If waking and dreaming consciousness became de-differentiated, would schizophrenia result?

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If both waking and dreaming consciousness are functional, their de-differentiation would be doubly detrimental. Differentiation between waking and dreaming is achieved through neuromodulation. During dreaming, without external sensory data and with mesolimbic dopaminergic input, hyper-cholinergic input almost totally suppresses the aminergic system. During waking, with sensory gates open, aminergic modulation inhibits cholinergic and mesocortical dopaminergic suppresses mesolimbic. These neuromodulatory systems are reciprocally interactive and self-organizing. As a consequence of neuromodulatory reciprocity, phenomenologically, the self and the world that appear during dreaming differ from those that emerge during waking. As a result of self-organizing, the self and the world in both states are integrated.

Some loss of self-organization would precipitate a degree of de-differentiation between waking and dreaming, resulting in a hybrid state which would be expressed heterogeneously, both neurobiologically and phenomenologically. As a consequence of progressive de-differentiation, certain identifiable psychiatric disorders may emerge. Ultimately, schizophrenia, a disorganized-fragmented self, may result.

1. Introduction

This paper elucidates the hypothesis that schizophrenia results from the progressive de-differentiation of waking and dreaming consciousness. In support of this, research is synthesized from several domains which have tended to be discrete: the differentiation of conscious states; phenomenological and neurobiological differences in waking and dreaming; phospholipid metabolism in neuronal membranes; dreaming and memory consolidation and work on the neurobiology, phenomenology and heterogeneity of schizophrenia. The methodology is hermeneutic in the sense that a more holistic understanding of schizophrenia may emerge through considering the interrelationships between these areas of work-essentially, iterative steps of 'what is the significance of this evidence in this domain in the context of that evidence in that domain'. Keshavan, Tandon, Boutros, and Nasrallah (2008) comment that the accumulating 'fact-base' on schizophrenia requires new integrative approaches that can generate testable predictions, this particular hypothesis is in this spirit.

Differentiation is achieved between the conscious states of waking and dreaming (see later). If differentiation has to be achieved, it can fail to be achieved, resulting in de-differentiation and a hybrid waking/dreaming state. The mind–brain processes that achieve differentiation are complex and, although reciprocally interactive, can vary independently. Therefore, de-differentiation would be expressed heterogeneously. However, one unifying thread is that conscious experience of the world and the self would both be disrupted in a de-differentiated state. The integrity of waking and dreaming consciousness...
underlies the differing experience of the self and the world in the two states. De-differentiation would compromise the integrity of both states (although not necessarily to the same extent at the same time). The progressive de-differentiation of waking and dreaming consciousness and associated loss of self-organization in both states may gradually result in schizophrenia: a fragmented self. It is argued that understanding of both the heterogeneity of schizophrenia and the disruption to self-experience can be advanced through this de-differentiation hypothesis.

2. Consciousness, conscious experience and the heterogeneity of schizophrenia

Schizophrenia is a heterogeneous condition. In classic work Bleuler (1911/1950) referred to the ‘group of schizophrenias’. In a recent comment, Carpenter (2008, p. 1003) reiterated that, ‘Schizophrenia has the nosological status of a clinical syndrome rather than a validated single disease entity. Heterogeneity in clinical presentation and course is routinely observed, and heterogeneity of disease processes is likely’. Tandon, Nasrallah, and Keshavan (2008, p. 1) refer to an ‘...admixture of positive, negative, cognitive, mood and motor symptoms whose severity varies across patients...’. Moreover, there is acceptance that distinctions between all psychiatric illnesses are fuzzy and symptomatic profiles overlap (Brockington and Leff, 1979). Kendall and Jablenski (2003, p. 4) conclude that, ‘...there is little evidence that most currently recognized mental disorders are separated by natural boundaries. Researchers are increasingly assuming that variation in symptoms is continuous’. For example, obsessive-compulsion disorder (OCD) is not infrequently a co-diagnosis with schizophrenia (Krüger et al., 2000). OCD symptoms have been found in up to 50% of patients with schizophrenia (Berman, Merson, Viegner, et al., 1998). In particular, it appears that schizophrenia and bipolar disorder may be closely related, they share the same etiology (Craddock, O'Donovan & Owen, 2006; Craddock & Owen, 2005; Lichtenstein et al., 2009; Maier, Zobel & Wagner, 2006), exhibit symptomatic overlap (Lake, 2008; Lake & Hurwitz, 2006; Pope & Lipinsks, 1978), can follow a similar deteriorating course (Addington and Addington, 1997; Zuibietil, Huguetel, O'Neil, & Giordani, 2001), involve sleep disturbances (Benson, 2006; Costa e Silva, 2006), can be difficult to distinguish clinically (Craddock & Owen, 2005; Owen & Craddock, 2009; Walsh, 2009) and partly share genetic determinants (Craddock, O'Donovan & Owen, 2005, 2006; Lichtenstein et al., 2009). Indeed, the diagnosis of schizoaffective disorder recognizes the overlap between schizophrenia and bipolar disorder. Craddock and Owen (2005) comment on the multidimensional space occupied by functional psychiatric pathologies, ‘The recent findings are compatible with a model of functional psychosis in which...[there is] susceptibility to a spectrum of clinical phenotypes...[or indeed]...a multidimensional space [where] in addition to bipolar disorder and schizophrenia there is genetic overlap between the functional psychoses and major depressive disorder – and, indeed, other disorders – with extension into sub-clinical (or normal) variation’. Reviews of population surveys indicate that attenuated psychotic experiences are reported by 5–8% of respondents (Polanczyk et al., 2010; van Os, Linscott, Myin-Germey's, Delespaul, & Krabbendam, 2009). Over 10 years, for a representative sample of adolescents and young adults from the general population, Dominguez, Can Saka, Lieb, Wittchen, and van Os (2010) reported a 12% cumulative prevalence rate for both negative/disorganized and positive psychotic symptoms.

Heterogeneity in presentation and course, overlap with other psychiatric disorders and extension (in attenuated form) into the general population implies difficulty (indeed, most probably, impossibility) in delimiting schizophrenia to a core constellation of signs and symptoms. As compared to other illnesses, the diagnosis of schizophrenia has been described as an art (Eaton, Hall, Macdonald, & McKibben, 2007). One statement that can be definitively made, however, is that schizophrenia involves consciousness and disrupts normal conscious experience. As consciousness is a notoriously elusive and contest concept (see, for example, Chalmer, 1996), this may not advance matters much unless there is clarification of how ‘consciousness’ and ‘conscious experience’ are understood here.

In this paper, ‘Consciousness is the appearance of a world’ (Metzinger, 2009, p. 15). A world appears in both waking and dreaming consciousness (but not in deep dreamless sleep), moreover, when a world becomes present, a self also emerges as a part of this world (Metzinger, 2009, p. 57). The world and the self that appear during waking differ, in certain respects, from those that appear during dreaming. (This paper focuses on REM dreaming, rather than the more literal and thought-like NREM, for a review of the differences see, Nielson, 2003).

‘Conscious experience’ is understood in the sense of a ‘cognitive global workspace’ (Baars, 1988, 1997, 2002). At any point in time conscious experience focuses on a particular aspect of the world. Conscious experience, ‘...resembles a bright spot on the stage of immediate memory, directed there by a spotlight of attention under executive guidance’ (Baars, 2005, p. 46). Again conscious experience differs in waking and dreaming, although there is a ‘spotlight’ of attention in both, during (non-lucid) dreaming ‘executive guidance’ or the intentional control of that ‘spotlight’ is lacking. Moreover, as working memory is much attenuated in dreaming, the ‘stage of intermediate memory’ dissolves, so, from the perspective of waking consciousness, it is unclear why the spotlight is focused where it is in dreaming.

In sum, waking and dreaming consciousness are differentiated both with respect to the world that emerges and the self that becomes present. The next section addresses how this differentiation is achieved at the neurobiological level.

3. Achieved differentiation between waking and dreaming consciousness

Achieved differentiation between waking and REM dreaming consciousness can be partly understood through the ‘AIM Model’ (Hobson, 1990, 1992, 1997; Hobson & Stickgold, 1994; Kahn, Pace-Schott, & Hobson, 1997). AIM suggests that all conscious states reflect three processes: the level of brain activation (‘A’); the origin of inputs (‘T’); and dynamic reciprocity...
between aminergic and cholinergic neuromodulation (‘M’). Although these processes do tend to vary in concert, they can also exhibit independent variability (Hobson, Pace-Schott and Stickgold, 2003).

Activation (‘A’) is a measure of conscious experience (the ‘spotlight’ of attention), ‘... as reflected in the length, intensity and complexity of subjective reports of mental activity...’ (Hobson, Pace-Schott, and Stickgold, 2003, p. 40). Although, phenomenologically, reports of conscious experience in dreaming and waking differ in kind (see later), both states are equally activated (Hobson, 2002). Indeed, Tononi (2004) argues that during REM dreaming activation may be even higher than in waking. The origin of inputs (‘I’) reflects the extent to which the brain–mind is making sense either of external sensory data or internally generated percepts, this difference is driven by both input–output gating of external stimuli and the strength of internal data sources (Hobson, Pace-Schott, & Stickgold, 2003). As would be expected from the reported measures of conscious experience, thalamocortical activation is at normal (waking) levels during REM dreaming (Behrendt, 2006; Paré and Llinás, 1995), so, to achieve differentiation between the two states, external input must be excluded (Hobson, 1999).

When sensory stimuli from the external world are blocked, hallucinatory imagery suffuses consciousness (Behrendt, 2003; Behrendt & Young, 2004). In contrast, during waking, internal perceptions are gated by sensory afferents so hallucinatory images do not normally arise (Behrendt, 2003; Llinàs & Parè, 1991). Fictive hallucinatory movement in dreams is compelling. Not only are motor commands issued to motor neurons but they are also copied to the sensory system, motor output during dreaming is blocked only through postsynaptic inhibition (Hobson, 1999).

Neuromodulation ensures unified, coherent mind/brain responses. In the AIM model, modulation (‘M’) is the ratio of aminergic (noradrenergic and serotonergic) to cholinergic inputs, the predominance of aminergic modulation during waking supports directed thought, insight, judgment, abstract thinking, decision-making, working memory and space–time orientation, the almost total lack of aminergic input to dreaming suppresses these executive functions (Hobson, Pace-Schott, & Stickgold, 2003). Process changes in the three AIM parameters go some way to explaining how the differentiation between waking and dreaming is achieved. However, Hobson, Pace-Schott and Stickgold (2003) recognize that the three dimensions of ‘AIM’ do not exhaust the conscious state space; indeed, they propose that more dimensions exist. It is suggested here that to advance understanding of the achieved differentiation between waking and dreaming and, hence, to grasp the consequences of a de-differentiated state, a more comprehensive account of ‘M’ is required (to include mesolimbic–mesocortical dopaminergic modulation) and a self-organization dimension (‘O’), which is dependent upon ‘I’ and ‘M’, should be included. The reasons for these modifications are discussed, in turn, next.

Solms (1999, 2002) argues that the role of dopamine (DA) in directing the dreaming state has been neglected. Indeed he posits that the mesolimbic–mesocortical dopaminergic pathway is crucial for dream generation. Although it is possible that aminergic demodulation may facilitate dopaminergic effects, these DA effects are primary (Solms, 2000). A dynamic reciprocal relationship between mesocortical and mesolimbic DA projections has been proposed (Davis, Kahn, Ko, & Davidson, 1991; Pycock, Kerwin, & Carter, 1980). It has also been shown in rodents that serotonergic systems can modulate the impact of mesolimbic DA (Barr et al., 2004). There is overwhelming evidence that the prefrontal DA inhibits subcortical DA activity (for a review see Guillain, Abi-Dargham, & Laruelle, 2007). Mesocortical DA enables prefrontal neuronal activity whereas mesolimbic DA has a critical role in motivation and emotion (Mathé, Nomikos, Blakeman, & Svensson, 1999). As discussed below, on the phenomenological level, dreaming is a hyperemotional state where thinking reaches its nadir. Hence, it is intuitively plausible that, during dreaming, mesolimbic DA may predominate over mesocortical DA, with the reverse during waking. A sensitized mesolimbic dopaminergic system is correlated with an increase in cholinergic input (Sarter, Nelson, & Bruno, 2005). So, during dreaming, increasing mesolimbic DA is associated with increasing cholinergic neuromodulation. Thus, dynamic reciprocity characterizes not only aminergic/cholinergic systems but also mesocortical/mesolimbic dopaminergic systems and mesolimbic dopaminergic/cholinergic systems.

The combined impact of shifts in ‘I’ and ‘M’ most probably account for the changes in regional activation of the brain that also differentiate dreaming and waking (Hobson, Pace-Schott, & Stickgold, 2003). For example, the gating of external stimuli along with the suppression of aminergic and mesocortical DA input may explain the de-activation of the frontal lobe during REM dreaming. Conversely, the selective activation of the limbic system during REM dreaming may be due to cholinergic and mesolimbic DA stimulation accompanied by strong internally generated percepts. Specifically, Hobson (1999, p. 152) argues that dreaming is initiated in the pontine brainstem triggering activation, aminergic demodulation and cholinergic hyperactivity which selectively engages the limbic areas. The engagement of the limbic lobe then triggers primary emotions (e.g. fear, elation and rage). These emotions drive dream plot elaboration which is constituted through the visual imagery centres of the associative cortex, the spatial centres of the parietal lobe and the narrative organizing centres of the temporal lobe. Solms (2003) argues that dreaming (unlike REM architecture) is preserved with pontine brain stem lesions whereas a complete cessation of dreaming is observed with lesions of the parieto-temporo-occipital (PTO) junction. Despite their differences, both Hobson and Solms agree on certain features of the differential regional activation of the brain during REM dreaming: the dorsolateral prefrontal cortex and the primary visual cortex are deactivated, whereas limbic areas and the occipitotemporal cortical areas are selectively activated. Normal spatial cognition through the spatial centres of the parietal lobe is preserved in both waking and dreaming.

Both waking and dreaming are self-organized mind-brain states (Kahn, Pace-Schott, & Hobson, 1997; Tononi, 2004; Tononi, 2008; Tononi & Edelman, 1998). The mind–brain self-organizes through sensory data and neuromodulation (Freeman, 2005; Singer, 1986). As noted above, although external sensory input is largely ignored during dreaming (Mahowald, Woods & Schenck, 1998) self-organization through neuromodulation and internally generated percepts continues (Kahn, Combs, & Krippner 2002). During waking, self-organization generates a brain ‘forward model mechanism’ that enables
Differentiation between waking and dreaming depends on dynamic reciprocity and is a ‘constantly negotiated compromise’ (Hobson, 2002, p. 102). Hence, as is predicted by chaos theory, the achieved differentiation between waking and dreaming is both vulnerable and volatile. Indeed, this is confirmed by the existence of transient dissociated states: lucid dreaming; REM sleep disorder; and waking fantasy or ‘daydreaming’ (see Hobson, Pace-Schott and Stickgold, 2003, for a review). De-differentiation differs from state dissociations in that de-differentiation would impact upon both waking and dreaming consciousness. Also de-differentiation be an enduring (and sometimes progressively deteriorating, see later), rather than a transient condition.

Systems, such as the mind-brain, that are poised between order and disorder are optimal for complex tasks (Kauffman, 1993). If de-differentiation between waking and dreaming occurred then the ability to undertake complex tasks in both states would be effected detrimentally. One of the key tenets of chaos theory is uncertainty over the precise ways in which these effects would be manifested. In a state of self-organized criticality, a small perturbation in the mind-brain (in this hypothesis a small shift into de-differentiation) can precipitate fluctuations which are largely unpredictable on the basis of initial conditions (Bak, 1996; Orsucci, 2006).

4. How could de-differentiation occur?

The implication of the above arguments is that the heterogeneity associated with schizophrenia (and, possibly, other related psychiatric/neurodevelopmental disorders) may reflect the multifarious possibilities inherent in the process changes that achieve differentiation between waking and dreaming in the mind-brain. Or, in other words, schizophrenia is a condition of the ‘anomalous dynamics’ (Orsucci, 2006) that can emerge within any system poised at self-organized criticality. But not all humans descend into schizophrenia, so the question arises of how these anomalous dynamics are triggered in susceptible individuals.

There is evidence of neuronal cell membrane phospholipid abnormalities in patients with schizophrenia (Glen et al., 1994; Horrobin, Manku, Hillman, Iain, & Glen, 1991; Nuss et al., 2009; Yao & Reddy, 2000; Yao, van Kammen, & Welker, 1994). These membrane abnormalities are observed prior to treatment, so they may be trait-related (Jayakumar et al., 2003; Reddy, Keshavan, & Yao, 2004). Schizophrenia may be associated with deficient uptake or excessive breakdown of membrane phospholipids (Fenton, Hibbeln, & Knable, 1999). Excessive breakdown may flow from the over-activity of calcium-independent phospholipase A2; such over-activity has been demonstrated in schizophrenia (Horrobin, 1996; Macdonald et al., 2004; Smesny et al., 2005). Evidence of abnormal fatty acid metabolism in schizophrenia includes: reduced fatty acid levels in cell membranes; reduced skin flush response to topical niacin; abnormal electroretinogram; increased levels of calcium-independent phospholipase A2 in blood and brain; and abnormal 31P magnetic resonance spectroscopy of brain phospholipids (Peet, 2002).

Essential fatty acids (EFAs) are key components of neuronal cell membranes. Eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) are common essential fatty acids in the brain (see, for example, Horrobin, 1998). Supplementation with EPA has been shown to be effective as an adjunct treatment for already medicated patients with schizophrenia (Emsley, Myburgh, Oosthuizen, & van Rensburg, 2002; Peet, Brind, Ramchand, Shah, & Vankar, 2001; Peet & Horrobin, 2002; Shah, Vankar, Telang, Ramchand, & Peet, 1998). There is indicative evidence that drug-naïve patients may respond too (Puri et al., 2000; Richardson et al., 2000). EPA has also been shown to reduce the risk of psychosis in high-risk individuals (Amminger et al., 2007), young people with subthreshold psychotic states (Amminger et al., 2010) and enhance treatment response in patients with a first episode (Berger et al., 2007).

Moreover, EPA, along with DHA, has also been found to be efficacious for bipolar disorder (Stoll, Severus & Freeman, 1999; and unipolar and bipolar depression (Frangou, Lewis, & McCrone, 2006; Freeman et al., 2006). In particular, the therapeutic effects of EPA have been shown to be equal to fluoxetine in treating major depressive disorder (Jazayeri et al., 2008). The inclusion of EPA along with other essential fatty acids, vitamins and minerals in the diet of prisoners reduced the incidence of anti-social behaviour (including violence) by 26.3% (Gesch, Hammond, Hampson, Eves, & Crowder, 2002). The administration of EPA and DHA decreased feelings of anger in substance abusers (Buydens-Branchey & Branchey, 2008). Ultra pure ethyl-eicosapentaenoic acid (E-EPA) diminished aggression and depressive symptoms in women with untreated borderline personality disorder (Zanarini & Frankenburg, 2003). Essential fatty acid deficiencies (with improvements after supplementation)
have been found to be implicated in three developmental disorders which are frequently co-morbid: dyslexia (Richardson, Easton, & Puri, 2000; Richardson & Ross, 2000; Richardson et al., 1999; Richardson et al., 2000a, 2000b; Stordy, 1995; Stordy, 1997; Taylor & Richardson, 2000; Taylor et al., 2000), dyspraxia (Richardson & Ross, 2000; Stordy, 1997) and ADHD (Richardson & Puri 2000, 2002; Stevens et al., 1995; Stevens, Zentall, Abate, Kuczek, and Burgess, 1996). In a review, Garland and Hallahan (2006) concluded that decreased levels of essential fatty acids are implicated in psychiatric conditions characterized by disordered impulsivity (ADHD; borderline personality disorder; suicide, deliberate self-harm, aggression and homicide) and that improvement occurred when deficiencies were corrected. Although these results are preliminary and further investigation is required, they are indicative of a similar therapeutic role for fatty acid supplementation across a range of psychiatric conditions, thereby lending weight to the hypothesis that these conditions are related.

Normal phospholipid metabolism ensures the dynamic fluidity of cell membranes and, thus, preserves multiple neuronal structures and functions including receptors, ion channels, signal transduction and second messengers, moreover, lipid products derived from phospholipid membranes regulate cell signaling systems (Hallahan & Garland, 2005; Kinsella, 1990). Specifically, essential fatty acids are also thought to be associated with dopaminergic and serotonergic transmission, brain glucose metabolism, eicosanoid synthesis, gene expression, cell growth and may guard against apoptosis (Liperoti, Landi, Fusco, Bernabel, & Onder, 2009). Thus abnormal phospholipid metabolism, associated neuronal cell membrane abnormalities and, consequent disrupted neurotransmission may serve as a point of convergence for the generalized dysfunction associated with schizophrenia.

The ‘membrane theory of schizophrenia’ Horrobin, Glen, and Vaddadi (1994) is compatible with disruption to all neurotransmitter systems. Horrobin et al. (1994, p. 204) comment, ‘...disruption of the fundamental structure of membranes could lead to serious brain malfunction, with demonstrable abnormalities of brain histology and abnormalities in the functioning of all neurotransmitter systems: the components of such transmitter systems are all associated with membranes and all function through changing membrane behaviour.’ Specifically, membrane abnormalities have been shown to cause disruption to dopaminergic (Malnoe, Milon & Reme, 1990) and cholinergic (Fong & McNamee, 1986) neurotransmitter systems. Reviewing evidence on dietary manipulation of membrane phospholipid composition in rats, du Bois, Deng, and Huang (2005) found impacts on dopamine, serotonin and muscarinic receptor densities; they deduced that there is a tight relationship between membrane phospholipid status and normal neurotransmission. In a review, Chalon, Vancassel, Zimmer, Guilloteau, and Durand (2001) concluded that strong links characterize phospholipid status, neurotransmission processes and behavioral disorders. Phospholipid status is determined by genetic markers in conjunction with diet and stress as environmental factors, the enzymes that control phospholipid synthesis and breakdown are under genetic control but the essential fatty acids in neuronal membranes cannot be manufactured in the body, they must be included in the diet (Horrobin, 1998). In cross-national studies, there is epidemiological evidence that dietary consumption of essential fatty acids modifies the risk for neuropsychiatric disorders (Hibbeln, 1998; Peet, 2004; Young & Conquer, 2005). Specifically, in such studies, greater seafood intake was associated with lower incidence of bipolar disorder (30-fold range) and major depression (50-fold range) (Hibbeln & Salem, 2001).

In addition to effective transmitter-receptor interaction, of particular importance to normal neurotransmission is the scavenging of excess transmitter from the synapse to terminate the signal (Gazzaniga, Ivy, & Mangun, 2002). Moreover, feedback through membrane receptors coordinates the rates of neurotransmitter release with rates of reuptake and rates of breakdown to ensure the appropriate concentration level for each neurotransmitter in the synapse (Freeman, 1992). Membrane abnormalities would impede neurotransmitter-receptor interaction, disrupt the processes that control the concentration of neurotransmitters, interfere with the reuptake and reentry of excess neurotransmitters into the presynaptic neuron and result in a loss of control over neurotransmitter release. Thus the consequence of membrane abnormalities would be some chaotic intermingling of neurotransmitters within the synapse. Ultimately, due to the impact of membrane abnormalities and anomalous phospholipid metabolism on multiple transmitter systems, the waking and dreaming states may become de-differentiated as some dynamic reciprocity between aminergic/cholinergic/dopaminergic systems is lost. Clearly, this loss of dynamic reciprocity would be a matter of degree. The impact would be slight if membrane abnormalities and/or anomalous metabolism were minimal or considerable if they were grossly aberrant.

Although genetic factors along gene–environment interplay have been shown to contribute 80% to a liability for developing schizophrenia (Tandon, Nasrallah, & Keshavan, 2008), there must be a neurobiological substrate through which this interplay is expressed (Horrobin, 1998). The impact of membrane abnormalities and anomalous phospholipid metabolism on multiple transmitter systems (leading to the de-differentiation of waking and dreaming consciousness) may be, among others, the expression of this genetic-environmental susceptibility. Why would this be most likely to happen in late adolescence?

Huttenlocher (1979) first reported the 30–40% decline in synaptic density in the frontal cortex during adolescence. This finding has since been confirmed by Glantz, Gilmore, Hamer, Lieberman, and Jaruskog (2007) who remark that this decline is consistent with the superior performance of adults (over children) in frontal cortical–dependent tasks that rely on this pruning process to enhance the efficiency of synaptic connectivity. As synaptic elimination occurs at the same developmental stage as schizophrenia emerges it is intuitively likely that the two are linked in some way. Feinburg (1982) first suggested that some pruning defect may cause schizophrenia. EFA metabolism is implicated in synaptic pruning (Crawford, 1992; Richardson & Puri, 2000) so metabolic abnormalities may precipitate pruning defects. However, an alternative (or contributory) explanation is that the 30–40% decline in synaptic density would greatly exacerbate an underlying cumulative (throughout childhood) de-differentiation process, as, after pruning, neurotransmission takes place through fewer circuits. The underlying cumulative de-differentiation process would, therefore, become manifest as psychiatric symptoms in adolescence.
De-differentiation would be 'underlying' as phospholipid status is determined through gene-environment interplay. During infancy and childhood this interaction would be expressed differentially due to the impact of diet (including, contemp-orarily, fatty acid supplementation, see above) and stress. As discussed above, phospholipids have highly significant structural and functional roles in the brain, so phospholipid status would have fundamental consequences for brain development.

It has been proposed that schizophrenia is a neurodevelopmental disorder (Murray & Lewis, 1987; Weinberger, 1986, 1987). The earlier onset and more severe form of schizophrenia in men (than women) have been attributed to neurodevelopmental differences (Castle & Murray, 1991). The 'membrane theory of schizophrenia' was developed within the neurodevelopmental paradigm, as gene-environmental interplay influences the way that neurons are laid down, differentiated, culled and re-modelled (Horrobin, 1998). Moreover, although high oestrogen levels have been shown to be protective against schizophrenia, differences in EFA metabolism may also play a part, as female rats metabolize EFAs more rapidly and, in the context of EFA deficiency, retain EFAs within membrane phospholipids more readily than males (Horrobin, 1998; Marra & de Alaniz, 1989). However, the neurodevelopmental approach is in no way in contradiction with disrupted neurotransmission models of schizophrenia. As argued above, there is a tight relationship between membrane phospholipid status and normal neurotransmission. Moreover, disrupted neurotransmission would be a fundamental aspect of the de-differentiation of waking and dreaming consciousness.

Any adequate understanding of the consequences of de-differentiation must make reference not only to neurobiology but to conscious, lived experience. Phenomenology, as the study of lived experience, is essential to the elucidation of psychopathology (Gallagher & Zahavi, 2008; Parnas, Sass, & Zahavi, 2008). If schizophrenia results from the progressive de-differentiation of waking and dreaming consciousness, resulting in a hybrid waking/dreaming state, to understand schizophrenia it is necessary to understand the phenomenology of both waking and dreaming.

5. The phenomenology of waking and dreaming

What is experience like during waking and dreaming consciousness? How far does conscious experience differ in the two states?

The world that appears during dreaming consciousness is completely internally generated in the sense that, as discussed above, external stimuli are excluded in favour of internal precepts. The world that appears during waking is also entirely internally produced but a major aspect of the production is making sense of external sensory data. Waking consciousness generates a strong sense that an external reality exists. To create this outer reality (and a self in this world) the brain-mind systematically externalizes the sense that 'I am present in a world outside my brain' (Metzinger, 2009, p. 23). As a result of this externalization, during waking my experience is of a subjective private 'inner' world that is present within an objective public 'outer' world. During waking I know my subjective world is self-generated whereas the objective external world is not. Moreover, I experience myself as both a subject and an object in this external world (Legrand, 2007). If I am of a philo- sophical inclination, I accept I have no direct access to the external world except through the representations my brain generates. Nonetheless, in normal waking consciousness, I do not distrust the objective material reality of the external outer world nor do I doubt that I exist in this domain.

During dreaming consciousness only one world appears, the distinction between subjective and objective worlds is lost, or very much attenuated. Consequently I am not aware that my dreaming world is self-generated. Much follows from this. In waking I reflect on and have insight into my own state of mind-brain. I know I am awake and am aware of another possibility- dreaming. In contrast, during dreaming I do not attribute a state of mind-brain to myself; also I am unaware that there is an alternative state-waking. Only during lucid dreaming is there awareness of the mind-brain state (Voss, Holzmann, Tuin, & Hobson, 2009)). Whilst I am awake I can travel mentally in time. Moreover, I am aware that I am re-visiting the past or anticipating the future (Metzinger, 2009). During dreaming my situation feels very immediate. I am always present. I do not travel mentally in time. Nor do I think about my situation (Kahn & Hobson, 2005a). Concomitantly, there is loss of sub jective thinking about the thinking, feeling and situations of those who populate my dreams. So 'theory of mind' is much diminished (although see, Kahn & Hobson, 2005b) and dreamers are not inclined to empathy. This loss of reflexivity means that I do not monitor the actions of self and others. Hence, during dreaming, I do not ponder ('Am I taking the 'right' action?'). So I cannot form flexible responses to situations. I do not think on the causes of my actions nor do I contemplate their consequences for self or others. What happens during (non-lucid) dreaming appears not to be under my control; or rather the issue of 'control' (like 'state of mind', 'causes' and 'consequences') does not occur to me. I do not run an internal commentary on external reality. Archer (2003) has termed this commentary the 'internal conversation'. During waking, in the subjective, private, internal conversation I 'talk to myself' as I try to adjust the world to my goals or make sense of 'what's going on' externally. In the world that my mind-brain has externalized I may, for example, converse politely with a colleague about organizational politics whilst, in my internal world, I command myself 'Extractre yourself, you're going to be late for the meeting at 1400' or advise myself, albeit from, sometimes, conflicting perspectives, 'She is looking bored, maybe she actually dislikes you. . .Do not be stupid, you know that she is on your side'. In sum, during dreaming all executive functions fade, without subjectivity, there is little abstract thinking, decision-making, sustained concentration or applied logic (Dietrich, 2003), self-monitoring and the inner conversation are also lost. However, despite (or, arguably, because of) all of these losses the authenticity of the dreaming world is not doubted.
Dreaming has been described as remarkably single-minded (Rechtschaffen, 1978). In part, this ‘single-mindedness’ comes about because the subjective inner ‘evaluating, sense making and advising’ mind is lost. With subjectivity ‘switched off’ the dreamer’s attention is totally caught up in a uni-dimensional, unravelling drama. Loss of reflexivity (including reality monitoring, ‘That can’t be true’) allows the dreamer to accept the highly unlikely, indeed, sometimes, the impossible (Nir & Tononi, 2010), the dead return, distinguished and famous public figures become close acquaintances, flying is effortless and dream characters are sometimes blended (in the sense that aspects of two persons are united into one). Consequently, from the perspective of waking consciousness, dream experiences are often bizarre with a disregard for space/time parameters (Scarone et al., 2008). The periodic, mundane, habitual behaviours that characterize waking are notably absent (e.g. washing, dressing and grooming). Also lack of self-monitoring implies that this neglect of self-care fails to register. The relatively dramatic content of dreaming consciousness (as compared with waking) is driven by primary emotions like anxiety, fear, rage and elation (see Hobson, 1999; Hobson, 2002). More subtle emotions, like serenity, humility and compassion are rare. Although, sustained concentration is negated as dream scenes rapidly appear, shift and dissolve, attention is focused and perception enhanced -as Leonardo da Vinci’s famous comment about ‘seeing more clearly in dreams’ attests. This focused attention/enhanced perception constitutes another aspect of the ‘single-mindedness’ (Rechtschaffen, 1978) of dreams.

However, during waking, focused attention equates to a spotlight under executive control (Baars, 2005). Such control ensures that only certain features of any visual scene fall under this spotlight and are, therefore, consciously perceived. The peculiarity of this was demonstrated by Simons and Chabris (1999) when, with their attention focused elsewhere, 50% of participants in their study failed to perceive a man dressed as a gorilla as he crossed their visual field. This is a dramatic illustration of the importance of attention to perception. As noted above, executive control is missing in dreaming, so attention cannot be directed. Directed attention is lost but perception is enhanced in dreams (Hobson, 2002). This may follow from the sparsity of dream scenes. Rather like stage sets, the scenes constructed during dreaming appear edited when compared with the usual complexity of the waking visual field. Therefore, the significance of the directed attention for perception may drop as there is less to distinguish and assimilate. In a computer simulation Hoffman and McGlashan (1997) showed that perception is enhanced when working memory capacity is reduced, as working memory is much diminished in dreaming this may be another contributory factor to the augmentation of perception in the absence of directed attention.

As thinking reaches its nadir in REM dreaming there is a concomitant peak in visual imagery (Fosse, Stickgold, & Hobson, 2001). Dreaming consciousness elaborates internally generated precepts into highly associative narratives dominated by this visual imagery (Hobson, 2002). Although the dreamer and other dream characters sometimes speak in (REM) dreams, this is relatively rare; when attempted, dreamers have difficulty in vocalizing their thoughts (Hobson, 2002). Moreover, the speech is stereotyped and does not form part of a coherent two-way conversation. As deliberate thought is absent these dreaming narratives cannot be ‘thought about’ as are waking narratives. Hobson (2002) argues that dreaming narratives are driven by emotional salience rather than directed thought. Therefore, the subjective inner ‘evaluating, sense making and advising’ mind is lost. Loss of reflexivity (including reality monitoring, ‘That can’t be true’) allows the dreamer to accept the highly unlikely, indeed, sometimes, the impossible (Nir & Tononi, 2010). In both dreaming and waking, the self surveys a spatially coherent environment (Nielsen & Stenstrom, 2005). Dreams feel real in the same way that the external world feels real during waking. Dreams do not present meaningless random images, dream material is organized and selective (Revonsuo, 2003). Solms (2003) argues that dream images are actively constructed through complex cognition. Phenomena that arouse motivated interest during waking (e.g. people, faces, animals, movement, places and landscapes) also dominate dreaming (Nir & Tononi, 2010). In both dreaming and waking, the self surveys a spatially coherent environment (Nielsen & Stenstrom, 2005). Dreams do not resemble abstract art. As noted above, dreaming, like waking, is sensorimotor; dreams simulate a very convincing sense of the self moving through space (Hobson, 2002, p. 30). The embodied self is central to waking consciousness (Merleau-Ponty, 2002), similarly, I experience myself as embodied and at the vortex of the unfolding drama that is my dream (Llinàs, 2002). Dreaming consciousness, like waking consciousness, is integrated. In both states a conscious scene is unified and seamless; hence, a scene cannot be deconstructed into components and is experienced from a private, single point of view (i.e. during waking one cannot simultaneously perceive both the old witch and the young woman in the visual illusion, similarly, during dreaming there is a single perspective on a scene) (Tononi, 2008; Tononi & Edelman, 1998).

The differences in phenomenology between waking and dreaming are most probably underpinned by shifts in input specificity and neuromodulation (Hobson, 2002), whereas, the similarities are likely to flow from self-organization, integration and comparable levels of activation. Specifically, the self-organized nature of dreaming, the active, complex, cognitive
processes required for dream construction and this phenomenological data on dream coherence indicate that dreams do not emerge from a disorganized brain.

6. How would de-differentiation be expressed?

Through synthesizing the above evidence on the neurobiology and phenomenology of waking and dreaming consciousness, some preliminary observations can be made on how de-differentiation would be expressed. Although, in line with chaos theory, the precise effects would be uncertain, the parameters of de-differentiation will flow from some loss of order (i.e. the diminution of the self-organized integrity of the waking and dreaming states). So the prime characteristic of de-differentiation will be a degree of disorder. This disorganization would follow from shifts in those parameters that enable the differentiation of waking and dreaming (i.e. input specificity and neuromodulation).

Hence any or all of the following may be involved, to a lesser or greater extent. First, shifts in ('I') which would equate, during waking, to a loss of focus on external sensory data, accompanied by increasing concern with internal precepts. During dreaming shifts in 'I' may include disturbed sleep due to increased sensitivity to external sensory data (see later). Second, changes in neuromodulation ('M'), so that, during waking, cholinergic and mesolimbic dopaminergic modulation rise (and amnnergic and mesocortical modulation fall). During dreaming, amnnergic and mesocortical dopaminergic modulation would rise (and cholinergic and mesolimbic modulation fall). Although both waking and dreaming are activated, in the sense that lengthy, complex and intense accounts of mental activity can be elicited, ultimately, this activation may not be preserved in a progressively de-differentiated state.

These neurobiological shifts will, naturally, result in phenomenological changes. If the degree of de-differentiation was slight then, for example, during waking there could be merely some ‘dreaminess’ through an increased focus on internal precepts. Or a modest increase in anxiety, as anxiety is the predominant emotion in dreaming (Hobson, 2002). Equally, as dreaming is a hyperemotional state characterized by primary emotions allied with a loss of executive control, waking problems with managing rage or controlling aggression may emerge. Or relationship difficulties may present themselves, as during dreaming there is little empathic connection with others. Or psychomotor control in waking may be affected as motor outputs are blocked in dreaming. Or difficulties with sustained concentration may be experienced, as concentration is not maintained during dreaming. Or thinking during waking may become less linear-logical and more highly associative, as dreaming is a hyper-associative state. If such shifts remain modest then the results of de-differentiation would hard to distinguish from ordinary functioning. Indeed, as any conscious state is a ‘constantly negotiated compromise’ (Hobson, 2002, p. 102) between waking and dreaming, it could be argued that such shifts are the essence of normality. That this is a matter of debate, however, is evidenced in the current draft of DSM-5 (see, www.dsm5.org), where new syndromes (e.g. temper dysregulation disorder with dysphoria) proliferate, this has elicited the criticism that under DSM-5 ‘normality’ will become a highly restricted zone (Wykes & Callard, 2010).

Leaving this debate aside, if the intrusion of an aspect of dreaming into waking becomes more pronounced and/or waking consciousness takes on multiple facets of dreaming, then various disorders may emerge. Slight anxiety may progress to ‘anxiety disorder’. Or if a lack of sustained concentration becomes combined with impulsivity (due to a disregard for consequences – another aspect of dreaming) this may progress into ‘attention deficit hyperactivity disorder’. Or if mood became highly reactive with a notable increase in impulsive aggression ‘borderline personality disorder’ may be the result.

As discussed earlier, as with all non-linear complex systems, a small disturbance in the mind-brain can give rise to large fluctuations which are unpredictable on the basis of initial conditions (Bak, 1996; Orsucci, 2006). Hence a small disturbance (equating to a minor neuronal membrane defect and/or metabolic abnormality) could give rise to any of the above possibilities. On the other hand, a small disturbance only implies some loss of self-organization. Moreover, it has been shown that the mind/brain strives towards self-organization (Cicchetti & Tucker, 1994; Fingelkurts & Fingelkurts, 2004; Goodwin, 1994). Any self organized state finds its own inherent configurations (Krippner & Combs, 2002). So, when understood in the terms of chaos theory, the progression (or not) of de-differentiation would depend upon complex dynamics. For example, a modest increase in anxiety could stabilize or may progress to ‘anxiety disorder’ whereupon a new stable configuration may be reached. So the progression of any disease process would depend upon the extent of the loss of self-organization as, only modest losses would enable new stable, albeit anomalous (e.g. anxiety disorder) configurations.

At the neurobiological level, minor neuronal cell membrane phospholipid abnormalities (which may impact on only a limited number of neurotransmitters) would limit the possibilities for disease progression. However, as discussed earlier, gross neuronal membrane defects and associated metabolic abnormalities could precipitate a chaotic intermingling of neurotransmitters in the synapse with the progressive loss of self-organization and subsequent on-going de-differentiation of waking and dreaming consciousness. Ultimately, as more and more aspects of dreaming consciousness become incorporated in the waking state, identifiable psychiatric disorders (including psychosis) may emerge. Such a situation would not, however, equate precisely to ‘dreaming during waking’. A de-differentiated state would be a hybrid one, not dreaming nor waking but an anomalous ‘in-between’ condition. As argued earlier, this ‘inbetween’ state could be expressed in a myriad of different ways and would be a matter of degree.

The paper will now assess how far de-differentiation can explain the positive, disorganized/cognitive, motor and mood aspects of schizophrenia on a phenomenological level, before considering if this de-differentiation hypothesis is compatible with
the neurobiology of schizophrenia. Negative symptoms will be discussed later. These particular dimensional distinctions (positive, disorganized/cognitive, motor, mood and negative) are those suggested in Tandon et al. (2008), cited earlier in this paper.

### 6.1. Positive symptoms

As argued above, internally generated precepts are the very essence of dreaming and the dreamer readily accepts the impossible. Many have noted that with regard to hallucinations (perceptions without external stimulus) and delusions (false, fixed beliefs about reality) there are connections between psychosis and dreaming. In The Interpretation of Dreams Freud quotes from Kant, Krauss, Schopenhauer, Hagen and Wundt to this effect. The idea continues to convince (see for example, Koersko, Snyder, & Feinberg, 1963; Rechtschaffen, Schulsinger, & Mednick, 1964). More recently, Hobson (2004) suggested that the dream is a model for (psycotic) madness. Similarly, Gottesmann (2006) proposed that REM sleep is a useful model for schizophrenia.

Whilst this de-differentiation hypothesis predicts similarities between psychosis and dreaming it departs from the idea of dreaming as a model for psychosis. Rather, this paper will argue that both waking and dreaming are functionally adaptive (see later). Similarities between psychosis (positive symptoms) and dreaming do not imply equivalence between the two states; dreaming is self-organized, whereas psychosis is disordered. As noted earlier, to achieve a dreaming state of mind-brain there must be cholinergic suppression of aminergic neuromodulation, mesolimbic dominance over mesocortical dopaminergic function, sensory gates must be closed, the prefrontal cortex de-modulated and so on. Clearly all of these conditions are not met in psychosis which is a condition that occurs during waking. Although their performance may be defective, patients with psychosis do retain some frontal lobe functionality and are still processing external data. However erroneous their conclusions may be, such patients are trying to make sense of their experience. During dreaming, dreamers do not engage in sense making. Therefore, phenomenologically, the experience of psychosis will differ in from that of dreaming.

For example, although hallucinations characterize both dreaming and psychosis, dreaming is totally hallucinated, whereas, in psychosis, hallucinations only emerge sporadically and do not take up the whole visual field, Collerton, Perry, and McKeith (2005) argue against the case for hallucinations as dream intrusions, in part on the grounds that dreams occupy the whole visual field whereas hallucinations do not. However, during dreaming the sensory gates are closed and the frontal cortex de-modulated whereas in a de-differentiated state there would still be some aminergic input, with the sensory gates only partially closed, sensory input would diminish so there would only be an increase in the possibility for hallucinatory imagery to emerge (see Behrendt, 2003, 2006; Behrendt & Young, 2004). Dreaming is essentially visually hallucinated, whereas auditory hallucinations predominate in psychosis (see, for example, Schiffer, Rao, & Fogel, 2003). Although speculative, this may also be explained through considering psychosis as a hybrid state. As argued earlier, during waking consciousness, the brain-mind systematically externalizes the sense of an external world (Metzinger, 2009). As a result of this process, in waking there is experience of a subjective private ‘inner’ world that is present within an objective public ‘outer’ world. Moreover, during waking, the mind-brain makes a distinction between these worlds, without this distinction, internally generated precepts may be experienced as having external sources (Ford et al., 2007). During dreaming consciousness there is no externalization of the sense of an outer world and, therefore, the distinction between the internal subjective world and the external world is lost. During psychotic waking the externalization process would still be present (at least to some extent) but the distinction between an external ‘public’ world and internal ‘private’ precepts is being eroded due to the partial intrusion of the dreaming state (which conlates the external and internal worlds). Therefore, in a de-differentiated state, when waking has incorporated some (not all) of the characteristics of dreaming it is unsurprising that internal precepts become experienced as external reality. During waking, as argued earlier, the main internal ‘running commentary’ on the external world is not visual but takes the form of an ‘internal conversation’ (Archer, 2003). During this internal conversation, sometimes based on my fears, I advise myself over what to do next. For example, ‘Hurry up, slowcoach, you are getting old, you’re going to miss the train’, sometimes, I equivocate giving rise to multiple internal ‘voices’, ‘Don’t have chocolate, you’ll get fat, you are supposed to be on a diet... Oh, go on, it’s Ok if you only have a small piece’. Due to the externalization process, along with some loss of self-monitoring (owing to the partial de-activation of the frontal cortex), in a de-differentiated state, this internal advice may be experienced as external threats or commands. Based on evidence from functional magnetic resonance imaging, Lawrie et al. (2002) conclude that auditory hallucinations emerge consequent upon the defective monitoring of inner speech processed in temporal areas which are unconstrained by prefrontal inputs. Auditory hallucinations in schizophrenia are either voices conversing amongst themselves or voices commanding, threatening or commenting on the patient (Tandon et al., 2008). In most cases, auditory hallucinations are whole phrases or sentences and are often not in responses to external speech (Hoffman and McGlashan, 1997). Internal advice to oneself is also in sentences and often not in response to the speech of others. Increases in anxiety and fear due to the carry over of such primary emotions from the dreaming state (see mood disorders later) would tend to pitch this ‘internal advice experienced as external commands’ more negatively.

Similarly, although delusions characterize psychosis and dreaming, their nature differs in the two states. Persecutory delusions and delusions of body/mind control are most common in schizophrenia (see, for example, Appelbaum, Robbins, & Roth, 1999). Paranoia and feelings of body/mind control do not pervade dreaming. As argued above, the more or less complete loss of executive functions in dreaming implies that the dreamer does not consider the issue of control. In a de-differentiated state the partial intrusion of some phenomenal aspects of dreaming means that some loss of intentional control may be experienced during waking. Sufficient prefrontal functioning would be retained, however, for awareness of this loss to be registered. If one’s actions are not under the control of the self then it would be a reasonable sense making assumption that they may be under...
the control of another. During dreaming, emotions such as fear and anxiety are tied to the dream plot (Hobson, 2002). If increases in fear and anxiety from the dreaming state start to permeate everyday waking situations (where such emotions are inappropriate and non-explicable in terms of on-going events) then the fear and anxiety appears free-floating and in need of alternative explanation. In a de-differentiated state the presence of free-floating fear and anxiety may arouse suspicion that, in turn, may give rise to an inference of concealed threats thus fuelling a paranoia which would tend to be augmented by any felt experience of loss of control.

Particularly at an early stage, people with schizophrenia often become highly religious (Mohr & Huguelet, 2004). Moreover religiosity can persist; religious delusions are one of the least well understood phenomena in schizophrenia (Brüne, 2009). Again religious delusions are not common in dreaming. As argued above, chaos theory predicts that when a degree of self-organization is lost the consequent state fluctuations are unpredictable. In a de-differentiated state where felt loss of intentional control is not accompanied by increased fear and anxiety, this loss may be interpreted benignly as, for example, well intended guidance from an (unknown) external source. This more positive interpretation could then precipitate not paranoia but religiosity.

In sum, although psychosis and dreaming do have striking parallels there are also significant differences. It is argued that psychosis as a de-differentiated, hybrid state (where only variable aspects of dreaming consciousness are incorporated in waking and to a greater or lesser degree) accounts better for these differences than the thesis that dreaming is a model for understanding psychosis.

6.2. Cognitive/disorganized symptoms

As some self-organization has been lost, a de-differentiated state would, by definition, be a disorganized one. Cognitive/disorganized symptoms include: positive formal thought disorders (e.g. pressure of speech, tangentiality, derailment, incoherence, and illogicality), poverty of the content of speech, alogia, bizarre behaviour, difficulty in abstract thinking, poor attention, unusual thought and poor insight (see, for example, Hardy-Baylé, Sarfati & Passerieux, 2003). As argued earlier, thinking reaches its nadir in REM dreaming (Fosse et al., 2001) so some thought disorder would be anticipated in a de-differentiated state. Dreaming is, of course, a highly associative visual (rather than a spoken) mode where the dreamer often engages in bizarre behaviour. As noted earlier, although dreamers do sometimes speak in dreams, this is relatively infrequent and when attempted, dreamers struggle to vocalize their thoughts (Hobson, 2002). Working memory along with other executive functions (abstract thinking, decision-making, sustained concentration and applied logic) are greatly suppressed in dreaming (Dietrich, 2003). During (non-lucid) dreaming insight is lost (Voss et al., 2009). If waking consciousness has encompassed some aspects of dreaming many of these cognitive/disorganized symptoms could be explained.

Motor output is blocked in dreaming and thinking is greatly suppressed but, in a hybrid state, unusual thought may occur and bizarre urges may be acted out. Whilst bizarre impulses are activated in psychosis, normal routines are neglected (or poorly performed) in schizophrenia. The disorganization syndrome is associated with poor self care (Liddle, 1987) and inappropriate attire (Tandon et al., 2008). Routine, habitual self-care activities are rarely undertaken during dreaming, so in a hybrid state these may be neglected. Difficulty in abstract thinking, poor insight and poor attention (as sustained concentration) would follow from the suppression of executive functions in dreaming. 'Loosening of associations' has been seen as fundamental to schizophrenia since Bleuler (1911/1950). During dreaming, linear-logic is lost to a loosely parallel associative, rapidly changing visual narrative, the details of which are not vocalized. If this non-linear-logic, very loosely associative, rapidly changing mode started to drive waking thought and was vocalized, from the point of view of the audience, there would be pressure of speech with illogicality, tangentiality and derailment. Working memory deficits would exacerbate these difficulties. If the degree of penetration of the dreaming state into waking was even greater then vocalization may become incoherent, there may be poverty of speech content, lack of unprompted speech and, even, ultimately, alogia.

Uhlhaas and Mishara (2007) argue that in the literature on the cognitive/disorganization syndrome, changes in basic perception have been neglected. They report that patients with schizophrenia experience a fragmentation of the normally integrated perceptual field. Moreover, patients focus only on details of a scene to the neglect of the wider background, rendering relatively unimportant features accentuated and hyper-significant. In a hybrid dreaming–waking state the sensory gates would be partially closed with some loss of self-organization and visual integration, so perceptual anomalies would be anticipated. In addition, as argued earlier, the world that appears during dreaming is relatively sparse (compared to the one that materializes in waking) with an enhanced perception of these limited phenomena. So in a de-differentiated waking state some loss of scene background (or context) may be experienced with an unusual accentuated focus on specific details. In waking an appreciation of context is essential to make sense of ‘what’s going on’. So, in a de-differentiated state, perceptual anomalies could only exacerbate sense making difficulties consequent upon the diminution of executive functions.

A cardinal aspect of schizophrenia is lack of insight (Carroll et al., 1999). As argued above in a de-differentiated state there would be a partial de-activation of the frontal lobes, this would result some loss of self-monitoring. Moreover, the reality of what conscious experience is ‘telling one’ is not questioned in either dreaming or waking. So this sense of the authenticity of experience would be likely to be preserved in a de-differentiated state. Thus, in a de-differentiated state, both some loss of self-monitoring and a preserved sense of authenticity would mitigate against insight.
6.3. Motor symptoms

Some inhibition of psychomotor function is present in schizophrenia (see Morrens, Hulstijn, & Sabbe, 2007, for a review). As motor output is blocked during dreaming, de-differentiation could result in some slowing of movement during waking. Morrens, Hulstijn, Lewi, De Hert, and Sabbe (2006) suggest that the repetitive, purposeless motor activity seen in schizophrenia may be the result of impaired executive function due to reduced activity in the dorsolateral prefrontal cortex. As argued above, the frontal lobe is deactivated during REM dreaming, with an associated suppression of executive functions, so the existence of stereotypy would also be compatible with the de-differentiation thesis. Only the oculomotor pathway functions during the eponymous REM dreaming (see, for example, Hobson & McCarley, 1977); these rapid eye movements are not under intentional control. Some carryover of rapid eye movements in a de-differentiated state may account for both pursuit and antisaccade abnormalities. Goldman (2000) reports that 80% of patients with schizophrenia (who are not medicated) exhibit abnormally jerky saccadic eye movements when their eyes pursue a moving target. Over and above this pursuit abnormality, people with schizophrenia also have an antisaccade abnormality (see, for example, Turetsky et al., 2007).

6.4. Mood symptoms, bipolar disorder and autopoiesis

Anxiety is a prominent feature of the early stages of schizophrenia (Chapman, 1966). Social anxiety is highly prevalent in out-patients with schizophrenia, their social anxiety scores did not differ from those of subjects with social anxiety as their primary diagnosis (Pallanti, Quercioli, & Hollander, 2004). This indicates that the social anxiety suffered in schizophrenia does not differ in kind from that in social anxiety syndrome. As argued above, anxiety is the most common emotion in dreaming and as social interaction is attenuated in dreaming, it may be anticipated that in a de-differentiated state social situations would precipitate anxiety.

Depression is common in schizophrenia (Tandon et al., 2008). Depression does not characterize dreaming. However, depression is, of course, integral to bipolar disorder. As noted earlier, although modern psychiatric practice continues to recognize and diagnose two major psychiatric conditions (schizophrenia and bipolar disorder), there is current debate on whether they are indeed distinct (Craddock, O’Donovan & Owen, 2005, 2006; Craddock & Owen, 2005). Disturbances to affective experience and expression are fundamental aspects to schizophrenia, particularly in the early stages (Yung & McGorry, 1996). Altered prefrontal function characterizes not only schizophrenia but also affective disorders (Stoll, Renshaw, Yurgelun-Todd, & Cohen, 2000). The formal thought disorder (including a loosening of associations) that occurs in schizophrenia also characterizes mania (Solovay, Shenton, & Holzman, 1987). Even auditory hallucinations arise in 15% of patients with mood disorders (Hoffman, 2003). Auditory hallucinations occur in between 50–70% of patients diagnosed with schizophrenia (Andreasen & Flbaum, 1991; Sartorious, Shapiro, & Jablonsky, 1974). Keshavan et al. (2008) provide a review on the many areas of neurobiological overlap between affective disorder and schizophrenia.

Some light may be shed on the relationship between schizophrenia and bipolar disorder through considering the concept of autopoiesis. ‘Autopoiesis’ expresses the idea of holistic self-producing systems (Maturana & Varela, 1980; Varela, Maturana, & Uribe, 1974). The mind-brain is such a system, it produces itself as a unitary, bounded entity. Autopoiesis accounts for the holistic ‘feel’ of both waking and dreaming, both are experienced as unified. Self-producing systems are also self-organizing ones, ‘...an autopoietic system has a domain in which it can compensate for perturbations through the realization of its autopoiesis, and in this domain it remains a unity’ (Varela et al., 1974, p. 188). This implies that if ‘perturbed’ the mind-brain (as an autopoietic, self-organizing system) can reconstitute itself—such organizing potential is inherent in the production of a coherent whole from constituent parts. As Goodwin (1994, p. 169) puts it, ‘...complex non-linear dynamic systems... tend to settle dynamically at the edge of chaos. If it [the system] moves too far into the chaotic regime it will come out again of its own accord; and if it strays too far into the ordered regime it will tend to “melt” back into dynamic fluidity where there is a rich but labile order, one that is inherently unstable and open to change’. If, as argued earlier, order in the mind-brain is represented by an achieved differentiation between waking and dreaming and ‘chaos’ or disorder (schizophrenia) results from their de-differentiation, then bipolar disorder may equate to autopoietic attempts to restore self-organization (Llewellyn, 2009).

Self-organizing occurs though the brain’s modulatory systems (Kahan, LaBerge, Levitan, & Zimbardo, 1997; Singer, 1986) so any autopoietic response would also be expected to occur through neuromodulation. As argued earlier, de-differentiation would result in cholinergic and mesolimbic dopaminergic modulation rising (and aminergic and mesocortical modulation falling) during waking. During dreaming, de-differentiation would cause aminergic and mesocortical dopaminergic modulation to rise (and cholinergic and mesolimbic modulation to fall). If an autopoietic response to correct these ‘imbalances’ occurred, mania/hypomania may flow from self-organizing to produce a ‘purer’ waking state (though aminergic hyperactivity and cholinergic hypoactivity). In contrast, depression may result from autopoietic attempts to induce a ‘purer’ dreaming state (through cholinergic hyperactivity and aminergic hypoactivity). If psychosis predominated (with the waking state more affected) then it would be predicted that bipolar disorder would begin with a manic episode. Alternatively, if the dreaming state were more affected, the first episode would be more likely to be a depressive one. Or both aminergic and cholinergic hyperactivity could alternate (resulting in rapid cycling bipolar ‘disorder’). Evidence is suggestive of this possibility.

Classic cholinergic–adrenergic imbalance theories of mania and depression have been proposed (Janowsky, El-Youssef, Davis, Hubbard et al., 1972; Janowsky, El-Youssef, Davis, & Sekerke, 1972; Janowsky, Risch, & Gillin, 1983). Over-activity of catecholamine neurotransmission may explain mania/hypomania, whereas depletion of amines may account for depression.
Cholinergic reversal of manic symptoms has been demonstrated (Janowsky, El-Yousef, Davis, Hubbard et al., 1972; Janowsky, El-Yousef, Davis, & Sekerke, 1972). People with depression exhibit shorter REM latency and heightened REM density, which may be interpreted as hyper-cholinergic activity (Riemann et al., 1994). Mahmood and Silverstone (2001) found that central serotonergic activity is reduced in depression and reported similar findings in euthymia but failed to consistently substantiate an increase in mania. Selective serotonin reuptake inhibitors can precipitate mania (Breggin, 2004; Peet, 1994).

Once established, schizophrenia tends to be chronic but characterized by remissions, albeit that these are incomplete (Tandon et al., 2008). Such a trajectory is, at least, suggestive of some autoepoietic response. If schizophrenia and bipolar disorder are mutually implicated, so that the latter can be an attempt to restore order to the former, then this may partly explain the uncertain trajectory of schizophrenia. It has been noted that schizophrenia is potentially reversible in the prodrome stage (Lee, McGlashan, & Woods, 2005). If an autoepoietic response to incipient schizophrenia started early and was successful, then the de-differentiation process may be arrested. On the other hand, a more severe form of schizophrenia may induce more dramatic manic and depressive symptoms—precipitating classic bipolar disorder. Observations from clinical practice note a sometime temporal progression from classic bipolar disorder to schizophrenia, with the reverse being rare (Walsh, 2009). This would be the case if bipolar disorder was an autoepoietic response but, in some cases, ultimately failed.

This hypothesis that bipolar ‘disorder’ may be an autoepoietic attempt to restore order to schizophrenia does not exclude the possibility that bipolar disorder could also be expressed as an independent condition of anomalous neuro-dynamics.

7. How does ‘de-differentiation’ compare with current thinking on the neurobiology of schizophrenia?

Clearly it is not possible here to review all possible relevant research findings on schizophrenia but some comparisons are made with classic and continuing areas of work. In psychiatry, thinking on schizophrenia has been dominated by the ‘dopamine hypothesis’. The early hyperdopaminergic theory of schizophrenia was superseded by one positing (implicitly, in the waking state) hyper-mesolimbic dopamine and hypo-cortical dopamine (Carlsson, 1988; Davis et al., 1991; Weinberger, 1987; Weinberger, Aloia, Goldberg, & Berman, 1994). Howes and Kapur (2009) have recently argued, however, that the picture on schizophrenia is more complex than this mesolimbic–mesocortical DA imbalance hypothesis implies. They state that while DA imbalance may account well for psychosis, it does not explain the totality of schizophrenia, reasoning that multiple transmitter systems are likely to be involved in negative symptomatology and cognitive deficits. Such arguments reflect those of others. Multiple ‘imbalance’ hypotheses have been suggested. Schizophrenia (as observed in the waking state) is characterized by serotonergic and noradrenergic demodulation and cholinergic hypermodulation (Bymaster, McKinzie, & Felder, 2004; Gottesmann, 2006; Sarter et al., 2005). Sarter and Bruno (1999) have argued that both hyper- and hypo-cholinergic activity characterizes a broad range of neuropsychiatric conditions including schizophrenia. Noradrenergic instability has been suggested in schizophrenia (Van Kammen & Gelernter, 1987). Cholinergic–dopaminergic interactions have been implicated in the expression of positive and negative symptoms (Tandon et al., 1999). Dysfunctions in both serotonergic and dopaminergic systems have been proposed, with the serotonergic mediating negative symptomatology and dopaminergic precipitating the positive symptoms (Bleich, Brown, Kahn, & van Praag, 1988). Tandon et al. (1999) suggest that altered balance between DA and one or more other transmitter systems are likely to be involved in schizophrenic symptoms. In a more recent review, Keshavan et al. (2008) argue for the implication of dopamine, glutamate, GABA, acetylcholine and serotonin (although they conclude that the evidence for 5-HT involvement is indirect).

Second, there has also been a substantial body of research exploring frontal lobe dysfunction (see, for example, Berman, Zec, & Weinberger, 1986; Buchsbaum, 1990; Weinberger, Berman, & Zec, 1986). Weinberger et al., 1994 point out that this work can be traced back to Kraepelin; they argue that, although there is evidence of prefrontal dopaminergic deafferentation, this is likely to be secondary to a failure of prefrontal activation. Functional brain imaging of patients with schizophrenia confirms the lack of activation of the dorsolateral prefrontal cortex when confronted with cognitive tasks (Berman & Meyer-Lindenberg, 2004). Such failure results in impairment on tests of ‘executive’ functions involving abstract thought and working memory. Frontal lobe dysfunction has not been clearly linked to any structural pathology, however (Bilder & Goldberg, 1987; Goldberg & Bilder, 1987).

Disturbed information processing and attentional dysfunctions constitute a third major area of work with a long history (Braff, 1993). Sensory gating deficits are well documented in schizophrenia (Adler et al., 1982; Hirano et al., 2010; Judd, McAdams, Budnick, & Braff, 1992; Potter, Summerfelt, Gold, & Buchanan, 2006; Venables, 1964). These gating failures result in people with schizophrenia being unable to filter out irrelevant data, all sensory signals tend to be perceived as equally significant hence, sensory overload is the consequence. Judd et al. (1992) suggest that there is a tendency for gating deficits to be more prominent in frontal areas. Specificity on attentional and gating deficits has been gained through studies on event related potentials. Mismatch negativity (MMN) represents the pre-attentive phase of auditory information processing; MMN is disturbed in schizophrenia, a finding that has been consistently replicated (Umbricht & Krijés, 2005). The P300 waveform has been seen as the neuro-correlate of a working memory update on environmental changes. Blunted amplitude of the auditory P300 response to salient stimuli is present in schizophrenia (Bramon, Rabe-Hesketh, Sham, Murray, & Frangou, 2004; Jeon & Polich, 2003). Although the data is not as consistent as for P300 (possibly due to methodological differences), P50 suppression (the normal decrease in P50 amplitude when the second of two paired auditory stimuli is presented) is disrupted in schizophrenia (Bramon et al., 2004; de Wilde, Bour, Dingemans, Koelman, & Linszen, 2007; Patterson et al.,
Pre-pulse inhibition (which is implicated in sensory gating) is reduced in schizophrenia (Braff & Light, 2005; Geyer, Krebs-Thomson, Braff, & Swerdlow, 2001).

Work on frontal lobe dysfunction and research into disturbed information processing/sensory gating deficits both link into the disconnectivity hypothesis of schizophrenia. Friston (2002, p. 66) defines this, ‘Schizophrenia can be understood in cognitive terms, and in terms of pathophysiology as a failure of functional integration within the brain.’ Friston and Frith (1995) proposed that there may be disruption of fronto-temporal interactions; they also note work which has highlighted some abnormal integration of prefrontal activity with that in subcortical, limbic and temporal structures. Lawrie et al. (2002) implicate reduced frontotemporal connectivity with auditory hallucinations in schizophrenia. Keshavan et al. (2008) suggest that P50 gating deficits in schizophrenia may reflect frontal–hippocampal miscommunication.

Clearly there are some convergences between this de-differentiation thesis and neurobiological findings in schizophrenia. Both ‘de-differentiation’ and current thinking on schizophrenia implicate not only mesolimbic–mesocortical DA imbalance but altered balance between other transmitter systems. Differentiation implies both hyper-cholinergic input into waking and hypo-cholinergic into dreaming, both are thought to characterize schizophrenia. The de-differentiation of waking and dreaming consciousness would disrupt reciprocity between dopaminergic/serotonergic/noradrenergic/cholinergic systems. Current thinking on schizophrenia is moving towards implicating multiple and interacting imbalances between modulatory systems (Carlsson, Waters, Waters, & Carlsson, 2000; du Bois et al., 2005).

Input–output gating dysfunctions would characterize de-differentiation in a similar manner to those seen in schizophrenia. De-differentiation would result in diminution of sensory input during waking (due to sensory gates being ‘partially closed’). This may intensify the hallucinatory positive symptoms induced by heightened mesolimbic DA. De-differentiation would result in fewer sensory signals being registered; those that are processed may induce the ‘sensory overload’ seen in schizophrenia-as the dreaming state is non-adaptive for the processing of external sensory information. Moreover, as dream scenes are sparse compared to waking ones, it appears that there is less of a requirement to filter information (compared to waking) so, in a de-differentiated state, filtering problems may emerge.

As argued earlier, during dreaming the dorsolateral prefrontal cortex and the primary visual cortex are deactivated, whereas limbic areas and the occipitotemporal cortical areas are selectively activated. De-differentiation would be expected to reduce frontal lobe activation and impair frontal lobe functionality during waking. As the regional activation of the brain varies between waking and dreaming, resulting in two different functionally integrated states, it would be anticipated that de-differentiation would engender a loss of functional integration in both cognitive and physiological terms in a similar way to that outlined in the disconnection hypothesis.

8. Negative symptoms and sleep disturbances

The above sections have outlined the possible implications of the de-differentiation of waking and dreaming consciousness for the functionality of the waking state. The discussion has also considered the compatibility of the de-differentiation thesis with the neurobiology of schizophrenia but, implicitly this has also been in terms of impact in the waking state. But if the de-differentiation idea has any validity then effects on dreaming consciousness and sleep would also be anticipated. De-differentiation involves some loss of dynamic reciprocity between neuromodulatory systems. This loss is not absolute, however, but a matter of degree. So, for example, the retention of some dynamic reciprocity between neuromodulatory systems would imply that if aminergic and mesocortical dopaminergic input decline during waking, they would rise in REM dreaming and also during other sleep stages. An increase in aminergic and/or mesocortical dopaminergic modulation during sleep (and dreaming) would be expected to disrupt sleep. As noted earlier, schizophrenia and bipolar disorder are both characterized by sleep disturbances (Benson, 2006; Costa e Silva, 2006). Ferrarelli et al. (2007) report reduced spindle density in schizophrenia. In a review Monti and Monti (2005) conclude that sleep-onset and maintenance insomnia is a fundamental feature of schizophrenia, regarding of medication status or whether the clinical phase is chronic or acute. They also remark that most studies show that stage 4 sleep is reduced, as is REM latency, but REM sleep duration remains unchanged. Moreover, it would be expected that the partial projection of waking into dreaming would partly disinhibit motor function during sleep. Restless legs syndrome in sleep has been found to be higher in patients with schizophrenia than normals (Kang et al., 2007).

Studies on whether dreaming consciousness (as opposed to REM sleep) is disrupted in schizophrenia face clear difficulties. First, in neuroscience there is profound split between those ‘...holding that the dream is a mere random byproduct of REM sleep physiology, and [those holding] that the dream is an organized subjective experience that performs a specific function.’ (Ardito, 2003). If the conscious experience of dreaming serves no function, then disruption has no consequence. Second, the idea (discussed earlier) that dreaming is a model for psychosis (or schizophrenia) has tended to mask the possible functions of dreaming. Third, experimental approaches to studying the functionality of dreaming are hard to design and execute. If dreaming does have a function, could the loss of this functionality be linked to the negative symptoms of schizophrenia?

Negative symptomology (social withdrawal, diminished affect, loss of motivation and apathy) is generally accepted to be more debilitating (Andreasen, 1990) and less curable, being resistant to pharmacological approaches (Erhart, Marder, & Carpenter, 2006; Samuelian, 1996). Indeed, it has been argued that the anti-psychotic medications currently used to treat positive symptoms may induce negative symptomatology, engendering a loss of interest in the world (Breggin, 1980; Charlton,
2006; Panksepp, 1985). This would be expected under the de-differentiation thesis as blocking mesolimbic dopamine may result in a rise of mesocortical DA, due to some retained reciprocity between these modulatory systems (Davis et al., 1991; Guillin et al., 2007; Pycock et al., 1980). This would improve both positive and disorganized symptoms during waking but may exacerbate negative symptoms due to the impact of less mesolimbic and more mesocortical DA during dreaming, as described in 8.1 below. Thus the efficacy of anti-psychotic medication would depend upon the balance of positive and negative symptoms, which would, in turn, be contingent on the relative impact of de-differentiation on the waking and dreaming states. Specifically, if de-differentiation affects the waking state more than the dreaming one then anti-psychotics would be of more benefit. In contrast, if the dreaming state is more negatively affected, then anti-psychotics would be less efficacious, indeed, as described above, they may exacerbate the negative syndrome.

The intrusion of some aspects of dreaming into waking (as discussed above) may have implications for negative symptoms, for example, motion slowing may be linked to apathy and/or loss of intentional control and inability to make sense of waking experience may lead to a loss of motivation. But these possibilities seem insufficient to account for the devastating impact of negative symptoms. Would the intrusion of some physiological and phenomenological of waking into dreaming cause problems? Admittedly, this is highly speculative but this paper will go onto explore the idea that dreaming consciousness may be functional for emotionally salient memories. Indeed, that a dream may be a mnemonic device for such memories. If this is the case, then the intrusion of aspects of waking into dreaming may result in pathologies, indeed, specifically, may have implications for negative symptomatology.

8.1. Is dreaming functionally adapted to support imaginative association and memory?

Hartley (1749) proposed that perceptions of an integrated, meaningful world emerge from ‘joint impression’ or association. He also believed that personally meaningful psychological associations create a sense of self. He held that this fundamental principle of association works at the level of the brain and the mind (Buckingham & Finger, 1997). Dreaming is an activated, conscious, cyclically periodic brain/mind state; this suggests that dreaming has some function. Indeed dreaming consciousness may be complementary to waking (Hobson, 2009). Association may be the source for self-organizing and integration (a unified experience from a single perspective) in both waking and dreaming consciousness. The basis for association in the two states may be different, however. Freud contended that dreams reflect ‘free’ or loose association. This idea has proved a robust one. Stickgold (2003) argues that, during dreaming, the brain ignores predictable associations, instead novel connections are sought out and strengthened as appropriate. Strengthening associations, particularly novel ones, is integral to long term potentiation and the enhancement of memory (see, for example, Lynch, 2004; Malenka & Bear, 2004).

Throughout Classical Antiquity and into the Early Middle Ages, memory was recognized to be a creative process where material to be remembered (in current terms, ‘encoded’ and ‘stored’ in long term memory) was made memorable through imaginative, personal association. In this ancient art of memory (AAOM) personally significant events/people/emotions were interlinked in ways that did not reflect how these events/people/emotions had actually been related in day-to-day experience but how they were meaningfully, emotionally associated from a personal point of view (Bolzoni, 2001; Carruthers, 2008; Yates, 1966). The aim of AAOM (incorporating the method of loci) was to produce memory cues or ‘…chains of association that regenerate memories.’ (Bolzini, 2001:xviii). The more bizarre, dramatic and absurd is the cue, the more resilient is the memory to which the cue has been associated (Hughes, 1997). As compared to replay or rehearsal, AAOM can still produce extraordinary memory ‘feats’ during waking (Wilding & Valentine, 1997). But with sensory gates closed, and with around 2 h each day devoted to the task, REM dreaming consciousness may self-organize specifically to support associative mnemonic functioning and, hence, be much superior to waking in this respect. Some indicative evidence of this is provided by Sprenger et al. (2010) who demonstrated that REMs, when imagining vivid scenes in dreaming, were similar to eye movements with open eyes when imagining remembered scenes during waking.

Memory is not unitary but multi-faceted. Classic work in psychology recognizes six types of memory. Memory is traditionally divided into declarative (explicit) and non-declarative (implicit) categories, declarative memory can be further divided into episodic and semantic, non-declarative is sub-divided into procedural/skill, conditioning, non-associative and priming, such a division gives six categories of memory (although sometimes autobiographical memory is seen as a subset of episodic, on some schema it is considered an independent category). Sleep is implicated in all of these six types of memory and there is no known memory category for which sleep does not provide benefit (Stickgold, 2009). Stickgold (2009) calculates that with six different kinds of memory, five different stages of sleep and six stages of post-encoding memory processing, there are 144 distinct sub-questions involved! This paper, however, is not directly concerned with all of the complex questions that the relationship between sleep and memory raises.

Rather this section of the paper is focused on this question: ‘If dreams are functional for memory, what kind of memory might dreams enable?’ Clearly we need to remember what is important to us – concerns. Concerns include some episodic and autobiographical memories but are not limited to them. Concerns also encompass projects – things to accomplish in the future (from writing a paper on dreaming consciousness to meeting a friend for coffee next week). In relation to our own concerns and projects, we need to remember our interactions and conversations with significant others, as these may converge with or cut across our plans. In relation to our own thoughts, intentions and goals, memory is not only about the past, ‘…memory pulls us forward…’ (Robinson, 2005, p. 192) as memory helps us decide what to do next. Concerns are emotionally salient. We cogitate on our concerns. So, we need to remember waking thoughts about concerns. Concerns drive personal identity. Archer (2000, p. 10) argues that personal identities are formed by the way that concerns are monitored and
prioritized. So if projects, emotional responses to concerns, waking thoughts about concerns and, even, the nature of concerns are forgotten, personal identity is eroded. In terms of this paper, there is no exact fit between the classic memory definitions in psychology and the type of memory that may be enhanced in dreaming consciousness. ‘Episodic’ memory is the closest to ‘emotional concern memory’ but, as discussed above, the latter is much wider than the former.

8.1.1. A possible example of bizarre, creative dream association and memory enhancement

Nielsen and Stenstrom (2005) suggest that the links between dreaming and memory will only be elucidated when subjects are able to identify the non-obvious source memories for their dreams; they also propose that self-identification of these sources, in the mode of Freud, William James and others, may yet prove to be the most productive method. In this spirit, I narrate the following dream fragment as an example of bizarre, absurd and creative dream association.

I dream I am having my eyes tested and instead of the normal chart the ophthalmologist uses ‘Bagpuss’. In my dream, the usual ‘chart’ letters are apparent under Bagpuss’s fur, I fail the test because I can’t see them.

‘Bagpuss’ is an old, fat, saggy cloth cat—a 1970’s UK children’s TV character. I often get my eyes tested because I am very short-sighted. The Bagpuss dream occurred shortly after a recent test for glaucoma. Instead of my normal optometrist I was referred to the hospital I used to be taken to as a child. I was most certainly worried, even afraid. What did the occasion mean to me? Well, I was very conscious of the hospital visits I made as a child when I feared that the stronger and stronger spectacles I was being prescribed indicated that I was going blind. Now many years later I was returning to this same hospital for a test for glaucoma— which can cause blindness and is quite common in the elderly. So, for me, the visit had associations of both being young and growing old. Bagpuss is old, not to mention fat and saggy! And Bagpuss is for the young— he is a children’s TV character. But this does not seem enough, does it? Why associate Bagpuss with a test for glaucoma? Glaucoma starts with a loss of peripheral vision. Bagpuss is always in scenes which shade out into hazy, lost, blankness (see ‘Bagpuss’ on youtube). Predictably, I always disliked Bagpuss without ever analyzing the reason why. For me, in my dreams, Bagpuss is associated with being young, growing old and fear of blindness through glaucoma.

If, in waking consciousness, I was asked for a personally meaningful, strikingly bizarre, visual image that was associated with glaucoma, being young and growing old. I think it highly unlikely I would be able to come up with anything. But, in my dreams, I easily make an imaginative, associative ‘leap’ across 35 years to connect blindness with a saggy, old cloth cat. Dreaming may differ from waking consciousness primarily because it forges more imaginative, more bizarre and less time/space bound associations. Such associations may act to enhance memory. Lake (2008) has demonstrated that loose association or ‘flight of ideas’ in schizophrenia becomes explicable if the patient is able to narrate the personal associations that link the ideas. Similarly here, the association between Bagpuss and ophthalmology in my dream is understandable in the light of my past history. This suggests that ‘flight of ideas’ in schizophrenia may emerge consequent upon the intrusion of loose associations from the dreaming state.

8.1.2. Other phenomena that both enhance memory and characterize dreaming

Along with association, dreaming has several characteristics that have been shown to aid memory. First, dreaming creates sequential visual imagery; visualization improves recall (Bower, 1970). Pictorial representation, particularly when the images are interacting, also enhances memory (Richardson, 1980). Second, dreams are frequently more dramatic and emotionally driven than everyday waking experience. Unusual, dramatic and emotionally salient events are more memorable (Dutta & Kanungo, 1975; Hunt, 2008; Rubin & Friendly, 1986; Rusting, 1998). This memory effect is exemplified and extended in ‘flashbulb memory’; as the name suggests, what is surprising about this phenomenon is not that dramatic events are easily recalled but that the personal circumstances surrounding unusual occurrences are also vividly remembered (Brown & Kulik, 1977). This may suggest that, in dreams, introducing emotionally charged, bizarre and dramatic events improves recall of this content but also aids memories for the more ordinary, but connected, personal context. Third, dreams portray organized scenes (for example, the sky is always above the characters, the ground always below). And these scenes form a narrative. Organization and narration support memory (Mandler, 1984). Finally, in my dreams, I am embodied and feel I am living out the events. Embodied memories are the most resilient (Merleau-Ponty, 2002).

8.2. Evidence on the relationship between dreaming and memory

This evidence will be addressed through discussing work on how and why dreams embed memories and reviewing neurobiological data indicating that mnemonic processes function during dreaming.

Freud (1999) thought that all dream material relates in some way to remembered experience, this he asserted as undisputed knowledge. Similarly, Stickgold (2002) argues that with sensory signals excluded, memories along with derived symbols and meanings are the only possible sources for dreams. Hall and Van de Castle (1966) found extensive support for Freud’s proposal that recent events are the basis for dream plot elaboration. The new associative links forged during dreams may be vital for understanding the meaning of events as they unfold (Stickgold, 2002). Dreams do not replay whole episodic memories but around 65% of content can be clearly traced to memory fragments of waking experiences (Fosse, Fosse, Hobson, & Stickgold, 2003). The presence of new and bizarre associations may account for the 35% of dream content that is not readily traceable to waking experiences. For example, at first I am unlikely to understand the connection between a saggy old cloth cat and my visit to the ophthalmologist but on reflection I may see the connection. If dream content is
entirely composed of meaningfully associated memory fragments, everything in dreams is relevant to the dreamer. During waking emotional salience (or relevance) has to be discerned from a myriad of sensory signals, it may be that during dreaming consciousness emotional salience can be assumed.

During memory ‘encoding’ the hippocampus enables the creation of novel meaningful associations (Achim & Lepage, 2005; Davachi & Wagner, 2002; de Vogelaere, Santens, Achten, Boon, & Vingerhoets, 2010; Henke et al., 1999). Activity in the hippocampus is increased during REM dreaming compared to both waking and NREM (Nielsen & Stenstrom, 2005). Recent memories may be stored in the hippocampus temporarily and then reactivated during dreams (Hobson, 2002). In dreams, access to remote memories is also enhanced (Hobson, 2002). Evidence is consistent with the processing of emotionally charged memory traces during REM sleep, limbic areas, especially the amygdala, are activated (Revonsuo, 2003). Wagner, Gais, and Born (2001) demonstrated that recall of emotional charged (but not neutral) text was significantly enhanced following REM enriched late night sleep. Similarly, Rauchs et al. (2004) reported improvement for episodic declarative memories after REM enriched late night sleep. Nielsen and Stenstrom (2005) suggest that, during dreaming, memories may be relocated from the hippocampus to be ‘stored’ in the associative neocortex. In sum, this physiological evidence is, at least, suggestive of the dream as a hyper-associative mnemonic narrative that embeds memory ‘fragments’ to form chains of association that can regenerate whole memories.

However, it may be objected that dreams are forgotten, so how can they have a mnemonic function? Dreams are not generally recalled into waking consciousness- this does not prove that they are forgotten. It has been suggested that 95% of cognition is unconscious (Lakoff & Johnson, 1999; Thift, 2008). Joordens, Wilson, Spalek, and Paré (2010) state there is now general agreement that memories have both conscious and unconscious influences; moreover, they produce experimental data indicating that conscious and unconscious processes operate independently. So there would be nothing surprising about dreams functioning mnemonically at an unconscious level. The Penfield experiments lend intuitive support to this idea; stimulation of the exposed cortex of patients elicited reports of dream-like memories. Indeed, if dreams embed personally meaningful associative cues that are not easily understood in waking consciousness it is clear that the cues must kept out of the waking mind. To return again to the Bagpuss example, if whenever I go to the ophthalmologist I consciously associate this with Bagpuss without knowing the reason why I am making the association I will be confused. If psychosis is ‘dreaming hallucination partially projected into waking’ then in a psychotic state I may hallucinate Bagpuss when visiting the ophthalmologist. Behrendt (2005) comments that ‘...what we see in a hallucination or dream is usually expected unconsciously.’ This would be the case if waking hallucinations, like dreams, are constituted from memory fragments associated in ways that resonate at an unconscious level but are not readily understood in waking consciousness.

The ‘continuity hypothesis’ suggests that dreams portray concerns and experiences from waking life (Domhoff, 1996; Hartmann, 1998; Schredl, 2003). Revonsuo (2003) argues there is overwhelming evidence that dreams incorporate waking concerns. But such suggestions have to be supplemented with clarification of why -in dreams- our concerns and experiences appear (from the perspective of waking consciousness) in such a fragmented and, sometimes, bizarre and dramatic way. The idea of the dream as a mnemonic associative narrative that conflates memory fragments could explain this. Payne, Stickgold, Swanberg, and Kensinger (2008) have demonstrated that sleep preferentially enhances memory for the emotional (but not the neutral) components of waking scenes. Only the emotional essence (or essential ‘fragment’) of personally significant waking experiences may feature in dreams. Also dreaming experiences always differ from the ‘same’ waking experiences because they always form part of a chain of association. For example, Bagpuss was not present at my recent visit to the ophthalmologist nor was there an ophthalmologist around in my remote memory of seeing Bagpuss on TV. Bagpuss and ophthalmology are not related in any logical, linear, cause and effect way. But they are related in an emotionally salient way. The Bagpuss’ dream conflates the two memory fragments because it expresses what the visit to the ophthalmologist means to me- glaucoma and fear.

Foster (2009, p. 30) states that ‘...information in long term memory is thought to be stored primarily in terms of the meaning of the information’, moreover, Solms and Turnbull (2002, pp. 173 and 174) suggest that long term memories are stored, organized and connected in very different ways to the ‘rational’ memory forms that are retrieved into waking consciousness. This paper suggests that dreams are both the media for associating recent and remote memories and the way that long term memories are ‘stored’ and organized – as chains of emotionally associated memory ‘fragments’. Crick and Mitchison (1983) point out that the richly interconnected assemblies of the neocortex could serve to store associations; however, they postulate that such an associative net may become overloaded and produce bizarre associations. Their hypothesis is that dreams rid the net of such incongruities. But AAOM shows that it is precisely those bizarre associations that make memories more memorable. The more absurd and striking the cue – the more resilient the memory to which the cue is associated. Bizarre associations have high mnemonic functionality. Hebbian principles (Hebb, 1949) dictate, first, that a memory ‘fragment’ can elicit a more complete memory and, second, a memory with a particular element can invoke other memories with that element. This is possible through chains of association. Korsakoff proposed that the accessibility of a memory is dependent not only on the strength of the trace but also on the number of associations made (Banks, 1996). Habitual actions (for example, visits to an ophthalmologist) are particularly resilient because script memories are available (Mandler, 1984). In the Bagpuss dream, there is little detail of the routine events that constitute eye-testing. However, this fragment could trigger the appropriate associated script memory (drops, dilation, examination and discussion). The presence of Bagpuss ‘tells me’ what kind of variant occurred on the routine eye examination – a test for glaucoma.

Such suggestions are congruent with the reconstruction of memories on retrieval (Bartlett, 1932; Neisser, 1962; Schachtel, 1947). Declarative memories are also dynamic (Paller & Voss, 2004) and labile, not only before encoding, but also upon
retrieval (Maquet, 2001; Nader, 2003). Lability is necessary, first, for the meaningful 'updating' of memories as new related experiences are assimilated and, second, as remote memories influence the meaning and significance attributed to recent experiences. Stickgold (2009) propose that memories evolve over time but 'evolution' may be a slightly misleading metaphor. For example, as a child my memory of visits to the ophthalmologist were characterized by fear, followed by a long period of time when they evoked only mild anxiety, now fear has returned as the possibility of glaucoma looms. Whilst it may be said that my memory 'evolved', this evolution was not driven by random events but personal emotional salience.

If dreaming is particularly functional for 'emotional concern memory' and, ultimately, personal identity then the partial intrusion of aspects of the dreaming state into waking along with the partial intrusion of aspects of the waking state into dreaming will start to erode memory, concerns and over time, personal identity will diminish. For example, as argued earlier, if dreaming incorporates memory fragments, when dreaming is partially projected into waking, some personal memory fragments (which function as bizarre memory cues in dreams) will start to appear during waking experience. At first, these may only present as puzzling intrusive thought associations but, if psychosis emerges, they may develop into visual or auditory hallucinations (e.g. a Bagpuss hallucination during a visit to the ophthalmologist). Badcock, Waters, Maybery, and Michie (2005) found that, in schizophrenia, the presence of auditory hallucinations was associated with an inability to suppress memories that were not relevant to the on-going situation. To an external observer, Bagpuss seems totally irrelevant to a psychotic patient, associative memory cues will be forged to what are already associative memory cues (e.g. Bagpuss). Thus a false memory (Bagpuss actually was heard or seen at the ophthalmologist) will be formed and strengthened. This process would allow a hallucination to become delusion (a fixed false belief). If the association–hallucination–delusion process occurred in connection with a fairly commonplace event in the life of the patient then a particular delusion may come to dominate consciousness.

Moreover, as argued earlier, the projection of aspects of dreaming into waking will inter alia impair working memory, impedes abilities to filter out irrelevant non-emotionally salient data and, hence, diminish focus on new truly emotionally salient material. So memories will be formed of non-salient data and, over time, memories of emotionally salient concerns will diminish. Although it is possible to form associative emotionally charged memory cues during waking, this paper has argued that dreaming is functionally adapted to this task and, therefore, performs at a much superior level. Hence, the projection of aspects of waking consciousness into dreaming will curtail the ability to form chains of imaginative, personally associative memory cues. In consequence, over time memories for concerns (even delusional ones) will fade and personal identity will be eroded. The loss of memory for concerns and the erosion of personal identity may be expressed in the clinically manifest negative symptoms of de-motivation, apathy and social withdrawal.

Solms and Turnbull (2002, p. 312) observed that patients who do not dream become 'as spontaneous, inert and apathetic'. This is predictable if dreams forge the associative cues that are necessary to remember concerns/projects and, thus, maintain the coherent identity that enables (in waking) the imagining and planning of a future. Patients with schizophrenia would dream but, over time, the partial projection of aspects of waking physiology and cognitive into dreaming would result in dreams losing mnemonic functionality. People with pronounced negative schizophrenic symptomology would, therefore, come to resemble those who do not dream.

9. Is this congruent with findings on memory impairments in schizophrenia?

In sum, the above discussion presents indicative evidence that phenomena are selected for memory formation during waking consciousness but are strengthened, organized and re-organized during dreaming consciousness, through being associated to personal emotionally salient cues. The de-differentiation of waking and dreaming consciousness would, therefore, have a negative impact on memory selection, formation, strengthening/enhancement ('consolidation'), organization and reorganization.

There is a history of experimental research into memory impairment in schizophrenia that is consistent with this thesis, in the sense that it shows generalized dysfunction across a wide range of memory tasks (see, for example, Aleman, Hjiman, de Haan, & Kahn, 1999; Calev, 1984; Clare, McKenna, Mortimer, & Baddeley, 1993; Gold, Randolph, Carpenter, Goldberg, & Weinberger, 1992; Goldberg, Weinberger, Plokin, Berman, & Podd, 1989; Manoach & Stickgold, 2009; McKay et al., 1996; Stickgold, 2005; Tamlyn et al., 1992). Experimental testing during schizophrenia for loss of emotional concern memories due to disrupted dream-dependent consolidation processes is clearly fraught with difficulty. There has been interest, however, in the specific relationships between emotionally charged memories, personal identity and schizophrenia.

To be functional, memory has to be selective. Concern memories are formed of emotionally salient events/people/places. Dopamine mediates the emotional salience of both external events and internal representations (Kapur, 2003). Linked to dopamine dysfunction, during waking, schizophrenia is characterized by aberrant salience; false significance is attributed to irrelevant phenomena, whilst, at the same time, there is adaptive failure to discern truly emotionally salient events (Kapur, Mizrahi, & Li, 2005; Roiser et al., 2009). Such dysfunction may lead to memories being fashioned from insignificant stimuli, whilst memories of salient events fail to be formed. Moreover, in support of the iterative scenario outlined above, Roiser et al. (2009) found that aberrant salience correlated with both delusions and negative symptoms in schizophrenia. As mentioned
earlier, Badcock et al. (2005) report that auditory hallucinations in schizophrenia are related to the intrusion of activated memory representations. Thus aberrant salient in schizophrenia is explicable through the de-differentiation idea.

Conscious recollection of episodic memories is significantly impaired in schizophrenia and it is argued that this dysfunction results from encoding failures (Danion, Huron, Vidailhet, & Berna, 2007; Gold et al., 2000; Neumann, Blairy, Lecompte, & Philippot 2007). Achim and Lepage (2005) argue that people with schizophrenia may use less associative encoding strategies. This would be congruent with the disruption of personal associative memory processes, if waking is partially projected into dreaming, consequent upon de-differentiation. Conscious recollection involves mentally re-living events, such reflection is vital for rethinking the past and projecting into the future. If the thread that connects the present self with the past is broken, a meaningful life narrative cannot be sustained. Without a meaningful life narrative, the future cannot be envisaged and enacted. In schizophrenia the loss of conscious recollection of past experiences leads to an erosion of the sense of the present self being continuous with the past self (Danion et al., 2007). People with schizophrenia have difficulty in re-living the past and find it even harder to imagine the future (D’Argembeau, Raffard & Van der Linden, 2008). They also have difficulty in discerning the meaning in what should be personal self-defining memories (Raffard et al., 2009). To argue that personal identity is eroded (Blairy et al., 2008; Riutort, Cuervo, Danion, Peretti, & Salamé, 2003) hardly captures the devastation, unlike having an illness, schizophrenia is something a person can become (Estroff, 1989). Although ‘person with schizophrenia’ is now used rather than ‘schizophrenic’ as this language is thought to be less alienating and stigmatizing, there may be an important sense in which this understanding does not adequately reflect the way in which the condition transforms the self (Sass, 2007).

10. If waking and dreaming consciousness became de-differentiated, would schizophrenia result? Concluding comments

If waking and dreaming consciousness became de-differentiated, would schizophrenia result? Based on the arguments of this paper, the answer is: ‘possibly’. Whether schizophrenia emerged would depend upon the degree of de-differentiation which, in turn, would be contingent upon the extent of membrane defects and abnormalities in phospholipid metabolism. The extent and nature of phospholipid aberration would drive the extent and nature of neurotransmitter imbalance and the consequent phenomenological expression of psychiatric symptoms. Susceptibility to these symptoms would depend upon gene-environment interplay (including diet and stress) which predict the impact of phospholipid aberrations on multiple transmitter systems.

If membrane defects and/or abnormalities in phospholipid metabolism were less serious and transmitter systems less disrupted, rather than schizophrenia, such conditions as anxiety syndrome, ADHD or borderline personality disorder may result. However, in the mode predicted by chaos theory, more severe phospholipid abnormalities may precipitate a process that, ultimately, leads to unpredictable dynamic fluctuations between interacting neuromodulatory systems, as some reciprocity between these systems is lost. Two related and sometimes intertwined possibilities, emergent from this flux, would be schizophrenia and bipolar disorder.

The progression of the schizophrenia may be partly dependent upon the efficacy of bipolar disorder as an autopoietic, self-organizing response and partly driven by the iterative processes of memory formation during waking and memory organization/consolidation during dreaming. As suggested in 8.2, in a de-differentiated state, the first psychotic episode may emerge when some personal memory fragments (which function as bizarre memory cues in dreams) become conscious, externalized, visual hallucinations—through being triggered by unconscious association during waking. Or, as suggested in Section 6.1, auditory hallucinations may start to appear during waking experience when inner ‘voices’ (or speech memories) are externalized. Whether visual or auditory, these hallucinations (as emotionally salient phenomena) will be selected for memory formation to the possible neglect of truly (in relation to concerns) emotionally salient material. Diminished functionality for memory organization/consolidation during dreaming would lead to further diminution in emotionally, meaningful memories. Iterations of this process may result in a more or less complete loss of the personally meaningful associations that create and sustain a sense of self (Hartley, 1749).

This de-differentiation thesis should be testable. For example, Voss et al. (2009) were successful in using EEG to demonstrate the physiological correlates of lucid dreaming. As yet, imaging techniques cannot capture the dynamics of neuromodulation, but this should change as the field matures. Nahas (2010) pinpoints multimodal imaging techniques and cross-validations with genetic studies as the way forward.

Although schizophrenia is a heterogeneous condition, one unifying focus is the fragmentation of the self (see, for example, Frith, 1992; Parnas et al., 2008; Sass & Parnas, 2003). The self that emerges in waking. The dreaming self does not do much thinking, and does not think about this loss of thinking. The dreaming self is driven by primary emotions. The engagement between the dreaming self and others in dreams is less empathic. The dreaming self does not inhabit a private subjective realm within a public objective world. But, because of these losses, the dreaming self may be able to make more imaginative hyper-associative connections between personally meaningful memories to consolidate and organize these in long term memory. The waking self does think and can cogitate upon this thinking, is able to engage with others in meaningful joint endeavour, and does dwell in a private inner space whilst experiencing the sense of an embodied self within an externalized world, but may be less able to achieve leaps of associative imagination.
Some of the strangest phenomena in schizophrenia may be explained by the de-differentiation of these two disparate selves. For example, an incomplete or discontinuous externalization of the sense of a self who inhabits an external world may give rise to an uncertain sense of existence. A loss of the distinction between the private internal realm and the external world may lead to a failing sense of the privacy of the inner world and/or aspects of the self may begin to feel like external objects. Diminishing self awareness and self-monitoring may precipitate feelings of anonymity. Such experiences sometimes characterize schizophrenia (Parnas et al., 2008; Sass & Parnas, 2003) As pointed out earlier, personally meaningful psychological associations create a sense of self (Hartley, 1749). If the personally meaningful associations which may be forged in both waking and dreaming dissolve due to de-differentiation then the sense of self may fragment. Bleuler (1911/1950) believed that the core deficit in schizophrenia is the loss of experience of an integrated, continuous sense of self.

Of all the puzzling things about schizophrenia, one of the most perplexing is the question of why, given the majority of people with schizophrenia do not marry or have children, the condition persists, another is that, although full-blown schizophrenia is so disabling, some people with an attenuated form are highly talented (for example, James Joyce and Isaac Newton had psychopathological symptoms, John Nash was diagnosed with schizophrenia, Bertrand Russell and Albert Einstein had first degree relatives with the condition), both the persistence of schizophrenia and the relationship to creativity suggest that a predisposition to schizophrenia may convey some benefit (Andreasen, 2000). Redfield-Jamison (1994) has investigated the relationship between artistic creativity and bipolar disorder, many distinguished poets, artists, writers and musicians (e.g. Blake, Byron, Van Gogh, Woolf and Schumann), although not usually diagnosed as such at the time, seem to have experienced either bipolar disorder or schizophrenia or both. This link to creativity may be explained as a small degree of de-differentiation may be of advantage. If the linear-logic of normal waking consciousness became interspersed with the highly associative leaps of imagination that characterize dreaming then cognition, problem-solving and creativity would be augmented.

Waking and dreaming consciousness are both utterly normal states of mind-brain. Why two conscious states? Why not just one? If schizophrenia is the devastating possibility inherent in the dynamic neuromodulation that enables the achievement of two differing states of consciousness, then this suggests that both these conscious states are necessary and, indeed, may be complementary. Moreover, this de-differentiation thesis can explain why in persons with talents and abilities, just a ‘touch of schizophrenia’ may enhance their contribution to scientific endeavour and artistic creativity.

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References


