The Quantum Account of Anorexia Nervosa Continued: Probability Amplitudes, Microtubules and the Amygdala

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ABSTRACT

In this paper, the author builds on ideas about a quantum approach to anorexia nervosa developed in a previous paper. A basic equation demonstrating the role of probability amplitudes relating to the answers to the question 'am I fat?' is proposed, and mechanisms by which these abstract amplitudes might relate to the physiology and biology of the human brain are discussed. In particular, it is suggested that the probability amplitudes for the abstract states 'fat' and 'not fat' might relate physically to the superposition of tubulin orientations in cytoskeletal microtubules (as suggested by the Penrose-Hameroff Orchestrated Objective Reduction), and that the question 'am I fat?' might arise in the threat-detection mechanisms of the amygdala.

Key Words: orchestrated objective reduction, anorexia nervosa

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Introduction

Why Develop a Quantum account of Anorexia Nervosa at all?

Anorexia Nervosa is a mental disorder characterised by a refusal to maintain body weight at or above 85% of the minimally normal weight for age and height, an intense fear of gaining weight and a disturbance in the way in which one's body weight or shape is experienced such that the sufferer is overly influenced by body weight or shape on self-evaluation and/or denies the seriousness of the current low body weight. It can be characterised as either restricting type or binge-eating/purging type, the difference being the former does not engage in bingeing and purging behaviour, while the latter does (APA, 1994). While it typically only affects between 1% and 10% within a population (Levine & Smolak, 2002), it has one of the highest mortality rates of all psychological disorders, leading to death in 6-15% of cases (Lock, le Grange, Agras & Dare, 2001). Despite its morbidity, it remains a poorly understood illness, with no unified approach to its etiology and with even the most widely lauded methods of treatment, Cognitive Behavioural Therapy (CBT) and Family Therapy, still showing a worrying lack of demonstrable efficacy (Wilson, Grilo & Vitousek, 2007). With this in mind, it is necessary to pursue every possible avenue that might improve our understanding of this disorder.

The reason for suggesting that *quantum physics* might be such a relevant avenue for understanding AN specifically or psychopathology in general flows from the fact that certain authors have suggested that quantum physics might be relevant for

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understanding ordinary brain function. The theories suggested by these authors are known collectively as Quantum Theories of Mind, and include such ideas as the Penrose-Hameroff Orchestrated Objective Reduction hypothesis (Hameroff, 2007), Bohm's quantum ontology (Bohm, 2002), Stapp's 'interactive dualism' (Stapp, 2011; 2012) and Quantum Brain Dynamics (Jibu & Yasue, 1995) or Thermofield Brain Dynamics (Vitiello, 2001; 2003), both based on Quantum Field Theory. While each of these theories varies in their details, they each suggest that quantum mechanics is in some way relevant to understanding human consciousness and brain function. If these theories represent valid approaches for understanding ordinary mental functioning, then they should also represent valid approaches for understanding abnormal mental functioning such as that found in mental disorders such as AN; and, while any hypotheses about Quantum Paradigms of Psychopathology (QPP) are necessarily speculative, there are reasons to be optimistic about their validity.

The first reason is the finding that quantum probability theory represents a better model of human cognitive functioning than does ordinary classical Bayesean probability theory. In a recent paper, Pothos & Busemeyer (2013) noted the failure of classical probability theory to account for many empirical findings in cognitive science, and suggested that quantum probability theory might fit these results better, in particular noting the potential of quantum concepts such as superposition, entanglement and context- and order-dependency for modelling, inter alia, the fuzziness and conflict in everyday thought, the way in which certain concepts are irredeemably linked in the mind, and the way in which the answer to a question depends on the context in which it is asked. While their thesis has not been universally accepted, it does leave open the question: if quantum probability theory *does* model human cognition better than classical probability theory, does this imply that it is quantum processes, and not classical processes, that underlie human cognition on the physical level? Authors such as Hameroff (Pothos & Busemeyer, 2013) suggest that it does.

The second reason, particularly relevant for this paper, is the suggestion that quantum formalism describes human binocular perception. Manousakis (2007; 2009) has suggested that we may distinguish between *potential* and *actual* states of consciousness, wherein the *potential* state of consciousness evolves in time as a quantum system and the *actual* state of consciousness occurs when the wavefunction of that potential state collapses into a conscious event, observable as a particular 'neural correlate of consciousness' (NCC). Thus, when a perceptual event is observed, we may consider that multiple potential NCCs existing in superposition in a *potential* state of consciousness have collapsed into one *actual* NCC associated with the observation of that particular perceptual event. Manousakis finds that this approach accurately describes the observed probability distribution of dominance duration (PDDD) in binocular rivalry (2007) and that his distinction between potential and actual states of consciousness could account for certain optical illusions (2009).

The third reason is the recent observation by Loewenstein (2013) that the computations of the human brain take place in parallel, not in series, and that "*quantum computing fits that to a T*" (Loewenstein, 2013, p.273). This is because it allows the simultaneous computation of superposed quantum states in such a way that the state representing the most efficient transmission of information may be chosen (Loewenstein, 2013, p. 268). Thus, based on the observation that evolution often takes the most information-efficient route (which is invariably that of quantum computing), and that quantum computing has already been found empirically to take place in photosynthesis and in visual perception (via rhodopsin carbon chains found in the retina), he proposes that the neuronal web of the human brain can operate in two computational modes, the first being a quantum mode that serves the function of parallel computation, the second being a macroscopic (ion-based) mode that serves the function of integration and low-order polynomial computation (Loewenstein, 2013, p.270). The advantage of his approach is that it is not a hypothesis of consciousness, nor a hypothesis related to quantum gravity, but simply the claim that quantum computing is "biologically important in its own right" (Loewenstein, 2013, p.273). In this way he avoids two areas of potentially great controversy to put forward a

simpler idea that may help us to understand normal and abnormal mental function.

Thus, as quantum probability theory and quantum formalism have been found to accurately model human cognition in general and binocular perception in particular, and as quantum computing could contribute greatly to our understanding of parallel computation in the brain, it is appropriate to consider further what a quantum approach might add to our understanding of the perceptual deficit found in Anorexia Nervosa.

I. Developing the theory: the perceptual deficit in Anorexia Nervosa and Quantum Probability Theory

In a previous paper (Marlow, 2013), it was suggested that Anorexia Nervosa may be characterised by the question "am I fat?" and the two prospective answers "yes" and "no". In classical probability theory, these are seen as mutually exclusive. In quantum probability theory, they are seen as existing in a state of superposition until the wavefunction collapses, whereupon one answer ("yes" or "no") is measured classically. In the quantum state of superposition, each answer has associated probability amplitude (a_n), and the probability of this answer occurring is related to the modulus squared of this amplitude ($|a_n|^2$).

It was suggested (Marlow, 2013) that subjective experiences stored as subconscious memories in the form of bosonic condensates (Jibu & Yasue, 1995; Vitiello, 2001) might influence the probability amplitudes of these two answers, and that in anorexic patients, this might result in a dangerously underweight person becoming convinced that s/he is fat and embarking upon the path of self-starvation. My previous paper simply made that suggestion. This paper attempts to demonstrate the mechanism by which it might occur.

In Quantum Mechanics, a quantum state may represent a particular observable for a particular quantum system, whether that observable is energy, position, momentum, or something else. This quantum state is represented by the symbol $|\Psi\rangle$ and denotes a complete set of amplitudes for the quantum system. By this, we mean that it represents a set of probability amplitudes such that, if we know these amplitudes, we can calculate the outcome of any measurement of the system. While $|\Psi\rangle$ denotes a complete set of amplitudes, the quantum state it encapsulates is independent of the particular set of amplitudes with which we choose to quantify it.

In the present case, we may initially take $|\Psi\rangle$ to represent a human mind and we may consider that this human mind is asking the question 'am I fat?' A typical example where this may occur is when an anorexic is looking in the mirror. In this instance, we may write in Dirac notation:

$$|\Psi\rangle = a_Y|Y\rangle + a_N|N\rangle \quad (1)$$

where $|Y\rangle$ represents the state of 'yes' or 'fat', $|N\rangle$ represents the state of 'no' or 'not fat', and a_Y and a_N represent the probability amplitudes associated with both of these states. If this representation of the anorexic state of mind is accurate, a_Y will be much larger than a_N, such that somebody suffering from anorexia will perceive himself/herself to be fat most of the time when s/he looks in a mirror or turns his/her mind to the subject.

Precisely why this amplitude should be so high has not yet been explained. It is suggested that it is the strength of past experiences, both those considered important by psychoanalysis and other experiences that may be 'triggering', that render the amplitude a_Y so much larger than that of a_N. These experiences may be stored as bosonic condensates in the brain, as suggested by Jibu & Yasue (1995) and by Vitiello (2001). In each of these bosonic condensates, each individual boson in the quantum state is coherently linked to each other individual boson, such that we may consider the bosonic condensate as a whole to be one quantum state of the form $|\Psi_n\rangle$. This is appropriate as, if this condensate is measured, all the bosons within it will be found to have the same value as every other boson of any relevant observable.

We may consider that each individual bosonic condensate encodes an individual memory represented by its own ket $|\Psi_n\rangle$, and that it is the strength of many or multiple memories that gives our amplitude a_Y such a high value compared with a_N. Thus, we may expand the above equation to state that:

$$|\varphi\rangle = |\Psi_1\rangle + |\Psi_2\rangle + |\Psi_3\rangle + \ldots + |\Psi_n\rangle \quad (2)$$

Where $|\varphi\rangle$ represents the quantum state of the mind as a whole, while each $|\Psi_n\rangle$ represents the quantum state of a particular memory stored as a bosonic condensate.

This may be expanded to:

$|\varphi\rangle=(a_{Y1}|Y\rangle+a_{N1}|N\rangle)+(a_{Y2}|Y\rangle+ a_{N2}|N\rangle)+(a_{Y3}|Y\rangle+a_{N3}|N\rangle)+ \dots +(a_{Yn}|Y\rangle+a_{Nn}|N\rangle)$ (3)

and:

$|\varphi\rangle = (a_{Y1} + a_{Y2} + a_{Y3} + \dots + a_{Yn})|Y\rangle + (a_{N1} + a_{N2} + a_{N3} + \dots + a_{Nn})|N\rangle$ (4)

where a_{Yn} represents the amplitude of the nth state $|Y\rangle$ and a_{Nn} represents the amplitude of the nth state $|N\rangle$.

It is thus clear that, if the memories one unconsciously consults before receiving the answer to the question "am I fat?" are predominantly those with large vales of a_Y, or if at least some of those values are excessively large, then the amplitude associated with $|Y\rangle$ in the equation for $|\varphi\rangle$ will also have a large value, resulting in a large probability of perceiving oneself as fat:

$P(Y)=|a_Y|^2=|(a_{Y1} + a_{Y2} + a_{Y3} + \dots + a_{Yn})|^2$ (5)

and a low probability of perceiving oneself as not fat:

$P(N)=|a_N|^2=|(a_{N1} + a_{N2} + a_{N3} + \dots + a_{Nn})|^2$ (6)

Of course, the memories in question ($|\Psi_n\rangle$) need not be exclusively concerned with whether one is or is not fat (although they often are in the case of AN). We can conceive of an operator F that measures a particular quantum state for the quality of being 'fat', such that $\langle Y|F|\varphi\rangle$ represents the overall probability amplitude of measuring $|Y\rangle$ given $|\varphi\rangle$ and $\langle N|F|\varphi\rangle$ represents the overall probability amplitude of measuring $|N\rangle$ given $|\varphi\rangle$. This analysis would mean that the memories contained in state $|\varphi\rangle$ could be 'measured' using a different operator to cause a different kind of perception, and that the individual bosonic condensates $|\Psi_n\rangle$ might be memories of a more general nature.

The processes described above would probably be unconscious, as literature on the subject of Quantum Paradigms of Psychopathology has thus far considered quantum logic to be the logic of the unconscious (for example, Zizzi & Pregnolato, 2012). On this basis, all of the above would occur without the anorexic patient being aware of it: s/he would only experience the fact that, when s/he looks in a mirror or turns his/her mind to the subject of whether or not s/he is fat, the answer that comes back is, almost invariably, and despite reality, 'yes'.

Parts II and III of this paper will make some suggestions regarding the physical location in the anorexic brain of the abstract logic discussed above.

II. Locating the effect I: microtubules, vesicles and 5-HT$_{2a}$ receptors

The first *possible* location of the abstract quantum logic discussed in Part I is in the microtubules of the neuronal cytoskeleton.

In commentary to a recent paper by Pothos and Busemeyer (2013), which suggested that human cognition could be better modelled by quantum probability theory than by classical Bayesian probability theory, Hameroff outlines the way in which such quantum cognition might be explained by locating quantum effects (including superposition, entanglement, non-commutability and quantum computation) in the brain's cytoskeletal microtubules. These microtubules are made up of tubulin proteins, in which aromatic rings of phenyl or indole are arrayed along "quantum channels". Collective dipoles along such channels may exist in quantum superposition of alternative orientations. According to the Penrose-Hameroff Orchestrated Objective Reduction (Orch OR) hypothesis, this quantum state of both alternatives in superposition is said to reduce every 25ms to a classical state where there is only one orientation in existence.

The role of the microtubule within the neuron is to transport vesicles containing molecules of neurotransmitter to the terminal button of the axon, where the neurotransmitter will be released into the synapse and will find its way to postsynaptic receptors (Carlson, 2010). One such receptor is the 5-HT$_{2a}$ receptor, which detects serotonin (5-HT) in the synapse and thereupon triggers an action potential in the receiving neuron. It has been found that patients with AN have significantly reduced 5-HT$_{2a}$ binding potential in the left frontal cortex, in the left and right parietal cortex, and in the left and right occipital cortex when compared with healthy volunteers (Audenaert *et al.,* 2002).

The analysis in Part I of this paper is an abstract one; however, if it is true, it may be made more physical by reference to the account given in this Part of the Orch OR hypothesis. If the role of the microtubule is to transport vesicles to the terminal button, and if it is reasonable to suppose that different orientations of tubulin within the microtubule would alter the overall macroscopic structure of

the microtubule, then it is reasonable to suppose that the alternative orientations of tubulin that exist in quantum superposition would have alternative consequences for the larger-scale organisation and function of the microtubule, such that one orientation might end up transporting vesicles to the terminal button at a faster rate or larger quantity than the other.

We may thus suppose that the 'yes/fat' state discussed in part I might represent one orientation of tubulin, and the 'no/not fat' state might represent another. Based on the analysis in Part I, the 'yes/fat' state would have a larger probability amplitude than the 'no/not fat' state, such that it would occur, in patients with severe AN, almost all of the time. It may be supposed that the structure and function of the microtubules whose tubulin reduces into the 'yes/fat' state would be such that fewer vesicles reach the terminal button, or that they are transported more slowly to it. These vesicles would be carrying serotonin, and the reduced amount of serotonin in the terminal button would account for the reduced amount of serotonin being released into the synapse, which in turn would account for the reduced binding potential of 5-HT_{2a} receptors in certain parts of the anorexic brain, since there would be fewer 5-HT molecules in the synapse to bind with the 5-HT_{2a} receptors.

III. Locating the obsession: the insula, the amygdala, and Pribram's Holonomic Brain Theory

The second *possible* location of the abstract quantum logic discussed in Part I is in the threat- and equivalence-detection functions of the amygdala.

As well as implicating the role of 5-HT_{2a} receptors in the anorexic brain, recent research has also implicated the insula, or insular cortex, as a site of special interest. This will be discussed here in relation to the role of the amygdala in Pribram's Holonomic Brain Theory (Pribram, 2011).

Nunn, Frampton, Gordon and Lask (2008) propose that the clinical phenomena of AN may be explained by insular dysfunction. In particular, it is suggested that it is the insula's role in certain neural circuits that is important, and that dysfunction of the insula leads to dysfunction of the whole circuit. They write that: "*precipitating factors… may… trigger threat detection in the amygdala with a subsequent anxiety response… the amygdala then communicates with the insula… Because the insula is unable to integrate this affective information load with a reliable sense of body perception… or state of hunger/satiety… eating will be restrained*".

Pribram (1991) does not deal with the insula itself; he does, however, discuss the role of the amygdala in processing equivalence. He notes experiments with monkeys in which amygdalactomy seemed to reduce their perception of threat. Amygdalactomy also seemed to reduce their ability to recognise the equivalence of values, in that monkeys that had undergone amygdalactomy were unable to transfer the value of 'largeness' from circles to squares and were unable to transfer the value of 'lighter grey' from one task to another, while monkeys that had not undergone amygdalactomy had no trouble in these tasks. The monkeys that had undergone amygdalactomy seemed to treat each of these tasks as being entirely novel, implying that the amygdala's role in detecting threats and carrying values from one task to another is related to a more general role of searching for equivalences. Those without an amygdala will treat every situation as novel, while those with an (over)active amygdala will see more situations as being equivalent, whether that equivalence is defined by the presence of a threat, the largeness of the shapes, or the shading of some colours.

Which brings us back to the question of AN. In the view of Nunn et al. (2008), the key dysfunction in the anorexic brain is found in the insula. However, this dysfunction must not be considered on its own, but rather in the context of a wider neural circuit beginning with the amygdala. Part I of this paper, and indeed my previous paper (Marlow, 2013), suggested that AN could be understood, at least partially, by reference to the question "am I fat?" and the probability amplitudes associated with each possible answer. This question must arise somewhere, and it is suggested that it arises in the amygdala. Just as an active amygdala will look for equivalences relating to 'danger', or 'largeness', or 'lighter grey' shading, so it might also look for equivalences relating to 'fatness'; or, to put it another way, 'fatness' is viewed as a threat in the anorexic mind, and thus the amygdala is overactive in its detection of whether the anorexic is fat or not, thus

triggering the question above and the probability amplitudes associated with its answers. Given that the state of 'yes/fat' will have a much greater probability of occurring than the state of 'no/not fat', the amygdala will almost constantly be in a state where the threat of 'fatness' is being detected.

It is worth noting that when an anorexic perceives herself to be fat, s/he does not just *see* it, but can also *feel* it. This observation points to the important role of the amygdala in AN, as it is in the amygdala and the orbitofrontal cortex that the visual sensory information converges with the somatosensory and olfactory sensory information (Loewenstein, 2013, p.179). Thus, if the problem were related solely to the his/her *visual* perception, one would be looking at other parts of the brain, but because it involves *visual* and *somatosensory* perception, it is appropriate to look not at these individual systems, but at the part of the brain where these systems converge.

Moreover, insofar as the notion of 'grandfather cells' or 'grandmother cells' (clusters of neurons that fire in response only to very specific stimuli, such as a grandmother's face, and thus facilitate recognition of that very specific stimulus) is correct[2], these are found in the amygdala (Loewenstein, 2013, p.197). It may be suggested, then, that there is such a cluster of cells in the amygdala that responds to recognition of the category 'fat', and, in the anorexic brain, in response to visual and other sensory signals , that fires when the category 'fat' is perceived. The abstract analysis in Part I of this paper would thus be expressed as an increased probability amplitude for this cluster of cells to fire in the anorexic brain, even in the absence of an appropriate outside stimulus.

If the hypothesis of Nunn et al. (2008) is correct, this detection of threat or equivalence will cause an anxiety response in the anorexic's mind and will cause the amygdala to communicate with the insula. Given the insula's dysfunction, causing it to be unable to integrate this threat detection with a reliable sense of body perception or of hunger/satiety, the anorexic will have no other guide by which to act except the threat detection of his/her amygdala and the ensuing anxiety, and will thus restrain his/her eating or pursue other means of losing weight, such as excessive exercise, vomiting, or abuse of diuretics. Moreover, while the amygdala can communicate with the insula, it is also possible for the insula to communicate back to the amygdala; thus, this feedback loop may reinforce the large probability amplitude associated with the amygdala detecting the state 'yes/fat'. Thus, while the insula's dysfunction is indeed a crucial factor in this psychopathology, so is the underlying quantum logic discussed in Part I and localised to the amygdala in Part III of this paper; the two cannot be considered separately.

V. Concluding remarks

In this paper, I have developed the quantum account of AN begun in my previous paper and have suggested some tentative explanations by which the abstract account found in Part I of this paper might be explained on a physical level, for example by locating the quantum logic of Part I to the orientation of tubulin channels in cytoskeletal microtubules (Part II) or to the threat detection mechanism of the amygdala (Part III). It must be emphasised that the ideas developed in this paper are highly speculative: the idea that quantum mechanics might be useful to understand normal brain function is itself a controversial idea (see, for example, Part 2 of the author's previous article (Marlow, 2013) for a critical discussion of Quantum Theories of Mind), and so the idea that it might be useful in understanding abnormal brain function and psychopathology is even more so. Nevertheless, it is hoped that, as more evidence from quantum biology is collected in support of *quantum theories of mind*, this new and speculative science might become more concrete and practical, and that these first speculative steps into the unknown might one day form the basis of a new understanding of, and hopefully treatment for, mental disorders such as Anorexia Nervosa.

[2] The notion of 'grandfather cells' or 'grandmother cells' is not in itself uncontroversial. See, for example, Jibu & Yasue (1995, p.108-9) for a discussion of how the existence of an individual 'grandfather cell' that fires in response to a specific stimulus might merely be representative of an underlying dendritic network: "[a]s a result of cooperative activity within the dendritic network, one of the participating neurons fires when a person sees the face of his or her grandfather" (p.109).

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