Commentaries on Articles

The Cell Membrane: Is It A Bridge From Psychiatry To Quantum Consciousness?

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Abstract

Many current mainstream neuropsychiatric models show inconsistencies, equivocal evidence and failure to explain neuropsychiatric illness. Completely new approaches could help greatly to improve the current situation. A very promising pathway seems to be quantum models of mind, brain, and consciousness; however, an open question is how to link them to the psychiatric world. Cytoskeletal proteins have been credibly proposed as a starting point, but the cell membrane should be taken into account as well. In fact, G-protein dynamics and membrane fatty acid profiles are deeply involved in classical mechanisms of psychiatric illness, and additionally they may play a much different though important role within quantum models of mind, brain, and consciousness. Indeed, G-protein dynamics and membrane fatty acid profiles may represent a substantial bridge between the psychiatric world and quantum theories. Might the nexus of cell membrane investigations therefore lead to a diagnostic tool able to identify psychopathology in a way also comprehensible in terms of a patient's altered conscious state? Toward the end of answering this question, brain cell membranes should be studied, but some experimental clues suggest that platelet membranes may ultimately provide an alternative practical assay with the virtues of low cost and ease of accessibility. In any case, cell membranes (i.e. G-protein dynamics and/or fatty acid profiles) show much promise as a starting point for the linkage of psychiatry to quantum models of mind, brain, and consciousness.

Key Words: cell membrane, G-protein, fatty acids, brain, platelet, quantum brain, quantum mind, quantum consciousness.

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Introduction

The three target articles of the present NeuroQuantology issue (Globus, 2010; Mender, 2010; Woolf, 2010), depict the current mainstream state of art in neuropsychiatry. The status quo does not seem at all adequate. Mender explores many inconsistencies of the biopsychosocial paradigm for psychiatric disease. Globus uses schizophrenia as an example of ways that classical nonlinear dynamical brain modeling fails to explain neuropsychiatric illnesses. Woolf and her colleagues show that there exist both equivocal and unambiguously negative evidence for theories of "chemical imbalances" across networks of neurotransmitters.

Moreover, Globus suggests changing the definition itself of the known psychopathologies in light of open controversies surrounding the *Diagnostic*

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and Statistical Manual of Mental Disorders (DSM). DSM-V remains a work in progress, starting in 1999, still years from finalization, and presently subject to ongoing discussions. The current DSM-IV has been in use since 1994 but its design has been linked to deleterious taxonomic flaws (Bowden, 2001; Ruggero, 2009) raising serious ethical issues (Kocmur, 2009).

There is a need for deep change. Perhaps a completely different approach might prove to be the right solution to the present problematic situation. Quantum theories of mind seem to offer a promising path toward a better understanding of psychopathology. New quantum paradigms of mental illness could be a great help in comprehending the role of mind-brain relations in psychiatric pathogenesis.

Good starting points might be the most notable quantum-neurodynamic models: the thermofield approaches of Umezawa, Jibu, Yasue, Vitiello, and Globus (Globus, 2003; 2009; Jibu and Yasue, 1995; Vitiello, 2001) and the Orch OR hypothesis of Hameroff and Penrose (Hameroff, 1996, 1998; Penrose, 1994). However, other contributions should be taken into account, e. g. the innovative membrane-oriented ideas postulated by Bernroider (Bernroider, 1999; 2003; 2004; 2005; Summhammer, 2007).

At present, it seems that no strong pragmatic contribution to the clinical psychiatric world has yet been made by quantum theories. Moreover, psychiatric applications have not been considered seriously by the quantum community, at least in a theoretically systematic manner or by means of planned experimental design. Hence, one of the first problems to overcome is the link between the domain of psychiatric illness as understood today and the realm of normative quantum mind. We should find common points in order to study them together at the same time. The two different worlds of psychiatry and quantum mind theory at present remain mutually detached, and we have to build a bridge linking them.

In this sense, Woolf and her colleagues (Woolf, 2010) have made an important contribution. They are working on an approach to cytoskeletal proteins operating at two levels. First, from a psychiatric point of view, they adduce evidence that cytoskeletal proteins (microtubules in particular) are dysfunctional in at least some mental illness. Second, they remind us convincingly that quantum theories of mind are closely related to cytoskeletal proteins, particularly those implementing information processing in microtubules.

Are there other possible roads bridging psychiatry and quantum models of consciousness?

The Cell Membrane Has a Key Role in Psychiatric Illness

Woolf and co-authors have successfully identified a relationship between quantum mind/brain models and psychiatry through cytoskeletal proteins. There may be yet another link: the cell membrane.

Brain cell membranes seem to play a key role in psychiatric illness. Diverse approaches have provided evidence with respect to this topic; perhaps one of the most important is a proposal by Rasenick's group (Allen 2007; Donati, 2008). This research focuses on the dynamics of G-protein within its membrane lipid raft microdomain as a basic element of vulnerability in depression and suicide. Different cell membrane states (i.e. different G-protein and lipid microdomain configurations) lead to differences in activation of Gα subunit coupling with adenyl cyclase in the subsequent cAMP signaling cascade. Since the international scientific literature for more than two decades has reported cAMP signaling cascade abnormalities within brains of suicidal and depressed human subjects (Cowburn, 1994; Pacheco, 1996; Dowlatshahi, 1999; Stewart, 2001; Dwivedi, 2002; 2004; Pandey, 2005), this locus of apparent pathology constitutes a promising avenue of further investigation regarding a potential role for the cell membrane in psychiatric illness.

Rasenick's group has studied brain cells but argues as well that Gα raft localization in non-neural peripheral tissue may serve as a biomarker for depression. In particular, a wide scientific literature supports the suggestion of Rasenick's group that human platelets serve as biological

markers for depression (Mooney, 1988; 1998; Garcia-Sevilla, 1990; Pandey, 1990; Menninger and Tabakoff, 1997; Hoffman, 2002; Hines and Tabakoff, 2005).

The authors of the present paper have studied platelet membranes of depressed subjects, enlisting profiles of fatty acids (FAs) as a possible measure of the membrane status (Cocchi and Tonello, 2006; 2007a; 2007b; Cocchi, Tonello *et al.*, 2006; 2008; 2009). In particular, the authors have compared the platelet membrane FA profile of a group of clinically depressed subjects against a group of healthy subjects. The pattern of the two groups has been found to be statistically different.

The FA findings in the authors' experiments appear highly consistent with the G-protein and lipid raft dynamics delineated by Rasenick's group. In fact, this result is in agreement with an extensive body of scientific evidence suggesting that polyunsaturated fatty acids (PUFAs) affect cellular functions by modulating the structure and function of specific lipid domains, such as lipid rafts (Chen, 2007; Sottocornola, 2008; Stulnig, 1998; 2001). It has been suggested that the PUFAs in the cellular bilayer may enhance the transfer of cholesterol, potentially modifying raft composition and thus the local function of a membrane (Kucerka, 2009). In any case, recent growth of clues and evidence points toward a link between FAs and G-protein dynamics (Han, 2009; Bok, 2009) and reveals that lipid rafts from diseased brains exhibit aberrant lipid profiles in comparison to healthy brains (Martín, 2010).

The authors of the present paper have performed further analysis of the previously cited experiments, using several mathematical methods that provide additional results. Data studied by means of a Self Organizing Map (SOM), a kind of artificial neural network - the so called "*Kohonen Network*" - (Kohonen, 2001), suggest three FAs as the main actors in depression (and suicide) and thus as possible unique platelet biomarkers. They are arachidonic, palmitic and linoleic acid. The authors hypothesize that these three FAs might suffice to summarize the whole cell membrane status, i.e. the full FA profile, and hence the G-protein dynamics.

Some clues indeed seem to support the fundamental role of the three FAs. For example, consider that G-proteins could be targeted to raft domains by several mechanisms. The most plausible one is that Gα subunits are subject to palmitoylation (Allen, 2007), i.e. the covalent attachment of FAs, such as palmitic acid, to cysteine residues of membrane proteins. Thus palmitic acid emerges as a leading component in G-protein dynamics as well as one of the biomarkers postulated by the authors. Recently, the role of palmitic acid in relation to G-protein coupled receptors has been demonstrated (Chini, 2009). Matters could thereby turn out to be surprisingly simple: just three FAs could be enough to describe the membrane status and hence the depressive substrate.

Of overarching importance here is that the cell membrane seems to be deeply involved in depression and perhaps also in other neuropsychiatric illness. In particular, G-protein dynamics in brain cells and FA profiles in platelet membranes could be a very promising way to better understand psychopathology. These two molecular entities seem to give different measures of the same thing, the brain cell locus being of central import but the platelet site perhaps easier practical access.

The Cell Membrane: Possible Roads to Quantum Consciousness

Once again, let us look at the cell membrane. There are many clues suggesting multiple strong connections between FA profiles or G-protein dynamics and current quantum brain theories. We shall now try to shed light on these links by honing in on some of the most notable quantum models and theories about brain, mind and consciousness.

The Orch OR model and Hameroff's "Conscious Pilot"

Let us start from the platelet membrane's FA composition, which, as previously described, may be seen as an alternative expression of the brain G-protein dynamics. But consider also that G protein dynamics (and thus lipid raft) are strongly related to microtubule and cytoskeleton dynamics; there is a wide literature demonstrating the existence of such a relation (Layden, 2008;

Roychowdhury, 1999; Chichili, 2009). FAs (including platelet FAs) may be linked to cytoskeletal protein in this way. Indeed, according to some recent works, FAs may be understood to possess an explicit and direct connection to microtubule and cytoskeleton dynamics (Han, 2009; Bok, 2009).

Now, as clearly shown by Woolf's group (Woolf, 2010), microtubules are the main site of the most notable quantum mind models. In fact, tubulin is the fundamental building block of the Orch OR model introduced by Stuart Hameroff and Sir Roger Penrose.

Let us now consider another of Hameroff's ideas. This recent so called "conscious pilot" model (Hameroff, 2010) deals with brain gamma-synchrony and focuses particularly on the neuronal GAP junction as its fundamental engine. In Hameroff's words: "*placement, openings, and closings of gap junctions are regulated by intraneuronal calcium ions, cytoskeletal microtubules, and/or phosphorylation via G-protein metabotropic receptor activity*."

Therefore by inference it seems clear that G-protein and membrane FAs are linked with Orch OR and the new "conscious pilot" model.

Thermofield Brain Dynamics

Thermofield Brain Dynamics (TBD) is another very well known theory of mind on which many important authors are working [for a detailed list see Globus (Globus, 2010)]. It happens that membrane FAs harbor multiple links to TBD.

The first link is quite clear. In the words of Globus, TBD has its roots in the "*Quantum Electrodynamics of the brain's water dipole field inside the microtubules*". According to the TBD model, the neuronal cytoskeleton interacts with a coherent water dipole field, once again endowing the microtubule with a leading role. The same considerations discussed above, linking microtubules within the Orch OR model and membrane FAs, can easily be extended to TBD. This is the first strong connection.

But there is a second point of contact. According to Vitiello (Vitiello, 2003), the brain can be modelled as a mixed system involving two separate but interacting levels. The memory level is quantum dynamical while electrochemical activity is at a classical level. The long-range quantum dipolar correlation in the water matrix plays a crucial role in the electrochemical activity. As Vitiello notes, "this points to the role of the glial cells in…biochemical brain activity and may constitute a link between the two levels" (Vitiello, 2001; see also Jibu, 1994; 1996). It is well known that glial cells are closely related to FAs. For example astrocytes, a kind of glial cell, are deeply involved in arachidonic acid production.

Hence, multiple potential links exist between TBD and cellular FAs or G-protein.

Bernroider ion channels

Bernroider's conception of quantum brain function is built upon voltage controlled ion channels (Bernroider, 2005). These may be involved in mental phenomenology such as the well-known "*binding problem*."

In Bernroider's model, the classical view on gating has to be changed with respect to the high-resolution structure elucidated by MacKinnon's group (Zhou, 2001; Morais-Cabral, 2001; Jiang, 2003). In voltage controlled channels, changes in the imposed membrane field induce a charge transfer transition (12-14 electron charges in K channels) carried by amino acids called voltage sensors (Bezanilla, 2000). The transition moment in gating correlates with the open probability of the channel. However, in the "paddle-model," the transition moment couples with the neighbouring lipids of the channel protein, and this latter view gains strong support from an increasing number of studies demonstrating that protein surrounding lipids engage in the control of voltage gated Kv channels (Oliver, 2004).

In this latter way, membrane FAs would be linked to the quantum dynamics of the ion channels and thus, according to Bernroider, to mind processes.

Other clues: Stapp's idea

Many other mind-brain quantum theories show connections, coupling, and direct or indirect links to FAs. As an example, consider Stapp's model of consciousness as a quantum Zeno effect locating quantum

dynamics within the Ca^{2+} ion channel (Stapp, 2005). This model, though controversial, clearly relates to membrane FAs, in particular arachidonic acid (Roberts-Crowley, 2009).

The foregoing survey demonstrates that cell membrane status (i.e. G-protein and/or FA composition) can be linked with a variety of quite different quantum brain/mind models. Actually, membrane status of this kind seems to be a sort of common, mutual element, shared by all of the considered brain, mind and consciousness models. This could be seen as a very important foundational feature, a robust starting point for whatever quantum approach one might decide to explore. If we were to envision membrane status as the principle bridge connecting psychiatry to quantum models, it would be a road with many lanes!

A Short Discussion

G-protein dynamics and membrane FA profiles seem to be strongly related to the concerns of psychiatry. There is evidence suggesting their wide involvement in psychopathology. From another viewpoint, they can be seen as important building blocks for the most notable quantum models of brain, mind and consciousness. Indeed, they seem to be a common foundational element shared by all of these quantum models and theories. Therefore, they might well constitute an ideal starting point to link the world of psychiatry and the quantum universe.

The proposals of Woolf and her colleagues in the present issue of NeuroQuantology hold out the promise of direct and vital applications to basic psychiatric research. This promise is also highly compatible with cheap and easy FA profiles in platelets, which may afford practical ease for eventual routine clinical use.

Conclusion

Many mind/brain models such as Orch OR are best characterized as theories of consciousness. It is therefore potentially fruitful in neuropsychiatric terms to consider the various forms of psychopathology, such as depression and predisposition to suicide, as altered states of consciousness.

Comparison between healthy and psychiatric subjects yields evidence of differences in cytoskeletal proteins, G-protein dynamics, and FA profiles. At the same time, these three biomolecular entities are fundamental elements of quantum consciousness models.

If such correlations eventually lead us to a tool providing a measure of the depressed state, not in a dichotomous manner (simply present or not present) but in a continuous fashion (e.g. as proposed by the authors), then will we thereby be able to "measure consciousness"? The authors' current work on platelets as the place at which to find biomarkers of depression may contribute to such an outcome.

The quantum world may be useful in better understanding psychiatric illness, but psychiatry as well may contribute to improving the quantum research applicable to consciousness. In both cases, the cell membrane might be a good starting point, linking the two worlds in a powerful way.

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References

Allen JA, Halverson-Tamboli RA, Rasenick MM. Lipid raft microdomains and neurotransmitter signalling. Nature 2007; 8: 128-40.

Bernroider G. On Form, Mind and Matter (with special reference to 'Crystal Souls' by Ernst Haeckel, translated by A. L. Mackay). Forma 1999; 14:185-198.

Bernroider G. Quantum neurodynamics and the relation to conscious experience. NeuroQuantology 2003; 2:163-8.
Bernroider G and Roy S. Quantum-Classical Correspondence in the Brain: Scaling, Action Distance and Predictability behind Neural Signals Forma 2004; 19:55-68.
Bernroider G. and Roy S. Quantum entanglement of K ions, multiple channel states and the role of noise in the brain. SPIE 2005; 5841 (29):205-14.
Bezanilla F. The voltage sensor in voltage-dependent ion channels. Physiol Rev 2000; 80: 555-592.
Bowden CL. Strategies to reduce misdiagnosis of bipolar depression. Psychiatric Services 2001; 52: 51-55.
Bok E, Hryniewicz-Jankowska A, Sikorski AF. The interactions of actin cell and membrane skeleton proteins with lipids. Postepy Biochem 2009; 55(2):207-22.
Chen W, Jump DB, Essekman WJ, Busik JV. Inibition of cytokine signalling in human retinal endothelial cells through modification of caveolae/lipid rafts by decosaexaenoic acid. Invest. Ophthalmol. Vis Sci 2007; 48: 18-26.
Chichili GR, Rodgers W. Cytoskeleton-membrane interactions in membrane raft structure. Cell Mol Life Sci 2009; 66(14):2319-28.
Chini B, Parenti M. G-protein-coupled receptors, cholesterol and palmitoylation: facts about fats. J Mol Endocrinol 2009; 42(5):371-9.
Cocchi M and Tonello L. Biological, Biochemical and Mathematical considerations about the use of an Artificial Neural Network (ANN) for the study of the connection between Platelet Fatty Acids and Major Depression. J Biol Res 2006; LXXXI, 82-87.
Cocchi M and Tonello L. Platelets, Fatty Acids, Depression and Cardiovascular Ischemic Pathology. Progress in Nutrition 2007a; 9 (2): 94-104.
Cocchi M and Tonello L. Depressione Maggiore e Patologia Cardiovascolare Ischemica. Un Network fra piastrine, cuore e cervello. Reti Neurali Artificiali, potenzialità predittiva di alcuni acidi grassi della membrana piastrinica. Bologna; CLUEB. 2007b.
Cocchi M, Tonello L, Tsaluchidu S, Puri BK. The use of artificial neural networks to study fatty acids in neuropsychiatric disorders. BMC Psychiatry 2008; 8 (Suppl 1):S3. doi: 10.1186/1471-244X-8-S1-S3.
Cocchi M, Tonello L, De Lucia A, Amato P. Platelet and Brain Fatty Acids: a model for the classifcation of the animals? Part 1. International Journal of Anthropology 2009; 24(1) :69-76.
Cocchi M, Tonello L, Cappello G *et al.,* Biochemical Markers in Major Depression as interface between Neuronal Network and Artificial Neural Network (ANN). J Biol Res 2006; 81: 77-81.
Cowburn RF, Marcusson JO, Eriksson A, Wiehager B,O'Neill C. Adenylyl cyclase activity and G-protein subunit levels in postmortemfrontal cortex of suicide victims. Brain Res 1994; 633:297–304.
Roberts-Crowley ML, Mitra-Ganguli T, Liu L, Rittenhouse AR. Regulation of voltage-gated Ca2+ channels by lipids. Cell Calcium 2009; 45(6):589-60.
Donati RJ, Dwivedi Y, Roberts RC, Conley RR, Pandey GN, Rasenick MM. Postmortem brain tissue of depressed suicides reveals increased Gs alpha localization in lipid raft domains where it is less likely to activate adenylyl cyclase. J Neurosci 2008; 28(12):3042-50.
Dowlatshahi D, MacQueen GM, Wang JF, Reiach JS, Young LT. G protein-coupled cyclic AMP signaling in postmortem brain of subjects with mood disorders: effects of diagnosis, suicide, and treatment at the time of death. J Neurochem 1999; 73:1121–1126.
Dwivedi Y, Rizavi HS, Conley RR, Roberts RC, Tamminga CA, Pandey GN. mRNA and protein expression of selective alpha subunits of G proteins are abnormal in prefrontal cortex of suicide victims. Neuropsychopharmacology 2002; 27:499–517.
Dwivedi Y, Rizavi HS, Shukla PK, Lyons J, Faludi G, Palkovits M, Sarosi A, Conley RR, Roberts RC, Tamminga CA, Pandey GN. Protein kinase A in postmortem brain of depressed suicide victims: altered expression of specific regulatory and catalytic subunits. Biol Psychiatry 2004; 55:234–243.
Garcia-Sevilla JA, Padro D, Giralt MT, Guimon J, Areso P. Alpha2-adrenoceptor-mediated inhibition of platelet adenylate cyclase and induction of aggregation in major depression. Effect of long-term cyclic antidepressant drug treatment. Arch Gen Psychiatry 1990; 47:125–132.
Globus G. Quantum closures and disclosures. Amsterdam: John Benjamins; 2003.
Globus G. The transparent becoming of world: a crossing between process philosophy and quantum neurophilosophy. Amsterdam: John Benjamins; 2009.
Globus G. Toward a quantum psychiatry: hallucination, thought insertion and DSM. NeuroQuantology 2010; 8(1): 1-12.
Hameroff S. Quantum computation in brain microtubules? The Penrose–Hameroff 'Orch OR' model of consciousness. Phil Trans R Soc Lond A 1998; 356: 1869–1896.
Hameroff S. The "conscious pilot"—dendritic synchrony moves through the brain to mediate consciousness. J Biol Phys 2010; 36:71–93.
Hameroff S and Penrose R. Orchestrated reduction of quantum coherence in brain microtubules: a model for consciousness. In: Hameroff S, Kaszniak A, Scott A, editors. Toward a science of consciousness: the first disucssions and debates. Cambridge (MA): MIT Press; 1996. p. 507-40.
Han X, Smith NL, Sil D, Holowka DA, McLafferty FW, Baird BA. IgE receptor-mediated alteration of membrane-cytoskeleton interactions revealed by mass spectrometric analysis of detergent-resistant membranes. Biochemistry 2009; 48(27):6540-50.
Hines LM and Tabakoff B. Platelet adenylyl cyclase activity: a biological marker for major depression and recent drug use. Biol Psychiatry 2005; 58:955–962.
Hoffman PL, Glanz J, Tabakoff B. Platelet adenylyl cyclase activity as a state or trait marker in alcohol dependence: results of theWHO/ISBRA Study on State and TraitMarkers of Alcohol Use and

Dependence. Alcohol Clin Exp Res 2002; 26:1078–1087.
Jiang Y, Lee A, Chen J, Ruta V, Cadene M, Chait BT, MacKinnon R. X-ray structure of a voltage-dependent K+ channel. Nature 2003; 423, 33-41.
Jibu M and Yasue K.Quantum brain dynamics and consciousness. Amsterdam: J. Benjamin, 1995.
Jibu M, Pribram KH, Yasue K. From conscious experience to memory storage and retrivial: The role of quantum brain dynamics and boson condensation of evanescent photons. Int J Mod Phys 1996; 10:1735-1754.
Jibu M, Hagan S, Hameroff SR, Pribram KH, Yasue K. Quantum optical coherence in cytoskeletal microtubules: implications for brain functions. BioSystems 1994; 32:195-209.
Kocmur M. Psychosis: what it is and what it is not. Psychiatric diagnosis an an ethical problem. Psychiatr Danub 2009; 21 (Suppl 1) :98-101.
Kohonen T. Self-Organizing Maps. 3. Berlin: Springer, 2001.
Kucerka N, Marquardt D, Harroun TA, Nieh MP, Wassall SR, Katsaras J. The functional significance of lipid diversity: orientation of cholesterol in bilayers is determined by lipid species. J Am Chem Soc 2009; 18; 131(45):16358-9.
Layden BT, Saengsawang W, Donati RJ, Yang S, Mulhearn DC, Johnson ME, Rasenick MM. Structural model of a complex between the heterotrimeric G protein, Gsalpha, and tubulin. Biochim Biophys Acta 2008; 1783(6):964-73.
Martín V, Fabelo N, Santpere G, Puig B, Marín R, Ferrer I, Díaz M. Lipid Alterations in Lipid Rafts from Alzheimer's Disease Human Brain Cortex. Journal of Alzheimer's Disease 2010; 19(2): 489-502.
Mender D. Post-classical phase transitions and emergence in psychiatry: beyond George Engel's model of psychopathology. NeuroQuantology 2010; 8(1): 29-36.
Menninger JA and Tabakoff B. Forskolin-stimulated platelet adenylyl cyclase activity is lower in persons with major depression. Biol Psychiatry 1997; 42:30–38.
Mooney JJ, Schatzberg AF, Cole JO, Kizuka PP, Salomon M, Lerbinger J, Pappalardo KM, Gerson B, Schildkraut JJ. Rapid antidepressant response to alprazolam in depressed patients with high catecholamine output and heterologous desensitization of platelet adenylate cyclase. Biol Psychiatry 1988; 23:543–559.
Mooney JJ, Samson JA, McHale NL, Colodzin R, Alpert J, KoutsosM, Schildkraut JJ. Signal transduction by platelet adenylate cyclase: alterations in depressed patients may reflect impairment in the coordinated integration of cellular signals (coincidence detection). Biol Psychiatry 1998; 43:574–583.
Morais-Cabral HJ, Zhou Y, MacKinnon R. Energetic optimization of ion conduction rate by the K+ selectivity filter. Nature 2001; 414, 37–42.
Oliver D, Lien C-C, Soom M, Baukrowitz T, Jonas P, Fakler B. Functional conversion between A-type and delayed rectifier K+ channels by membrane lipids. Science 2004; 304, 265-270.
Pandey GN, Pandey SC, Janicak PG, Marks RC, Davis JM. Platelet serotonin-2 receptor binding sites in depression and suicide. Biol Psychiatry 1990; 28:215–222.
Pandey GN, Dwivedi Y, Ren X, Rizavi HS, Mondal AC, Shukla PK, Conley RR. Brain region specific alterations in the protein and mRNA levels of protein kinase A subunits in the post-mortem brain of teenage suicide victims. Neuropsychopharmacology 2005; 30:1548–1556.
Pacheco MA, Stockmeier C, Meltzer HY, Overholser JC, Dilley GE, Jope RS. Alterations in phosphoinositide signaling and G-protein levels in depressed suicide brain. Brain Res 1996; 723:37–45.
Penrose R. Shadows of the mind. Oxford: Oxford University Press, 1994.
Roychowdhury S, Panda D, Wilson L, Rasenick MM. G Protein Subunits Activate Tubulin GTPase and Modulate Microtubule Polymerization Dynamics. J Biol Chem 1999; 274 (19): 13485-13490.
Ruggero CJ, Zimmerman M, Chelminski I, Young D. Borderline personality disorder and the misdiagnosis of bipolar disorder. J Psychiatr Res doi:10.1016/j.jpsychires.2009.09.011
Sottocornola E, Berra B. Modulation of membrane lipid raft by omega-3 fatty acids and possible functional implication in receptor tyrosinekinase activation. Progress in Nutrition 2008; 10 (4): 210-213.
Stewart RJ, Chen B, Dowlatshahi D,MacQueen GM, Young LT. Abnormalities in the cAMP signaling pathway in post-mortem brain tissue from the Stanley Neuropathology Consortium. Brain Res Bull 2001; 55:625–629.
Stulnig TM, Huber J, Leitinger N. Polyunsaturated eicosapentaenoic acid displaces proteins from membrane rafts by altering raft lipid composition. J Biol Chem 2001; 276: 37335-40.
Stulnig TM, Berger M, Sigmund T, Raederstorff D, Stockinger H, Walhausl W. Polyunseturated Fatty Acids inhibit T cell signal transduction by modification of detergent-insoluble membrane domains. J Cell Biol 1998; 143: 637-44.
Stapp HP. Quantum interactive dualism. Journal of Consciousness Studies 2005; 12(11):43–58.
Summhammer J, Bernroider G. Quantum entanglement in the voltage dependent sodium channel can reproduce the salient features of neuronal action potential initiation. arXiv:0712.1474v1[physics.bio-ph] (2007)
Vitiello G. My double unveiled: the dissipative quantum model of the brain. Amsterdam: John Benjamins; 2001.
Vitiello G. Quantum dissipation and information: a route to consciousness modeling. NeuroQuantology 2003; 2: 266-279.
Woolf NJ, Craddock JA, Friesen DE, Tuszynski JA. Neuropsychiatric Illness: A Case for Impaired Neuroplasticity and Possible Quantum Processing Derailment in Microtubules. NeuroQuantology 2010; 8(1): 13-28.
Zhou Y, Morais-Cabral JH, Kaufman A, MacKinnon R. Chemistry of ion coordination and hydration revealed by a K+ channel-fab complex at 2.0 A resolution. Nature 2001; 414: 43–48.