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Auto-in fammation 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

followed discursively via n question involves an inclusion of change over spac

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