Interesting in this regard is the observation that grapefruit and grapeseed extract and green tea extract are known to substantially diminish both imanitib and its activated desmethyl metabolite desmethyl imanitib, resulting in enhanced clearance that is correlated to diminished therapeutic exposure to tissues to which imanitib is applied. Another study observes that imanitib can cause aberrant methylation by promoting the release of methyl groups through cytochrome CYP3A4, while imanitib is experimentally susceptible to increased methylation of DNA at cpg islands which promotes resistance to imanitib. Uniprot presents catalysis of CYP3A4 as being an organic molecule or R group, and a reduced redox factor such as NADPH hemoprotein reductase being translated into an alcohol, molecule of H^+ , a molecule of H_2O and an oxidized NADPH Hemoprotein reductase. These present an interestingly observable perspective of how current is utilized in physiological processes, and presents how the leaving of a methyl group from a molecule causes the molecule to become an active molecule with methylene bridges that begin to interact more directly and more completely with redox transactions constituting movement of hydride, electrons of eV^- , although the release of CH_3 in the activation of imanitib results in synthesis of a lactam which is among the foundational categories of derivatives first produced from thioglycolic acid in the first several decades of the 1900s regarded as being centers of medicinal chemistry. Uniprot presents CYP3A4 catalysis as a unidirectional transaction.

Grapefruit, as another example, can increase the AUC, area under the curve, which is an observation of the bioavailability of therapeutic over a duration to ascertain level of bioactivity of a such therapeutic at thresholds of availability indicative of such bioactive activity, particularly increasing such bioavailability for felodipine although grapefruit is a wellknown to change bioavailability for therapeutics. Although grapeseed extract and green tea extracts are known to change CYP3A4 catalysis, there may be multiple mechanisms involved, such as phosphatidylcholine being known to enhance transit of digestive pathway membranes by nutritional and therapeutic factors and such as similar enhanced absorption produced by piperazine. This present accompaniment of natural or modulative therapeutics as way of changing the availability, effect and duration of effect of pharmacological factors or therapeutics otherwise for consideration in therapy. However, these analysis are included in a compendium research which suggests utilization computational proteomics that include providing therapeutics

that have been locked into inactive status followed by providing separate proteomics that modulate release of the therapeutic payload presented in the therapeutic proteomic molecules. Strategies include providing an activating therapeutic vector that can be monitored to determine its activation graph or its affect to the therapeutic molecules, while also an inactivating vector could be provided that can also be utilize diminish the effect of a therapeutic molecule, thus enable are upregulators, downregulators and abrupt abatement capabilities for any therapeutic molecules provided to affect physiology, biology and behavior. Similarly presented in such compendium of research associated with this analysis is the ability to have such modulative vectors assay each cellular entity individually for availability, for exhibition of particular characteristics, exhibition of genetic or epigenetic characteristics, pathology proteins or conditions, thermodynamic characteristics, or other factors, followed by activating or deactivating a therapeutic molecule. Most interesting, also, is the potential of having any therapeutic molecule not only having different molecular sequences which can be released independently of one another, but also having having each molecular therapeutic payload sequence release an activation sequence that automatically activates another molecular therapeutic sequence payload, resulting in walking an individual cellular entity, tissue base, or physiology through a rehabilitative process that impose a development, therapeutic or other complex procedural outcome. It is also to have each released therapeutic payload sequence merely release another computational assay domain that assays for how effective therapeutics have been or the current status of such process, followed by enhancing the availability of therapeutic payload to move the rehabilitative process forward, followed transition of the therapeutic focus forward for each phase of a therapeutic process. Such assay, response, control loops may be performed using a therapeutics followed by assay, followed by instrumentation of additional therapeutic levels until subsequent phases have been achieved, then, again, followed by instrumentation of therapy to traverse the next phase of a therapeutic process. Likewise, one therapeutic with multiple payloads sequence for one therapeutic rehabilitative process or one therapeutic with multiple payload sequences for multiple disease, conditions or statusus, all may be controlled by instrumentation or remote control of which therapeutic payload is release, when it is release and what level of such therapeutics payload is release. Remote activation factors might include energy, infrared or ultraviolet light, different frequencies and wavelengths, sound, thermodynamics, or conditions that can be changed such as postprandial status, activity level, or other interactivity.

Importantly, these represent a way to impose a ceiling and a floor for the effects of therapeutics as well as enable an emergency abatement mechanism for therapeutics to enhance quality of outcomes. Importantly, protein transduction therapy already has enabled very large 250 dalton or more biologically activity therapeutics to transit every cellular entity in physiology with the efficiency, or near efficiency of a water molecule. These present an opportunity to produce a holographic platform that transcends physiology encompassingly, resulting in promoting, escalating, or enabling transparent observability, as well as modulation of any physiological factor or mechanism, pathology, pathology vector, or atypical developmental nuances, followed by implementing a therapeutic or rehabilitative process that is personalized down to individual molecules in physiology, individual cellular entities, structures, organs, tissues and individuals.

These can even be instrument, monitored, reported, and interactively managed by automated capabilities and technologies.

Although the changes possible to therapeutics manufacturing include plug and play therapeutic modules and processes that personalized all the way to the protein level, such that focus on safety, instrumentation, and control can proceed as priorities in separate focus areas, these enable enhanced coverage of the different contexts of disease, injury, impairment and developmental impairment which have escaped economic justification, although there are now organizations focused on developing therapeutics for conditions that are unlikely to provide adequate return on investment and net present value typically, otherwise, included industry development and manufacturing decisions.

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The implications of methylene bridge sequestration of current may seem controversial. However, the exhibition of methylene bridge sequestration of current is plainly observable in CH₃ which constitutively integrates e⁻ as the extra e⁻ in in Hydride, H(2e⁻) (1p). However, although photons are known to cause emission of electrons when encountering matter, the literature does plainly differentiate such emission spectra from being caused by displacement of electrons by photons. The potentials of methylene bridge sequestration of current are clearly observable as polymerization potential and are clearly observable in methylene bridge cysteine once CH₃ has been removed, because this results in one of the most biologically destabilizing factor known to exist as an endogenously produced metabolite. Importantly, a particular research article analyzes the palladium complexes with methylene bridge diphosphines which is interesting for multiple reasons. Palladium is known to be among the most potent storage capabilities for energy, electrons or hydride, because its lattice expose a series of electron holes that extend deploy to its structure, resulting in remarkable disproportionate ability to receive and store energy compared to it structural density or size. The diphosphine's phosphines are joined to palladium by methylene bridges, in the study.

Phosphines are known to occur in space. Diphosphines have been ascertain in interstellar space. CH₂ has been ascertained in interstellar space. Diphosphines joined by CH₂ in interstellar space clearly suggests sequestration of e⁻ in space without constitutive integration of hydride that includes its constitutive integration of additional e⁻, such that direct integration of e⁻ from interstellar space, absent of matter as vacuums may be, implicating that light may be integrated

from environment by methylene bridges when the encompassing aether is not constitutive of matter. Correlatively, the integration of e^- or eV^- is known to occur through RET in physiology in which e^- or eV^- is integrated to reconstitute NADH from NAD^+ . Most importantly, the compendium of research associated with this analysis clearly found experimental production of electrons from hydridic fields, photons and freed eV^- or current within a context of qubit function in quantum computing architecture and design. These suggest that light or photons have energy and that energy may possibly be translated by CH_2 or methylene bridges into polymerization potential, although a correlative and more simple explanation might be constitutive of or include enhanced energy of existing electrons produce by photons. Regardless, the translation of photons into structure is very likely represented by the function of methylene bridges which can, minimally, translate the energy conferred by photons into structural or polymerization potential.

Likewise, the literature observes phosphoric acid, phosphonic acid and pyrophosphoric acids as occurring in interstellar space with structure exhibiting that in which hydride has been integrated between the phosphate groups as oxonium. The popular literature observes oxygenated acids or oxyacids or oxoacids, all as having and acidic hydrogen integrated into an oxygen atom with the length or strength characteristics of this linkage being mitigate because such molecules include nonmetal hydrides. These suggest, again, that hydride is being integrated into the structure phosphate complexes in interstellar space.

Thus, where there is no matter, methylene bridges sequester e^- . In physiology, freeing of e^- results in excitation of environment and exhibition of fluorescent influence, all of which enable synthesis of ATP and integration of hydride between the phosphate groups of ATP as oxonium. Thus, derived from aether of the universes, excitation, eV^- , current, electrons or e^- integrated into hydrogen to produce hydride, are able to be sequestered by CH_2 and these can be translated into structure or polymerization potential or integrated into polymers.

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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 CH_3 , 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 CH_3 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.

Antihistamines ethanolamine, Phosphoethanolamine, CDP Ethanolamine, phosphatidylethanolamine, PMME, PDME and Phosphatidylcholine are each included in the literature as being relevant to the management and clean up of industrial wastes, while each perform as inorganic to organic phase transfer agents, deteriorate pervasive carcinogens at structural levels, and perform in myriad capacities that sequester a space or place in the biome for biology and human development to emerge and persist. Although catalytic relevancy is not excluded in this observation, hydridic character may be among the causes of effectiveness of such molecules. Thus, this compendium of research explores the nature of the detrimental effect of homocysteine and this may be relevant to understanding how the effectiveness of the antihistamines emerges. This specific context suggest that homocysteine focuses on accessing

deactivating the hydridic centers of biologically active molecules, affecting the shape, twist and writhe of biological molecules which, then, impairs the ability of molecules to respond, move, twist, turn, reshape themselves and exhibit other activity that occurs in three dimensional aspects of biological activity. Homocysteine may also fit or integrate, as well a catalytically interact with loci and molecules that methionine and s adenosyl methionine interact with, obscuring, deactivating or even activating such molecules with disparate results from what biology requires.

There is also the possibility that homocysteine performs and signals exhibition of a biological resection cascade that may be intended to be ephemeral, enabling resection of tissue, followed by increased levels of homocysteine performing as substrate for biosynthetic and regenerative biosynthesis. Homocysteine, correlatively is utilized by a numerous biosynthetic pathways, particularly by being recycled into methionine, explaining why methionine is required in the transcription of 99.5 percent gene translation products through each essential role as priming molecule for T RNA translation at ribosomal molecular machines.

PMME and PDME both promote inorganic to organic phase transfer, enable serine protease function, perform as antihistamines, synthesis the exceptional fibrinolytic known as tissue plasminogen activator, and assist by promoting embryonic plasticity that includes environment cleaning. PEMT synthesis phosphatidylcholine with enriched fatty acid species that include increased diversity, docosahexaenoic acid, extended length arachidonic acid, oleoylate, palmitate first fatty acid in diverse fatty acid synthesis pathways known as fatty acid beta oxidation, ether linked fatty acids that enhance insulation of cellular membranes, as well as omega 3 fatty acids. These factors are diversified in cellular membranes phospholipids by phospholipases and diesterases, but particularly by noninflammatory phospholipases such as phospholipase A2 and noninflammatory calcium independent phospholipases that free fatty acids from membrane phospholipases followed by reintegration of fatty acids into phospholipids such as LPCAT/MBOAT acyl transferases that produced phosphatidylcholine from fatty acids and Lysophosphatidylcholine, which comprise aspects of the lands cycle.

These group of factors even frack, reduce, methylate, sulphonate or otherwise deteriorate pervasive carcinogens in nature, and obviously include deterioration of many synthesized industrial wastes.

Such fatty acid diversity ablates 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 lipoxxygenases. 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.

Thus, hydride as negatively polarized hydridic center or character in molecules produces a background pH near between 7.2 and to 7.6 which contrasts with the strong ions calculated in acute medicine. This results in a gradient upon which biological activity occurs in numerous redox pathways but particularly involving NAD^+/NADH , $\text{NADP}^+/\text{NADPH}$ or between the background pH range and circulating or otherwise exhibited H^+ . pH is presented with a definition of “potential of hydrogen.” Thus NAD^+ exhibits hydride the energy that fuels stars, and precursors of nucleotides, DNA, RNA, as well as phosphinic acid, ethanolamines, CH_3 and other factors are found in interstellar space.

Near 7.2 to 7.6 range of background pH is typically regarded as the range essential for biological function, consciousness and conscious cognitive function. Hydride in NADH , NADPH and other redox factors are oxidized in metabolic redox catalytic activity to release hydride which can be accompanied by release of H^+ in some instances. However, the central pathway of energy synthesis in physiology is comprised the electron transport pathway or oxidative phosphorylation which frees hydride from NADH and integrates 42 percent of the energy between the phosphate groups of ATP synthesized by such process, while about 58 percent of such energy is utilized by such process which utilizes load balanced democratized process to efficiently apply the 58 percent of energy to the storage of 42 percent within ATP.

The freeing of Hydride results in release of fluorescent energy as free energy that activates, influences, is observed by, causes spin within or otherwise effect ambient material, particles and factors, although the process can be inverted to produce NADH using energy in molecular and metabolic pathways. Cellular membranes provide insulation and produce capacitors of cellular entities and produce capacitance or potentials that are changed upon activation of neurological receptors as well as which participate in the hydridic effect that produce background physiological pH near between 7.2 and 7.6. Hydride can be mined, fracked and released to produce chemical energy, free energy, capacitance and potentials. Pyruvate, ATP, and Glucose contribute to, can be derived from and includes exhibition of Hydride. A study of popular sweeteners produced by research linked to this analysis clearly presented a correlation of sweetness with hydride and hydrogen density, although the efficiency of integrating these into structure seemed to be diminished by inhibition of glut produced in choline deficiency as well as trapping of glucose in glycogen cycling during choline deficiency, while empirical metabolic pathways suggested that glucose might otherwise be directed into biosynthetic pathways with glycine as a substantial output of processing instead of being retained as glycogen.

Subcellular compartments can compete for capacitant influence or potentials with Mitochondria, Endoplasmic reticulum and Nucleus being major competitors, although PARP signaling can sequester enough NAD^+ to change capacitant field balances, although the hundreds of mitochondria in the typical cellular entity and the attachment of these hundreds of mitochondria to the endoplasmic reticulum to result in PEMT production of enriched, CH_3 dense, phosphatidylcholine changes these in favor of the Mitochondria, particularly changing these to the favor of the Mitochondrial Associated Membrane that links these numerous mitochondria to the endoplasmic reticulum.

PEMT enables and leads to synthesis of oxytocin, a molecule integrally involved in the development of Human relationships and emotional linkages essential in the emergence of relationships, familial linkages, groups, communities, and civilizations. The diminishing of PEMT and homocysteine levels, both, explain changes to reproductive health and decisions that are involved in whether or not a gestationally developing Human is allowed to be successful in completion of the gestational status. Oxytocin is a neuropeptide, derived from the hypothalamus, and is an integral factor in relationships, reproduction, birthing processes as well as interactions and statuses of those involved reproduction after childbirth. Its competition with synthetic peptides, social processes, and cognitive influences, may integral to outcomes and risk involved in reproduction. An imperative observation in this context is that vast aspects of synthetic therapeutics and environmental particulate, as well as electromagnetic influence increase levels of homocysteine and result in inhibition of PEMT, thereby, also, affecting synthesis of oxytocin.

There is clear correlation between age, development and levels of Homocysteine. The inference that homocysteine itself may be a factor in emerging Human development may be observationally accurate, although its causal participation in Human development may be less resultant of level of homocysteine than it may the result increase nutritional obtainment of xenobiotics, exposure to environmental particular and the increased volume of the growing physiological system. Certainly, homocysteine's contribution to methionine and biosynthetic processes are reasonable correlated with integration of the xenobiotic management system which produces Homocysteine along with a result potential upregulation of PEMT and other biosynthetic pathways that may utilize homocysteine. There does not seem to be many studies that are observationally relevant in this regard, although this integrated systems of toxicity management supplying biosynthesis with substrate seem reasonable and in many ways is more simplistic and directly competent to density of cellular entities per micrometer as well s density of cholesterol in cellular entities.

A review of the literature observes that maternal carriers have lower homocysteine than females otherwise, generally. Also, there are clear correlations between elevated Homocysteine and decreasingly optimal gestational status as well as decreasingly optimal gestational outcomes. However, homocysteine increases in the second and third trimester. However, the compendium of research with which this analysis is associated, clearly observes distinct changes occurring in trimethylglycine and other biosynthetic metabolites that are center upon density of cellular entities per micrometer and foundational levels cellular membrane structure and metabolism. These metabolites, interestingly, are integral to the existential characteristics of tissue, glands, organs, neurological centers, integral with cellular metabolism and number of cellular entities per micrometer of biological structure, but also includes levels of homocysteine, correlation to optimal characteristics hematopoietic fluid or blood, status of the brains loci for rewards systems and control function, as well as, obviously, perception, cognition, behavior, and health status. Correlatively, the analytical data and these analyses suggest that when these become increasingly less than optimal, the locus of health and behavior determining factors move in two directions, inward to molecular and metabolic systems as oscillating mechanisms, along with outward migration in which systems, influences and statuses in the environment perform as oscillating mechanisms that interact with these emerged internal molecular and metabolic oscillating

mechanisms. Perhaps most if not every metabolic, chemical, emotional, molecular, perceptive, cognitive or other aspect of reproductive behavior may be being shadowed, modulated or changed by such factors. Hardly any of these affective factors are required to understand their effects. Information. "Review." Biomed Res Int. Volume 2021. Article 6652231. 2021.

These clearly explain how homocysteine results subjective elevation of influences of civilization in which humans have inclination, potential behavior, behavior and physiological status that are anathema to their own interests, anathema to Human priority, and may be pervasively constitute of less than competent influence to their own outcomes. Certainly, plants that produce financial currency in nature do not exist, although humans are required to respond to metabolic and molecular changes with an empirical and incipient correlation or association with currency resulting in insertion of all manner social, political, economic and other systems of civilization in the Human/behavioral/physiological/environment synapse. These are factors integrally affecting, also, decisions regarding reproduction. There may such diverse social constructs, decisions, financial, economic, socioeconomic, cultural, and other factors of influence that this splinter into diverse shaping influences which nature has not ever intended to be included in or affect reproduction.

Although NAD⁺ is linked to electron transport and DNA structure as well as DNA transcription, NADP⁺ is linked to biosynthesis, protection and maintenance, according to the some of the literature although are interactions between NAD(H), NADP(H) pathways. One study observes 20 mg of niacin as a foundational daily requirement for NAD⁺ levels. NAD⁺/NADH and NADP⁺/NADPH levels are each regarded as balances to observed therapeutically. A complete complement of B vitamins including methylcobalamin version of B12 among others, are considered essential daily requirements, along with choline, 6s 5678 methyltetrahydrofolate, phosphatidylcholine, a complete vitamin, and between 7 to 4 mg per kg of anatomical mass each day of choline or phosphatidylcholine. Sulphones such methsylsulfonylmethane, trimethylglycine, and s methylmethionine sulfonium are useful requirements for daily supplementation.

Ancient pink Himalayan Sea Salt is recommended because it diminishes vascular striates, has a most diverse group of minerals although these may sometimes be at minuscule supplies a cofactor for the primary choline transporters which are sodium coupled, and prevents the upregulation of VLDL in PEMT pathways required to coat vasculature to help repair vasculature.

Choline supplementation must include uncooked, insubstantially processed choline and foods from which choline is derived should be warm, all to prevent impaired absorption and utilization of choline, phosphatidylcholine or cholesterol in physiology.

Importantly, and confirming, diminished production of phosphatidylcholine, particularly including diminished de novo synthesis of choline as phosphatidylcholine by PEMT, results in impairment of bulk lipid integration into VLDL. The context was observed in small nonhuman mammalian experimental organism hepatocytes.

These, then lead to consideration of laminins that are transcriptionally produce and then modified post translationally to assist in rigidity of subcellular compartments, extracellular matrix and

connective tissue, as well as participate in synthesis or comprise subcellular compartment structural components along with cholesterol which comprises, according to some of the clinical information, up to 87 percent of cellular membranes. Laminins also are used to comprise extracellular matrix and can be used in synthesis of connective tissue. High molecular mass Hyaluronic acid is also optimal compared to overprocessed, nonresolution phase low molecular mass hyaluronic acid. Hyaluronic acid sequesters water through absorption and impedes infiltration of tissues by inflammatory monocytes and leukocytes. Post translational modifications of Laminin determines if it is unchanged or promoted to the plasma membrane or promoted for translocation to the nucleus.

However, Agrin has emerged as galvanizer of pioneering development, aggregation of acetylcholine receptors used in innervation of tissues in a gender modulated way, as well as is essential in regenerative repair along with particular metalloproteinases, C3 complements activation, and PEMT function. Agrin is known to emerge at conception in a way in which its participation with hydridic or other fields is observed to occur in coordination with areas of emerging, growing and developing gestational Human beginning with conception. Agrin also is involved with managing monocyte, hematopoietic or blood, circulating and tissue resident stem cellular entities, particularly in marrow. The canonical function of Agrin is its integration into extracellular matrix where it performs as signal conducting sensory mechanism for extracellular matrix flexibility, stimulating synthesis of more matrix or less matrix in a way that maintains extracellular matrix and connective tissue.

Agrin can inserted into Cardiac tissue where it stimulates regeneration of Cardiac tissue, although homocysteine should be managed also in this context. The literature observes decellularization of cardiac organs and pulmonary organs, followed by reseeded these with stem cellular entities produces regeneration of organs and establishment of spontaneous Cardiac Rhythms and Spontaneous Pulmonary Rhythms ex vivo without requirement of anatomical compartmentalization, illustrating the importance of managing inflammatory cytokines which prevent or delay regenerative repair.

Agrin integrates with the HIIPPO pathway, YAP/TAZ, LAT1/LAT2 to manage mitogenic potential, which includes acquiescence to or surmounting of confluence. Confluence is the inherent signaling pathways that stimulate cellular entities to proliferation until they are encompassed by other cellular entities or until they interact with extracellular matrix or connective tissue otherwise. Agrin and the HIPPO pathway as well as YAP/TAZ, LAT1/LAT2 all participate in receiving, emitting or fielding mitogenic signals that determine if confluence fundamental foundational signaling pathways are surmounted or are acquiesced to.

However, it is the hydration shell that encompasses particles, atoms, molecules and enzymes that must be considered, also, as foundational factors in development and homeostasis. Hydration characteristics at the molecular interface with biological fluids might be regarded as an integral aspect of the potential of hydration because involve Hydrogenic interactions, the hydration shell encompasses all particular, molecules and enzymes, regulates internal molecular dynamics, external molecular dynamics, and the characteristics of intermolecular interactions.

This is an efficient, noncomprehensive distillation, representing focused presentation of wholistically and pharmacologically manageable platform, with obviously promoted interactive interfaces.

Homocysteine emerges as the central indicator of systemic efficiency, sustainability and homeostasis when optimal and increasingly potentiates inverse outcomes in correlation to its exhibition at non optimal levels.

Homocysteine is, thus, an indicator not only of cellular structure and metabolism status, but also bridges to the genomic, metabolic, tissue, structural and organ, as well as systemic, cognition and behavioral levels. Thus, it is indicator of density of cellular entities per micrometer among these other factors.

However, homocysteine has been known of since 1810 and has been able to be managed at 700 times the therapeutic benefit of hardly any factor accepted and utilized in care, even in modernity, although this is rapidly changing as care entities and care providers become aware of this platform, its clinical API and its stack of modulation capabilities.

Homocysteine causes deterioration of areas of the brain required for social behavior, rewards system function of known appropriate boundaries for behavior and attachment of social norms to behavior. Homocysteine causes impaired conditioning, impaired blocking within condition, deterioration of recall in which condition in one context can be conjured within other contexts, as well as causes a diminished ability to withstand external shaping influences and externally impose stimuli/response pairings. Most importantly, when including electromagnetic fields, inherent capacitant fields emanated from mitochondria, nucleus and endoplasmic reticulum as well as emanated from neurological system becomes crowded out, displaced and sometimes replaced, not only by stimuli, conditioning and deteriorated environment or social conditions, but directly by electromagnetic fields themselves. These explain why particular zip codes have the most diminished social, behavioral and physiological outcomes, particularly when including environmental particulate.

The literature is reluctant to indicate that electromagnetic fields from electricity infrastructure and wiring, or from wireless technologies are singularly causal of disease, although this is changing mostly because the inflammation pathways invoked by such artificial electromagnetic fields participate in inhibiting PEMT and thus move aggregate PEMT inhibition as well as aggregate homocysteine toward thresholds causal of disease, enabling of disease or impair pathways that would prevent disease. The literature regards this multiple causal factor causing of disease as multiple factor theory and has been controversial because until recent aspects of modernity, multiple factors participating in causing any one disease was considered to be only theory. This model of biological systems and susceptibility for pathology clearly presents that multiple factor theory is the canonical context for exhibition of disease, since incipient susceptibility, existential challenge to cellular entities, cellular membranes and tissue exhibition of cellular entities per micrometer, are substantially or even pervasively essential to diminished Human outcomes. The major pathways for electromagnetic energy exposure known in general and consensus context, although there are likely to be more intricate contexts and factors which

are to emerge and which may be less widely known, include NOS2 inducible, phospholipase D, phospholipase C gamma, phosphatidylcholine specific phospholipase, and 2 palmitoyl phosphatidylcholine surfactant specific phospholipase, both as phospholipases and phosphodiesterases. Luminal expression of these are particularly detrimental and describe why diseases in areas with comparatively increased levels of electromagnetic field exposure are correlated with more substantial epidemiological conformation of particular diseases as well as more substantial advancement of particular disease

Regardless of the role which electromagnetic fields from infrastructure, wiring, electronics, devices, and other sources might have in disease and diminished human behavior, it is certain that shielding massive levels of electromagnetic energy to which humans are being exposed may produce remarkable changes in duration of span of being, levels of detrimental behavior, levels of chronic disease, susceptibility to sudden adverse health events and susceptibility to sudden adverse behavior. Since electromagnetic field exposure was not created or produced by any known human which continues to exhibit vital being, there should be limited reluctance in organizations producing factors that emit electromagnetic fields to endeavor upon a path of Human priority by integrating insulating, absorption and covering capabilities into products, capabilities services, devices and infrastructure. Pervasively, these electromagnetic field mitigation capabilities in minuscule differences in costs and complexity of products and services. Similarly, these increase the value of investments in permanent magnet energy solutions and water synthesis from atmosphere solutions, resulting in regeneration of hydrological systems, vegetation system, atmospheric quality, and context for improve Human outcomes. Information. "Health." International Journal of Molecular Sciences. Volume 2021. Number 22. Page 3772.

The response is that physiology becomes programmed to find factors that alleviate homocysteine levels, which have particular innate shapes, tastes, smells, correlations, associations, textures, sounds, and other characteristics including less than conscious cues that are well known enough that about 90 percent of consumer behavior is derived by interactions among systems competing for benefit for increasing diminished control of inclination, associations, behavior and decisions. Thus, each iteration of sales, marketing, advertising or other campaigns provides intricate data about Human associations and behavior, allowing systems to frack deeply into the areas of biological function and cognitive function hat program Human outcomes. The physiological response to inadequate choline, inadequate NAD⁺, NADH, NADP⁺, NADH, enriched phosphatidylcholine, and other essential factors, including unimpeded access to clean fresh water are complex because rapid interaction with the brain occurs from even interaction with these with areas of physiology used for taste, texture and smell, for instance. However, these essential factors are so important that that the influences, systems, organizations and interests which control these bend Human inclination, behavior and outcomes to their own interests, sometimes allowing benefit to be obtained from exhibition of detrimental outcomes.

Physiologically, homocysteine and trimethylaminenoxide which is the primary causal factor in sudden adverse health events and perioperative complications, although also participating in

exacerbating choline inadequacy, result in inhibition of glycolysis, and rapid translation of NADH to NAD⁺ through the function of lactate dehydrogenase which produces lactate anion while metabolizing NADH to NAD⁺. This metabolic juncture supplies NAD⁺ to Parp signaling which occurs persistently because of the thymidine block resultant of inhibition of the hexose monophosphate shunt by P53 and NAD⁺ depletion. Thymidine is unable to be reduced to thymidine monophosphate and the accumulation of thymidine impedes Nucleotide Synthesis. However, the depletion of NAD⁺ and pyruvate by lactate dehydrogenase results in enhanced competition for the multiple fates or direction of pyruvates metabolism, and includes an upregulation of lactate anion which can be metabolized lactic acid. This moves the response to changing physiological conditions from a redirection of pyruvate to a change in NADH, NAD⁺, lactate anion, lactic acid, and level or Parp signaling. Parp signaling has numerous interactions that affect cellular division in the foundational confluence pathways as well as produces more homocysteine by continuing to deplete NAD⁺ in a way that requires nicotinamide methyltransferases to reduce Nicotinamide in a manner that produces more homocysteine.

The physiological response, then, includes the exhibition of this diminished ability of cellular entities including nonexercised muscle tissue to exhibit stamina while exercised or exercising muscle tissue are less affected. Along with diminished cognitive interactions through physiological fluids and among the cognitive, perceptive synapse, the result is a greater utilization of brain stem and innate response, increase circumventing of the conscious cognitive context, a diminished capacitance among the cognitive and behavioral synapse, and comparative increase in actively exercising muscle tissue.

All of which constitute an enhanced potential for detrimental behavior, particularly enabling powering through the conditions, circumstances and impedance in nature that are causing inadequate obtainment of homocysteine alleviating factors. Increasingly, civilizations have become aware of, learned and utilize the shapes, colors, smells, tastes, textures, associations and other factors that would lead to alleviation of homocysteine in nature, such that these are utilized to shape consumer behavior, resulting, also, in the diverse group of potential human outcomes in civilization, including detrimental outcomes, particularly those outcomes from which benefit might be obtained. These extend the platform and API to Human, social, behavioral and physiological outcomes, and represent on more specific loci or mechanisms at which systems of civilization may integrate into this example. However, the subjectiveness produced by homocysteine can even be shaped into detrimental health outcomes explaining why even health services entities exhibit marking, sales and advertising campaigns. However, the data observes that a mere increase in health facilities typically increases the level of detrimental or diminished Human outcomes, while a study of homocysteine below or above about 7 $\mu\text{m/L}$ produced a 500 to 1 difference in the instances unassured vital being over decade of observation, favoring those with homocysteine less than 7 $\mu\text{m/L}$. The study includes all cause unassured vital being such that even victims of detrimental Human outcomes had increased potential for being victimized, along with an increase of participants becoming victimizers, when homocysteine was above 7 $\mu\text{m/L}$.

The data analyzing ranges of homocysteine were varied, as were intent of such studies. Thus, in order to derive a general application of homocysteine levels to guide level and modality of care, correlations of homocysteine during admittance, direction to outpatient, care, office visit care, and general management were become priorities. Data in each of these areas were mixed with other study criteria. However, one study in particular integrated both objective and subjective factors in analyzing usefulness of homocysteine as an indicator of and triage factor in general aspects of care. The study indicates that homocysteine is useful as a health management indicator, although the same study observes wild fluctuations in homocysteine that make it difficult to be considered as detrimental factors, although this study clearly presents the hundreds of aspects of pathology required in every disease that homocysteine produces integrally with these or even independent of disease or diagnosis. Information. "Homocysteine." Medicine. Volume 100. Number 33. Page e26893. 8th Month, 20th Day, 2021.

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 50th 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 the homocysteine methyltransferase to prevent intramolecular disulfide links within the homocysteine methyltransferase. Intramolecular links within the homocysteine methyltransferase occur when inadequate sulfur is exhibited in blood and in the microenvironment, such that this enzyme's 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.

The emergence of homocysteine, choline inadequacy and PEMT inhibition, along with PP53 upregulation at changes to existential aspects of cellular membranes and its role as the fundamental basis for biological compartmentalization is important because it explains a continued exhibition and utilization of compartmentalization in diminished Human outcomes, even though thousands of years of experience clearly present compartmentalization across this Boltzmann transition from microbiology to social systems has not been effective at improving human outcome, while causing somewhat massive collateral and generational effects, although clearly this context seems to be providing information instead of being utilized as a rational bona fide mechanism to specifically decrease diminished outcomes. This projection of biology and physiology into systems, patterns in systems and outcomes are essential in producing wholistically applied improving change. Inadequate focus on assuring, repairing, reconstituting and sustaining the fundamental existential aspects of cellular structure and cellular metabolism are request in pervasive, if not all, disease, as well as explains therapeutic inadequacy.

Similarly, the emergence of homocysteine, choline inadequacy, and PEMT inhibition, along with P53 upregulation at decreased number of cellular entities per micrometer of tissue, also presents

existential level challenges to physiology. Together with challenges to cellular existence, these are translated into changed behavior, which the analyses with which this analysis is associated clearly present as resulting in impaired electromagnetic synapse, chemical synapsis, neurological synapse, perception, cognition, circumventing of insertion of stimuli for conscious cognitive processing, impaired rewards systems function utilize to determine appropriateness of behavior to circumstance, impaired conditioning, impaired recall of conditioned responses in other contexts, impaired blocking, impaired ability to withstand externally impose stimuli/response pairings, deterioration of areas of the brain required for learning, memory and behavioral control, such that all of these begin to emerge as detrimental physiological effects even during gestation.

It is not difficult to conclude that these are involved maternal risk, maternal making of decisions regarding gestation and in determination of human outcomes, particularly introducing patterned risk for outcomes in correlation with inadequacy, including detrimental behavior, behavioral health conditions, and diminished physiological outcomes.

However, the role in which choline status, homocysteine status, PEMT status and P53 upregulation are subtly at the foundational and empirical aspects of pervasive health status and human outcomes. A study observes that homocysteine disrupts D2 dopamine receptor activation. Homocysteine performs as an allosteric D2 receptor agonist which selectively diminishes the affinity of D2 receptors in a manner that excludes antagonists, while homocysteine effect in this regard utilizes Arginine/Thiol electrostatic influence to produce noncovalent complexes that include 2 arginine dense epitopes as well as which includes the 3rd intracellular loop of the D2 dopamine receptor which can include the A2A/D2 receptor homodimerization intracellular loop of D2 dopamine receptors. This among the hundreds or maybe thousands of detrimental effects of homocysteine explain impaired dopaminergic function and impaired synthesis of dolichol and neuromelanin in Parkinson's disease, diverse and myriad diseases with impaired movement or coordination, neurodegenerative disease, but diseases of impaired focus, attention and impaired behavior. Most importantly, these explain how homocysteine dissociates conscious biological and cognitive function from autonomous and innate physiological interacts that are programmed to resolve choline inadequacy, inhibition of PEMT, homocysteine increase and P53 upregulation. The context of dopaminergic fasting in which humans consciously exclude peripheral stimuli to focus on specific reduced sets of objective attainment, are relevant.

Importantly, the dopaminergic involvement of homocysteine is an important assertion that extends the model of P53 diminishment of pyruvate availability, while PAPR signaling depletes NAD⁺ in a manner that causes NADH and pyruvate to be increasingly metabolized by lactate dehydrogenase toward lactate anion which potentiates also lactic acid along with NAD⁺ production. For cellular entities and muscle tissues not actively exercising, this produces a high sensitivity to glucose depletion because PEMT inhibition, choline inadequacy, homocysteine, and P53 upregulation prevents systemic absorption of glucose by inhibiting GLUT glucose endocytosis and preventing glucose from any source such as from gluconeogenesis to be trapped in glycogen cycling where amyloid fibrils are extended and catabolized along with diminished endocytosis of glucose and diminished directing of glucose into glycolysis, pentose phosphate pathway and hexose monophosphate shunt. The insulin receptor is also inhibited by P53 in this

context. Canonically, the insulin receptor can enhance metabolic throughput within glycolytic pathways through phosphofructokinase, although, however, an increasingly rapid depletion of pyruvate can be produced in tissues and nonexercising muscle tissue, resultant in lactate accumulation, depletion of pyruvate and lactic acid accumulation sensitivity that requires conditioning to diminish as an impendence to performance.

Most importantly, it is the context of actively exercising or actively utilized muscle tissue, fibers and cellular entities which is most interesting because these escape impendence to endocytosis of glucose and escape glucose direction into glycolysis, such that the glucose that accumulates in circulation resultant of choline inadequacy, PEMT inhibition and P53 upregulation, is, instead, able to be directed toward these actively utilized muscle tissues. This explain hypertrophic growth in and required utilization of particular muscle tissue basis to sustain hypertrophy, sustain leanness, and avoid hypertrophic growth toward adiposity, all of which seem to be the result of decisions in development which occur in a continued endocytosis of glucose without function of PEMT and without assured choline, cellular structure and metabolically essential cholesterol, as well as diminished levels of cellular entities per micrometer.

Impairment of these aspects of enhanced muscular endocytosis of glucose, impaired involvement of conscious cognitive function in the stimuli/response pairings, and impaired dopaminergic function, increasingly explain how existential aspects of the basic biological compartmentalization as well as how existential aspect of the density of cellular entities per micrometer become translated into cognitive function, behavior, and integrated sets of inclinations, compulsions, decisions and outcomes. These explain inclination, addiction, compulsion and impaired behavioral factors linked to diminished outcomes. Resultant upregulation of Cytochrome C, Bax and Bak which promote massive deterioration of cellular entities and P53 promotion of pause in hypertrophic phases of mitosis, are integral to this context because these require upregulation survival signaling to prevention somewhat complete deterioration of cellular bases and tissues. NOS2 inducible version expression which occurs also in this context extends this paradigm because NOS2 is detrimental if expressed in more than ephemeral duration, can become uncoupled to produce acute phase, and is expressed to improve the turgor of cellular entities in assistance of supporting anatomical structure. NOS2, illustratively, is expressed in astronauts returning from space travel to assist in supporting physiological structure. Interestingly, and concluding, these factors clearly present that tissue density per micrometer, cellular structure nutrient density, both, are existential aspects of being that affect factors that transcend location, space and time affect human inclination, perception, cognition, physiology and behavior. Information. "Allosteric." *Journal of Proteome Research*. Volume 5. Pages 3077 to 3083. 2006.

The unassured existential aspects of Human physiology and behavior, thus, emerge as susceptibilities and human outcomes provide intricate information about such susceptibilities as well as the universes level influences, into antecedent aspects of time and future aspects of time, as well as those of immediacy, act upon these susceptibilities. Pervasively, unassured human, social, behavioral, physiological and other requirements are the empirical basis for diminished human outcomes and diminished human events, elute these as

outcomes, information and opportunities for analysis, understanding, prevention, intervention in the ways that prioritize humanity, but inclusively in resolution thereof.

Parenteral instrumentation of choline and phosphatidylcholine, illustratively, are recommended by health services authorities to prevent nosocomial microbial conditions which emerge in particularly when choline is not included in nutritional preparations during hospitalization. Hospitalized populations can emerge as a source of systematically produced resistant microbes not because of proximity to microbes being therapeutically, but because it is diminished PEMT function and choline inadequacy that are pervasively linked to susceptibility for opportunistic microbial affliction as an entry point for hospitalization this context and because of inadequately pervasive management of choline, PEMT, P53 and homocysteine before admission for hospitalization and within parenteral nutrition or other therapy. Information. Volume 137 Supplement S. Pages S119 to 128. Gastroenterology. November, 2009. Information. Choline. Fact Sheet for Health Professionals. Office of Supplements. National Institutes of Health.

CRISPR, importantly, as a gene therapy may be utilized to impair the genome of bacteria therapeutically as well as produce antisense nucleotide sequences that impeded transcription of bacteria. This Important potential should become a priority for human populations, particularly as exploration of extraterrestrial contexts increasingly emerge, although there may already be enough instances or contexts in which such application may be improve human outcomes. Crispr is important because it allows genetic sequences which have become integrated through the activity of pathogens, which have been changed through other impairing circumstances, or which are within pathogens, all to be excised, changed and replace to the favor Human health. These include emerging ability to produce antisense RNA or DNA to ablate disease enabling proteins, ablate the transcription of genes by pathogens or by genes introduced by pathogens into Human genome, as well as implementing specific cellular intervention that causes pathogens or diseased cellular entities to deteriorate. Advances included instrumentation of personalized therapies as well as instrumented generic application of CRISPR along with protein transduction domains to assure efficient complete anatomical transducing of cellular entities which is a challenge in pervasive therapeutics. Also, activating designer proteins can be produced which release CRISPR activation sequences when a particular genetic sequence, protein sequence, pathogen protein or even a particular temperature or particular tissue type is encountered.

These tissue specific therapies are very interesting since it is now known that pathogens pervasively, particularly viral vectors, require destabilization of G quadruplexes in telomeric regions along with TNF and particularly SP1 contents, in order to produce latent disease. Q quadruplex destabilization commandeers L arginine and Ca^{2+} from NOS2 inducible, producing cyclic enhanced cycling between L arginine and L citrulline to polarize macrophages toward the inflammatory macrophage phenotype and escaping L arginine begin directed toward Arginase which is a resolution phase macrophage phenotype. Latent disease produced by viruses or other conditions are emerging more and more as being enabled by G 4 destabilization, SP1 Release from G4 quadruplexes, inflammatory pathway persistence enabled by destabilized G4, SP1 stimulated downregulation of CD4+ and CD8+ immunological synapse receptors which receive MHC complexed immunological monitoring proteins and present these for lymphocyte

processing in reverse sequence into lymphocyte genome known as V(D)J recombination, movement of the integrated antisense sequences to the thymus by lymphocytes where pruning, priming, and nursing processes produce efficient lymphocyte monitoring processes which attached to CD4+, CD8+ receptors during immunological monitoring, division into immunological cascade by lymphocytes, or presentation of the antigen sequences to lymphatic center B cellular entities which assist with immunological cascade.

SP1, along with diminishing CD4+ and CD8+, also upregulate PDL1 and PD1, both of which obscure diseased or impaired, as well as inflamed or pathogen commandeered cellular entities from lymphocyte search and monitoring capabilities as well as prevents lymphocyte attachment to ligands and receptors exhibited by these diseased cellular entities. Similarly, SP1 upregulates telomerase which causes the telomeric regions that have increased numbers of G quadruplexes and SP1 transactivation loci to escape transcriptional depletion during each cellular division. Thus, stabilization of the genetic loci at which destabilized G 4 are occurring as a feature oSP1 upregulation of telomerase, although SP1 is an inhibitor of PEMT and upregulator of the CDP choline pathway at CTP Choline Cytidylyltransferase . Importantly, SP1 upregulation of telomerase or hTERT potential prevents AP1downregulation of telomerase from causing depletion of telomeres which would result in exit of inflamed, impaired or diseased cellular entities from mitosis through chromosome fusing that emerges when telomeres are depleted in cellular entities. The removal of limitation on AP1 by SP1 causes latent disease from a transcriptional perspectives because this results in the continued upregulation of AP1, inhibition of PEMT by AP1 although SP1 inhibits PEMT by this same or another mechanisms, while Ca2+ depletion by G 4 participates in this pathology context to cause dissociation of the mitochondrial associated membrane which results in inadequate supply of Ca2+, Phosphatidylserine, Phosphatidylethanolamine, and possibly phosphatidylinositol useful in activating autophagy, all occurring through transfer from the endoplasmic reticulum to the mitochondria through the shared emerged mitochondrial associated membrane which these two cellular subcompartments share. These explain why catalytic activity of PEMT2 , in particular in the most advanced or detrimental of disease and diminished health statuses, is typically regarded as being strongly deteriorated although some diseases linked to trimethylaminenoxide as well as participation of diminished PEMT1 or PEMT3 also participate in disease, diminished physiological capacitance or diminished cognitive capacitance, and diminished Human outcomes.

Its important to present that PEMT1 is activated in development while PEMT2 emerges in later aspects of gestational development or even following birth, suggesting that PEMT2 may be a regulator of growth and development, particularly balancing size or growth with energy levels required to sustain physiology, cognition, and movement through electron transport pathway, oxidative phosphorylation, and the hundreds of mitochondria that can be exhibited in each cellular entity which exhibit PEMT2 activity.

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.

Choline inadequacy impairs the immunological synapses, impairs nonspecific permeability of the outer plasma membrane, impairs specific permeability of the inner plasma membrane, impairs V(D)J adaptive immunological genetic repair in immunological cellular entities, impairs lipid raft characteristics in the caveolae, and along with NOS2 inducible version impairs dilatation of the caveolae, as well as along with NOS2 inducible enables microbes to escape the toxic plasma membrane interstitial space using can include NOS2 and phospholipase D enabled endosomes within which microbes move into the intracellular space. A characterization of NOS2 function when expressed ephemerally is that it supplies toxic reactive molecular species used in intracellular and extracellular microbial defense to flush microbes from the intracellular space and supplies toxic molecular species for exhibition in the plasma membrane intracellular space. These toxic molecular species can also be emitted in the extracellular space, such as from NOS2, NOS1 and NOS3 to participate in extracellular defense, although these can include uncoupling of nitric oxide synthase versions that can become participative in the acute phase.

Most imperatively, hepatic organ and thymus, both deteriorate resultant of choline deficiency and correlative to homocysteine exhibition, including in correlation with typical patterns in detrimental aspects of aging. Impaired development, nursing, and sustainment of T lymphocytes in thymus also emerges, diminished the immunological synapse resultant of choline inadequacy. However, beginning with choline inhibition of P53 downregulation of biosynthesis pathways that occurs with PEMT inhibition or choline deficiency or both, diminished expression of genes and biosynthetic enzymes emerges which are replaced with inflammatory signaling, including upregulated expression of MHC antigen presentation proteins which systematically integrate with proteins and molecules in the intracellular space, then move these to the extracellular interface of the plasma membrane, most aggregately within lipid rafts and most aggregately at the Caveolae, resulting in presentation of this within CD4+ and CD8+ receptors which lymphocytes and leukocytes utilize for antigen reception, antigen reporting, antigen monitoring and copying of the inverted protein sequences into their own DNA using adaptive immunological DNA repair such as V(D)J recombination. Innate and compliment immunological function can also utilize these receptors for marking, amplification and enhancement of immunological cascade. However, these processes present not only how microbes are found directly in circulation or anatomy in in the humoral immunological response, are found in the cellular response by cellular level reporting pathway, and, however, presents the context in which T cellular entities or Humoral as well as cellular level cascade can result in presentation of antigen by these immunological factors to lymphatic centers where immunological be cellular entities are activated in a similar extended immunological cascade.

Foxn1 is known to promote regeneration of thymus. Information. "Thymus Organogenesis and Development." *Eur J Immunol*. Volume 46. Number 8. Pages 1826 to 1837. 9th Month, 2016.

Regeneration of Islet Beta Cellular entities has been presented in the literature using IGF 1 or insulin growth factor I. Islet beta cellular entities are hyperactivated by accumulation in circulation of glucose resultant of P53 inhibition of GLUT endocytosis of glucose, overloaded

from toxic contents of cellular entities catabolized during massive apoptosis which emerges resultant of choline inadequacy, both which of which produce such levels of oxidative distress that cellular entities produce insulation that is distributed to much of physiology using arginine pathways in vasculature as which produce such levels of oxidative distress that Islet Beta cellular entities can activate an autoimmunological response to themselves as well as which can cause Islet Beta cellular entities to dedifferentiate into other cellular entity types. This process of dedifferentiation is perhaps a most underconsidered modality of impaired differentiation, mesenchymal phenotype emergence and impaired mitotic characteristics exhibited in disease. Information. "Regenerative Medicine." World J Gastroenterol. Volume 26. Number 22. Pages 2948 to 2966. 6th Month, 14th Day, 2020.

Immunological cascade can, then, also involve leukocyte activation of diseased or impaired cellular entities using T, B, or other cellular entity marking and activation of intracellular immunological pathways to cause hyperactivation such phosphorylation cascade or activate specific pathways such as Trail, Caspase or other pathways causing cellular deterioration of destabilization. An interesting context to present is that NOS2 inducible is an inherent inhibitor of PEMT. Also, upregulation of choline kinase alpha and thus upregulation of the CDP choline pathway, increases phosphocholine and phosphocholine upregulation causes low to middle level activation of the complements immunological system and this pathway is the underconsidered pathway for both vascular deterioration and participation in particular nuances of vascular repair such as with Estradiol instrumentation.

GSK3B inhibitors which are invoked in SIP 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 selpercatinib and vandetanib. Information. “Ret Tyrosine Kinase.” RSC Adv. Volume 12. Number 2. Pages 1194 to 1207. December 22, 2021.

The dynamics of managing homocysteine and assaying homocysteine, then, emerge in the context of other assay of molecular mass in hematopoietic fluid. Hb1AC assay, for instance, are complicated by prandial status, such as before, after, during fasting periods of nutritional obtainment. Hemoglobin, also, through red blood cell recycling, results in somewhat pervasive cycling ever 4 months or so. Thus, Hba1c, should assay the duration that a hemoglobin has been exhibited empirically, the duration of which hemoglobin has been glycosylated, and perhaps, although this seems to be the priority of such assay, the levels of glycosylation of hemoglobin. There may be extreme levels of glycosylation in this regard, but therapies typically are not interventional unless utilized in acute care, resulting in a chronic management of glycosylated hemoglobin from what could be periodic increase. The recommendation for fasting before testing improves the relevance of chronic management with an instance of assay.

Trimethylaminenoxide is another inhibitor of PEMT. The literature does not pervasively acknowledge PEMT status and trimethylaminenoxide even in extended duration studies, while it is known that some of the major pathways supplying oxygen , metabolites, nutrients and repair

factors to the brain are impeded by deterioration of carotid intima media which can only be repaired to adequate plasticity by managing trimethylaminemethoxide unless mechanical mechanisms are applied to open such pathways without improving carotid intima media plasticity.

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 homocysteine was assayed within 24 hours of admission which diminishes reliability of the study because even Saline can be utilized to 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 $\mu\text{M/L}$, which are interesting because objective consideration of homocysteine in the translational wellness clinical platform clearly observes that at 15 $\mu\text{M/L}$ without any symptomology and at 10 $\mu\text{M/L}$ 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 experienced unassured vital being, 37 had recidivist ischemia, 22 were not included in follow up, resulting only 471 participants being reviewed while subsequent to this review at 3 months, 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 a 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 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.

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 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 it is 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 have been required or B vitamins may be deficient. These also suggest that hypothyroidism before therapy is a lesser

pathology status at least 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 well 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 methionine 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.

Mitochondria experience recycling through a version of autophagy known as mitophagy. PTEN products P53 from being deteriorated through ubiquitylation, while PTEN also enables expression of PINK1. PINK1 accumulates in the inner membrane of stable mitochondria but also accumulates in the outer mitochondrial membrane of unstable mitochondria. TOM and TIM23 both perform import of PINK1 into the outer or inner mitochondrial membrane while PARKIN integrates with PINK1 in the outer mitochondrial membrane, while the mitochondrial directing sequence, MTS, that directs PINK1 to mitochondrial members is segmented from PINK1 by Mitochondrial peptidase processing enzymes. Parkin1 interaction with PINK1 in the outer membrane cause fragmentation of mitochondria, although typically mitochondrial potential has already been diminished when PARKIN and PINK1 accumulated in the outer mitochondrial membrane, resulting in fragmentation. Submitochondria produced as a result are then recycled into other mitochondria or other mitochondrial fragments when potentials in such submitochondria are adequate, resulting in recycling.

Cellular entities exhibit sometimes hundreds of mitochondria and mitochondria can even move between cellular entities in muscle tissue, particularly in sheathed cellular in shared sheathing. Mitochondria attach to endoplasmic reticula through an emerged shared membrane known as the

mitochondrial associated membrane through which sharing of phosphatidylserine, Ca^{2+} , phosphatidylethanolamine and other factors are exchanged, while phosphatidylserine, Ca^{2+} , phosphatidylethanolamine stabilize mitochondria, supply PEMT with substrate, and enable mitochondrial control of cellular outcomes including programmed deterioration that causes apoptosis, other cellular outcomes and statuses, as well as rescuing cellular entities from control imposed by membrane level existential dynamics that occur during choline inadequacy, cholesterol inadequacy and existential challenges that include promoting of proteolysis and survival signaling pathways such as upregulation of choline kinase as an aspect of upregulated CDP Choline pathways.

PEMT is also potentially reliant upon transmembrane weaving between the endoplasmic reticulum and mitochondria through the mitochondrial associated membrane or at least in the mitochondrial aspect of the mitochondrial associated membrane. Although PEMT2, mitochondrial PEMT version, may be functional elsewhere in the mitochondria. However, the typical pathway to pervasive disease includes existential challenge to cellular membranes and as diminished cellular entities per micrometer in tissue, along with PEMT inhibition, P53 upregulation, survival signaling to counteract massive apoptosis that occurs when PEMT is inhibited and choline is deficient, upregulation of proteolysis and choline kinase alpha, upregulation of the CDP choline pathway, diminished enriched phosphatidylcholine which PEMT would produce if not diminished also, culminating in deterioration of the mitochondrial associated membrane, and obliteration of PEMT2 function as well as potentially impaired function of PEMT versions generally. A particular article presents the importance of nutritional sustainment of mitochondria, including utilization as therapy for conditions which emerge with aging. Information. "PINK1." *Journal of Neurochemistry*. Volume 139. Issue S1. Pages 232 to 239. October, 2016. Information. "A Strategy for Healthy Brain Aging." *Antioxidants*. Volume 9. Page 932. 2020.

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. Trimethylamine oxide 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, while also clearance of trimethylamine oxide is a major status change that occurs with dialysis although clearance of trimethylamine oxide 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 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.

Management of homocysteine, creatinine, blood urea nitrogen, trimethylamine oxide, asymmetrical dimethylarginine, symmetrical dimethylarginine, B12, Folate, and, thus, emerge as correlated and supporting pathways in emergency, inpatient, outpatient, and proactive modalities of care.

Information. "Mechanism." PLoS One. Volume 10. Number 12. Article e0143731. 2015.

Information. "Levels." Scientific Reports. Volume 10. Article 18050. 2020.

Information. "Treatment." Clinical Treatment. Volume 47. Issue 9. Pages 1738 to 1741. 9th Month, 1st Day, 2001.

The factors presented in this analysis suggest that impaired focus on the priorities that resolve and assure health status, behavioral control and management of homocysteine may acculturated, typical and imposed in civilizations, often utilizing substitute factors as intermediaries in analyzing, considering, obtaining and instrumenting the factors that resolve homocysteine, assure pemt, and which are integral to behavior in context of control and conscious levels of control, particularly when compared to control mechanisms instrumented in civilization through comprehensive groups of stimuli and response pairings. Information. Chapter 57 Level Consciousness, in Clinical Methods. The History, Physical and Laboratory Examinations. ISBN-10 0-409-90077-x.

It is well known that background ph of near between 7.2 to 7.6 are typically essential in sustaining consciousness, particularly conscious cognitive function. The literature does not present as much context for less than conscious statuses with regard to ph out of these ranges other than paralytic status which are to be remediated, although certainly, there may be correlation to ph, conscious cognitive control and potential for interaction at less than conscious levels when this range of ph may not be exhibited. However, the literature presents a continuum of levels of consciousness that include clouding which includes inattention and reduce wakefulness, confusion which includes disorientation along with bewilderment and difficulty translating commands into compliance, lethargy which includes particular stimuli' ability to effect arousal along with a propensity otherwise to reenter less than conscious status, obtundation which includes similar specific stimuli' effecting of arousal although with an diminished interested in the environment amid slow responses to stimuli as well as enhanced propensity for reentering less than conscious statuses. Stupor is constituted of requirement of repeated and vigorous stimuli to effect arousal while such repeated and vigorous stimuli are required to persist the aroused status. A coma is typically constituted of inability to effect arousal from a less than conscious status or inability to effect arousal to a conscious status.

It is clear that consciousness may be promoted or sustained by stimuli and these stimuli perform as a framework for more substantial or focused aspects of behavior, cognition and activity. A relevant example may be the exhibition of a context or environmental group of prominent stimuli, along with the conjuring of associations that are linked from those stimuli or exhibition of stimuli which conjures into relevance a stimuli and response pair from another context, such as less than conscious interactions moving into consciousness or such as conditioned linkages in other context or learning environment being conjured into relevance within a different environment or context. Both of these are explored in the literature with regard to homocysteine, choline deficiency enabled deterioration of the brain beginning even in gestation, as well as in other aspects of the literature. It is very difficult to exclude electromagnetic fields, freakish anatomical structural elements which the literature present as emerging in luminal areas of

physiology along with expression of NOS2 inducible, phospholipase D, and phosphatidylcholine specific phospholipases that are stimulated into expression in luminal areas by electromagnetic fields.

Another aspect of the NIH stroke scale includes horizontal eye movement. The literature again seems to exclude direct linkage of the NIH stroke scale with homocysteine, but the literature clearly links homocysteine with deterioration of visual tissues and deterioration of aspects of the brain linked to visual function. A particular studies specifically presents exhibition of impaired horizontal eye movement in association with homocysteine increases along with improvement of atypical horizontal eye movement characteristics resultant of managing elevate levels of Homocysteine. Information. "Vitamin B12." Intern Med. Volume 59. Number 24. Pages 3229 to 3233. December 15, 2020. Information. "Folates." Eye (London). Volume 22. Number 8. Pages 989 to 993. 9th Month, 22nd Day, 2008.

The literature presents some of the neurological pathways that are involved in impaired eye movement and these include medial rectus, lateral rectus, oculomotor nerve of the cranial nerve III, adducens nerve of the cranial nerve IV, Brainstem medial longitudinal fasciculus MLF, thereby comprising the 3 major disorders of eye movement such as lateral gaze palsy, one and a half syndrome, and internuclear ophthalmoplegia. These provide the integral effect to brain and brainstem function which homocysteine may impose, although this is merely one dimension of one aspects, of one organ's function, revealing the potential widespread reprogramming of perception, cognition and behavior impose by homocysteine. Information. "Correlation." Radiographics. Volume 33. Number 1. Information. "Folates." Eye (Lond). Volume 22. Number 8. Pages 989 to 983. 8th Month, 2008.

The literature is careful to consistently include gestational, after gestation and developmental aspects of neurological, neural tube closure, spina bifida, and impair myelin synthesis and maintenance, all in peripheral consideration of homocysteine and choline, as well as phosphatidylcholine adequacy, converging making of decisions, cognitive clarity, gestation, maternal carriers of gestational instances regarding health and making of decisions, all in an interesting focused view on homocysteine, opportunities to vastly improve diminished Human outcomes, along with extraordinarily expansive groups of very specific pathophysiology.

Cognitive capacitance seems to be derived from electrons transport pathway synthesis of ATP which involves freeing of Hydride from NADH factors, about 58 percent of the 2 eV- freed from NADH being exhibited as fluorescent and molecular energy that is evenly distributed among the phases of the electron transport pathway or oxidative phosphorylation, while about 42 percent typically is integrated into the oxonium that is exhibited between the phosphate groups of ATP. ATP integration into {methionine enhances the hydridic character within methionine causing the sulfonium to become Ionized into a Cation through a Methyl Group and Hydride carbocation. Mitochondrial fraction of capacitant and fluorescent influence is a major component resultant of both PEMT2 activity at the mitochondrial associated membrane and because of the hundreds of mitochondria in a particular cellular entity along with mitochondrial recycling that occurs in cellular entities, typically. Nucleus and Endoplasmic reticulum are contributors of variable

levels of capacitance also, particularly as result of changed signaling that redirects enzymes and substrate to different subcellular compartments.

A study, contrast to other studies that suggest that DHA in particular downregulates PEMT activity, observes that omega 6 and omega 3 fatty acid supplementation in a small nonhuman mammalian organism upregulates PEMT activity. This upregulation of PEMT by omega 6 and omega 3 resulted, in the study, included selectivity for species of phosphatidylethanolamine which exhibited extended length nutritionally obtained n6 and n3 fatty acids. Omega 6 fatty acid enriched nutritional regimen, in the study, observes also a high ration of both omega 3 and omega 6 fatty acids in the synaptosomal membrane fraction of phosphatidylethanolamine, concurring with the literature's observation that omega 3 is derived from particular junctures of omega 6 fatty acid processing pathway. The synaptosomal membranes are linked to neuronal junctures in tissue, according to the literature, suggesting that acetylcholine processing from pyruvate processing toward acetyl CoA followed by Acetyl CoA decarboxylation by cholineacetyltransferase which determines acetylcholine levels, acetylcholine storage levels modified by acetylcholine esterase activity, all of which are downregulated by inhibited PEMT and occurs along with the NKCC1 to KCC2 switch as developing humans emerge into maturity to result inversion of polarization direction of action potential in neurons, also resulting in a change in maternal to infant capacitant exchange during hyperpotentiation such as during breast feeding, all may be factors involved in synaptosomal phosphatidylethanolamine processing. Agrin is also known to be active in capacitant, hydridic, polarized contexts as potent actuator of acetylcholine receptor aggregation linked to innervation and neurological development, beginning from conception and into other phase of development. Information. "Phosphatidylethanolamine." *Biochim Biophys Acta*. Volume 918. Number 2. Pages 97 to 105. 4th Month, 3rd Day, 1987.

Another longitudinal study observes a decrease in muscle function, coordination and grip strength which occurs in correlation with elevated homocysteine. Information. *J Gerontol A Biol Sci Med Sci*. Volume 73. Volume 4. Pages 545 to 551. March, 2018.

General the NIH stroke scale assays factors that invariably involve the detrimental aspects of homocysteine. Another study presents that homocysteine can be useful in estimating the exhibition of causal deteriorating conditions causal of and correlated with aphasia including DCI, aSAH, and factors producing a spectrum of linked disorders such as hemiparesis, apraxia, aphasia, hemianopia or deprivation. Information. "Admission." *Front Surg*. Volume 8. Article 813607. 2021.

The literature observes that PEMT selects newly synthesized phosphatidylethanolamine exhibiting tails that are unglycosylated or lightly glycosylation. Other relevant features of PEMT pathway processing include an about 30 percent contribution of PEMT to membrane phospholipids as an aspect of the CDP ethanolamine pathway, compared to the about 70 percent contribution of phospholipid through the CDP ethanolamine pathway, at least in the hepatic tissues. Also, phosphatidylcholine constitutes more than 50 percent of all phospholipids as well as 30 percent or more of lipids in the cellular membrane, typically. The de novo synthesis of choline as phosphatidylcholine is major determinant of the existential aspects of cellular

structure, cellular metabolisms, tissues, glands, organs, connective tissue and other factors. Phospholipases and Phosphodiesterases can deteriorate or catabolize cellular membranes during inflammation, deficiency or impairment to produce or synthesis, or mimic availability of choline, although de novo synthesis and nutritional obtainment of about between 7 mg per kg of anatomical mass is essential to prevent advancing deterioration of cellular membrane and tissue existence that is known as detrimental aspects of aging and enabling conditions for pervasive disease, diminished behavior and diminished aspects of cognitive, neurological and social function.

Supplemental choline should be raw, uncooked, unchanged by high cooking temperatures or warm when ingested as food to enable chemical, thermodynamic and structural readiness for digestion. Some supplemental versions of choline and phosphatidylcholine are produced ready for ingestion, although typical translation into bioavailability of supplemental factors can be about 5 percent of ingested portions, phosphatidylcholine enhances bioavailability of other ingested supplemental factors and although lecithin is hybridized version of choline that has both choline and phosphatidylcholine.

The literature, contrasting earlier conclusions, suggests that phosphatidylethanolamine methyltransferase has an individual typical location to which phosphatidylethanolamine, phosphatidylmonomethyl ethanolamine, and phosphatidyl dimethylethanolamine each integrate, with the first methylation or attachment of CH₃ to the nitrogen within phosphatidylethanolamine being the rate limiting or Michaelis juncture step or phase of the 3 sequential methylations required to produce enriched phosphatidylcholine from phosphatidylethanolamine. The three individual sequential methylations sometimes are presented as occurring independently, with multiple PEMT enzymes performing interactions with a phosphatidylethanolamine as it becomes PMME, PDME and then phosphatidylcholine. However, other literature suggests that PEMT as transmembrane protein exhibits structure turns as each methylation occurs, which seems most precise because 3 dimensional changes, rotations and movement are often essential to exposing areas of a biological molecule that introduce different interactive potential as posttranslational modifications occur.

This compendium of reach presents this as shape, twist and writhe, which includes rotations, turns, twists, bends, movement and 3 dimensional as well as quaternary changes to a biologically active movement, along with resonant changes in which atoms near the molecule causes changes to structure, shape or reactivity. These can include carbocation rearrangements, managed separation of hydride from an integral molecule to produce energy release or fluorescence in a way that abdicates control to a molecule into which a hydride exhibiting molecule is integrate, as well as potentially introducing angular characteristics to such fields such as changes frequency, wavelength, and even introducing susceptibilities such as red shift resultant of quality, characteristics, homocysteine levels and other factors in microenvironment and in other aspects of physiology. Interestingly, these seem to be a way in which classical nuances of physics may be reentering biophysics and allow more expansive participation of the sciences in understanding nuances of physiology and biophysics. These dynamics, however, widen the possibility and equation verifiable nuance of physiological interactions into antecedent eras, into future eras, and

certainly transcendent of distance, location, space and time. Hydridic fields, light, fluorescence, free energy, electromagnetic fields and current, all are not only produced and used by physiology, but are integrated within the components that comprise foundational biological structure and function.

Information. "A Brief History of Time." Volume 61. Number 3. Pages 254 to 258. 2012.

Information. "A Short History of Rubisco." Current Opinion in Biotechnology. Volume 49. Pages 100 to 107. February, 2018. Information. A Brief History of Time. ISBN 978-0-553-38016-3.

Interactions occur between events and humans in eras of immediacy, future and antecedent epochs. These interactions occur in ways that effect phosphate groups in neurological and cognitive centers and are essential to contrive typical cognition. These interactions may be important factors in establishing stable advancement of events, while it is order of events that most determines stability, suggesting the time is only a figment of the imagination, although order most contrives tangible nuances of time, although Human interaction with photons and electrons, as well as particles causes these to collapse from superposition into particles and characteristics of tangibility. Hydride performs as a universes level field among perhaps billions of fields that human physiology, cognition, perception, cognition, expression, and constructive activity connects in ways not possible otherwise or uniquely compared to other ways in which fields or factors of the universe interact. Humans perform as oscillating mechanisms that connect, conduct, conclude or otherwise affect fields that extend to all aspects of the universes.

Adjudicative interactions occur in any context, including human context, to determine which macroscopic, tangible, microscopic or other system of dynamics has the most prevalent or differential influence to an outcome, while it is the defined space that determines which system of dynamics is most precisely affective to an outcome. Nanoplasm is the most empirical representation of solution, system, or molecular factor and at such levels systems of dynamics can cumulatively produce such a context, but a system of dynamics typically describes most differentially and most precisely the characteristics of activity or conditions in such defined space. Atoms can behave as other atoms, as long as there are adequate subatomic material, particular conditions, and, especially in biology, resonant, nonlocal, disconnected, aromatic interactions that are typical of biology, including through space jumps of electrons, just in time movement of electrons between molecules and atoms, hydridic character, carbocation, emitted fluorescent or 2 eV- energetics, tunneling of electrons through potential, and other interesting activity exhibited in biological systems.

Events in one era continue to compete with alternative outcomes in that era, even after an adjudicative outcome has occurred, requiring interactions with antecedent and future eras to strengthen the factors, events and conditions that rely upon the status quo. Activity in defined space can enable a metabolic interaction that is required by physiology by not completely satisfied by canonical nanoplasm, such that unless replenishment or stabilization of such metabolic interaction occurs nutritionally or therapeutically, biological systems can become destabilized and such metabolic interactions can be outcompeted by alternative statuses in antecedent instances, suddenly not having occurred in antecedent instances, and produce systemic destabilization multiple organ distress syndromes. Choline adequacy and

phosphatidylcholine adequacy through PEMT are potent stabilizers of existential aspects of physiology, counteracting alternative events that compete for deterministic influence over other events that have occurred or other outcomes that have already occurred. Interactions into antecedent eras connect Humans and human systems to creative forces and contexts of Universes, such that increasingly conscious understanding of these nuances of cellular existentially, anatomical existentialism, cognitive interactions through phosphate groups of neurons which typically encapsulate hydride between the phosphate groups of ATP as oxonium, all produce interactive reinforcement of individual and Human existentialism.

The most interesting perspective is quantum entanglement in which material that interact in the universe exchange subatomic particles and become entangled with a somewhat permanent interactivity, such that this interactivity can be very strong in some instances, less than strong in others, exhibit multiplicity, and result in changes in one entangled factor causing response changes in the other entangled factor or factors. This interaction occurs at 30,000 times the velocity of light, and enables Humans to interact with much of the universe before such aspects of the Universe are as they are observed. This perspective allows Human observation to result collapse of a electron, which is subatomic material exhibited in a orbital within one or more energy levels that has probabilistic aggregation resultant of interactions with other atoms, from superposition to particle characteristics although an electron or any material may be caused to exhibit wave characteristics, energy characteristics, angular influence, all become emerged by interactivity. Experimental contexts were able to produce two sequential entangled relationships with one common particle between these two entangled contexts, followed by experimentally changing one of the entangled factors in the first relationship without viewing the effect to the second entangled factor. Subsequent production of the second entangled relationship using the first or controlled varied factor from the first relationship along with a newly entangled second particle, resulted in the ability to introduce changes to the particle involved in both entangled relationships that not only affected the newly entangled particle in the second relationship, but changed the characteristics of the unviewed changes in the first entangled relationship. The result was an ability, as a distribution but not 100 percent on a one to one basis, to change what hat may have occurred in an antecedent instance.

A more specific explanation of this phenomenon is as follows. The observational conclusions are that management of choline, phosphatidylcholine, homocysteine levels, cellular membrane stability, number of cellular entities per micrometer, as well as Human, social, behavioral and physiological requirements produces a prioritization and resilience of status quo, particularly how human events have resulted in the population members exhibited at any instance in time or in any instance of the Human experience. Management of these factors also enable individual capacitant aspects of vital being, cognition, and decisions to emerge in context of adequate or optimal characteristics of aspect of neurological tissue and systems which enable interactions with antecedent and future eras in a way that interactively enables Humans and an emerged status quo among Human social systems to make the kind of decisions and iterative review or cyclic decision making that navigates the influence of alternative outcomes in antecedent or future eras which endeavor to bend the Human experience as well as bend Human events toward paths or events that prioritize such alternate outcomes. A river with a meander has competing

interests influencing its path until the influences which causes the meander result in an oxbow lake after having achieved prominent influences in producing the meander. However, the competing influence of the forces which cause the river to flow eventually supersede the meandering influences and cause the river to again flow adjacent to and omitting the oxbow lake. Wilderness and vegetation likewise reclaim uninhabited areas.

Thus, unassured Human, social, behavioral and physiological requirements, increased levels of Homocysteine, diminished function of PEMT, diminished levels of choline and phosphatidylcholine in cellular membranes, decreasing levels of cellular entities per micrometer, inadequate housing, inadequate nutritional quality and stability, diminished safety, diminished access to health services, health services which do not prioritize existential aspects of physiology and behavior, all result in susceptibility of Humans to influences emitted from other eras, impairing perception, cognition, physiology and behavior to produce detrimental outcomes that benefit competing alternate events, alternate outcomes, interests which endeavor to change the status quo, such that detrimental aspects of the status quo might be destabilized as should occur with advancement, but also causing new detrimental aspects of the status quo to be established and causing massive level of detrimental Human outcomes or tumult to occur in these event pathway displacement dynamics. Pandemics, detrimental artifacts in which Humans may abated vital being with hardly any effort, inadequate resolution of causal factors of detrimental outcomes to produce general level cycles of detriment, collaterally detrimental effect and generational detrimental effect, famines, recessions, and other conditions which could only possibly occur because they are wrongly presumed to be necessary aspects of the Human experience, all emerge as convenient mechanisms by which such displacement dynamics imposed by competing events in different impose their influence on Human outcomes in eras of immediacy.

Thus, as a distribution, allowed challenges to Human existentialism, particularly when these included allowed obtainment of benefit from exhibition of detrimental Human outcomes, allow interactions through distance, space and time, to enable spooky action at a distance within Human events and civilizations, much like experimental management of quantum entanglement between an incipient and subsequent relationship that shares a particle is able to cause hidden results of a relationship in an antecedent instance to, as a distribution, to be changed by controlled changes made in a subsequent relationship. However, in Human events, existential level Human inadequacy lessens the existential nature of vital being, physiology, cognition, and civilizations which seems to inherently cause the paths of ordered events which produce the status quo to become to be likewise challenged by competing Human events and competing ordered paths of events that compete with the status quo. Thus, like a civilization constructed on the bends of a meander, civilizations must rapidly advance in assuring human priority, understanding of the nature the hydrological system, and advance toward sustainable agility to continue benefiting from the flow of such a river, thereby preventing becoming isolated within an oxbow lake as the sustaining waters of the river potentially omit them.

Thus, outcomes which seem to be the result of system workers, and outcomes of Humans, all are the result of systemically imposed or systemically unalleviated inadequacies which systems have

been incipient produced to understand, prevent, filter out through training and awareness or during worker selection and monitoring, or empirically understand such that the causal factors of diminished outcomes are managed or mitigated. The generational exhibition of diminished Human outcomes, thus, were not intended to be exhibited, and when understanding becomes adequate where not intended to persist, such that allowed exhibition of such collateral and generational detriment becomes a mechanism of control by which interest, influences and competing events in other eras might impose control over the status quo and bend Human outcomes to often less than insightful, less than accurate, or openly contrived opinion, cognition and psyche. These can cloud, impair or dissociation decisions, outcomes and cyclic exhibition of Human outcomes from incipient nuances of reason, such as Life, Liberty and the Pursuit of Happiness or from other incipient, empirical impetuses including the now known incipient impetus for the construction of Independence which was a vehement decrying of involuntary servitude, although such a decrying of involuntary servitude became diminished as the Silver Frame of the Constitution and systems of civilization were constructed through filtering feedback of a consensus required to sustain unity during synthesis of civilization level systemic foundations. The unfortunate context of attributing diminished outcomes to Humans is that detrimental outcomes can be produced on demand by Roemer's Dynamics which are systemic nuances that shape Human outcomes to the priorities of systems. Similarly the attributing of outcomes to humans diminishes the priority of analysis, understanding, ascertaining empirically causal factors, contexts, artifacts, conditions, metabolic factors, physiological factors, environmental factors, and essential enabling factors otherwise.

The result is that the allowed exhibition of diminishing influences, factors, conditions, artifacts, and influences results in cyclic exhibition of outcomes while attributing causality to Humans along with exhibition of sanctions wrongly acculturate Humans into believing that the detrimental outcomes which they experience as symptoms of inadequacy are somehow inherently inhibited by or are derived from Human nature. These result in imposed exhibition of detrimental outcomes and programmed expectation and presumption that detrimental outcomes have to occur and are to occur. The result has been that instead of moving systemic perspective of these factors as immunity outward to alleviate the way I which sanctions cause generational and collateral detriment, social constructs and sanctions have been moved into systems to affect systems workers, allowing systems and systemic allowed exhibition of human susceptibility as well as systemic exhibition of detrimental aspects of the status quo, all to escape improvement at the juncture of change which are most capable of improving human outcomes. The status quo, thus, seems to focus on producing social constructs and decisions that attribute causality to humans for outcomes produced by allowed human inadequacy, increasingly complicating the roles performed by systems workers, increasing risk among systems worker, and imposing sanctions as manner of achieving an equity that cannot be reasonably attained after detrimental outcomes have occurred, while the causal factors and empirically causal factors are allowed to produce massive levels of diminished Human outcomes.

A useful perspective in this instance is that increased levels of homocysteine increase risk of all causes of unassured vital being, including increasing the risk of being a victim resultant of diminished Huma outcomes. This context presents why entities, groups and systems branches

which synthesize social constructs are pervasively challenged in producing effective social constructs and programs, because such systems are compelled to favor victims, but may inadequately perceive victimizers as the victims which all Humans experiencing detrimental outcomes are. Thus, such entities and branches must be equipped with data science and analytics along the numerous correlates presented in this compendium of research. Policies, programs and decisions, likewise, should be the result competently implement applied policy and program analysis, development, feedback and continuous monitoring as well as continuous improvement. Every role making decisions that affect Human outcomes should have Data Science implemented that allows individual, group, branch and systems levels understanding of the affect of their activity, decisions, social constructs and outcomes. Data sciences observers should be shared among roles, branches, and systems, along with exhibition of Program office that specifically correlates and analyzes, as well as correlates activity, affects, decisions, and risks, as well as opportunities produced by the great work that civilizations do in observing, capturing, reporting, intervening and managing Human outcomes. 7 ore levels of causal factors should be included in all outcomes, correlates, mechanistic links, dualities, tuples or other concluding observations in this context of Data Science. A useful way to begin can include having Data Science analysis participate in meetings, analysis and other activity, with an incipient directive of finding outcomes, conditions, contexts or complexities that should be explained, should be understood, or are not understood, as well as to dispel inaccurate opinion, conjecture, derived conclusions, misinformation, or contexts in which Humans are attributed outcomes while causal factors are allowed to persist, particularly when such allowed persistence of causal factors and attributing o causality to Humans results in humans, vital being, liberty, care quality, health or wellness to become dispensable. Analytics enabled electronic health information system which managed outcomes toward optimal are useful examples of how such data science might be implemented at multiple levels.

Consider that if one peruses causal linkages in any Human outcome, it can be clearly ascertained that Humans have been foundationally caused to incur the outcomes they incur. Pervasively, systems or of most substantial causality and when those outcomes occur more than singularly, as pervasive outcomes are cyclical and generationally occurring, it is not only systems, but both allowed continued exhibition of such outcomes and allowed obtainment of benefit from diminished human outcomes which are integrally involved. However, like all systems of the universe, every system has a propensity, correlative to duration, regardless of if such characterization occurs resultant of duration of exhibition or if such characteristic occurs because of increased duration of the exhibition of such system, to prioritize itself over its incipient circumstance, incipient utility, and over Humanity. This phenomenon results in exhibition of the status quo in which particular contexts emerge in which benefit is allowed to be obtained from sustaining exhibition of diminished Human outcomes and allowing benefit to be obtained from exhibition of diminished Human outcomes. These can result in attributing causality for outcomes to individuals in away that diminishes the priority of ascertaining empirical causal or enabling artifacts, contexts, conditions, statuses, metabolic indicators, or other shaping influences and correlates. These can also result in producing social constructs and priorities, as well as decisions that do not prevent detrimental outcomes, but allow such outcomes to occur while benefit is obtained from the exhibition of diminished Human outcomes. These promote

the acculturation among populations that such diminished outcomes are inherently potentiated and are aspects of Human nature, both of which are falsely objective conclusions. These propensities must be managed, diminished and mitigated in assurance of Human priority.

These aspects of systems are ubiquitously potentiated in systems of the Universe, thus, enabling Humans to consider such propensities objectively, particularly since hardly any Human exhibiting vital being now has been integral to either the synthesis of systems of civilizations or particularly have not been involved in how such dynamics are potentiated in systems of the universes pervasively.

The practical perspective of this is that biology, development, and changes to human characteristics over time are produced by course improvement, directional advisement, interactive changes, and obtaining direction from the creative forces of the Universes, particularly those which favor humanity and have allowed Humanity to emerge, persist and advance. Hydride and hydridic character are each included. Hydride is the energy that fuels the stars of the Universes. What is most important is that sustaining existential aspects of physiology, including the foundational aspects of biological compartmentalization and foundational aspects of tissue existentialism, the ability to mine, translate, intensive and capture hydridic fields is diminished, such cognitive capacitance is diminished, susceptibility to other fields is enhanced, and such that a systems interactivity context emerges in which other exogenous systems begin to impose their priority, changing biology to the potentiate the outcomes of other competing systems in the biome, in antecedent eras, in future eras and in eras of immediacy.

Time advances in minuscule or expansively correlative differences in condition, as well as independently for any entities exhibiting differences in velocity or mass, $e = mc^2$. The popular notion is that time in a defined space may progress more slowly in a defined space with an increased comparative velocity because light has to move across a longer distance to arrive at the same receive point because the increased velocity produces a disparity in which emitted light within a context of increased velocity must move, bend or have an angled trajectory to catch up to the receive point. This change in time is regarded as change to the characteristics of light that are changed by motion. However, this perspective may be enhanced by considering that time is a requisite dimension of any factor in the Universe. Humans perceive time because systems of civilization pervasively produce adjudicative interactions that reduce Human events to an outcome, although the Universes and Human civilizations were most likely not intended to have many adjudicative contexts which result in outcomes imposed upon them, as the context of nanoplasm, defined space and metabolic interactions in defined space clearly suggest.

Similarly, differences in the advancement or characteristics of time seem to be unimportant except for order or events and except for interactions imposed upon 'humans in civilizations. Humans, for instance, near the polar regions of the Earth move at a much slower velocity than Humans near the equator, because the thousands of miles that the Earth must move at the equator each day compares to the hundreds of miles or several miles that the Earth must move as one nears the poles. There are differences in Human outcomes in such regard, but the differences in average lifespan are often mitigated by environment, access to resources, and lifestyle. Humans

interact in ways that synchronize time, such as through communications, sharing of information and utilizing time keeping devices that synchronize to similar indexing factors, along with synchronizing of time pieces manually. However, the systems of dynamics seem to manage such disparity in time by providing encompassing contexts such as being upon the Earth, and emergence of evenings and mornings, all in a way that synchronizes contexts that include factors with different mass and imprecisely synchronized velocity.

Time can be considered to exist in a dimension into which geodesics or curvature is introduced and it is this dimension that exhibits curvature when differences in mass and velocity are exhibited by different factors. Two individuals traveling at massively different velocities and different mass, thus, may interact with one another because the disparity is move to the dimension of time and a geodesic or curvature is exhibited in a way that enables the two distinct factors to interact. Linear travel in the same direction or linear travel in the opposite direction introduces an increasingly ascertainable effect because interaction or communication eventually becomes affected by distance. However, traveling in circles that result in a common shared locus in such circular pattern potentiates limited level of distance between such factor and even potentiates exhibition of nearness between such objects at the start of differential characteristics of travel. The differences in time, thus, occur as geodesics. These describe mundane aspects of interactions through distance, location, space and time.

Systems of civilization have what is called polynomial time and nonpolynomial time in which polynomial time is the exhibition of resources, energy, and time applied in analyzing, resolving or exploring any problem such that only reasonable, practical, and sustainable levels of these are applied, particularly when considering other tasks, problems, or issues or explorative contexts are considered. Nonpolynomial time occurs when extensive, excessive, impractical, or particularly expansive or overly focused application of resources occurs, resulting in objective consideration becoming, instead, deterministic influence in which systems themselves, objectives of systems, or presumptions all begin to become causal to outcomes that occur in antecedent eras, future eras and eras of immediacy. These conditions can be particularly influential and complex when benefit, economic, political, social or other, is allowed to be obtained from the exhibition of diminished human outcomes.

These describe how systems benefiting from diminished human outcomes in any way, including obtaining economic benefit, political or social influence, as well as in confirmation of inaccurate opinion or antiquated perspective, may eventually begin to become integral enablers or integrally causal to the outcomes which such system may intervene, interact, manage or regulate. This context is particularly in regard to fee for service payment in which are is provided on the basis of exhibition of diminished Human outcomes, as well as logically similar contexts otherwise. Humans were intended to interact with antecedent and future eras, and have susceptibilities that emerge when inadequate satisfaction of human and social requirements occur, such that how these factors change physiological and behavioral outcomes were not intended to produce stereotypes or provide substrate for systemic activity, but were intended to provide information about intangible or inadequately understood influences to physiology, perception, cognition, and behavior. Civilizations were intended to utilize differences in Human outcomes to improv e

understanding, knowledge, and awareness, as well as develop capabilities to more adequately assure Human, social, behavioral and physiological requirements among increasingly encompassing aspects of Humanity.

Hardly any Human now exhibiting vital being has derived the nuances of systems that may be causing the very outcomes for which such system may be instrumented, such that there should be hardly any reluctance to implement essential nuances of change or improvement in this regard. Likewise, it should be apriority for the synthesis of decisions and social constructs that affect human outcomes to alleviate or remove the artifacts, factors and conditions that are essential in causing detriment to workers in systems as well as which may be detrimental Humans in any regard, particularly by linking such decisions, social constructs, care, service and outcomes to empirical aspects of ideals and reason, including assuring that activity in the Silver Frame of systems of civilization are always as empirically as possible resolved to Life, liberty and Pursuit of Happiness, assurance of vital being, opportunity, or other declarations exhibiting empirical or defining utterances which invoke or connect Humanity to the favor of the Universes which have enabled Humanity to emerge, persist and advance along the course of the Human experience.

Importantly, these nuances of spooky action at a distance manifest themselves as what is known as Roemer's dynamics in which it became known in the 1940s that construction of health facilities in an already existing service area typically resulted in commensurate increase in disease, unassured vital being and diminished Human outcomes such that the newly exhibited facilities become occupied or utilized at rates similar to already existing service area. The phenomena came to be described as "A bed made is a bed filled." , and resulted in construction of nationalized health infrastructure in some other nations while in United States the Hill Burton Act and its certificate of needs provisions emerged such that it became required for public funding used in health facility construction to be accompanied by an assessment of needs in the existing service area to be sure that bona fide adequate demand was exhibited. The Hill Burton Act Provisions were relegated to public funding, thus enabling privatized health industry to flourish without such restriction unless restriction to private interests occurred through regional regulation.

Analysis of the structure of estrogens and hormones reveal that hydroxyls perform as loading points for attached molecules or resonant influence that can provide current, while the aromatic rings exhibit a controlling influence to the environment that coordinates molecules and extramolecular space toward galvanizing biological reactivity, interactions and metabolism. However, most remarkably, the offset of the uppermost purine in estrogens and androgens such as testosterone along with the offset of the lowermost ringed hydrocarbons are precisely correlated or even coordinate with the offset the cardiac organ as well as offset of the major digestive organs. The third and fourth transmembrane domains exhibit integration loci for PEMT catalysis that involve Gly98, Gly100, Glu181 and Glu180. Impaired PEMT genomic sequences result in impaired production of VLDL, impaired ability to store fats, reduced adiposity, and increases in glucagon, glucagon receptor, insulin receptor substrate 1 phosphorylation at serine 307 linked to downregulation of insulin receptor I, as well as diminished AMPK, all of which impair typical responses to fat obtainment nutritionally. The

result is that insulin resistance risk is upregulated, gluconeogenesis is upregulated, producing a decision in which increased levels of fat obtainment in this context is prevented from causing adiposity while inadequate fat obtainment results in insulin resistance along gluconeogenic production of glucose. PEMT impaired metabolism can result in particular physiological characteristics typically including inadequate achievement of adiposity.

PEMT is suggested by the literature to exhibit a ping pong mechanism of catalysis which involves S-adenosyl methionine interaction to obtain CH_3 , integration with phosphatidylethanolamine to attach the CH_3 , release of phosphatidylethanolamine as phosphatidylmonomethylethanolamine, such that the lower affinity for phosphatidylethanolamine exhibited by PEMT enables PMME and PDME to have higher affinity, causing PMME and PDME to attach more efficiently and rapidly PEMT as it recovers from each catalytic interaction. However, release of substrate and products by PEMT seems to be only the most obvious of the catalytic function of PEMT, potentiating carbocation and other intramolecular transactions that have the potential to be as important as the ingredients to product or solution which are then packed as substrate, leaving groups, catalytic output otherwise.

PEMT exhibits four spans that permeate hydrophobic regions of cellular membranes.

It is known that phospholipids increase the temperature at which superconductivity occurs, enabling such superconductivity to occur nearer to physiologic thermodynamical 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.

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 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.

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.

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.

Cytosolic NAD^+ density is observed in some literature as about 100 μM , while Mitochondria exhibited about 250 μM , although NADH to NAD^+ to NADPH can be as high as 1 to 1000 in the cytosol with mitochondrial gradient of NADH to NAD^+ at about 1 to 10.

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 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⁺ 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 preventing 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 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.

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 interesting linkage of Agrin to incipient exhibition of fields and capacitance that is developed into the conscious aspects of cognition and being includes, imperatively, an inherent focused preference for hydridic centers to find the most stable configuration in a molecule which is an integral priority of conscious cognitive function and a preference even for less than conscious cognitive function in which metabolic and physiological factors, along with populations in similar regard, endeavor to find or produce stability. Carbocation rearrangements exhibit living characteristics, cause molecules to exhibit living activity along with and against inherent gradients or potentials, while these including focused priority on ascertaining stability in a way that begins at conception.

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 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.

Another version or characteristic of carbocation arrangements include a pattern, Saytzeff's or Zaitsev's axiom in which stability of an alkene is highly potentiated when the alkene is excreted 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 enhance 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 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.

PEMT produces more diverse, more lengthy extent, more polyunsaturated, and increased extent arachidonate characteristics in fatty acids while CDP choline exhibits more diminished extent and more polyunsaturated fatty acid species. PEMT is a substantial contributor of energy metabolism, according to the literature. Digestive fluids produced from membrane phospholipids using MDR2 result in depletion of phosphatidylcholine which has to be replaced by nutritional obtainment of choline or phosphatidylcholine to prevent exacerbating of existential aspects of

cellular and tissue existential status, such that low calorie nutritional regiment linkage to enhanced span of being in experimental conditions can involve diminished catabolism of phosphatidylcholine from diminished digestive processing as well as resulting an greater fraction of retained membrane phosphatidylcholine. PEMT activity is a major contributor of VLDL and increases in levels of VLDL can be produced to coat vasculature to counteract striates such as exhibited in typical stable salt or other striates which scratch and causes bleeding of the vasculature. The increased levels of lipoprotein analyzed in assay includes cholesterol, although cholesterol is integrated into transport proteins known as LDL, VLDL, etc. Because cholesterol is a major component of cellular membranes, estimated above 80 percent and as much as 87 percent, and cholesterol aggregated at the cellular membrane is integrated into the pocket protein of START domains of star proteins in membrane phospholipases such as phosphatidylcholine for shielded transport to subcellular compartments such as the mitochondria where carnitine assisted traversal of cholesterol into the mitochondria flowed by processing by cytochrome p450 scc results in pregnenolone to stimulate steroidogenic hormonal processing, each clearly present that is oxidation, peroxidation or glycation of cholesterol that presents the most substantial risk instead of mere exhibition of cholesterol. Ancient pink Himalayan sea salt supplementally and in replacement of table salt, if organic and natural, can alleviate and prevent complexities and complicated nuances of cholesterol and sodium management, particularly therapeutically.

The interesting linkage of Agrin to incipient exhibition of fields and capacitance that is developed into the conscious aspects of cognition and being includes, imperatively, an inherent focused preference for hydridic centers to find the most stable configuration in a molecule which is an integral priority of conscious cognitive function and a preference even for less than conscious cognitive function in which metabolic and physiological factors, along with populations in similar regard, endeavor to find or produce stability. Carbocation rearrangements exhibit living characteristics, cause molecules to exhibit living activity along with and against inherent gradients or potentials, while these including focused priority on ascertaining stability in a way that begins at conception.

Carbocation rearrangements in molecules exhibit hydridic character that is continuously seeking stability in the intramolecular space which as a foundational characteristic or priority exhibited by Life or living systems. Hydride can be separated from its integral molecules resultant of carbocations in a managed way that controls the fluorescent moment, fluorescence and release of 2 eV-, as well as potentially opening a circuit through energy continuously flows into the hydridic integral molecule and out of the managed macromolecule as hydride continues to be separated from the integral molecule in a carbocation rearrangement. The literature observes that carbocation rearrangements are dynamic not only through molecular structural change, such that this can include interaction of the managed macromolecule with energy fields or influence that resupplies current or energy, addition of atp which causes more current to be available, but also because carbocations try to find the most stable configuration. This dynamic seems to be similar to how conscious capacitance emerges at conception and then beings to respond to influences in the gestational environment or influences in the wider biome, in a way that becomes more complex through phases of development to become the conscious cognitive contexts and less than conscious cognitive context.

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 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.

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 d 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, magnetoelastic 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 pathways involved in hydridic or hydride eluting, mining, transport and application. The hydrides RCo_5 , R_2Co_{17} , $Nd_2Fe_{14}B$, and $RFe_{11}T$ 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.

The following pattern of homocysteine integration into care, thus, emerges.

Acute Care

During acute care, homocysteine should be considered a constitutive causal and participant factor in all pathology.

Inpatient Care

Homocysteine at 10 $\mu\text{m/L}$ or more should be considered for therapy that includes Homocysteine management specifically. Referral for outpatient care should be performed to complete the continuum of care to below 10 $\mu\text{m/L}$, while management to 6 or 7 $\mu\text{m/L}$ can be performed interactively with office visits, with a therapeutic objective to near 3.7 $\mu\text{m/L}$ or lower. Wholistic care and services can be integrated increasingly when care is near and below 10 $\mu\text{m/L}$.

Outpatient Care

Homocysteine above 10 $\mu\text{m/L}$ along with a condition that requires outpatient care, particularly if not improved over the course of outpatient care, should be recommended for inpatient care to management homocysteine below 10 $\mu\text{m/L}$. Homocysteine near 6 or 7 $\mu\text{m/L}$ should be a candidate for therapy with office visits to assist monitoring and prescription as well as nonprescription therapy. This can include wholistic aspects of medicine. The objective such therapy should be near 3.7 $\mu\text{m/L}$. Homocysteine over the course of any health intervention that is at 15 $\mu\text{m/L}$ or higher, regardless of exhibited in the context of an existing health condition, presenting condition, or other an a wellness visit, should result in referral for inpatient, outpatient or specific off visit management of such status, varying with other factors indicative of health homeostasis. Homocysteine, elevated to 10 or particularly at 15 $\mu\text{m/L}$, should be considered constitutive of an adverse health status or adverse health event.

Office Visit

Homocysteine above 10 $\mu\text{M/L}$ along with a condition that requires office visit care, particularly if not improved over the course of office visit care, should be recommended for inpatient care to manage homocysteine below 10 $\mu\text{M/L}$. Homocysteine near 6 or 7 $\mu\text{M/L}$ should be a candidate for therapy with office visits to assist monitoring and prescription as well as nonprescription therapy. This can include wholistic aspects of medicine. The objective such therapy should be near 3.7 $\mu\text{M/L}$. Homocysteine over the course of any health intervention that is at 15 $\mu\text{M/L}$ or higher, regardless of exhibited in the context of an existing health condition, presenting condition, or other an a wellness visit, should result in referral for inpatient, outpatient or specific off visit management of such status, varying with other factors indicative of health homeostasis. Homocysteine, elevated to 10 or particularly at 15 $\mu\text{M/L}$, should be considered constitutive of an adverse health status or adverse health event.

Behavioral Health

A priority for these indicators and how these result in care modality implementation includes the correlation of homocysteine and trimethylaminenoxide with both sudden and emergent, as well as chronic exhibition of detrimental behavioral potential. The objective thus is not only health but managing safety by reducing risk for adverse behavior and adverse health outcomes.

Emergency Medicine

Homocysteine should be considered to be an integral aspect of emergency conditions and sudden adverse health events, as well as chronic conditions and conditions that become increasingly detrimental. Emergency medicine, however, particularly with regard to transport, should be careful to obtain fluids and samples for assay rapidly before instrumentation of stabilization protocols that manage homocysteine.

Level 4 indicators are an aspect of the API. Level 4 indicators are presented immediately following this item.

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.
 1. This suggest 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 Menaquione-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, Indolethylamine 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 Methylpherase, and used toward S-Adenosyl Methionine/Dimethyl Sulfide.

Level 1 indicators represent an API interactive with homocysteine status.

Level 2 indicates an extended API.

However, wholistic therapeutics and the complete group of pharmacological and nonpharmacological therapies, including nutraceutical, naturopathic, traditional, Eastern and other modalities of care, such as nutrition, diet, exercise, environment, behavior, lifestyle, learning, regenerative medicine, and other, all may interact with any level of the API, although direct interaction with this centered platform model is also possible.

Places, organizations, services or systems which people interact with, obtain services from, which affect, or for whom humans perform work, all should have flexibility with such exhibition of homocysteine as an indicator as well as should have incentives that enable compliance with such parameters for homocysteine shaping of care. Imperatively, such parameters may be essential in enhancing productivity, duration of productivity, exclusion of impairment, and decreasing of duration of care and decreasing level of impairment, while also improving outcomes. Although schemes of wellness and healthy industry operational management costs and worker compensation may optimally involved a weekly, biweekly or monthly model that has an assured minimum payment that assures costs coverage along with a margin, each presented in a transparency costs/expense/finance model, and such may include model of worker compensation that includes minimum assured levels adjusted for ancillary contributions, ancillary achievement, active/inactive practice status, acuity adherence and pay for performance quality bonus, the use of homocysteine as an indicator may result in a substantial increase in volume of lower cost, lower complexity, decreased duration, higher volume services that displace the exhibition of more complex, longer duration, higher cost instances of care.

Care pathways in any human system, particularly those which involve the effects of homocysteine, the translational wellness clinical example context, and any of the indicators in

the level 1, Level 2 and Level 4 list of indicators, should include in procedure, systems and protocols only pathways, care plans or paths that lead to improvement and, when possible, optimal human outcomes, including gating mechanisms that focus on assurance of foundational aspects of cellular membrane and tissue level existentialism, but also including gating mechanisms that implement the indicators in assurance of improvement and feedback as to which factors are producing bona fide improvement compared to those which only seem to provide improvement. These improvements should also link indicators to statuses such as housing, food security, nutritional security, emotional stability, safeness of areas in which habitation occurs, access transportation, opportunity access, and optimally assurance of the 1.25 to 1.50 income level for each individual Human being, including vicarious allocation of tis level of benefit to a parental assurer of a another's achievement of this subsistence level.

Managed Care and cost sharing arrangements may have a distribution of such assured minimum costs for a service provider or care entity allocated using a formula that adjusts their contribution to each such entity in accordance with the percentage of covered members or covered subscribers service by the care entity compared to the aggregate population serviced by the covered entity. A monthly or yearly formula for such proportions might be utilized and adjusted yearly or monthly, even though the distribution of funds may be weekly, biweekly or monthly. The importance of this potential model of managed care service emerges to prevent fluctuations in epidemiology and other factors from causing disruption of health services infrastructure and health services access. Also, such a model of cost sharing emerges because managing homocysteine may cause substantial shift from fee for service only payment that is based upon actual emergence of tangible pathology toward proactive management of health services infrastructure that manages detrimental outcomes before they emerge as tangible, high cost, high risk outcomes.

Area health planning and Hill Burton Certificate of Need Programs can be utilized as a model to which acuity analytics can be applied, allowing health providers and facilities to be added according to bona fide demand and allowing changes to ratios of specialists, ratios of facilities, and quality analytics to emerge in ways that assist payer entities and groups to produce innovative ways of payment on a context in which providers, carers, provider entities and carer entities are assured achievement of operational subsistence and individual ranges of assured compensation.

Already, managed care entities have started to shift their funding toward enabling, producing, and sustaining health services provider offices, providers, entities, groups and associations, as well as facilities, particularly to sustain the regulatory cost ratios required for managed care entities.

Regardless of the changed role for managed care, the role of managed care entities and health services reimbursement coverage may optimally be maintained through encounters and claims processes because, importantly, a strongest role of health care coverage entities may be monitoring of human outcomes as well as the known way in which managed care coverage improves human outcomes in a comprehensive group of circumstances, systemic interactions and

interventions, regardless of if managed care coverage is utilized and regardless of if managed care coverage is directly relevant to such outcomes.

A useful way of understanding these modalities of activity and consideration is that these represent a unifying, interactive, shared rhythm for wellness, health, medical and Human service with which all services affecting Humanity might eventually interact with or which all services might eventually link their priorities.

A particularly obvious conclusion that these analyses have been required to consider is that innovation, development, advancement, and particularly, value synthesis and delivery in any one industry within an civilization seems to often require the aggregate contributory and interactive function of a somewhat comprehensive group of industries and function in a civilization.