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Auto-infammation is a Diaeventontological Trigger in Neuropsychiatric disorders

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Single biochemical or cellular events cannot be expected to explain complex neuropsychiatric disease and, while animal models are indispensable for pharmacotherapy and pharmaceutical discovery, they will not fully apprehend these human disorders. Clinical studies have the advantage of authentic examination of human neuropsychiatric pathology, but they cannot holistically arrive at sound theories or practice for surmounting these debilitating diseases, even when precise molecular tools are employed. is is because 'Systems' approaches such as the standard OMIX platforms or cell Sorting/Screening from

However, the environment also generates a 'plastic response' on the chromatin that leaves behind covalent modi cations including methylation of CpG islands and acetylation of lysine residues of histone proteins in association with duplex DNA. ese epigenetic alterations, along with micro-RNA mediated control of mRNA translation, function to maintain a perpetual existing into each successive temporal sequence and therefore represents the unique manifestation of each existing individual as a perpetual becoming at the molecular and cellular levels.

is is the neuroeimmunoepigenome that ultimately carries the phenomenological signatures including healthy and pathological erefore, a paradigm shi is necessary to neuropsychiatric states. approach and indeed engage neuropsychiatric disease. A Diaeventontological method is therefore proposed. It starts with a dialectic approach that transcends dogma and conventional principles while preserving truths via coherence and foundational ascendency by isolating and verifying premises that can be used to generate sound epistemological arguments. is is sequentially followed by the generation of hypotheses via the deductive method by implementing careful hermeneutical analysis of both cross-sectional and cohort-based published research. Once theses/ antitheses / syntheses have been proposed according to a justi cation of truth qualia that better explains the necessary and su cient relational competency of rational foundational concepts, temporally centered experimentation using human subjects allow for a comprehension of biochemical interactions as a vectorial dynamic ux within cells/tissues/organs and entire organisms that recognize these as processes having three phases: Initiation, Extension and Termination. A case in point are Tlymphocytes that react in the intact human dynamic event ontology to respond to the environment, maintain cellular and physiological health and to prepare for future change that includes nutrition, neurological imprinting, disease and aging. T lymphocyte lineages and associated biochemical communication are modi ed via changes in the epigenome as well as canonical inducible and repressible gene expression and membrane recombination. Synergy between IL-12 and IL-18 for the induction of IFN- production and subsequent involvement of the heterodimeric IL-12 receptor leads to STAT4 phosphorylation a er recruitment of the kinases Tyk2 and JAK2. STAT4 then transactivates IFN- transcription and upon binding of IL-18 to its receptors, there is activation of the MAPK pathway downstream leading to the stabilization of IFNmRNA and enhanced IFN- secretion by NK cells. Secreted IFN- also activates B cells to mature to IgG producing plasma cells from germinal centers thus inducing a potential autoimmune disease when initial antigen presentation involves host metabolites.IL-12- co-activation of STAT1 and STAT4 mediates histone modi cation, with a sequentially expanding T follicular helper - 1-like cells activation and recruitment. When these biochemical and cellular pathologies align in speci c CNS nuclei, an autoimmune neuropsychiatric event may result. Using the conventional scienti c methods in neuroimmunopsychiatry needs to be ere is always an initial proposition that works as a premise which requires data as transformed into de nitive evidence (because of disinterested experimentation). is rst movement of the "classical method" functions a priori with the avor of both necessity and universality in subsequent neuropsychiatric or neuroimmune evaluations and diagnoses being prejudicially deployed as a foundation for treatment and conceptually, to inform fur(d co/W(iden)4 (ce)TJren)18

observer and the observed as eventuated by the observer himself. . ese changes can be followed by close scrutiny of the immune responses during treatment by examining innate immune pattern recognition receptors (e.g. TLR's) and their activation of circulating neutrophils and monocyte -macrophage lineages ultimately leading to dendritic cell mobilization to lymph glands thus involving lymphocyte-mediated acquired immune responses in the pathology of the neuropsychiatric status From these biological data , the analyst can have a discrete individualized neuroimmune event-status of each individual patient

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by analyzing patterns of change in cytokine, chemokine and growth factors in circulation. In my view, the ontology is the environment that also changes over space-time but is independent of its substance as de ned by the observer and yet only apprehended by his owned phenomenology. I conclude therefore that all science is approximation and any judgement based on research can lead to ideas only pretending to be justi ed true beliefs. Knowledge depends upon coherent and

foundational aspects of nature called truths plus argumentation based