Letter to the Editor

Achilles' Heels of the "Orch OR" Model

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Abstract

It seems that the "Orch OR" model violates conservation of energy and does not match with experience. In this article we focus on these subjects.

Key Words: consciousness, "Orch OR" model, space-time, parallel universe, false subjective experience, uncertainty principle, gap junction, X-linked form of Charcot-Marie-Tooth disease.

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1. The "Orch OR" Model and Conservation of Energy

Penrose and Hameroff have proposed a model of consciousness (Hameroff and Penrose, 1996; 2003) based on the Penrose "objective reduction" in quantum gravity (Penrose, 1996). In Penrose's view, discrete space-time or foams like bubbles in a bubble bath will eventually reduce to one particular curvature at the OR threshold in a *reasonably short time*. The "Orch OR" model involves quantum computation in microtubules within the brain's neurons. When enough entangled tubulins are superpositioned long enough to reach the OR threshold, an objective reduction (conscious event or "occasion of experience" in Whitehead's language (Hameroff, 2003)) occurs. During the pre-conscious superposition phase, there are quantum superpositions of all possible perceptions or choices which then

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reduce/choose one particular set of qualia at the moment of OR. It seems this idea violates conservation of energy. Look at this (anthropocentric!) problem:

> *Just after childbirth, other mammals can recognize their young. However, a human mother can not. Actually, she accepts any infant as her child! If a mom looks at her "false" infant, then she will feel a "false" subjective experience.*

Note that this situation is more complex than you had earlier thought! "Orch OR" can solve one part of this problem. There are a zillion universes for human and the number of possible space-time configurations is enormous, so the number of combinations of states is quite large. These choices for human can be thought of as consciousness. The conscious human, by making choices, creates his/her universe. Each choice of states creates a universe. Notice, however, there is only one real universe and all other possible universes are *false universes*. The false (virtual) universes allowed by *Heisenberg's uncertainty principle* of quantum mechanics and therefore the similar "virtual particles" (which also follow from the uncertainty principle), exist for only *a short time* and their continued existence would violate the conservation law for energy. But a mom can *create* a virtual universe. This violates the law of conservation of energy. While for other mammals that *consciousness is meaningless* ,there is no conservation of energy problem, since all of the parallel universes are the same (and actual). Therefore, in the language of philosophers of mind such as Thomas Nagel (Nagel, 1993) and John Searle (Searl,1993), "Orch OR" model can explain two of three features of consciousness: its unitary (binding) nature and intentionality, but still can not solve the major difficulty of the three features of consciousness which derives from its subjective qualities.

2. "Orch OR" Model and Experience

The "Orch OR" model faces at least two important obstacles: first, quantum computation requires isolation (decoherence) to prevent thermal interactions which apparently destroy quantum processes (Tegmark, 2000); and, second, it is unclear how quantum states isolated within individuals neurons could extend across membranes and anatomical regions. To overcome the first problem, the model assumes acetylcholine binding to muscarinic receptors acting through secondary messengers to phosphorylate MAP-2, thereby decoupling microtubules from the outside environment, and to overcome the second problem it assumes quantum states or fields could extend across membranes by quantum tunneling across *gap junctions* of neurons, which would allow intracellular quantum states to spread among neurons (Woolf, 2001).

Therefore, if we block muscarinic receptors (with atropine: $C_{17}H_{23}NO_3$), or impair gap junctions, we'll expect abnormality in cognitive behaviors. I have not checked the first proposal, but in 2001, Guldenagel *et al*. (Guldenagel, 2001) produced a mouse with no gap junctions, but apparently normal cognitive behavior. In addition, connexin hemi channels have been proposed as a diffusion pathway for release of extracelluar messengers, based on connexin expression models and inhibition by gap junction blockers. Naturally, in the X-chromosome-linked form of Charcot-Marie-Tooth disease(CMTX), mutations in one of the connexin genes (connexin-32) expressed in Schwann cell prevent this connexin from forming functional gap junction channels (Bruzzone, 1994). However, there is apparently no reported abnormality in cognitive behaviors (Rubinsztein, 1997).

In conclusion, I am persuaded that we should seek some other approaches such as the top-down (from large to small) of Chris Clarke (Clarke, 2004) (which apparently, at least, could solve the decoherence problem) to solve the problem of consciousness.

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Box: Connexin 32 and CMT Disease

Charcot-Marie-Tooth disease comprises a group of genetically heterogenous disorders of the peripheral nervous system. *The X-linked form of Charcot-Marie-Tooth disease (CMTX) is associated with mutations in the gene encoding connexin32 (Cx32), which is expressed in Schwann cells.* Among the 20 proposed members of the connexin family of proteins that form gap junctional intercellular communication channels in mammalian tissues, over half are reported to be expressed in the nervous system. There have been conflicting observations, however, concerning the particular connexins expressed by astrocytes, oligodendrocytes, Schwann cells and neurons.

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The connexins are a family of homologous integral membrane proteins that form channels that provide a low resistance pathway for the transmission of electrical signals and the diffusion of small ions and non-electrolytes between coupled cells. Individuals carrying mutations in the gene encoding Cx32, a gap junction protein expressed in the paranodal loops and Schmidt-Lantermann incisures of myelinating Schwann cells, develop a peripheral neuropathy - the X-linked form of CMTX. Over 160 different mutations in Cx32 associated with CMTX have been identified. Some mutations will lead to complete loss of function with no possibility of expression of functional channels. Some mutations in Cx32 lead to the abnormal accumulation of Cx32 proteins in the cytoplasm, particularly in the Golgi apparatus; CMTX may arise due to incorrect trafficking of Cx32 or to interference with trafficking of other proteins. On the other hand, many mutant forms of Cx32 can form functional channels. Some functional mutants have conductance voltage relationships that are disrupted to a degree which would lead to a substantial reduction in the available gap junction mediated communication pathway. Others have essentially normal steady-state g-V relations. In one of these cases (Ser26Leu), the only change introduced by the mutation is a reduction in the pore diameter from 7 A for the wild-type channel to less than 3 A for Ser26Leu. This reduction in pore diameter may restrict the passage of important signaling molecules. These findings suggest that in some, if not all cases of CMTX, loss of function of normal Cx32 is sufficient to cause CMTX.

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