A diaeventontological paradigm for brain dysfunction research

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Brain disorders, like all pathologies, have a contributing genetic component, if not via direct mutation such a nucleotide polymorphism, copy number variation or splice variation of a suite of structural genes. This genomic contribution includes gene products that (for example) regulate neuroimmune responses, subcellular signal transduction, programmed cell death, autophagy, or the driving of action potentials, synaptic trafficking and the cycling of membrane polarization. This genetic contribution can be well described or occult but, in neither case, does it fully explain the presentation of neuropsychiatric disorders just as any human disease cannot be explained by examining dysfunction in healthy physiological processes as underscored by myriad biochemical events. There are two other interacting and cohering mechanisms which must be included in order to begin an architectonic reasoning of human disease of which neuropsychiatric disorders are the most complex and least understood. The second member of this trigonal planar network to consider is the molecular cellular and physico-chemical environment which is in constant flux both internally and externally. For example, the environment within the CNS is changing stochastically with respect to oxidative metabolism and the production and subsequent removal of reactive oxygen and nitrogen species. These inorganic compounds move through autocatalytic and metal-ion activated oxidation states that produce highly reactive free radical unpaired electrons that will partially reduce other molecular species including fatty acid and cholesteryl ester unsaturated lipids. These oxylipids then induce local inflammatory responses that can further damage membranes and macromolecules such as protein, RNA and DNA. These interactions, when occurring in the CNS at specific neural loci, impart the prodromal phases of neuropsychiatric pathologies and thus interact with the genome either by removal of the toxic molecular environment or a now mutated axis of dysregulation leading to frank disease with its presentations and deficits as described in psychiatry. The external environment including biofuel accommodation (glucose vs. ketone bodies) oxygen availability including ischaemic and traumatic brain injuries and unconditioned senescence -biological aging- are all equally involved in the process. Finally, the natural-native system that encompasses all of the features describing this neuropsychiatric interactome and its axis is the systemic immune system. The neuro immune system offers defensive and offensive biochemical pathways and in conjunction with epigenetic mechanisms, generates the existing individual. This is a perpetuating neural network that can learn, via attention and ascent to stress, to function within the world. Such a biologically adaptive phenomena is accomplished via homologous recombination of variable regions of both the immunoglobulin family and the T Cell receptor in concert with chromatin remodeling, the Histone code and both the acetylome and methylome of cohering DNA. This connection might be the molecular and cellular adaptive immunological interactome that serves to generate neural tracts according to developmental, endocrine and peripheral stimuli while maintaining repair processes in the CNS by using the complex interactions among astrocytes, oligodendrocytes, microglia and neurons. An event ontology that reaches across the neural network of genetic and environmental interaction

is articulated and synthesized through epigenetic modifications of the classical immune system thus creating a responsive plastic and elastic memory field capable of learning, ideation, imagination and understanding through time. This diaeventontologicome involves the central existing individual agentically interacting through the events obtained across the inherited genome, the changing environment and the transcendental immunoepigenome through time. This presentation will address the potential to take a paradigmatic shift in our research and therapy for brain disorders. Here I will implement this diaeventontological perspective within current neuroscientific research, including brain disorders.

Biography

Daniel Guerra has completed his Ph.D. in Biochemistry and Physiology-Plant Science at Utah State University in the year 1984. In 1981 M. S in Plant Biochemistry at University of Arkansas and in 1978 he did his B. S in Agriculture, Agronomy at University of Illinois. He has Authored Over 40 scientific publications.

As a founder and CSO of VerEvMed, his credibility emerges from well-established and highly respected US universities with over 35 years of experience in the biochemistry/biomedical field. His company is here to determine and explain the true evidence about biosciences, pharmaceuticals, therapies, diet, exercise, and how these services, substances and practices impact traditional health care and medical practice. Accuracy is our mission and authenticity our goal.

Podcast: Authentic Biochemistry https://podcasts.apple.com/us/podcast/authentic-biochemistry/id1454408625?mt=2

Teaching: He have recently taught General Biochemistry, Graduate Biochemistry, Lipid Biochemistry and Physiological Biochemistry, Graduate Seminar, Graduate Research Methods and two additional graduate courses in the biochemistry, genomics and molecular biology of exercise physiology and nutrition in the Department of Nutrition and Exercise Physiology WSU Spokane Riverpoint Campus. He offered students a variety of biochemical, and molecular biological, courses depending on the needs of the department. These included any of the following courses he have previously taught at university: biochemistry (basic, graduate, physiological, clinical), biotechnology, genetic engineering, lipids and membranes, physiology, anatomy, molecular biology, genetics, cell biology, microbiology, pharmacology, forensic biochemistry, neuroscience, molecular microbiology, research methods and instrumentation, and graduate human nutrition. He have taught several other courses for NEP including professional development, cellular signaling and research design and continue to develop these in the future.

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