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Review Article

Nutrition Science-An Altered Evolving Role in Maintaining Natural Health Driving Illnesses and Cure

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Abstract

Nutrition science refers to knowledge related to providing the nourishment to body. In recent years, the published literature indicates that nutrition carries additional roles as therapeutic and prophylactic agents, that are also evident in case of COVID 19 pandemic such as use of diet preparations like ginger fortified honey mixture and chicken nutrifortified broth which have played a critical role in eradicating COVID 19 pandemic across the globe. Cells exhibit diverse features but consistent characteristics under physiological conditions as a consequence of possessing shared cellular metabolism and physiological functional pathways that are regulated at genes operational networks level in pre-programmed or unprogrammed manners; micronutrients and metallic ions obtained from food play a critical role in modulating energy demand, homeostasis and oxidative stress flux bursts production in addition to acting as stimuli driving the natural health, illnesses and cure by common mechanisms that intertwine the different cellular processes in the body and by unusual auto-regulated isolated but molecularly interdependent ongoing processes in brain. This work discusses recently revealed evolving role of nutrition, reporting a few diet preparations that serve multifaceted roles based on latest scientific knowledge. There is an urgent need to critically review the published literature independently for saving lives and well-being of global community. This will enable science to move on in correct direction as many recent findings such as reported and unpublished and published findings have transformed the current understanding of knowledge, making previously reported work invalid.

Keywords: *Nutrition; Neurodegenerative Diseases; Alternative Scientific Narrative; Evolutionary Biology; Food Therapy; Epigenetics*

Introduction

Nutrition

By classical definition, nutrition refers to substances that a living entity takes in to build the components of its body structure, to produce instant energy or to save the energy in reserved form, to support the ongoing biochemical reactions responsible for maintaining life characteristics features including the synthesis of biochemical and/or chemical entities having multifaceted roles performed under physiological conditions or acquire altered roles under non-physiological conditions or under situation raised in response of stress, common stimulus that is triggered as an outcome of unusual consequences or events which occur within the cells or in their environments disturbing the homeostasis across the cells composing tissues, and interconnected physiological systems in multicellular entities [1,2].

Types of food and diet intake dependent consequences

The type of food intake plays a crucial role in governing the health status of individuals [1]. Certain food types, including food items generally considered safe and healthy, are reported to slow down the aging process [3-9] whereas, the intake of certain other types of food or their ingredients is associated with a heightened risk of illnesses onset either by affecting body metabolism or through other independent means [1,6,7,10-12]. Since ancient times, food preparations have served a medicinal purpose when used regularly, owing to their prophylactic and therapeutic properties. In recent history, use of diet preparations such as ginger fortified honey mixture and chicken nutrifortified broth has played a significant role in eradicating the COVID 19 pandemic from Pakistan and many other regions of the globe [1,5,8,9,13-19].

Biological complexities-Yeast an alternative research model for molecular biology tools

Studies of biological systems and their functions, conducted on multicellular entities and their cell lines often do not reflect real world occurrences. Due to considerable genomic homology between *Saccharomyces cerevisiae* and human genome as well as consistency and reproducibility of the findings, studies are being conducted on yeast to understand molecular mechanisms underlying biological processes in living systems. It is reported in literature that the *SOD1* gene that encodes for Cu-Zn superoxide dismutase (sod1p) and *CTR1* gene which encodes for copper transporter 1 protein (ctr1p) are part of same gene regulatory network and are reciprocally regulated in response to copper concentration gradient levels dependent manner modulated by iron-copper associated and dissociated ionic equilibrium balance gradient shifts driving the oxidative stress shifts through feedback mechanism at the transcriptional level. It demonstrates that the physical, chemical and biochemical stimuli or biological events can alter the effects of mutations that impair the function of essential genes operating within the same regulatory network. This process can enhance the activity of weaker genes.

When biochemical reaction cascades function under stress conditions, their framework is altered. Rab (2007) revealed that, under stress conditions, the operational regulation of gene networks' pathways and ongoing biochemical reactions modify their functional, biochemical and chemical potentials, targets and consequences. For example, under stress condition in del *CTR1* gene (that encodes for copper transporter1 protein (ctr1p)) yeast cells, when copper ions are sufficiently supplied to Cu-Zn superoxide dismutase (sod1p) by the *Lys7/CCS* gene's product (*Lys 7/CCS* gene is down-regulated or is turned off in non-functional or in absence of *CTR1* gene that encodes for copper transporter 1 protein (ctr1p)) the cell populations regain their viability proportion same as the wild type yeast cell populations exhibit on exposure with same strength of stressor. This restoration of activity of *SOD1* gene (that encodes for Cu-Zn superoxide dismutase (sod1p)'s product, a protein that binds copper and zinc ions in its molecular structure, and destroys free superoxide radicals including those generated by electron transport chain (respiratory chain) in absence of *CTR1* gene, which encodes for copper transporter 1 protein (ctr1p), a cell membrane protein that is a high affinity membrane copper transporter; such as Cu-Zn superoxide dismutase (sod1p) molecules acquiring their multifaceted chemical and biochemical potentials involving stereochemistry and enzymatic activity and their shifts enabling the knock out yeast cell populations to regain the survival strength against the stressor in a manner similar to that observed in wild-type yeast strains. These findings question the credibility of traditional understanding of the relation among protein dysfunction, cell survival and disease. It also underscores the complexity of genomic operations and biochemical processes which may be governed by genetically and/or epigenetically pre-programmed biochemical cascades alone or modulated in combination with environmental and dietary drivers. These act upon universal evolutionary capacitor switch, such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch driving regulatory circuit through universal evolutionary switch complex such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch complex acted upon by modulators in parallel which drive evolution of the healthy or unhealthy poorly-adopted or well-adopted cell phenotypes influencing the trajectory of disease and recovery.

These findings confirm the presence of universal evolutionary capacitor switch, such as*SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch driving regulatory circuit through universal evolutionary

switch complex such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch complex carrying multifaceted roles, that includes deciding fate of the cells, their evolution and regeneration potentials, sensing and transmitting triggered consequences of homeostasis shifts driving stimuli across the cells being regulated by genes operational networks altering the impact of genes' mutations including deletions with modulating the metabolic cascades in genetically and/or epigenetically preprogrammed and unprogrammed driven manner mainly depending on availability of types and levels of metabolizable sugars in primitive biological entities whereas in animal kingdom these biological features are driven by sustainable availability of insulin-dependent-sugars such as sugars that require insulin to enter target cells-and insulin-independent-sugars such as sugars that do not require insulin to enter target cells, by sustainable supply of micronutrients particularly metallic ions supply, by levels of free molecular oxygen availability, affected by fluctuation in their individual levels and the energy demand, in addition to, depending on environment and on the climate, any change in atmosphere of cells is additionally sensed by detecting shifts in cellular oxidative stress fluxes [20-41].

Nutrition-novel alternative roles

These latest interpretations of findings have altered the understanding related to nutrition and its potential roles in the body. It is evident in the reported literature that cell membrane physiology and membrane transporters drive the biological and biochemical role selection and the targets of different biological biochemical and chemical entities approaching the environment of the cells whereas the complex chemical structures, stereochemistry, ligand sequestration potentials, interaction and binding mechanisms of free ions, radicals, ligands, apo enzymes (inactive, protein part of an enzyme that becomes fully functional only when it binds with a non-protein cofactor) holoenzyme (a complete, catalytically active enzyme, which includes both the protein part called, the apo-enzyme, and any necessary non-protein components called cofactors) drive equilibrium shift between compounds, complexes and other forms of chemical and biochemical species originated from food (ingested foreign substance) define cellular functions and cellular structures under physiological conditions and modulate changes under stress or non physiological conditions by altering their innate roles, targets and cellular structures, in addition to, modulating the genetically and/or epigenetically driven preprogrammed or unprogrammed evolutionary, physiological and metabolic cascades operating within cells through the prime role of universal evolutionary capacitor switch, such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch driving regulatory circuit through universal evolutionary switch complex such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch complex [38,42-107].

Food serves as a source of micronutrients and sugars in addition to playing a significant role by its components and containing microorganisms in modulating cell membrane physiology and the role of cell membrane transporters in dependent and independent manners by affecting oxidative stress flux triggering and transmitting process.

Nutrients driving organ functions

The cumulative health of individuals is governed by normal physiology of body systems comprising of interconnected organs composed of tissues which are built from various cell types sharing the common partitioned environment defined by sustainable homeostasis under normal conditions which fluctuates under non-physiological conditions and unusual situations on weakening the buffering systems, repairing, regeneration and/or immune systems or with their cumulatively coordinated involvement, illnesses can occur with or without appearance of symptoms [108].

Nutrition oxidation and energy

Living entities including human beings require chemical and biochemical entities originated from organic and inorganic sources as well as sources of carbon, nitrogen, oxygen and sulphur including amino acids, vitamins etc. in addition to other components that can not be synthesized in the body to produce energy by oxidizing them, that is required to perform normal cellular and physiological functions

and to build cellular structures and to grow the body. The essential and non-essential fatty acid, essential and non-essential amino acid and all the metabolizable sugars are inter-convertible except into essential nutrients and can form components of cell organelles, in addition to, transforming into precursors of various metabolic cascades involved in synthesis of different metabolites and other biochemical and chemical entities. Recent research reveals that nutrients are involved in defining the fate of living entities by molecularly interacting at cellular levels, in addition to governing genetically preprogrammed and/or epigenetically preprogrammed and/or unprogrammed genomic functions.

In presence of free molecular oxygen functional catalase (an enzyme that breaks down hydrogen peroxide into water) works alongside with Cu-Zn superoxide dismutase (sod1p) a dimeric metallo-protein containing one zinc ion (Zn^{2+}) and one copper ion (Cu^{2+}) per subunit, that scavenges and dismutates the superoxide anion (O^{2-}) into molecular oxygen (O_2) and water (H_2O), subsequently, all these enzymes catalyze the same reaction converting the oxygen radical in molecular oxygen (O_2) and hydrogen peroxide (H_2O_2) through the alternate reduction and reoxidation of Cu^{2+} moiety from Cu-Zn superoxide dismutase (sod1p); the hydrogen peroxide (H_2O_2) is then enzymatically converted by catalase and glutathione peroxidase in molecular oxygen (O_2) and water (H_2O), produced in the final stage of respiration process driving energy from nutrients and transforming them into products or in their precursors, particularly from glucose that undergoes insulin dependent absorption in the target cells to breakdown, producing instant energy ultimately releasing carbon dioxide (CO_2) and water (H_2O) as products. The sustainable free oxygen supply and the presence of functional mitochondria dictate and select the fate of the end products of glycolysis such as pyruvate, opting to oxidatively metabolize it through the respiratory chain to produce adenosine triphosphate (ATP) or reductively metabolize it through lactate or ethanol fermentation to regenerate nicotinamide adenine dinucleotide (NAD^+) for continued glycolysis. Aerobic respiration produces a higher amount of energy as previously mentioned, this process occurs under steady supply of molecular oxygen and requires the functional enzymatic activity of catalase and Cu-Zn superoxide dismutase (sod1p). In cases of higher energy demands and/or limited molecular oxygen supply, catalases enzymatic activity is diminished and functional Cu-Zn superoxide dismutase (sod1p) molecules lose their dismutase enzymatic activity and acquire peroxidase activity, which may alter the regulation of *SOD1* gene and the other genes present within same genes regulatory network. In healthy cells loss of dismutase enzymatic functional Cu-Zn superoxide dismutase (sod1p) is associated with down-regulation of *SOD1* gene. In contrast, cancer cells that exhibit over-expression of *SOD1* gene under hypoxic conditions, leading to post-transformational conformational changes or altered stereochemistry of the protein molecules associated with altering enzymatic roles. It may be the primary underlying reason for the suppression of the metabolic cascade responsible for oxidatively metabolize the end product of glycolysis, pyruvate, through the respiratory chain (electron transport chain and oxidative phosphorylation) to produce adenosine triphosphate (ATP) in presence of steady supply of molecular oxygen but oxygen scarcity drives metabolic cascade to metabolize reductively through alcohol fermentation or lactate fermentation to regenerate nicotinamide adenine dinucleotide (NAD) from nicotinamide adenine dinucleotide hydride (NADH) ensuring continued glycolysis process. This shift is coupled with the release of fluctuating heightened cellular and systemic oxidative fluxes that alter the innate roles of chemical and biochemical entities, metabolic cascades, cellular organelles' structural definition and functions and oxidation-reduction potentials (redox potential) associated with changes in pH. Parallel to glycolysis, the pentose phosphate pathway, also known as the phosphogluconate pathway or the hexose monophosphate shunt (HMP shunt), generates nicotinamide adenine dinucleotide phosphate (NADPH), and ribulose-5-phosphate during oxidative phase (irreversible). This is followed by non-oxidative phase (reversible) comprising of pentose sugar interconversions. By oxidizing glucose into glycolytic intermediates including the conversion of pentose phosphates into glycolytic intermediates or other sugars like ribose-5-phosphate, overall pathway functions an anabolic process.

This makes insulin dependent sugars (sugars that require insulin to enter target cells) such as glucose, one of the key molecules that modulate cellular functions, genomic operations regulated genetically or epigenetically etc. Furthermore, they effect the accumulation of metabolites including the toxic composites that drive biological systems towards a genetically unprogrammed state such as seen in case of metabolic syndrome, in COVID 19 virus driven illness manifestations, in cancer and in other diseases which are the outcomes

of consequences of emergence of phenotypic individuality at cellular as well as at systemic and organismal levels. Metallic and non-metallic ions, micronutrients, biological factors, alongside intracellular cholesterol biosynthesis Cu-Zn superoxide dismutase (sod1p) and glutathione (GSH) levels, and other key drivers modulating shifts in oxidative stress and homeostasis balance equilibrium give rise to altered cellular and organismal energy demands and aging process that dictate cells to decide their fate ranging from cells death to evolution of well adopted cell phenotypes with or without undergoing premature cellular aging process or become cells surviving as cell population comprising of unhealthy deprived or damaged cells such as metabolically dormant cells. This variation in cellular responses is further influenced by localized access to steady supply of free molecular oxygen, metallic ions and electrolytes, nutrients depending on their types and levels, and other dietary factors, including Zn-Cu superoxide dismutase (sod1p), glutathione (GSH) and alongside intracellular cholesterol biosynthesis levels. Additionally, it is shaped by exposure to infectious and/or non-infectious agents, immunogens, among other factors including the broader impacts of cellular environments and climate change. One of the consequences of disturbed homeostasis is the appearance of a common pathological feature leading to the formation of plaque-like structures composed of cellular debris in vessels, in brain tissues and in other organs, escaping the death events in response of increased cell-to-cell variability. This is one of the outcomes of hemoglobin molecules driven oxidation particularly of cell membranes' cholesterol structure following the release of free iron during heightened oxidative stress. These findings suggest that food serves as a key driver for defining and modulating health status, immune profile, recovery and regeneration potentials, mental well-being and behaviors of individuals, mainly by virtue of sugars intake, depending on sugar types and levels, that appears as one of the primary factors, playing a critical role in dictating the cellular fate, thereby effecting the natural health, disease onset and recovery.

Natural health depends on types of nourishment and respiration

As previously discussed, in presence of free molecular oxygen and functional catalase (an enzyme that breaks down hydrogen peroxide into water) works alongside with Cu-Zn superoxide dismutase (sod1p) a dimeric metallo-protein containing one zinc ion (Zn^{2+}) and one copper ion (Cu^{2+}) per subunit, that scavenges and dismutates the superoxide anion (O^{2-}) into molecular oxygen (O_2) and water (H_2O), subsequently, all these enzymes catalyze the same reaction converting the oxygen radical in molecular oxygen (O_2) and hydrogen peroxide (H_2O_2) through the alternate reduction and reoxidation of Cu^{2+} moiety from Cu-Zn superoxide dismutase (sod1p); the hydrogen peroxide (H_2O_2) is then enzymatically converted by catalase and glutathione peroxidase in molecular oxygen (O_2) and water (H_2O), produced in the final stage of respiration process driving energy from nutrients and transforming them into products or in their precursors, particularly from glucose that undergoes insulin dependent absorption in the target cells to breakdown, producing instant energy ultimately releasing carbon dioxide (CO_2) and water (H_2O) as products.

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A scarcity of steady supply of free molecular oxygen, metallic ions and electrolytes, nutrients depending on their types and levels, and other dietary factors, including Zn-Cu superoxide dismutase (sod1p), glutathione (GSH) and intracellular cholesterol biosynthesis levels including heavy intake of insulin dependent sugars (sugars that require insulin to enter target cells) such as glucose are associated with the appearance of a common pathological feature in response of disturbed homeostasis leading to the formation of plaque-like structures composed of cellular debris in vessels, in brain tissues and in other organs, escaping the death events in response of increased cell-to-cell variability. This is one of the outcomes of hemoglobin molecules driven oxidation particularly of cell membranes' cholesterol structure following the release of free iron during heightened oxidative stress.

This makes insulin dependent sugars (sugars that require insulin to enter target cells) such as glucose, one of the key molecules that modulate cellular functions, genomic operations regulated genetically and/or epigenetically etc. Furthermore, they affect the accumulation of metabolites including the toxic composites that drive biological systems towards a genetically unprogrammed state such as seen in case of metabolic syndrome, in COVID 19 virus driven illness manifestations, in cancer and in other diseases which are the outcomes of consequences of emergence of phenotypic individuality at cellular as well as at systemic and organismal levels. Metallic and non-metallic ions, micronutrients, biological factors, alongside intracellular cholesterol biosynthesis Cu-Zn superoxide dismutase (sod1p) and glutathione (GSH) levels, and other key drivers modulating shifts in oxidative stress and homeostasis balance equilibrium give rise to altered cellular and organismal energy demands and cellular aging process that dictate cells to decide their fate ranging from cells death to evolution of well-adopted cell phenotypes with or without undergoing premature cellular aging process or become cells surviving as cell population comprising of unhealthy deprived or damaged cells such as metabolically dormant cells. This variation in cellular responses is further influenced by localized access to steady supply of free molecular oxygen, metallic ions and electrolytes, nutrients depending on their types and levels, and other dietary factors, including Zn-Cu superoxide dismutase (sod1p), glutathione (GSH) and alongside intracellular cholesterol biosynthesis levels. Additionally, it is shaped by exposure to infectious and/or non-infectious agents, immunogens, among other factors including the broader impacts of cellular environments and climate change. These findings suggest that food serves as a key driver for defining and modulating health status, immune profile, recovery and regeneration potentials, mental well-being and behaviors of individuals, mainly by virtue of sugars intake, depending on sugar types and levels, that appears as one of the primary factors, playing a critical role in in dictating the cellular fate, thereby effecting the natural health, disease onset and recovery [10,15-18,22-24,32- 36,38,42,47,49,52-56,58-61,63-96,98-107,109-161].

The link between nutrition-genes regulatory networks and energy demands - another narrative

As reported by Rab (2007) and Bishop., *et al.* (2007) that the *SOD1* gene, (that encodes for Cu-Zn superoxide dismutase (sod1p)), a protein that binds copper and zinc ions in its molecular structure, destroys free superoxide radicals including those generated by electron transport chain (respiratory chain) and the *CTR1* gene, which encodes for membrane copper transporter 1 protein (ctr1p), a cell membrane protein that is a high affinity membrane copper transporter, are linked through the same genes network operational regulatory pathway. These genes are reciprocally regulated at transcriptional level in response to copper concentration gradients modulated by iron-copper associated and dissociated ionic equilibrium balance gradient shifts, which drive oxidative stress shifts through a feedback mechanism. Genes encoding glutathione (GSH) biosynthesis lie upstream of pathways that reciprocally regulate intracellular biosynthesized cholesterol and vitamin D biosynthesis in presence of sunlight, that in turn modulate respiration modes, energy demands, cell aging and cell fate in genetically and/or epigenetically preprogrammed manners and also in genetically or epigenetically unprogrammed manners in response to unusual stimuli sensed as altering oxidative stress flux release leading to shifts in energy demand varies from cell to cell over time such as fluctuating oxidative stress seen in case of COVID 19 virus driven illness manifestations and in many neurodegenerative diseases where scarcity of free molecular oxygen and copper ions sustainable supply are the key drivers to modulate illnesses, by affecting the immune functions, regulating the responses against the driving stimuli such as oxidative stress and free iron catalyzed manifestations, augmented

by presence of non- physiological hemoglobin. They are cumulatively responsible for sustainable homeostatic disturbances and pH shifts affecting the cellular energy demands in diverse manners, which dictates the cells to decide their fate. These effects are transmitted at the organismal level modulating natural health, disease onset, and recovery. A scarcity of steady supply of free molecular oxygen, metallic ions and electrolytes, nutrients depending on their types and levels, and other dietary factors, including Zn-Cu superoxide dismutase (sod1p), glutathione (GSH) and intracellular cholesterol biosynthesis levels including heavy intake of insulin dependent sugars (sugars that require insulin to enter target cells) such as glucose are associated with the appearance of a common pathological feature in response of disturbed homeostasis leading to formation of the plaque-like structures composed of cellular debris in vessels, in brain tissues and in other organs, escaping the death events in response of increased cell-to-cell variability. This is one of the outcomes of hemoglobin molecules driven oxidation particularly of cell membranes' cholesterol structure following the release of free iron during heightened oxidative stress [10,20-41,46-50,52-56,58-114,117-172].

Intracellular cholesterol biosynthesis regulation and its multifaceted consequences

The oxidation of cholesterol particularly cholesterol biosynthesized besides altering cell membranes, physiology and structure, adversely affects vitamin D synthesis by reducing precursor availability, in addition to causing other harmful effects. The outcome of cholesterol oxidation present in erythrocytes is reflected in the altered physiology of cell membranes affecting the selective permeability and oxygen carrying potential of red blood cells (RBCs) [172,173]. The binding affinity of hemoglobin depends on free oxygen attachment to hemoglobin gradient that is dependent on pH. This pH dependency is lost in case of hemolysates [174,175] generated during hemolysis leading to accumulation of free iron and denatured partially degraded hemoglobin. Any event that destabilizes the structure of hemoglobin as seen in advanced stage of COVID 19 virus driven illness manifestations, diminishes its oxygen binding potential. Simultaneously alterations in membrane structure play a critical role in dispersing these effects. This is evident in case of COVID 19 virus driven manifestations, where COVID 19 viral proteins invade target cell membranes destabilizing the cholesterol infrastructure and affecting transporter proteins. This process augments oxidative stress, leading to extensive intracellular cholesterol biosynthesized molecules' oxidation and selective expression of viral genes that increase energy demands varyingly within affected cells [176]. Cu-Zn superoxide dismutase (sod1p) molecules, a prerequisite for energy generation via aerobic respiration particularly for insulin dependent sugars (sugars that require insulin to enter target cells) lack dismutase activity in healthy cells but acquire peroxidase activity in certain diseases, such as in cancer under scarcity of free molecular oxygen and copper ions sustainable supply [102]. This results in uncontrolled augmented shifts in dispersed oxidative flux, that spread from cellular level to affect the physiological systems and bodily functions, adversely affecting natural health leading to the onset of disease and poor recovery. Heme biosynthesis involves mitochondria, which can disturb iron homeostasis under sustained stress conditions [177]. Increased oxidative stress in response to the deficiency of Cu-Zn superoxide dismutase (sod1p) activity in red blood cells (RBCs) augments triggered autoimmune responses. In contrast, the intake of antioxidants may prevent certain autoimmune responses by maintaining an appropriate redox balance in red blood cells (RBCs) and in other targeted cells [178]. Cu-Zn superoxide dismutase (sod1p) functional deficiency is reflected as elevated levels of intracellular nitric oxide (NO) in red blood cells (RBCs), as seen in COVID-19-virus-infected-patients [179,180]. The crosstalk between Cu-Zn superoxide dismutase (sod1p), calcium (Ca) ions and other metallic ions connects neuron and muscles through co-regulated interconnected junctions; this process is regulated at transcription level by genes lying within shared gene operational regulatory networks [143]. Enhanced Cu-Zn superoxide dismutase(sod1p) enzymatic activity which is dependent on metallic ions availability and the homeostatic balance equilibrium between iron and copper prevents initiation of apoptosis [68]. Glutathione (GSH) modulates apoptotic process; for this reason glutathione (GSH)-depleted cells do not undergo apoptosis in an epigenetically driven manner [68,133,134]. Lower activities of Zn-Cu-superoxide dismutase (sod1p), catalase, and glutathione peroxidase (GSH-Px) have been reported in the red blood cells (RBCs) of elderly people. These lower levels indicate an impairment of antioxidant defense during aging in biological entities, occurring at both cellular and physiological systemic levels. Furthermore, the appearance of intensified peroxidized lipid structures suggests an imbalance in intracellular cholesterol biosynthesis. This is accompanied by altered energy demands that vary from cell to cell over time [113].

Link between energy demand-gene network operation regulation, biological events and cell fate

Published literature suggests that the redox status of the cell is one of the key drivers mediating the apoptotic pathway in which glutathione (GSH) plays a critical role in driving apoptosis either via nitric oxide (NO) and reactive oxygen species (ROS) or independently of nitric oxide (NO) whereas enzymatically functional Cu-Zn superoxide dismutase (sod1p) resists the initiation of apoptotic process under steady supply of copper and free molecular oxygen.

The body's energy demand and expense depend on many factors; however, latest understanding of genome has altered the conventional narrative on nutrition, suggesting energy demand varies from cell to cell over time depending on circumstances.

Asprosin is a fasting-induced hormone that promotes hepatic glucose production. Plasma asprosin crosses the blood-brain-barrier and directly activates Agouti-Related Peptide (AgRP) (a powerful appetite-stimulating (orexigenic) neuropeptide produced in the hypothalamus that strongly promotes feeding and reduces energy expenditure) production in orexigenic AgRP+ neurons via a cyclic adenosine monophosphate (cAMP)-dependent pathway. This signaling results in inhibition of downstream pro-opiomelanocortin (POMC) production from anorexigenic POMC+ neurons in a gamma-aminobutyric acid (GABA)-dependent manner, resulting in appetite stimulation and leading to increased adiposity and body weight [181,182].

The connection between augmented oxidative stress in COVID 19 virus driven illness manifestations including lethargy and weight loss stems from a failure to meet heightened energy demands. This deficit results from impaired metabolic cascades that modulate cellular and organismal physiology and from lower level of asprosin (glucogenic hormone) a hormone that affects different metabolic pathways including glucose metabolism. The decrease in copper supply caused by diminished neutral amino acid absorption following the expression of Angiotensin-Converting Enzyme 2 (ACE2) protein during COVID 19 virus infection in targeted cells is a problem that can be resolved through nutritional supplementation. The underlying reasons reveal a new role for metallic ions and oxygen scarcity in causing extreme weakness and weight loss in COVID19 patients. This occurs through unprogrammed shared gene networks operational regulation at transcription level targeting the metabolic cascades [183,184].

Universal evolutionary capacitor switch-shared pathways lead to common consequences

Multicellular organisms evolve from a zygote according to a given respective preprogrammed genomic roadmap that dictates developmental cascades' pathways. These developmental pathways that sustain individual phylogenetic characteristic features, exist within respective species. They vary among individuals within a given specie, depending on exposure of triggering stimuli, biological events and environmental factors that can mimic common outcomes when similar sequences of events take place at same intervals in response of given triggering stimuli initiating them under the control of universal evolutionary capacitor switch, such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch driving regulatory circuit through universal evolutionary switch complex such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch complex regulating the common cascades' pathways [20-41,139]. A detailed paper by the author, covering different aspects in this area has already been published.

For instance, structural pathologies found in platelets and erythrocytes, together with spontaneously formed amyloid microclots [185] mimic the amyloid formation that is a common feature associated with progression of neurodegenerative diseases and aging [25,32-35,49,55,56,58-62,64-66,68-74,78,79,82,86-88,91,101,102,106,107,113,114,118,119,126-129,132-134,138,143-145,162,183,186-196].

Evidence of these features in brains of some COVID 19 infected patients confirms that COVID 19 virus triggers altered biological events initiated by elevated, fluctuating oxidative stress. These events involve heightened cellular energy demand that varies from cell to cell over time [25,49,55,56,58,59,61,62,64-66,70-74,78,79,82,88,91,101,102,106,118-120,126-129,132,138,143,144,150-156,158,164,165,176,179,180,183,187,192,194,195,197-203].

In addition to this, COVID 19 infected patients suffer from thrombotic microangiopathy, disseminated intravascular coagulation, and large-vessel thrombosis; they also demonstrate emergence of ground-glass opacities similar to those ground-glass opacities, seen in progression of *Mycobacterium tuberculosis* infection [35,36,49,55,56,58,59,61,62,64-66,68,70-74,78,82,86-88,90,92,99,101,102,107,114,119,126,128,132,135,138,151,153,155,156,165,173,175,176,179,180,183,185,197,201-205].

As discussed earlier, appearance of similar pathological features in different types of illnesses such as those found in lungs and in brains, confirming the common role of universal evolutionary capacitor such as universal evolutionary capacitor switch, such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch driving regulatory circuit through universal evolutionary switch complex such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch complex. This universal evolutionary capacitor switch, such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch driving regulatory circuit through universal evolutionary switch complex such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch complex modulates ionic homeostasis disturbance, copper and free molecular oxygen scarcity, and iron overload. These factors varyingly affect cellular molecular oxygen and energy demands, resulting in heterogeneous cellular premature aging as an outcome of targeting the co-regulated interplay of Cu-Zn superoxide dismutase (sod1p), glutathione (GSH), cellular cholesterol biosynthesis [55,56,58,59,61,62,64-66,70-74,79,82,88,91,92,101,102,106,107,118-120,126-129,132,138,143,144,164,165,176,179,180,187,192,194,195,197-199].

The scientific interpretation of impact of diet on the body

Literature describes that a scarcity of sustainable molecular oxygen supply is mostly coupled with an inadequate availability of copper. This deficiency impairs mitochondrial functions by reducing oxygen consumption, thereby augmenting oxidative stress-triggered damage, lipid peroxidation and disturbed glucose metabolism. Furthermore, reduced mitochondrial respiration drives abnormal metal ions distribution, specifically manganese copper and zinc alongside accumulation of scattered free iron clusters [62,146,158,206,207].

Reduced mitochondrial manganese may be coupled with mitochondrial dysfunctions, likely due to decreased activity of mitochondrial manganese-dependent superoxide dismutase, an enzyme that protects mitochondria from respiration-generated free superoxide anions [206,208,209]. Parallel to this, hyperferritinemia (scattered free iron loads) drives a disturbance in mitochondrial homeostasis, shifting mitochondrial respiration from an aerobic to an anaerobic state. Anaerobic respiration favors pyruvate reduction into lactate, catalyzed by lactate dehydrogenase (LDH), which is a highly upregulated marker in COVID-19 virus driven illness [155,210,211-216].

Additionally, iron overload, specifically in form of loosely bound iron is a major concern. This is due to its potential to catalyze production of reactive oxygen species (ROS) across various pathological scenarios, sharing the common features, indicating the occurrence of common biological events leading to a disease onset including neurodegenerative diseases, COVID-19 virus driven illness manifestations and tuberculosis (TB). These conditions are governed by common genes operational regulatory networks, targeting the common metabolic cascades [62,78,119,126,132,217-226].

Literature indicates that iron overload, reduced glutathione (GSH) and mitochondrial superoxide facilitate *Mycobacterium tuberculosis* to trigger ferroptosis of infected macrophages [90,133-135,227].

It has already been established that release of unbuffered oxidative stress fluxes, exhibited in response to a given biological event or injury, targets cellular components, including mitochondria and lipids etc., and interferes with cellular integrity, membrane fluidity, and permeability [228-231].

Under the proposed model, dysbiosis occurs in response of different biological events such as COVID 19 virus infection driven manifestations. These infections drive shifts in oxidative and pH, augment varyingly cellular energy demands and alter sequestration

potential and competitiveness for chelating electrolytes and other chemical and biochemical species including metallic ions. These factors alter the normal flora and gut physiology, often by increasing intestinal mobility [205]. Agents with sequestration potentials, specifically capable to chelating electrolytes and metallic ions, can act as carrier molecules or ligands, particularly for metallic ions. Specifically, by modulating the supply of metallic ions such as copper ions and iron ions, these agents facilitate to restore enzymatic role of Cu-Zn superoxide dismutase (sod1p) which regulates cellular energy generation process affecting systemic energy demands [78,93,156-162].

Food can be a critical source of loaded Cu-Zn superoxide dismutase (sod1p) (Cu-Zn superoxide dismutase (sod1p) refers to the copper (Cu^{2+}) and zinc (Zn^{2+}) bound, enzymatically active form of the Cu-Zn superoxide dismutase (sod1p) protein molecules) for the body. It plays a vital role in protecting against a wide range of illnesses and modulates both the onset of illnesses and the recovery process. These bioactive entities play additional specialized chemical and biochemical roles when used as active pharmaceutical ingredients or as a part of food-based diet preparations or in combinations with antimicrobial anticancer etc. drugs including the drugs those have lost their efficiency; for instance, drugs those have become inactive against resistant infectious agents. Research suggests that they can cure various illnesses and offer a prophylactic role during prolong use. For example, use of diet preparations, such as chicken nutrifortified broth and ginger fortified honey mixture, played a significant role in eradicating COVID 19 virus pandemic across the globe [1,5,7-10,13-19,150,160,199,232]. The three main challenges of drug driven recovery include its absorption in the blood, penetration across biological barriers, and unaltered transport to the target site with sustainable activity and acceptability by the most of the host bodies. Diet preparations alone exhibit all these characteristic features at negligible cost and in an environment friendly manner. In contrast, other medicated or non-medicated candidates carry a wide range of severe risks, adversely affecting both the environment and health of consumers [233].

Unusual brain roles-electrolytes metallic ions-the key players for brain function modulation

Recently published literature reveals unusual roles of brain, which operates independently at nervous system level and also through its glands. These interconnected systems work in coordination, modulated by shifts in oxidative stress status and altered homeostasis; together they act as primary drivers for governing health status, illness onset and recovery. The hypothalamus and pituitary glands are involved in modulating electrolytes balance, partly by sustaining homeostasis and buffering potential against systemic and cellular oxidative stress flux shifts. They regulate the roles of common drivers including iron, copper and calcium ions balance, independently, across the nervous system and body physiological systems. Furthermore, they regulate the function of thyroid gland by building a web of interconnected, co-regulated circuits across the physiological systems, connecting cells by sharing the common environment, modulating the preprogrammed genomic operation via interconnected genes regulatory networks that respond to transmitted signals, act as triggering stimuli when homeostasis is altered, ultimately resulting in release of unbuffered oxidative stress bursts. In this process, universal evolutionary capacitor switch such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch drives regulatory circuit through universal evolutionary switch complex such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch complex partly by its varying molecular stereochemistry which plays a key role in modulating interconnected co-regulated circuits. Through the varying biochemical and chemical roles, universal evolutionary capacitor switch, such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch driving regulatory circuit through universal evolutionary switch complex such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch complex connects physiological systems with cellular gene-regulation networks, modulating metabolic cascades and/or exhibits other roles in the body involving cells and their cellular gene regulatory networks [63,113,143,148,149,234-244].

Brain unusual role modulates its unusual functions

Brain is mainly composed of cholesterol, which is synthesized by brain cells. These cells build an isolated ecosystem that is partly partitioned by the blood brain-barrier, making access to brain limited and selective. Consequently, brain physiology and pathology

differ from rest of the body. They are driven by molecules such as Cu-Zn superoxide dismutase (sod1p), glutathione (GSH), intracellular biosynthesized cholesterol and free molecular oxygen levels and by glucose including other insulin dependent sugars (sugars that require insulin to enter target cells) and insulin independent sugars (sugars that do not require insulin to enter target cells) etc. Furthermore, these processes are modulated by electrolytes and metallic ion balance equilibrium; for instance, shifts in the iron-copper associated and dissociated ionic equilibrium balance gradient drive oxidative stress shifts.

As previously mentioned, for healthy body to function, glucose is broken down under steady supply of molecular oxygen and copper. Furthermore, copper must be loaded onto Cu-Zn superoxide dismutase (sod1p) molecules for dismutasing peroxide ions produced during oxidative respiration and to mitigate oxidative stress shifts released as an outcome of localized reactions producing peroxide anion. Under excessive supply of sugars requiring insulin for their absorption in target cells, a sudden spike in glycemic index occurs on commercial food products intake, including high sugar containing drinks, leading to deficiencies in free molecular oxygen, copper ions, loaded Cu-Zn superoxide dismutase (sod1p), glutathione (GSH) etc. Consequently, shifts in intracellular biosynthesized cholesterol levels drive glutathione (GSH) synthesis imbalance or glutathione (GSH) cycle imbalance, particularly in the brain, leading to an accumulation of free iron loads, which also occurs with consumption of non-halal or non-kosher meat. This results in exhibiting insulin resistance, contributing to the onset of metabolic syndrome. This process is driven by the uncontrolled oxidation of intracellular biosynthesized cholesterol, accumulation of unloaded Cu-Zn superoxide dismutase (sod1p) molecules as well as Cu-Zn superoxide dismutase (sod1p) molecules that lack dismutase activity but exhibit peroxidase activity especially under hypoxia condition in presence of unbuffered redox agents. Under scarcity of oxygen, copper and in presence of unbuffered oxidizing and reducing agents, these shifts drive uncontrolled oxidative stress fluctuations. They alter the role of various chemical and biochemical entities and organelles by involving universal evolutionary capacitor switch such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch driving regulatory circuit through universal evolutionary switch complex such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch complex regulated cascades' pathways that determine the fate of cells, choosing between apoptosis ferroptosis necrosis or autophagy or leading to evolution of well-adopted cell phenotypes with and without undergoing cellular premature aging. These cell phenotypes may exhibit altered characteristics; capable to form amyloid microclots found to be related to Cu-Zn superoxide dismutase (sod1p) molecules aggregates which deposit in body vessels, particularly in brain. The deposition of amyloid protein (amyloid β protein) molecules triggers the initiation of neurodegenerative pathological cascade similar to those seen in case of COVID 19 driven manifestations; both appear to be driven by electrolytes and metallic ion balance equilibrium shifts such as by iron-copper associated and dissociated ionic equilibrium balance gradient shifts. These shifts drive oxidative stress fluctuations and time-dependent variably altered cellular energy demands, suggesting that dietary interventions are the primary means to inhibit the onset of these illnesses with common pathological cascades [10,46-50,52-56,58-66,68,70-108,118-120,126-129,132,133,136,138-140,143-146,150,157-162,188,196,207,208,210,211,217-223,232,243-250].

An unnatural or hyper-processed commercial diet which is devoid of natural source of copper, particularly rich in insulin-dependent sugars (sugars that require insulin to enter target cells) and high in salts when consumed regularly can increase the risk of oxidative stress driven illnesses onset. These include metabolic syndrome, neurodegenerative and autoimmune diseases. This may diminish healing potential by altering enzymatic roles, activity and stereochemistry of Cu-Zn superoxide dismutase (sod1p) molecules [3,4,7,11,12,93,123,150,151,153,159-161,165,166,187,192,195,199,201,220,242,250].

Nutrition connecting neurobiology with psychology

The role of calcium ions in neuronal signal transduction is mediated by several calcium ions Ca(2+)-binding proteins, such as calmodulin, calcineurin, and synaptotagmin. Structural changes in response to shifts in calcium ion concentration allow these proteins to function in memory formation and other neurochemical roles. Metallochaperones help to achieve metal ion homeostasis and thus

prevent the onset of neurological diseases mainly triggered by metal ions imbalance. The concentration of copper in the brain is partly controlled by metallothionein, and zinc is released in the hippocampus at glutamatergic synapses. Glutamate, the principal excitatory neurotransmitter of the brain, participates in a multitude of physiologic and pathologic processes, including learning and memory. It modulates the concentration of glutathione, a tripeptide comprising glutamate cysteine and glycine which plays an additional critical role as an antioxidant. This provides a functional junction (bridge) between brain physiology and pathology, modulated by shifts in oxidative stress flux bursts release that acts as a common stimulus for various cascades. Glutathione (GSH) may serve as a reservoir of neural glutamate because of its high concentration in brain. Inhibition of the synthesis of glutathione (GSH) in the brain may trigger sustained, heightened oxidative flux release. This is demonstrated by impaired cognitive functions, such as reduced learning and memory capacity, diminished hippocampal neurogenesis, and an altered dendritic network. These characteristic features are universal in the aging brains, providing further evidence of its multifaceted roles played by involving, universal evolutionary capacitor switch such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch driving regulatory circuit through universal evolutionary switch complex such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch complex serves as a junction, providing a common connection for cascades involved in modulating the accumulation of enzymatically inactive copper-unloaded Cu-Zn superoxide dismutase (sod1p) molecules and copper-loaded Cu-Zn superoxide dismutase(sod1p) molecules deprived of dismutase activity but exhibiting peroxidase activity particularly under molecular oxygen scarcity in presence of unbuffered oxidizing and reducing agents. This process drives uncontrolled oxidative stress shifts producing reactions, altering the roles of chemical and biochemical entities and cellular organelles in brain and transmitting the consequences to rest of the body.

Dyshomeostasis of redox-active metal ions, such as copper and iron appears to be a key driver in the brain that triggers the initiation of degenerative diseases including Alzheimer's disease (AD). Regulating the levels of copper ions with different oxidation states (such as cuprous ions denoted by copper (I) and cupric ion denoted by copper (II) in terms of oxidation-reduction states) Cu(I/II) and the levels of iron ions with different oxidation states (such as ferrous ions denoted by Fe(ll) and ferric ions denoted by Fe(lll) in terms of oxidation-reduction states) Fe(II/III) and their interdependent balance equilibrium is essential for normal brain function. This is modulated through universal evolutionary capacitor switch such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch driving regulatory circuit through universal evolutionary switch complex such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch complex driving physiological cascades' pathways regulatory operation and glutathione (GSH) and glutamate interdependent functional levels. By regulating physiological cascade pathways' regulatory operation and glutathione (GSH) and glutamate interdependent functional levels, it protects cellular biosynthesized cholesterol from oxidation to maintain normal functions of brain and body in interdependent manner, effectively bridging neurobiology with psychology to support healthy behavior. In other words, nutrition bridges neurobiology and psychology. This connection is regulated by the universal evolutionary capacitor switch such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch driving regulatory circuit through universal evolutionary switch complex such as *SOD1* gene encodes for Cu-Zn superoxide dismutase (sod1p) - Cu-Zn superoxide dismutase (sod1p) (*SOD1*-sod1p) switch complex mainly modulated in interdependent manner by glutathione (GSH) and cellular cholesterol biosynthesis levels, by metallic ions levels and by their interdependent association and dissociation equilibrium balance gradient shifts, by sustainable free molecular oxygen supply and by insulin dependent sugars (sugars that require insulin to enter target cells) and insulin independent sugars (sugars that do not require insulin to enter target cells) levels.

The literature indicates that Cu-Zn superoxide dismutase (sod1p) molecules play additional roles in regulating metabolism. These include modulating cholesterol metabolism by regulating energy flow, sensing micronutrients to bridge neurophysiology and neuropathology as well as influencing muscle functions by co-regulating mitochondrial respiration [53,56,59,60,71,74,75,78,79,82,84,88,93,97,102,106,108,114,118-120,126-129,132,133,135,138,139,143-145,150,161,166,186-193,195-201,207,208,211,218-222,229-231,243-255].

Current concepts in nutrition science

However, the latest understanding in nutrition science, revealed in reported data by Rab (2007) and Bishop., *et al*. (2007) [256] has challenged the findings of Schlessinger., *et al.* (2011) [257] which suggested a mutual relation among defects in proteins, cells survival and disease. This shift in understanding has transformed the previously established concepts of nutrition science [2,245].

Research reveals that cumulative impact of electrolytes intake, metallic ions intake, insulin dependent sugars (sugars that require insulin to enter target cells) and insulin independent sugars (sugars that do not require insulin to enter target cells) intake, architecture of food, origin of food ingredients such as naturally derived from biologically origin or synthesized or extracted from biological and/or non-biological sources, their combination composing the food items and their processing etc. the extent of absorption in blood and their post-absorption metabolic roles etc. drives the health, mental well-being and physical status of the body, partly by influencing the oxidative stress shifts and their consequences, in addition to their individual multifaceted roles depending on many other factors.

For instance, glucose, but not fructose, ingestion suppresses the activation of the hypothalamus, insula, and striatum-brain regions that regulate appetite, motivation, and reward processing; glucose ingestion also strengthens functional connections among the hypothalamic-striatal network and increases satiety. The disparate response to fructose intake is associated with reduced systemic levels of the satiety-signaling hormone insulin. The underlying reason may be an inability of fructose to cross the blood-brain barrier into the hypothalamus or it may be the exhaustion of copper pools-down-regulates or inhibits the hypothalamic expression of genes necessary for fructose metabolism. Previous investigations revealed that the central metabolism of glucose suppresses food intake through the hypothalamic adenosine mono phosphate (AMP)-activated protein kinase (AMPK)/coenzyme A derivative of malonic acid (AMP-kinase/malonyl-CoA) signaling system. Unlike glucose, centrally administered fructose actually increases food intake. Evidence presented herein indicates that the more rapid initial steps of central fructose metabolism deplete hypothalamic adenosine triphosphate (ATP) levels, whereas the slower regulated steps of glucose metabolism elevate hypothalamic adenosine triphosphate (ATP) levels. Consistent with effects on the [adenosine triphosphate]/[adenosine monophosphate] [ATP]/[AMP] ratio, fructose increases phosphorylation/activation of hypothalamic adenosine monophosphate (AMP) kinase causing phosphorylation/inactivation of acetyl-CoA carboxylase, a vital, biotin-dependent enzyme that catalyzes the crucial, rate-limiting step in fatty acid synthesis: converting Acetyl-CoA into coenzyme A derivative of malonic acid (malonyl-CoA) providing the building blocks for fats whereas glucose has the inverse effects. The changes provoked by central fructose administration reduce hypothalamic, coenzyme A derivative of malonic acid (malonyl-CoA) levels and thereby increase food intake [245,258-261].

Another interesting finding revealed in the data reported by Rab (2007) shows that under non-physiological states, such as under stress conditions, cells, their organelles, biochemical and chemical entities deviate from innate paradigms, to drive the cells in genetically and/or epigenetically programmed manners as well as in genetically and/or epigenetically unprogrammed manners in parallel. These parallel processes lead to consequences that differ significantly from those observed under physiological conditions for a given set of parameters [32,47,56,262,263].

Latest nutrition science understanding based diet preparations

Following are diet preparations designed by using latest understanding of nutrition science, which have multifaceted health roles

Pomegranate fruit mix juice

Peel 2 - 3 pomegranate fruits, add 3 - 4 teaspoons of brown sugar, ½ - 1 teaspoon of unpasteurized organic honey to a blender and then blend the mixture for 5 - 7 minutes with intermittent intervals of 30 seconds. Leave the ground pomegranate fruit mix sit at room temperature for an hour. Add 2 liters of potable water. Mix it well; refrigerate it in a glass bottle with a lid on. Drink 2 glasses of pomegranate fruit mix juice daily; shake well to ensure that ground seeds are included. Mix 2 - 3 teaspoonfuls of psyllium husk in lukewarm water to consume daily, ensuring a 5 - 6 hours interval between taking pomegranate fruit mix juice and psyllium husk mixture.

Consuming preparations like pomegranate fruit mix juice and psyllium husk water can decrease visceral fat while improving the sleep quality and duration.

Pomegranate apple grapefruit mix juice

Peel 3 - 4 pomegranate fruits, and cut 1 red apple (with peel) into small pieces. Add fresh juice extracted from 1 grapefruit, 150 ml of potable water, 3 - 4 teaspoons of brown sugar and ½ - 1 teaspoonful of unpasteurized organic honey to a blender then blend the mixture for 9 - 10 minutes with intermittent intervals of 30 seconds. Keep ground pomegranate apple grapefruit mix juice at room temperature for 1½ hours. Add 3 liters of potable water. Mix it well and refrigerate it in an airtight glass bottle with a lid on. Drink two glasses of pomegranate apple grapefruit mix juice daily after shaking; it contains ground seeds. Pomegranate apple grapefruit mix juice is helpful in preventing and treating many virus infections, particularly chikungunya viral disease.

Results and Discussion

A body of knowledge including in nutrition science has been overturned by Rab (2007) and Bishop., *et al.* (2007), whose findings represent the most groundbreaking discoveries of this era [32,33,262,263,265]. In addition, these finding have raised questions regarding work of Schlessinger., *et al*. 2011 which indicate a mutual relation among defects in proteins, cells survival and disease [257]. This paper utilizes the data reported by Rab (2007) and Bishop., *et al.* (2007) [32,33,262,263,265] to explain basic concepts in nutrition science through alternative scientific narratives. The composition, processing, origin of food ingredients, their microstructure and the combination of food items consumed and their absorption potentials, etc., cumulatively play a key role in modulating physical health, mental well-being and illness onset by influencing the oxidative stress shifts driven by sugars, electrolytes and metallic ions etc. intake. Finally, a few diet preparations have been reported which can be used as diet therapy or an adjunct therapy [32,33,262-265].

Conclusion

Most of the current understanding of biological sciences, including nutrition science, became invalid following the data reported by Rab (2007) and Bishop., *et al* (2007) [32,33,262,263,265]. It has raised questions about the validity of the work by Schlessinger., *et al*. (2011) whose findings indicate a mutual relation among defeats in proteins, cells survival and disease) [257]. It is the need of time the hour to critically and independently review published literature to differentiate between legitimate and fraudulent work. Doing so is essential for protecting global public health and ensuring that scientific progress remains on the right track [32,33,262-265].

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