The Fundaments of Factor-X

RICHARD L AMOROSO

Noetic Advanced Studies Institute
Escalante Desert, Hogan's Wash
Beryl, UT 84714-5401 USA
amoroso@noeticadvancedstudies.us

Abstract. *Factor-X,* postulated by Nobelist S. Prusiner is the causal agent in prion protein, PrP propagation. The Prusiner Group suggests that Factor-X is a proteinaceous compound triggering normal cellular surface prion protein (PrP^{C}) to transform to the misfolded prion protein (PrP^{Sc}) causing transmissible fatal neurodegenerative diseases by its aggregation. PrP gene ablation in mice, has empirically demonstrated a *protein-only hypothesis*: Misfolded prion protein (PrP^{Sc}) is the only disease-causing agent. Furthermore, the Prusiner view claims prion propagation occurs by a form of *Dominant-Negative Inhibition* where PrP^{Sc} interferes with PrP^{C} function in conjunction with an auxiliary molecule called protein-X, because PrP^{Sc} exhibits more avid binding properties. Our model of Prion propagation alternatively postulates that factor-X is not a proteinaceous molecule, rather a discordant phase alignment in the cyclical propagation of the coherent noetic action of the Unified Field (UF), F_N. The founders of quantum mechanics claimed quantum theory did not explain living systems. In contrast, Einstein believed that *life* would be described by emergent properties of his Unified Field Theory. The three known forces are phenomenological (their fields are mediated by exchange quanta). Ongoing development of Unified Field Theory (UFT), suggests the UF does not entail a 5th force, but an ontological-energyless unifying *force of coherence*, transferring information by topologically switched phase transitions. A primary assumption is that spacetime is comprised of a cellular automata-like tessellation of Least Cosmological Units (LCU), a dichotomy of localized quantum field phenomenology and a nonlocal extension of the *hypertube* model first proposed by Dirac and as embellished by Vigier, where local temporality becomes superluminal at the electron core. We further extend this construct to an M-Theoretic form of Einstein's UFT, where the superluminal concept becomes instantaneous (EPR) within the tenets of an Ontological-Phase Topological Field Theory (OPTFT). In this scenario, a single pixel is not a discrete Planck area quantized qubit; but rather a hyperspherical close-packed 12-sphere LCU – likely to be configured as a dual mirror symmetric Calabi-Yau 3-brane comprising a 6D M-theoretic encoded qubit embedded in a 6D manifold. We assume that spacetime (surface of a Dirac polarized vacuum) is tessellated with cellular automata-like close-packed Least Cosmological Units (LCU), a duality of localized geometric QED cavities and nonlocal topological M-theoretic brane transitions of an Einsteinian Unified Field. We operate under the assumption that this spacetime is programmable. We propose an empirical mediation of the primary mechanism initiating protein conformation in prion propagation utilizing a configuration of two entangled Hadamard gates as a circuit representation for a possible quantum logic gate configuration for PrP^{C} Propagation, H generates a superposed intermediate conformation of PrP^{C} called PrP* in state $|0\rangle \pm |1\rangle / \sqrt{2}$, illustrating the possibility that the Prion's pathological process acts like a quantum Hadamard Controlled-Not Gate; where $|A\rangle$ is the control qubit and $|B\rangle$ is the target qubit in order to mediate topological phase transitions of the UF at an energetic correlation with PrP^{C} - PrP^{Sc} protein conformation misfold. This noetic postulate is compatible with Prusiner's view that prion propagation appears to occur by a form of what Prusiner's group calls *Dominant-Negative Inhibition* where PrP^{Sc} interferes with PrP^{C} function in conjunction with an auxiliary molecule called protein-X because PrP^{Sc} exhibits more avid binding properties. However as stated our interpretation for a protein-X differs; we postulate instead that QED cavity dynamics within the cavity where PrP* binding occurs. This noetic postulate is compatible with Prusiner's view that prion propagation appears to occur by a form of what Prusiner's group calls *Dominant-Negative Inhibition* where PrP^{Sc} interferes with PrP^{C} function in conjunction with an auxiliary molecule called protein-X because PrP^{Sc} exhibits more avid binding properties. However as stated our interpretation for a protein-X differs; we postulate instead that QED cavity dynamics within the cavity where PrP* binding occurs can be described as a form of logic-gate for interactive computing. This is a boundary condition problem; possibly of the Born-von Karman (BvK) type where boundary conditions restrict the wave function to periodicity on a Bravais lattice of hexagonal symmetry. BvK boundary conditions are periodic conditions which impose the restriction that a wave function must be periodic on a particular Bravais lattice.