Quantum Paradigms of Psychopathology

Consciousness and Hallucinations: Molecular Considerations and Theoretical Questions

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Abstract

This paper focuses on aspects of quantitative and quantum approaches to the phenomenon of hallucination. Consideration is given to the molecular hypothesis of consciousness, to consciousness as perception of the self, and to hallucinatory perception as consciousness detached from reality. The possible transitional sequence *viscosity of membrane / GS-alpha protein / tubulin* in altered states of consciousness is explored.

Key Words: consciousness, hallucination, quantitative psychiatry, platelet cel membrane, Penrose-Hameroff model, serotonin, membrane viscosity, interactome.

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Introduction

Psychosis is defined by psychiatrists as a state of being "out of touch" with reality. In this context, hallucinations experienced by patients suffering from schizophrenia and other psychotic disorders are commonly considered to represent a perceptual subset of detachments from reality, with non-perceptual detachments categorized separately as delusions. Psychiatric designations of any and all "unreal" subjective experiences, whether hallucinatory perceptions or delusional cognitions, depend on metaphysical controversies about what counts as "real"; these historically protracted theoretical tensions among philosophers remain unresolved.

One way of bypassing the above metaphysical morass with respect to hallucinations is to regard them as failures of metacognitive discrimination between two sets of equally "real" informational sources: self-generated and external (Kumar, 2009). The perceptual release theory (West, 1975) operationalizes such a notion by postulating the existence of a neural censorship mechanism which actively excludes from consciousness most of the sensory information continuously received by the brain.

The censor can function only when there is a continuous flow of afferent inputs. If by chance there is an interruption or impairment of such input (e.g. in "functional psychosis" associated with prolonged periods of sensory deprivation), perceptional fragments of previous memory traces emerge in consciousness, and the individual then experiences corresponding hallucinations. This model specifically explains the occurrence of those hallucinations resulting from sensory deprivation (Lilly, 1956, 1977; Sireteanu, 2008).

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A possible relationship between aspects of consciousness, including perception, and quantum theory has been considered by several authors (Vannini, 2008; Smith, 2009). From those considerations have emerged possible formal descriptions of the most empirically elementary mental events, i.e., the subjective experience of the perceptual process (Manousakis, 2009). Quantum foundations of conscious processes as theorized in particular by Hameroff and Penrose (1996) fit especially well with the interactome hypothesis of depressed consciousness described by us in the current issue of this journal (Cocchi, 2010c). In that light it seems reasonable to ask whether or not both perceptions detached from external "reality" and commensurately altered states of consciousness are mediated by the same molecular pathway that subserves ordinary states of consciousness. In other words, might such psychopathological manifestations as depression (Cocchi, 2010a; b) and/or schizophrenic psychosis (van Woerkom, 1990; Benitez-King, 2004), implying different kinds of consciousness (Morin, 2006), involve cytoskeletal quantum-nanowire network substrates?

Toward the end of addressing this issue, the present paper has been organized into the following sections:

1. Quantitative and quantum approaches to psychopathology
2. The temporal latency of antipsychotic drug action
3. The platelet membrane: a bridge between enteric and brain serotonin
4. The molecular hypothesis and quantification of hallucinatory consciousness
5. Conclusion.

Quantitative and quantum approaches to psychopathology

Recent articles by Mender (2010), Globus (2010), and Woolf et al., (2010) have compared traditional mainstream neurobiological models of psychopathology to new quantum-oriented theories. All three papers elaborate ways in which orthodox perspectives offer less adequate foundations for explaining mental illness than do quantum neurodynamical approaches. Mender's critique explores many internal inconsistencies of the now dominant biopsychosocial paradigm, which is based on "classical" formalisms antedating quantum physics. Gordon Globus demonstrates the inadequacy of classical nonlinear dynamics, shorn of any quantum elements, as a sole basis for understanding schizophrenia and other psychiatric illnesses. Woolf and her collaborators show that theories based only on disruption of chemical neurotransmission and neuromodulation in non-quantum neural networks contradict at least some empirical evidence, and that quantum wetware components of the brain may provide a more plausible fit with experimental data. Mender, Globus, and Woolf *et al.,* all argue that, since standard frameworks employed by psychiatrists fall short of quantum neurodynamical potentialities, remedies mobilizing a deep, radical paradigm shift incorporating quantum perspectives are needed.

New neurodynamical models incorporating quantum principles may well lead to an improved understanding of mental illness. Nevertheless quantum theories of psychopathology have so far not made substantive progress in moving beyond abstraction into the concrete domain of experimentally validated biological structure and function (Bruza, 2010). Hence, there exists a need to establish links between higher level, qualitative concepts of psychiatric illness and biophysically grounded, quantified instantiations of quantum neurodynamics (Woolf, 2009).

In fact, Woolf and her colleagues (2010) have begun the process of inquiring into ways that malfunctions of cytoskeletal proteins, i. e. of microtubules and their subunits, could underlie some psychiatric diseases. She and her co-authors have drawn upon the work of Penrose and Hameroff (1996; 2010) to argue that aberrations of tubulin superposition might subserve quantum-computational anomalies relevant to psychopathology.

Building upon this proposal by Woolf *et al.,* we have already sketched a hypothetical pathway linking cell membranes and cytoskeletal structures. We have recognized that, while the step leading

from membrane fatty acids to tubulin harbors a merely classical albeit strongly non-linear sensitivity to initial conditions of membrane viscosity (Figure 1), quantum concepts of psychiatric illness may play a crucial role in the transitional sequence *viscosity of membrane / Gsα protein / tubulin* encompassing the interactome. The molecular hypothesis of the interactome proposed by Cocchi et al., (2010c) implies powerful correlations among membrane viscosity, G protein dynamics, and quantum neurophysics vital to states of consciousness which may include pathological experiences such as hallucinations.

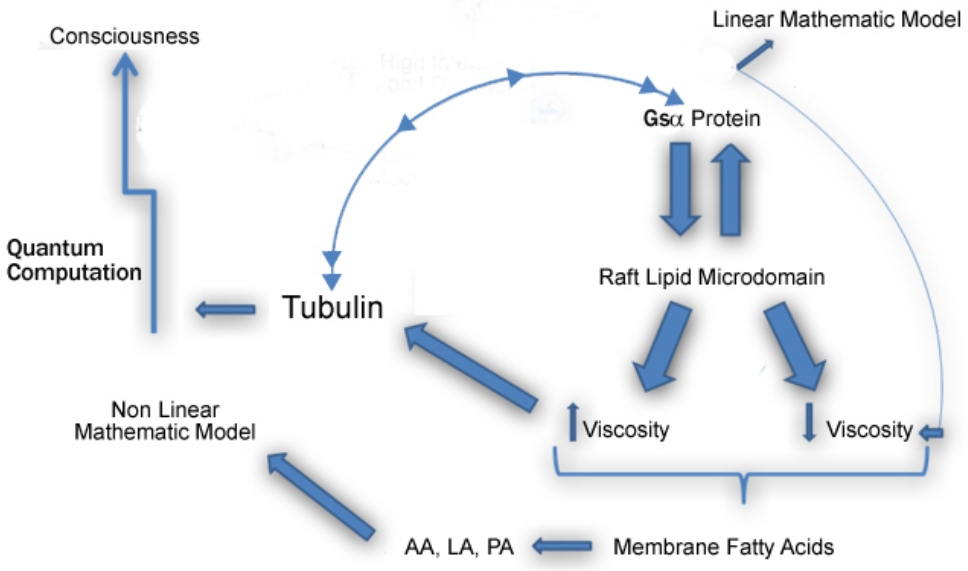

Figure 1. The figure describes the mathematical steps delineating at a cellular level the main hypothetical molecular pathways involved in major depression (MD) and in consciousness (see, text for explanations).

Temporal latency of psychotropic drug action

As Woolf (2010) points out, antidepressants and antipsychotic drugs require a latency of 2-6 weeks to exert their therapeutic actions. The fact that the first part of this period (2 weeks) corresponds to reorganization of the neuronal cytoskeleton suggests that psychopharmacological agents may initiate ameliorating clinical effects through cytoskeletal alterations (Woolf, 2009). Additional actions, including not only dopaminergic and glutaminergic effects but also more pointedly inhibited serotonin re-uptake by many psychotropic drugs, raise the possibility that such phenomena in neurons are heralded by similar events in platelets (Axelson, 2005). Woolf's suggested cytoskeletal substrate for the therapeutic effects of psychotropic drugs, linked to the additional context of serotonin re-uptake, may imply, as outlined by Cocchi *et al.*, (2009a; b), that a multi-staged chain of pharmacologically induced consequences, mediated by the bidirectional transfer of arachidonic acid between platelets and the brain, modifies molecular steps underlying psychopathological processes, i.e., those involving membrane viscosity, Gsα protein, and tubulin, in turn influencing neural correlates of pathologically altered consciousness.

Rasenick (Donati, 2008) has specifically proposed the so-called "*suicide shift*," i.e., rapid Gsα protein changes associated with suicide. Depressed patients with suicidal tendencies demonstrate a high proportion of Gsα protein in their neuronal lipid raft membranes. A similar finding may perhaps occur in platelets. This kind of modification depends on changes in membrane viscosity (Donati, 2008). Under conditions of elevated arachidonic acid concentration in platelets, one would expect the normal exchange of the arachidonic acid between platelets and the brain to be disrupted (Cocchi, 2009a; b); a significant increase of arachidonic acid in the brain with

a decrease in the viscosity of neuronal membranes would reduce serotonin uptake by neurons and platelets (Heron, 1980). A clinical outcome with impact on the mood coloration of consciousness, possibly including suicidal depression, might follow.

The platelet Membrane: a bridge between enteric and brain serotonin

The connections between neuronal and platelet cell membrane viscosity (Tonello, 2010) together with its probable central role in regulating the interactome are often overlooked. Heron (1980) describes the correlation between membrane viscosity, related to fatty acid characteristics, and serotonin receptor binding capacity, the latter influencing the emergence of psychopathological states. It is now possible, due to the creation of a mathematical model for the classification of psychiatric disorders, to demonstrate that platelet membrane viscosity mediates at least some forms of psychopathology with arachidonic acid (AA) as a critical element (Cocchi, 2010c).

However, this research alone does not explain the platelet-brain-serotonin connection. The hypothesis of a platelet-brain-serotonin connection, formulated by Cocchi (2010c), proposes that in major depression a high concentration of platelet membrane AA (Cocchi, 2008) and consequently decreased viscosity generates a defect in platelet receptors binding serotonin issuing from enterochromaffin cells, thus decreasing the 5-HT concentration. The high concentration of AA in platelets will prevent mutual exchange with the brain (Cocchi, 2009a; b), increasing neuronal concentration of AA, which tends toward greater accumulation as the brain receives AA from other sources. A significant reduction in neuronal membrane viscosity should thus lead to a commensurate reduction of serotonin. This would explain similarities between neurons and platelets in major depression (Takahashi, 1976; Edwards, 1978; Marangos, 1979; Kim, 1982; Rotman, 1983; Dreux, 1985; Wirz-Justice, 1988; Camacho, 2000; Plein, 2001; Maurer-Spurej, 2007).

Theoretical developments have elaborated the basic steps of a complex network that might plausibly support consciousness in not only normal but also depressed states (Tonello, 2010) involving the molecular circuitry through which serotonin and AA interact with platelets and neuronal membrane viscosity. Microtubules (MTs) and actin filaments are the primary interactive partners of lipid rafts. Tubulin, the bulding block of MTs, is anchored on the lipid raft of the plasma membrane. It is known that Gsα proteins in concert with tubulin promote GTPase activity and increase the dynamical behavior of MTs (Wang, 1991; Layden, 2008). Tubulin activates signal transduction in 5-HT receptors coupled to protein Gq/11α through a direct transfer of GTP from tubulin to Gq/11α (Popova, 2002). This substrate comprises a series of molecular events leading to the activation of protein kinase C (PKC) through a mechanism involving hydrolysis of phosphatidyl inositol-2 (PIP2) by the membrane-bound enzyme phospholipase C (PLC), which through the same reaction produces inositol trisphosphate (IP3) and diacylglycerol (DAG). IP3 diffuses into the cytosol and stimulates release of calcium ions from the smooth endoplasmic reticulum while DAG remains within the plasma membrane and activates PKC. The activated PKC is involved in receptor desensitization, in modulating membrane structure, in regulating transcription, in mediating immune responses, in controlling cell growth, and in learning and memory. These functions are achieved through PKC-mediated phosphorylation of other proteins.

The above molecular events, which map a route converging on tubulin (Wang, 1991), are closely related to the Penrose-Hameroff model linking consciousness to quantum processes in microtubules (1996). A related construct, invoking gamma synchrony, offers the prospect of quantifying a variety of psychopathological conditions like major depression and hallucinatory psychosis as expressions of different states of consciousness (Hermann, 2005; Flynn, 2008).

This entire framework considered as a whole argues strongly for the workability of a quantitative approach to psychopathology (Cocchi, 2010b). Membrane viscosity, platelet modulation, serotonin re-uptake,

and exchange of AA between platelets and the brain may prove to be some of the basic parameters quantifying major depression. From that perspective it might also be possible to understand whether similar molecular substrates in addition apply to consciousness that is detached from reality, as in hallucination.

The molecular hypothesis and quantification of hallucinatory consciousness

The foregoing considerations along with "many worlds" arguments advanced by Werneke in this issue of NeuroQuantology (Werneke, 2011) suggest that hallucinations may be regarded as expressions of temporarily hidden phenomenological "*reality*" i.e. of a latent, alternate, quantum-superposed life dimension. Structures comprising the cytoskeleton, insofar as they relate to the problem of "normal" consciousness, could also contribute in aberrant form to the emergence of hallucinatory experience. Perceptual detachment from ordinary reality testing during hallucinations specifically might entail dysfunctional modifications of the interactome (Joachim, 2006; Rual, 2005).

The well-established involvement of serotonin among other neurotransmitters in hallucinations (Kumar, 2009) strongly implies a connection between hallucinosis and the molecular bases of other psychopathological conditions. One can reasonably expect to find correlations between hallucinations and not only cellular-molecular interactions but also measurably aberrant gamma synchrony, the latter germane to variations of consciousness framed in terms of meditative states and some psychiatric diagnoses (Flynn, 2008; Hameroff, 2010). Psychopathology notwithstanding, connecting molecular aspects of cell membrane viscosity with hallucinations might facilitate theoretical and experimental investigation of "normal" non-waking phenomena in the form of dreams, which are detached from the ordinary reality of wakefulness (Tonello, 2010). Relevant interpretations in terms of quantum neurophysics must take into account what has already been outlined regarding the movement of serotonin, since as demonstrated 5-HT is inextricably bound to physico-chemical properties of the cell membrane interfacing with the cytoskeletal quantum-nanowire network. Major or minor changes in membrane viscosity may increase or decrease the responsiveness of serotonin receptors (Heron, 1980) and thereby modulate platelet as well as neuronal function, potentially impinging upon psychopathological phenomenology including hallucinatory symptoms. Despite our lack of definitive experimental proof at this juncture, it is not difficult to imagine a map potentially leading us from the relationship between serotonin and membrane viscosity to specific expressions of alternative consciousness, including dreams and hallucinations.

If the foundations of that map prove to be consistent, we might reasonably argue that perception detached from ordinary reality originates in molecular changes at the cellular level and unfolds in an unusual dimension, whose extension depends on a self-governing phenomenological process conditioning the kinds of perceptions that, whether real or unreal in classical terms, may seem real in a superposed sense. Regarding this point one wonders whether hallucinations might reproduce perceptions rooted in experience initially overlapping the normal conscious process of perception, but in such a way that molecular changes within the cell induce an entirely new, divergent level of consciousness. In the course of that divergence, do hallucinogenic molecular alterations, once having determined their own birth, then collapse, thus boot-strapping a new, second, independently liberated level of consciousness, albeit upon the back of the original singular molecular mechanism underlying all conscious perception? We are probably facing here an exotic form of quantum "self-reference," the proximate genesis of which will most likely prove difficult to understand, given that the subject subsequently lives through a whole anomalous experience of him- or herself.

Perhaps identifying critical molecular changes in tubulin function will be the means by which crucial missing knowledge relevant to this issue will be filled in. According to Hameroff and Penrose's Orch OR theory, during natural or induced sleep

(another condition of detachment from reality), conscious processes become quiescent. Might this interlude of quiescent consciousness correspond to a steady, perhaps unitary state of activity within the molecular-cytoskeletal "interactome"? Clearly, the interactome must work even during sleep, but at which level does it operate during dreams and, by extension, during hallucinations? Jonathan Winson's theory concerning the neurobiology of dreaming (Winson, 1986) may pertain to these questions. Winson speculates that off-line information processing within the brain merges new information with old memories to produce strategies for future behavior. He offers empirical support for the idea that dreams are the bridge between "the conscious and the subconscious." Winson's ideas, adapted to the classical and quantum perspectives of the interactome, might be useful in understanding not only dreams but also hallucinations.

Are there grounds for thinking that the mechanisms subserving perception and consciousness escape normal "on-line" molecular pathways when generating the dream-like psychopathology of hallucinations? What are the alternative mechanisms that might be activated at the point of bifurcation? Specifically, given that cytoskeletal proteins implicated in consciousness contribute to the regulation of neuroplasticity (Woolf, 2010), how may they participate in the modulation of hallucinatory perception?

These problems are complex and imply several further salient questions: a) Is it possible or impossible to tease apart complex changes of the interactome detached from reality during hallucinations and dreams in the same way that we might analyze the molecular substrates of other psychopathological conditions? b) Is it possible or impossible to model a divergent molecular pathway for hallucinatory consciousness beyond other manifestations of psychopathology? c) Is any heuristically constructive scientific advantage offered by a consideration of hallucinations and their "unreal" perceptual status as off-line guards against a total blackout of the mind? How might such a function fit with Winson's theories?

There are reasons to think that we can address these questions through the transitional sequence *viscosity of membrane / Gsα protein / tubulin*, which will not only bolster the plausibility of other levels of consciousness but may also lend credence to a quantum-biomolecular basis for perceptual detachment from reality. The phenomenon, notwithstanding its neuroanatomical context, should also demonstrate molecular parameters characteristic of dreaming and hallucinatory experience; debate will then hinge on empirical comparison of those parameters with expected normal waking conditions. If Winson's work can be shown to be fully consistent with ours, then a unified quantum-biomolecular paradigm for hallucinations and dreams supporting a truly scientific approach to phenomenological detachment from reality can move forward in earnest.

There is a third condition of detachment from realty; it is known as anesthesia. Anesthetics are thought to cause unconsciousness by blocking the brain's ability to integrate information (Alkire, 2008). A model enlisting anesthetic drugs that induce complete loss of consciousness might lead to correlations with platelet membrane viscosity and Gsα protein-tubulin interactions. It should be possible in turn to extend that research toward inquiries into changes involving the cytoskeletal network of microtubules. Hence, studies on molecular modifications during anesthesia might become a template against which to compare interactome function and malfunction during dreams, hallucinations, and other psychopathologies (major depression, bipolar disorder, etc.) potentially characterized by different levels of consciousness. It might then be possible to understand in concrete, measurable terms whether divergent conscious dimensions do in fact exist under bifurcating conditions of detachment from realty.

Conclusion

The present paper makes no claim to provide answers but rather only asks whether quantum-biomolecular processing alterations operate in different levels of consciousness. This inquiry is central to the debate on the dreaming and/or hallucinating

body, starting with the following questions: 1) Is consciousness on-line or off-line? 2) If dreams, in Freud's words, provide the royal road from consciousness into the subconscious, then what defines the subconscious in terms of a quantum-biomolecular conception of dreams? 3) Can we map a route by which the functional response arising from afferent "variables," whether attached to or detached from "reality," may rejoin the concrete steps subserving the physics of quantum consciousness, i.e. through the tubulin interface as interpreted by Hameroff and Penrose, or must a "non-quantum consciousness" be assumed?

For purposes of argument let us stipulate, despite many dissenting voices, that there is a consciousness which identifies itself with our personal ego; this assumption is unlike, for instance, Metzinger's Ego Tunnel (Metzinger, 2010) and the related "aporia" (απορια), according to which everything is played out in the ego tunnel – these examples constituting but useful fictions in plumbing our fundamental relationship with reality. Given the identification of consciousness with our personal ego, then all conscious experiences will possess quantum or non-quantum legitimacy. For example, during hallucination there can be no interruption of the self but only a different narrative, operating "off line" at another level. In short, even dreams and hallucinations, though in different ways, shades, and colors, participate in the stream of consciousness and are not necessarily confined to a non-conscious domain.

In the Greek word "syneidesis" and the German word "Bewusstsein," which appear to carry the meaning of consciousness, the particles "*be* and *syn*" indicate an intentional link between consciousness and the world as developed through the phenomenological tradition of Brentano, Husserl, Heidegger, Bergson, Merleau-Ponty, and Binswanger. The relationship between subjectivity and the world belonging to each conscious perception, whether online or offline, suggests that even in hallucination the subject organizes perceptual data according to his or her own forms of lived experience (*Erlebnisse*). Phenomenologically hallucinatory consciousness assumes a bracketed role a la Husserl similar to that played under "normal" conditions. Therefore, it must be subject to the same interpretive keys, i.e. the quantum and/or non-quantum processes of "normal" consciousness.

From these insights follow important questions: 1) what does the hallucinating person see or hear? 2) What does he or she think or believe, and might or must it be delusional? 3) If the content of his or her vision, audition, thinking or belief were confined to a quasi-solipsistic off-line consciousness without any connection to "reality," would that confinement necessarily have to be assigned an a priori non-existent or false status?

In this exploration we have traveled a long distance, whose beginning and end remain blindfolded. We are likely with sufficient effort to learn all the intermediate steps, although we must recognize the difficulty of penetrating those mysteries enshrouding the origin and terminus of anything. In any event, our goal remains access to knowledge inspiring newer, safer, and more effective therapeutic measures that can alleviate the suffering of psychotic patients.

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