Translational Wellness Clinical Platform, Clinical API and modular interactive stack

A translationalwellness.com Care Artifact.

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 Citidylylcholine Phosphotransferase, hallway 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 a 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 r lease, inhibition of glucose absorption, trapping of intracellular glucose from absorption or gluconeogenesis int 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." Themedicialbiohemistrypage.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 include 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, Ca2+, 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 CH3 and its hydridic contingent into phospholipids by attaching deriving CH3 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 CH3 that has assumed hydridic character, then attaches the CH3 to the open location of the ethanolamine in phosphatidylethanolamine using adhesion that includes the lone pair of the CH3 becoming shared by the Nitrogen of the phosphatidylethanolamine molecule. This first attachment of PEMT produces PMME, the second attachment of CH3 produces PDME, and the third attachment of CH3 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 word homocysteine is a euphemism and is misleading, such that monocysteine should always be regarded as a euphemism for the physiologically precise word homocysteine.

The defining CH3 that is added to methionine to produce methionine from homocysteine occurs in the synthesis of methionine from homocysteine by BHMT, BHMT2, Methionine Synthase, as well as thetin homocysteine methylpherase and even the synthesis of S adenosyl methionine in its complete structure by INMT which interconverts S Adenosyl Homocysteine directly into s adenosyl methionine, while also S adenosyl homocysteine hydrolase bidirectionally produces homocysteine and s adenosyl homocysteine according nad+/nadh balance, although TTMT production of trimethylsulfonium and 5 methyltetrahydrofolate enables both thetin homocysteine methylpherase with trimethylsulfonium substrate and enables methionine synthase with 5 methyltetrahydrofolate. BHMT uses trimethylglycine while BHMT2 utilizes S methylmethionine sulfonium to produce Methionine from Homocysteine. Dimethylthetin and trimethylsulfonium are known to decrease homocysteine linearly in a graph of such depletion, although the transsulfuration pathway also is known to irreversibly deplete homocysteine toward Cystathionione, Cystine, glutathionine, cystine and HS molecules. Serine and homocysteine are used by cystathionine beta synthase to produce H20 and cystathionine while cystathionine gamma lyase utilizes H20 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 1diphosphate, 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 form 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.

Methyl shift involves a similar movement of Hydrogen from the 1st Carbon to the second Carbon in molecular structure, exhibiting movement of a hydrogen from a methyl group with 3 CH3, and resulting movement of the hydrogen across a hydrogen adhesion to the second carbon in the structure. Compared to hydride shift or primary carbocation in which only two CH3 are exhibited along with a hydrogen connected to the originating carbon, the secondary carbocation exhibited in methyl shift exhibits 3 CH3 moieties connected to the originating Carbon. The result of the secondary carbon is a CH3 and two Hydrogens connected to the Carbon that receives the shifted Hydrogen. The result of primary carbocation, in contrast, results in three hydrogens attached to the carbon that receives the shifted hydrogen. Secondary carbocations are typically more stable than primary carbocations, and phenyl carbocations can occur to enhance stability even more substantially, while generally, resonances or intermolecular influences are considered to be typically more stabilizing for carbocations. The delocalization of the Cationic polarization enhances stability, a benefit for resonance such as in CH3 and Choline once the nitrogen of phosphatidylethanolamine has received 3 CH3 to become choline within phosphatidylcholine. Tricyclo Propane carbocation, illustratively, is considered to be a most stable carbocation.

It is also important to know that the electron transport pathway of oxidative phosphorylation exhibits freeing of Hydride from NADH to produce 2 eV- of energy as freed fluorescent influence that supplies energy to the electron transport pathway which equalizes the utilization of energy through process democratization among each of its phases, utilizing about 58 percent of such energy to produce ATP, such that ATP receives about 42 percent of such freed energy by integrating such energy into the oxonium exhibited between its phosphate groups. The attachment of ATP to Methionine results integration of this hydridic source of energy into S adenosyl methionine, resulting in carbocation rearrangement that Ionizes the Sulfur of Methionine, changing sulfur to a Cationic or positively polarized Sulfur. Information. Carbocation. IT JEE Study Material. JEE. byjus.com website. Information. “Selenium.” Chem Soc Rev. Volume 42. Number 23. Pages 8870 to 8894.

Some of the literature presents phosphatidylcholine and phosphatidylethanolamine with the same structure near the nitrogen, with three open locations at the nitrogen or sometimes with three hydrogenase linked to the nitrogen, while in those depictions of the nitrogen in choline and the nitrogen in phosphatidylethanolamine the oxygen between the second carbon from the nitrogen and the other aspects of the these two molecules is presented as negative polarized or an oxygen anion. These suggest that either CH3 are not structurally attached to the Nitrogen, resulting in no change to the polarization of the Oxygen, or, this suggests that when 3 CH3 molecules are linked to the Nitrogen, a carbocation or shift occurs at hydride, methyl or other levels, resulting in change in polarization of this linking oxygen.

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, eicosotriene, and leukotriene pathology cascades as

well as many other detrimental factors including lipoxygenases. However, not all prostaglandins enable cascading detriment, and prostanoids, poxytrins, elovanoids, (R) - Resolvins, (S) - Resolvins, are all considered linked to specialized Pro - Resolvin Mediators which resolve pathogenic cascades. Versions of these mediators include sulfido - peptide conjugated mediators which, like other versions of these mediators, methylthioglycolic acid and other factors, acquire specialized characteristics in the acute phase or cascading pathology microenvironment, derived from the molecular, ionic, atom and quantum characteristics of such microenvironment, very much being adaptive in ways that are similar to molecular therapeutics. These mediators are searched for, screened, tested and used to produce therapeutics.

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 occur in numerous redox pathways but particularly involving NAD+/NADH, NADP+/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, CH3 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, CH3 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 methyltetrhydrofolate, 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 grop 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 confirmingly, 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, inflammation enabled 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 reseeding 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 are 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 less than 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 lactase 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 um/L produced a 500 to 1 difference in the instances unassured vital being over decade of observation, favoring those with homocysteine less than 7 um/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 um/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 thetin homocysteine methylpherase to prevent intramolecular disulfide links within thetin homocysteine methylpherase. Intramolecular links within thetin homocysteine methylpherase occur when inadequate sulfur is exhibited in blood and in the microenvironment, such that this enzymes role as one of the most abundant anatomical enzymes becomes ablated and the enzyme becomes packed and stored away in tissues in a gelatinous phase that is bereft of catalytic activity.

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 form microbiology to social systems has not been effective at improving

human outcome, while causing somewhat massive collateral and generational effects, although clearly this contexts seems to be providing information instead of being utilized as a rational bona fide mechanisms 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 Cytochodrome 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 of 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 s 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 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 Ca2+ 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, SP! 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 causses 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 underconsidred pathway for both vascular deterioration and participation in particular nuances of vascular repair such as with Estradiol instrumentation.

GSK3B inhibitors which are invoked in S1P receptor pathways and methylsulfonylmethane both are used in oncology therapy. Methylsulfonylmethane is used in breast oncology and prostate oncology, bridging the disparity between estrones and androgens. Androgen upregulation is a causal factor in prostate oncology while oncology that does not exhibit upregulation of androgens are known to respond anyway to androgen inhibition therapy. GSK3B inhibition surmounts and prevents endothelial to mesenchymal transitions and as well as surmounts and prevents chemoresistance in oncology of the breast while GSK3B is known inhibitor of upregulated androgens exhibited in prostate oncology. Moreover, the patterned characteristics of oncology generally and somewhat inclusively involve increased catalytic activity in particular cellular lineages which are redirected as bona fide catalytic activity, mitosis or differentiation, although kinase activity upregulation is typically linked to each of these. Catalytic activity can be upregulated by nutritional obtainment, such as chREBP activation which can power through choline deficiency enabled cellular cycle pause, although catabolism of cellular membranes by phospholipase can mimic choline availability in this regard, and although ankyrin repeats in

molecules are also able to deactivate P53 impose cellular cycle pause, such that also in this regard postprandial activation of protein kinase C may also upregulate kinase phosphorylation cascades. Although T lymphocyte activation of cellular entities can involve such phosphorylation cascade, another Kinase, the protein tyrosine kinase ret+, is also known to be involved phosphorylation cascades that lead to increased catalytic activity redirected as differentiation, mitosis, or bona fide catalytic activity.

There should not be a confusing of ret+ with RET that is the inverse of NADH release in the electron transport pathway in which Hydride is released to sue 58 percent of hydridic energy in loadbalanced energy utilization in a democratized process during oxidative phosphorylation, while 42 percent of hydridic derived energy is integrated between the phosphate groups of the ATP which is the product of oxidative phosphorylation. RET involves reintegration of hydride into NADH at the incipient phases of oxidative phosphorylation instead of hydride being abdicated from NADH to produce free energy as florescent energy that molecules, particles and processes can absorb, interact with, entered excited status as result of, or experience spin as a result of. However, differently, ret+ is a transmembrane protein tyrosine kinase that is able to receive mitogenic signals from the extracellular environment resulting in division, mitosis, intracellular phosphorylation cascade, or other change representing redirectable catalytic potential. Ret+ upregulation has been observed in particular oncology and its catalytic activity has been observed as a differential factor in some such oncology, including NSCLC, while ret+ genetic polymorphism has been linked with EGFR polymorphism, MET amplifications, both without requirement of combustive use of detrimental tobacco products. Importantly, in order of therapeutic efficacy, Cabozantinib, vandetanib, Lenvatinib, selpercatinib, and pralsetinib have been tested or indicated for management of ret+. “Ret Inhibitors.” ‘Oncologies’(Basel). Volume 13. Number 17. Pages 4415. 9th Month, 2021. Information. “GSK3B.” Breast ‘Oncology’ Research. Volume 21. Number 1. Page 37. 3rd Month, 7th Day, 2019. Information. Int J Mol Med. Volume 28. Number 1. Pages 95 to 100. 7TH Month, 2011. Information. PLoS. ONE. Volume 7. Number 4. Article e33361. 4th Month, 2nd Day, 2012. Information. “GSK.” ‘Oncology’ Letters. Volume 380. Number 2. Pages 384 to 392. Pages 384 to 392. October 1, 2016.

The literature presents configuration of propanoic acid, hexanoic acid and cyclohexane carboxylic acid, as natural compounds, that exceeded the integration free energies of the selpercatanib and vandetanib. Information. “Ret Tyrosine Kinase.” RSC Adv. Volume 12. Number 2. Pages 1194 to 1207. December 22, 2021.

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 repair to adequate plasticity by managing trimethylaminenoxide 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 change levels of aggregate Homocysteine. The quartiles established were group as less than 9.94, less than 12.7 exclusive of less than 9.94, less than 16.8 exclusive of less than 12.7, and greater than or equal to 16.8 um/L, which are interesting because objective consideration of homocysteine in the translaitonalwellness clinical platform clearly observes that at 15 um/L without any symptomology and at 10 um/L with symptomology, these patients are pervasively exhibiting substantial risk for sudden adverse health events, sudden adverse behavior or are within a pathophysiological status that is leading to an adverse diminished outcomes.

The study followed up with patients at 3 months and 1 year after admission. Resultantly, at 3 months 64 of 594 participants had experience unassured vital being, 37 had recidivist ischemia, 22 were not included in follow up, resulting only 471 participants being reviewed while subsequent to this 3 month review, 48 participants experienced unassured vital being, 44 had experienced recidivist ischemia, and 40 were not included in the review at 1 year. 339 participants were reviewed at 1 year. The study concluded that homocysteine was not correlated with functional outcome among the 339 participants, although homocysteine is known to be direct causal correlated factor in detrimental behavior, victimization, becoming a victim of victimization, accidents, disease, detrimental behavior resulting in deprivation of liberty, disease and all of the reason by 594 participants might be reduced to 339 participants.

The NIH scale for stroke, however, was correlated with functional outcome. Exploration of the NIH stroke scale revealed 1A as being Level of Consciousness Responsiveness, which is interesting because the Criteria for LOC 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 is it unlikely that this context of pathology is any different than the diverse spectra of diminished physiology that occurs when PEMT catalysis is downregulated. Temporal analysis of cholesterol changes informed, also, changes in homocysteine over the same time, confirming that PEMT upregulated production of VLDL is not a likely source of the increased levels of cholesterol, in both hypothyroidism and hyperthyroidism, which involves increased levels of homocysteine which is an allosteric inhibitor of PEMT.

The study of thyroid status included assay of study parameters at baseline in which homocysteine, folate, cholesterol and creatine were higher in hyperthyroidism compared to lower

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

However, during therapy, there was a substantial decrease in homocysteine, creatinine and cholesterol among hyperthyroid patients, suggesting that renal clearance or methionine synthase processing involving these factors, as we;; as other pathways of homocysteine processing or each of these were improved. Serum folate decreased while also cobalamin was unchanged, more strongly suggesting that both renal clearance, methionine synthase processes or other pathways of homocysteine recycling were improved. However, in hyperthyroidism, typical therapeutic instrumentation resulted in the same changes except that both folate and methionine processing were unchanged, suggesting that these improvements involved alternative processing pathways for homocysteine and improvement renal clearance, while changes in s adenosyl homocysteine synthase or changes to methionine synthase activity did not occur during therapy for hypothyroidism. These observations are qualified by the observation that vast aspects of therapeutics increase homocysteine because of methyl group attribution involved in detoxifying therapeutics as well as exhibition of prodrugs which require methyl groups to become activated. Information.

These confirm inoculation processes linked to PEMT inhibition, as well as canonical patterns of cellular membrane existential inadequacy, cellular entity per micrometer of tissue existential inadequacy, anomaly in homocysteine processing, homocysteine, B Vitamin, and general spectra of factors linked to these, all as causal factors in thyroid dysfunction, including hormone or steroidogenesis pathways that included star proteins, phosphatidylcholine adequacy, transport of cholesterol from cellular membranes to the mitochondria by star proteins of start domains in phospholipids such as phosphatidylcholine, resulting transfer of cholesterol to the mitochondria where cytochrome p450 scc enables synthesis of pregnenolone to begin processing of hormones systemically.

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 PINK! Into the outer or inner mitochondrial membrane while PARKIN integrates with PINK1 in the outer mitochondrial membrane, while the mitochondrial directing sequence, MTS, that directsPINK1 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 PINK1accumulated 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, Ca2+, phosphatidylethanolamine and other factors are exchanged, while phosphatidylserine, Ca2+, 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 isdefici9enty, 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 important 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. Trimehylaminenoxide 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 trimethylaminenoxide is a major status change that occurs with dialysis although clearance of timrethylaminenoxide in dialysis is typically comparable by percentage or fraction to the creatinine levels resultant of dialysis. The li9terature observes these in a study of limitations tons to dialysis clearance of excretion susceptible metabolities sch 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, trimethylaminenoxide, asymmetrical dimethylarginine, symmetrical dimethylarginine, B12, Folate, al, 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.

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 opthalmoplegia. 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 cholineacetyltranseferase 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 Biophsy 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 phosphatidyldimethylethanolamine 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 require d 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. Volume49. 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 variated 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 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 individua, 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 hat 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 = mc2. 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 CH3, integration with phosphatidylethanolamine to attach the CH3, 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 import 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 rations 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 uM, while Mitochondria exhibited about 250 uM, although NADH to NAD+ to NADH can be as high as 1 to 1000 in the cytosol with mitochondrial gradient of NADH to NAD+ at about 1 to 10.

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, preventied from being diminished and rescued. These might challenge heretofore exhibited opinion in this regard. Cytosolic NAD+ density is observed in some literature as about 100 uM, while Mitochondria exhibited about 250 uM, although NADH to NAD+ to NADH can be as high as 1 to 1000 in the cytosol with mitochondrial gradient of NADH to NAD+ at about 1 to 10.

Managed NAD+/NADH ratio is widely stabilizing and 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.

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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 a alkene is highly potentiated when the alkene is excrete from a molecular interaction involving carbocation of from a carbon atom with low number of hydrogens. These likely are reflected in 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 n 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 stables status. This suggests that carbocations can result abdication of a hydride in the intramolecular space that results in fluorescence and release of free energy. This suggest 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, magnetoeleastic properties, and magnetic order occur along with RKKY interactions in magnetism. 3d metals Mn, Fe, Ni and Co are known to participate in biologically active molecular and hydridic interactions, provide one context of what may be other molecules or factors that are sequestered into biological compartments, biotic phases and organic phases by inorganic to organic phase transfer, abiotic to biotic phase transfer and eluting activity perform b pathways involved in hydridic or hydride eluting, mining, transport and application. The hydrides RCo5, R2Co17, Nd2Fe14B, and RFe11T have emerged as extraordinarily applicable to high performance, highly efficient Permanent Magnet application for substrate bereft, clean, nonpolluting, indefinitely sustainable energy production that provides indefinite, modular and extraordinary energy to form factor size energy for all manner of application. Information. Magnetism of Hydrides in Handbook of Magnetic Materials. Volume 17. Pages 293 to 456. 2007.

Magnetism in this context explains how the background characteristics of the extracellular environment are changed by capacitant cellular entities and capacitant subcellular compartments. These suggest that intracellular and systemic energy production and recycling, as well as energy

sustainment may be occurring as an aspect of physiology and that it is a deterioration of rare earth element, mineral and other nutrient obtainment that destabilizes the endogenous permanent magnet energy homeostasis, superconductivity which translates this source of energy through assurance of foundational nuances of cellular existential characteristics and tissue existential characteristics, into subcompartment, cellular compartment, and tissues, and anatomy level traps that sustain physiology. However, the civilization level and biological microenvironment level exhibition of magnetism as a manner of producing sustainable energy seems to emerge amid a context of universes levels fields which physiological compartmentalization seems to be trapping, even at conception, to produce physiological capacitance that is response to external influence and eventually develops to exhibit interactivity, more complexity and ability to be intermediated by conscious cognitive cognition emerging from brainstem intermediation and into areas of the neurological context involved in enhanced conditioning and enhanced control.

Phospholipids are affected by nano level factors and exhibit nano level change along with imposing nano level or greater influence that it includes structure and thermodynamics, resulting in phases that emerge as structure moves through Botlzmann 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 um/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 um/L, while management to 6 or 7 um/L can be performed interactively with office visits, with a therapeutic objective to near 3.7 um/L or lower. Wholistic care and services can be integrated increasingly when care is near and below 10 um/L.

Outpatient Care

Homocysteine above 10 um/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 um/L. Homocysteine near 6 or 7 um/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 um/L. Homocysteine over the course of any health intervention that is at 15 um/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 um/L, should be considered constitutive of an adverse health status or adverse health event.

Office Visit

Homocysteine above 10 um/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 um/L. Homocysteine near 6 or 7 um/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 um/L. Homocysteine over the course of any health intervention that is at 15 um/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 um/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 PPARy-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 Methylpherase 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 tRNAMet from ATP, L – methionine and tRNAMet. 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.