ONEIRIC RELEASE
—
PHENOMENOLOGICAL ORIGINS OF ABNORMAL PERCEPTION

by

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"Find out all about dreams and you will have found out all about insanity."[1]

—John Hughlings-Jackson

"It is obvious, it cannot be but obvious that the dream and madness spurt from the same source."[2]

—Henri Ey

[1] Source: Sulloway, 1992: 270; and Windt and Noreika, 2011; [2] Source: Ey, 1967: Translated by Gottesmann, 2010.

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Abstract

Oneiric activity (i.e., dreaming) and schizophrenia share a number of important characteristics. Specifically, both mental conditions can: (1) reliably induce vivid, realistic, and fully integrated multifaceted objectless perception in all sensory modalities, e.g., visual, auditory, gustatory, olfactory, and somatosensory mentations; (2) trigger immersive hallucinatory interactions with non-real objects, humanoid-entities, and landscapes; (3) seamlessly integrate hallucinatory precepts within the larger domain of mental content, i.e., objectless precepts rarely appear "out of place" when they are experienced; (4) produce a disconnection between external and internal narrative structures; (5) induce temporary "breaks-in-reality"; and (6) induce multiple forms of such cognitive impairments as deficiencies in meta-awareness abilities.

These similarities have been noted and commented on in various capacities by philosophers, neurologists, physiologists, sleep researchers, and psychiatrists for more than four-hundred years. To date, however, no model has been able to explain why oneiric activity shares such a substantial number of phenomenological features with schizophrenia. Because of this, the exact interrelationship between these two phenomenon remains elusive. My thesis remedies this gap in knowledge by not only presenting and outlining the detailed shortcomings of previous oneiric formulations of schizophrenia, but also developing a new explanatory framework "Oneiric Release Theory" (ORT) that accounts for the psychotic symptoms of schizophrenia (i.e., waking hallucinations, delusions, and psychosis) through the neurophysiological dynamics of oneiric activity. A cohesive summary of ORT is found on p. 99 of this work.

Résumé

L'activité onirique (c'est-à-dire rêver) et la schizophrénie partagent un certain nombre de caractéristiques importantes. Spécifiquement, les deux conditions mentales peuvent: (1) induire de manière fiable une perception sans objet multiforme, vive, réaliste et totalement intégrée dans toutes les modalités sensorielles, par exemple des mentations visuelles, auditives, auditives, gustatives, olfactives et somatosensorielles; (2) déclencher des interactions hallucinatoires immersives avec des objets non réels, des entités humanoïdes et des paysages; (3) intégrer de manière transparente les préceptes hallucinatoires dans le domaine plus vaste du contenu mental, c'est-à-dire que les préceptes sans objet apparaissent rarement «déplacés» lorsqu'ils sont expérimentés; (4) produire une déconnexion entre les structures narratives externes et internes; (5) induire des «ruptures dans la réalité» temporaires; et (6) induisent de multiples formes de déficiences cognitives telles que des déficiences dans les capacités de méta-conscience.

Plusieurs philosophes, neurologues, physiologistes, chercheurs en sommeil et psychiatres ont noté et commenté ces similitudes à divers titres depuis plus de quatre cents ans. À ce jour, toutefois, aucun modèle n'a été en mesure d'expliquer pourquoi l'activité onirique partage un nombre aussi important de caractéristiques phénoménologiques avec la schizophrénie. De ce fait, l'interrelation exacte entre ces deux phénomènes reste insaisissable. Ma thèse remédie à ce manque de connaissances en présentant et soulignant les lacunes détaillées des formulations oniriques précédentes de la schizophrénie, mais aussi en développant un nouveau cadre explicatif appelé «Oneiric Release Theory» (ORT), qui prend en compte les symptômes psychotiques de la schizophrénie (hallucinations, délires et psychose) à travers la dynamique neurophysiologique de l'activité onirique. Un résumé cohérent de l'ORT se trouve sur page 99.

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Alex Enescu

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Preface

Throughout most of the twentieth century, oneiric activity has been suspected to be involved in the aetiology of schizophrenia. The advent discovery of rapid-eye-movement (REM) sleep in the 1950s allowed researchers to put together the first oneiric formulations of schizophrenia. These models posited that components of REM sleep (i.e., dreaming) can intrude into the waking consciousness of people who suffer from schizophrenia and disrupt their mental activity.

Although, REM intrusion frameworks were able to successfully account for the phenomenological similarities between schizophrenia and dreaming, they were unable to explain the neurophysiological discrepancies between the two phenomena.

The **first part** of this thesis identifies two primary reasons why previous oneiric formulations of schizophrenia have failed to generate any meaningful empirical results. The first of these reasons is that REM sleep is not the neurophysiology basis of dreaming —dreaming can not only occur throughout all stages of sleep, but may also extend into wakefulness through mind-wandering activity. The second reason is that schizophrenia is not a singular homogenous disease entity —it is a cluster of loosely associated syndromes, each with its own diverse neurophysiological signature.

The **second part** of this thesis presents a detailed cross-examination of the phenomenological features of oneiric activity and the psychotic symptoms of schizophrenia, introduces the concept of a "hybrid-state-of-awareness" into the literature (i.e., mixed phenomenological states that rely on components from multiple perceptual states to exist), and lays out a new explanatory framework to account for both the phenomenological *similarities* and neurophysiological *discrepancies* between oneiric activity and the psychotic symptoms of schizophrenia.

In accordance with McGill University's *Graduate and Postdoctoral Studies* (GPS) regulations, I hereby affirm that I am the sole contributor to each section of this work. No co-authors were involved in the production of this manuscript.

Introduction

This work addresses a longstanding problem in the field of psychiatry and sleep research. Specifically, there exists an intricate phenomenological interrelationship between oneiric mental activity (*from the Greek *oneiros* 'dream' + -ic), and the psychotic symptoms of schizophrenia (i.e., waking hallucinations, delusional thinking, and psychosis). Some of these similarities include cognitive deficiencies (e.g., a diminishment in meta-awareness, a reduction in self-referential abilities, loss of insight, and memory retrieval impairments), analogous perceptual experiences, such as objectless perception that can extend to all sensory modalities (i.e., visual, auditory, gustatory, somatosensory, kinaesthetic, vestibular, tactile, visceral, and olfactory systems), and the encounter of complex hallucinated figures, including forms of humanoids entities, animals, hybrid entities, and the experience of complex multi-sensory hallucinatory environments (Hartmann, 1975; Tholey, 1989; Hobson, 2003: 31; Windt and Noreika, 2011; Kraepelin 1915: 11; Bleuler, 1966: 95 & 96; Arango and Carpenter, 2011; Waters, 2014; Goldsworthy and Whitaker 2015; and D'agostino et al., 2013b and 2013c; Gerrans, 2014; Dresler et al., 2014b; Hobson, 2015: 152; Waters et al., 2016; Lim, et al., 2016; Benson and Feinberg, 2017; and Pace-Schott et al., 2017).

On a psychological level, both oneiric activity and the psychotic symptoms of schizophrenia can induce overarching delusional narratives. Common delusional structures that are manifested in both conditions include: (1) delusions of persecution e.g., being followed or monitored by para-governmental agencies, clandestine institutions, monsters, or otherworldly entities; (2) religious or supernatural delusions; (3) delusions of misidentification; and (4) cotard delusions (Nielsen et al., 2003; Arango and Carpenter, 2011; and Gold and Gold, 2014: 59, 60, 62 & 63; and Gerrans, 2014a).

These similarities have lead many researchers to the hypothesis that dreams, waking hallucinations, and psychotic symptoms are likely to have similar phenomenological origins (Thiher, 2004: 172 & 173; Gottesmann, 2010, and Windt and Noreika, 2011). Indeed, thinkers have explored this idea at least since the seventeenth century (Esquirol, 1838; Moreau, 1845; Feinberg, 1970: 125; Sulloway, 1992: 270; Thiher, 2004: 172 & 173; Foucault, 2006/1964: 239; and Windt and Noreika, 2011). But, it was not until the discovery of different stages of sleep and the advent of the rapid-eye-movement (REM) theory of dreams that a proper scientific framework was proposed to account for the intricate phenomenological similarity between oneiric activity and psychotic symptoms (those of schizophrenia in particular). The most influential of these models was developed by William Dement et al., in 1969: "Phasic REM Intrusion".

Dement et al., (1969) posited that components of REM sleep can intrude into wakefulness to produce vivid and seemingly real hallucinatory experiences, in addition to the cognitive deficits that are characteristic of schizophrenia (McCreery 2008; and Limosani 2011). This model conceptualizes dreaming as the phenomenological origins of hallucinatory perception. Unfortunately, REM sleep intrusion theories of schizophrenia cannot account for the neurophysiological discrepancies between waking hallucinations, psychotic symptoms, and REM sleep. Subsequent research has revealed that this was in part due to the incorrect assumption that oneiric activity is the exclusive product of REM sleep (Zarcone 1979; Carney, et al. 2005; and Chokroverty 2011).

Recent data demonstrates that oneiric mentations can be produced throughout all stages of sleep and may even extend outside of their neurophysiological architecture through the process of mind-wandering activity (Schredl, 2010; Domhoff, 2011; Cipolli et al., 2017; and Siclari et al., 2017). Moreover, a thorough analysis of the nosological history of "schizophrenia" emphasizes that the disorder has always been proposed as a group of loosely associated heterogenous syndromes rather than a singular homogenous disease entity (Claridge, 1997; Kandell 1987: 511; Os et al., 1999; and McNally, 2016: 41, 47, 49, 50, 51, 53, 54,93 & 96). It should therefore come as no surprise that REM sleep intrusion formulations of schizophrenia, as well as their subsequent non-REM (NREM) renditions, did not produce any meaningful empirical results. They could not have. Even if dreaming is the phenomenological origins of psychotic symptoms, researchers have erroneously searched for the neurophysiological signature of particular stages-of-sleep in groups of people who were given an immensely heterogeneous psychiatric diagnosis.

Recent research into such hybrid-states-of-awareness as sleep paralysis, lucid dreaming, and somnambulism, has revealed that the underlying neuro-phenomenological characteristics of sleep, wakefulness, and dreaming can become functionally imbricated to form new compounded perceptual states. These findings offer a unique opportunity to revisit oneiric formulations of schizophrenia, and to develop new frameworks through which its psychotic symptoms can be accounted for. This thesis will determine whether a reformulated and expanded version of previous oneiric formulations of schizophrenia can sufficiently explain both the neurophysiological discrepancies between sleep, dreaming, and psychotic symptoms and their phenomenological similarities. The modern oneiric-based model of abnormal perception proposed will be constructed with four primary arguments.

First, a revised oneiric explanatory framework of schizophrenia needs to account for the neuro-phenomenology of such specific psychotic symptoms as delusional thinking, waking hallucinatory perception, and psychosis. It will not attempt to address the entire symptomatological spectrum of

schizophrenia. Second, a new oneiric formulation of schizophrenia should acknowledge that neither REM sleep, nor NREM sleep, is the neurophysiological basis of oneiric activity. Indeed, as recent studies have shown, oneiric activity can extend far beyond the neurophysiological architecture of any specific sleep stage. Third, the existence of such hybrid-states-of-awareness as lucid dreaming, sleep paralysis, and somnambulism, demonstrate that the elementary components of sleep, wakefulness, and oneiric activity can become functionally imbricated to create new perceptual states. Last, it needs to be emphasized that the explanatory framework of "dream intrusion" is insufficient to account for psychotic symptoms. The dynamic range of functional hybridization and the true complexity of psychotic symptoms requires a new hybridization mechanism, i.e., what this thesis proposes as Oneiric Release Theory (ORT). A succinct summary of the model can be found on p. 99 of this work.

These four arguments will be developed in two parts and broken into five different sections. Part one of this work is composed of Section I and II, whereas part two is composed of Section III, IV, and V. The first section "Oneiric Formulations of Schizophrenia", presents the neurophysiological framework through which REM sleep intrusion and NREM sleep models of schizophrenia were developed. It also offers a detailed overview of the empirical studies that lead to their invalidation. The second section, "Rationale for Revisiting Dream Intrusion Models of Schizophrenia", examines evidence from sleep and mind-wandering studies that illustrates the many ways in which oneiric activity can extend beyond the neurophysiology of traditional sleep stages. This section also argues that schizophrenia was never intended to be conceptualized as a singular "homogenous" psychiatric condition and that a new oneiric formulation of schizophrenia should target specific psychotic symptoms within the spectrum of schizophrenia (i.e., the psychotic symptoms of the disorder) rather than the condition as a whole.

The second part of this work (comprising of Section III, Section IV, and Section V) builds on these two insights and shows that (1) oneiric activity most resembles the psychotic symptoms of schizophrenia; (2) different combinations of sensory and higher-order cognitive demodulation will give rise to different perceptual states; (3) through the process of sensory and higher-order cognitive demodulation, the elementary components of wakefulness, sleep, and dreaming can become "imbricated" to create what I call "hybrid-states-of-awareness"; (4) sensory and higher-order cognitive demodulation is a functional process that does not require structural impairments to occur and can be reversed; and (5) the demodulation of sensory and higher-order cognition is the root mechanism through which the "oneiric release" process occurs.

Section III begins with a detailed phenomenological overview of the psychotic symptoms of schizophrenia and oneiric activity that outlines and documents the many remarkable similarities between these perceptual states. The fourth section of this work, "Hybrid-States-of-Awareness", presents a unique interpretation of sleep paralysis, lucid dreaming, and somnambulism. Specifically, this section argues that the elementary components of sleep, wakefulness, and oneiric activity can become functionally imbricate to create new perceptual states and suggests that psychotic symptoms can be conceptualized as a fourth genre of hybrid-states-of-awareness.

The final section, "Oneiric Release as Possible Imbrication Mechanism", lays out the framework for a new mechanism to explain the full dynamic range of functional hybridization. The model, "Oneiric Release Theory" (ORT), is predicated on a modified version of Rodolfo Llinás formulation of wakefulness as a sensory modulate dream state, Louis West's Perceptual Release Theory (PRT) of hallucinations, and Hughlings-Jackson's Dissolution of Higher Centres theory of psychosis. More specifically, ORT postulates that the "imbrication/hybridization" of different perceptual states occurs when the natural sensory demodulatory processes of sleep or the sensory modulatory processes of wakefulness occur in a disconsonant and incongruous manner. In the case of psychotic symptoms, ORT posits that the phenomenological integrity of wakefulness (composed of the assembly of properly modulated sensory and cognitive impressions) "collapses" back onto its underlying oneiric architecture, and effectively "substitutes" each incongruously demodulated sensory or cognitive pathway with an analogous oneiric impression. The final section of this work also provides empirical evidence in support of ORT and lays out the groundwork for two future studies that can be used to empirically test and further develop the neurophysiological framework of ORT.

PART ONE

PART ONE INTRODUCTION

The first part of this work is composed of two sections. Section I ("Previous Oneiric Formulations of Schizophrenia") provides an overview of previous oneiric formulations of schizophrenia that analyzes their findings, limitations, and shortcomings. Section II ("Rationale For Revisiting Oneiric Formulations of Schizophrenia") identifies two primary reasons why previous oneiric formulations of schizophrenia have not been supported by empirical data. First, because dreaming is not the exclusive product of any particular sleep stage, and second because schizophrenia is not a singular disease entity. These collective insights are applied in the second part of this work to develop a new oneiric formulation of schizophrenia, namely, Oneiric Release Theory (ORT).

SECTION I

PREVIOUS ONEIRIC FORMULATIONS OF SCHIZOPHRENIA

Section I Introduction

Thinkers and researchers have been commenting on the phenomenological similarities between dreaming and schizophrenia for centuries. But, in the absence of a neurophysiological understanding of oneiric activity, the phenomenological similarities between these two phenomena could not be integrated into a larger neurophysiological model. The advent discovery of REM and NREM sleep in the 1950s encouraged researchers to develop the first empirical frameworks that could test if schizophrenia and oneiric activity might be conceptualized as the products of similar neurophysiological structures (Koresko et al., 1963; Rechtschaffen, 1964; Feinberg et al., 1964, 1965 and 1970: 125; Stern et al., 1969; Caldwell et al., 1967; and Benson and Feinberg, 2016). This section presents important oneiric formulations of schizophrenia that were modelled on the neurophysiology of either REM or NREM sleep. It examines their empirical findings, limitations, and shortcomings in chronological order.

It is important to note here that twentieth-century researchers rarely provided a detailed comparison between the phenomenology of oneiric activity and schizophrenia. The two phenomena were suspected to have a similar phenomenological origin strictly because both could generate hallucinatory and delusory activity. Therefore, a detailed analysis of the exact phenomenological relationship between oneiric activity and schizophrenia will not be presented in Part I of this work. In fact, it would be anachronistic to do so. Instead, an exhaustive cross-examination of oneiric activity and schizophrenia will be presented in Section III (p. 41). The reader is invited to consult this section first if they are unfamiliar with the phenomenological similarities between the two phenomena.

The Discovery of Rapid-Eye-Movement (REM) Sleep

Throughout the 1950s, 1960s, and 1970s, dream and sleep researchers sought to establish oneiric activity as the phenomenological origins of schizophrenia. The effort was facilitated by a number of recent technological advancements, most notably the ability to measure electroencephalographic waves in humans through the usages of the electroencephalogram (EEG), the application of EEG scans to human sleep, and the discovery of rapid ocular movements during sleep (Raehlmann and Wikowsky, 1877; Ladd, 1892; Berger, 1928; Loomis et al., 1935; and Aserinsky and Kletiman, 1953).

The story begins in 1938 with Edmund Jacobson (1888-1983) who discovered that subjects who were awoken during periods of "ocular movements activity" were "highly likely" to produce dream reports (Jacobson, 1938). This observation was later independently confirmed by Aserinsky and Kleitman in 1953 and verified by Dement et al., in 1957. Specifically, Aserinsky and Kleitman

observed not only that cyclical rapid-eye-movements (REM) took place in ninety minute intervals between three to four times each night, but also that they were followed by periods of ocular stillness, or "non-REM" (NREM) sleep (Aserinsky and Kletiman, 1953). The two researchers also confirmed that REM-sleep (REM sleep) awakenings yielded significantly higher dream reports than NREM-sleep (NREM sleep) awakenings. Specifically, they found that 70% of REM sleep awakenings yielded a dream report, whereas only 17% of NREM sleep awakenings did so. This discovery gave birth to the first neurophysiological model of oneiric activity, i.e., the REM sleep hypothesis of dreams (Feinberg et al., 1964; Fagioli, 2002; Schredl, 2010; and Cipolli et al., 2017).

Physiologically, it is now known that REM sleep begins in the brainstem (i.e., in the pons and caudal midbrain), where cerebral cortex projecting pontine cholinergic neurons activate such parts of the thalamus as the lateral geniculate area and initiate ponto-geniculo-occipital (PGO) waves, or "spike potentials", that occur in three to ten waves, in lower electroencephalogram (EEG) amplitude (Winson, 1993; and Siegel, 2017). Additionally, the brainstem and hypothalamus contain cells that [either maximally, or minimally] activate during REM sleep (Siegel, 2017). "REM-on cells" include acetylcholine, gamma-aminobutyric acid (GABA), and glutamate. "REM-off cells" are generally limited to norepinephrine, epinephrine, serotonin, and histamine molecules (Siegel, 2017). Destruction or damage to the medulla, pons, or midbrain has been shown to either change the physiological characteristics of REM sleep or inhibit it altogether (Siegel, 2017).

REM sleep is marked by a series of unique features, including, low voltage activity in the neocortex (40Hz gamma activity), intracerebral disconnection from the peripheries and the precuneus, incomplete dorsomedial prefrontal cortex connectivity, loss of auditory-evoked responses, deactivation of the dorsal lateral prefrontal cortex (DLPFC), the emergence of waking theta waves levels in the hippocampus, thalamus, extrastriate and temporo-occipital cortices, irregular heart rate and respiration, desynchronized optical EEG, decreased thermoregulation, and the inhibition of spinal motor neurons (i.e., loss of muscle tone). Researchers divide REM sleep into two states: (1) phasic REM sleep, during which muscle twitches and ocular movements can be detected; and (2) tonic REM sleep, i.e., REM sleep without its phasic features (LaBerge, 1988; Winson, 1993; Mirmiran, 1995; Braun et al. 1997: 1190; Kahn and Gover, 2009; and Gottesmann, 2010; Hobson, 2010; Dresler et al., 2012; Voss, 2014; Filevich et al., 2015; Siegel, 2017; and Cipolli et al., 2017).

On a neurophysiological level, REM sleep is remarkably similar to wakefulness (Werth et al., 2002; and Llewellyn, 2009). In particular, both the neurocortex and the thalamus are equally as active during REM sleep as during wakefulness (Stickgold, 2017). In fact, the only substantial differences

between wakefulness and REM sleep is the presence of muscle atonia, which is responsible for inducing such markedly changed external behavioural output as unresponsiveness to external stimuli (Llinás et al., 1991; Mahowald et al., 1998; Espãna, 2013; Norman, 2005; and Stickgold, 2017). Indeed, because of its similarity to wakefulness, researchers have nicknamed REM sleep as "paradoxical sleep" (Llinás et al., 1991; Winson, 1993; Mahowald et al.,1998; Norman, 2005; Kussé et al., 2010; Christoff et al., 2011; Espãna, 2013; and Stickgold, 2017).

The function and purpose of REM sleep remains elusive. What is known, however, is that REM sleep only occurs in homeothermic mammals and birds —including infants and fetuses (Hartmann, 1982; Crick et al., 1983; Hunt 1989; Winson, 1993; Mirmiran, 1995; Werth et al., 2002; Franklin et al., 2005; Carney et al., 2005; Hobson, 2010; Sándora et al., 2014; and Siegel, 2017). These observations have lead to the hypothesis that REM sleep may be somehow involved in the mammalian thermoregulatory process (Hartmann, 1982; Crick et al., 1983; Hunt, 1989; Hobson, 1999: 187 & 188; McGinty, et al., 2008: 13; and Hobson, 2010).

Some other biological functions that have been suggested for REM sleep include: (1) conserving energy; (2) driving discharge; (3) prevention of brain inactivity; (4) "re-charging" essential neural systems; (5) preventing a central nervous system collapse; (6) programming behaviour; (7) modulating brain temperature; (8) facilitating binocular coordination; (9) stimulating nerve growth; (10) encoding memory traces; (11) erasing superfluous memory traces; (12) maintaining vigilance systems; (13) activating the central nervous system (CNS); and (14) stimulating the brain during the absence of sensory input, among other formulations (Snyder, 1966; Roffwarg, 1966; Dement et al., 1969; Berger, 1969; Jouvet, 1975; Crick et al., 1983; Hunt, 1989; Wehr, 1992; Vertes, 1992; Winson, 1993; Mirmiran, 1995; Hobson, 1999: 188; Vertes and Kathleen, 2000; and Norman, 2005).

Non-Rapid-Eye-Movement (NREM) Sleep

The function and purpose of NREM sleep is equally ambiguous. Generally speaking, NREM sleep is characterized by low voltage activity in the neocortex, K-complexes, regular heart rate and respiration, synchronous (high-amplitude), low-frequency EEG —"with delta (<4 Hz), theta (4-8 Hz), and spindle (12-14 Hz) waves" —thalamocortical activity, and low muscle tone (Norman, 2005; Espãna, 2013; Carskadon et al., 2017; and Stickgold et al., 2017). Traditionally, NREM sleep had been divided into four continuous, fluid, albeit physiologically distinct, stages of sleep, namely, NREM 1 (N1), NREM 2 (N2), NREM 3 (N3), and NREM 4 (N4) (Siegel, 2017; and Stickgold et al., 2017). In 2007, however, the American Academy of Sleep Medicine (AASM) merged N3 sleep with

N4 sleep into a single sleep stage that is now referred to as "slow-wave-sleep" (SWS) or NREM 3 (N3).

N1 is described as a "low-arousal" transitional state. During this stage, such elements of both wakefulness and sleep as partial sensory and muscular modulation can congruently coexist (Hobson, 1999: 52; and Carskadon et al., 2017). N2 has a lower arousal threshold than N1, is marked by the gradual emergence of high voltage biphasic slow-wave activity, and produces sleep spindles or K-complexes in the EEG (Hobson, 1999: 52; Norman, 2005; Carskadon et al., 2017). N3 generates twenty to fifty percent of its onset EEG activity as (at least) 75 μV voltage in the neocortex and slow-waves (Carskadon et al., 2017). The latter part of N3 (formally N4) is characterized by slow-waves (>50%), low arousal rates, and 75+ μV activity.

The first sleep cycle ends through a "reversed ascendency" of NREM sleep 3 back into NREM sleep 2, NREM sleep 1, and REM sleep, followed by a "brief" period of wakefulness (Hobson, 1999: 52; and Carskadon et al., 2017). The second sleep cycle emerges from that "brief" period of wakefulness (as opposed to full wakefulness), and then goes on to reproduce the architecture of the previous cycle. As the sleep cycles progresses, three alterations are produced. (1) the period spent in REM sleep substantially increases; (2) after two hours of sleep, the neurophysiological signature of the latter part of N3 sleep is no longer produced; and (3) roughly five hours into the sleep cycle, the neurophysiological features of N3 sleep also cease.

In general, an eight hour sleep period, will produce about four to five sleep cycles. Each can last anywhere between 90 and 120 minutes (Tononi, 2009; and Carskadon et al., 2017). During this period, episodes of "brief" wakefulness will account for 5% of total sleep, N1 for about 2-5%, N2 for about 45-55%, N3 for anywhere between 13-23%, and REM sleep will account for about 20% to 25% of each sleep cycle (Hobson, 1999: 52; Norman, 2005; Carskadon et al., 2017; and Carskadon et al., 2017). As such, NREM sleep 2 (and NREM sleep in general), not only form the bulk majority of sleep (>80%), but also constitute the architectural basis of sleep (Hobson, 2003: 124).

Alterations in Functional Connectivity (REM Sleep, NREM Sleep and Wakefulness)

Although the brain continues to be just as active during sleep as during wakefulness, its functional connectivity undergoes substantial changes (Hobson, 1999: 59). For example, the relay of information between the striate and extrastriate cortices, as well as the relationship between the amygdala and temporal occipital cortex, become inverted during REM sleep (Kussé et al., 2010). Some other major functional differences during REM sleep include: the suspension of higher-order

association and unimodal sensory areas, and the adoption of a "closed-loop" circuit in the prefrontal areas (Waters et al., 2016; and Stickgold, 2017).

During REM sleep, the thalamus operates in the relay mode such that sensory information continues to be transmitted to the cortex just as during wakefulness. On the other hand, during NREM sleep the thalamus operates in an oscillatory mode (burst mode) and environmental information becomes suppressed. In other words, during NREM sleep, sensory data no longer reaches the cortex and internal physical output can no longer become physically enacted (Mahowald et al., 1998; and Hobson, 2010). Put differently, the brain becomes differentiated during NREM sleep and inattentive to external input during REM sleep (Mahowald et al., 1998; Hobson, 2010; and Espãna, 2013).

Local Sleep

It should also be noted that sleep was originally believed to be a top-down/whole-brain phenomenon with a universal "on/off" switch. However, repeated studies have shown that no specific brain area or brain part is required to generate sleep (Mahowald, et al., 2011b; Vyazovskiy et al., 2011; Krueger, et al., 2013; and Krueger, et al., 2019). In fact, sleep appears to be localized in highly interconnected and semi-autonomous/self-organizing individual neuronal/glial networks that continuously oscillate between wakefulness and sleep (Vyazovskiy et al., 2011; Krueger, et al., 2013; and Krueger, et al., 2019). This means that different parts of the brain can be both awake and asleep at the same time (Vyazovskiy et al., 2011; Krueger, et al., 2013; and Krueger, et al., 2019). As such, components of wakefulness and sleep can become periodically hybridized to generate new neurophysiological states (Mahowald, et al., 2011b). More on this in Section IV.

Proto-REM Sleep Intrusion Models

The first empirically testable proto-REM sleep intrusion model of schizophrenia was proposed by William Dement in 1955. Dement speculated that if oneiric activity, then believed to be the exclusive product of REM sleep, was the phenomenological origins of schizophrenia, then people who suffer from schizophrenia were likely to display some type of alteration in the architectural structure of their REM sleep cycles (Dement, 1955).

The underlying premise behind Dement's hypothesis (1955) was that people who suffer from schizophrenia might fulfil some of their REM sleep requirements during wakefulness, and hence display unusual REM sleep patterns during sleep. Dement's study (1955), however, detected no differences between the REM sleep architecture of people who suffer from schizophrenia and those

who do not. In 1963, Koresko and Feinberg expanded Dement's model (1955), to hypothesize that if schizophrenia is the product of a compromised dream generating mechanism (i.e., if people who suffer from schizophrenia spend their waking hours "dreaming/hallucinating"), then it would follow that they should also implicitly —either partially, or impartially— fulfil their "REM requirements" during wakefulness instead of sleep. Koresko et al., posited that schizophrenia might "require" less total REM sleep or perhaps no REM sleep at all (Koresko et al., 1963; and Hartmann et all., 1965).

Koresko et al.,'s study (1963) yielded no data in support of the hypothesis. They were unable to observe a statistically significant difference in the overall REM sleep architecture of people who suffer from schizophrenia (Koresko et al., 1963). A year later, Feinberg et al., set out to replicate the 1963 study. In particular, Feinberg hoped to discover previously overlooked REM sleep anomalies in people who suffer from schizophrenia. This study also failed to produce any meaningful empirical results (Feinberg et al., 1964). Variations of Dement's hypothesis (1955) were developed and tested throughout the 1960's by Feinberg et al., (1965), Gulevich et al., (1967), Stern et al., (1969), and Kupfer et al., (1970). None detected any significant differences between the REM sleep cycle of people who suffer from schizophrenia and those who do not (Caldwell et al., 1967; Zarcone, 1979; and Benson and Feinberg, 2016).

In 1964, Rechtschaffen et al., took a different approach. Instead of assuming that people who suffer from schizophrenia experience "less" REM sleep, they inverted the causal relationship, to postulate that schizophrenia might be the result of excessive REM sleep that *protrudes* into wakefulness. In other words, Rechtschaffen et al., (1964) set out to test if people who suffer from schizophrenia produce REM sleep mentations *during* wakefulness. They studied the waking eye movements, EEG, and electromyogram (EMG) tracings of a small group of people who suffer from schizophrenia (Rechtschaffen et al., 1964). Yet the group of researchers was unable to find evidence of REM sleep in the waking architecture of their subjects. Rechtschaffen et al.,'s study (1964) was reproduced in 1969 by Stern et al., with a minor twist. In addition to EEG and EMG tracings, the group also analyzed potential differences in the eye movements of people who suffer from schizophrenia and those who do not through the usage of electrooculography (EOG). The study, like its predecessors, also failed to produce any meaningful results (Stern et al., 1969).

By the early 1970s, not a single study had been able to detect REM sleep differences in either the waking or sleep architecture of people who suffer from schizophrenia (Kupfer et al., 1970; Benson and Feinberg, 2016). In spite of this, optimism still ran high. In fact, many researchers were still convinced that it was just a matter of time before the neurophysiological interrelationship between oneiric mentations and schizophrenia was discovered (Feinberg, 1970: 125). Researchers

started to look at alternative theoretical frameworks to account for the phenomenological, cognitive, and psychological similarities between oneiric mentations and schizophrenia. In their search, they found the "REM rebound effect".

In 1960, Dement observed that selective REM sleep deprivation induced prolonged subsequent REM sleep periods, or what he termed: "the REM rebound effect" (Dement, 1960). Dement believed that REM sleep rebound was how losses in REM sleep were compensated for during subsequent periods of sleep. This suggested that REM sleep must fulfil a crucial bio-physiological purpose —otherwise, why would compensation occur? (Dement, 1960; Hernández-Peón, 1967; and Vogel, 1974; Crick et al., 1983; and Winson, 1993; and Roehrs et al., 2017). Dement's observation that periods of REM sleep deprivation were followed by subsequent periods of "psychologically disturbed" wakefulness further supported his hypothesis (Dement, 1960).

Some of the psychological disturbances Dement had observed included, anxiety, depression, and increased irritability (Hernández-Peón, 1967; and Vogel, 1974). This observation suggested that REM sleep deprivation might be implicated in the aetiology of abnormal psychology (Dement, 1960).[3] Indeed, the relationship between waking psychology, REM sleep, and the "REM rebound effect", made "it look as if the dream state could intrude into wakefulness to produce psychiatric symptoms", or as "if REM sleep [could act] like a [psychological] regulating valve" (Vogel, 1974). This lead to the hypothesis that the inability to maintain proper REM sleep (i.e., endogenous REM sleep deprivation), might be indirectly responsible for the induction of psychological disturbances —including psychosis (Hartmann, 1965; and Vogel, 1974).

The model that ultimately became known as the "REM rebound formulation of schizophrenia" was primarily outlined by Ernest Hartmann (1965). Specifically, Hartmann argued that hypogenic and tranquilizers, in addition to such other drug as phenothiazines and alcohol, have a tendency to either reduce or altogether inhibit REM sleep. He further speculated that people who suffer from schizophrenia could be overproducing endogenous "REM sleep inhibiting neurochemicals". In this view, the overproduction of these endogenous compounds would be responsible for the induction of "severe and sudden" REM sleep rebound periods and contribute to the possible development of multiple types of psychological disturbances during periods of subsequent wakefulness (Hartmann, 1965; Dement, 1967; and Gulevich et al., 1967).

A number of researchers, including Vogel (1974), Gillin (1974), and Zarcone (1975), made multiple attempts to test the model. It is important to note that, even though the theoretical

[3] These effects were later shown to be statistical artifacts and did not resurface in better controlled studies (Vogel, 1974; and Gillin, 1975).

framework of Hartmann's model was based on the "REM rebound effect", its empirical dimension varied little from previous study designs. The studies also sought to determine if people who suffer from schizophrenia experience less REM sleep than healthy controls. It should not come as a surprise that Hartmann's hypothesis was ultimately unsupported by the empirical data (Vogel, 1974; Gillin, 1974; and Zarcone, 1975). Indeed, none of the controlled studies carried out throughout the 1970s observed any statistically significant differences in the duration, efficiency, or quality of REM sleep in the sleep cycles of people who suffer from schizophrenia (Vogel, 1974; Gillin, 1974; Zarcone, 1975; Gillin et al., 1975; and Zarcone, 1979).

The studies, however, were not fruitless. It shocked all expectations to discover that people who suffer from schizophrenia do not display any signs of REM rebound. Instead of finding that people who suffer from schizophrenia experienced prolonged periods of REM sleep rebound that could extend into wakefulness, the exact opposite effect was discovered. People who suffer from schizophrenia proved to be effectively immune to the "REM sleep rebound effect" (Gulevich et al., 1967; Gillin 1974; Zarcone 1975; Gillin et al., 1975; Zarcone, 1979; and Benson and Feinberg, 2016).

Dement's REM Sleep Intrusion Hypothesis

The discovery of "REM sleep rebound immunity" in people who suffer from schizophrenia led to the development of an entirely new genre of REM sleep models of schizophrenia (Vogel, 1974). The new framework was once again pioneered by Dement, who postulated that people who suffer from schizophrenia may not experience a "REM sleep rebound effect" because they are already dreaming during wakefulness. Dement further speculated that people who suffer from schizophrenia must be fulfilling their REM sleep requirements outside of sleep —just as Koresko and Feinberg had suggested in their 1963 paper.

Dement named this model the "phasic REM sleep event intrusion" (or the "REM sleep intrusion") hypothesis of schizophrenia. Specifically, he argued that objectless perception (waking hallucinations), delusions, and breaks with reality (i.e., psychoses) were produced through the leakage of REM sleep mentations into unequivocal wakefulness (Dement et al., 1969). Fischman describes how the "hypothesis held that a defective serotonin gating mechanism allowed some phasic events of REM sleep to intrude into the waking state [of people who suffer from schizophrenia]" (Fischman, 1983).

Dement's model conceptualizes wakefulness as a porous states that can be periodically punctured —or suspended— through the interjection of intrusive oneiric mentations. Dement was aware of the previous empirical failures of proto-REM sleep intrusion models and argued that REM

sleep intrusion would not be detectable through the presence of REM sleep during wakefulness or through immediately observable alterations in the REM sleep cycle of people who suffer from schizophrenia (Fischman, 1983). He denounced previous research methods, and posited that pontine-geniculate-occipital (PGO) waves are the underlying neural correlates of oneiric activity (Dement et al., 1969; Laurent et al. 1977; Jouvet 1979; and Fischman, 1983).

Dement found partial support for his theory in an animal study that detected traces of PGO waves in the waking cycle of abnormally behaving (possibly hallucinating) cats (Dement et al., 1969). Dement further hypothesized that if dreams are the phenomenological basis of objectless perception, then PGO waves (i.e., what he now speculated to be the "neurophysiological correlates of dreaming") may be detectable during the waking cycle of people who suffer from schizophrenia. The hypothesis proved difficult to test because PGO waves can not be reliably monitored in the absence of intrusive surgery on human subjects since they originate in the pons and the lateral geniculate nucleus (LGN).

Unable to carry out the intrusive surgical procedures, Dement adapted the hypothesis to the technical capabilities of the period and developed a number of non-intrusive methodologies to test the REM sleep intrusion hypothesis. In particular, he used the electromyogram (EMG), to monitor such alternative REM sleep markers as periorbital integrated potentials (PIPs) and middle ear muscle activity (MEMA) in people who suffer from schizophrenia. He also considered looking for possible signs of abnormal NREM sleep distribution through EEG recordings (Dement et al., 1969; Vogel, 1974; and Benson et al., 1985). Using these alternative physiological markers, Dement's hypothesis was rigorously tested by Feinberg (1964, 1965 and 1967) Guleyich et al., (1967), Stern et al., (1969) Kupfer et al., (1970), Itil et al., (1972) and Jus et al., (1973). But, these studies were also unable to establish a correlation between existing REM sleep markers and schizophrenia (Vogel, 1974; Benson et al., 1985; McGreery, 1997; and Benson and Feinberg, 2016).

In 1974, Vogel summarized the predicament: "The bright promises of a decade ago have dimmed". Indeed, twenty years after the discovery of REM sleep, not a single controlled study established a definitive link between the neurophysiological characteristics of REM sleep and schizophrenia. As for PGO waves, although never directly studied in people who suffer from schizophrenia, later research showed that dreaming can occur even in the absence of the ascending PGO system, (i.e., even after it has been impaired or destroyed). This later finding demonstrates that PGO waves are not the "minimal neural substrate" of oneiric activity that Dement's "phasic REM intrusion" model posited (Dement et al., 1969; Laurent et al. 1977; Jouvet 1979; and Fischman, 1983). Perhaps, researchers were looking in the wrong place?

Post-REM Sleep Intrusion Models

In 1983, Francis Crick et al., became fascinated by the phenomenological and cognitive similarities exhibited by schizophrenia and oneiric activity, and contributed to the development of an entirely new genre of REM sleep intrusion models. Specifically, Crick et al., (1983) speculated that REM sleep (still considered the neurophysiological basis of dreaming) functioned as a "reverse learning" mechanism. In other words, he postulated that REM sleep may remove non-essential memory traces from waking consciousness. Since not every waking moment is remembered, Crick posited that memories must be "selectively stored", or "selectively removed". He argued this to be the function of REM sleep and believed that dreams are the phenomenological by-product of this process (Crick et al., 1983).

Crick et al., went on to speculate that if both schizophrenia and oneiric activity have the same phenomenological origins, then it would follow that schizophrenia can be conceptualized as a "REM sleep reverse learning disorder," or a disorder caused by the inability to efficiently "remove" superfluous memory traces during sleep (Crick et al., 1983). Under this formulation, the memory systems of people who suffer from schizophrenia are considered, overloaded. Indeed, according to Crick et al., (1983), the symptomatological spectrum of schizophrenia is produced through the "over-accumulation" of superfluous memory traces. Even though Crick et al.,'s (1983) model of schizophrenia failed to produce empirical results, it was nevertheless expanded by Kelly in 1998, who argued that "superfluous" memory traces may foster a mental environment that is conducive to delusional thinking and delusional beliefs. More recently, D'Agostino et al., (2013) contended that imperfect memory consolidation during REM sleep may be the source of schizophrenia. None of these subsequent models, however, were able to generate an empirically testable hypothesis.

REM Sleep Intrusion Models in the Early 21st Century

The repeated failures of REM sleep intrusion models over four decades did not deter researchers from further investigating the possible interrelationship between dreaming and schizophrenia (Collerton et al., 2005). Dement's REM intrusion hypothesis was reformulated by Claude Gottesmann in a 2006 paper. Gottessmann (2006) acknowledged the repeated failures of previous REM sleep intrusion models, yet he insisted that the similarities between oneiric mentations and schizophrenia are too formidable to be accidental. Furthermore, Gottesmann argued (2006 & 2007) that even if the physiological properties of REM sleep may not necessarily "intrude into wakefulness" during periods of psychosis, REM sleep may nevertheless be used as a "model for

schizophrenia." In other words, he contends that by studying REM sleep, it may be possible to discover overlooked neurophysiological characteristic of schizophrenia.

Gottesmann's position was further developed by Scarone et al., in 2007, Limosani et al., in 2011, and by Allan Hobson in 2011 and 2014. Collectively, these researchers accepted the empirical failures of previous REM sleep intrusion hypothesis, but still maintained that because REM sleep shares so many neurophysiological features with schizophrenia further research is warranted. Specifically, they point to schizophrenia's and REM sleep's shared intracerebral disconnections, disturbed responsiveness, sensory deafferentation processes, breakdown in forebrain-inhibitory influences, dorsolateral prefrontal deactivation, and "disinhibition of auditory-evoked potentials at the cortical level." They also note that both display identical pharmacological and neurochemical variations (Gottesmann 2006, 2007, and 2010; Hobson, 2004; 2011; and 2014; Scarone et al., 2007; Hobson, 2015: 153; Schredl, 2010; Windt and Noreika, 2011; Limosani et al., 2011; Domhoff and Fox, 2015). However, none of these researchers were able to offer new empirical frameworks that allowed their hypotheses to be tested.

In the theoretical domain, Sue Llewellyn (2009) proposed that schizophrenia should be seen as an "in-between" state that borrows components from both REM sleep and wakefulness. Under her formulation, schizophrenia can be seen as a "mixed-state" that simultaneously relies on components of wakefulness and REM sleep to exist. Llewellyn further refined her model in 2011 to add that different psychiatric conditions might be associated with different degrees of REM sleep dreaming "intrusion", i.e., more severe conditions would be predicated on a higher degree of REM sleep integration, whereas milder conditions would only rely on fragmented components of REM sleep (Llewellyn, 2011).

Llewellyn's model, like those that came before it, still relies on the assumption that REM sleep is the neurophysiological basis of dreaming (2009 and 2011). She attributes the empirical failures of previous REM sleep intrusion models of schizophrenia to inadequate neuroimaging techniques (Llewellyn, 2011). In her view, the exact neurophysiology of REM sleep has not yet been adequately mapped out. As a result of this, she speculates that REM sleep intrusion researchers may have been looking for the wrong neural signature in people who suffer from schizophrenia (Llewellyn, 2011).

NREM Sleep Disturbances in People who Suffer from Schizophrenia

REM sleep models of schizophrenia did not produce the expected empirical data. The studies nevertheless produced a number of serendipitous discoveries. In particular, REM sleep intrusion studies documented that people who suffer from schizophrenia exhibit substantial NREM sleep

abnormalities. These abnormalities include disruption in slow-wave-sleep (SWS), sleep onset, and NREM sleep 3 (Caldwell et al., 1967; Feinberg et al., 1964, 1965, 1969; Dement et al., 1969; Stern et al., 1969; Gillin et al., 1975; Tandon et al., 1992; and Hudson et al., 1993; and Benson and Feinberg, 2016).

In 1970, Kupfer et al., discovered that NREM sleep disruptions can reliably foreshadow psychotic episodes. This finding was initially promising, but further research revealed that NREM sleep abnormalities are not unique to schizophrenia (Feinberg, 1969, and 1970). In fact, NREM sleep impairments are also predictive of depression, somnambulism, and anxiety (Feinberg, 1969). Moreover, NREM sleep abnormalities in people who suffer from schizophrenia display enormous intra-individual variability and do not exhibit an intrinsically recognizable pattern (Kupfer et al.,'s 1970). This made it difficult to correlate any particular NREM sleep disruption to schizophrenia (Kupfer et al.,1970; Kupfer et al., 1970; Zarcone, 1987; and Gillin et al., 1975).

In 1997, Lauer challenged the NREM sleep data from the 1970s by showing that non-medicated people who suffer from schizophrenia do not appear to display any NREM sleep abnormalities. Keshavan et al., (1998) replicated Lauer's (1997) study and reported contradictory results. Keshavan et al's research posits that NREM sleep abnormalities do exist even in non-medicated individuals who suffer from schizophrenia. These two studies ignited a debate on the interrelationship between NREM sleep abnormalities and schizophrenia. It was during this period that Zarcone published a seminal paper (1997) to establish that a relationship between psychotic episodes and a number of preceding sleep disturbances, such as difficulties in falling asleep, a reduction in SWS, and lowered overall sleep efficiency, do indeed exist.

Zarcone's model (1997) was further developed by Poulin et al., (2003), Manoach et al., (2004), Forest et al., (2007), and Llewellyn (2015). All of these models posit that the various cognitive deficiencies exhibited by people who suffer from schizophrenia are likely to be caused by the fragmentation of NREM sleep 3 (a sleep stage associated with the maintenance of voluntary attentional processes during wakefulness). Such other researchers as Peterson (2008), found that insomnia and psychotic episodes frequently occur together. Peterson's findings were corroborated by polysomnographic (PSG) studies that "validated subjective complaints of poor sleep quality" in people who experience frequent episodes of psychosis (Benson and Feinberg, 2016). Furthermore, thalamocortical network dysfunctions (correlating with anomalous sleep spindles activity) have also been detected in people who suffer from schizophrenia (Kelly, 1998; Wamsley et al., 2012; and Manoach et al., 2016).

The evidence for an interrelationship between abnormal sleep patterns and psychotic symptoms has now become indisputable (Manni, 2005; Yang et al., 2006; Sarkar et al., 2010; Sekimoto et al., 2011; Palagini 2011; Benson and Feinberg, 2016; and Manoach et al., 2016). Indeed, such researchers as Kamath (2015) have gone so far as to suggest that psychotic symptoms may be reversible through the administration of antipsychotic medication because of their regulatory effect on sleep. It is now widely accepted that an irregular NREM sleep architecture is a core characteristic of schizophrenia (Benson and Feinberg, 2016). In spite of this, the exact interrelationship between schizophrenia and NREM sleep remains poorly, if at all, understood.

NREM Sleep Intrusion Models of Schizophrenia

In 1970, Feinberg proposed the first NREM sleep model of schizophrenia in a paper entitled, "Hallucinations, Dreaming and REM Sleep" (Feinberg, 1970). Feinberg discusses the empirical failures of previous REM sleep intrusion models and argues that these failures are likely because dreaming may not be an exclusive REM sleep phenomenon.

The new model drew on earlier studies conducted by Foulks (1962), Rechtschaffen (1963), and Goodenought (1965), who all offered early evidence against the REM sleep hypothesis of dreams (more on this in Section II). Feinberg (1970) proposed an oneiric framework for schizophrenia that was predicated on a multi-sleep-stage model of dreaming. More specifically, Feinberg (1970) argued that both REM sleep and NREM sleep are equally capable of generating oneiric mentations. For this reason, he believed it behoved researchers to investigate the neurophysiological interrelationship between NREM sleep dreaming and schizophrenia.

The general consensus of the period was that REM sleep was involved in the production of visual oneiric mentations, whereas NREM sleep was involved in the production of "thought-like" sleep activity (Feinberg, 1970). Feinberg (1970) proposed that different kinds of oneiric mentations (i.e., NREM sleep/REM sleep mentations) are responsible for different types of psychotic symptoms. For example, Feinberg's (1970) model pre-supposed that auditory hallucinations (given their "thought-like" nature) are caused by a form of "NREM intrusion", whereas delirium, which tends to be more visual in nature, is most likely produced by periodic REM sleep intrusion episodes (Feinberg, 1970). Feinberg's model was more flexible than previous REM sleep intrusion formulations of schizophrenia, but the model was still unable to establish a functional interrelationship between the different sleep stages and psychotic symptoms.

Twenty seven years after, Feinberg's (1970) hypothesis was revisited and expanded by Charles McGreery (1997). McGreery noted that the neuro-phenomenological similarities between waking

hallucinations, delusional thinking and oneiric activity "have never been satisfactorily explained" and concluded that REM sleep intrusion researchers "have been looking in the wrong place" (McGreery, 1997; and 2008). He postulated that psychoses are most likely caused by the exclusive architectural displacement of NREM sleep (McGreery, 1997). A decade later, McGreery further revised his model to argue that the symptomatology of schizophrenia is caused by different degrees of "NREM sleep intrusion" (McGreery, 2008).

NREM sleep models of schizophrenia are supported by better empirical data than REM sleep intrusion hypotheses. Episodes of chronic sleep deprivation, for example, can induce periods of "micro-sleep" in healthy individuals. These episodes usually occur outside of conscious perception (or volitional control). They can cause severe impairments in sustained attention, stimuli response, and the ability to accurately scan external information (Forest et al., 2007). Furthermore, episodes of micro-sleep are marked by the combined EEG characteristics of both NREM sleep and wakefulness. This evidence demonstrates that sleep and wakefulness can become imbricated on a neurophysiological level (Roehrs et al., 2017).

This line of research suggests that a type of "endogenous NREM sleep deprivation" may force people who suffer from schizophrenia to spend an abnormal amount of their waking time in a "micro-sleep" state. Indeed, if people who suffer from schizophrenia fail to meet their NREM sleep requirements during sleep, it is possible that a type of "NREM sleep rebound effect" takes place to compensate for the loss. Unfortunately, the exact neurophysiology of NREM sleep remains elusive. For this reason, "NREM sleep intrusion" empirical formulations of schizophrenia are yet to be exhausted (Roehrs et al., 2017).

Hypnagogic Intrusion Models of Schizophrenia

There also exists a genre of NREM sleep models of schizophrenia that is entirely predicated on the neuro-phenomenological similarities between psychotic symptoms and the hypnagogic state. The first model of this kind was formulated by Vogel in 1974 and has been recently revisited by Waters et al., (2016), and D'Agostino et al., (2016). In "Dreaming and Schizophrenia" (1974), Vogel correctly inferred that (1) oneiric mentations are not the exclusive domain of REM sleep; and (2) hypnagogic hallucinations are a type of dreaming that occurs during sleep-onset (SO). Based on these two suppositions, Vogel reasoned that schizophrenia may be caused by a form of "hypnagogia intrusion", or, at the very least, that hypnagogia and schizophrenia may share a partial or common neurophysiology. Unfortunately, hypnagogia has been studied more by poets, writers, and artists than

by scientists and philosophers (Schacter, 1976; and Mavromatis 1987). Because of this, it remains a poorly understood perceptual state.

Some characteristics of hypnagogia are nevertheless clear. (1) the experience takes place during sleep-onset (SO) or NREM sleep 1; (2) it can generate hallucinatory mentations in all sensory modalities; and (3) it is reported to regularly occur in more than 70% of the general population (Waters et al., 2016). Perceptually, hypnagogic hallucinations can induce a complex range of visual features, auditory cues, and somatosensory impressions. Hallucinations can include, but are not limited to, humanoid figures, kaleidoscopic imagery, distorted faces, animals, therianthropes, lilliputian figures, landscapes, mountains, forests, rivers, hearing people talk or whisper out in the distance, vibrations, tactile cues, olfactory sensations, hearing the sound of incongruous chatter, imprecise words, questions, threatening commandments, door-bells, and even one's name being called out (Schacter 1976; Mavromatis, 1987; Manford, 1998; Jones et al., 2010; and Waters et al., 2016).

The phenomenology of hypnagogia is similar to oneiric mentations, albeit not identical. Such sleep researchers as Dement (1967), Schacter (1976), and Mavromatis (1987) suggest that both oneiric activity and hypnagogia may represent different facets of the same underlying phenomenon. Compared to oneiric activity, hypnagogic mentations tend to display an unusual level of "intrusiveness", or "externality" (i.e., they are perceived as having a predominantly "external" origin), and tend to be described as "real", vivid, and well integrated within an individual's perceptual field (Foulkes et al., 1965; Schacter, 1976; Mavromatis, 1987; Manford, 1998; Jones et al., 2010; and Waters et al., 2016). Moreover, hypnagogic hallucinations, in contrast to oneiric mentations, more easily integrate such ongoing external sensory as sounds, sights elements, tactile feelings, and vibrations, into the matrix of their phenomenology (Mavromatis, 1987). Oneiric activity does so to a far lesser extent.

The models developed by Vogel (1974), Waters et al., (2016) and D'Agostino et al., (2016), posit that hypnagogia is the phenomenological origins of schizophrenia. The models are based on four main arguments: 1. hypnagogic hallucinations are more similar to the phenomenology of schizophrenia than REM sleep mentations; 2. Hypnagogic mentations and waking hallucinations display similar degrees of veridical integration (i.e., alongside real sensory input); 3. hypnagogic mentations simultaneously exhibit the neurophysiological features of both wakefulness and sleep (indicating that the state is inherently conducive to functional hybridization); and 4. the characteristic features of auditory hallucinations in schizophrenia (i.e., hearing threatening commands or one's

name being called out, etc) are strikingly similar to the type of auditory cues that are reported by people who experience hypnagogia (Vogel 1974; Waters et al., 2016; and D'Agostino et al., 2016).

Hypnagogic frameworks of schizophrenia have at least two major advantages over REM sleep and NREM sleep intrusion models. First, the phenomenology of hypnagogia, as opposed to that of REM sleep or NREM sleep dreaming, resembles more the content, form, and features of schizophrenia (e.g., veridically integrated threatening hallucinations are a feature of hypnagogia, but not of REM sleep). Second, hypnagogia, unlike REM sleep or NREM sleep dreaming, is a condition that occurs on the threshold of wakefulness and sleep. In spite of this, it still remains unclear if hypnagogia is related to dreaming or if it should be considered an independent perceptual state.

Current evidence suggests that hypnagogia is an imbricated phenomenon. Because of this, there is no good reason to believe that hypnagogia can become further "hybridized" with wakefulness. Put differently, if hypnagogia is not a neuro-phenomenological state onto itself, then it follows that it cannot further "hybridize" with or "intrude" into wakefulness because it is already a "hybrid-state-of-awareness". This means that the mechanisms that enable hypnagogia, whatever they may be, are likely to be of more potential value to schizophrenia research than the actual hypnagogic state itself.

Section I Summary

This section presented a thorough overview of REM sleep, NREM sleep, and hypnagogic formulations of schizophrenia. The section detailed each major iteration of these models and analyzed their shortcomings, limitations, and overall contribution to the field. The next section will present "a rationale for revisiting oneiric formulations of schizophrenia", and identify two main reasons why previous oneiric formulations of schizophrenia failed to yield the expected empirical results.

SECTION II

RATIONALE FOR REVISITING ONEIRIC FORMULATIONS OF SCHIZOPHRENIA

Section II Introduction

This section identifies two main reasons why previous oneiric formulations of schizophrenia failed to produce meaningful empirical results. Previous studies have wrongly assumed that dreaming is the exclusive product of a particular sleep stage and that schizophrenia is a singular disease entity, and not a cluster of loosely associated heterogeneous symptoms. In addition, this section argues that given the limitations of previous oneiric formulations of schizophrenia, and in light of recent discoveries made in both the fields of dream and schizophrenia research, the neuro-phenomenological relationship between oneiric activity and schizophrenia should be revisited.

REM Sleep is not the Neurophysiological Basis of Dreaming

REM sleep, NREM sleep, and hypnagogic intrusion models of schizophrenia all presuppose a relationship between oneiric activity and schizophrenia. The investigation of this relationship were primarily predicated on the assumption that a specific sleep stage, most often REM sleep, is the neurophysiological basis of oneiric activity. This claim has been significantly undermined in recent years.

Data about oneiric activity that occurs outside of REM sleep has been systematically and reliably collected for over six decades now. In fact, NREM sleep dream reports were even collected in Aserinsky and Kleitman's original 1953 study (17% of their NREM sleep awakenings yielded a dream report). Nevertheless, the REM sleep hypothesis of dreaming was strongly supported by a number of early studies (Dement et al., 1957). These researchers concluded that NREM sleep oneiric activity is sufficiently infrequent as to be either a "statistical anomaly" or a type of "memory spillover event" from a previous REM sleep cycle (Dement et al., 1957).

Studies carried out a few years later by Goodenough (1959), Kamiya (1961), Foulkes (1962 cited in Domhoff, 2011), and Rechtschafen (1963), uncovered how NREM sleep awakenings yield significantly more dream reports than expected. In a study conducted by Goodenough et al., (1959), for example, dream reports were collected in at least 7% of NREM sleep awakenings. Foulkes (1962) later expanded the definition of "dreaming" from "visual mentations that occur during sleep" to include "any type of mental activity that occurs during sleep". With this redefinition he collected dream reports from an astonishing 74% of NREM sleep awakenings (Foulkes, 1962). Foulkes' publication (1962) introduced into the literature the claim that REM sleep may not be the exclusive neurophysiological domain of oneiric activity (Goodenough et al., 1959; Roffwarg, 1962; Goodenough et al., 1965; Foulkes, 1967; Feinberg, 1970: 125, 126; Schredl 2010; and Stickgold et al.,

2017). His approach was challenged by Berger (1967) and Feinberg (1970: 126), who argued that "thought-like" mentations should not be classified as "dreams".

The debate between these researchers drew attention to the fact that "oneiric mentations" lacked a universal definition (Domhoff, 2011). It was also during this period, i.e., the late 1960s and early 1970s, that REM sleep dreaming came to be characterized as more "immersive", "visual", "sensorial", and "emotional", whereas NREM sleep mentations came to be described as less "sensorially developed", and more "thought-like", "conceptual", and "cognitive" in nature (Rechtschaffen, 1963; Goodenough et al., 1965; and Stickgold et al., 2017). Yet, many studies conducted during this period also showed that "immersive", "visual", "sensory-based", "developed", and "emotional" dreams can equally be produced during NREM sleep (Rechtschaffen 1963; Berger, 1967; Hernández-Peón 1967; and Feinberg, 1970: 126). The same studies also gathered evidence that "thought-like", "conceptual", "plausible", and "realistic" oneiric mentations can likewise be produced during periods of REM sleep (Rechtschaffen 1963; Berger, 1967; Hernández-Peón 1967; and Feinberg, 1970: 126). Indeed, the collective findings of the 1960s showed that (1) the supposedly "exclusive" phenomenological, psychological, and cognitive features of both REM and NREM sleep can regularly transcend into other's neurophysiological boundaries and that (2) REM sleep is unlikely to be the exclusive neurophysiological basis of "fully-formed" oneiric activity (Hernández-Peón 1967; Berger, 1967; Feinberg, 1970: 126; Tononi, 2009; and Kussé et al., 2010).

In 1977, Hobson et al., proposed the activation-synthesis model (AIM) of dreaming. The model, by and large, ignored the extensive literature on NREM sleep oneiric activity to argue that dreaming is the exclusive product of REM sleep. AIM proved to be surprisingly popular among dream and sleep researchers. Because of this, it dissuaded the research community from searching for alternative neurophysiological oneiric frameworks. By the early 1980s most sleep and dream researchers had come to rely on some version of Hobson's AIM theory in their work (Antrobus, 1983).

Yet, evidence of "fully formed" oneiric activity outside of REM sleep continued to mount (Suzuki et al., 2004; and Schredl, 2010). In 1982, Ernest Hartmann declared that REM sleep only bears a coincidental —as opposed to causal— relationship with dreaming. He interpreted the then current literature on NREM sleep as making it beyond dispute that "fully-formed" oneiric activity can occur outside of REM sleep. Hartmann's position received support from Antrobus (1983), who joined the chorus deriding how an uncritical acceptance of the REM sleep hypothesis and Hobson's AIM theory had overhastily squashed the development of alternative models.

Evidence for the existence of "fully-formed" NREM sleep dream mentations continued to accumulate throughout the 1980s, 1990s, and early 2000s (Cavallero et al., 1992; Flanagan, 1995; Mahowald et al., 1998; Nielsen, 2000; Franklin et al., 2005; Schredl, 2010; Domhoff, 2011; Cipolli et al., 2017; and Siclari et al., 2017). Even so, Hobson (1994 and 2000), who was still influential in the field, refused to relinquish the idea that REM sleep is the neurophysiological basis of dreaming. In 1994, he not only argued that the similarities between REM sleep and NREM oneiric activity had been overemphasized (even "exaggerated"), but also proposed that the two types of sleep mentations were likely generated by two different neurophysiological mechanisms.

This position became known as the Gen. 2 model of dreaming and posits that REM sleep and NREM sleep oneiric activity are separate phenomena (Hobson, 1994; Hobson, 2000; and Stickgold et al., 2017). In 1997, Solms proposed an alternative model, in direct contradiction to the underlying premise of Hobson's Gen. 2 model. Specifically, Solms (1997) posited that both REM sleep and NREM sleep mentations appear to be produced by similar, if not identical mechanisms, and that no substantial phenomenological difference exists between the two types of experiences. Solms' model became known as the Gen. 1 model and posits that the neurophysiological basis of dreaming was still undiscovered (Solms, 1997; Takeuchi et al., 2001; and Stickgold et al., 2017).

The field was now at an impasse. Either dreaming is caused by REM sleep *and* a still undiscovered NREM sleep dream generating mechanism (Hobson's Gen. 2 model) or oneiric activity is caused by a singular undiscovered dream generating mechanism (Solm's Gen. 1 model). A way forward was offered by Nielsen in 2000. In particular, Nielsen (2000) both adopted the underlying premise of Solms' Gen. 1 model by arguing that dream mentations are likely produced through a single neurophysiological mechanism, and *also* sided with Hobson's Gen. 2 model by insisting that the neurophysiology of all oneiric activity must invariably be REM sleep. For Nielsen (2000), the absence of the core neurophysiological characteristics of REM sleep did not automatically imply the absence of REM sleep (Nielsen, 2000). Nielsen's theory (2000) posits that REM sleep can periodically occur "covertly" during NREM sleep (forming the underlying neurophysiological basis of NREM sleep oneiric activity). This model became known as the "covert REM sleep" (cREM sleep) hypothesis.

The cREM sleep hypothesis of dreaming is paradoxical. Yet, despite the fact that REM sleep cannot occur during NREM sleep, it nevertheless received a fair amount of support in the early 2000s (Takeuichi et al., 2001; Werth et al., 2002; Suzuki et al., 2004; Nielsen et al., 2005; and Gottesmann 2010). In 2004, however, Wittman and Schredl published the result of a study that directly refuted the covered REM sleep hypothesis. The two researchers found that subjects who

were awakened during SWS (a sleep stage that Nielsen himself acknowledged should not be susceptible to cREM sleep) reported a substantial number of unambiguous "fully-formed" dream reports (Witmman and Schredl, 2004). Even more damningly, their study yielded an overall 60% more dream reports from NREM sleep awakening than from REM sleep (Witmman and Schredl, 2004). The two authors were unequivocal that their study implies that there is more cREM sleep than actual REM sleep: "Obviously, it makes no sense to name a process after a specific stage of sleep if this process occurs more often outside than inside that sleep stage" (Wittman and Schredl, 2004).

A year later (2005), Valli et al., argued that REM sleep is most likely involved in the consolidation of memories and serves little —if any— function in the production of dreams. Based on the available data, the authors concluded that REM sleep "is neither a necessary or sufficient condition for dreaming" (Valli et al., 2005). This conclusion has been supported by many researchers in following years, including: Fagioli (2002), Fosse et al., (2004), Eiser (2005), Llewellyn (2009), Limosani et al., (2011), Christoff et al., (2011) and Palagini (2011).

In 2012, the REM sleep hypothesis of dreaming was dealt its final blow by the landmark study, "Dreaming Without REM Sleep". Oudiette et al., (2012) hypothesized that were the REM sleep model of dreaming correct, then dream mentations should not be produced in the unequivocal absence of REM sleep. To test their hypothesis, they administered clomipramine (a potent REM sleep suppressor) to a group of healthy volunteers and ensured that all of the neurophysiological features of REM sleep (including those of Nielsen's hypothetical cREM sleep) were effectively suppressed. Using EEG and other standard measurements, Oudiette et al., (2012) woke their subjects during random periods of sleep and asked them if they had "anything to report". To everyone's surprise, the participants reported the presence of fully-formed, detailed, vivid, and multi-modal dream mentations despite the clomipramine (Oudiette et al., 2012). The experiment unambiguously demonstrated for the first time in more than six decades of research that fully-formed oneiric activity can occur even in the complete absence of REM sleep.

Oudiette et al.,'s (2012) study persuaded even Hobson and Nielsen. In 2015, Hobson finally admitted that there is no substantial difference between NREM sleep and REM sleep dream mentations (Hobson, 2015: 18 & 20). In 2016 Nielsen adopted the view that developed mental experiences can take place during all stages of sleep (Nielsen et al., 2016). Yet, the question still remains —why are dream reports more easily collected from REM sleep awakenings and why does REM dreaming tend to display higher levels of emotional arousal than NREM sleep (Maquet et al., 1996)? Based on current estimates, dream mentations are collected from as many as 81.8% of REM

sleep awakening. Less than half of NREM sleep awakening yield a dream report (Pace-Schott et al., 2017). Many now believe that this discrepancy is the result of memory consolidation occurring differently across sleep stages (Wittman and Schredl, 2004; Eiser, 2005; Kussé et al., 2010; Cipolli et al., 2017; Peigneux, et al., 2017 and Siclari et al., 2017).

This is not a new idea. In fact, in 1959 Malcolm argued that sleep memory "may be distorted in ways we do not understand", and that because dreams can only be reported during periods of awakenings, their recall rate is invariably subject to different types of distortions (Malcolm, 1959: 58, 66, and 74). This position is still supported by many researchers, including Goodenough et al., (1959), Oswald (1962b), Rechtschaffen (1967), Hernández-Peón (1967), Crick et al., (1983), Hartmann (1982), Franklin et al., (2005), Schredl (2010), Kussé et al., (2010), Nir et al., (2010), Palagini (2011), D'agostino et al., (2013) , Sándora et al., (2014), Stickgold et al., (2017), and Siclari et al., (2017).

Malcolm's observation that dream experiences cannot be recorded directly (i.e., they must be verbalized during periods of awakening), reminds us that the integrity of a dream report (including its emotional aspects) is invariably contingent on the availability of multiple memory recall systems (Malcolm, 1959: 74, 75, and 83; Nir et al., 2010; D'agostino et al., 2013; Sándora et al., 2014; and Siclari et al., 2017). More specifically, given that the limbic circuits in the medial temporal lobe are highly active during REM sleep and mostly deactivated during NREM sleep, it is possible that the former sleep stage may be better equipped to facilitate the recall of dreams, whereas the latter may be antithetical to it (Hartmann, 1982; Nir et al., 2010; Palagini, 2011; and Stickgold et al., 2017). This would explain why REM sleep generates more dream reports than NREM sleep without it being the neurophysiological basis of oneiric activity.

The relationship between different sleep stages and impaired memory recall has not yet been sufficiently studied. It still remains unclear if disparities in recall rates between different sleep stages are due to different memory consolidation processes or some other unknown factor(s). Having said that, the discovery of fully-formed dream reports in subjects who are given clomipramine, makes it clear that existing neurophysiological models of dreaming are insufficient to account for the full spectrum of oneiric activity. New formulations are needed.

Lessons From Mind-Wandering Research

The recent dismantlement of the REM sleep hypothesis of dreaming has produced a conceptual-vacuum in the field of dream and sleep research. Indeed, more than one-hundred years after Freud published his magnum opus, *The Interpretation of Dreams* (1900), the underlying architecture involved

in the production of dreams still remains largely unknown. A number of recent studies, however, have revealed an unexpected interrelationship between mind-wandering and oneiric activity (Nir et al., 2010).

Specifically, these studies have shown that both mind-wandering and oneiric activity: (1) occur during a state of sensorial-perceptual decoupling (i.e., they are stimulus independent); (2) induce multi-modal, fictive, albeit, mentally navigable, worlds; (3) are predominant "hallucinatory" in nature; (4) have reality-simulation properties (i.e., a tendency to generate social rehearsal scenarios, re-assess past engagements, and plot out future possible scenarios, events, and activities, such as autobiographical planning); (5), allow for imaginative wish-fulfillment, the mental enactment (and-reenactment) of fantastical narratives, and repose from sensory modulation; (6), are characterized by various degrees of meta-awareness deficiency; (7) and often display elaborated narratives structures (Oswald, 1962a; Klinger, 1971; Starker, 1977; Rechtschaffen, 1978; Hobson, 1999: 28; Domhoff, 2003; Fosse et al., 2004; Henry, 2009: 48; Nir et al., 2010; Killingsworth et al., 2010; Schooler et al., 2011; Domhoff, 2011; Baird et al., 2011; Christoff et al., 2011; Mcmillan et al., 2013; Zellner, 2013; Gerrans, 2014b; Smallwood et al., 2015; Domhoff and Fox, 2015).

These findings are especially significant given that anywhere between 50% and 60% of waking time is spent in a mind-wandering state (Seligman and Kirmayer, 2008: 34; Killingsworth et al., 2010; Christoff et al., 2011; and Smallwood et al., 2015). Yet, studying the mind-wandering phenomenon has proven difficult because the activity lacks a rigid taxonomy. Indeed, it is referred to by at least a good dozen common and interchangeable names, including: "mind-wandering activity", "daydreaming", "imaginary activity", "imagination", "thought intrusion", "task irrelevant thoughts", "spontaneous thought", "spontaneous cognition", "stimulus independent thought", "internally generated thoughts", "self-generated thoughts", "absent-mindedness", "zoning out", "offline thought", "undirected thought", and "unconscious thought" (McMillan et al., 2013). The wide variety of words used to describe the mind-wandering phenomenon means that volunteers can significantly underreport their experiences simply based on the vocabulary used by investigators. Even so, it has nevertheless been established that mind-wandering activity can be initiated both deliberately or spontaneously and that it tends to occur in the absence of directed thought and cognitively demanding tasks (Oswald, 1962a; Wolmann et al., 1986; Seligman and Kirmayer, 2008: 34; Christoff et al., 2011; Schooler et al., 2011; Gerrans, 2014; Andrews-Hanna et al., 2014; and Smallwood et al., 2015). Furthermore, it has been shown that such sensory stimuli as visual, auditory, tactile, gustatory, olfactory, or kinaesthetic inputs, have an antithetical effect on mind-wandering activity (Klinger, 1971; and Wolmann et al., 1986).

In terms of content, mind-wandering episodes, just like oneiric activity, are frequently shaped by autobiographical preoccupations, are rarely related to immediate circumstantial contexts, and are marked by the unambiguous absence of meta-cognitive awareness (Killingsworth et al., 2010; and Schooler et al., 2011; Stawarczyk et al., 2011). These observations have lead researchers to posit that mind-wandering and oneiric activity may be generated by the same underlying architectural structure. Such a framework implies that mind-wandering episodes could be placed on the same "phenomenological continuum" as oneiric activity (Klinger, 1971; Domhoff, 2003; Llewellyn, 2009; Nir et al., 2010; Christoff et al., 2011; Domhoff, 2011; Cipolli et al., 2011; Smallwood et al., 2015; and Domhoff and Fox, 2015). In the absence of a readily recognizable term to refer to this "continuum", this work introduces the term "imagioneiric" (mixing imagination from the Latin "imaginari" [to picture to oneself,] and oneiric from the Greek "oneiros" [to dream]) to flag that both phenomena may have similar phenomenological origins. Throughout the rest of this work, I use the term "imagioneiric" to refer to the collective content, structure, and phenomenology of both oneiric and mind-wandering activity.

Current research continues to show that imagioneirc activity is neither stable, nor homogenous. Its content, intensity, and degree of absorption fluctuate based on the neurophysiological context in which it is produced, i.e., wakefulness, REM sleep, NREM sleep 2, NREM sleep 3, etc (Fox et al., 2013; Hobson, 2015: 199; Domhoff, 2015; and Cipolli et al., 2017). Put differently, different neurophysiological states either enable or accentuate different facets, elements, or characteristics of the imagioneirc continuum. As such, oneiric mentations can be conceptualized as more immersive forms of mind-wandering activity, whereas mind-wandering mentations can be conceptualized as either "less-immersive" forms of dreaming or as a form of "dreaming" that is more susceptible to external sensory information and motor output (Wolmann et al., 1986; Domhoff, 2003 & 2011; Fox et al., 2013; and Domhoff and Fox, 2015). In terms of underlying core features, imagioneirc mentations share the ability to generate multi-modal, stimulus-independent, virtual environments that reversely correlate with the degree to which sensory, cognitive, and motor decoupling has taken place. In other words, deeper levels of sensorial, cognitive, and motor decoupling will generate proportionally more immersive virtual realities (Gerrans, 2013). In order for this to occur, imagioneirc mentations must be generated by a network that is active during both wakefulness *and* sleep.

A functional network that partially meets these criteria was proposed in 2004, the "default-mode-network" (DMN) (Morcom and Fletcher, 2007; Andrews-Hanna et al., 2014; Gerrans, 2014b; and Smallwood et al., 2015). The DMN was discovered via nuclear imaging studies, and it was found

to correlate with episodes of mind-wandering activity and both periods of waking restfulness and sensorial-perceptual decoupling (Morcom and Fletcher, 2007; Morcom and Fletcher, 2007; and Scheibner et al., 2017). Because of this, many researchers now hypothesize that the DMN could be the neural correlate of mind-wandering activity (Christoff et al., 2011; Domhoff, 2011; Andrews-Hanna et al., 2014; Domhoff and Fox, 2015; Vatansever et al., 2015; and Davey, et al., 2016).

A number of positron emissions tomography (PET) and functional magnetic resonance imaging (fMRI) studies support this hypothesis by showing a substantial overlap between DMN activity and periods of daydreaming (Raichle and Snyder, 2009; and Scheibner et al., 2017). Specifically, DMN activity appears to correlate with the demodulation of sensory cortices through the ventral/dorsal medial prefrontal cortex (mPFC) (an area that is mostly confined to the Bromann Area 10). Additionally, activity in the posterior cingular cortex (PCC), the lingual gyrus (located in the medial occipital lobe), the caudate nucleus, the amygdala, hippocampal formation (hippocampus and parahippocampal cortex), and the inferior parietal lobule (IPL) also appears to be demodulated (Christoff et al., 2011; Smallwood et al., 2015; Domhoff 2011 & 2015; Scheibner et al., 2017; and Pace-Schott et al., 2017). Knowing this, Domhoff and Fox have speculated that mind-wandering activity may be produced through the functional disconnection of the parietal and prefrontal structures, and the decoupling of affective somatosensory information processing (Domhoff 2011; and Domhoff and Fox, 2015).

This framework suggests that if mind-wandering mentations are indeed produced through the same mechanism that produces oneiric activity, as Mahowald et al., (1998) and Domhoff (2011) speculate, then both phenomena may invariably be by-products of DMN connectivity, or at the very least, by-products of a subsystem of the DMN (Zellner, 2013; and Scheibner et al., 2017). As Domhoff and Fox (2015) write, if the DMN "becomes ascendant whenever there is a decline in vigilance and a loss of volitional control, then there is reason to believe that people can indeed drift into dreaming during periods of relaxed wakefulness and mind-wandering". In other words, it is possible that the phenomenological, cognitive, and perceptual similarities between oneiric and mind-wandering activity exist because both phenomena are produced through the same sensory/cognitive/motor decoupling process. Increasing evidence appears to support this claim. For example, Zellner (2013) explains that lesions in areas that are not normally associated with the DMN (e.g., sensorimotor areas) do not disrupt the production of oneiric mentations, whereas lesions in areas that are associated with the DMN (the occipital lobe, ventral medial pre-frontal cortex) do. Additionally, components of the DMN are believed to be active during multiple sleep stages (Gerrans, 2014b; Domhoff and Fox, 2015; and Windt, et al., 2016).

By conducting PET and fMRI studies, Fox et al., (2013) discovered that multiple areas of the DMN are active during REM sleep. These include the medial temporal lobe structures, posterior cingulate, and the medial prefrontal cortex (PFC). Moreover, DMT connectivity also appears to be partially preserved during NREM sleep. As Waters et al., (2016) explain, "Connectivity to the Dorsal Attention Network remains high during NREM, but connectivity to the Central Executive Network (CEN) is slightly reduced". Put differently, the preliminary data appears to support the claim that (1) imagioneiric activity is produced by the same underlying structure; (2) that structure is preserved across multiple arousal and sleep stages, and (3) the structure in question could (potentially) be the DMN network (or parts of the DMN network). That said, these claims also suffer from a number of severe limitations.

DMN connectivity is not entirely preserved throughout all stages of sleep. The inferior parietal cortices, for example, remain largely inactive during REM sleep whereas medial prefrontal cortex decoupling and reductions in frontoparietal correlations are observed during NREM sleep (Horovitz et al., 2009; Nir et al., 2010; and Spoormaker et al., 2012). In fact, most of the DMN remains disconnected during NREM sleep altogether (Koike et al., 2011; Zellner, 2013; and Pace-Schott et al., 2017). Other differences between DMN, mind-wandering activity, and dreaming include a deeper quiescence of the prefrontal cortex (PFC) during sleep and an overall diminishment in meta-cognitive awareness (Fox et al., 2013). Interestingly, because meta-cognitive tasks (external or internal) appear to reduce DMN connectivity, it has been suggested that a distinction should be made between perceptual and cognitive decoupling (Scheibner et al., 2017). In fact, controlled mind-wandering activity (i.e., mind-wandering episodes in which meta-awareness is preserved) appears to be antithetical to DMN connectivity (Scheibner et al., 2017). This means that not all forms of mind-wandering mentations are reliant on the presence of DMN activity. Additionally, mind-wandering episodes have been shown to recruit multiple non-DMN brain regions, including the dorsolateral prefrontal cortex, dorsal anterior cingulate cortex, temporal cortex, Broadmann area 38, the lingual gyrus, and secondary somatosensory cortex (Fox et al., 2015). These findings have lead researchers to conclude that "DMN activation alone is insufficient to adequately capture the neural basis of spontaneous thought" (Fox et al., 2015).

As things currently stand, it is still premature to declare a one-to-one correlation between imagioneirc mentations and DMN activity. In fact, it is becoming increasingly clear that imagioneirc activity recruits multiple non-DMN areas (Koike et al., 2011; Zellner, 2013; Pace-Schott et al., 2017; and Cipolli et al., 2017). Because of this, the only thing that can hitherto be stated with confidence is that DMN activity may be partially involved in the generation of imagioneirc mentations (Cipolli et

al., 2017). Nevertheless, the current neurophenomenological data indicates that both mind-wandering and oneiric activity are products of exceedingly similar sensorial, perceptual and cognitive decoupling neurophysiological processes. This suggests that a neurophysiological system of patterned sensorial, cognitive, and motor deactivation likely underlies both activities. More importantly, the current neurophenomenological data suggests that both mind-wandering and oneiric activity are expressions of an underlying neurophysiological process that can be active throughout multiple sleep stages *and* wakefulness. Whether or not the neurophysiological dynamics of this process is generated by the DMN or subsections of the network remains to be seen.

Oneiric Activity and Schizophrenia

The primary reason why REM sleep intrusion models of schizophrenia have not generated the expected empirical results, is not because their underlying premise was incorrect. (It is true that the perceptual, phenomenological, and cognitive features of schizophrenia are remarkably similar to oneiric activity). Rather, REM sleep hypotheses failed because they did not capture the entire neurophysiological spectrum involved in the production of oneiric activity. As the preceding summary demonstrates, oneiric mentations can be generated in a plurality of neurophysiological settings and can even extend into wakefulness during periods of mind-wandering. In retrospect, searching for the neurophysiological traces of REM sleep or NREM sleep in people who suffer from schizophrenia was a misguided effort because the characteristic features of specific sleep stages are not the neural correlates of oneiric activity.

Schizophrenia is Not a Homogenous Diseases Entity

A secondary impediment to oneiric formulations of schizophrenia comes not from the field of dream research, but from nosological discrepancies within the field of psychiatry itself. Specifically, dream researchers have incorrectly assumed that schizophrenia is a single, unitary, and homogenous disorder. A close examination of the psychiatric literature reveals that schizophrenia was never intended to be conceptualized a singular disease entity, but as a nebulous cloud of heterogeneous symptoms (Bleuler, 1966: 8; Andreasen, 2011; McNally, 2016: 3, 24, 40 & 41; and Benson and Feinberg, 2016). The official definition of schizophrenia has remained in constant flux throughout most of the twentieth and twenty-first centuries. Indeed, its definition has varied even within the same research institutes (Gone and Kirmayer, 2010: 88 & 90; Gold and Gold, 2014: 52; McNally, 2016: 4, 6, 7, 8, 36 & 106; and Lewis-Fernández et al., 2017: 2). Moreover, schizophrenia does not have established biomarkers, neurophysiological correlates, or objective diagnostic criteria. The

disorder is diagnosed on the basis of verbal reports and vague behavioural patterns. Thus, it remains unclear what exact psychiatric condition, or "psychiatric phenomena", previous oneiric formulations of schizophrenia targeted in their models.

The nosological roots of schizophrenia can be traced back to the late nineteenth century. In particular, Emil Kraepelin noted symptomatological similarities in a group of patients who exhibited marked cognitive decline, disorientation, auditory hallucinations, loosening of associations, difficulty of speech, delusional thinking, absence of emotions, defective judgment, and impairment of voluntary attention, among other symptoms (Kraepelin, 1915: 222-225). Kraepelin then went on to established that the same cloud of patterned heterogeneous symptoms had been reported across clinics. In 1893 he decided to name the group of symptoms "Dementia Praecox" (Stone, 2006). His formulation of dementia praecox drew on his own clinical experiences, Bénédict Morel's (1809-1873) notion of *démence précoce,* introduced in *Études cliniques* (1852) and further expounded on in *Traité des maladies mentales* (1860), Ewald Hecker's (1843–1909) formulation of "hebephrenia", and Karl Ludwig Kahlbaum's (1828–1899) conceptualization of "catatonia" (Thiher, 2004: 232; and Stone, 2006). Indeed, in order to identify the disorder, Kraepelin had to synthesize a number of previously unassociated symptoms and psychiatric conditions into a single taxonomical entity.

In the seventh edition of *Clinical Psychiatry* (1915), Kraepelin wrote that "dementia praecox" was a provisional name for, "a large group of cases which are characterized in common by a pronounced tendency to mental deterioration of varying grades" (Kraepelin, 2002: 23). He further divided the condition into three broad subtypes, known as: (1) hebephrenia; (2) catatonia, and (3) paranoid conditions, and at least 53 individual cognitive, perceptual and behavioural conditions (Kraepelin 1915: 230; and McNally, 2016: 4 & 90). This classificatory scheme is not entirely dissimilar to how cancer (a single conceptual disorder) is currently subdivided into more than one-hundred different malignant tumour types (each with their own unique features and properties).

Kraepelin's hebephrenic forms were characterized by mental deterioration, temporary delusions, emotional indifference, hypochondria, depression, visions of the deceased, olfactory hallucinations, and other forms of delusional thinking (Kraepelin, 1915: 231, 232, 233 & 257). The catatonic forms of dementia praecox were also marked by mental deterioration, but included religious delusions, negativism, hyper-suggestibility, uniform muscular tension, and impulsiveness (Kraepelin, 1915: 241, 243 & 257). The paranoid forms of the disorder were the only group not marked by mental deterioration. Instead, they were characterized by manic-depressive episodes, delusions, and brief episodes of psychosis (Cohen, 2008: 208; and Kraepelin, 1915: 233). Yet these subdivisions drew criticism. They were clearly ill-defined, had multiple overlapping symptoms, and

were not the product of a rigid nosology —a fact that Kraepelin himself openly acknowledged (Kraepelin, 2002: 23).

A rigorous attempt to rectify the problem was made by Swiss psychiatrist, Eugene Bleuler (1857-1939), who argued that since not all cases of dementia praecox are deteriorative they should not be all categorized as "dementias" (Noll, 1992: xxii; Cohen, 2008: 210; and Andreasen, 2011). Bleuler went on to write an influential book on the subject matter, *Dementia Praecox or the Group of Schizophrenias* (1911), which amounted to a rigorous effort to refine, update, and expand Kraepelin's nosological entity (Bleuler, 1966: 7; McNally, 2016: 23). Bleuler coined the term "the schizophrenias" (with an "s") to capture the diverse symptomatological spectrum of Kraepelin's Dementia Praecox and proposed it to describe a patterned group psychoses, various forms of behavioural, cognitive, emotional and perceptual disturbances, and to convey the general idea of a "splitting of the mind" ("*skhizein*" is Greek for "splitting", whereas "*phren*" is Greek for the mind) (Bleuler, 1966: 8; Andreasen, 2011; McNally, 2016: 3, 24, 40 & 41; and Benson and Feinberg, 2016). Indeed, Bleuler's reformulation of Dementia Praecox was not intended to signify a single nosological entity.

Briefly, Bleuler divided the symptomatological characteristics of the various schizophrenias into two major groups; Fundamental and Accessory Symptoms. On the one hand, Bleuler's Fundamental Symptoms were further divisible into four subtypes: paranoid, hebephrenia, catatonic, and schizophrenia simplex (Bleuler, 1966: 227; and Jablensky, 2010). Additionally, each Fundamental subtype was believed to manifest in conjunction with at least one of four cognitive abnormalities, namely, Loss of Association, Loss of Affect, Ambivalence, and Autism (Cohen, 2008: 210; and Bleuler, 1966: 14; 40, 53 & 63). Accessory Symptoms, on the other hand, were presented as by-products of the four Fundamental subtypes, i.e., they were not seen as disorders themselves, but as "side effects". Bleuler's core Accessory Symptoms included both waking hallucinations and delusional thinking (Andreasen, 2011; and Bleuler, 1966: 10).

Mapping out the full taxonomy of the schizophrenias proved to be an insurmountable challenge. In fact, Bleuler realized early on that there were simply too many variations of the condition to integrate all of them into a single symptomatological spectrum (Bleuler, 1966: 8). To make matters worse, the plural name of the condition was linguistically awkward and ultimately proved unpopular among clinicians. Because of this, Bleuler deliberately dropped the "s" from the name (Bleuler, 1966: 8). A name change that unsurprisingly created additional confusion. Specifically, it fostered the misconstrued notion that "schizophrenia" is a single disease entity (McNally, 2016: 42). To further muddy the waters, by the 1920s, both Bleuler's "schizophrenia" and Kraepelin's "Dementia Praecox" were used interchangeably, and remained in concomitant use until "Dementia

Praecox" became obsolete in the late 1950s (McNally, 2016: 41, 47, 49, 50, 51, 53, 54 & 93). By this time, the symptomatological spectrum of both conditions had become nearly impossible to differentiate.

This meant that two supposedly "provisional" disease entities (neither Kraepelin, nor Bleuler intended their nosological entities as more than a "temporary place holder") became amalgamated into a singular disease entity (McNally, 2016: 28, 41, 49, & 54). This prompted such prominent psychiatrists as William Alanson White (1870-1937) and Richard Bentall (1956-) to derogatorily describe schizophrenia as an "imprecise waste-basket" or "a ghost within the body of psychiatry" (Greene, 2007; and McNally, 2016: 48 & 90). In 1972, Bleuler's son, Manfred, concluded that "one is impressed with the variability of [schizophrenia], more than with [its] consistency" (McNally, 2016: 95). By the end of twentieth century, "schizophrenia" had become a catch-all term for uncategorizable mental disorders and was mostly used as a default category for unclassifiable patients (Greene, 2007; and McNally, 2016: 43, 48, 50 & 90). Indeed, researchers were becoming increasingly concerned that schizophrenia had no clear taxonomical boundary, no precise nosology, and no objective diagnostic criteria (Kandell 1987: 511; Claridge, 1997; Os et al., 1999; Gold and Gold, 2014: 49; and McNally, 2016: 96). To this day, the symptomatology of schizophrenia continues to be defined by its heterogeneity, rather than its homogeneity (Arango and Carpenter, 2011).

It is now well recognized within the field of psychiatry that schizophrenia is not a singular disease entity, but a plurality of associative syndromes. Broadly speaking, the disorder is now defined as a general "loosening of associative thought" or an alteration in thinking patterns caused by the collapse of normal perceptual-cognitive mechanisms (Claridge 1997; Arango et al., 2011; and Bob and Mashour, 2011). These alterations are believed to deform the unitary, sensorially-congruous, cognitive systems of wakefulness, and indirectly contribute to the formation of discordant perception and such disconsonant internal narratives as delusional thinking and hallucinatory perception (Claridge 1997; Arango et al., 2011; and Bob and Mashour, 2011; Arango and Carpenter, 2011; and David et al., 2014).

Behaviourally, people who suffer from schizophrenia display an inability to read social cues, relate to social activities, effectively plan for future events, distinguish between internal and external narratives, express their emotions, and effectively organize their internal thinking (Cohen, 2008: 207; and Andreasen 2011). On a cognitive level, schizophrenia is characterized by a loss of insight, a drastic reduction in meta-awareness, impaired reality testing, single-mindness, deficits in self-awareness, a reduction in abstract thinking, and ineffective self-reflection (Amador et al, 1991;

Amador, 1994; D'Agostino et al., 2013c; Gerrans, 2014, and Dresler et al., 2015). Intriguingly, these meta-cognitive impairments mean that more than 70% of people who suffer from schizophrenia are unaware that they suffer from the condition (Lincoln, 2006; Dresler et al., 2014; and David et al., 2014).

Schizophrenia induces oscillating, independent, or combinatory fragmentations in each and every cognitive, sensory, and perceptual system (Claridge, 1997; and Arango and Carpenter, 2011). As such, the symptomatology of the condition can vary extraordinarily on an intra, inter, and cross-individual level (Gone and Kirmayer, 2010: 88 & 90; Andreasen, 2011; Gold and Gold, 2014: 52; McNally, 2016: 6 & 7; and Lewis-Fernández et al., 2017: 2). Because of this, the exact definition, taxonomy, and nosological structure of schizophrenia remains evasive (McNally, 2016: 4, 8, 36 & 106).

Section II Summary

This section has identified two existing limitations of previous and current oneiric formulations of schizophrenia. First, dream intrusion models of schizophrenia have incorrectly assumed that either REM sleep or a specific NREM stage of sleep is the neurophysiological basis of oneiric activity. Second, oneiric formulations of schizophrenia have wrongly approached schizophrenia as a singular disease entity. Both of these limitations have been systematically overlooked by all major oneiric formulations of schizophrenia, so it should come as no surprise that these models have produced close to no meaningful empirical results. Nevertheless, the insightful observation by such nineteenth century researchers as Hughlings-Jackson still remains true today: oneiric activity bears a close similarity to psychotic symptoms. Indeed, upon closer examination, it becomes clear that oneiric activity shares a number of remarkable phenomenological characteristics with a specific sub-group of schizophrenia symptoms, namely, the "psychotic symptoms" of the disorder. These symptoms include delusional thinking, waking hallucinations, and psychosis (Arango and Carpenter, 2011; and McNally, 2016: 65). The next section will compare and contrast these three psychotic symptoms of schizophrenia with the phenomenological characteristics of oneiric activity to demonstrate that they display sufficient overlaps to provide evidence for their similar phenomenological origins.

PART TWO

PART TWO INTRODUCTION

The second part of this work builds towards proposing a new oneiric formulation of schizophrenia. The first section (Section III) presents a detailed cross-examination of the phenomenological characteristics of schizophrenia and oneiric activity. The second section (Section IV) offers a detailed analysis of such hybrid-states-of-awareness as lucid dreaming, sleep paralysis, and somnambulism to show that the combinatory elements of oneiric activity, sleep, and wakefulness, can become imbricated to form new perceptual states. Moreover, Section IV also argues that the psychotic symptoms of schizophrenia, namely, hallucinations, delusions, and psychosis, can also be conceptualized as hybrid-states-of-awareness. The last section (Section V) proposes an underlying framework that accounts for the hybridization process, Oneiric Release Theory (ORT).

SECTION III

AN OVERVIEW OF THE PSYCHOTIC SYMPTOMS OF SCHIZOPHRENIA AND ONEIRIC ACTIVITY

Section III Introduction

This section provides a detailed cross-examination of the phenomenological features of oneiric activity and the psychotic symptoms of schizophrenia. The symptomatological spectrum of schizophrenia is generally divided into "positive" and "negative" symptoms. While the two terms only became commonplace in psychiatry in the late 1980s, they were first proposed by Hughlings-Jackson at the end of the nineteenth century (Arango and Carpenter, 2011; and McNally, 2016: 65). Hughlings-Jackson posited that the "positive symptoms" of schizophrenia, namely perceptual alterations, delusions, and psychotic states, are caused by an "enhancement" or "overclocking" of normal cognitive and sensory processes, whereas the "negative symptoms" (i.e., such cognitive and behavioural deficiencies as lethargy and apathy), are produced by a reduction in normal cognitive processes (Arango and Carpenter, 2011; and Andreasen, 2011).[4]

The section begins by first outlining the neurophenomenological features of hallucinations, delusions, psychosis, and oneiric activity before proceeding to cross-examine their similarities. The cross-examination allows this section to argue that the psychotic symptoms of schizophrenia are most similar to the content, structure, and features of oneiric activity and suggests that oneiric formulations of schizophrenia should target these particular symptoms instead of the entire disorder. Moreover, this section also presents an overview of contemporary "virtual-reality" theories of dreaming. The collective insight of these theories is used in the fifth section of this work to formulate some of the conceptual elements of Oneiric Release Theory (ORT).

Hallucinations

The word "hallucination" first appears in English literature in the late sixteenth century, when it was originally used to refer to "mind-wandering" activity (from the Latin *alucinatus*, and the Greek *alyein* i.e., "to wander in mind"). At that time, the experiences that are normally associated with the word "hallucinations" (as the term is understood today, namely seeing "something that is not there") would instead have been called "apparitions" (Corballis 2014: 128). The term "hallucination" was first employed, in a psychiatric sense, by Jean-Étienne Esquirol (1774-1840) in *Des Maladies Mentales* (1838) (Corballis 2014: 128). In this textbook, the French psychiatrist differentiated the phenomenology of "illusions" from that of "hallucinations" (Arango et al., 2011). Illusions, he

[4] Hughlings-Jackson's original definition of the "positive symptoms of schizophrenia" (i.e., perceptual alterations, delusions, and psychotic states) are now generally referred to as the "psychotic symptoms" of schizophrenia (i.e., hallucinations, delusions, and psychosis). This is because the former term, namely, "the positive symptoms of schizophrenia", is no longer used as it was originally intended by Hughlings-Jackson. In addition to the psychotic symptoms of the disorder, the "positive symptoms of schizophrenia" now also include, confused thinking, disorganized speech, difficulties in concentration, and catatonic behaviour (i.e., movement disorders).

argued, are misinterpretations of real events, whereas hallucinations are a form of "objectless perception" (Arango et al., 2011; and Goldsworthy and Whitaker 2015). Today, just as Esquirol had proposed, hallucinations are defined as sensory experiences that are produced in the absence of external sensory input (West, 1975: 288; Assad, 1986; Campbell, 1989; Henry, 2009; Arango et al., 2011; Corballis, 2014: 128; Goldsworthy and Whitaker, 2015; and Blom, 2015b). It should be noted that by this definition, dreaming, hypnagogia, and even mind-wandering activity would equally qualify as "hallucinations" (West, 1975: 288). Yet, in its psychiatric usage, the term deliberately excludes these three phenomena (Kraepelin, 1915: 4; and Rosenhan, 1973). As such, a "hallucination" is currently defined as an objectless sensory perception that occurs outside of the context of dreaming, hypnagogia, or mind-wandering activity.

Objectless perceptions can occur in all sensory modalities, not only auditory, olfactory, gustatory, and visual, but also the somatosensory (proprioceptive, tactile, and visceral), kinaesthetic, and vestibular modalities (Kraepelin, 1915: 262; Assad, 1986; and Goldsworthy and Whitaker, 2015). Hallucinatory experiences are exceedingly common. More than 40% of the general population has experienced a hallucinatory experience, and at least 5% of individuals experience them on a regular basis (Mahowald et al., 1998; Waters, 2014; and Goldsworthy and Whitaker 2015). Within the context of schizophrenia, more than 75% of people who have been diagnosed with the condition report experiencing them (Waters, 2014). It has been traditionally assumed that people who suffer from schizophrenia experience most of their hallucinations in auditory form, but recent data indicates that the overwhelming majority of people who suffer from schizophrenia report experiencing hallucinations that also include other sensory systems (Waters et al., 2016; and Lim, et al., 2016).

When experienced outside the confinements of mental illness, hallucinations tend to be quickly identified as "imposter" sensorial cues, uncorroborated by the rest of external reality (Kraepelin, 1915: 3-4 & 9; and Sanati, 2012). When hallucinations occur within the context of schizophrenia or other psychiatric conditions, however, they are more easily mistaken for real sensory data (Bleuler, 1966: 95). This suggests that hallucinations, *per se*, do not cause a break with reality. Rather, "breaks in reality" are caused by such concomitant cognitive impairments as reductions in meta-awareness, loss of insight, and impaired reality testing (D'Agostino et al., 2013b and 2013c; Gerrans, 2014a, Amador et al, 1991; and Dresler et al., 2015).

Hallucinations occur more frequently during the morning and evening period, and they often become less noticeable, or even entirely absent, during the afternoon (Kraepelin, 1915: 11). The content of hallucinations exhibits great variability too. Auditory hallucinations, for example, can be

experienced as threatening, commanding, soothing, or comforting human voices, as well as animal sounds and music (Bleuler, 1966: 95 & 96; Manford, 1998; Arango and Carpenter, 2011; Waters, 2014; and Goldsworthy and Whitaker, 2015).

On a phenomenological level, hallucinations are perceived as fully embedded in the external world (Corballis 2014: 129; and Blom, 2015b). For instance, they are experienced as emanating from outside of the body and alongside normal perception (Bleuler, 1966: 95 & 96; Arango and Carpenter, 2011; Waters, 2014; and Goldsworthy and Whitaker, 2015). Hallucinated images, figures, and objects are seamlessly integrated with correctly modulated sensory input and respect the spatio-temporal boundaries of the physical environment. They can produce realistic noises and appear to act as if by their own volition (Bleuler, 1966: 104; Corballis 2014: 129; Blom, 2015b; Waters et al., 2016).

Psychosis

Psychosis is generally defined as a temporary "break with reality", during which a mixture of hallucinations, delusions and paranoid thinking occurs (Henry, 2009). On a cognitive level, the condition is marked by a significant reduction in "self-reflective", "meta-cognitive", and "strategic metacognitive function" (Quee et al., 2010; David et al., 2014; and Dresler et al., 2015). Psychosis can occur in multiple affective pathologies and in such non-pathological conditions as bi-polar disorder; organic brain syndromes (e.g., Alzheimer's), cases of substances abuse, consumptions of psychedelics, metabolic and neurological disorders, and schizophrenia (Henry 2009; and Limosani et al., 2011).

The severity of psychotic symptoms as well as the long term psychosocial impact of the condition, directly correlate with the degree of impaired insight (Amador 1994; and David et al., 2014). Insight is a multi-faceted structural system with different possible breakage points that can be divided into at least three general categories: (1) recognition of psychosis; (2) recognition of hallucinations and delusions as abnormal mental phenomena; and (3) recognition of the need for medical treatment (Quee et al., 2010; and David et al., 2014).

On a phenomenological level, psychosis —given its element of fantasy— is most comparable to a dream state. It is marked by a breakdown between external and internal cognitive sensory feedback structures (Rechtschaffen, 1978; and Limosani et al., 2011). Mood swings and mood-incongruent delusions, in addition to multi-sensorial hallucinations and disorganized thinking patterns, are commonly reported during these episodes. Psychoses can be triggered by any number of pharmaceutical, cultural, psychological, social, genetic, or historic circumstances.

Delusions

Delusional thinking has been associated with a wide range of psychopathological conditions (Gold and Gold, 2014: 59). In fact, over seventy different conditions, many caused by brain and genetic disorders, can lead to the development of delusional thinking (Gold and Gold, 2014: 59). Nevertheless, delusions, just like hallucinations, are not exclusively restricted to the domain of the psychopathological, and can readily occur in healthy individuals (Gold and Gold, 2014: 60).

Delusions are primarily defined as non-amenable fixed beliefs that persist in spite of overwhelming evidence against them (Henry, 2009). Delusions are also characterized as "bizarre", in the sense that they either don't conform to accepted cultural, social and educational narratives, or contravene the most basic standard of logic (Henry, 2009; and Arango et al., 2011). Within the context of schizophrenia, delusions have been argued to develop in response to previous trauma, or in an effort to rationalize the content of such hallucinatory experiences as hearing disembodied voices or seeing things, objects, or people that are not there (Arango and Carpenter, 2011; and Gerrans 2013).

Delusional thinking can also arise within the context of non-hallucinatory perceptions and in the absence of trauma (Arango et al., 2011). This implies that they can be generated in heterogeneous psychological contexts. People who suffer from delusional thinking are unable to detect flaws in their logic (even when these flaws are pointed out to them) and can go to great lengths to defend their delusional narrative. For example, when the consistency of their delusional narrative starts to breakdown, they often resort to including an ever increasing number of fictional co-conspirators, including family members, friends, social workers, or medical staff (Kraepelin, 1915: 257; Bleuler, 1966: 119; and Henry, 2009). Intriguingly, people suffering from delusional thinking are usually able to correctly identify delusional narratives and logical inconsistencies in other individuals (Kraepelin 1915: 259; Bleuler, 1966: 125 & 126; and Gerrans, 2014a).

The content of delusions can vary immensely and tends to be adapted to an individual's history, cultural background, fears, desires, and psychological predispositions (Bleuler, 1966: 117; Arango and Carpenter, 2011; and Gold and Gold, 2014: 59 & 63). Yet the overarching structure of delusional thinking (delusional forms), display remarkable intra-individual, cross-cultural, and historic stability (Gold and Gold, 2014: 65 & 66). The most common delusional form is the "persecutory" or "paranoid" type. This type of delusion is characterized by the irrational conviction that one is being followed, monitored, or surveilled by a group of people, institutes, organization, or supernatural entities (Kraepelin, 1915: 257; Henry, 2009; Arango et al., 2011; and Gold and Gold,

2014: 64). Persecutory delusions are incredibly malleable to an individual's autobiographical circumstances and tend to reflect immediate psychological contexts.

Less common delusional forms include: (1) delusions of influence and control, i.e., the conviction that one's thoughts, actions, and emotions are being controlled by such external forces as aliens, magic, or hypnosis; (2) delusions of thought withdrawal or insertion; (3) delusions of thought broadcasting; (4) such delusions of misidentification as the (a) Fregoli and (b) Capgras syndrome, (c) delusions of grandeur, (d) religious delusions; (5) cotard delusions, i.e., conviction that one is (i) already dead, or (ii) has had their organs replaced, or stolen; and (6) such somatic delusions as hypochondria (Arango and Carpenter, 2011; and Gold and Gold, 2014: 59, 60, 62 & 63). Capgras and Fregoli delusions are especially peculiar. In the former case, individuals become convinced that their spouse, family, and/or friends have been replaced by indistinguishable imposters, whereas in the latter case, they believe that different individuals are in fact a single entity that can manifest itself in different forms or "disguises" (Gerrans, 2014a).

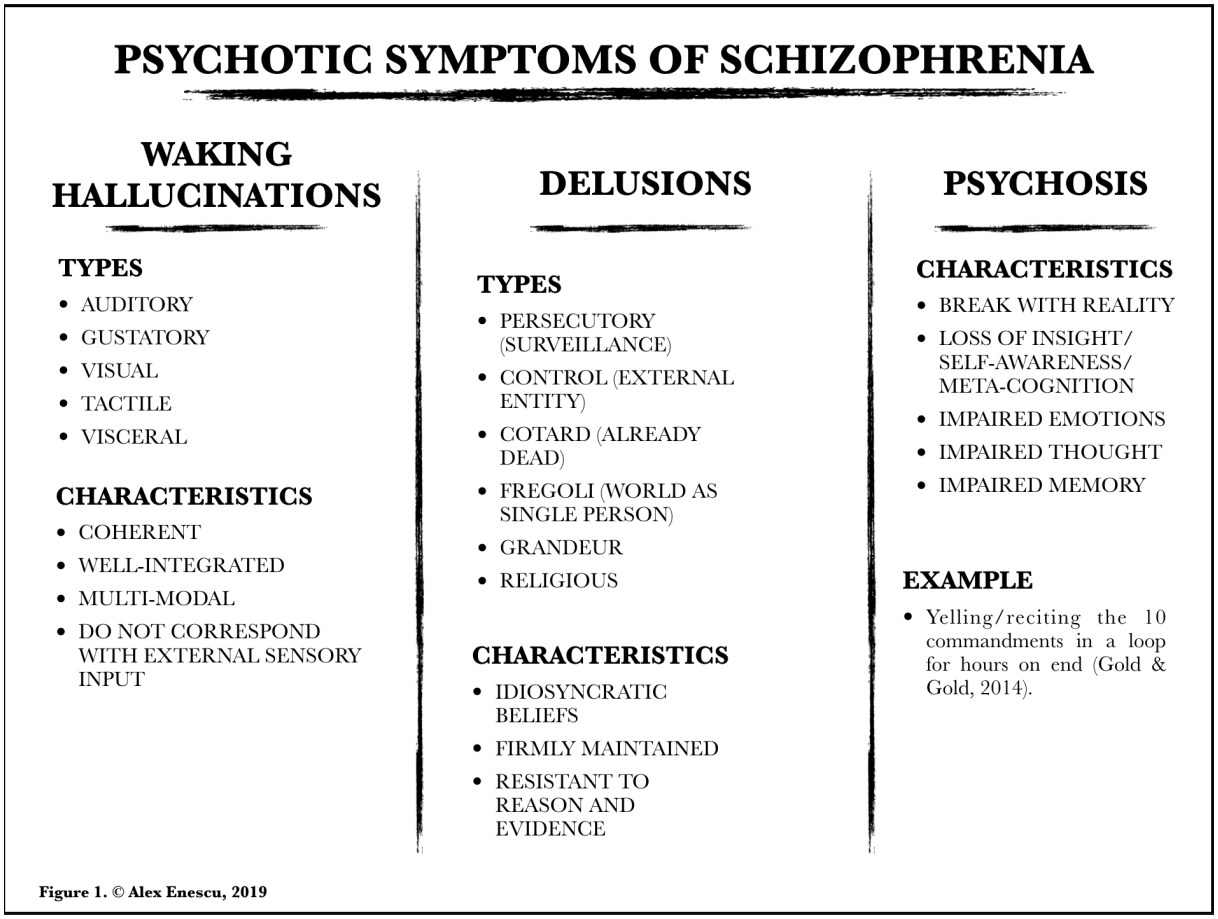


Figure 1. © Alex Enescu, 2019

It merits noting here that the line between delusional narratives, religious beliefs, and other dominant cultural narratives is thin and difficult to delineate. For example, the conviction that the universe is a "computer simulation", which is an increasingly popular idea among silicon valley elites, would not qualify as a delusion. Indeed, what may qualify as a delusional belief in one historic period, may be orthodoxy in another social context. For example, believing that Thor is directly monitoring your mental activity would qualify as a delusional narrative in today's cultural setting, whereas, believing that the Jewish, Christian, or Muslim deity is engaged in the same activity would not (Gold and Gold, 2014: 66).

Nosological Considerations

Culture plays an undeniable role in the nosology and overall diagnosis of mental illness. Diagnosis criteria are not "cognitively innocent" activities (Canino et al., 1997: 172; Berrios and Elias, 2002; and Gone and Kirmayer, 2010: 83 & 90). Psychiatric diagnoses are shaped by prevailing socio-cultural presuppositions, overarching cultural narratives, and individual biases (Berios and Elias, 2002: 2; Thiher, 2004: 233; Kirmayer, 2007: 11; Gone and Kirmayer, 2010: 80; and Kirmayer and Ban, 2013: 107). For example, the psychiatric disorder known as "drapetomania", which was coined in 1851 by Samuel Cartwright (1793-1863) and defined as: "An ailment that causes a slave to be possessed by a desire for freedom and want to escape", is racist and in violation of modern conceptions of inalienable human rights (Jarvis, 2008: 230). The same can be said about homosexuality, which was only removed from the Diagnostic and Statistics Manual (DSM) in 1974 —and only by a marginal vote. Moreover, perceptual experiences that are unanimously considered as "abnormal" in today's context, such as objectless perceptions and hearing voices, have been alternatively praised as messages from the gods or as signs of spiritual enlightenment (Pagel 2014; Corballis 2014; and Lewis-Fernández et al., 2017: 21). Systematic transcultural studies reveal to just what extent the boundary between the "normal" and the "psychopathological" can be shaped and determined by dominant cultural narratives and the degree to which this dichotomy exhibits great cross-cultural variability.

In contemporary Western societies, psychiatric diagnoses are issued based on loosely defined syndromes that can vary between diagnostic manuals, clinics, and individuals psychiatrists. They remain susceptible to personal opinions, prejudice, political, historical and dominant cultural narratives (Gone and Kirmayer, 2010: 88 & 90; and Lewis-Fernández et al., 2017: 2). The lack of viable bio-physiological markers, the cross-individual heterogeneity of psychopathology disorders, the imprecision of psychiatric nosologies, and the unclear boundary between "normal" versus

"abnormal" mental experiences, pose a great challenge to the field of psychiatry as a whole (Canino et al., 1997: 172).

More recent diagnostic manuals take these facts into consideration. The term "mental illness" is now exclusively reserved for debilitating expressions of psychiatric symptoms instead of psychiatric forms in general (Amador et al., 1991; and Nordenfelt 1997). Hallucinations and delusions, for example, are only labeled as "pathological" *if* they directly interfere with an individual's work performance, lifestyle, or emotional stability (Amador et al., 1991; and Nordenfelt, 1997). Psychopathology, in general, is now increasingly seen as an exaggeration of normal mental functions channeled, shaped, and chiseled by individual psychology, cultural variations, and historic contexts (Canino et al., 1997: 171 & 172; Kirmayer, 2007: 11; Gone and Kirmayer, 2010: 80; Gold and Gold, 2014: 132; Arango et al., 2011; and Kirmayer et al., 2015: 1). This means that the content, classification, and social tolerance for psychiatric conditions will continue to change over time.

Nevertheless, universal structured patterns of "abnormal modes of behaviour" have been systematically identified in cross-cultural settings, diverse demographies, and a plurality of historical contexts. There is consensus that the psychotic symptoms of schizophrenia (i.e., delusional thinking, hallucinatory perception, and psychosis) are deviating forms of mental activity in heterogeneous human environments (Berrios and Elias, 2002: 2 & 11). Indeed, these three psychotic symptoms are so dissimilar to how the majority of people experience reality that they warrant their designation as "abnormal mental states". The cross-cultural similarities of psychotic symptoms strongly suggest that such a transcultural neurophysiology as the one that generates oneiric activity can likely form the substrate of these three phenomena.

Defining Oneiric Activity

The traditional definition of hallucinations (i.e., sensory experiences produced in the absence of sensory input) also applies to oneiric mentations (Keep, 1970; West, 1975: 288; Hartmann, 1975; Assad 1986; Campbell 1989; Henry 2009; Arango et al., 2011; Blom, 2015b; Goldsworthy and Whitaker 2015). Dreaming is commonly defined in opposition to wakefulness or as a non-waking form of consciousness (Searle, 2015: 47). Others use the term "dreaming" to describe any perceptual, cognitive, or phenomenological activity that occurs during sleep (Hartmann, 1995; and Limosani et al., 2011). A more specific definition of dreaming is offered by the *American Heritage Dictionary*: "series of images, ideas, emotions and sensations occurring *involuntarily* in the mind during certain stages of sleep" [my emphasis] (Hobson et al., 2000). But, none of these definitions capture the full dynamic range of oneiric activity that has been uncovered by recent research.

To overcome these difficulties, some sleep researchers have decided to discard the term "dreaming" altogether in favour of "mental-sleep-activity" (MSA) or "mental-sleep-experiences" (MSE) (Fagioli, 2002). In a similar vein, Domhoff and Fox (2015) have proposed to define the phenomenon as any form of "spontaneous" mind-wandering activity that persists during sleep. Yet, even these definitions fail to capture the full dynamic range of oneiric activity. For example, neither one of these definitions captures the oneiric dimensions of such hybrid-states-of-awareness as sleep paralysis, somnambulism, and lucid dreaming, nor do they fully apply to hypnagogic imagery (all of which concomitantly display features of wakefulness, sleep, and dreaming). For these reasons, a thorough definition that accounts for the broad dynamic range of the phenomena is still lacking (Hunt 1989; Hartmann 1995; and Domhoff and Fox, 2015). The parallels between what happened with the nosology of schizophrenia during the twentieth century, and the absence of an encompassing definition for dreaming are intriguing. In fact, usages of both terms still remain in a state of perceptual semantic flux that can significantly vary between research institutes, investigators, and individuals.

One way around this problem is to define oneiric activity as: sensory and perceptual experiences produced in the partial or complete absence of uni-modal, or multi-modal sensory input. This would place dreams on the same continuum as waking hallucinations and would allow the two phenomena to be differentiated based on their level of sensory modulation, i.e., the ratio at any given moment between real sensory input and simulated sensory cues. Within this taxonomical formulation, or what I call the "Oneiric Release Theory Spectrum Scale" (ORTSC) (see p.89), oneiric mentations would be defined as full-blown sensorial hallucinations and would be placed closer to the "sensory de-modulated" end of the imagioneirc continuum, whereas waking hallucinations would be placed closer to the "sensory modulated" end of the spectrum. This definition would not only capture the full range, scope, and perceptual complexity of oneiric activity, but also the dynamic range of hybrid-states-of-awareness, hypnagogic imagery, and mind-wandering episodes. Moreover, by defining both dreaming and waking hallucinations not as dichotomized (sleep/waking) experiences, but as different degrees of objectless sensory impressions that belong to the same imagioneiric continuum, it becomes possible to form new conceptual models of oneiric activity that can account for the entire perceptual range of both waking and sleep phenomena.

Function of Dreams

Defining oneiric activity is far simpler than establishing its bio-evolutionary function. Freud argued that the sole purpose of dreams is to preserve sleep (Freud, 1920: III, ¶12-14 & IX,¶6; and Van de

Castle, 1994: 128). In particular, the Austrian neurologist believed that inhibited desires for prestige, sexual activity, hunger, thirst, success, etc., induced psychological tensions that —under normal circumstances— would be antithetical to the formation (or maintenance) of sleep (Freud, 1933). Given the intrinsic phenomenological similarities between dreaming and waking perception, Freud proposed that dreams evolved to create a "simulation of wakefulness" in which fantasies can be enacted (Van de Castle, 1994: 128).

While the formulation of dreaming as a wish-fulfillment mechanism may be persuasive on a theoretical level and pervasive in the media, the existence of nightmares, and other sleep related anxieties, immediately casts the hypothesis into question. Freud was aware of this problem and made numerous attempts to reconcile nightmares with his general theory of dreams. For example, in *Dream Psychology* (1920), Freud tried to salvage the model by arguing that nightmares are not a core feature of dreaming, but a form of neurosis, or a sign of innate psychopathology.

Another challenge to Freud's theory of dreams is that the overwhelming majority of dreams are not fantasy-enabling. Indeed, most of them are exceptionally mundane (Hobson, 1999: 117 & 131; and Schredl; and Cipolli et al., 2017). In order to circumvent this additional hurdle, Freud argued that the "manifest" content of a dream, or what a dream appears to be about, is not *actually* what a dream is about (Freud, 1933: 47; and Freud, 1920/2013: VI: ¶4 & I: ¶25). This allowed the Austrian neurologist to maintain that all dreams, irrespective of their appearance, invariably serve the function of "wish-fulfillment" (Van de Castle, 1994: 117).

Freud also believed that dream symbolism is universal and can be deciphered in the absence of psychological parameters (Van de Castle, 1994: 158). The Swiss psychiatrist Carl Jung (1875-1961), one of Freud's early closest disciples, vehemently opposed this view and argued that the content of oneiric states is unequivocally heterogeneous. Indeed, he posited that a dream can only be understood within the context of individual mental features, personal history, and current concerns —a view that remains dominant among contemporary psychologists (Van de Castle, 1994: 158).

The first neurophysiological model of dreaming was developed by Hobson and Robert McCarley (1937-2017). Briefly restated, Hobson's Activation-Synthesis-Model (AIM) postulates that dreaming is produced by random residual electric activity that occurs during REM sleep in the pontine brain stem, limbic system, amygdala, and hippocampus. The "random electric" activity is then synthesized by the brain into internal approximations of external sensory input, emotional activity, and cognitive experiences (Hobson and McCarley, 1977). Although the AIM hypothesis successfully accounts for the unpredictable, peculiar and incongruous nature of oneiric activity, it

was unable to account for the existence of re-occurrent dreams. Re-occurrent dreams are experienced by more than 70% of adults and cannot be explained away as "random" electric brain impulses (Zadra, 1996). Moreover, Hobson's model failed to account for the existence of fully formed NREM sleep oneiric activity (see previous section).

Other bio-evolutionary theories of dreaming include: (1) Montague Ullman's (1916-2008) formulation of dreaming as a "problem-solving/insight-generating" mechanism; (2) Harry Fiss' (1926-2009) model of dreaming as an ego coalescing mechanism; (3) Richard Jones' (1925-1994) model of dreams as an ego developing system; and (4) Milton Kramer's model of dreams as an adaptation to stress (Ullman, 1959; Jones, 1962; Fiss, 1986; and Kramer, 1993). Memory consolidation formulations of dreaming often posit that there is a direct interrelationship between the content of oneiric activity and the consolidation of memories during sleep (Maurizi, 1987; and Llewellyn, 2013). In general, these models assume that the phenomenology of dreams is directly (or indirectly) produced by the memory consolidation process itself (Nir et al., 2010; Warmly, 2010; D'agostino et al., 2013c; and Stickgold 2017).

While memory consolidation models of dreaming have been persuasive and indeed popular, they have not been supported by the empirical data. In particular, neither REM sleep, nor SWS deprivation has been shown to impact memory formation. Moreover, neither semantic memory, nor procedural learning, appears to be significantly enhanced by sleep (Hobson, 2010). In fact, over 80% of dream content is entirely unrelated to waking memories (Hobson, 2010). The notion that dream content is a by-product of memory consolidation is also unsupported by the empirical data.

An alternative "memory-consolidation" hypothesis was suggested by Crick et al., in 1983. Specifically, the theory stated that oneiric activity serves the function of preventing "the obsessive persistence of over learning" (Crick et al., 1983). In other words, it posited dreaming as a by-product of the scanning and erasing process of "superfluous memory traces" (Hobson, 2003: 121). The model was never empirically tested, and its impact was overshadowed by the popularity of "memory consolidation" frameworks.

The theories outlined above are all fundamentally reductionist in their approach. Indeed, each focused almost exclusively on the relationship between dreaming and specific psychological functions or features: "memory consolidation", or "emotional regulation", or the "maintenance of the integrity of the self". Because reductionistic formulations of oneiric activity have failed to generate a comprehensive understanding of dreaming, contemporary dream researchers have instead begun to investigate oneiric activity as a phenomenological "whole". This has led to the development of an entirely new genre of theoretical frameworks. Specifically, these newer

formulations present oneiric activity as a type of "virtual-environment" that has exclusively evolved for the rehearsal of different psychological, social, and survival tactics.

The first of these models was proposed by Antti Revonsuo in the year 2000. The "Threat Simulation Theory" (TST) argues that because: (1) dreaming must have evolved to serve a specific bio-evolutionary function, and (2) given that oneiric activity faithfully mimics the phenomenological intricacies of physical reality, produces fully-developed 3D environments, and has the tendency to induce threatening, menacing, or anxiety generating narratives, dreaming could serve the function of sharpening threat-avoidance skills, strategies, and tactics in a "safe virtual space" (Revonsuo, 2000; and Hobson, 2003: 3, 9 & 29).

Revonsuo's model is both original and persuasive. It attributes a direct function to the intrinsic phenomenology of dreams which helps explain why dream environments are often indistinguishable from reality. Furthermore, by portraying dreams as a survival-enhancement-system, TST embeds oneiric activity into a larger a bio-evolutionary *raison d'être*. And unlike many earlier models, Revonsuo's formulation of oneiric activity can successfully account for re-occurrent dreams, i.e., skills that have not been mastered will continue to be rehearsed in "virtual space".

Yet in spite of its many strengths, the model is contradicted by empirical data. In fact, fewer than 20% of dreams, and less than 15% of recurrent dreams, involve threatening, anxiety inducing, or frightening content (Desjardins and Zadra, 2006; and Malcolm-Smith et al., 2008). The overwhelming majority of dreams (80-85%) are non-threatening, and hence cannot serve to improve "threat-avoidance skills". In addition, threatening dream scenarios are resolved in fewer than 20% of dreams, and, their resolution does not appear to lead to more effective avoidance strategies in real life (Desjardins and Zadra, 2006; and Malcolm-Smith et al., 2008). In spite of these failures, Revonsuo's model has re-shaped the scale, scope, and philosophic framework through which oneiric activity is conceptualized.

In 2003, William Domhoff declared that dreams are indistinguishable spatio-temporal replicas of the physical world (Domhoff, 2003: 19). In 2005, David Kahn expanded the framework of TST to encompass the social dimensions of human interactions (Kahn and Hobson, 2005). In 2011 Katja Valli argued that dreaming enables the rehearsal of perceptual, cognitive, *and* emotional social cues.

Revonsuo and his team have since integrated Domhoff (2003), Kahn (2005), and Valli's (2011) insight into a new framework (Revonsuo et al., 2015). The framework, renamed Social-Simulation-Theory (SST), posits that dreaming acts as a "social simulation" environment (Revonsuo et al., 2015). SST focuses on the fact that dreams are centred around lively social interactions and often

include people, animals, or entities that appear to act under their own volitional control (Tholey, 1989; Windt and Noreika, 2011; and D'agostino et al., 2013c). This framework accounts for more oneiric narratives than TST, but, still faces many of the same underlying problems. It too cannot account for the totality of dream experiences. Specifically, while dreams do in fact exhibit a significant "social" component, they also exhibit threatening, uncanny, and other non-social scenarios. It also remains unclear if "social-oneiric-rehearsal-activity" has any meaningful impact on real social activity.

Another "virtual-reality" model was developed by Voss et al., in 2013. In particular, the group argued that dreaming is just a virtual space, with no immediately identifiable function (Voss et al., 2013). Hobson went on to postulate that oneiric activity can be conceptualized as a residual by-product of an "early-stage fetus-neural-rehearsal mechanism", what he termed "proto-consciousness" (Hobson, 2010; and Hobson, 2015: 11, 13, 20, 22, 107, 177, 178 & 230). Hobson's model did not produce an empirically testable hypothesis. When Llewellyn revisited Revonsuo's SST framework in 2016, she argued that the neural algorithm involved in the production of dreams has evolved to predict, simulate, and prepare individuals for future possible events (Llewellyn, 2016b). This formulation presents dreams as serving the purpose of "preparing" individuals for an array of future circumstances (Pace-Schott et al., 2017). This model of dreaming as a "future-rehearsing-mechanism" remains untested.

Virtual, reality-simulation, and rehearsal models have been influential. Their timely appearance alongside the many "virtual-reality" theories now coming out of Silicon Valley merits notice. Indeed, they are expressions of a larger cultural zeitgeist. The bio-evolutionary functional approach and their portrayal of oneiric activity as fully developed spatio-temporal replicas of wakefulness has been met with great interest by the greater scientific and academic community. Unfortunately, the heterogeneity of these models, equivocal characteristics, and their inability to account for the full cognitive, perceptual, and behavioural spectrum of oneiric activity, makes them difficult to test.

I will end this subsection restating that 120 years after Freud first published the *Interpretation of Dreams* (1899), the bio-evolutionary adaptive value of dreaming, and the implications of having to live with minds that are neurobiologically wired to dream on a daily basis still remains unknown (Valli et al., 2005; Gottesmann, 2010; Valli, 2011; and Stickgold 2017). None of the current formulations of dreaming shed any real light on the purpose, function, or evolutionary advantage of having evolved the capacity to generate oneiric activity (Hartmann, 2010; Valli, 2011; Stickgold, 2017). These failures have led some to hypothesize that dreaming may simply be a residual product

of wakefulness, or an epiphenomenon of basic brain activity with no specific function related to survival, well-being, or health (Domhoff, 2011).

Phenomenology of Dreams

In terms of perceptual modalities, dreams tend to be predominately visual and can replicate the full spectrum of waking optical perceptions. This includes shapes, movement, colour, texture, shadows, contrast, people, animals, faces, objects, and inanimate objects, among others (Rechtschaffen 1978; Kahn and Gover, 2010; Kussé et al., 2010; Nir et al., 2010; Pagel 2014; and Kahan and Claudatos, 2016). Auditory hallucinations are the second most common type of oneiric mentation, and are reported in more than 50% of awakenings (Nir et al., 2010; and Zadra, 2017). Oneiric auditory mentations are equally capable of reproducing the full spectrum of waking auditory cues, including music, voices, dialogue, and environmental sounds (Van de Castle, 1994: 11, 12, 13, 16, 18, 22, 35 & 36; Kahn and Gover, 2009; and Kussé et al., 2010). Gustatory, olfactory, somatosensory (pressure, pain, and warmth), proprioceptive, visceral, kinaesthetic, and vestibular oneiric hallucinations, are also routinely reported, though less frequently (Kahn and Gover, 2010; Nir et al., 2010; and Kahan and Claudatos, 2016). The exact phenomenology of these secondary mentations has not been yet fully mapped out. It is clear, however, that all oneiric mentations display wake-like perceptual-motor vividness and can induce the full range of sensorial experiences that are normally associated with wakefulness (Rechtschaffen 1978; Hobson, 2003: 48; Kahn and Gover, 2010; Kussé et al., 2010; and Limosani et al., 2011).

Perceptually, the embodied experience of selfhood (or phenomenological subjectivity), as encapsulated by the pronoun "I", remains intact during dreaming (Gillespie 1997; and Hobson, 2015: 26). In particular, the centre of phenomenological perception, normally associated with physical embodiment (somewhere immediately behind one's face), is replaced or repositioned to within the confinements of a "dream body" (Kahn and Gover, 2009; and Tononi and Laureys, 2009). The "oneiric homunculus" (my term) is like all other oneiric hallucinations in its remarkable ability to mimic the real physical body. It can move, hear, walk, run, taste, smell, feel, and perform any type of normal physical bodily motion (Kahn and Gover, 2009). In addition, it can fly, walk through walls, change its size and shape, and disregard many of the other physical constraints imposed on a person's physical body (Kahn and Gover, 2009 & 2010).

On a structural level, the phenomenology of dreams is characterized by a marked dichotomy between an embodied presence (the oneiric homunculus) and the perception of an externalized oneiric environment. This environment, for all intents and purposes, behaves in a similar fashion to

the real "external" world (Gillespie, 1997; Occhionero and Cicogna, 2011; and Hobson, 2015: 26). In some cases, dream environments are altogether indistinguishable from a person's real living environment. For example, during false awakenings, dreamers report waking up to a perfect oneiric replica of their room, house or apartment (Hobson, 2003: 158 & 160). This phenomenon suggests that oneiric activity can project internal precepts into fully actualized spatio-temporal imitations of the real world (Kahn and Gover, 2010).

Just as with the psychotic symptoms of schizophrenia, and as Jungian theory would anticipate, the content of oneiric mentations is influenced by personality traits, individual interests, waking concerns, and to some degree, pre-sleep conditions (Hobson, 1999: 117 & 131; Schredl 2003; Nir et al., 2010; and Cipolli et al., 2017). The exact relationship between a dreamer's immediate physical environment and the content of their oneiric activity remains unclear (Arkin and Antrobus, 1991; Nielsen, 1993; Lesilie and Ogilvie, 1996; Mahowald et al., 1998; Eiser, 2005; and Cipolli et al., 2017). For example, about 30% of people who have slept in a sleep laboratory regularly report dreams that reflect their experience of the "sleep laboratory environment" (Rechtschaffen, 1978). Conversely, people who suffer from sleep apnea, neurological disorders, or chronic conditions rarely dream about their symptoms (Gross and Lavie, 1994; Nielsen et al., 1993; and Mahowald et al., 1998). This suggests that not all external physical circumstances impact the content of oneiric activity in an equal manner.

Despite the common association between dream content and the bizarre, the vast majority of oneiric activity is mundane (Strauch and Meier, 2004; Schredl, 2010; and Kahn and Gover, 2010). In fact, less than 5% of dreams contain any "bizarre" elements (Rechtschaffen 1978; Strauch and Meier, 2004; Eiser, 2005; and Schredl, 2010). Moreover, most dreams tend to develop in accordance to a specific theme (Rechtschaffen 1978; and Siclari et al., 2017). These themes can be experienced as basic images, inclusive narratives, or as fully-developed phenomenological environments.

Dreams also exhibit predictable forms. For example, "persecutory experiences" —especially that of being chased— are the single most reported oneiric narrative form (Nielsen et al., 2003; and Windt and Noreika, 2011). It is worth noting that "persecutory delusions" are also the most commonly reported waking delusional form. Less common oneiric delusional forms include encountering the deceased, encountering the living as deceased, encountering uncanny humanoid presences, interacting with deities, aliens, and meeting "supernatural entities" (Nielsen et al., 2003). Many of these figures appear to act as if they are in possession of their own consciousness, psychology, individual goals, fears, concerns, and ambitions (Tholey, 1989; Hobson, 2003: 31; Windt and Noreika, 2011; and D'agostino et al., 2013b). Indeed, influencing the behaviour of oneiric

figures is reportedly just as difficult as influencing the behaviour of real people (Hobson, 2003: 31; and D'agostino et al., 2013b). This holds true even for lucid dreamers who have actively cultivated the ability to consciously control their oneiric activity (Tholey, 1989). Strangely, oneiric figures appear to be susceptible to dialogue, reason, argumentation, and discourse (Tholey, 1989).

Individual dream content can vary immensely, but, the phenomenological architecture of oneiric activity exhibits a universal structure (Hobson, 1999: 131; Revonsuo, 2000; Kahn and Gover, 2009; and Kahan and Claudatos, 2016). Specifically, in addition to some predictable oneiric narrative themes (like persecution), all dreams that are remembered well enough to report: (1) advance in a temporal episodic sequence; (2) occur in congruence with real time; (3) give rise to fully developed multimodal sensorial experiences; (4) construct 3D spatio-temporal environments; and (5) are experienced from the vantage point of a sensory-motor enabled oneiric homunculus (Oswald 1962b; Rechtschaffen 1978; Valli et al., 2005; Hobson et al., 2007; and Limosani et al., 2011).

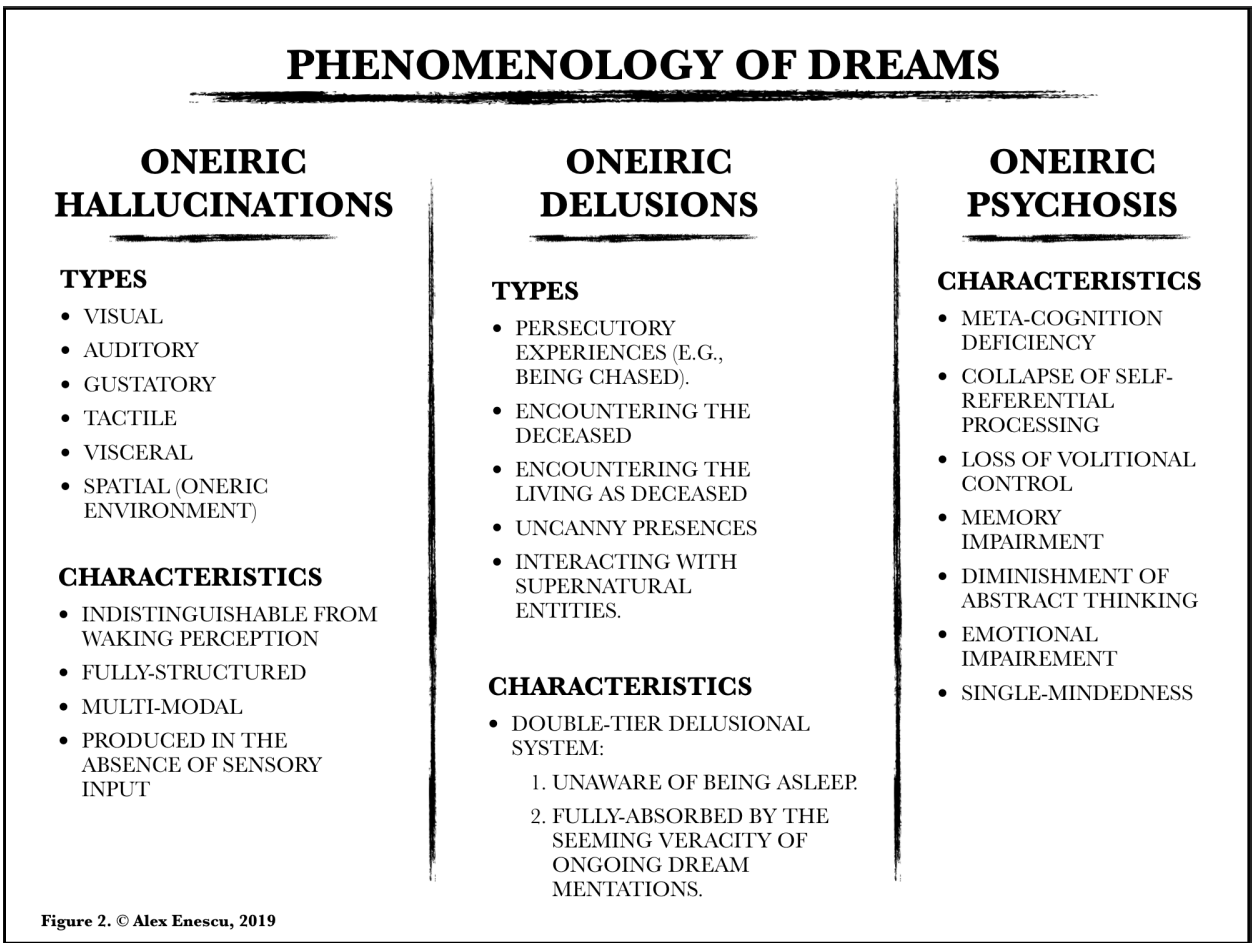


Figure 2. © Alex Enescu, 2019

The cognitive dimensions of oneiric activity are also surprisingly similar to those that are normally displayed during wakefulness. Dreamers can engage in logical thinking, perform cognitive analysis, connect internal narratives (or different dream plots), evaluate perceived events, and make judgment calls (Rechtschaffen 1978; Kahn and Gover, 2009; and Kahan and Claudatos, 2016). Be that as it may, the dreaming mind is also dissimilar from its waking counterpart. Specifically, dreaming induces severe deficiencies in meta-cognition, self-referential processes, feedback processes, attention, volitional control, memory retrieval, abstract thinking, and the ability to engage in mind-wandering activity (James, 1890/1981: 264; Rechtschaffen 1978; Hartmann, 1982; Kahan 1997; Mahowald et al., 1998; Hobson, 1999: 32; Franklin et al., 2005; Limosani et al., 2011; D'agostino et al., 2013b and 2013c; and Filevich et al., 2015).

Perceptually, these deficits lead to the formation of a narrowly channeled stream of "single-minded" consciousness. This greatly inhibits the dreamer's ability to alternate between internal mental cues and the "external" oneiric environment or to reflect back on their own condition (Rechtschaffen 1978; Hartmann, 1982; Franklin et al., 2005; Kahn and Gover, 2009; and Gerrans, 2012). Even so, the overall phenomenological and cognitive features of oneiric activity are strikingly similar to those that are exhibited by waking mental states.

Comparison Between Oneiric Activity and the Psychotic Symptoms of Schizophrenia

Oneiric activity shares a remarkable number of features with the psychotic symptoms of schizophrenia (i.e., waking hallucinations, delusions, and psychosis). Dream cognition, for example, closely mimics the symptomatology of delusional thinking and psychosis. Specifically, dreams induce a severe deficiency in meta-awareness, self-referential ability, volitional control over mental content, ability to maintain organized thinking, meta-attention, associative thinking, memory retrieval, abstract thinking, mental continuity, emotional stability, reality testing abilities, self-reflective capacities, and the ability to discern between internal and external cues (Oswald, 1962a, 1962b, 1962c, 1962d, and 1962e; Koresko et al., 1963; Vogel, 1974; Hartmann, 1975; McGreery, 1997; Mahowald et al., 1998; Hobson, 1999: 43; Hobson, 2003: 80; Gottesmann, 2010; Nir et al., 2010; Limosani et al., 2011a and 2011b; D'agostino et al., 2013 and 2013c; Gerrans, 2014a; Dresler et al., 2014b; Benson and Feinberg, 2017; and Pace-Schott et al., 2017). Dreams and psychosis are also both marked by a significant "loss of insight", during which internally generated imagery, cues, and narratives seamlessly replace the rigidity and structure of external attention. The rigidity, logic, and ongoing stream of causal connectivity between events is lost in both states (Hartmann, 1982; Kahn and Gover, 2009; Gerrans, 2012; Dresler et al., 2012; Dresler et al., 2014; and Hobson, 2015: 152).

Sensorially, both dreams and waking hallucinations are experienced in multiple, as opposed to uni, simultaneous sensory cues and can re-produce the full-spectrum of waking sensorial perceptions (Vogel, 1974; Hartmann, 1975; McCreery, 1997; Mahowald et al., 1998; Hobson, 1999: 32 & 43; Hobson, 2003: 80; Nir et al., 2010; Limosani et al., 2011a; Dresler et al., 2014; Waters et al., 2016; Waters et al., 2016; Lim, et al., 2016; and Pace-Schott et al., 2017). Whereas dreams are predominantly visual, waking hallucinations tend to be predominantly auditory (Domhoff et al., 2015). Conversely, auditory hallucinations are the second most common oneiric sensory experience and visual objectless perceptions are the second most reported type of waking hallucinations (Kraepelin, 1915: 262; Rechtschaffen 1978; Assad, 1986; Nir et al., 2010; Goldsworthy and Whitaker 2015; and Zadra, 2017). Furthermore, olfactory, gustatory, somatosensory, kinaesthetic, vestibular, tactile, and visceral hallucinations, are infrequently reported in both conditions (Kraepelin, 1915: 262; Assad, 1986; Nir et al., 2010; Goldsworthy and Whitaker 2015; and Kahan and Claudatos, 2016). It has been argued that waking hallucinations and delusional narratives can impact a person's belief system in a way that dreaming cannot (Waters et al., 2016). Yet this argument is blatantly false. Oneiric experiences can significantly alter a person's mental state, waking mood, thought stream, philosophic outlook, ideas, and general worldview (Revonsuo 2000; Strauch and Meier, 2004; Schredl; 2010; Nir et al., 2010; Noreika, 2011; and Palagini, 2011)!

This is clear both inside and outside of the laboratory setting. For example, dreaming played a major inspirational role in William Blake's (1757-1827) paintings, the cinematographic production of Ingmar Bergman (1918-2007), Carlos Saura (1932-), Orson Welles (1915-1985), and Federico Fellini (1920-1993), and in the literary works of Marry Shelley (1797-1851) and William Burroughs (1914-1997). Moreover, oneiric activity played a pivotal role in Frederick Banting's (1891-1941) discovery of pancreatic insulin extraction, the creation of the periodic table, and the discovery of the benzine molecule (Van de Castle, 1994: 11, 12, 13, 16, 18, 22, 35 & 36; Revonsuo 2000; Strauch and Meier, 2004; Kahn and Gover, 2009; and Kussé et al., 2010; Schredl; 2010; Nir et al., 2010; Noreika, 2011; Palagini, 2011).

On a phenomenological level, both dreams and waking hallucinations can generate spatio-temporally situated voices, figures, people, animals, objects, and landscapes, in addition to objectless somatosensory impressions (Bleuler, 1966: 104; Corballis 2014: 129; Blom, 2015; and Waters et al., 2016). In both cases, oneiric and waking hallucinated figures are experienced as conscious, goal oriented, and in possession of their own volitional control (Kraepelin, 1915: 11; Bleuler, 1966: 95 & 96; Tholey, 1989; Hobson, 2003: 31; Windt and Noreika, 2011; Arango and Carpenter, 2011; D'agostino et al., 2013 and 2013c; Waters, 2014; and Goldsworthy and Whitaker 2015).

Psychologically, dreaming is a two-tier delusional system. On the one hand, dreaming is always a delusional mental state because it is incorrectly perceived as wakefulness. On the other hand, dreaming can reproduce the entire spectrum of waking delusional narratives —in the same exact order of frequency (Vogel, 1974; Hartmann, 1975; Hobson, 1999: 32, 43; Hobson, 2003: 80; Kahn and Gover, 2009; Nir et al., 2010; Limosani et al., 2011; Gerrans, 2012; D'agostino et al., 2013; Gerrans, 2014a; Gerrans, 2014b; Dresler et al., 2014; and Pace-Schott et al., 2017). For example, the most common oneiric and waking delusion is that of persecution, i.e., being followed by monsters, or para-governmental agencies, being monitored by supernatural entities, or being detained, punished, and disciplined in the absence of a crime (Kraepelin, 1915: 257; Nielsen et al., 2003; Kellerman, 2009; Arango et al., 2011; and Gold and Gold, 2014: 64). Other common types of oneiric and waking delusional narratives that occur in the same frequency include: (1) delusions of misidentification; (2) religious and supernatural delusions; (3) cotard delusions; and (4) delusions of control (Arango and Carpenter, 2011; and Gold and Gold, 2014: 59, 60, 62 & 63; Nielsen et al., 2003; and Gerrans, 2014a). This suggests that similar systems are involved in the production of both waking and oneiric delusions.

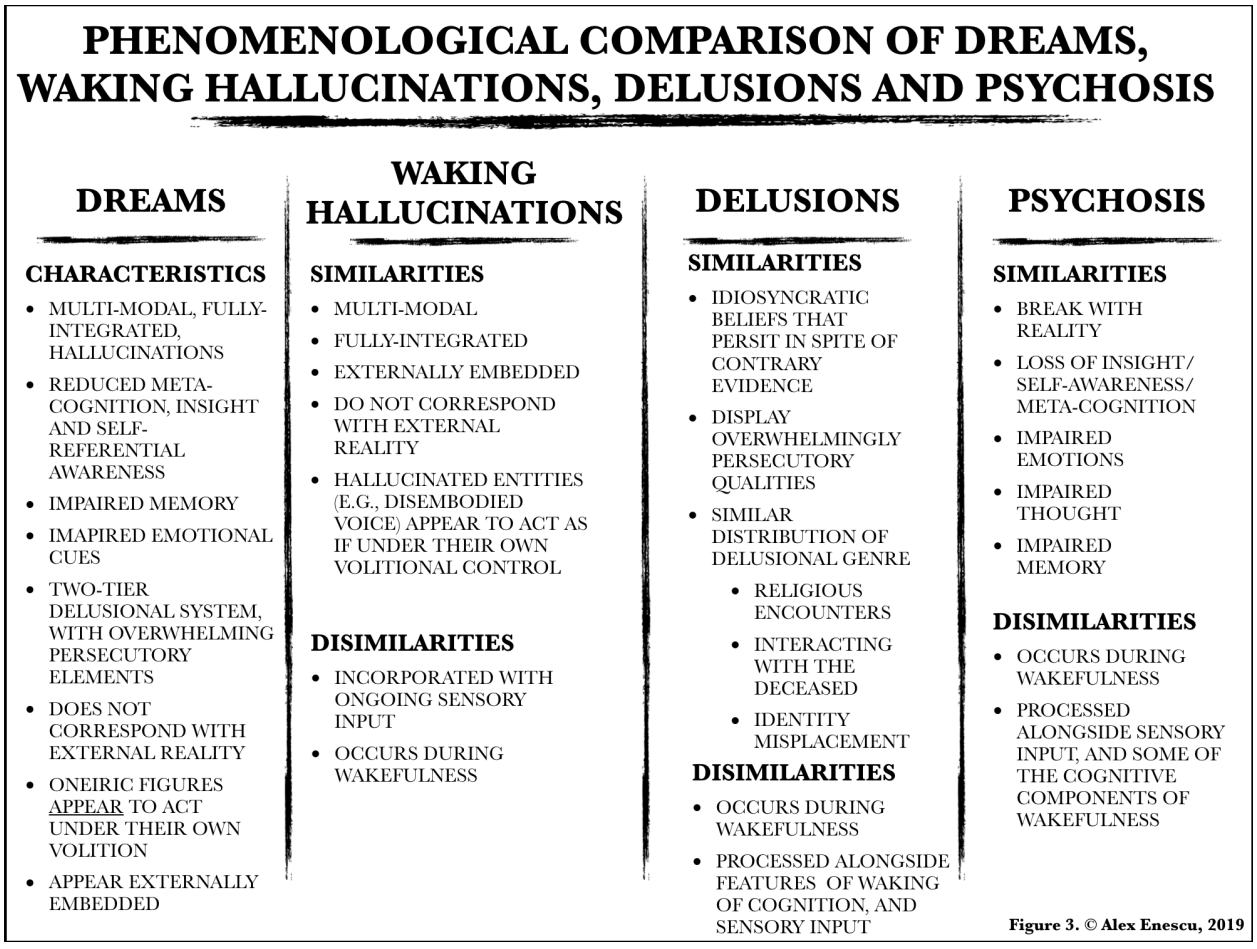


Figure 3. © Alex Enescu, 2019

Section III Summary

The perceptual, cognitive, structural, and phenomenological similarities exhibited by oneiric activity and the psychotic symptoms of schizophrenia, strongly suggest that the two conditions have similar phenomenological origins (Mahowald et al., 1998; and Windt and Noreika, 2011). Moreover, given that the formation of dream mentations is not contingent on structural brain abnormalities, this further suggests that the psychotic symptoms of schizophrenia can be produced by an "intact" brain architecture. This would help explain why no significant neurophysiological abnormalities have been found in people who suffer from schizophrenia or in any other type of psychotic disorder (Hobson, 2015: 151; and Windt and Noreika, 2011). In fact, if the psychotic symptoms of schizophrenia are invariably caused by a form of dreaming that has "gone out of control", or by oneiric activity that has extended beyond its normal architectural confinement (i.e., the physiological boundaries of sleep), then it follows that the mechanisms that enable the development of psychotic symptoms must be inherently present in everyone. To explain this, an endogenous mechanism capable of hybridizing elements of dreaming (i.e., multi-modal hallucinations, meta-cognitive deficits, and delusions) with the variant characteristics of wakefulness (i.e., meta-awareness and contextual insight) and sleep (i.e., sensory demodulation) must exist. The following two sections present neuro-phenomenological evidence for the existence of just such a mechanism.

SECTION IV

HYBRID-STATES-OF-AWARENESS

Section IV Introduction

This section proposes that dreaming, wakefulness, and sleep are diaphanous states that can periodically "bleed" into each other's phenomenological, cognitive, and psychological domains (Mahowald and Schenck, 1992; Llewellyn, 2009; Limosani, et al., 2011; and Hobson, 2015: 46). More specifically, it argues that the elementary components of dreaming, wakefulness, and sleep can become "imbricated" in order to form new perceptual states of awareness, or what I now call "hybrid-states-of-awareness". Although the combinatory potential of the elementary components of dreaming, wakefulness, and sleep could very well be too great to map out, this section focuses on the neuro-phenomenological dynamics of three unambiguous hybrid-sates-of-awareness, namely sleep paralysis, somnambulism, and lucid dreaming.

The latter part of this section argues that the psychotic symptoms of schizophrenia can be conceptualized as a fourth genre of "hybrid-states-of-awareness". That said, it should be noted that at no point in Section IV is a specific imbrication mechanism discussed. A possible mechanism for hybridization, oneiric release, is presented in Section V of this work. Instead, this section (Section IV) demonstrates that imbrication can occur on a regular basis in individuals who do not suffer from any structural brain abnormalities and that hybrid-states-of-awareness can be generated within the context of "normal" brain architectures.

Sleep Paralysis (SP)

Sleep Paralysis (SP) is one of the most easily recognizable hybrid-state-of-awareness (HSA). The condition tends to occur either immediately prior to falling asleep or during the process of awakening. Specifically, SP combines the core neurophysiological features of REM sleep (i.e., skeletal muscular motor paralysis) with multi-modal oneiric hallucinations, and such features of wakefulness as the partial restoration of meta-cognitive awareness, the ability to self-reflect, and sensory modulation (Cheyne, 1999; Sharpless et al., 2011; Goldestein, 2011; Sharpless 2016; Avident, et al., 2011; Ohayon et al., 1999; Occhionero and Cicogna, 2011; Sara et al., 2016; and Denis et al., 2018).

Throughout most of the twentieth century, SP was considered a sleep disorder related to anxiety, neurosis, and psychopathology (Sevilla, 2004). Early twenty-first century researchers, however, unequivocally demonstrated that SP is not the product of psychiatric disorders and occurs more frequently in healthy individuals (McNally, 2005). In fact, one of the primary diagnostic criteria for SP is the absence of an identifiable psychiatric condition (Sevilla, 2004). Secondary diagnostic criteria for SP include: repeated motor paralysis experienced during periods of awakening (or during

sleep onset) and the presence of oneiric hallucinations alongside real sensory modulated perception (Sevilla, 2004; and McNally, 2005).

SP is not a rare phenomenon. It has been recorded in as little as 2% and as much as 50% of the general population (Sevilla, 2004; and Denis et al., 2018). According to the most conservative estimates, roughly 25% to 40% of people will experience at least one SP attack during their lifetime (Goodare, 2013). In most of these cases, such oneiric hallucinatory components of the condition as encountering "supernatural" entities, shadow entities, or the deceased, are not experienced. Nevertheless, at least 5% of the population will experience some type of oneiric hallucination during an SP attack (McNally, 2005).

Certain personality traits, behaviours, gender, and lifestyle choices have been found to increase the likelihood of an SP episode (Denis et al., 2018). In particular, irregular sleep cycles, shift work, caffeine and alcohol consumption, insomnia, regular sleep deprivation, napping, and sleeping on one's back, have all been established as SP risk factors (Ohayon et al., 1999; Hufford, 2002; McNally, 2005; and Hurd, 2011). Moreover, fantasy-prone individuals, artists, and people who work in creative industries, in addition to those who are prone to experiencing anxiety and low moods, have a greater chance of experiencing an SP episode than the general population (Hurd, 2011).

The exact neurophysiological processes involved in the production of SP remain unknown (Denis et al., 2018). Yet it is generally agreed that SP is produced through the physiological, perceptual, and cognitive combination of REM sleep, wakefulness, and dreaming (McNally, 2005; and Goodare, 2013). In particular, SP is believed to enable the amalgamation of real sensory input from the dreamer's immediate environment with oneiric visual, auditory, and somatosensory hallucinations, REM sleep atonia, and waking levels of meta-cognition (see figure 4 on p.65).

The core characteristics of sleep paralysis are the following: (1) waking up to skeletal body muscular paralysis while being able to scan the immediate physical environment through eye movements; (2) experiencing objectless auditory cues, such as thunder claps, howls, shrills, hisses, and whispers; (3) suffering from such somatosensory hallucinations as tingling sensations, numbness, and a feeling of lightness or flotation; (4) encountering aggressive, threatening, or non-threatening humanoid entities; (5) experiencing an uncanny felt presence, like an invisible "monitoring" entity; and (6) suffering from panic or intense emotional distress (Thorpy, 1990; Spanos, 1995; Cheyne, 1999; Ohayon et al., 1999; Blackmore and Parker, 2002; Cheyne, 2003; Occhionero and Cicogna, 2011; and Jalal, 2016).

Some SP sufferers never report encountering humanoid figures. Instead, they describe being "monitored" by an "invisible" presence (Nielsen 2007; Cheyne, et al., 2007; Solomoneva, et al., 2011;

and Hurd, 2011: 35). Contrary to popular belief, in more than 56% of cases, the felt presence (FP) is experienced as a non-threatening or neutral entity. Only one third of encounters with an FP exhibit menacing characteristics (Nielsen, 2007). Intriguingly, the felt presences tend to induce a "carry-over" effect and continue to be "felt" long after the SP episode has ended.

The humanoid entities encountered during SP often behave in an aggressive manner (Ohayon et al., 1999; Solomoneva, et al., 2011; Lišková et al., 2017; and Denis et al., 2018). These apparitions are frequently described as actively attempting to suffocate the SP experiencer, either by sitting on their chest or by strangulation (Jalal, 2015; and Denis et al., 2018). Because of these frightening hallucinatory assaults, SP can induce a state of panic, acute anxiety, breathing difficulties, and a general sense of horror (Liddon, 1967; Ness, 1978; Ohayon et al., 1999; and McNally et al., 2005b).

The entities are most often experienced as "self-conscious" shadow figures, dwarf-like entities, or as old hags. But, people also report encountering space aliens, demons, and even deceased and/or living relatives (Ness, 1978; Ohayon et al., 1999; Appelle, 2002; Cheyne, 2003; McNally et al., 2005b; D'Agostino et al., 2010; Hurd, 2011: 51 & 52; Solomoneva, et al., 2011; and Denis et al., 2018). Non-aggressive apparitions are rarer and do not differ in form. Instead of physically assaulting the SP victim, non-aggressive apparitions tend to induce a soothing emotional state or make active attempts to comfort the sufferer (Solomoneva, et al., 2011; Hurd, 2011: 51; and Lišková et al., 2017).

It remains unclear why SP suffers universally encounter the same genre of humanoid entities (Hufford 2002: 15; Hurd, 2011: 35 & 43; and Sevilla 2004: 29). For example, in Newfoundland and many parts of Western Europe, the SP entity generally takes on the shape of an "Old Hag"; in Japan, SP sufferers report encountering the "Kanashibari", a popular folklore figure who resembles the ghost character from the 1998 Japanese ghost-horror film (リング Ringu) (Hufford, 2002: 1 & 13; Adler, 2011: 20; and Hurd, 2011: 43). During the fifteenth and sixteenth centuries in Europe, the SP entity became associated with the incubus and succubus phenomenon —believed to visit people during sleep to sexually assault them (Adler, 2011: 47 & 48). In traditional East Asian folklore the SP entity is portrayed as a ghost or as the spirit of a recently deceased relative; while in Ancient Greek and Middle Eastern literature, SP apparitions are depicted as demonic entities, similar to the incubus and succubus entities described in Renaissance Europe (Adler, 2011: 41; and Lin Fang, 2013). During the Scottish witch-hunt of the eighteenth century, SP entities took on the image of witches and warlocks (Goodare, 2013: 128). In contemporary Western societies, the apparitions are most often depicted as shadow entities, dwarfs, old hags, and space aliens (Appele, 2008; and Sevilla, 2004). Nevertheless, there are also reported cases of insect-like creatures, "sentient balls of lights", and other "irregular" entities (Hurd, 2011: 36; and Hufford 2002: 63). What remains unclear is why

the hallucinatory components of SP are expressed in the form of a "monitoring" presence as opposed to an "earthquake", "asteroidal impact", "explosion", or other types of natural phenomena (Hurd, 2011: 36 & 37).

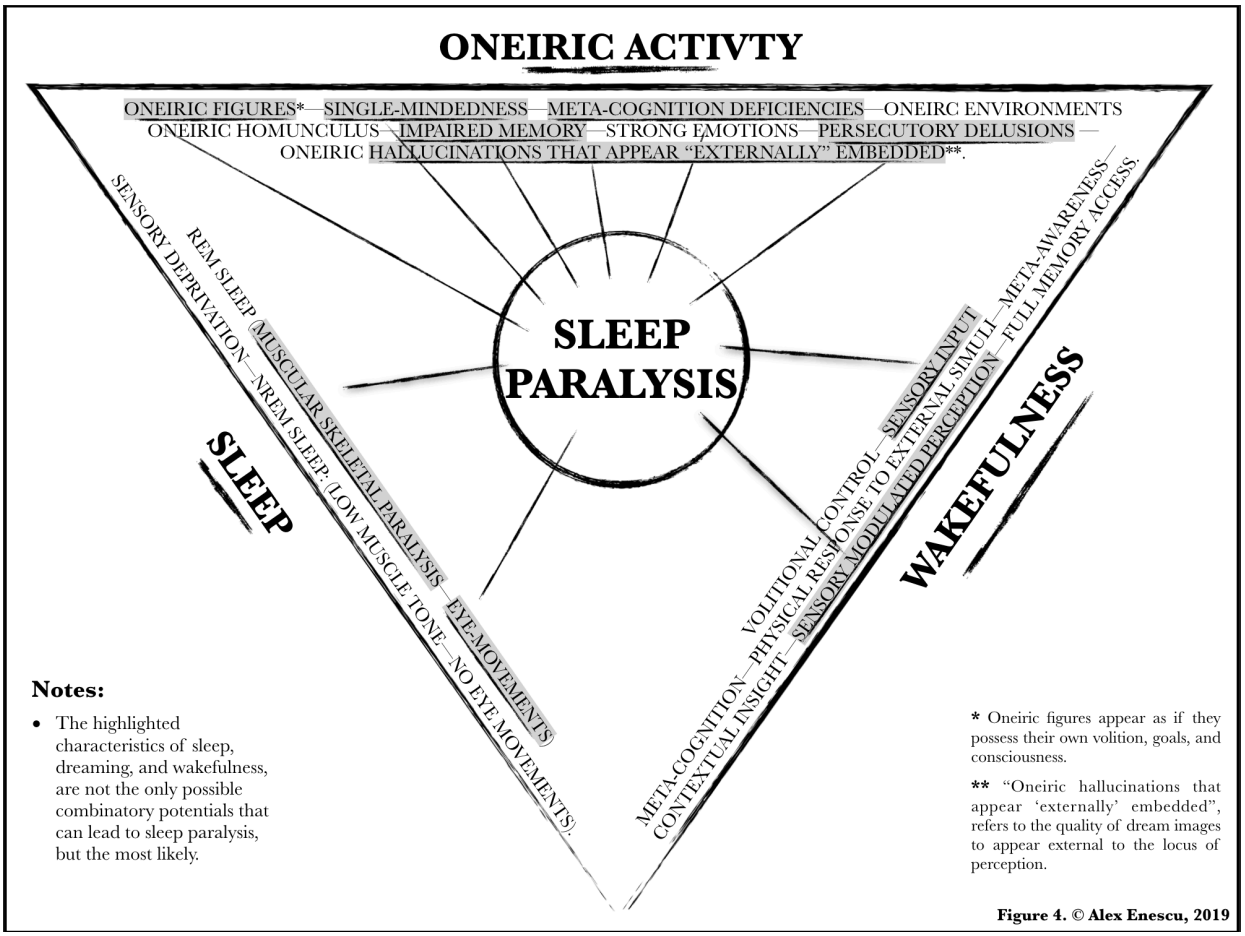


Figure 4. © Alex Enescu, 2019

The presence of muscle atonia during SP correlates with some of the phenomenological components of the experience, namely the sensation of being physically oppressed and having difficulty breathing. Current formulations of SP postulate that apparitions are created by an overactive threat-activated vigilance-system (TAVS) triggered by the sensation of paralysis (Cheyne, 2007b; and Jalal, 2016). According to this view, the experience of immobility in the absence of an immediately identifiable source of physical constrainment, causes the brain to "invent" an oneiric "intruder" to complement the physiological symptomatology of paralysis (Nielsen, 2007, Cheyne, 2007b; and Jalal, 2016). However, the presence of skeletal muscular paralysis does not explain why SP sufferers regularly report encountering benign felt presences or why the overwhelming majority of SP apparitions do not engage in threatening behaviour (Nielsen, 2007).

Sleepwalking (SW)

In the 1950s, Michel Jouvet (1925-2017) hypothesized that impairing the pontine tegmentum, or dorsal pons (found in the brainstem), would permanently disable motor skeletal muscular paralysis during REM sleep (LaBerge, 1990; and Kripnner, 2002). He went on to theorize that when uninhibited by REM sleep atonia, oneiric homunculus activity was likely to translate into real physical motor action. Jouvet tested his model on a group of lab cats in the 1950s, and, to everyone's surprise, the hypothesis panned out. During each REM sleep cycle, the post-surgical lab cats stood up, moved, ran, and stalked imaginary prey (LaBerge, 1990; and Kripnner, 2002). In other words, the cats became "somnambulic".

Similarly to Jouvet's pontine-tegmentum-impaired cats, people who experience somnambulism can engage in a host of wake-like behaviour during sleep. They might stand up, move, run, get dressed, climb up or down ladders or windows, do housework, cook, eat, play musical instruments, engage in sexual activity, safely drive motor vehicles for prolonged distances, and even write (Gaudreau et al., 2000, Lam et al., 2009 and Dang-Vu et al., 2015). In addition, sleepwalkers can express complex states of mental distress, such as pain, various forms of anxiety, excessive agitation, and aggression (Gaudreau et al., 2000, Oudiette et al., 2009; and Lam et al., 2009).

For a long time, somnambulism was believed to be caused by the unconscious enactment of dreams during unequivocal sleep (Jacobson, 1965). This belief drew on the observation that sleepwalkers behave in a manner that corresponds with their most immediately remembered dream activity (Pillmann, 2009). In the 1960s, however, the "dream enactment" model was challenged by the discovery that SW occurs exclusively during NREM sleep —a sleep stage that was then incorrectly assumed to be antithetical to dreaming (Jacobson, 1965; and Broughton, 1968). As such, dream enactment models of somnambulism were discarded in favour of "arousal" theories and other models that disregarded the notion that SW activity may correspond to ongoing oneiric mentations (Jacobson, 1965 ; Broughton, 1968; Gaudreau, et al., 2000; Szelenberger, et al., 2005; and Oudiette, et al., 2009).

The exact neurophysiology of somnambulism remains elusive (Szelenberger 2005, Espa et al., 2000 & Dang-Vu et al., 2015). Sleepwalkers exhibit lower EEG slow-wave-activity (SWA) during slow-wave-sleep (SWS), in addition to continuous episodes of micro-arousals, followed by a sudden and unaccounted for EEG electrical spike (103 c/s—1-3Hz) ten to thirty seconds prior to a SW incident (Halasz, et al., 1985; Broughton, 1990 & 1991; Blatt, et al., 1991; Gaudreau, et al., 2000; Espa, et al., 2000; Szelenberger, et al., 2005; Pilot et al., 2012; and Zadra, et al., 2013). The correct analysis of this data still remains the subject of debate.

Most today believe that somnambulism emerges out of a "dysfunction in slow-wave-sleep regulation" and/or an "incomplete transition from SWS to wakefulness"; episodes appear to be precipitated by possible genetic factors and lifestyle choices, including the use of recreational drugs like alcohol, caffeine, nicotine, or recreational drugs, excessive consumption of carbohydrates and sugar, exposure to particular types of music before sleep, shift-work, irregular sleep patterns, insomnia, sleep deprivation, and/or the pre-existence of such disorders as anxiety, depression, and psychosis (Broughton, 1982; Mahowalkd, 1990; Ettinger, 1990; Espa, et al., 2000; Gaudreau, et al., 2000; Lecendreux et al., 2003; Zadra, et al., 2013; and Dang-Vu, 2015).

Sleep disruption increases SW incidents in susceptible individuals, but not in healthy controls (Espa, et al., 2000; Szelenberger, 2005; Zadra, et al., 2013; and Dang-Vu et al., 2015). In fact, a number of studies indicate that somnambulists exhibit abnormal sleep patterns even when not SW (Espa et al., 2000; and Guilleminault, et al., 2005). This evidence suggests that although a disrupted build-up of SWA or the inability to maintain SWS are reliable characteristics of people who suffer from SW, these factors alone are insufficient to induce a somnambulic episode (Broughton, et al., 1994; Crisp, 1996; Espa et al., 2000; Joncas et al., 2002; Pilot, et al., 2008; Jaar, et al., 2010; and Zadra, et al., 2013).

Somnambulism is more prevalent in children and individuals who suffer from at least one psychiatric disorder (Kales, et al., 1980; Abe et al., 1984; Szelenberger, 2005; and Lam, et al., 2009). In particular, about 44% of all psychiatric patients experience periodic somnambulic episodes and more than 60% of people who regularly experience SW will eventually be diagnosed with a psychiatric condition (Szelenberger, 2005; and Lam et al., 2009). Psychiatric patients share many physiological overlaps with sleepwalkers. Lower delta activity, as well as dysfunctional interplay between the motor and cumulate cortices and the medial prefrontal and lateral parietal cortices have been routinely observed in both sleepwalkers and people who suffer from schizophrenia (Hiatt, et al., 1985; Ganguli, et al., 1987; Szelenberger, 2005; and Zadra, et al., 2013). Nevertheless, SW can and does occur outside the context of psychopathology.

Sleepwalkers report that their episodes are triggered by "an intrinsic sense of urgency" that is often accompanied by a nightmarish dream mentation (Oudiette et al., 2009 and Zadra et al., 2013). In fact, it is entirely possible that the sudden EEG spike observed in sleepwalkers, ten to thirty seconds prior to an episode, is a direct visual representation of the subjective sense of this "intrinsic sense of urgency" that is reported. Indeed, the concurrence of frightening dream mentations during SWS also appear to act as an internal trigger for somnambulism. This would also explain why somnambulic episodes cannot be reliably induced in a laboratory setting, i.e., they require a

subjective trigger (Zadra et al., 2008). These observations suggest that somnambulic behaviour is a condition that requires both a particular physiology (i.e., fragmented SWS) *and* a particular psychological state.

Due to the dominance of the arousal model of SW, phenomenological reports are virtually absent from the SW literature. Indeed, somnambulic dream reports have been systematically disregarded since the 1960s as remnants of unorganized thoughts, waking visual imagery, or faulty memory systems (Oudiette, et al., 2009). In spite of this, an increasingly convincing body of literature suggests that dream mentations are unequivocally present during episodes of somnambulism (Pilmann, 2009; and Oudiette, et al., 2009). Furthermore, we now know that sleep stages are more fluid and interactive than originally thought (see p.12) and that REM sleep-like dream reports can be routinely produced throughout all stages of sleep, including during NREM sleep (Oudiettet, et al., 2009; and Zadra et al., 2013). Yet collecting dream mentations from sleepwalkers is challenging.

Sleepwalkers exhibit difficulties in remembering not only their somnambulic activities, but also any dream mentations that they may have experienced in close proximity to the SW period (Bassetti et al., 2000; Gaudreau et al., 2000; Oudiette et al., 2009; Pillmann et al., 2009; and Lam et al., 2009). Even so, a number of studies demonstrate that a sleepwalker's dream activity often corresponds with their somnambulic behaviour (Pillmann, 2009; and Zadra, et al., 2013). There are at least a good dozen cases of well-documented sleepwalkers whose somnambulic behaviour perfectly mimicked their dream activity (Oudiette, et al., 2009; and Pillmann, 2009). For example, Oudiette et al., (2009) documented one sleepwalker who described how "she was going to get run over by a truck", and "leapt out of bed and out of the mezzanine to avoid it". Another sleepwalker in the same study dreamed that her "baby was jeopardized," and "grabbed her [real] baby and ran out of the room with it". Yet another sleepwalker dreamed that she "was locked in a box she could not escape […,] felt herself suffocating" and "pushed back [against the real] walls around her" (Oudiettet, et al., 2009). In all of these cases, sleepwalkers report dream mentations that structurally correspond to their immediate physical environment (Pillmann, 2009). This suggests that somnambulic dream environments must overlap to some degree with real sensory input (Gaudreau et al., 2000; and Zadra, et al., 2013). Otherwise, by physically reacting only to supposedly "sensory isolated" dream mentations, sleepwalkers would not be able to simultaneously and accurately navigate both the oneiric and physical environment (Jaar, et al., 2010).

Dream actions including breathing, muscle twitches, and even embodied actions, stimulate the same neuronal responses as their physical counterpart (Fenwick et al., 1984; Laberge, et al.,1981;

Fenwick et al., 1984; Kripnner 2002: 37; and Pace-Schott et al., 2017). Because of this, in the absence of skeletal muscular paralysis, dream activity can —just as Jouvet had hypothesized— become physically enacted in the outside world (Harris et al., 2009).

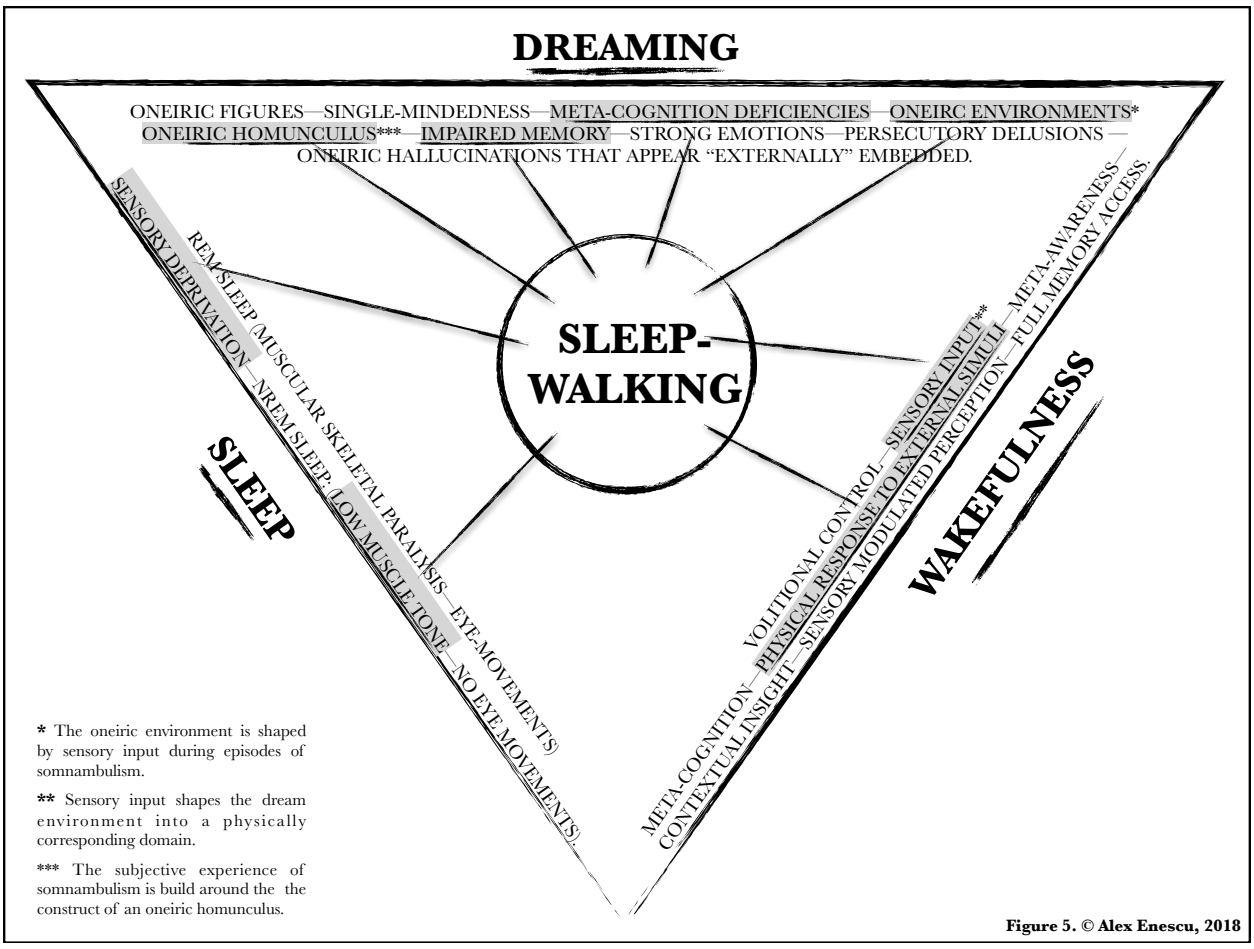


Figure 5. © Alex Enescu, 2018

Given that muscle atonia reaches its lowest point during SWS, and this is also when somnambulic activity reaches its peak, many propose that SWS dream mentations are the most likely internal source of somnambulic activity (Gaudreau, et al., 2000; and Zadra et al., 2013). This also suggests that SW is produced through the hybridization of three separate mental and physiological characteristics: (1) the physiology of SWS (i.e., reduced skeletal-muscular paralysis); (2) partial waking sensory modulation that feeds and shapes the spatio-temporal phenomenology of ongoing multi-modal dream mentations; and (3) the meta-cognitive deficiencies that are normally associated with dreaming (Bassetti et al., 2000, Szelenberger 2005, Harrist et al., 2009, Nobili et al., 2011 and Zadra et al., 2013). The imbrication of these three mental and physiological attributes give rise to a behaviourally functional temporary hybrid-state-of-awareness that can re-combine existing elements of sleep, wakefulness, and dreaming into a unique neuro-phenomenological perceptive state.

Lucid Dreaming (LD)

The term *lucid dreaming* was first used in 1913 by Dutch psychiatrist Frederik van Eeden (1860-1932). In *A Study of Dreams*, van Eeden reports having experienced more than three-hundred-twenty-five lucid dreams, during which he was able to fully recall the intricate details of his waking life, act voluntarily, and consciously navigate the dream world (van Eeden, 1913). Historically, this phenomenon had been observed and commented on by such diverse thinkers as Aristotle, St-Augustine, and in the 19th century by Marquis d'Hervey (1822-1892) in *Les rêves et les moyens de les diriger; observations pratiques*, to name a few (LaBerge et al., 1981; LaBerge, 1988; Stumbrys, et al., 2012; Hobson, 2015: 44; and Dresler, 2017: 539).

Lucid dream reports were met with skepticism by the scientific and academic community of the twentieth century (LaBerge, 1988; Bogzaran 2014; Hurd, 2014; and Dresler, 2017: 539). The phenomenon was not only at odds with the dominant Freudian dream theory that posited dreaming as an unconscious process, but it was also discredited due to its traditional association with occultism, esoteric, and magical practices (Kripnner, 2002; LaBerge, 2009; and Olsen, 2014). Lucid dream critics argued that lucidity, if real, would at best be an "illusory" sleep experience that is produced during periods of brief micro-awakenings (Antrobus et al., 1965; Schwartz et al., 1973; Hartmann et al., 1975; LaBerge et al., 1981; and LaBerge, 1988).

The phenomenon was nevertheless legitimized in the late 1970s when two independent researchers, Keith Hearne and Stephen LaBerge, discovered that dreamers can communicate with the outside world during REM sleep (Stumbrys et al., 2012). In particular, they demonstrated that REM sleep skeletal muscular paralysis does not affect eye movements, fingers, toes and feet, and that oneiric homunculus activity produces the same neuronal activity as real physical movements (Laberge, et al., 1981; Fenwick et al., 1984; Kripnner 2002: 37; Dresler et al., 2012; Pagel, 2014; and Pace-Schott et al., 2017). This insight was then used to test if LD occurs during unequivocal sleep or periods of micro-awakening. To everyone's surprise, electrooculogram (EEG) monitors revealed that lucid dreamers can remember pre-sleep tasks, regain metacognitive abilities, and intentionally signal back to the outside world through pre-determined volitional eye, finger, toe, and feet movements, during periods of unambiguous REM sleep (LaBerge et al., 1981; Fenwick et al., 1984; LaBerge 1988; Dresler, 2007; Dresler et al., 2011; Laberge, 2011; and Filevich et al., 2015).

The exact definition of a lucid dream remains elusive. It is unclear whether "lucidity" is best defined as: (1) the mere awareness of being in a dream; (2) the presence of self-reflection, self-evaluation, insight, and access to waking memories during sleep; (3) the ability to exert conscious volitional control over the overall dream plot, its content, and environment; or (4) a combination of

all of the above (Kahan et al., 1994; LaBerge, 1988; Kahn and Gover, 2010; Hobson et al., 2010; Dresler et al., 2012; Voss et al., 2013; Voss et al., 2014; Taitz, 2014; Sanders, 2014; Sparrow, 2014, Voss and Georg, 2014: 27; Filevich et al., 2015; Dresler et al., 2017; and Pace-Schott et al., 2017).

Most lucid dreams are marked by transient episodes of lucidity and varying degrees of control (Hobson, 2010). Meta-cognition during sleep is often described as a "balancing act"; it can easily disappear and just as quickly reappear (Barrett 1992; Hobson, 2003: 97; Kahn and Gover, 2009). Volitional control is not a binary —"all-or-nothing"— phenomenon either (Dresler, 2017). It varies in degree, reach, and comprehensiveness (Pagel, 2014; Hufford, 2014; and Dresler, 2017). In most circumstances, volitional control is limited to eye movements, direction of gaze and basic oneiric homunculus activity (Kripnner, 2002; and Hufford, 2014). In more exceptional circumstances, control may extend to manipulating oneiric landscapes or even the direction of the dream plot itself (LaBerge, 1990; Kripnner, 2002; Pagel, 2014; Hufford, 2014; Johnson, 2014; and Zimer, 2014).

The ability to control particular elements of oneiric activity while lucid implies that *other* elements must remain outside of conscious control. There is no evidence that "complete" control over a dream is possible. In fact, dream figures are notoriously difficult to control (Post, 2014; and Filevich et al., 2015). LD, therefore, is most accurately described as a distinct mental state *within* the phenomenological architecture of dreaming, rather than as the unequivocal ability to shape the totality of ongoing oneiric activity (LaBerge, 1988; Hobson, 2010; Voss et al., 2013; Voss, 2014; Bulkeley, 2014; and Erlacher, 2014).

LD is a rare phenomenon (Dresler et al., 2017). Less than one percent of the population reports regularly experiencing it (Schredl et al., 2011; Green et al., 1994; and Schredl and Erlacher, 2011). But, it is estimated that most people will experience at least one lucid dream during their lifetime (Green et al., 1994). In spite of this, the unpredictable nature of LD and its transience makes it more difficult to study than most other sleep related phenomena (Filevich et al., 2015).

LD can be somewhat reliably induced through at least three different methods (LaBerge, 1980; Dresler et al., 2012; Kahan and Laberge, 2011; and Stumbrys et al., 2012). The first is known as the "wake initiate lucid dream" (WILD) technique, and it consists of falling asleep with the explicit intent of having a lucid dream (LaBerge 1980; Hobson, 2003: 98; and Stumbrys et al., 2012). The second approach, the "dream initiated lucid dream" (DILD) method requires performing periodic mnemonic reality tests during wakefulness to help engrain the habit and remind the dreamer to become lucid once asleep (LaBerge 1980; Hobson, 2003: 98; and Stumbrys et al., 2012). The third method involves external intervention, namely administering external stimulation, either in the form of 40-Hz trancranial electrical current in the lower gamma band over the dorsolateral prefrontal

cortex (DLPFC) areas during REM sleep or through pre-determined light, acoustic, vibro-tactile, electro-tactile, and vestibular shocks, vibration, or flashes (Stumbrys et al., 2012; and Voss et al., 2014; and Dresler et al., 2017). All three methods have produced various degrees of success, but none guarantee lucidity (LaBerge, 1980; Kahan and Laberge, 2011; and Dresler et al., 2012).

On a phenomenological level, lucid dreams are reportedly more vibrant, colourful, and immersive than both wakefulness and non-lucid oneiric activity (Garfield 1995; LaBerge, 2009; Occhionero and Cicogna, 2011; Gackenback 2014, Olsen, 2014, and Hobson, 2015: 43). In fact, the experience is often described as being "more real than real" (Hobson et al., 2010: 40). What is more, lucidity can be maintained throughout multiple levels of dreaming, i.e., the dreamer can become lucid in a dream within a dream, and so on (Pagel, 2014).

Many regions that are deactivated during REM sleep, and are associated with a loss of self-awareness (e.g., the DLPFC and precuneus), are reactivated during lucid dreams (Kahn and Gover, 2010; Dresler et al., 2012; Voss et al., 2014; and Filevich et al., 2015). Specifically, LD is marked by bursts of alpha and gamma EEG frequency, greater cortical connectivity, —especially in the neocortical regions (i.e., lateral prefrontal, frontopolar, and metal parietal cortices)— increased activation in the precuneus, the bilateral dunes, and the occipitotemporal cortices, and high-frequency (40-Hz) EEG gamma band power over dorsolateral prefrontal areas (Tyson et al., 1984; Voss et al., 2009; Hobson, 2010; Kahn and Gover, 2010; Dresler et al., 2012; Pagel, 2014; Voss, et al., 2014; Dresler et al., 2017; and Pace-Schott et al., 2017).

While LD was first conceptualized as strictly a REM sleep phenomenon, LaBerge's original study mentions at least two subjects who became lucid during NREM sleep but were unable to signal back to the outside world (Pagel, 2014; Laberge, Taylor et al., 1981; and LaBerge, 1988). Subsequent research has confirmed that LD can occur throughout all stages of sleep, albeit, the ability to eye and muscle signal appears to be limited only to REM sleep (LaBerge, 1988; and Dresler et al., 2015). Furthermore, the cortical activity of LD appears to expand beyond all traditional sleep stages, simultaneously exhibiting both the physiological features of wakefulness and sleep (Hobson, 2003: 93; Voss, et al., 2009; Kahn and Gover, 2009; Hobson, 2010; Voss, et al., 2013; Voss, et al., 2014; Pagel, 2014; Filevich et al., 2015; Hobson, 2015: 42 & 46; and Dresler et al., 2017). The condition enables the emergence of such waking mental characteristics as full waking memory recall, self-reflection, and metacognition throughout periods of unambiguous sleep and unambiguous dreaming (Rechtschaffen, 1978; and Voss and Georg, 2014: 25-26). The fact that lucidity is a learnable skill, clearly demonstrates that functional hybridization is <u>not</u> contingent on the existence of structural brain abnormalities.

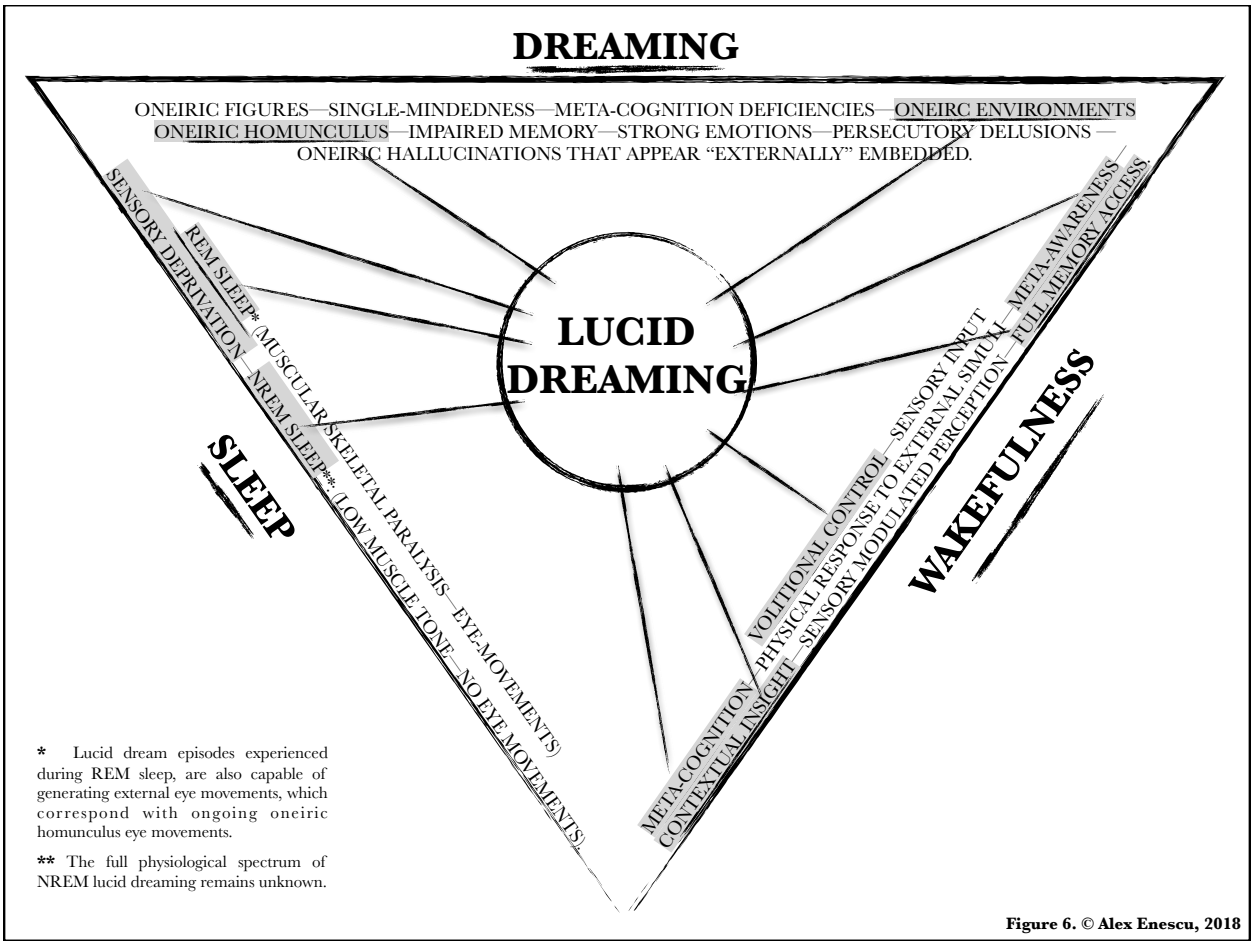


Figure 6. © Alex Enescu, 2018

SP/SW/LD Summary: Sleep paralysis, somnambulism, and lucid dreaming are all produced through a functional hybridization process. The unique psychological, cognitive, and perceptual characteristics of each hybrid-states-of-awareness demonstrates that components of wakefulness, oneiric activity, and sleep can become imbricated to create new phenomenological states. As Mahowald, et al., (2011b) explain, different arousal states: "are not necessarily mutually exclusive, and components of [wakefulness, REM sleep, and NREM sleep] may appear in various combinations, with fascinating clinical consequences". This observation is supported by data from local sleep studies which have shown that different parts of the brain can be simultaneously asleep and awake at the same time (Vyazovskiy et al., 2011; Krueger, et al., 2013; and Krueger, et al., 2019).

Sleep paralysis combines the physiology of REM sleep (muscular skeletal paralysis) with waking sensory modulation and such other facets of oneiric activity as externally embedded *and* sensory modulated oneiric humanoid entities, single-mindedness, meta-cognitive deficiencies, impaired memory, and delusional elements (see figure 4). These combined features foster the mistaken notion that a felt presence or a culturally-shaped conscious entity is monitoring,

interacting, or even attacking the "paralyzed" sleeper. During episodes of somnambulism, waking sensory input is redirected to directly modulate the content of oneiric activity. This roundabout form of sensory modulation establishes a concomitant oneiric "carbon-copy" of the physical world and allows the sleepwalker to simultaneously navigate both their immediate physical surroundings and their oneiric environment through the movements of their oneiric homunculus. During episodes of sleepwalking, oneiric meta-cognitive deficiencies and memory impairments, are integrated in the somnambulic state. This effectively induces a partial state of post-hoc amnesia. (see figure 5). Lucid dreaming, as opposed to somnambulism and sleep paralysis, occurs during unequivocal sleep. It integrates such elements of waking cognition as volitional control, meta-cognition, full access memory, and meta-awareness into the dream state. In this hybridized mental state, the dreamer gains an unusual degree of control over their oneiric homunculus, ongoing dream narrative, and (in rare cases) even the general dream environment (see figure 6).

Psychotic Symptoms of Schizophrenia as a Fourth Genre of Hybrid-States-of-Awareness

The existence of these three hybrid-states-of-awareness (see above) demonstrates that the various components of wakefulness, sleep, and dreaming can become functionally hybridized in order to form new phenomenological states —each with their own unique features and patterned experiential outcome. While hybrid-states-of-awareness are expressed in predictable forms, they nonetheless lack rigid boundaries, are multi-faceted, and exhibit different degrees of combinatory potential. For example, the meta-cognitive features of lucid dreaming will reliably induce vibrant, hyperreal, and euphoric visual perception regardless of either oneiric or external context. Somnambulism, regardless of the dreamer's physical circumstances, will reliably engender an indirect, peculiarly reversed, or backward spatio-temporal oneiric navigating system. Each hybrid-state-of-awareness produces its own unique symptomatological spectrum that exhibits relatively few inter-individual differences, but substantial intra-individual variation. For example, while sleep paralysis produces predictable visual hallucinations in the form of culturally conditioned humanoid apparitions, as opposed to volcanos, earthquakes, or other natural phenomena in nearly everyone, the nature of those apparitions, nevertheless, exhibit great intra-individual variability.

Hallucinations, delusions, and psychosis also share a remarkable number of cognitive and phenomenological similarities with dreaming. Much like these three hybrid-states-of-awareness, the psychotic symptoms of schizophrenia also tend to be experienced in predictable pattern forms, namely as disembodied auditory hallucinations, persecutory delusions, and, more rarely, as externally integrated visual hallucinations. The lack of structural abnormalities in people who suffer from

schizophrenia, and the fact that both hallucinations and delusions can occur outside of psychopathology, suggests that these conditions have a functional origin.

Here I raise the possibility that hallucinations, delusions, and psychosis belong to a fourth genre of hybrid-states-of-awareness. The psychotic symptoms of schizophrenia, their universal, (albeit, diaphanous forms), their predictable symptomatological forms, and their intra-individual variation are best accounted for through a functional hybridization model. In fact, since dreaming reproduces the complete spectrum of psychotic symptoms, it makes little sense to look for their neurophenomenological origins in other architectural domains. There is no other known endogenous modality capable of reproducing the full symptomatological spectrum of hallucinations, delusions, and psychosis in such minute detail!

The explanatory framework of hybrid-states-of-awareness also helps solve some of the taxonomical limitations in the classification of psychiatric conditions. In particular, the psychotic symptoms of schizophrenia are only diagnosed as "pathological" *if* they interfere with a person's lifestyle and productivity. Supportive and soothing auditory hallucinations, for example, are not considered pathological, despite being phenomenologically indistinguishable from their threatening counterpart. Under the current classificatory system, phenomenologically undifferentiated experiences are disjoined and evaluated based on their disruptiveness, instead of their core neuro-phenomenological features or characteristics. This means that psychopathological research is currently focused on the symptomatology of abnormal perception, as expressed by a single personality (i.e., the affected type), rather than the larger underlying structural displacement of phenomenological perception that takes place in these individuals.

A functional hybridization model of the psychotic symptoms of schizophrenia can account for the full neuro-phenomenological spectrum of these symptoms without having to rely on post-hoc diagnostic methodologies. Such a model would not only help bridge the gap between pathological and non-pathological forms of perceptual alterations, but also enable systematic analyses of cross-cultural nosological variations of abnormal perception(s). Indeed, within the context of a "hybridization" model, research psychiatrists could abandon the fruitless endeavour of mapping the near-limitless intra-individual variational features and characteristics of schizophrenia, and instead focus on mapping out the basic neuro-phenomenological signs of functional hybridization. Moreover, a hybridization model could lead to the formulation of such new treatment methods as functional "de-hybridization therapeutic techniques", i.e., processes through which the symptomatological manifestation of abnormal perception can be systematically reversed rather than

merely attenuated through the administration of existing antipsychotics (see p. 90 for more details on this).

Section IV Summary

This section presented evidence for the existence of at least three hybrid-states-of-awareness: sleep paralysis, sleepwalking, and lucid dreaming. The existence of these imbricated perceptual states suggests that the elementary components of wakefulness, sleep, and oneiric activity can become functionally imbricated (in the absence of structural abnormalities) to create admix phenomenological states. These findings suggest that the psychotic symptoms of schizophrenia, namely waking hallucinations, delusional thinking, and psychosis are also likely to be created through a similar imbrication process. The last section of this work will provide an explanatory framework, "oneiric release", through which the process of hybridization is accounted for.

SECTION V

ONEIRIC RELEASE AS POSSIBLE IMBRICATION MECHANISM

Section V Introduction

Functional hybridization may account for the phenomenological similarities between oneiric activity, and the psychotic symptoms of schizophrenia better than other models do, but, it does not explain how hybridization occurs. In the following section, I present an original theory to explain, rather describe, one possible interpretation of the hybridization process. Oneiric Release Theory (ORT) combines the collective insights of (1) previous oneiric formulations of schizophrenia; (2) recent studies demonstrating that the formation of oneiric activity is not contingent on any particular sleep stage; (3) evidence that components of wakefulness, sleep, and oneiric activity can become functionally hybridized to create hybrid-states-of-awareness; (4) Rodolfo Llinás' conceptualization of wakefulness as a sensory modulated dream state; (5) Louis West's perceptual release theory of hallucinations; and (6) Hughlings-Jackson's theory of psychosis.

The first three axioms of ORT were presented in previous sections. Briefly restated: (1) dreaming and the psychotic symptoms of schizophrenia exhibit nearly identical phenomenologies; (2) the underlying architecture of oneiric activity can extend well beyond the neurophysiology of sleep; and (3) the neuro-phenomenological characteristics of oneiric activity, sleep, and wakefulness can become functionally "imbricated" to create new perceptual states.

The remaining three pillars of ORT are developed in the following pages and summarized on p. 99 of this work.

Llinás' Formulation of Wakefulness as a Sensory Modulated Dream State

Llinás first proposed his formulation of wakefulness as a sensory dream state by drawing on the observation that oneiric activity and waking cognition appear to influence each other and exhibit similar cognitive and sensory features (Llinás et al., 1991; Hobson, 2003: 119; Schredl, 2010; Christoff et al., 2011; Palagini, 2011; and Hobson, 2015: 233). Indeed, it has become clear that such "exclusive-waking-features" as the availability of the full phenomenological spectrum of sensory cues, speech, the experience of embodiment, perceptual brightness, colours, and even clarity, can be experienced to the same degree of intensity and vividness during dreams; and that such "exclusive-oneiric-features" as deficiencies in meta-cognitive awareness, memory retrieval system, and volitional control over mental content can equally manifest themselves during wakefulness (Rechtschaffen 1978; Hunt, 1989; Meier, 1993; Kahan, 1997; Zadra et al., 2006; Domhoff, 2011; Kahan and Laberge, 2011; Gerrans 2012; and Revonsuo et al., 2015). In fact, repeated studies have shown that oneiric activity and waking mental states display homogenous "processing levels of cognition", similar thought pattern distributions, nearly identical lapses in self-reflective consciousness,

rationality, and volitional control (Oswald, 1962c; Hall, 1966; Llinás et al., 1991; Kahan et al., 1994; Simon and Chabris, 1999; Schredl, 1999; Revonsuo, 2000; Schredl, 2003; Strauch and Meier, 2004; Schredl; 2010; Nir et al., 2010; Kahan and Laberge, 2011; Noreika, 2011; Palagini, 2011; Hobson, 2015: 121; and Bulkeley, 2017).

Such studies as Simons' and Chabris's (1999) attention test clearly demonstrate that "waking consciousness is just as fraught with discontinuity and incongruity as is dreaming" (Simons, 2010; and Hobson, 2015: 121). Conversely, the empirical validation of lucid dreaming has demonstrated that focused attention, voluntary control, self-awareness, reflective thinking, proper memory retrieval, and high-order cognition can equally be fostered during periods of unambiguous sleep (Kahan, 1971; Nir et al., 2010; and Kahan and Laberge, 2011). These collective findings support the conclusion that the perceptual, cognitive, and phenomenological features of wakefulness and oneiric activity do not exist in isolation of each other and can become interchanged.

Brain injury studies further corroborate this. In particular, waking perpetual and cognitive losses caused by structural damage produce deficiencies in corresponding oneiric features. For example, visual impairments caused by damage to the medial occipitotemporal regions or to the right parietal lobe not only impairs a person's ability to visualizing motion, see colours, and engage in proper facial recognition while awake but also in analogous dream processes (Llinás et al., 1991; Nir et al., 2010; and Domhoff, 2011).

Child developmental studies have shown that the "oneiric cognitive range" of children is closely paralleled by the development of associated waking abilities (Nir et al., 2010; and Domhoff, 2011). These findings indicate that (1) brain specialization does not alter between states, and (2) similar, if not identical, brain structures are involved in the generation of analogous perceptual, cognitive, and sensory experiences (Llinás et al., 1991; Kussé et al., 2010; Domhoff, 2011; Palagini, 2011; and D'agostino et al., 2013b).

Such researchers as Cartwright (1981), Langer (1989), and Kahan and LaBerge (2011) have long argued that the cognitive, perceptual, and sensory features of oneiric activity and wakefulness are better differentiated in terms of "degree" rather than "kind" (Kleitman, 1963; Kirpke & Sonnenschein, 1973; Folks & Fleisher, 1975; Cartwright, 1981; and Langer, 1989). In fact, many of these researchers propose that the differences in degree between the two states is better understood as the result of wakefulness being "entrained" by its external environment in a way that dreaming is not (Calkins, 1896; Llinás et al., 1991; Kerr, 1993; LaBerge, 1998; Franklin et al., 2005; and Kahan and Laberge, 2011). In other words, it is likely that the "continuity and congruity of waking

consciousness […] likely derive from the relative stability of the outside world", and not from waking consciousness *per se* (Hobson, 2015: 25).

On a neurophysiological level, models of wakefulness as a sensory modulated dream state reflect the fact that the central nervous system (CNS) is "primarily self-activating and capable of generating a cognitive representation of the external environment even in the absence of sensory input" (Llinás and Smith, 1996, 4). From this perspective, dreaming can be seen as an epiphenomenon of basic CNS activity (Flanagan, 1995; Llinás and Smith, 1996, 5; Mahowald et al., 1998; Flanagan, 2000; Valli et al., 2005; Pagel, 2014; Nir et al., 2010; Schredl, 2010; Valli 2011; Gerrans 2012; and Hobson, 2015: 28). With this in mind, Llinás argued that sensory input should be interpreted as a mechanism that modulates endogenous CNS activity (i.e., oneiric activity) into externally congruent perceptual fields (Llinás et al., 1991; and Llinás and Smith, 1996, 4). Llinás' model posits that perception can be seen as a "closed-loop" oneiric system that never directly interacts with the external world; or as he puts it: "we are basically dreaming machines that construct virtual models of the real world" (Llinás et al., 1991; Llinás and Smith, 1996, 5; Llinás, 2001: 94; and Llinás, 2009: 30). The underlying implications of this statement is that external objects can never be experienced as they really are, but must (instead) be matched to corresponding endogenous perceptual "themes and motifs" (Llinás, 2001: 192 & 193; Llinás, 2009: 30; Pagel, 2014; Hobson, 2015: 24; and Blom, 2015b). Put differently, external perception is constructed from oneiric endogenous architectural templates that are likely to vary on an inter-individual level —think about the perceptual differences that were revealed by the recent "Yanny versus Laurel" or the "white-and-gold/black-and-blue dress" internet debates (Llinás et al., 1991; Llinás and Smith, 1996, 6; Llinás, 2001: 94, 124, & 161; Windt and Noreika, 2011; Blom, 2015b; Hobson, 2015: 23; Jonauskaite et al., 2018; and Pressnitzer et al., 2018). In the absence of external input and content, endogenous brain activity cannot generate a reality-corresponding perceptual field and "dreaming" ensues (Llinás et al., 1991; Llinás, 2001: 94 & 130; and Blom, 2015b).

One advantage of this model is that it provides an explanatory framework that posits the perceptual, cognitive, and psychological differences between oneiric activity and wakefulness as a question of different degrees of sensory and cortical activation, the partial blockade of external sensory input, or brainstem neuro-modulation (Llinás et al., 1991; Schredl, 2010; and Gerrans, 2012). In other words, it portrays dreaming and wakefulness as expressions of the same underlying architectural substrate.

West's Perceptual Release Theory and Hughlings-Jackson's Theory of Psychosis

If wakefulness is a sensory modulated dream state as Llinás proposes, then "breaks in reality" must be produced through the demodulation of oneiric activity. A modified version of this view was first proposed in the 1950s by Louis Anthony West (West, 1975: 299). The Perceptual Release Theory (PRT) model states that sensory modulatory activity inhibits endogenous memory traces from "overwhelming" wakefulness (West, 1975: 299; and Mahowald et al., 1998). West proposed that sensory modulation is the primary mechanism through which internal mentations are shaped into "reality corresponding" mental experiences (West, 1962). Consequently, when sensory input is absent or diminished (including through excessive affective experiences), their underlying pathway becomes "demodulated" and trigger the "release" of memory traces (Assad, 1986; Mahowald et al., 1998; and Goldsworthy and Whitaker, 2015). Under this formulation, "hallucinations can be characterized as […] precepts that are insufficiently restrained by information from the senses" (Schacter, 1976). As West puts it, "in such instances, when the usual information input will no longer suffice completely to inhibit their emergence, perceptual traces may be 'released' and re-experienced either in familiar or new —even bizarre— combinations" (West, 1975: 300-301).

PRT's framework is predicated on two fundamental assumptions: (1) memory traces are permanently engraved in the brain and form the basis of endogenous phenomenological perception; and (2) phenomenological perception is continuously shaped by the interplay between internal and external "psychobiological" forces (Assad, 1986; and Mahowald et al., 1998). As West explains: "When sensory input is available it has an organizing effect on screening and scanning brain mechanisms. However, when the level of sensory information input is diminished, its organizing effect is also attenuated, and perceptual (neural) traces can become 'released' into wakefulness" (West, 1962). PRT thus explains hallucinations as being exclusively caused by the "perceptual release" of stored memory traces (West, 1975: 300-301).

West's model posits that hallucinatory perception occurs when: (1) the partial disinhibition of a particular sensory pathway takes place either on a functional or structural level, and (2) a sufficient degree of arousal (i.e., higher-order cognition) is maintained in other sensory and cortical pathways (Blom, 2015b). When these two conditions are met, endogenous mentations may become "hybridized" (my own) with ongoing sensory and higher-order cognitive input. This mechanism would explain why hallucinatory mentations are experienced in an integrated manner (veridically), i.e., because they are produced through the same sensory pathways that enable real perception.

PRT can also be seen as a mechanism through which the brain compensates for stressed, absent, or over-active sensory input by "releasing" corresponding endogenous cues (Waters et al.,

2016; and Pace-Schott et al., 2017). In particular, West argued that when "a general level of cortical arousal persists to a sufficient degree [during periods of sensory demodulation], the 'released perception' can enter awareness and be experienced as fantasies, illusions, visions, dreams, or hallucinations. The greater the level of arousal, the more vivid the hallucinations will be" (West, 1975: 301). Put differently, the difference between waking hallucinations, mind-wandering activity, and dreaming can be gauged by the amount of "cortical arousal" and sensory demodulation that takes place. Under normal circumstances, sensory activity and cortical arousal must undergo a certain degree of proportional demodulation to produce such non-pathological "perceptual-state-shifts" as wakefulness and sleep. When demodulation does not occur in a proportional manner, the balance between cortical arousal and sensory modulation becomes sufficiently incongruous as to generate abnormal perception. In other words, PRT presents abnormal perception as the product of divergent cognitive and sensory features that coalesce to generate a combinatory interplay between elements of wakefulness (arousal) and sleep (sensory demodulation).

PRT, however, is a theory developed to explain hallucinations. It does not extend to psychosis or other cognitive abnormalities. Even so, West's model can be expanded by incorporating it into the framework proposed by Hughlings-Jackson's theory of psychosis. Hughlings-Jackson argued that psychotic symptoms are produced whenever the inhibitory influence of higher cortical-control centres become "dissolute" (Assad, 1986). The model posits that such more recent evolutionary parts of the brain as the neocortex are responsible for "modulating" or "regulating" the more "primitive parts" (Andreasen, 2011). As Gilet and Franz (2013) explain, "Hughlings-Jackson suggested that conscious life involves memory, will, reason, and emotion which, when impaired, each results in partial dissolution of higher mental functions and causes both [the] negative and psychotic symptoms [of schizophrenia]". Put differently, Hughlings-Jackson argued that the demodulation of higher-cognitive centres enables "evolutionary anterior" forms of cognition to become the forefront of brain activity.

In many ways, this model can be seen as a "cognitive" version of West's PRT. Both models assume that the "dissolution/demodulation" of sensory pathways or "higher-order-cognitive-centres" is responsible for the "release" of abnormal perceptual/cognitive elements into wakefulness. Both models infer that wakefulness is a compounded phenomenon that is derived from more "primitive" or endogenous cognitive/perceptual structures. And both models imply that wakefulness is a "brittle" perceptual state. They also share the same limitations. Neither West's PRT nor Hughlings-Jackson's model of psychosis gives a satisfactory account of what exactly constitutes

the underlying structure of wakefulness. Moreover, they both fail to answer as to what exactly becomes "released/disinhibited" during periods of "cognitive disinhibition" or "perceptual release".

West attempted to overcome this lacuna by proposing that stored memory traces could be the phenomenological origin of hallucinations. This hypothesis, however, has at least three flaws: (1) Waking hallucinations do not interact with memory traces in any substantial or direct manner; (2) the content of hallucinatory perception is predominantly influenced by immediate circumstances, not past experiences; and (3) waking hallucinations are much more likely to complement ongoing sensory experiences than to diverge from them (Blom, 2015b). Indeed, periods of sensory deprivation do not implicitly induce memory recall. They trigger the release of imagioneiric mentations! Hughlings-Jackson's model of psychosis also has significant shortcomings. It cannot account for (1) the dichotomization of cognitive processes in the absence of structural damage, (2) the interdependent relationship between "higher-inhibitory" structures and older brain structures, and (3) the fact that the phenomenology of dreams, which is inherently "psychotic", is built out of the same underlying architecture that generates wakefulness. In other words, both models lack an elemental framework through which the phenomenology of wakefulness can be sustained in the absence of modulatory activity, i.e., they fail to breakdown the phenomenology of wakefulness into its "pre-modulated" form (Assad, 1986; and Andreasen, 2011).

Oneiric Release Theory (ORT)

The remainder of this section lays out the groundwork for a new "demodulatory" model that posits oneiric activity as the underlying architecture of wakefulness. Oneiric Release Theory (ORT) borrows components from Llinás' formulation of wakefulness as a sensory modulated dream state, West's Perceptual Release Theory, Hughlings-Jackson's higher-centres-dissolution formulation of psychosis, and elements of Dement's original REM sleep intrusion hypothesis of schizophrenia. ORT can account not only for the psychotic symptoms of schizophrenia, but also offers an explanatory framework for the structural processes that are involved in the generation of such hybrid-states-of-awareness as sleep paralysis, somnambulism, lucid dreaming, hypnagogia, and mind wandering activity.

ORT posits that wakefulness is a porous perceptual state balanced by continuous modulation of endogenous imagioneiric activity through sensory and higher-order cognitive input. Borrowing from West's PRT and Hughlings-Jackson's theory of psychosis, the model asserts that structural or functional sensory and higher-order cognitive impairments "disinhibit" the congruity between wakefulness and external reality. More specifically, when a particular sensory pathway becomes

incongruously disinhibited and other sensory pathways, or components of higher-order cognition do not, a hybridized field of perception is created. Put differently, ORT asserts that the phenomenological origins of hallucinations and abnormal mental activity are the integration of demodulated imagioneiric mentations and disinhibited higher-order cognitive activity within the perceptual context of a partially modulated state of arousal.

The model uses the insight that imagioneiric mentations can be interpreted as an epiphenomenon of basic CNS activity to expand Llinás' original formulation of wakefulness as a sensory modulated dream state to the entire imagioneirc continuum (Flanagan, 1995; Gillespie, 1997; Hobson, 1999: 131; Domhoff, 2003: 20; Hobson, 2003: 9; Tononi and Laureys, 2009; Damasio and Meyer, 2009; Searle 2015; and Revonsuo et al., 2015). Put differently, ORT presents wakefulness as a sensory modulated imagioneiric state.

The process of oneiric release is best illustrated through the gradient demodulatory nature of sleep, hypnagogia, and mind-wandering activity. During sleep both higher-order cognitive activity and sensory input gradually become "disinhibited" or "demodulated" (Kahn and Gover, 2010). The same process takes place during mind-wandering episodes and hypnagogia, albeit to a lesser extent. In all of these cases, sensory demodulation occurs in congruity with the dissolution of higher-order cognitive activity (Hernández-Peón, 1967; Ziskind, 1970: 149; Hobson, 1999: 75; Hobson, 2003: 57; and Pagel, 2014). Demodulation is not an "all or nothing process" (Hernández-Peón, 1967). Each sensory and higher-order cognitive system is modulated independently. Different perceptual states are produced at different stages of demodulation. As such, perceptual alterations are inherently dependent on the demodulatory process. Sleep, hypnagogia, and mind-wandering activity are produced when sufficient sensory and higher-order cognitive pathways have become demodulated in congruity to each other; conversely, when the same systems are incongruously demodulated, a hybrid-state-of-awareness forms.

ORT explains why dreaming, hybrid-states-of-awareness, and the psychotic symptoms of schizophrenia display such remarkable intra-individual features, patterns, and motifs, i.e., their content and structure is built out of the same underlying neurophysiological architecture (Hartmann, 1975; Hobson, 2015; 26; and Blom, 2015b). ORT also explains why uni-modal sensory demodulation will produce proportional and compensatory hallucinatory mentations in specific sensory modalities. When demodulations occur in a specific pathway, their underlying oneiric activity is "released" from the "constraints" of external input. Since the phenomenological properties of sensory pathways foster a "phenomenological whole", when incongruous demodulation occurs, the unmodulated "portion" of that particular pathway becomes "approximately" matched to the rest of

ongoing sensorial, cognitive, and perceptual impressions. Here, ORT draws on West's insight that abnormal perception is primarily compensatory in nature and occurs in proportion to the degree of inhibition.

The source of demodulation is either structural or functional. The former include brain injuries and sensory pathway impairments. The latter (functional demodulation) is an organic process that can generate abnormal perceptual cues even in the absence of structural abnormalities. During sleep, for example, all sensory and higher-order cognitive pathways undergo a significant degree of functional demodulation (Kahn, and Gover, 2009). When this occurs, oneiric activity becomes "de-anchored" from sensory input and leads to the generation of "unrestraint" mental content (Hobson, 2015: 25).

The degree of functional demodulation at any given moment varies enormously. Functional modulation levels periodically change during wakefulness, in between sleep stages, and even during single sleep stages (Hérnandez-Péon, 1967). Moreover, perceptual states possess an inherent ability to quickly alternate between themselves (Hobson, 2003: 153; Domhoff, 2011; and Lee, 2012). Think about how a sufficiently loud noise will immediately awaken you from sleep. The delineation of perceptual states into such specific categories as mind-wandering, oneiric activity, or hypnagogia, is predicated on normal mental activity exhibiting recognizable degrees of modulatory congruity at any given moment (Hobson, 2003: 153 & 234; Kussé et al., 2010, and Lee, 2012). Conversely, the asynchronous demodulation of any particularized sensory or cognitive pathway will generate a mental state that becomes labelled "abnormal" or as "pathological".

Cognitive and sensory asynchronous modulatory activity is what takes place during episodes of sleep paralysis, somnambulism, mind-wandering activity, and lucid dreaming. During periods of sleep paralysis, for example, motor activity remains disinhibited while such other sensory pathways as visual or auditory systems become partially restored. This enables partially modulated sensory mentations to become imbricated within the larger phenomenological domain of REM sleep. Conversely, during somnambulic episodes, partial sensory modulation and motor output can be restored in the entire absence of cognitive modulation. During mind-wandering episodes, near complete higher-order cognitive demodulation occurs in conjugation with partial sensory and muscular demodulation. This is evidenced by the fact that mind-wanderers can successfully navigate their immediate physical surroundings while remaining cognitively disconnected from their external environment. During lucid dreams, higher-cognitive systems are restored during a period of near total sensory demodulation. This allows individuals to consciously navigate a world whose phenomenological structure is produced in isolation from external reality. In the case of psychotic

symptoms, higher-order cognitive disinhibition and such specific sensory demodulation as visual or auditory demodulation occur incongruously. This creates a partial "disconnection" from the external world, i.e., modulated sensory systems produce "reality-corresponding" mentations, while unmodulated sensory pathways generate hallucinatory content.

It is important to note that even though the combinatory potential of cognitive and sensory modulatory activity can vary immensely, the process cannot produce content that extends beyond the phenomenological boundaries of oneiric activity. This explains why the patterned themes and motifs of hybrid-states-of-awareness (including psychosis) are remarkably similar, if not entirely identical, to those that are exhibited by oneiric activity. This also limits, at least theoretically, the total number of combinatory cross-over potential that can be generated in each sensory and higher-order cognitive pathway. Hybridization occurs in predictable patterns because there are only so many different ways in which the same systems can become imbricated.

Empirical data in support of ORT already exists. Ikuta et al., (2015) found that the same underlying structural regions that are activated by external sound, speech, and dialogue (i.e., the terminal of the primary auditory cortex, and both the Broca and Wernicke's areas), are also active during periods of oneiric auditory activity (Corballis, 2014; and Blom, 2015b). Moreover, damage to the peripheral areas of the auditory system has been shown to contribute to the formation of auditory hallucinations (Corballis, 2014; and Goldsworthy and Whittaker, 2015). PET scans in individuals who suffer from structural auditory impairments also reveal that the same underlying neural networks that are active during normal auditory input, are equally activate during periods of auditory hallucinations (Goldsworthy and Whittaker, 2015).

Evidence that structural demodulation is an inherent neurophysiological characteristic of schizophrenia is also readily available. For example, decreased blood perfusion in orbital-frontal regions has been observed in people who suffer from the condition (Bob and Mashour, 2011). This discovery indicates that higher-order cognition within the context of schizophrenia is likely "suppressed through orbital-frontal inhibitory impulses on posterior cortical areas" (Bob and Mashour, 2011). Visual and auditory information-processing deficits have also been discovered in people who suffer from schizophrenia (Yeap et al., 2008; and Turetsky et al., 2009). These findings suggest that sensory and higher-order cognitive demodulation negatively affects the organizational links between associative neural networks and that "neural discharges" can alter "activity in both sensory and motor pathways" (Bob and Mashour, 2011).

ORT, however, is not restricted to the domain of psychopathological perceptual abnormalities. One of ORT's major strengths is that it can distinguish between pathological and

non-pathological perceptual alteration based on the conjunctive modulatory relationship between sensory and concomitant higher-order cognitive activity. In the formal context, sensory demodulation is accompanied by loose-associative thinking, lapses in meta-awareness, and impairments in memory retrieval systems. When this happens, sensory-compensatory imagioneiric mentations (hallucinations) can easily be mistaken for real external cues. These cues can lead to the formation of secondary delusions and psychoses. In the latter context, sensory demodulation occurs outside of higher-order cognitive demodulation and individuals are able to correctly identify incongruous elements within their perceptual field.

This facet of ORT is best illustrated by the Charles Bonnett Syndrome (CBS). People who suffer from CBS experience such vivid perceptual abnormalities as visual and auditory hallucinations. These individuals, however, do not misinterpret their hallucinations as real external phenomena, and they rarely experience delusions or psychoses (Chen et al., 1996; and Corballis, 2014). Clinically, these types of hallucinations are referred to as "pseudo-hallucinations", or "lucid hallucinations" (Kraepelin, 1915: 4, 9, & 10; Mahowald et al., 1998; and Arango and Carpenter, 2011).[5] CBS is the result of structural damage to specific sense organs or to their underlying sensory pathways (Chen et al., 1996; Corballis, 2014; and Goldsworthy and Whittaker, 2015). The intensity of hallucinatory mentations are experienced in direct proportion to the degree of structural impairment (Corballis, 2014). Because CBS does not affect higher-order cognitive systems, people who suffer from the syndrome can correctly identify abnormal elements within their perceptual field. They do not mistake their hallucinations for reality.

People who suffer from CBS regularly report encountering humanoid entities (Corballis, 2014; and Goldsworthy and Whittaker, 2015). Similar experiences are also reported by people who suffer from minor structural damage to their visual sense organ (Mahowald et al., 1998; Chen et al., 1996; and Goldsworthy and Whittaker, 2015). The hallucinated figures bear a striking similarity to the humanoid entities reported by people who suffer from episodes of sleep paralysis. The similarities indicated that in both cases the content of hallucinatory experiences is likely to emanate from the same underlying neurophysiological "thematic bank".

Furthermore, these similarities also account for the effectiveness of such antipsychotic medication as carbamazepine to decrease the severity and intensity of hallucinatory activity in people who suffer from CBS and visual impairments (Chen et al., 1996; and Mahowald et al., 1998).

[5] It should also be noted here that the syntactical similarity between "lucid hallucinations" and "lucid dreaming" is not entirely irrelevant. In fact, during both experiences hallucinatory perception is correctly identified as "non-real". This is due to the fact that these types of hallucinatory mentations are experienced within the context of "uninhibited" higher-order cognitive activity.

Carbamazepine is also effective in treating waking hallucinations within the context of schizophrenia. Its cross-over effect suggests that both functional (and structural) induced hallucinations are generated through similar underlying structures.

The modulatory framework of ORT also explains the perception of oneiric embodiment. In fact, the presence of an oneiric homunculus is often taken for granted through the usage of the pronoun "I" in verbal dream reports e.g., "I moved", "I ran", "I saw", "I entered a new room", etc., (see Section III for more details). Furthermore, oneiric homunculi are perceptually indistinguishable from their sensory modulated counterpart (*ibid*). This should come as no surprise, given that the "body" is embedded at the deepest structural level and is represented in many areas of the brain, especially the cerebral cortex, the brainstem, and thalamus (Tononi and Laureys, 2009). In this case, the ORT framework posits that demodulatory activity that affects corporeal perception will invariably be substituted by an oneiric representation of the impaired body part. This is best exemplified by the "phantom limb" phenomenon "reported in patients who have lost extremities or who for other reasons have lost sensory information coming from parts of [their] body" (Mahowald et al., 1998). 100% of spinal cord injury, over 90% of amputees, and 23% of post-mastectomy patients report such an experience (Jarvis, 1970; Melzak, 1992; and Blumberg, 2017). In most cases, the "phantom limb" is manifested as a hallucinatory replica of the missing body part. Interestingly, just as with "oneiric body parts", patients report being able to exert volitional control over their phantom limbs (Goldsworthy and Whitaker, 2015; and Kikkert et al., 2017).

On a perceptual level, phantom limbs are capable of displaying the same spectrum of sensations as physical limbs (Andoh, et al., 2017). In fact, many patients "feel the lost limb so vividly that only when they reach out to touch it, or peer under the bedsheets to see it, do they realize it has been cut off" (Katz and Fashler, 2015). The prevailing explanatory framework of the phantom limb phenomenon assumes that the brain continues to generate the experience of missing (or impaired) body parts even in their absence and that "structural pathway demodulation" is responsible for this process (Mahowald et al., 1998, Katz and Fashler, 2015; Goldsworthy and Whitaker, 2015; and Andoh et al., 2017). As Mahwowald et al., (1998) explain: "It is clear that these sensations are arising from the higher levels of the CNS, as lesions of the peripheral nerves, spinal rootlets, pathways within the spinal cord, areas of the thalamus, and even extirpation of the primary sensory cortex". In other words, corporal perception is produced endogenously, and because its phenomenological expression is not dependent on peripheral CNS activity, amputated body parts can continue to be experienced even in their absence.

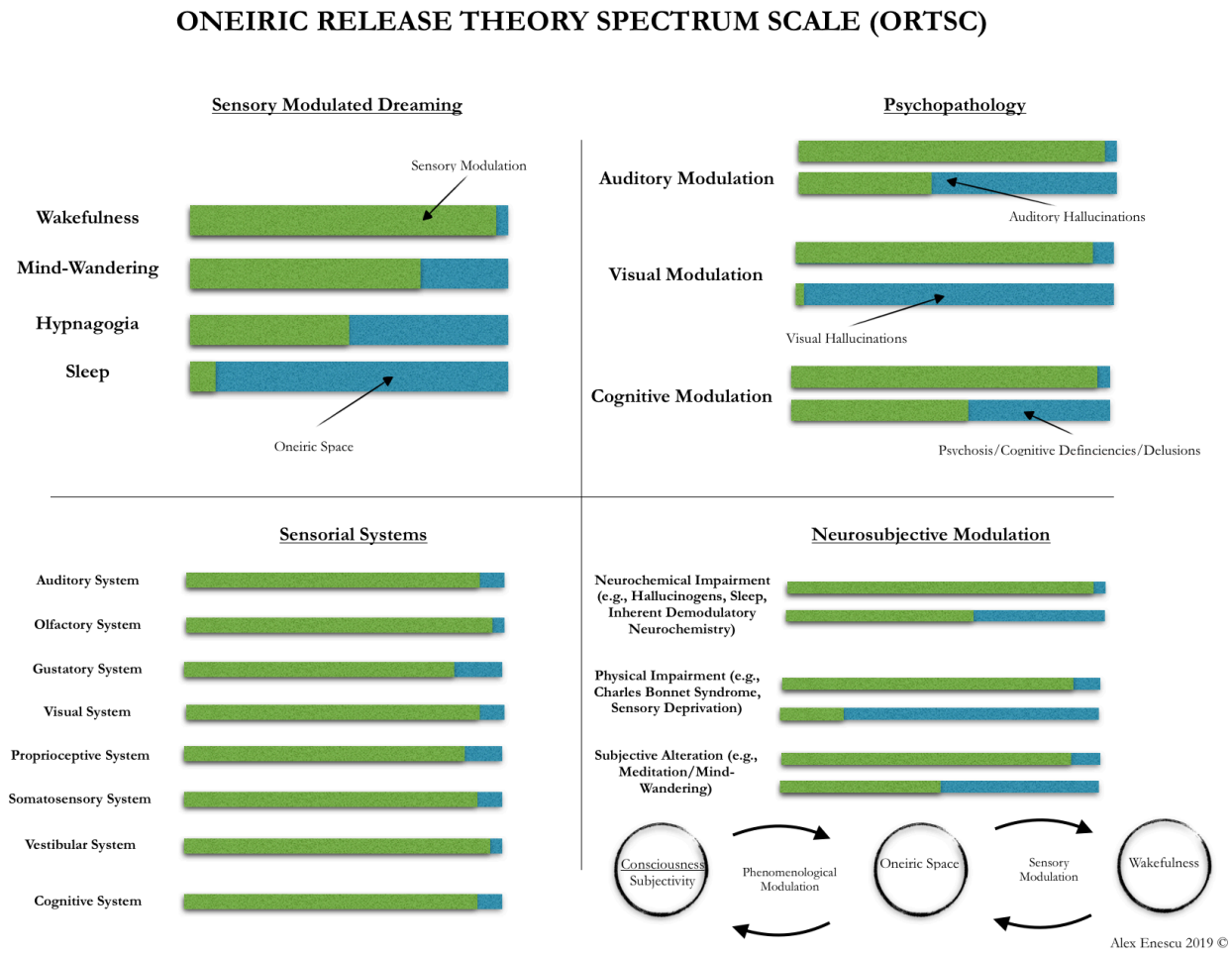
This interpretation is consistent with ORT. Specifically, if an oneiric homunculus forms the underlying phenomenological architecture of waking embodiment, as ORT suggests, it then follows that during periods of incongruous "peripheral demodulation" (i.e., demodulation that does not occur in congruity with other sensory pathways or body parts), oneiric-embodied-activity will continue to be generated in the affected pathways alongside correctly modulated sensory and bodily perception.

It is also important to note that functional or structural bodily impairments can occur at any point in a sensory pathway (Andoh et al., 2017). They can occur at the extremities, in the spinal cord, or near the receptor area for each sense impression (Mahowald et al., 1998; and Blumberg, 2017). In all of these cases, however, the underlying neural circuitry that is responsible for generating the phenomenological content of that particular sense organ or body part is left intact, and will continue to produce unmodulated oneiric activity. This explains why all forms of sensory or bodily impairments can contribute to the formation of objectless perception (Kubzansky and Leiderman, 1961; Freedman et al., 1961; Smith et al., 1961; Cranin, 1979; Mahowald et al., 1998; Limosani et al., 2011; and Corbalis, 2014). Furthermore, these findings strongly suggest that the underlying mechanism involved in the production of abnormal perception is predicated on the interplay between endogenously generated oneiric activity and the degree to which this activity can be "anchored" in external reality through sensory and oneiric homunculus modulation (see ORTSC below).

It should be reemphasized here that functional demodulation is an innate feature of normal brain dynamics. This is evidenced by both the rapid perceptual state alteration that occurs on a moment-to-moment basis in the form of mind-wandering activity and the rapid transitional pace that can occur between wakefulness and sleep. All together, these kinds of abrupt "perceptual shifts" indicate that functional modulation is sufficiently "dynamic" to produce multiple demodulator injunction points throughout a wide range of mental contexts.

If this holds true, then it follows that any process (endogenous or exogenous) that enables sensory, bodily, or higher-order cognitive modulation, must act as a counterbalance to functional demodulation and consequently be able to counteract, or even reverse, the formation of abnormal perception. This effect has been documented in the psychiatric literature at least since the late nineteenth century. Indeed, Kraepelin himself noted that patients who suffer from dementia praecox and are also engaged in manual or mental employment tend to display fewer psychotic symptoms (Kraepelin, 1915: 5). The same phenomena was observed throughout the twentieth century in many asylums, including the Pennsylvania Hospital (Rothman, 1971: 145-146). More

recently, it has been observed that patients who suffer from schizophrenia can stop their visual hallucinations by engaging in such visual reinforcement activities as counting ceiling tiles (Hobson, 2003: 80-81).



These types of clinical observations have been confirmed in at least one empirical study (Popova et al., 2011). Specifically, Popova et al., (2011) discovered that people who suffer from schizophrenia were able to normalize their sensory and cognitive functions by interacting with a computer-based program designed to reinforce higher-order cognitive and sensory pathways (Popova et al., 2011). Some researchers hypothesize that catatonic schizophrenia and other repetitive vestibular forms of behaviour may act as a form of self-administered sensory-reinforcement therapy (Lovaas et al., 1987). As Lovaas, et al., (1987) explains: "The most reliable and inevitable consequences of self-stimulatory behaviours are the perceptual or sensory stimuli that these

behaviours produce" (Lovaas et al., 1987). Indeed, catatonic behaviours may be fruitfully conceptualized as a form of intuitive, self-administered, "sensory-reinforcement" treatment method.

The idea that sensory demodulation plays a key role in schizophrenia is further given credence by the fact that people who suffer from the condition are much likelier to experience psychotic symptoms during periods of quietude or in the absence of social or sensory distractions (Kraepelin, 1915: 11; and Bleuler, 1966: 100 &107). Moreover, the same conditions that increase the likelihood of psychotic episodes (i.e., the absence of engaging external stimulus) are also contributing factors in the aetiology of mind-wandering activity and sleep (Gerrans, 2014b). These similarities suggest that people who experience psychotic symptoms develop neural-connective pathways that are more susceptible to asynchronous functional demodulation.

ORT also successfully explains the difference between neutral and negatively impactful hallucinations. To briefly reiterate, neutral hallucinations do not impact a person's productivity or quality of life and are generally not classified as "pathological" (see Section III). Conversely, impactful hallucinations are classified as "pathological" because of their ability destabilize a person's waking activities (*Ibid*). Indeed, ORT explains why hallucinations that are experienced within the context of schizophrenia are often misidentified as real sensory input, whereas hallucinations that occur with the context of such non-pathological disorders as Charles Bennett Syndrome or the Phantom Limb syndrome are immediately identified as non-real, i.e., the presence or absence of higher-order cognitive modulation will affect how objectless perception is processed at the cognitive level.

Future Studies to Test ORT

The phenomenological relationship between different kinds of endogenous hallucinations and psychotic states raises the intriguing possibility that non-endogenous perceptual alterations (such as those that are generated through the administration of psychedelic compounds) are likely to also be produced through the oneiric release process. A version of this idea was first proposed by Jace C. Callaway (1988), who argued that substituted-tryptamine-based psychedelic experiences can be seen as a form of exogenously induced dreaming, whereas normal oneiric activity can be seen as an endogenous psychoactive state. Unfortunately, Callaway's model was never fully developed, and remains primarily a suggestion.

The crux of the premise, however, may be used to map-out the neurophysiology of sensory and higher-order-cognitive demodulatory activity. This is important because while ORT offers a phenomenological model for hallucinatory perception and psychotic symptoms, it has little to say

about the underlying neurochemistry that is involved in the said process. This limitation is primarily due to the fact that neither the neurochemistry of oneiric activity, psychotic symptoms, nor non-pathological forms of hallucinations have been hitherto satisfactorily mapped out. The exact neurochemistry involved in the demodulation of perceptual and higher-order-cognitive pathways still remains to be established. Thus, Callway's formulation of dreaming as an endogenous psychedelic state offers an elegant way forward. In particular, if psychoactive experiences can be placed within the architectural framework of ORT, then it should be possible to "reverse-engineer" the neurochemistry of oneiric release through a phenomenological subtraction study (see p.93).

On a neuro-phenomenological level, the psychotic symptoms of schizophrenia, oneiric activity, and substituted-tryptamines-induced psychoactive experiences share a number of substantial features (Esquirol, 1838; Kraepelin, 1906; Koresko, et al., 1963; Hartmann, 1965; Vogel, 1974; Hartmann, 1975; Jacobs, 1979; Barker, 1980; Hartmann, 1982; Zarcone, 1985; Assad, 1986; Callaway, 1988; Mahowald and Schenck, 1992; Ciprian-Oliver et al., 1997; Mahowald, et al., 1998; Hobson, 1999; McBride, 2000; Vollenweider and Geyer, 2001; Hobson, 2003: 68-85; Hobson, 2004; Gottesmann, 2005; Gottesman, 2006; Gottesmann and Gottesmann, 2007; Gottesmann, 2010; Schredl; 2010; Nir and Tononi, 2010; Jacob and Presti, 2010; Potkonyak and Marshall, 2010; Shen et al., 2010; Christoff, et al., 2011; Oudiette, et al., 2012; Gerrans, 2014; Limosoni et al., 2011; Hobson, 2011; Gerrans, 2012 and 2013; D'Agostino, 2013, 2013b, and 2013c; Gerrans, 2014; Dresler et al., 2014; Szabo et al., 2014; Benson and Feinberg, 2017; Benson and ; Pace-Schott, and Picchioni, 2017; Stickgold, et al., 2017; and Sinclair, et al., 2017). Specifically, all three conditions are marked by five characteristics: 1. An inability to discern between internal and external cues; 2. the ability to induce vivid, realistic, and fully integrated hallucinatory environments including non-real objects, humanoid entities, animals, plants, and incongruous landscapes that are integrated within a partially sensory-modulated phenomenological field; 3. the ability to seamlessly integrate hallucinations within the larger domain of mental content such that objectless precepts do not appear "intrusive" when experienced; 4. by phenomenological content perceived as "unreal" through retrospective analysis; and (5) by the presence of similar ("sleep-paralysis-like") humanoid entities (Koresko, 1962; Hartmann, 1965; Vogel, 1974; Gillin and Wyatt, 1975, and 1976; Winters, 1975; Hartmann, 1975; Baker, 1980; Hartmann, 1982, Fischman, 1983; Assad, 1986; Strassman, 1996; Mahowald et al., 1998; Callaway, 1999; McBride, 2000; Winter et al., 2000; Vollenweider and Geyer, 2001; Hobson 2003: 69:85; Nichols, 2004; Hobson, 2004; Gottesman, 2005; Gottesman and Gottesman, 2007; Cardinaly et al., 2008; Reynolds, 2008; Wallach, 2009; Kahn and Hober, 2009; González-Maeso and Sealfon, 2009; Nir and Tononi, 2010; Emanuel et al, 2010; Shen et al., 2010; Limosani et al., 2011; Hobson,

2011; Bery et al., 2012; Hayward et al., 2012; D'Agostino et al., 2013b and 2013c; Gerrans, 2014; Hobson, 2015; and Riga, et al., 2016).

On a neurophysiological level, many endogenous psychoactive compounds including melatonin, N,N, dimethyltryptamine (DMT), 5-MeO-DMT, and bufotenin, are either directly involved or have been speculated to be involved in the aetiology of sleep, oneiric activity, and waking hallucinations (Winters, 1975; Jacobs and Trulson, 1979; Barker et al., 1980; Fischman, 1983; Strasman, 1996; Winter et al., 2000; Nichols, 2004; Reynolds, 2008; Kahn and Gover, 2009; Cardinali et al., 2008; Wallach, 2009; Riga et al., 2016; and Dean et al., 2019;). While such higher substituted tryptamines as bufotenine have never been detected in healthy human subjects due to their rapid catabolic effect, they *have* nevertheless been collected from the urine samples of people who suffer from schizophrenia and other types of psychoses (Emanuele, et al., 2010). Intriguingly, psychoactive experiences have also been shown to be reversible through the administration of antipsychotic medication —a finding that suggests similar neurophysiological processes are involved in the aetiology of both exogenous and endogenous abnormal states of perception (Winters, 1975; Gillin and Wyatt, 1976; Jacobs and Trulson, 1979; Barker et al., 1980; Fischman, 1983; Strassman, 1996; Strassman et al., 1996; Ciprian-Olliver and Cetkovich-Bakmas, 1997; allaway, 1999; CWinter et al., 2000; Nichols, 2004; Jacob and Presti, 2005; Reynolds, 2008; Cardonaly et al., 2008; Kahn and Gover, 2009; Wallach, 2009; González-Maeso and Sealfon, 2009; Emanuel et al., 2010; Shen et al., 2010; Barker et al., 2013; Riga et al., 2014; and Dean et al., 2019). To date, however, little to no research has rigorously investigated the neuro-phenomenological relationship between oneiric activity, the psychotic symptoms of schizophrenia, and substituted-tryptamines-induced psychoactive states. This dearth of evidence explains why the exact neurophysiological interrelationship between these three states still remains unclear. To remedy this gap, future research should seek to answer the following question: "why do dreams, the psychotic symptoms of schizophrenia, and substituted-tryptamines-induced psychoactive experiences share so many neuro-phenomenological characteristics, and to what extent do these characteristics overlap?".

If ORT is correct, studies should find that **1.** substituted-tryptamine-induced psychoactive activity shares a considerable amount of overlapping neurophysiological features with the psychotic symptoms of schizophrenia, oneiric activity, and waking hallucinations, and that **2.** these structural similarities exist due to an intrinsically shared underlying mechanism of action.

One possible method to test this hypothesis is a phenomenological subtraction study (PSS). Adopting a relatively new methodology that borrows multiple components from the common cognitive subtraction method used in neuroimaging studies, this experimental design compares two

or more different phenomenological states presumed to have similar neurophysiological origins (e.g., dreaming, psychosis, and substituted-tryptamines-induced psychoactive experiences (Grabowski et al., 2000). This approach would help identify the overlapping neurophysiological structures, patterns, and mechanisms involved in the aetiology of oneiric activity, the psychotic symptoms of schizophrenia, and substituted-tryptamine-induced psychoactive experiences. A PSS would provide the data to compare and contrast (in a detailed one-to-one collation) the neuro-phenomenological features of all three perceptual states, establish their relational ontological status to each other, and determine if they are indeed generated through similar neurophysiological mechanisms of action. The advantage of this study, over other approaches, lies in the fact that the neurochemistry of exogenously administered substituted-tryptamines is known. If the three states are indeed generated through the oneiric release process, it will then be possible to "reverse-engineer" the neurochemical pathways that are implicated in the production of the other two. Furthermore, these results would allow future empirical studies on schizophrenia to target possibly heretofore undocumented neurophysiological structures and develop new treatment methods.

If the first research project proves fruitful, ORT can be empirically tested through the administration of a low-tryptophan-diet (LTD). In particular, endogenous substituted tryptamines are built out of L-Tryptophan. Because of this, if the demodulation of sensory and cognitive processes is indeed produced through the over-production of such higher substituted-tryptamines as DMT, and bufotenin (as my preliminary research currently suggests), it should then be possible to negate this effect through the diet. I propose that the LTD should be administered within the larger dietary context of a carefully planned vegan diet that is supplemented with collagen. Collagen contains all essential amino acids except L-Tryptophan. In comparison to the Standard American Diet (SAD), the vegan regiment will drastically reduce essential amino acid intake without endangering the health of the participants. By supplementing with collagen, participants will able to restore their non-L-tryptophan essential amino acids back to their pre-study levels, and allow L-tryptophan to be isolated as the control amino acid.

The LTD study should be carried out in three parts. **1.** The administration of a high-performance liquid chromatography-mass spectrometry (HPLC-MS) assay to inpatients who suffer from the psychotic symptoms of schizophrenia (*N=40*) in order to replicate the previous discovery of substitute tryptamines in the urine samples of people who suffer from schizophrenia and to determine their exact typology, quantity, and inter-individual variation. **2.** If the presence of substituted tryptamines is established, an LTD should be administered over a seven-day period to a second group (*N=40*) of inpatients (now under around-the-clock-observation). Using the same

method of urine analysis, participants should be tested —initially, throughout, and at the end of the seven-day period— to determine the impact that an LTD has on endogenous substituted tryptamine production. In addition, during the same time intervals, the psychotic symptoms of schizophrenia should be assessed via semi-structured interview and the administration of the Psychotic Symptom Rating Scales (PSYRATS), and the Brief Psychiatric Rating Scale (BPRS), or similar scales. **3.** Ideally, the sleep cycle of the volunteers would also be monitored throughout the study via EEG scans. In addition, volunteers should be asked to keep a detailed dream journal that can be appraised at the end of the study to determine the impact of an LTD on dream frequency (DF) and theme diversity (DTD).

If an LTD reduces urine levels of substituted tryptamines, the severity of psychotic symptoms of schizophrenia, *and* the overall duration of total sleep (i.e., in an endogenously sensory demodulated state), then these findings would help pave the way towards new pharmaceutical treatment methods for psychotic symptoms, and also help establish a neurochemical empirical framework for ORT. Alternatively, should individuals respond differently to an LTD, this would indicate a neurochemical heterogeneity in people who suffer from psychosis and allow for the identification of distinguishable psychotic subtypes in schizophrenia.

Section V Summary

This section presented an original theory, oneiric release, to account for the functional hybridization process outlined in the previous section of this work. The model combines the collective insights of previous oneiric formulations of schizophrenia, Llinás' conceptualization of wakefulness as a sensory modulated dream state, Hughlings-Jackson's theory of psychosis, and elements from West's perceptual release theory (PRT) of hallucinations. These insights (and such recent findings in the field of sleep and dream research as the discovery that oneiric activity is not restricted to a particular sleep stage), are combined to propose a novel explanatory framework that is capable to account for a wide spectrum of perceptual alterations.

Oneiric Release Theory (ORT) posits that wakefulness is a sensory modulated imagioneiric state. Within this framework, perceptual shifts are produced through the functional demodulation of sensory and higher-order cognitive pathways. When a pathway is demodulated, its underlying oneiric activity becomes "de-anchored" from external reality. Such recognizable perceptual shifts as wakefulness —> mind-wandering —> hypnagogia — > sleep occur when multiple sensory and cognitive systems are demodulated in a congruous manner.

When demodulation occurs incongruously and affects only a restricted number of sensory and higher-cognitive pathways, a hybrid-state-of-awareness, or "abnormal perception" occurs. Hybrid-states-of-awareness (including psychotic symptoms) are expressions of patterned incongruous oneiric-modulatory degrees in sensory or higher-order-cognitive pathways. This explains why both the phenomenological content and structure of hybrid-states-of-awareness is preserved across a wide range of perceptual states. They are all produced through the hybridization of concordant oneiric substrates *and* from the same underlying "thematic bank".

ORT also differentiates between psychopathological forms of abnormal perception, such as the psychotic symptoms of schizophrenia and such non-psychopathological abnormal perceptual alterations in perception as the hallucinatory activity of Charles Bennett syndrome (CBS), sleep-paralysis, or lucid dreaming. Psychopathological perceptual states are produced when higher-order cognitive pathways are incongruously demodulated during periods of partial sensory and motor activity. Conversely, when sensory demodulation occurs in the absence of higher-order cognitive disinhibition, a lucid hallucination is formed.

The last section of this work ends by proposing two future studies to establish and empirically test the neurochemistry of the oneiric release process.

Conclusion

This thesis addresses a long-standing problem in the field of psychiatry, namely, why do the phenomenological, cognitive, and psychological features of oneiric activity resemble those of schizophrenia? To address this question, the work begins by presenting an overview of previous "Oneiric Formulations of Schizophrenia".

The second section of this work, "Rationale for Revisiting Oneiric Formulation of Schizophrenia", identifies two main reasons why previous oneiric formulations of schizophrenia have failed to generate any meaningful empirical results. The first reason being that oneiric activity is not the exclusive product of any particular sleep stage. In fact, as demonstrated by data from a large body of studies, collective findings suggest that oneiric activity is not only not the product of a particular sleep stage, but can even regularly extend into wakefulness during periods of mind-wandering activity. The second reason why oneiric formulations of schizophrenia have failed to produce any meaningful empirical results is because schizophrenia is not a unitary disorder —it is a cluster of loosely related syndromes. The section ends by recommending that future oneiric formulation of schizophrenia look beyond the neurophysiology of specific sleep stages and target specific psychotic symptoms instead of the entire symptomatological spectrum of schizophrenia.

The third section of this work provides a detailed comparison of the psychotic symptoms of schizophrenia and oneiric activity. This section reaffirms the fact that the two conditions share a remarkable number of cognitive, psychological, and perceptual features, and suggests that both phenomena are products of the same underlying architecture. The last two sections of this work develop a new explanatory framework to account for the remarkable phenomenological similarities between psychotic symptoms and oneiric activity, i.e., the concept of "functional hybridization" and the mechanism that facilitates it, namely "oneiric release".

In the fourth section of this thesis, "Hybrid-States-of-Awareness", I identify three hybrid-states-of-awareness (lucid dreaming, sleep-paralysis, and somnambulism) and argue that all three of these perceptual states are produced through the imbrication of the elemental features of wakefulness, sleep, and oneiric activity. This section further argues that the psychotic symptoms of schizophrenia (i.e., waking hallucinations, psychosis, and delusion thinking) should also be conceptualized as "hybrid-states-of-awareness".

Oneiric Release Theory (ORT), developed in the fifth section of this work, posits that natural perceptual shifts, such as wakefulness —> mind-wandering —> hypnagogia —> sleep, are products of congruous sensory and higher-order cognitive demodulatory activity. The model is

predicated on Llinás' formulation of wakefulness as a sensory modulated dream state and understands all perceptual states as fundamentally "oneiric" in nature (including embodied perception). The model also borrows components from West's perceptual release theory (PRT) of hallucinations and Hughlings-Jackson theory of psychosis to assert that incongruous demodulatory activity is the root mechanism through which "abnormal perception" is generated during wakefulness.

ORT further posits that perceptual alterations are produced through the "de-anchoring" of sensory and higher-order cognitive systems. When this process occurs in an incongruous manner, a "break-in-reality" occurs because unmodulated pathways continue to generate oneiric activity even in the absence of external structuring input. Moreover, the section outlines how imbrication is limited to the combinatory demodulatory potential of each sensory and higher-order cognitive pathway. This explains why hybrid-states-of-awareness share a remarkable number of cross-features, i.e., they are produced by the same underlying pathways and emerge out of the same endogenous "oneiric themes".

Hybrid-states-of-awareness can be formed at any segment in the "imagioneiric continuum" (a concept introduced in the second section of this work). For example, when higher-order cognitive activity is restored during sleep, a lucid dream is formed. Because all other sensory systems remain "demodulated", lucid dreamers experience a sensorially "de-anchored" environment in a "non-psychotic" mental state. Conversely, when the visual or auditory organ (or their underlying pathway) becomes impaired during wakefulness while all other sensory and higher-order cognitive pathways remain properly modulated, a "lucid hallucination" is formed.

Depending on the function, degree, and number of incongruously demodulated pathways, different forms of imbrication are produced. For example, the demodulation of meta-awareness *and* auditory sensory pathways during wakefulness will produce auditory hallucinations and secondary delusions. The larger the degree of demodulation and number of affected pathways, the more severe the experienced symptoms. Complete "breaks-in-reality", or psychoses, occur when multiple sensory pathways are severely demodulated alongside disinhibited higher-order cognitive systems.

The section ends by outlining suggestions for two future studies that can further develop the neuro-phenomenology of ORT and empirically assumptions.

SUPPLEMENTARY Section on Oneiric Release Theory (ORT):

Given the intricate nature of the model developed in this thesis, this brief supplementary section reiterates the core underlying principles of Oneiric Release Theory (ORT) for the reader. The overwhelming majority of the information presented in this supplementary section is derived directly from Section V of this work. Page number references (e.g., [p.82]) used in this section are to help re-direct the reader to specific arguments and evidence from this thesis.

The underlying premise of ORT (i.e., that sensory and higher-order-cognitive demodulation are the main mechanisms through which abnormal perception is generated) is partially supported by recent neurophysiological findings regarding cortical and sensory functional demodulation in people who suffer from schizophrenia [p.86], data on sensory/cognitive reinforcement therapy that shows that cognitive and sensory reinforcement (i.e., enforced "modulation" therapies) have an antithetical effect on psychotic symptoms) [p.90], the data on Charles Bonnett Syndrome (CBS) [p.87], and the phantom limb phenomenon [p.88]. CBS and the phantom limb phenomenon may not be considered "psychotic" phenomena, or part of the schizophrenia spectrum. Yet according to ORT, non-lucid hallucinations, or "psychotic/delusional" hallucinations, are only produced when sensory demodulatory activity is invariably accompanied by higher-order-cognitive demodulation [P.86]. Because of this, CBS and the phantom limb phenomenon are used to demonstrate that sensory, bodily, or CNS demodulation that occurs in the presence of higher-order-cognitive modulation (i.e., during periods of perception in which waking cognition is not demodulated) will produce lucid (as opposed to psychotic) hallucinations.

ORT posits that both sensory *and* higher-order-cognitive systems must be simultaneously demodulated in order to produce "psychotic" states. The degree to which each one of these systems is demodulated accounts for the different perceptual states generated. For example, higher-order-cognitive demodulation without sensory demodulation will produce delusions, whereas higher-order-cognitive demodulation produced during periods of concomitant sensory demodulation will produce psychosis. In all of these instances the same consciousness (i.e., dream consciousness — which is presumed by the model to be the basis of wakefulness [p.83]) is "re-structured".

Oneiric Release Theory asserts that non-pathological or non-abnormal perceptual state shifts (e.g., wakefulness—>sleep / sleep —> wakefulness) occur when sensory and cognitive demodulation occurs in a concomitant manner. Conversely, when sensory/cognitive demodulatory activity is incongruous, i.e., in a single sense organ at the time, then the underlying epiphenomenal activity of the affected organ is said to become "demodulated" and no longer able to produce phenomenological data in congruity with external events. Because of this, when a person suffers

from an impairment in a specific sense organ (as in the case of CBS patients), unmodulated oneiric impressions (i.e., "hallucinations") will be triggered in the affected modality. In the case of uni-modal sensory demodulation, the unimpaired sense organs (or cognitive pathways) remain correctly modulated and continue to produce oneiric impressions that correspond to external events. This is why hallucinations appear to be embedded in reality.

The model explains why auditory impairments produce auditory hallucinations, visual impairments produce visual hallucinations, and why higher-order cognitive demodulation produces corresponding "cognitive lacunae". Demodulation "de-anchors" oneiric activity from external reality. The simultaneous demodulation of sensory and higher-order-cognitive systems is posited as a cause of psychosis. In the case of schizophrenia, auditory hallucinations are more prevalent than other forms of hallucinations because people who suffer from schizophrenia are more likely to develop "auditory impairments" than impairments in other sense organs (see below). When people who suffer from schizophrenia develop impairments in other sense organs, non-auditory sensory hallucinations are also produced. What makes "schizophrenia" identifiable (as a specific cluster of symptoms) within the context of ORT is that people who suffer from schizophrenia are likely to experience concomitant higher-order-cognitive demodulation alongside partial sensory demodulation.

ORT posits that delusional thinking is produced when a person experiences lapses (partial demodulation) in higher-order-cognition, while the remaining sense organs remain properly modulated and continue to generate "reality-corresponding" input. When this occurs, a person's cognitive architecture reverts back to its antecedent unmodulated oneiric state. This is also why dream cognition exhibits the same features as delusional thinking. Both types of cognition are generated by the same underlying unmodulated systems. This process can be seen in all hybrid-states-of-awareness. For example, during lucid dreaming such components of higher-order-cognition as meta-awareness become modulated (creating waking forms of cognition within a larger dreamscape), while the remaining sense organs remain unmodulated, and continue to generate "unmodulated oneiric sense impressions". In this sense, ORT considers lucid dreaming as a form of "inverted-psychosis", i.e., a state where higher-order-cognition is restored in a sensory isolated context. Both delusional thinking and psychosis, in this model, are a product of an inverted demodulatory potential. Furthermore, ORT posits that combinatorial modulatory degrees exist in each sense organ and higher-cognitive pathway. Indeed, this is the strength of the theory: it can simultaneously account for the psychotic symptoms of schizophrenia as well as such non-pathological forms of perceptual alterations as sleep paralysis, somnambulism, and lucid dreaming.

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