

Health Industry Qualifying Rules Engine Algorithm, Deep Learning Tensor and Reinforcement Learning Tensor

AHEC, FQHC, and 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. Services performed in AHEC and FQHC 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, phosphatidylmethylethanolamine, phosphatidylcholine enriched with docosa-hexaenoic 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 resonant carbocation which transfers hydride into the sulfur atom to cause cationic Ionization of sulfur such that the enzyme s - adenosyl methionine begins to function as an enzyme with CH₃ sharing and other ability, and such that 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 and the lone pair electron configuration of CH₃ to Nitrogen of Phosphatidylethanolamine in three successive instances to produce another carbocation rearrangement that if resonant and not structural does not change the polarization of the oxygen between the 2 carbons and the other aspects of the phosphatide moiety or if including structural integration of these 3 CH₃ molecules produces a change in polarization of this oxygen or oxonium.

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 1-diphosphate, 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 with 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 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 nanoplast 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 μm/L. Asymptomatic 15 μm/L, symptomatic 10 μm/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 μm/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 nanoplastic 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 way that decreases the number cellular divisions that a divergent cellular lineage incurs before chromosomes fuse to disable additional proliferation. Counteracting SP1 and assuring stabilization of g quadruplexes prevents SP1 increase in telomerase and Prevents SP1 enabled obscuring of impaired cellular entities or impaired tissues from immunological control, as well as correlatively. SP1 deactivation of AP1 enabled rapid telomeric attrition toward senescent impedance to mitosis along with obscuring of cellular entities from immunological control by SP1 which allow proliferation of impaired or commandeered cellular entities, cellular lineages and tissues, are all counteracted by counteracting SP1 and assuring stabilization of G quadruplexes.

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

phosphatidylinositol and other factors from endoplasmic reticula to mitochondria through the mitochondrial associated membrane.

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

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

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

Salvation or Hydration shell constitutes a differentiated, molecule specific encapsulating H₂O sheath that is distinct in molecular, ionizing, and Michaelis as well as velocity of interactions and movement when compared bulk water beyond 2 angstrom base shell and 15 angstrom or more 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 salvation shell but also 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 between 10 and 70 water molecules comprise the Hydration shell of a molecule. 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 its connection between the 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. Experimental observation of poly ethyl acrylate has observed that it differs from poly methyl acrylate in that poly methyl acrylate has one less methylene bridge and this one less methylene bridge in poly methyl acrylate is accompanied by a methyl group which results in poly methyl acrylate being unable to promote or being enabled to actively diminish fibronectin polymer assembly. The potential of fibronectin polymer assembly when poly ethyl acrylate and its methylene cysteine bridges are inadequately managed contrasts with the prevention of fibronectin assembly by methylation in a similar molecule poly methyl acrylate.

Functional assay of diverse fibrillation integration molecules including those involved in conditions involving fibril polymerization reveals that methylated methylene bridges are unable to promote or participate on fibronectin polymer assembly. Precisely, the lead group of ethyl acrylate exhibits an increased number of methylene bridges and this increased number of methylene bridge moieties increases the motion of the lead group, producing a less dense and less stable hydration or solvation shell.

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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 the 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 phosphatidyl dimethylethanolamine 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.

