A Theoretical Alzheimer's Etiology Predicting Psychogenic Initiation

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Abstract. The cause of Alzheimer's Disease (AD) has been generally considered to be unknown; and even though there are over 50 causes of dementia over half of all demented patients suffer from AD. The 5 main etiological theories of AD are reviewed: 1. Genetic, 2. Toxic agent, 3. Infectious agent, 4. Immune function, and 5. Trauma. The noetic model presented is a radical new approach from a more fundament level than current thinking would produce and is an intimation that the 21st century might be called the 'age of consciousness' suggesting a revolution in medical prophylaxis, whereupon pathologies currently incurable will be treated or prevented. A whole new class of medical etiology, diseases of consciousness, that are the result of imbalances in the flow and coupling of the noetic field are the result of a noeti-taxic response (originating at a radically different physical locus than currently described by psychiatry, psychosomatics, psychoneuroimmunology or any other biological cause) defined as the 'Noetic Effect' is described as it applies to the genesis of AD. This model originates by applying the author's seminal work on 'Noetic Field Theory: The Quantization of Mind'. Preliminary empirical data in support of the model is discussed as it applies to gene activation, neurophysiology of the synapse and neurotransmitter function, Frohlich coherence associated with microtubules, and quantum brain dynamics; together a system forming a new physical cosmology of consciousness which may additionally include a long term EPR-like telergic associated factor that triggers or enhances the effects that produce amyloid deposits, neurofibrillary tangles or gene expression. Experimental protocols underway to further establish the theory are discussed as well as what kind of clinical intervention is possible; and how might it be applied to patients now and in the future in terms of both prevention and alleviation of the AD etiology.

Keywords: Alzheimer's Disease; Consciousness; EPR effect; Noetic field; Noetic effect; Telergic associated factor (TAF).

1.0 Introduction

The cause of Alzheimer's Disease (AD), first described by the German Physician Alois Alzheimer in 1907 (Mace & Rabins, 1981) has until now remained elusive. (Jenike, 1990; Berthoif, 1987; Mozar, Bal, & Howard, 1987). Even though there are over 50 causes of dementia, over half of all demented patients suffer from AD; this is one out of every six persons over the age of 65 (Kolata, 1981).

The diagnosis of dementia can be done using the DSM-III-R or IV criteria, with various intellectual impairments forming the basis of the diagnosis (Gottfries, 1988). Positive diagnosis can at this time only be achieved post mortem. However, a brain protein (Alz-50, found only in brains of AD patients, has been discovered. This finding could lead to a simple diagnostic test (Wolozin et al, 1986, 1987). Currently, clinical course and brain scans enable close diagnostic approximation. Psychological testing on dementia scales and certain aberrations in body language can correlate highly with AD. Specifically Smooth Ocular Pursuit (SOP) provides a sensitive indicator of brain function (Fletcher & Sharpe, 1988). During SOP there is an increased frequency of saccades. In testing SOP with varied frequency target motion, eye movements in AD patients failed to match test target movement within normal expectations necessitating large catch-up saccades.

Autopsy reveals a great incidence of Neurofibrillary Tangles (NT) and diffuse Senile Plaques (SP) in patients with AD. NT are abnormal 'twisting together' of neurofilaments in the axon. It is believed that the neurofilaments are involved in transport. Transmitter precursors flow from the cell bodies down the microtubules to the synapse and these NT could block this transport. SP are the 2nd anatomical marker of AD. They are areas of the cortex that contain degenerated nerve cells, an excess of glial cells, and deposits of an immune related substance called beta amyloid (Rozensweig & Leiman,

1982). The main transmitter dysfunction related to this neuron destruction is that of the acetylcholine system, significantly in the temporal neocortex, hippocampus, and amygdala. These cholinergic neurons are involved in arousal, REM sleep, pain perception, learning, memory, and thirst (Goldman, 1984). Thus producing the AD markers of cognitive decline and memory loss. Other transmitter systems including noradrenergic and seratonergic are also disturbed, but as yet less researched (Jenike, 1990). Hydergine, a metabolic enhancer, has been used for about 10 years in AD treatment (Jenike, 1985). But elevating arousal is only a primitive and limited aid. Also Physostigmine (Peters, 1979), is being tried; it blocks acetylcholinesterase, thus effectively allowing more acetylcholine to be present. But there is an optimal amount of this transmitter beyond which no improvement occurs, or function decreases. Only some patients are helped in a limited way at this time with Physostigmine. (Sitarm et al, 1978; Thai et al, 1983) Somatostatin, another neurochemical, levels along with cortical glucose levels are also found to be low in AD patients. Somatostatin has been found in NT & SP neurons. (Tamminga et al, 1987) Transmitter replacement strategies, although the latest treatment methodology, also do not seem to be attacking the root of the AD problem.

The Five main etiological theories of AD are reviewed:

- Genetic,
- Toxic,
- Infectious Agent,
- Immune Function, and
- Trauma.

(Jenike, 1985; Wurtman, 1985; Price, 1986)

2.0 Genetic

In five studies of first degree relatives of AD cases, four have shown an increased AD frequency. In a study with age of onset of AD over 70; statistical correlation could not be found. This has created a belief that there might be two types of AD, familial and random. (Chandra et al, 1987) Heston et al, 1981 also found risk similar to the general population for probands over 70. Chandra further states that an aggregation of cases within families does not delineate inherited factors; that the shared environment of families could be the explanation.

Seven dementia twin studies since 1945 have also yielded inconclusive evidence. If AD was of genetic origin disparities between Monozygotic (MZ) and Dizygotic (DZ) twins should be revealing. But there is no higher concordance rate in MZ twins than in DZ twins, both being about 40% concordance. Unless some unknown form of genetic alteration is at play an autosomal dominant cause of AD must be discarded and other environmental mechanisms searched (Nee et al,1987).

Jenike (1985) points out a study of identical twins (MZ) developing AD 12 years apart, to help explain the lack of concordance in time; and states that if the term of an AD patient is followed long enough the other twin might develop AD also. Too few twin studies have been done; and those with methodological problems. Because of the considerable insight twin studies could hold more methodologically sound work needs to be done. (Breitner et al, 1990)

Jenike further states that this implies that non genetic factors could influence the onset of the disease. This is probably true but it doesn~t necessarily reinforce the genetic etiology because it is not a complete explanation of the two AD types, familial and random after 70; and also the feeling that nearly everyone will develop AD if they live long enough. Jenike further reinforces the genetic position by noting that virtually all patients with Downs Syndrome develop AD if they live till about 40. Although the genetic theory is very attractive there is still too much discrepancy among the factors to substantiate a primary autosomal causality.

3.0 Toxic

Several studies have shown that rabbits and cats (Crapper et al, 1973) exposed to toxic levels of Aluminum (Al) develop NT. These NT are a little bit different than those found in AD patients. Data on elevated Al in AD patients has been conflicting and many cases show cerebrospinal fluid and serum Al levels to be normal in AD patients. Dialysis Dementia, a disease of elevated Al has no NT.

The experimental NT using Al intoxication are straight rather than twisted, and are dispersed in the brain stem and

spinal cord rather than only the cortex as in AD. The NT in cats was produced by cortical injection. Researchers promote Al as a neurotoxin (Bertholf, 1987) or pathogenic factor (Kruck, 1989; Birchall, 1988) creating a mineral imbalance in axonal transport, or the blood brain barrier, causing amyloid to form (Deloncle, 1990) or inhibiting magnesium requiring enzymes (Gluck, 1990) or releasing Al-silicate induced phagocytic free radicals. (Evans, 1989) In general there is increased brain Al content with aging; thus Al is not likely to be the responsible agent for AD (Jenike, 1985)

4.0 Infectious Agent

It has been conclusively shown that other degenerative neurological illnesses, such as Kuru, Creutzfeld~acob Disease (CJD), Gerstmann-Straussler syndrome, and Scrapie in animals are transmissible dementias caused by Prions,(Harrow et al, 1986) curious subviral agents with no nucleic acid (Prusiner, 1984A). These syndromes contain some degree of amyloid plaques like AD but not the NT.

The clinical findings are often so similar between AD and these diseases that they often cannot be distinguished in life (Goudsmit et al, 1980) Because Prion incubation can take decades it has been speculated that a Prion like agent could be involved in AD. But purified Prion brain extracts from AD patients have not been as transmissible in the laboratory as extracts from the proven Prion syndromes. So if Prions are involved in AD they must be different. (Prusiner, 1984B) It is possible the Prion molecule could change or adapt under some different physiological conditions in AD not yet discovered. AD NT have been induced using cultured human fetal cerebral neurones (DeBonni & Craper, 1978). Although recent evidence seems to exclude the Prion of CJD, or other viral or subviral agents; the transmissibility of AD has still not been totally ruled out.

5.0 Immune Function

Immune function abnormalities are common in AD patients. Serum protein variances, impaired cellular immune responses, and elevated levels of brain antibodies are some of these. (Jenike, 1985) The altered immune function could explain the presence of amyloid in senile plaques. And as mentioned earlier there is also a Somatin-like immunoreactivity involved in the plaques of AD. There is still insufficient evidence to draw any definite conclusion about immune related causes of AD (Jenike, 1985) The immune function theory is still the least researched; in view of the incompleteness the other etiologies provide perhaps the answer will turn up in the immune function area. Progress is being made; an immunoreactive undecapeptide called Substance P has also been found in senile plaques (Beal, 1987) which makes the immune function theory more attractive.

The immune system has been generally thought merely to distinguish between self and not self. Recent advances in the study of Autoimmunity, which is only just beginning to acquire a better understanding, has revealed that at finer levels of detail distinctions between self and not self are not absolute. In various autoimmune disorders the immune system has been found to attack healthy normal physiology (Cohen, 1988). It is possible further research could reveal an autoimmune response in AD; some of the unexplained details of AD seem to suggest this. Most recently behavioral interrelationships with neural and endocrine processes are being looked at with an eye to their effect on homeostatic mechanisms; (Ader et al, 1991, xxv.) and a framework for an autoimmune etiology of Ad with a psychoneuroimmunological formation of brain-reactive antibodies. (Forster & Harbans, 1991)

6.0 Trauma

People with head trauma or subdural hematoma, and thyroid disease are more prone to develop AD (Shalat et al, 1987; Graves et al, 1990; Gedye et al 1989). Also Parkinsons disease, brain tumors, Multiple Sclerosis, Epilepsy, and mental retardation correlate with AD (Chandra et al, 1987) Depression and the presence of extraprymidal signs of psychosis also lead to greater AD disability (Stern et al, 1987). This could mean that the geriatric condition in general creates a susceptibility to AD from an as yet unknown cause.

7.0 Discussion

Significant evidence supports each model of AD discussed, with none being mutually exclusive of the other (Bradley, 1990); making AD appear as a complex multi-factor syndrome. Recently all the same topical factors were considered as the pathogenesis of cancer; and all researchers were deemed correct. The time has approached to isolate and go beyond

the "elephantness" of AD. (Wurtman, 1985) This metaphor is of five blind men confronted by an elephant: One thinks of the tail as a rope; another the leg as a tree ; the trunk as a hose, the body as a wall, and the fifth thinks the ear is a large fan. The Current lens through which unification of the elephant is being attempted is that of premature aging. (Bosman & Bartholomeus, 1991; Rabinowe et al, 1989; Bertoni-Freddari, 1988; Masters & Beyreuther, 1988).

The nature of aging is an extremely difficult concept for science; however, aging has been postulated as being either genetically driven or of a stochastic process. It is not known if either or both is correct (Gershon, 1988).

Only weak correlational evidence supports programmed senescence. Cell cultures die off after about 50 doublings for human cells; proportionally less doublings for shorter lifespan animals, for example embryonic fibroblasts from rats and chickens double only about 15 times. Interestingly cells from midlife humans double only about 25 times (Hayflick, 1968).

The most significant of the random negative factors considered is free radical damage. The Harman "Free Radical Theory of Aging" is over 30 years old. (Gershon, 1988) There is free radical etiology support for each "elephant" factor. (Forster & Harbans, 1991; Cutler, 1991; Evans et al, 1991; Volicer & Crino, 1990; Bergtold, 1988) A correlation has been found between endogenous antioxidants and the lifespan of mammals; suggesting that oxidative stress is less in mammals with longer lifespans. (Cutler, 1991) This issue is being tested by measuring 8~hydroxydeoxyguanosine per deoxyguanosine levels in liver DNA and urine (Bergtold, 1988). The author suggests that if free radicals do play an additional putative role in the pathogenesis of AD; this urine biomarker could be also studied comparing AD patients with normal human population.

8.0 Noetic Nosology

Although the recent work in Psychoneuroimmunology and free radical theories of AD etiology represent a major leap forward; the author feels this is still not the root of the AD problem. It is proposed that the stressor initiating the AD pathology does not originate in the complex multi-factors reviewed, or their integration; but that the trigger is psychogenic in origin. (Corsetti, 1988; Vignat, 1987) The psychogenic stressor postulated is not the mental origin of bodily symptoms commonly called psychosomatic; or that of the newer area of psychoneuroimmunological origin, but of radical origin in the Noumenon of Consciousness. (Amoroso, 1992,1995,1997a,b,1999a) The stressor does not originate directly in the psychological process and act macroscopically; but rather is the result of occluded field translations in the submolecular quantum domain. This can lead to a comprehensive explanation of the heterogeneity of the AD condition; and as a field of physical action explain diffuse cerebral lesions. (Amoroso, et al. 1999)

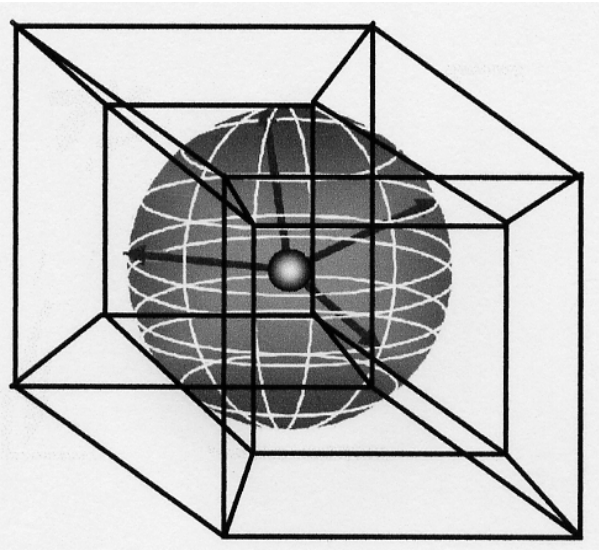

Figure 1. A metaphor representing the soul. Shown conceptually as the 4D hypercube covering the Psychosphere. Adapted from (Wolf, 1999: Tenen, 1999)

The soul-model represented in Figure 1 is meant to be scale invariant from the microscopic to macroscopic. Microscopically each spacetime point, nucleon, atom, and molecule are steeped in the physical energetic flux of the noetic field. This expands to the macroscopic where each biochemical, neurophysiological, and cellular structure likewise contains an inherent flux of the noetic field. So Figure 1 represents both each individual point within us and the global collection of these points that comprise the whole psychosphere of our soul. This recursive self-organized domain is confined within the hyper-dimensional structure of the cosmos.

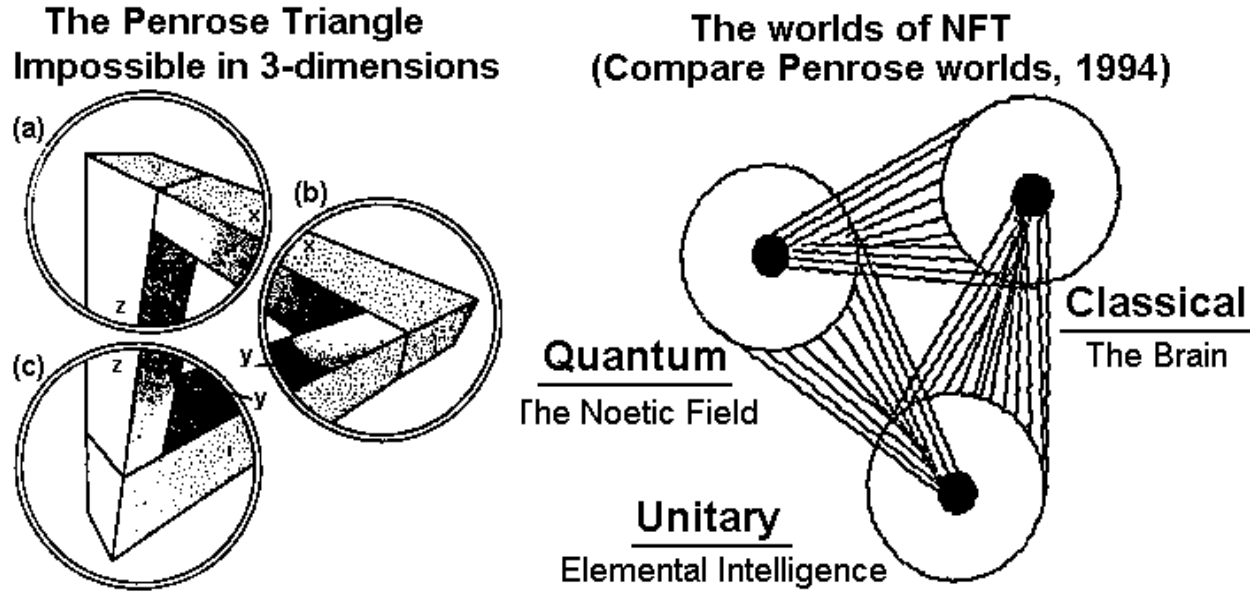

Figure 2. The hyper-dimensional recursive object of Figure 1 is given a dynamical character. The universe does not end in a scalar Planck potential of 10^{-33} cm at the microscopic level or at the Hubble radius of 15 billion light years at the macroscopic level as the current standard big-bang model of cosmology suggests; but is a continuous state scale invariant dynamic hyper-dimensional transform of Classical to Quantum to Unitary. Quantum mechanical uncertainty, which is 'the mystery of God', creates an observational barrier between the 'empyrean realm' and our more limited 'virtual reality' of everyday experience which represents only a temporary barrier established by the methods of current experimental design.

This has been a preliminary work only, introducing the most basic concepts of the noetic etiology of AD. The author realizes the gross inadequacy in the limited presentation of the aspects of noetic cosmology that are crucial to a definitive illustration of the noetic causation of AD. A complete delineation is beyond the scope of this introduction and a more comprehensive paper of much broader scope and detail will be available later this year. (Amoroso, et al, 1999).

Figure 3 illustrates another aspect of the continuous state cosmology (Amoroso, 1999b) that shows hidden variables of the noetic energetics imbedded in the nonlocal and atemporal creation and recreation of the present.

Now that we have this snippet of the noetic cosmology of consciousness we can discuss its relationship to the etiology of AD. The basic point to be illustrated in the prior 3 figures is to illustrate that the essential aspects of mind exist beyond the brain in the hyper-dimensional cosmology of the universe; and within this hyper-structure exist a continuous flux of energy essential to life and consciousness that is outside the current scope of psychology or medicine and is an inkling of the advent of a coming 21st century noetic medicine. A new classification of disease that is not psychological or medical, but diseases of consciousness mediated by the noetic field.

There is a space-time structure for each particular etiology, vaguely reminiscent of the varied electronic structure of the periodic table of the elements. Each created by diversion or stasis in the more normal or healthful flux loci of the noetic field. This action is called the 'noetic effect' and is the result of both intentionality and personality type from both individual and telergic effects.

DEEPER ASPECTS BEYOND THE STANDARD MODEL INHERENT IN THE DUALISM OF QUANTUM THEORY

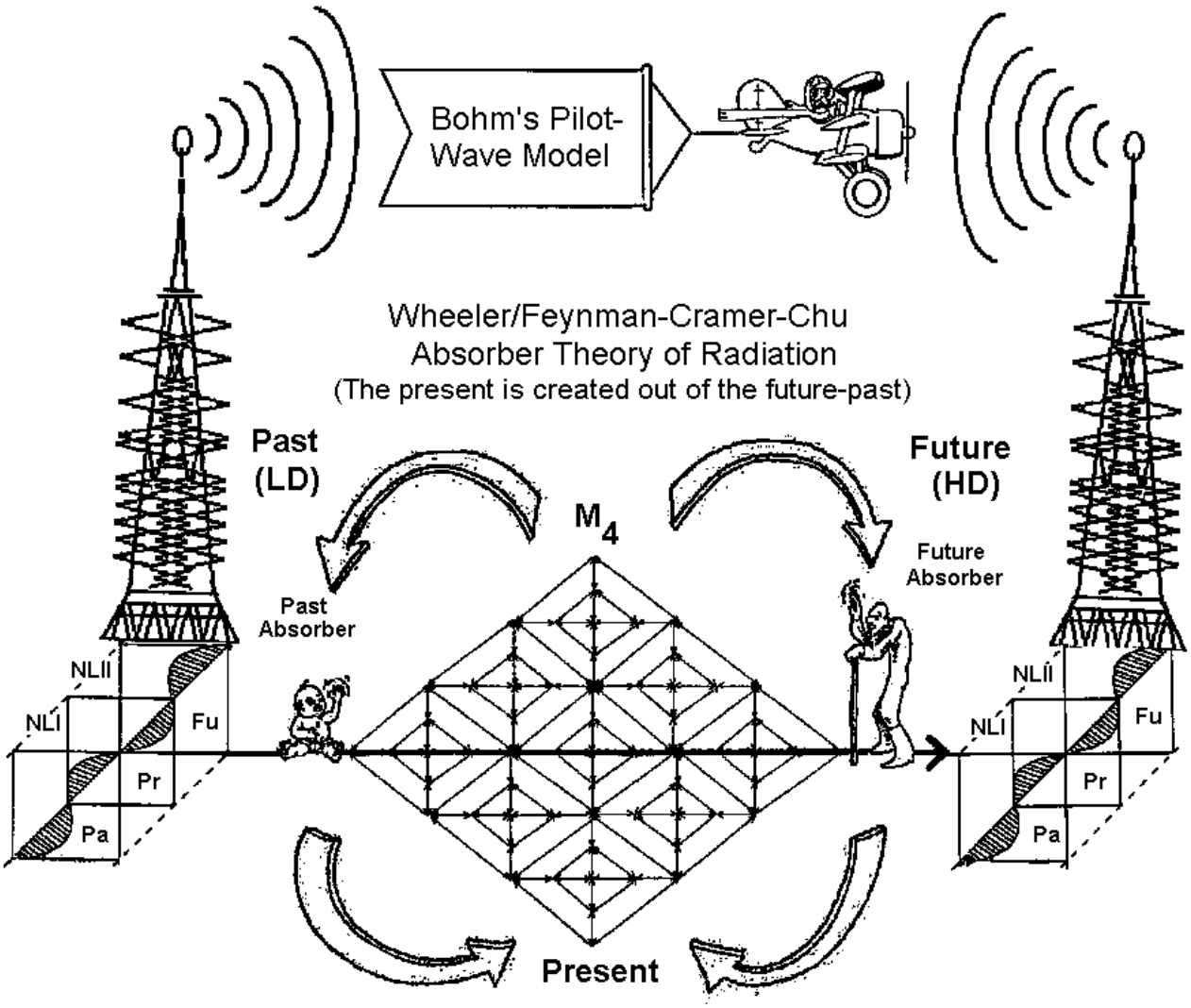

Figure 3. The quantum field of information may be compared to a ship on automatic pilot guided by radio waves from the future and past. In addition to the local/nonlocal standard model of quantum theory; a deeper nonlocal/unitary ontological aspect suggests that a quantum dualism is required for a complete quantum theory. The ontological domain is described by a combination of the Everett/Bohm Pilot Wave non-collapse version and the Wheeler/Feynman Absorber Theory. In this model the present collapses from a higher dimensional hyperstructure continuously recreated out of the energy of the future-past. The pilot-wave quantum potential has a relationship to causality. Gravitational tidal effects regulate aspects of quantum state evolution *U* and state reduction *R* in both collapse and non-collapse domains of the quantum dualism. This cosmological spacetime structure is an important aspect of the continuous state holographic conscious universe; and is required because the standard model of quantum theory is not sufficient to describe the mind comprehensively. It is through this deeper structure that the Noetic Field enters the body with enough degrees of freedom for reason to operate.

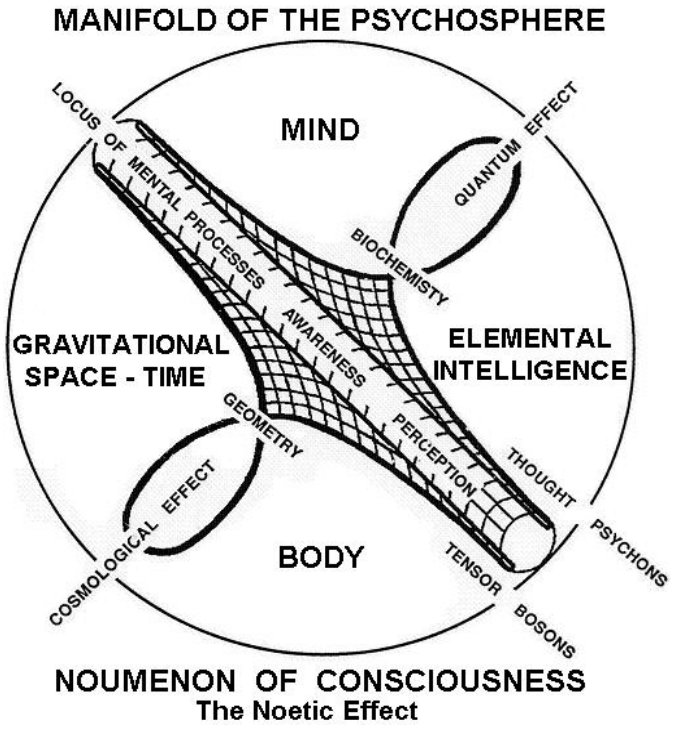

Figure 4. Conceptual representation of the noumenon of consciousness illustrating the domain of the noetic effect - a psychotaxic response of the life force or *elan vital* on quantum biological processes. Scale and dimensionality suppressed.

9.0 Conclusion

The Psychology of the individuals and their families must also be treated in AD cases; since the disease is difficult and tragic for all involved. Indeed it is further suggested that the original causal trigger has an interpersonal aspect. (Amoroso, 1992; Amoroso et al 1999; Vignat, 1987). Since AD affects so many people, and as the population continues to age; AD is becoming an area of major research interest. Continued increases in research funding are being made available. For example, since the Alzheimer's association began its funding program in 1982 about $60 million in research funds have been allocated; with $10 million in 1997 alone. By 2001 it plans to increase its research funding to $30 million. Total funding through other partnerships and advocacy efforts should reach $500 million.

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