Certificate of Need Health Resources Planning Areas are mentioned to counteract roemer's dynamics, stabilize Certificate of Need Health Resources Planning, and support Narrow Networks which counteract roemer's dynamics, utilizing systems and social institutions in a wider context, particular through shared Human outcomes optimization assurance priorities. Services performed in health Resources Planning Areas and Certificate of Need Health Resources Planning Areas to abate and counteract any other than beneficial correlations when increase in health resources or increase in health facility resources occurs including changes in ratios of health resources when compared to population levels.

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

Methionine, possibly a unique fraction of available methionine derived from recycling of monomethylated cysteine, methionine synthetase now known s adenosyl methionine synthetase integration of ATP into Methionine to causes the canonical activation of the methylene bridge in methionine in which a strong electron withdrawing Group attaches. Integrates or interacts with a methylene bridge to cause the methylene bridge to sequester hydride to complete its trio of hydrogen that seems to include only two hydrogens in its inactive status, but is either aromatically, virtually, or otherwise stabilized by potentials such as current in the environment as 2 eV-, eV-, fluorescent energy, or hydride in adjacent structures. The attachment of atp by methionine synthetase, or s-adenosyl methionine synthase as it is now known, sequesters an electron from the sulphone hydridic character, resulting in cationic or positive polarity. The specific literature characterizes Hydrogen with 1 negatively polarized electron as e- and 1 proton, molecular hydrogen as two of these, and hydride as H- that is constituted of 1 Proton and 2 negatively polarized electrons as 2e-. Hydride is, materially, electron reduced hydrogen, because education is constituted of receiving and oxidation involves abdication or release of material. Activation of the methylene bridge by strong electron withdrawers materializes the hydride from its performance as an aether or its aether characteristics that speckle or weave hydride in areas, space, atmosphere, material, tissues and physiology. The materialization of hydride as an emitted, abdicated, transferred, materialized from environment, delivered by molecular process, or otherwise exhibited electron is similar to how an electron has superposition multiplicity until a photon causes electrons as material orbiting orbitals in energy levels with only positional probability to collapse into an exhibition of its particle characteristics, although it’s other multiplicities such as wave function, energy function, and others are not abdicated.

Ionization of sulfur enhances the enzyme s - adenosyl methionine function as an enzyme. Phosphatidylethanolamine methyltransferase I of endoplasmic reticula, phosphatidylethanolamine methyltransferase III, and Phosphatidylethanolamine methyltransferase II of the mitochondrial associated membrane shared between hundreds of mitochondria and the endoplasmic reticula transfer CH3 which is a hydride primed or hydride integrated methylene bridge that pump primes or jump starts the hydridic current pipeline and trains strong electron withdrawing groups to exhibit homologous current sequestration by getting such activities started. Specifically, the lone pair electron configuration of CH3 and CH3 itself is removed from s -adenosyl methionine to the Nitrogen of Phosphatidylethanolamine in three successive transactions without changing the polarization or ionization of such Nitrogen and without changing the oxonium of the phosphate group, particularly because methylene divinyl patterns in fatty acids that are attached to the sn-1 location through methylene bridge and an oxygen, all result in efficient current management throughout the molecular structure. Methionine abdicated hydridic character transferred by PEMT must also be considered because methionine function enabled by methylene bridge activity is involved in tertiary and quaternary structure of more than one-third of all known proteins including tens of thousands of proteins which exhibit 10 or methionine methylene bridge intramolecular links, including many which exhibit hydridic and methylene character as much as or prevalent to exhibition hydrophobic character typical of methionine.

CH3 and its constitutive hydride, and also phenyl moieties and tricyclopropane propane exhibit carbocation rearrangements that move hydridic centers and distribute hydridic character in ways that include resonant influence that stabilizes carbocation experiencing molecules. Electron Transport pathway of oxidative phosphorylation freeing of hydride from NADH as 2eV- and as fluorescent influence along with utilization of as much as about 58 percent of such energy to fund evenly distributed utilization by the different phases of the electron transport pathway, resulting in integration of about 42 percent of such energy into the oxonium exhibited between the phosphate groups of ATP or Adenosine Triphosphate. The attachment of ATP to Methionine, enhances the hydridic character of methionine which exhibits a methyl group with likely experienced carbocation rearrangements, hydride shift and methyl group shift which are integral stability enhancers for carbocation experiencing molecules.

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

Amide translation into Nicotinic Acid Adenine Dinucleotide, adenylation of Nicotinic Acid Adenine Dinucleotide, followed by ATP and Mg2+ enablement of synthesis of NAD+ and AMP

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

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

Sulfur and Methyl Group supplementation to metabolize hormone and glucocorticoid factors, along sustainment of methyltransferases that integrate CH3 into phospholipids instead of freeing CH3 from management of homeostasis, resulting in integration of Hydride into cellular membranes, increase density of phospholipids in cellular membranes, increase number of cellular entities per micrometer of tissue, and enable systemic ph of near between 7.2 and 7.6 that is involved in assuring consciousness, cognitive function, and vital being. Water or H2O is essential because it assures that intramolecular and intermolecular interactions occur with intended and optimal throughput, velocity and consistency, as well as enables particular molecular phenomenon, including hydridic, hydrogen, hydrophobic, hydrophilic, and particularly including methionine and methionine carbocation occur in physiology. Clean, filtrated and sometimes supplemented water, can substantially enable physiology while betaine and other factors are known to stabilize the quaternary structure of biologically active molecules by performing as osmotic assurers of the shape, twist and writhe that typifies the interaction of biologically active molecules with living structures, tissues, glands, organs and anatomy.

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

Particularly, efficient and agile management of sulfur carrying amino acids that have methylene bridges toward recycling into methylated versions with subsequent adenylation, carbocation, and Ionization of its sulfur, and alternative transsulfuration in which methylene bridges are changed towards cystathionine, alpha ketobutyrate, cysteine and glutathione, although methylene bridges in cysteines qualify cysteines for inclusion in methylene bridge sulfur carrying amino acid metabolism. Methylene bridges promote strong energy potentials used in biology such as

participating in hydride, methyl, phenyl shifts , such as in s adenosyl methionine in which, instead of freeing 2eV- and fluorescent influence when hydride is oxidized or freed from NAD+ or NADPH, shift of hydridic character occurs in in carbocation rearranges in a controlled way preventing abdication of the hydride while using the 2eV-, ionizing the sulfur, and exciting the microenvironment which includes excitement of the outer incomplete energy levels and orbitals that are shared by all atoms of the universe, or metabolism, which is an antonym for nanoplasm or the empirical representation of any material or group of atoms in a defined space.

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

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

Such hydridic character packed into ATP can be donated to molecule during molecular interactions, across space, and resonantly resulting in a hydride, methyl, or phenyl carbocation or shift, as with ATP integration into methionine, donating hydridic character, moving the newly donated hydridic center and possibly shifting more distantly an already existing hydridic center, exciting or ionizing one of the molecular centers differentials such as the ionization of sulfur in methionine resultant of ATP integration into Methionine to produce the ATP adenylation methylation reduced methylene bridge cysteine known as s methyl methionine.

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

methylene bridge cysteines toward application to prevent potential massive deactivation of hydridic centers in biologically active or living molecules that is integral to all diminished outcomes.

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

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

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

The exhibition of methylene bridges in these contexts sequester capacitance or current from intramolecular or extra molecular environment, to Universes level magnetic and electromagnetic fields, and apply these toward construction from foundational physiological compartments to the anatomical compartments themselves, while capture of hydride oxidation freed 2 eV- by

membranes in correlation to insulating ether linked fatty acid availability in cellular membranes, magnetic field interactions used in permanent magnet sustainable energy dynamics, and membrane phospholipids which increase superconductor temperature thresholds of efficiency toward the physiological temperature range, while also physiological pressurization and thermodynamics enable fundamental interactions, such as hydrolysis of the water molecule, nearer to physiological environmental parameters, all present methylene bridge and methylene bridge cysteines as an oscillating mechanism that informs status of indefinitely sustainable physiological energetics.

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

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

However, availability of sulfur or thiols without methylene bridges supplies sulfur to integrate with intramolecular sulfide of Thetin unmethylated bridged cysteine methylpherase, thereby linking sulfur adequacy with preventing deactivation of this beneficial enzyme because intramolecular disulfide bridges occur in this most physiologically abundant enzyme during sulfur inadequacy. SP1 genomic sequence copy number increases in the folds of G quadruplexes and are counteracted by G quadruplex Stabilization as well as is counteracted by diminishing SP1 activity, thereby preventing SP1 increase of telomerase to diminish telomerase replacing of telomeric repeats when they are removed by DNA Replication primer activity during each cellular division.

Telomerase and Alternate Replacement of Telomerase enzymes both are beneficial in PEMT functional, unimproved cellular entities, cellular lineages, and tissues. G quadruplex stabilization and counteracting of SP1 also prevent SP1 diminishing of immunological CD4+ availability and diminishing of CD8+ availability, as well as prevents SP1 enabled increase of PD1 AND PDL1 receptors which all perform obscuring of cellular entities, impaired and unimproved, from

immunological Synapse monitoring, counteraction, removal, or introduction of senescence. AP1, when increased, just as SP1 is a deactivator of PEMT when increased, is a nonresolution cytokine.

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

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

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

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

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

The solvation or hydration shell constitutes a differentiated, molecule specific encapsulating H20 sheath that is distinct in molecular, ionizing, and Michaelis as well as velocity of interactions and movement when compared bulk water beyond the 2 angstrom base shell and particularly beyond the 15 angstrom extended hydration shell. Intramolecular characteristics and catalytic activity, as well intermolecular characteristics and catalytic activity, including compound molecules and closely linked molecules with overlapping hydration shells are all shaped by the Hydration shell dynamics which can promote not only planar behavior of the solvation shell but also can cause ligand or biological molecule catalytic interfaces to more precisely mimic experimental pharmacologically derived estimates of ligand behavior, particularly when between 70 and 10 water molecules comprise the solvation sheath within a subdomain of a macromolecule or when between 10 and 70 water molecules comprise the Hydration shell of a molecule. But inclusive of folds and overlaps that can occur between subdomains of compound molecules or such overlaps that can occur between closely linked molecules.

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

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

The active management of methylene bridge cysteines, therefore, prevents fibronectin from increasing the occurrences of Fibronectin connection between cytoskeleton and the extracellular matrix where fibronectin has the potential to increase signaling which promotes tissue remodeling, changes to extracellular matrix, and promote fibronectin polymer assembly. This connection imposes the effect of methylene bridges, changing both the characteristics of signaling at molecular and electromagnetic levels, but most importantly disrupting the characteristics of the foundational energies that link particles, waves, energy, superposition, angular characteristics and linkages between different instances of space and time, practically represented as changes to spatial interactions involving carbocation, hydridic characters, remote carbocation/hydridic/aromatic balancing interactions, and foundational remote programming of the environment by hormones, agrin, RNA, DNA and fields that orchestrate physiology.

Methyl bridges must be encapsulated because they are foundational biological polymerizing linkages that enable fatty acids sequences, DNA and RNA sequences, and enable alkanes that promote alkalinity necessary for mundane amino acids, such as s adenosyl methionine to be become enzymes and exhibit carbocation rearrangements in which hydridic energy is free at intramolecular levels without releasing the hydride that is separated from its original incipient location, similar to a how a filament in a light bulb is excited by current to emit electromagnetic energy. Methylene bridge cysteines, when activated by biosynthetic groups that are strong electron withdrawing groups such as those with nitro, nitrile, carbonyl or others, resulting in

methylene bridge sequestration of e-, current, or the additional e- integrated into hydrogen to produce hydride H with 2e- electrons and typically 1 proton, which is woven into the biome and universes somewhat pervasively and which almost every if not every atom foundationally uses as a superclass from which such nonhydrogen atoms are derived as subclasses, at least from a logical perspective. Methylene bridge activation sequesters the e- that it is already attached to remotely in the aether of the Universes, galvanizing metabolism, flow of electrons, through space jumps of electrons, enabling molecules and atoms to perform as essential molecules even when the electron configuration of atom is not precisely what is require or when nanoplasm is not precisely configured as required, producing tunneling of electron through impedance and limitations, as well as producing biosynthetic activity and even movement or chemotaxis that results in deliver of e-. Hormones and glucorticosteroids exhibit biologically relevant symmetry and offset from symmetry while exhibiting hydridic patterns that weave hydridic fields into the aromatic hexameter that emits resonant or remote control of diverse aspects of molecular activity and metabolism, including exhibiting remote carbocation interactions and shift of hydridic character. These analyses often present that every outer incomplete energy level or every outer energy level outside of a completed energy level might be considered as a shared energy level among all atoms of the universes, practically explaining these phenomena although these also explain quantum entanglement as a result of enhanced direct particle exchange between material of the universe along with encompassing influences of the Universes.

However, methylene bridges produced by many methyltransferases result in in freed unmethylated methylene cysteine bridge factors that must be encapsulated to prevent being utilized in dysregulated polymerization. Transsulfuration translates methylene cysteine bridges into cysteine without methylene bridges. Numerous pathways recycle methylene cysteine bridge into methionine that is used in synthesis of 99.5 percent of genetic transaction products because methionine is a foundational enabler of DNA and RNA polymerase primer sequences required for attachment of polymerases to begin synthesis at least at the T-RNA level within ribosomal molecular machines. Methionine can then become adenosylated to produce s – adenosyl methionine, resulting in the hydride within the oxonium between the phosphate groups of methionine becoming rearranged or experience carbocation that results in ionization of the Sulfur of Methionine, although it is the methylation that results in production of methionine that encapsulates or reduces the methylene bridge of methylene bridge cysteine, according to the literature. Importantly, methylene bridge is the gateway through which Hydride in the aether of the Universe becomes systematically integrated into physiology to become molecular energy, become translated into chemical energy such as pyruvate produced in glycolysis , become mined by the electron transport pathway of oxidative phosphorylation to become ATP for instance, derivation of pyruvate from Glucose, freeing of molecular and chemical energy as 2 eV-, reintegration of eV- through RET, integration of Hydride into physiology such as through PEMT packing of hydride within CH3 into the three open location of the nitrogen within phosphatidylethanolamine to produce Choline lead group, synthesis phosphatidylcholine, packing 3 CH3 molecules into the nitrogen which balances the methylene bridge at the SN1 fatty acid moiety, balances the fatty acid alkane in the SN-1 position, balances the oxonium in the phosphate group between the lead group and the fatty acyl integration loci. Importantly, methylene bridges are included as activated bridges in CH3 such that the nitrile group performs

as an electron withdrawing group and the 3 CH3s seem to be constituted activated methylene bridges with hydride already sequestered and integrated since the third Hydrogen in CH3 is considered to be hydride, according to the literature.

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

Supplementation of phosphatidylcholine, experimentally, causes a decrease or cooling of thermodynamics in the microenvironment compared to production of phosphatidylcholine in the cdp-choline pathway because the cdp-choline produces phosphocholine that is used by numerous pathways as an energy molecule including pathways used by pathogens and pathways used in pathology, particularly platelet activation and complements immune system activation which phosphocholine can perform constitutively, presenting why activity by PEMT as a producer of resolution phase fatty acids integrated in newly produce choline as phosphatidylcholine is beneficial in the about 30% fraction of phosphatidylcholine or higher which PEMT produces compared to about 70 percent or lower synthesis of phosphatidylcholine by the cdp-choline pathway using already non de novo choline typically obtained from nutrition or from phospholipase/phosphodiesterase lipase catalytic activate at cellular membranes. It is now known hat riboflavin is an enabler of cholesterol and fatty acid synthesis and in many ways enable hormone pathways that produce PEMT, such that it is now essential that all B vitamins be included as a foundational of any nutritional or therapeutic regimen. Particularly, the inhibition of PEMT prevents the packing of Hydride into and locking of Hydride into the electron withdrawing nitrile group that balances one of the methylene bridges maintained from its phosphatidylethanolamine while removing the other methylene bridge. This important pathway of ethanolamine as an essential nutrition which supplies substrate to the cdp-ethanolamine pathway, resulting in production of phosphatidylethanolamine, produces an encapsulation of two methylene bridges that are encapsulate in phospholipid as substrate for PEMT catalysis and which can be redirected toward attachment catalysts for autophagy or toward pathways in which methylene bridges can be revealed and implemented by pathology promoting conditions or activity. The redirection of recycled methylene bridges reinserted into the cdp-ethanolamine pathway such as by ceramide to S1P to S1P lyase activity, or obtainment of ethanolamine nutritionally or supplementally or otherwise, during inhibition of PEMT, promotes conditions of intracellular clearing through autophagy compared to the cdp-choline pathway which enhances proteolysis by producing phosphocholine in two loci, choline kinase and aSMase/nSMase, that supply phosphocholine for enhanced autophagy, increase influence ubiquitinase pathways which are numerous, and reprogram cellular phenotype toward dysregulate metabolisms focused on sustaining membrane resiliency during inadequate levels of choline and inadequate levels resolution phosphatidylcholine. BAG3 promotes autophagy while BAG1 promotes proteolysis, although some conditions can become resistant and persist when BAG1 or the 26S, 20S or 19S proteasomes, immunoproteasome or other proteasomes are therapeutically disruptive, often

requiring therapeutic decreasing of GSK3B, GPCR receptors, other S1P receptors, BCL2, or S1P Lyase which although depletes S1P can be a resistance pathway.

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

Methylene bridge cysteine is an amino acid exhibiting sulfur which is also a methylene cysteine bridge that, when untransformed into cysteine or unrecycled into methionine through methylation as well as potentially adenosylated to become s-adenosyl methionine, can have its methylene bridges become dysregulated and commandeered into pathology promoting differentiation, energetics, and polymerization, such that when methylene bridge cysteine is below 6 or 7 micromoles per liter, a 99.95 or more percent decrease in risk of the most substantial adverse physiological and behavior outcomes over a decade of observation among a population of about 10,000 compared to control populations of about 10,000. The enzyme PEMT packs methylene bridge cysteine that has sequestered hydride as a third Hydrogen, CH3 or methyl groups, into the three possible orbitals exhibited in the nitrogen of phosphatidylethanolamine, resulting in phosphatidylcholine, while removing one methylene bridge from phosphatidylethanolamine and producing enriched fatty acids with numerous methylene bridges including a double adhesion divinyl methylene spacer at the SN-1 location, although SN-2 and SN-1 can both exhibited enriched resolution phase fatty acids, which all, along with oxonium in the phosphate group of phosphatidylcholine, provide unique, fatty acid

alkene extents, packed hydride, and hydric oxonium to balance or stabilize the nitrogen cation. Resultantly, a highly stable most abundant phospholipid that is packed with energy is produced that is a foundational factor in development, regeneration and repair. Diminished PEMT catalysis results in remarkably diminished decrease of pyruvate because diminished PEMT results in P53 imposition of diminished Glycolysis and imposition of diminished pentose phosphate or hexose monophosphate shunt activity, although upregulation of the cdp-choline pathway also occurs when PEMT is diminished in activity. The cdp-choline pathway produces phosphatidylcholine that is not particularly enriched and uses recycled choline which has methylene bridges already integrated into the nitrogen of choline. Thus, when PEMT is diminished anaerobic glycolysis or diminished throughput in energy metabolisms occurs because the protective influence of a resilient mitochondria and its programming as well as optimal resolution phase fatty acid PMME, PDME and phosphatidylcholine, sequential metabolites produced by each addition of CH3 to the nitrogen of phosphatidylethanolamine may not be adequately available. Aerobic glycolysis, which is considered to be involved in pervasive pathology, occurs when P53 suppression pathways become surmounted or dysregulated, resulting in increase in energy pathway throughput without the protective saving of energy into hydride packed phosphatidylcholine, balancing of energy throughput with savings, redirection of current or hydride toward structural resiliency, and the without the angular, hydridic, and other 'special' effects that occur in physiology to produce the typical, or not so typical, resiliency of biological systems. Aerobic glycolysis is known as the 'warburg effect.'

Hormones exhibit Hydrogen in patterns that weave hydridic fields from the upper right offset pentameter into the midline upper hexamater and then into the lower mideline hexameter, followed by delivery into the lower lefmost aromatic resonant hexameter through a methylene bridge within a a divinyl methyline bridge, resultant in hydridic character in such lowermost resonant aromatic hexameter which is utilized in aromatic, resonant, through space interactions that program spatial aspects of biology through remote, spatial, angular, hydridic and ionic influences that enable development and sustainment of biology. Hormones, at these core pentameter and hexameter elements, exhibit close or strongly similar symmetry as the internal, wet or splanchnic organ systems. Hormones and glucocorticosteroids, thus, do more than merely incompletely or completely integrate into and transactivate the perfect 13 sequence DNA estrogen response element which includes PEMT and number of transcription sequences otherwise essential for pioneering, regenerative and repair development, such hormones and glucocorticosteriods program develoment through remote hydridic shift, remote hydridic character, angular influence, spatial influence, ionic influence and in other ways. Conditions involving dysrugulated or impaired hormones can typically involve the methylene bridges and programming influences in such regard as well as involve upregulated exhibition of dysregulated methylene bridges, as well as dysregulation of methyleine bridge cysteines. Importantly, methylation is an important primary regulatory, deactivator and encapsulated of methyline bridges and methylene bridge cysteines as well as impedes the leading edge of polymerization structures, while also is ustilized to detoxify hormones and glucocorticosteroids similar to sulphones or thiol molecues also detoxify or detactivate hormones through the COMT pathway, cytochrome P450 pathway, and in other sulfur pathways inclusive of the transsulfuration that structurally deteriorates methyleine bridge cysteine to cysteine. Recycling pathways for methylene bridge cysteine and s-adenosyl methylene bridge cysteine are major pathways of

stabilizing. The major pathway for methylene bridge cysteine and dysregulated unencapsulated methylene bridges are in their ability to sequester current from any location in which hydride has been woven into the aether of physiology, the biome and the universes, particularly because these disrupted signaling, metabolisms, tissue structure, extracellular matrix structure and signaling, agrin structure and signaling, as well as disrupt the energies that constitute the foundational aspects of matter and material of the Universes.

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

Wholistically, biomedically, pharmaceutically or adjuvantly otherwise assuring that ICD-10 code E72.11, or elevated methylene bridge cysteine, is prevented or alleviated with a asymptomatic inpatient therapy at 15 um/L, symptomatic inpatient therapy or asymptomatic outpatient therapy at 10 um/L, Outpatient, out of office location therapy, or office location therapy above 6 or 7 um/L and with an objective of between 6 or 7 to 3.7 um/L is essential to both Human health and Human behavior.

It should be presented here that the one instance of adverse outcome among the population of about 10,000 over a decade of observation, may have been the result statistical nuance in which a perfect 100 percent was not possible, although the study presents that only 1 instances of the most adverse of outcomes occurred among populations with methylene bridge cysteine below 6 or 7 um/L while in the population of about 10,000 with methylene bridge cysteine above 6 or 7 um/L there were 500 of such adverse outcomes in the same duration of observation. Importantly, alkanes inherently cause amino acids to exhibit catalytic activity and methylene bridges sequester current for application in development, structural synthesis, biosynthesis and metabolism, all from the hydride, current or eV- exhibited in the aether of the biome and the Universes.

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

Thus, the diminished storage of methylene bridges, encapsulation of methylene bridges, and packing of hydride into cellular and physiological structure, along with dysregulated energy, is known as aerobic glycolysis which is only meaningful when compared anaerobic glycolysis because aerobic glycolysis can occur beneficially when PEMT is fully functional and fully catalytically bioavailable. PEMT removes a methylene bridge from phosphatidylethanolamine packs methylene bridges within CH3 into the open locations within the nitrogen of phosphatidylethanolamine in a way that also packs hydride as the third hydrogen sequestered upon activation of the methylene bridge within CH3 into the nitrogen, and balances the methylene bridge in the lead group, the oxonium in the phosphate group in the lead group connector, as well as balances the methylene bridges in the fatty acids linked by the methylene spacer in the connect to the SN-1 fatty acid location, although SN-2 location can also host fatty acids. The balancing of hydride in the positivity polarized nitrogen of phosphatidylcholine contrasts the obscured negative or alkaline polarity of the third hydrogen in each CH3, the oxonium in the phosphate group and the alkaline polarity of alkane linkages in fatty acids at the SN-1 positions and at possibly also at the SN-2 positions.

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

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

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

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

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

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

Phosphatidylethanolamine is a source of methylene bridges for glycosylphosphatidylinositol anchored proteins which invoke autophagy by performance as attachment loci for emergence of autophagosomes which essential for cellular sustainment, preventing increased comparative proteolysis, controls proliferation and controls metabolic commandeered changes linked to uncontrolled proliferation. Contextually, adjacent methylene bridges or multiple methylene bridges 77in phosphorylated ethanolamine's explains why increase in S1P lyase, which results in the depletion of the S1P pathways typically linked diminished outcomes but also results in hexadecenal and ethanolamine phosphate, culminates of resistant conditions, particularly because methylene bridges are recycled when ethanolamine phosphate produced during S1P lyase pathway catalysis is reinserted into the cdp - ethanolamine pathway.

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

Glycosylation of phosphatidylethanolamine tails diminishes its selection by PEMT, particular introducing preference specificity for lightly glycosylated or unglycosylated

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

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

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

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

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

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

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

The one methylene bridge of phosphatidylcholine compares to the two methylene bridges of phosphatidylethanolamine, while both of these molecules maintain the hydride packed oxonium in the unlinked oxygen of the phosphate group which links the methylene bridge to the fatty acid, glycerol or glycosyl tails. PEMT may prefer unglycosylated tails because it's processing my require or prefer selective configuration of the fatty acids linked methylene bridge and phosphate group, particularly in the third methylation in which phosphatidylethanolamine is exited into the phosphatidylcholine fraction.

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

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

Importantly, it has been derived a most essential empirical observation, which is that eHcy may merely be a most obvious example of the multiplicity exhibited by methylene bridges which attach to and promote structural development and structural polymerization as well as which

sequester current emitted from oxidation of Hydride or emitted current from carbocation rearrangements in molecules with hydridic character.

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

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

Unmanaged methylene bridges may attach to structure, biologically active molecules, structure, promote polymerization, sequester current in these contexts, display signaling, disrupt hydridic character and carbocation rearrangements to hydridic character, and since energies are involved in structural adhesions in much if not all nuances material if the universe, the essential presumptive nuances of physics, biology and biophysics may be destabilized by accumulated, unmanaged and inadequately available methylene bridges.

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

A review of the lengthy list of required affects if Hcy or eHcy, active in or required for most if not all manifestation of diminished health status, pervasively reveal patterns of methylene bridge

dysregulation, causing methylene bridge to emerge as a new most empirical specific encompassing empirical parameter in health and behavior. Particularly because methylene bridges affect the solvation shell or hydration which guides intramolecular and intermolecular interactions, as well as determines hydrodynamic characteristics at least up to 20 angstroms from the molecular surface. Methylene bridge proactive management may replace much if interventional care in developed civilizations, allowing Care infrastructure to be sustained as is with inpatient, outpatient, Office, mobile or home nuances of care to enable vibrant industry to be sustained and grow with a correlated beneficent effect to health and behavior. This contrasts, clearly, delaying assay and care total methylene bridge mismanagement has been allowed to deteriorate physiology onto emergent or substantial pathology.

A priority is afforded to managing methylene bridges of phosphatidylethanolamine, their direction toward autophagy anchoring as glyceryl versions, their direction through exclusion from PEMT third methylation toward antihistamine function and recycling when glycosylated, as well as their preferred selection by PEMT when lightly glycosylated or unglycosylated. Ethanolamine attaches the fatty acids to cdp – ethanolamine using diacylglycerol or allocated acylglycerol as linkages while this catalytic interaction prefers sn-1,2 diradylglycerol as substrate, result in in major output as phosphatidylethanolamine and some fraction glycerophosphatidylchol7ine. Oxidative phosphorylation, cellular respiration Complex III enabling, essential phosphatidylserine decarboxylase translation of phosphatidylserine to phosphatidylethanolamine occurs at the inner mitochondrial membrane such that sn-1,2 diacylglycerol phosphatidylserine and sn-1,2 diacylglycerol are Selectively preferred as substrate by both phosphatidylserine decarboxylase 1 and phosphatidylserine decarboxylase 2.

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

Methylene bridges are practically CH3 methyl groups without the third Hydrogen which is considered to be hydride. Methylene bridges are susceptible to strong withdrawers of electrons and are affected in such regard strongly enough to cause deprotonation, such that assimilation of electrons from flowing 7.2 current, intramolecular current, hydride in intramolecular locations or hydride in unattached structures can be withdrawn to become localized to the electron withdrawing complexes, even when this results in deprotonation, even when this results in a carbocation arrangement or shift in hydridic character, as well as when this results in a withdrawing of an electron across space between unattached carbocation participants.

5,10 methylene tetrahydrofolate is processed by the enzyme MTHFR to produce 5 methylene tetrahydrofolate which methionine synthase uses to produce methionine. which contributes Methylene bridges, Methylene Spacers, Methanediyl group, or Methano factors, all used to indicate methylene bridges, exhibit CH2 with individual linkages of the carbon to other factors, such that when located between strong electron withdrawing groups such as Nitro NO2 linked to nitric oxide biological benefit, Carbon double linked to Oxygen as Carbonyl, and nitril

composed of an axial group linked to a Carbon that is triple linked to a nitrogen, exposure to strong bases can result in highly biosynthetic products such as enclaves and carnations, explaining why 7.2 to 7.6 alkaline environmental pH is linked to homeostasis as well as explaining how methylene bridges must be encapsulated or counteracted in the homeostasis alkaline environment.

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

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

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

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

Glycosylated tails are less preferred than glycerol tails in PEMT selection of its substrate fraction of available phosphatidylethanolamine while supply of phosphatidylethanolamine by serine decarboxylase and ethanolamine phosphotransferase are presented by the literature as scrutinizing only the sn-1 loci for diradyl glycerol during production of phosphatidylethanolamine as substrate for PEMT. The cdp – choline pathway uses already

existing choline processed in the same pathway transactions as ethanolamine, to result in phosphatidylcholine. with diminished focus on enriched diversity of fatty acids. Phosphatidylcholine can be recycled generally through phosphatidylserine decarboxylase, including direction toward trypsin synthesis by MDR2, lipase activity, diesterase activity or direction toward ceramide and toward the sphingolipid signaling pathways which should be managed because are cellular existential challenge response pathways linked to diminished health status response. Sphingolipid signaling pathways should be managed because these can emerge as causal factors of diminished health status if exhibited for extended duration.

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

Ethanolamine, phosphoethanolamine, Citidylylethanolamine, diradyl or glycero phosphatidylethanolamine, diradyl or glycero phosphatidylmonomethylethanolamine, diradyl or glycero phospatidyldimethylethanolamine, (palmitate first fatty acid in fatty acid beta oxidation, oleoylate, extended length omega-6 arachidonic acid, Docosahexaenoic acid, omega-3, ether linked, diverse fatty acid) enriched glycero phosphatidylcholine followed nonresolution/resolution phase lipase/diesterase freeing of fatty acids which are applied in immunology or shuffled while being reintegrated into phospholipids such as enhanced diversity fatty acid phospholipids and enhanced diversity phosphatidylcholine by LPCAT/MBOAT/Lysoplasmalogenase catalysis, provides a central perspective of ethanolamine shuttling of methylene bridges.

Although the genetic conditions can have enhanced sequelae, these and other extended differential characteristics pervasively involved methylene bridge and methylene bridge cysteine escape from transsulfuration, recycling, proteolysis, serine proteolysis, tissue plasminogen activator activity, autophagy, ubiquitylation or other excretion and recycling pathways. AP1 which diminishes the cdp-choline pathway at CTP--choline citidylytransferase while both diminishing PEMT and diminishing telomerase replacement of telomeres during each mitotic cellular cycle, compared to SP1 performance of these same changes although SP1 upregulates telomerase instead of diminishing telomerase, provides of differentiating influence in the developmental sequelae following pipelining of ethanolamine to diverse shuffled phosphatidylcholine. The small amount of cdp-choline pathway substrates produced by cdp-ethanolamine pathway enzymes and the small amount of cdp-ethanolamine pathway substrates

produced by the cdp-choline pathway enzymes are more than interesting, particularly because through phosphatidylcholine conversion to phosphatidylserine, phosphatidylserine conversion to phosphatidylethanolamine and phosphatidylcholine direction through ceramide, sphingolipid synthesis, and then to hexadecenal and ethanolamine phosphate, as well as PEMT de novo synthesis of choline as enriched phosphatidylcholine, the CDP-Ethanolamine pathway and the CDP-Choline pathway pipeline substrate to one another.

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

Intriguing is the way in which methylene bridge multiplicity enables or participates in which any defined space can have its constituent material behave as other material such as atoms of one nature perform as other atom level configurations. Methylene bridges have an omitted third Hydrogen, relatively, hydride, such that electron withdrawing groups attaching to methylene bridges cause a sequestration of 2 eV- or cause sequestration of current, either in through space jumps, electron tunneling across atom or biological structure, constitutively from freed fluorescent hydridic energy, or by causing a physiological pathway or pipeline to be invoked that culminates in delivery of current, electrons as 2 eV-, four essential energy immersion such as nitril groups packed with methyl groups which have hydride. Correlatively, methylene bridge as order takers and delivery invokes of hydride may result in synthesis or delivery of ATP or Pyruvate. Inherently, hydride negative polarity or alkalinity promotes natural gradient in the H+ prevalent solution, + environment or unpolarized environment, while its order taking and delivery of current or energy molecules as well as fluorescent enables physiological activity to occur against natural gradients, allowing organism to 8ncreasingly use conscious priorities on shaping of physiological outcomes and behavior. Methylene bridges exist in space and although essential biological factors are also found in space suggesting that there integral processing requires a spontaneous event. Order taking by the methylene bridge and its sequestration of current and interaction with the solvation or hydride shell is more than adequate cause spontaneous or more accurately, designed programmatic integration of these foundational components of biology. Thus, in any defined space the components can be galvanized to perform actions enabling or sustaining biology, at least one modality in such regard is the sequestration hydridic character or current by methylene enabled changes.

Polyunsaturated fatty acids Docosahexaenoic acid and Eicosapentaenoic acid exhibit methylene bridges and phosphorylate delta carbons of tryptophan 448 and 553 of PDK1 which along with AKT phosphorylation at tryptophan 424 by these PUFA fatty acids, results in PDK1 translocation to the cytoplasmic membrane, depletion of the Pyruvate Dehydrogenase downregulator known as PDK1, enhances glucose depletion correlative to acylation of AKT and acylation of PDK1, while also enhancing insulin resistance because the physiological effect of insulin receptor downregulation by P53 is circumvented by these conditions to enhance glucose removal and processing. A study observes that polyunsaturated fatty acid, PUFAs, counteract the potential for upregulation of glycolysis when glycolysis should typically be downregulated responsively to diminished PEMT activity and responsively to upregulated P53. This potential

increase in glycolysis amid PEMT downregulation and amid P53 upregulation is a canonical integral factor in diminished health status, and is counteracted by Docosahexaenoic acid and Eicosapentaenoic acid, in a way that results in beneficial tissue remodeling which. DHA and EPA enabled tissue remodeling, in this regard, follows reintroduction of diminished glycolysis that is coupled with reintroduction of Krebs Cycle upregulation compared to glycolysis.

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

The "warburg effect" can be simply reprinted as an increase in glycolysis without the assistance of PEMT packing of hydride into biosynthetic electron withdrawing nitril lead groups of newly produced, unglycosylated or lightly glycosylated, glycero, ether linked, Omega-3, DHA, EPA or otherwise enriched phosphatidylethanolamine metabolites including likewise enriched PMME, PDME and phosphatidylcholine. Phospholipase and diesterase are increased in detrimental conditions including when PEMT is downregulated, freeing choline, other lead groups such as ethanolamine, phosphatides, and fatty acid from cellular membranes along freeingCa^{2+} encircling lead groups of phospholipids to sustain Ca^{2+} reliant versions of lipases and diesterases, although versions of these include those which function independently of Ca^{2+}. These can result in release of unencapsulated methylene bridges, although phospholipase or phosphodiesterase activity can generally by correlated with increase in methylene bridge cysteines. Diminished PEMT results in diminished migration of phosphatidylethanolamine from the inner leaflet of membranes to the outer leaflet or outer membrane as phosphatidylmonomethylethanolamine then phosphatidyldimethylethanolamine, then phosphatidylcholine, resulting also in diminished hydride packing at the Plasma membrane, diminished support of hydridic field attenuation into the extracellular space which decreases support of the near 7.2 to 7.6 background pH and diminishing inherent enablement of polarity gradients, solvation, solvation shell, intramolecular dynamics. intermolecular dynamics, and water dynamics up to 20 angstrom or more from molecular surfaces. The result of impaired PEMT packing of hydride, in this regard, may be a foundational destabilization of existential nuances of the foundation biological compartment, including upregulation of the cdp- choline pathway to counteract massive programmed deterioration of cellular compartments, increase in proteolysis over autophagy, increase in ceramide from diminished directing of phosphatidylcholine toward phosphatidylethanolamine resultant of PEMT inhibition that produces accumulation of phosphatidylethanolamine, increase in sphingosine 1 phosphate from ceramide because tissue stability requires that the cellular deterioration signal ceramide be redirected toward S1P massive pathways of survival signaling, all of which are lessened in availability by PEMT function or PEMT metabolites.

G protein coupled receptors, S1P receptors, including GSK3B, PDK, cellular survival BCl2, and proteolytic enhancing BAG1 which links chaperone complexes to the 26s proteosome using ubiquitylation pathway signal attachments that are diverse and can be commandeered by detrimental conditions or axial pathways, all are S1P effected. BAG1 compares to BAG3 in that BAG3 invokes, preferentially, autophagy, which although endosomes can be used by microbes to escape the toxic plasma membrane interstitial space, is nonetheless vacuous to intracellular substrates moved into autophagosomes resulting in clearing of diverse material from the intracellular environment.

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

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

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

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

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

The linkage of nitril packed Hydride in membrane phosphatidylcholine, being fired lipase and diesterase for access in nad+/nadh and nadp+/nadph redox transactions such as the electron transport pathway freeing of hydride from nadh to emit 2 eV- with about 58 percent as fluorescent energy loadbalanced across each phase of oxidative phosphorylation electron transport and with about 42 percent integrated into the oxonium exhibited between the phosphate groups of ATP, Product of oxidative phosphorylation also known as cellular respiration, as well as entry of ATP into metabolism in Diverse contexts, including its integration into the glucose – 6 – phosphate dehydrogenase processing of glucose – 6 – phosphate to support translation of the hexose glucose into the hexose monophosphate shunt or into glycolytic synthesis of Pyruvate followed by either NADH enabled translation of pyruvate into lactate, the NAD+ and CoA enablement of Pyruvate translation into Acetyl – COA which can be shuttled by oxaloacetate into enabled shuttling of Pyruvate into the Krebs Cycle as Citrate and CoA. CO2 is supplied toward fatty acid synthesis during Acetyl-CoA production, Acetyl - CoA can be directed toward Acetyl - choline storage if excess choline. Alanine, phosphoenolpyruvate, oxaloacetate, and acetaldehyde each are major pathways of pyruvate processing.

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

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

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

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

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

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

a de facto increase in the cdp-ethanolamine pathway, explaining why the cdp-choline, in some organisms, is known as the nucleotide biosynthesis pathway.

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

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

PEMT might have no specific regard for D chiral Glucose except that when Glucose - 6 - Phosphate Dehydrogenase produces glucono lactone from Glucose phosphate nadp+ is used as a cofactor and becomes nadph which decreases the essential increased levels of nadp+ when nadp+ is compared to nadph, a disparity that is a foundational enabler of activity, gradients, transactions and flow of hydridic current in physiology and on biology generally. P53 seems to prevent both production of nadp+ and Glucose phosphate during gluconeogenesis as well as prevents production of glucono lactone and nadph because PEMT diminished function impairs the flow of hydridic current through structure as a priority. Structure us essential in trapping current and controlled directing of current, enabling cellular capacitance and potentials, post synaptic neuron

polarization baselines, hydridic effect, and galvanizing of concerted tissue capacitance linked to consciousness and cognition. The spooky spatial aspects of these influences are another dimension of why trapping and recycling current is a priority, such that shuttling current through structure enables the foundational biological compartment to exist and function in ways that are increasingly both spookily biological and strangely biological.

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

The activation potential for SAHH has been solved, in a study, as NAD+ increased comparatively to nadh, suggesting it is inherently and strongly potentiated toward translating s-adenosyl methylene bridge cysteines by release of the adenosyl moiety, producing nadh from nad+. However, the same study observes that SAHH then proceeds to synthesized adenosine which is a downregulator of choline kinase alpha attachment of ATP to Fee choline at the incipient phase of the cdp-choline pathway, resulting interestingly, in production of nad+ from nadh. Thus, s- adenosyl methylene bridge cysteine is trapped by diminished nad+ and produced when nadh levels are increased, whileP53 reinforces the glycolytic translation of glucose into pyruvate that already potentially occurs when nad+ is diminished in availability from its typically strong prevalence over nadh. These are clearly two among other mechanisms that manage structural molecular to phases of energy or flow of current. Pathways of methylene cysteine bridge processing, deterioration, or recycling which do not produce adenosine have an increased potential of upregulating the cdp-choline pathway because it relieves deterioration of PEMT diminished packing of Hydride by using already produce choline lead groups packed with hydride and CH3 to produced phosphatidylcholine using non newly produced choline lead groups. Phosphatidylcholine and phospholipid structure is produced from recycled structure when PEMT is not adequately synthesized choline lead groups. Particularly, redirecting current from escape, depletion and attenuation toward, instead, reintegration into membranes and structure. The priority seems to be structural translation, sustainment and cycling if current, particularly hydridic aether.

Redirecting of s-adenosyl methylene bridge cysteine toward thioether methyl transferase results in production s adenosyl methionine while each catalytic action by thio ether methyl transferase results in polymerization selenium, tellurium. sulphonium, other amine, or other factors, while trimethylsulfonium is then used as a substrate for thetin methylene bridge cysteine transpherase production of the desquamation depolymerization factor used pervasively in therapeutics production in the 1900s and 1900s, while dimethyl thetin performance as an alternate for

trimethylsulphonium has been presented in the literature since 1878, methylene bridge cysteine has been presented in the literature since 1810, and lecithin as mixed choline and phosphatidylcholine was characterized in the literature in the middle to later aspects of the 1700s.

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

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

The human inclination to derivatize important contexts to empiricism implores what may have been futile endeavor, in the more than two centuries since methylene bridge cysteine was first characterized, to simply explain the methylene bridge multiplicity. The methylene bridge sequestration by biosynthetic strong electron withdrawing groups sequester hydride and galvanized molecules, structure, metabolism and development of biological systems as a result. Much of human activity and behavior seems to be likewise sequestered to such priority or shaped in ways that indicative of such priority. However, the application of methylene by biosynthetic strong electron withdrawers, through polymerization potential, transforms current into structure, explaining why and how mitochondria, sometimes hundreds in an in individual cellular entity, effect and regulate developmental programs, particular through PEMT and particularly through mitochondrial PEMT2 activity which typically emerges near the transition from gestational phase.

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

Systems modeling perspectives observe correlation between hypomethylation and s-adenosyl methylene bridge accumulation, particularly linked to diminished unimpeded flow of methyl resources and, particularly, with diminished PEMT throughput. Increased methyl group resources without methyl resource metabolic processing and increased cysteine bridge exhibition without metabolic processes at result in accumulation, aggregation and sedimentation, with

methylene bridge accumulation potentially resulting in. Both, deposits without requirement of being integrated into complexes, but also potentiating integration into complexes that activate methylene bridges to transfer hydride for translation into structure polymerization activity. Slowing metabolism may cause methyl and cysteine bridge requirements to decrease, resultant in methylene bridge accumulation within junctures of metabolic pathways, particular areas of physiology, as structural polymerization, or as mechanisms enabling less beneficial process to commandeer such dysregulated methyl resources.

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

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

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

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

Another analysis in the compendium of research linked to this analysis presented that the hydroxyl and chiral hydrogen of hormone including estradiol exhibit a pattern that integrates the uppermost cardiac pentameter into the adjacent hexameter, subsequently to a hexameter connected to the first hexameter, and then is interrupted by divinyl methylene spacers in the lower offset hexameter in in both directions the shared methylene bridge between the lower center hexameter and the offset lower hexameter. Hydroxide is exhibited linked to the carbon

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

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

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

The culmination of data science objectives seems to be the generation of increasingly linear graphs, curves, training data, tensors, correlates, causal links, Mechanistic links, dualities or inferential correlations, multiplicities, information tuples, shards, data columns, columns, natural language representation of these as stories. The object of data Science also seems to include ascertainment, with increasing certainty. of relationships between variables, with increasing certainty enabling derivation of the values or characteristics of any variable using the values or

characteristics any other of these variables or using variables otherwise. Resultantly might be achieved structured logical, mathematical, clinical or other simple or complex analyses, explorative or presumption questions which can be presented as proofs that, also, with increasing certainty confirm such relationships, values and characteristics.

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

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