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Schizophrenia and the language of dreams

from Kraepelin's schizophasia to contemporary research

Armando D'Agostino

Associate Professor in Psychiatry

DISCOURSE IN PSYCHOSIS

2024 Consortium Satellite Meeting, Pavia

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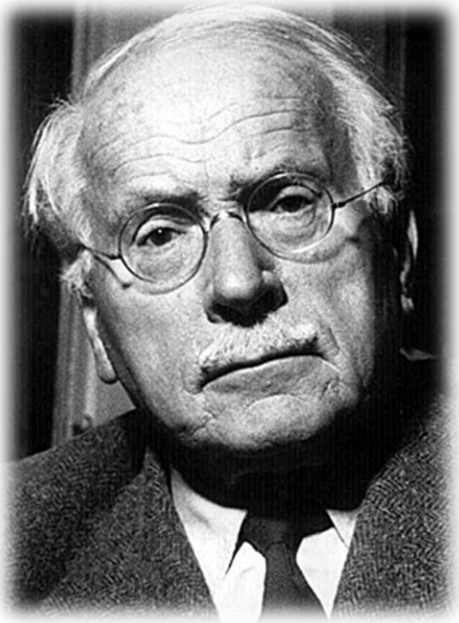
"Find out about dreams and you will find out about insanity"

John Hughlings Jackson, 1894

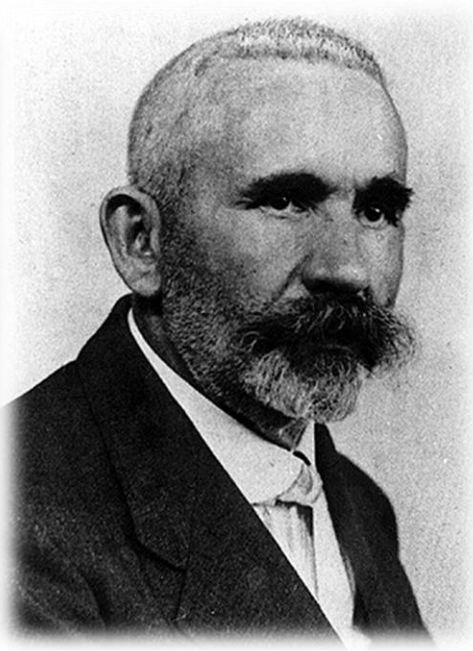
"If we could imagine a dreamer walking around
and acting his own dream as if he were awake,
we would see the clinical picture of dementia praecox"

Carl Gustav Jung, 1907



"Dream speech in every detail corresponds to schizophrenic speech disorder."

Emil Kraepelin, 1920



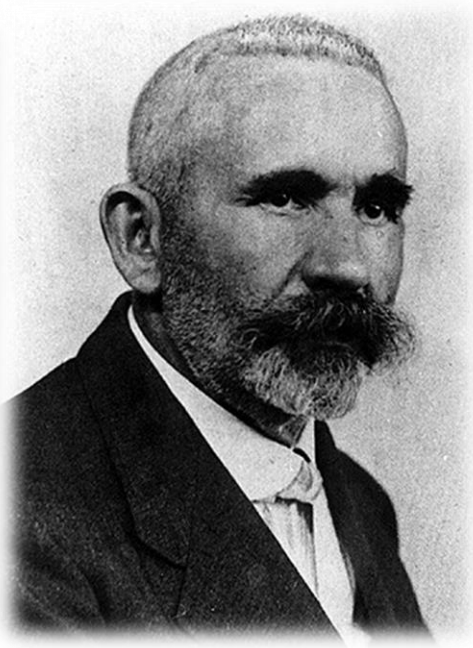
Traumsprache ("Dream speech")

1) disorders of word-selection ("paraphasias")

2) disorders of discourse (e.g. agrammatisms)

3) thought disorders.

The most frequent form
of dream speech is a **neologism**.



Über Sprachstörungen im Traume
("On Language Disturbances in Dreams"), **1906**

Kraepelin's schizophasia: Chaotic speech with preservation of comprehension and activities of daily living

Ricardo de Oliveira-Souza [a,b,*]

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Differential Diagnosis of Kraepelin's Schizophasia

Author(s)	Speech and Language	ADL	Anatomical Correlates
Kraepelin's schizophasia (Kraepelin, 1919)	Fluent paraphasic neologistic speech with severe impairment of communicative content; preserved ability to understand spoken and written language and repeat short and long sentences. Lack of insight.	Spared	Cortico-subcortical temporoparietal loops integrated by the anterior division of the arcuate fasciculus.
Schizophasia *sensu lato* (Anand & Wales, 1994)	A collection of ill-defined impairments of speech and language that are difficult to differentiate from formal thought disorder in some patients with a diagnosis of schizophrenia.	Impaired	Depends on the clustering of symptoms in each case.
Wernicke's aphasia (Binder, 2015)	Fluent paraphasic speech with impaired ability to understand and repeat spoken language. Often associated with alexia and agraphia.	Impaired	Corticosubcortical region corresponding to the territory of the inferior branch of the L middle cerebral artery, which includes the posterior segments of the superior and middle temporal gyri and insula, temporoparietal junction, and angular gyrus.
Transcortical sensory aphasia (Kertesz *et al.*, 1992)	Fluent paraphasic speech with impaired understanding and spared repetition.	Impaired	Damage to L posteromedial temporo-occipital junction and underlying inferior occipitofrontal and inferior longitudinal fascicles
Nonsense speech (amphigory) (Fisher, 1970)	Nonsense talkativeness with relative preservation of comprehension, naming, writing, and follow commands. Severe amnesia. Total lack of insight. Uncertain nosological status.	Not reported	Post-mortem exam: astrocytoma involving the optic nerves, hypothalamus, amygdala, fornix, corpus callosum, and mammillary bodies.
Incoherence with extravagant paraphasias (Guard *et al.*, 1983)	Dense amnesia and fluent paraphasic speech punctuated by extravagant words ("bombastic style") which are employed out of semantic context; preservation of comprehension, repetition and reading. Extravagant speech remitted in parallel with amnesia 6 weeks after evacuation hematoma.	Probably impaired	R intraparenchymal hematoma that destroyed the dorsolateral prefrontal lobe, the anterior cingulate gyrus, and the genu of the corpus callosum.
"A disorder of linguistic expression" (Mehler, 1987)	A 56-year-old woman developed semantic inconsistencies, intrusions and perseverations in speech and writing, but retained "excellent" praxis, repetition, oral comprehension and insight.	Not reported	Infarction of head of L caudate nucleus.
Thalamic thought disorder (Chatterjee *et al.*, 1997)	A woman with bizarre incoherent speech and dense amnesia.	Impaired	MRI: bilateral paramedian thalamic strokes involving the mammillothalamic tract, the internal medullary lamina, and the dorsomedial and ventrolateral nuclei bilaterally. SPECT: bilateral hypoperfusion of thalamus and basal ganglia, L temporal and posterior parietal, and R prefrontal cortices.
Schizophasia due to left thalamic lacune (Black *et al.*, 1997)	A 47-year-old woman with a diagnosis of schizophrenia suddenly developed incoherent speech with preservation of comprehension. Speech contained numerous associative alliterations, phonological perseverations, and illogical associations with delusional themes.	Not reported	CT and MRI: L anterosuperior thalamic infarct. SPECT: L frontal and R cerebellar diaschisis.

ADL: activities of daily living; L: left; MRI: magnetic resonance imaging; R: right; SPECT: single photon emission computerized tomography.

"**The dreaming man sleeps** and it is the sleeping condition which allows the desires to reveal themselves as bizarre and incomprehensible dreams, by temporarily stopping the connections with the real world

[…]

A patient suffering from schizophrenia doesn't sleep

but sometimes his inner life shows the same characteristics."

Eugène Minkowski, 1927

Schizophrenia and Physiological Indices of Dreaming

ALLAN RECHTSCHAFFEN, PhD
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ANN ARBOR, MICH

Commenting on the frequently noted similarity of thinking mechanisms in dreams and schizophrenia, Oswald [9] emphasized an obvious difference between the two states: ". . . psychiatric patients have typical schizophrenic experiences . . . while fully awake." While this difference precludes a complete identity of psychotic state and normal dreaming, it leaves open the possibility that although the schizophrenic state is manifest during wakefulness, it bears a phenomonological and physiological resemblance to dreaming in normals. In the present study, three physiological variables, electroencephalograph (EEG), eye movements, and electromyography (EMG), were examined during periods of wakefulness in five acute schizophrenics to determine whether there was any obvious similarity between the patterning of these variables in awake schizophrenics and dreaming normals. Acute schizophrenics were chosen as subjects because it seemed likely that their mentation would resemble dreaming more than other groups.

EEG, EMG, and eye movements were studied because research of the past ten years has shown that dreaming is most likely to occur during stages of sleep characterized by a distinctive patterning of these variables. Physiological characteristics of rapid eye movement (REM) periods, the stage of sleep from which subjects most frequently report dreams upon awakening, are: (1) stage 1 EEG.[1,4,5] This is a low voltage, irregular, relatively fast pattern containing slow components in the 4-6 cps range. (2) "Saw-toothed" waves of 3-4 cps in EEG tracings from frontal regions, usually just preceding bursts of rapid eye movements.[2,7,11] The saw-toothed waves are not an invariable con-

Submitted for publication Aug 17, 1963.

University of Chicago (Dr. Rechtschaffen); Kommunehospitalet (Dr. Schulsinger); University of Michigan (Dr. Mednick).

This research was supported by grants M-4151, MH-K3-18, 428, and MH-06867 from the National Institute of Mental Health, US Public Health Service. The study was conducted at the Psykologisk Institut, Kommunehospitalet, Copenhagen.

Schizophrenia Bulletin vol. 37 no. 1 pp. 1–3, 2011
doi:10.1093/schbul/sbq115
Advance Access publication on October 7, 2010

EDITORIAL

Corollary Discharge, Hallucinations, and Dreaming

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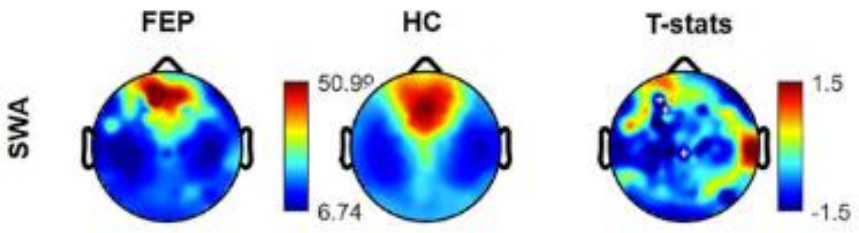
«the same process by which psychotic patients progress from salience attributed to irrelevant stimuli to a new, highly relevant meaning, appears to occur in dreams where a single hallucinatory image can convey an immediate emotionally compelling meaning that is not related to the image in any obvious way»

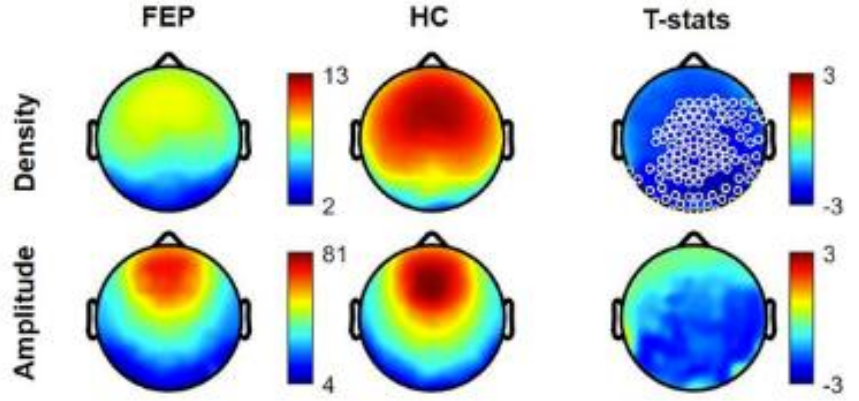
Proof–of–concept evidence for high–density EEG investigation of sleep slow wave traveling in First-Episode Psychosis

Anna Castelnovo[1,2,3], Cecilia Casetta[4,5], Simone Cavallotti[4], Matteo Marcatili[6], Lorenzo Del Fabro[7,8], Maria Paola Canevini[4,9], Simone Sarasso[10] & Armando D'Agostino[4,9]

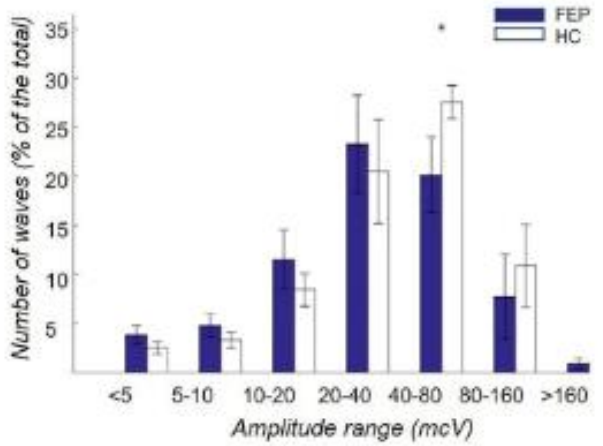
A. Slow wave Activity Scalp Power Topography



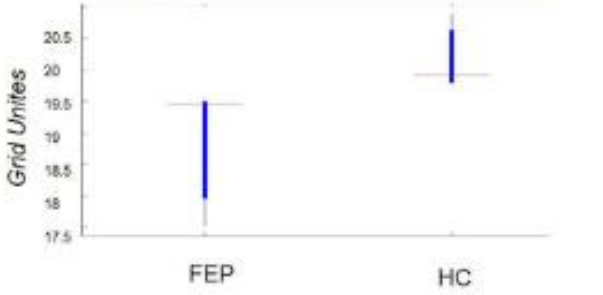
B. Slow wave Density and Amplitude Topography



C. Average Slow Wave Amplitude



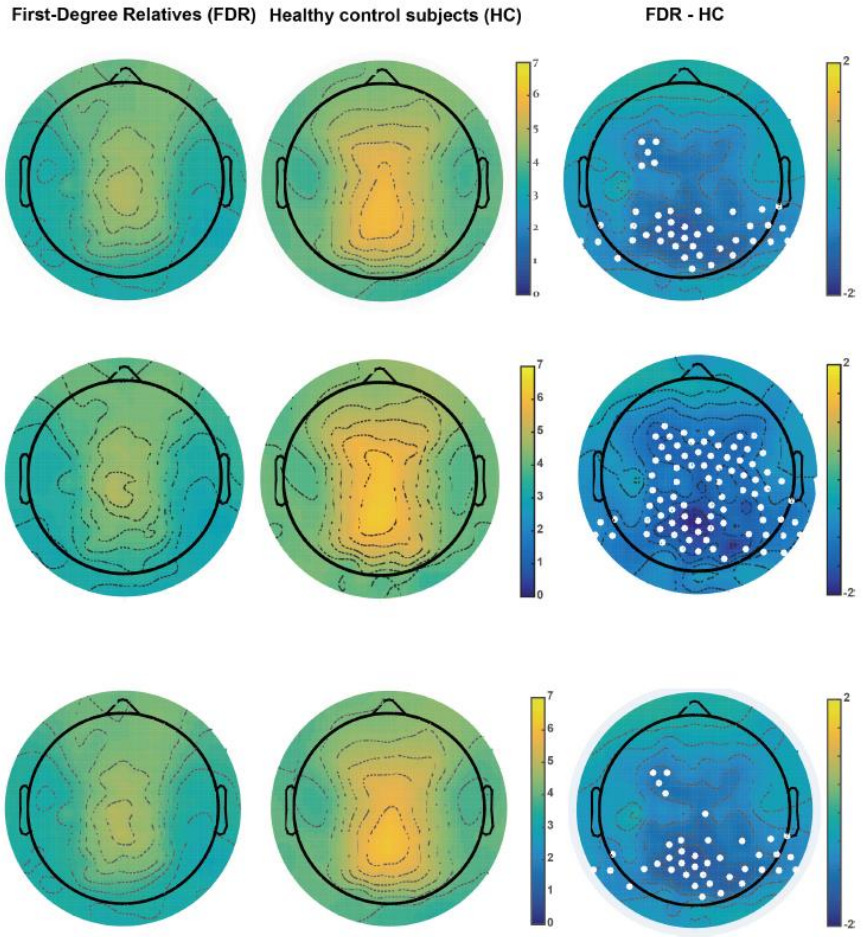
D. Slow Wave Traveled Distance

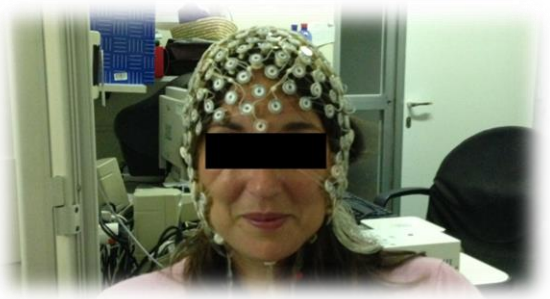


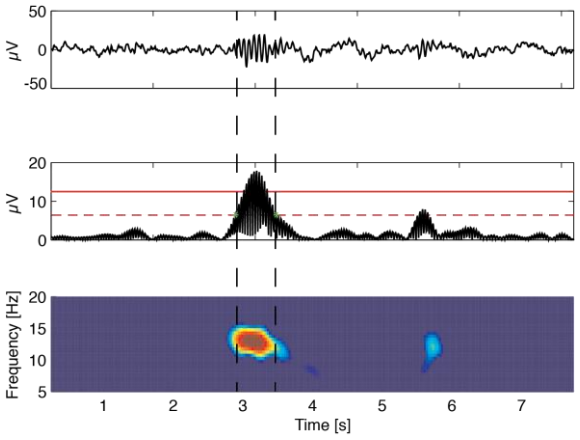
scientific reports

Sleep endophenotypes of schizophrenia: slow waves and sleep spindles in unaffected first-degree relatives

Armando D'Agostino[1,2], Anna Castelnovo[1], Simone Cavallotti[2], Cecilia Casetta[1], Matteo Marcatili[1], Orsola Gambini[1,2], Mariapaola Canevini[1,2], Giulio Tononi[3], Brady Riedner[3], Fabio Ferrarelli[4] and Simone Sarasso[5]





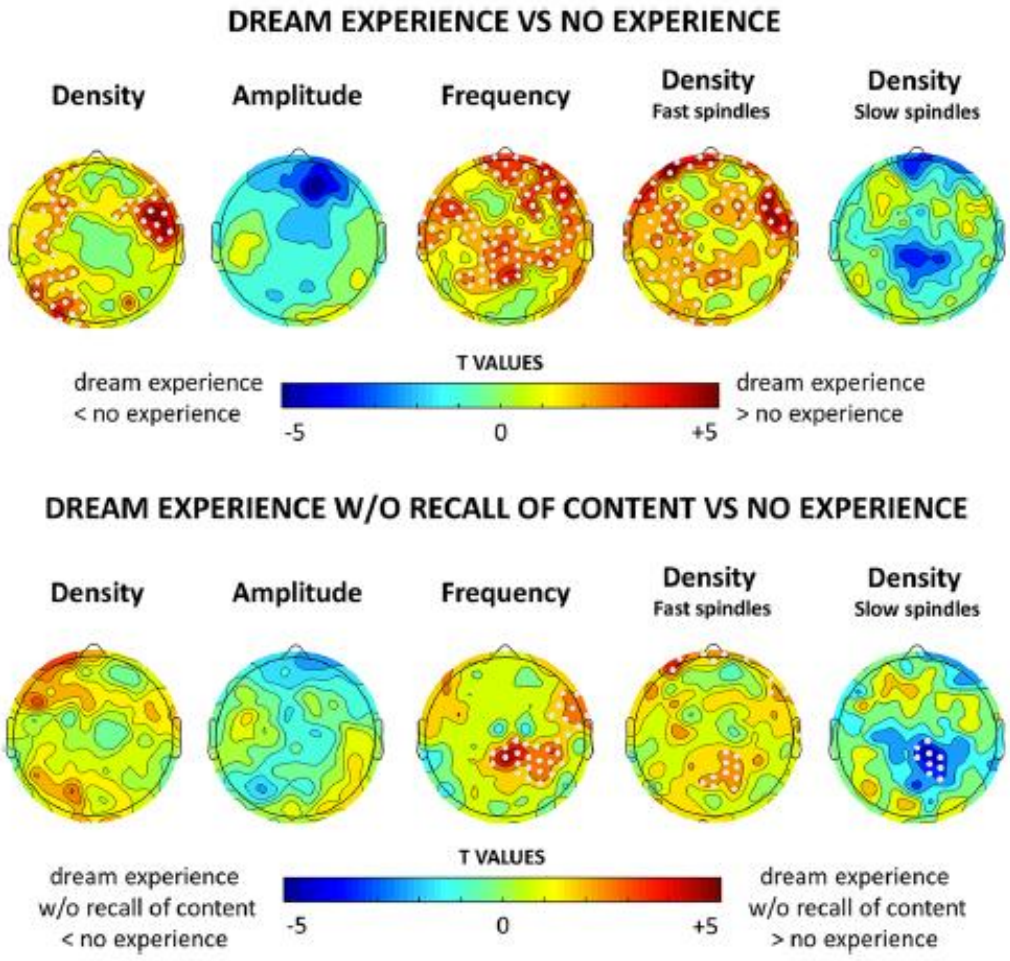


npj nature partner journals

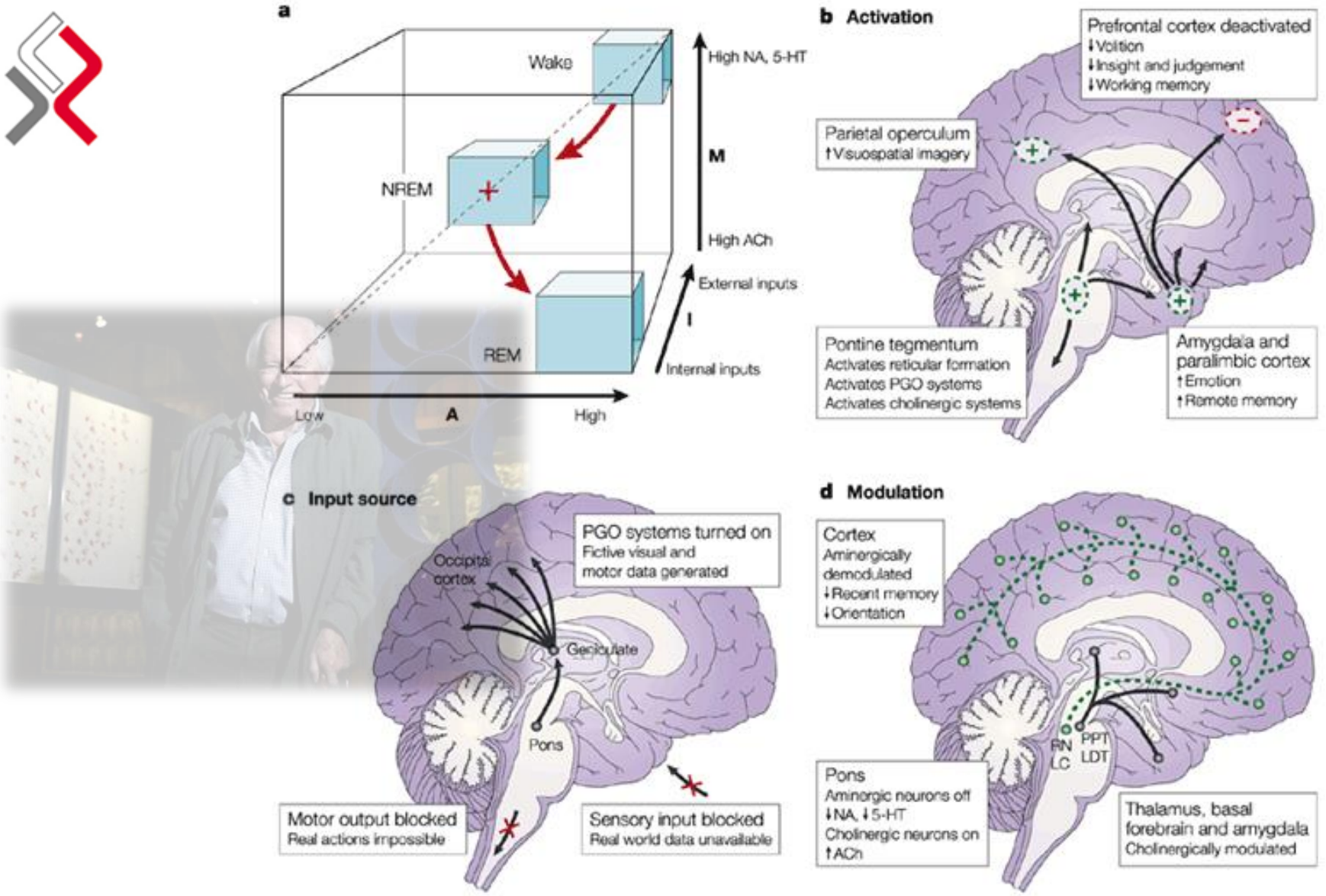
Dreaming in NREM Sleep: A High-Density EEG Study of Slow Waves and Spindles

JNeurosci
THE JOURNAL OF NEUROSCIENCE
An Official Journal of SOCIETY for NEUROSCIENCE

Francesca Siclari,[1] Giulio Bernardi,[1,3] Jacinthe Cataldi,[1] and Giulio Tononi[2]



- **dreaming** is more likely to occur in the presence of **fast spindles** in a central and posterior cortical region
- reports of **no experience** preferentially occur in the presence of **slow spindles** in the same areas
- these findings are consistent with the observation that subjects who report a high number of dreams have faster spindles (Nielsen et al., 2016)



Nature Reviews | Neuroscience

A model for madness?

Dream consciousness: our understanding of the neurobiology of sleep offers insight into abnormalities in the waking brain.

Allan Hobson

Sigmund Freud and Carl Jung noticed the kinship of normal dreaming with psychoses of severe mental illness, such as schizophrenic and affective disorders. The presence in both of hallucinations and delusions, cognitive abnormalities and emotional intensifications had also been emphasized by earlier, more physiologically oriented psychiatrists, but the psychoanalysts soon eschewed neurobiology and attempted to explain both dreams and psychosis psychodynamically, without reference to the brain. A century of progress in neurobiology and in sleep and dream research now encourages a new look at this old question.

Recent advances in brain imaging, coupled with cellular and molecular neurobiology data, have given us a remarkably clear picture of the differences in brain activity between waking and activated states of sleep, such as rapid eye movement (REM), when intense dreaming most commonly occurs. During waking, consciousness is dominated by externally driven percepts, whereas internally generated images are rare. During REM sleep, the exact opposite rule applies — externally driven percepts are rare, whereas consciousness is dominated by internally generated images. Conversely, descriptions of thinking, which are common in reports of waking consciousness, are rare in reports of dreaming. When the mind is dominated by internal percepts, as it is in dreaming and psychotic states, the capacity to generate thoughts is greatly weakened. Rationality can thus, understandably, fall victim to a change in brain state.

To summarize the extensive data it is useful to look at the results of recent positron emission tomography (PET) scans and brain injury data from humans, and to compare them with earlier results of laboratory experiments in other mammals. The emphasis is on the distinct physiology of REM sleep, but it should be recognized that dreams associated with non-REM sleep are also created by a brain whose physiology has shifted strongly from that of waking towards that of REM.

One of the important causes of dream hallucinations is the general and widespread activation of the brain that occurs in conjunction with a blockade of external sensory inputs and motor outputs in REM sleep. In REM, selective activation of the cortical region responsible for visuospatial integration is seen.

During REM sleep, there is also selective activation of the amygdala and other parts of the limbic system. This is relevant for our

LAURENT GIRAUDON/BRIDGEMAN ART LIBRARY

understanding of the heightened emotion — especially the feelings of anxiety, anger and elation — that so commonly dictate the development of a dream plot. An exaggeration of emotional activation is also common in mental illness, when it may contribute to impairment of rationality. An idea that helps to unite these findings is that emotion is itself an internally generated percept that powerfully influences conscious experience.

Human PET studies reveal a selective deactivation of the dorsolateral prefrontal cortex in REM state compared with waking. This brain region, which is assumed to be essential to working memory, directed attention and volition, remains inactive during REM. Subjects tell us that in this state they cannot use working memory effectively, are unable to sustain attention and do not will their dream actions. Selective frontal lobe deactivation has also been reported in PET studies of schizophrenia, and may account for the difficulties that these patients have in organizing their thoughts, integrating them with emotion, and translating them into appropriate actions.

Three aminergic neuronal systems — the noradrenergic, the serotonergic and the histaminergic — all shut down in REM. At the same time, the cholinergic system of the pontine tegmentum is activated. Thus, the entire brain is activated but aminergically demodulated except for dopamine (whose release continues at waking levels across all states of sleep). An abnormal sensitivity to dopamine is thought to mediate psychosis and its unmodulated action in REM may contribute to the madness of dreams. As norepinephrine is important for attention and serotonin is critical for learning and memory, it is likely that the decreases in thinking during dreams are due in part to these remarkable and profound changes in brain chemistry.

As well as showing a decline in the capacity to think, formal state studies of dreaming also reveal a decrease in the capacity for orientation (times, places and persons change without notice) and a corresponding increase in the capacity to confabulate (giving the cognitive defects a coherence which is emotionally salient however illogical). Dream bizarreness and the confabulatory quality, coupled with the memory defects and the increase in visual hallucinations, suggests that dreaming may be a functional delirium, much like the psychosis associated with alcohol and drug abuse. Indeed, the delirium-like dreams of normal sleep are produced by changes in the same brain neuromodulatory systems that are affected by drugs en route to clinical delirium.

Now that we have this preliminary, but precise, model for how consciousness is altered by normal and abnormal changes in the state of the brain, we can conduct more hypothesis-driven studies of the brain–mind connection. For example, we can study regional-brain activation in the same animals from which we have learned so much at the cellular and molecular level. In those animals, we can also use drugs to alter a brain state experimentally and predict changes in regional activation pattern. In humans, we can use behavioral techniques as well as drugs to experimentally alter the conscious state of our human subjects; and we can measure predicted changes in regional brain activation with the real-time technique of functional magnetic resonance imaging.

To say that the mind–body problem is solved would be provocative and invite derision, but we have made important progress, and future directions of this approach are now clearly indicated. The mind–body problem can now be confronted by taking advantage of the natural changes in brain–mind state that contrast waking and dreaming. ■

Allan Hobson is in the Department of Psychiatry, Harvard Medical School, Boston, Massachusetts 02115, USA.

FURTHER READING

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Neural correlates of insight in dreaming and psychosis

Martin Dresler [a, b, *], Renate Wehrle [a], Victor I. Spoormaker [a], Axel Steiger [a], Florian Holsboer [a], Michael Czisch [a], J. Allan Hobson [c]

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[c] Harvard Medical School, Boston, USA

dreaming

- hallucinations
- delusions
- bizarre experiences
- disorganized thought
- emotional intensifications
- lack of insight into current state

insight into the dreaming state

associated brain regions:

- dorsolateral PFC [35-36]
- frontopolar cortex [35-36]
- superior frontal gyri [36]
- middle frontal gyri [36]
- right inferior frontal gyrus [36]
- bilateral precuneus [36]
- left middle temporal gyrus [36]
- inferior temporal gyri [36]
- right lingual gyrus [36]

model

model

psychosis

- hallucinations
- delusions
- bizarre experiences
- disorganized thought
- emotional intensifications
- lack of insight into current state

insight into the psychotic state

associated brain regions:

- dorsolateral PFC [52, 59-62, 64, 67-68]
- frontopolar cortex [54-55]
- superior frontal gyri [52, 60, 62, 64]
- middle frontal gyri [52, 62, 67-68]
- right inferior frontal gyrus [64-68]
- bilateral precuneus [52, 57, 65-66, 68]
- left middle temporal gyrus [65, 68]
- inferior temporal gyri [63-65, 67-68]
- right lingual gyrus [56, 68]

Dream Bizarreness as the Cognitive Correlate of Altered Neuronal Behavior in REM Sleep

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Laboratory of Neurophysiology
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TWO STAGE SCORING SYSTEM FOR DREAM BIZARRENESS

Stage I identifies items as bizarre if they are physically impossible or improbable (probability of occurrence < 0.05) aspects of:

A. the plot, characters, objects or action
B. the thoughts of the dreamer or dream character
C. the feeling state of the dreamer or dream character

This stage establishes the dream domain or report *locus* of each item of bizarreness.

Stage II then characterizes the item as exhibiting:

1. discontinuity (change of identity, time, place, or features thereof)
2. incongruity (mismatching features)
3. uncertainty (explicit vagueness)

This stage establishes the character of each item of bizarreness.

SCIENTIFIC REPORTS
nature research
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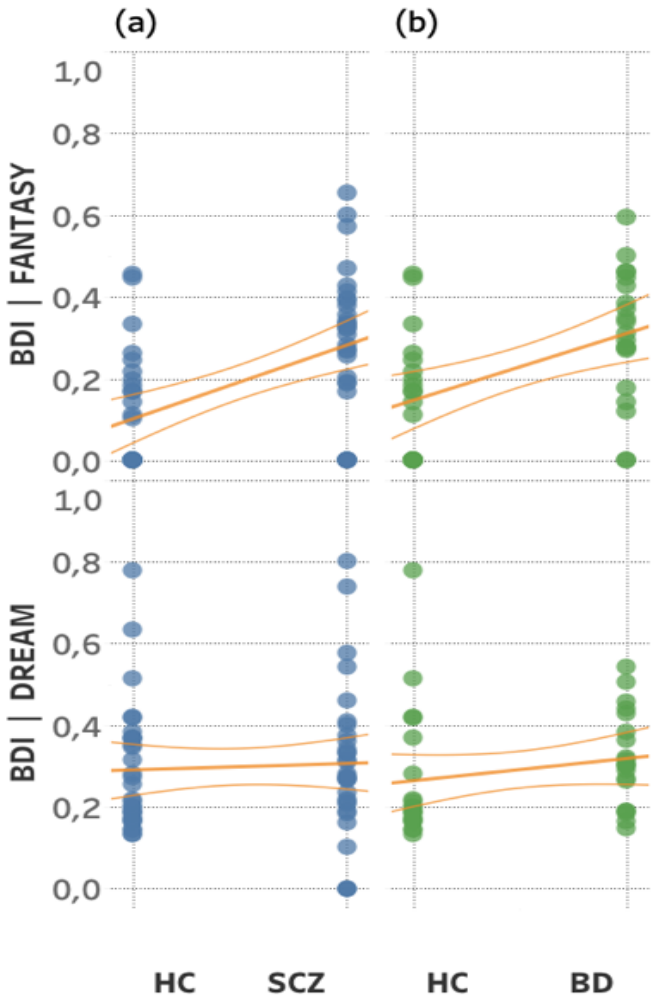
A cognitive profile of multi-sensory imagery, memory and dreaming in aphantasia

Alexei J. Dawes[1]✉, Rebecca Keogh[1], Thomas Andrillon[1,2] & Joel Pearson[1]

- compared to control participants with imagery ability, aphantasic individuals report decreased imagery in other sensory domains, although not all report a complete lack of multi-sensory imagery.

- they also report less vivid and phenomenologically rich autobiographical memories and imagined future scenarios, suggesting a constructive role for visual imagery in representing episodic events.

- interestingly, **aphantasic individuals report fewer and qualitatively impoverished dreams compared to controls**.

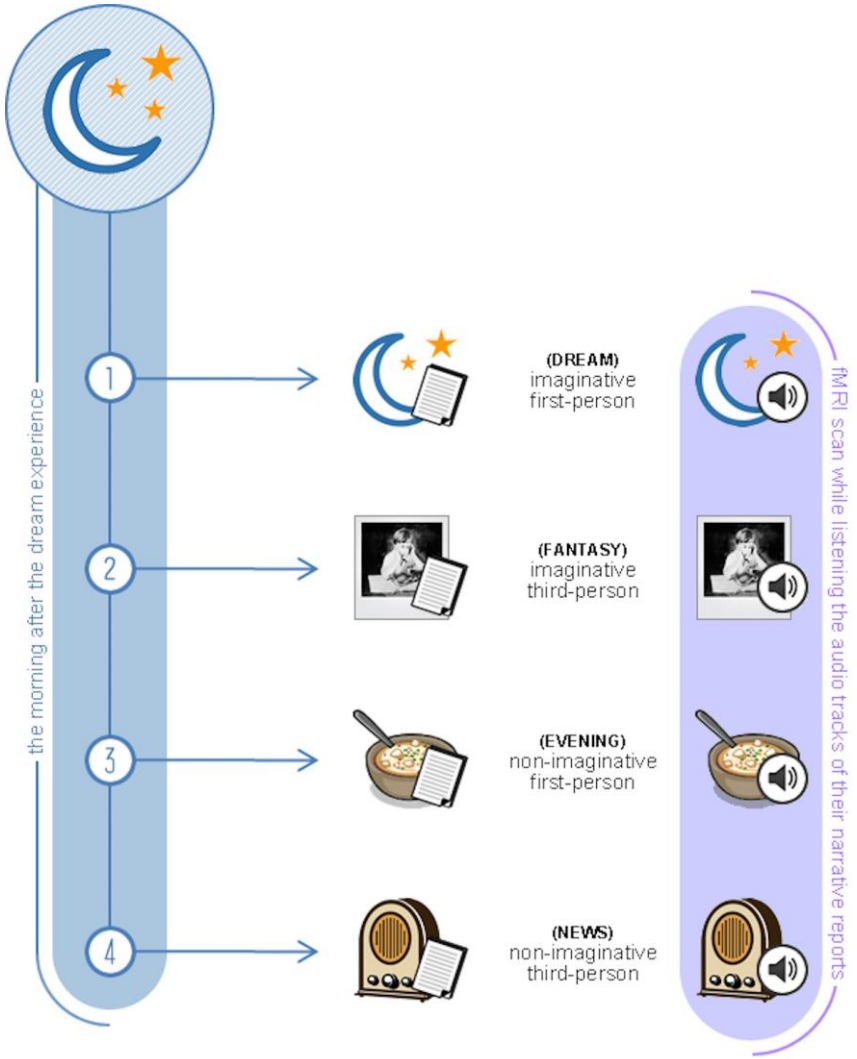


(a) Mean Bizarreness Density Index (BDI) values in the dream and fantasy reports of **acutely psychotic patients with Schizophrenia** (SCZ) and healthy control subjects (HC). **(b)** Mean BDI values in dreams and fantasy reports of **acutely psychotic manic patients with Bipolar Disorder** (BD) and healthy control subjects (HC).

Thematic Apperception Test (TAT) was used to elicit fantasy narratives. Bizarreness Density was calculated by dividing Bizarreness Intensity (BI) by the report word count. BI is scored as the number of bizarre events in the domains of plot, cognition and affect according to the Dream Bizarreness scale (Hobson et al., 1987).
Data retrieved from Scarone et al., 2008; Limosani et al., 2011.

is it possible to identify a model of brain activation that reflects differences in bizarreness across dreams and fantasies?

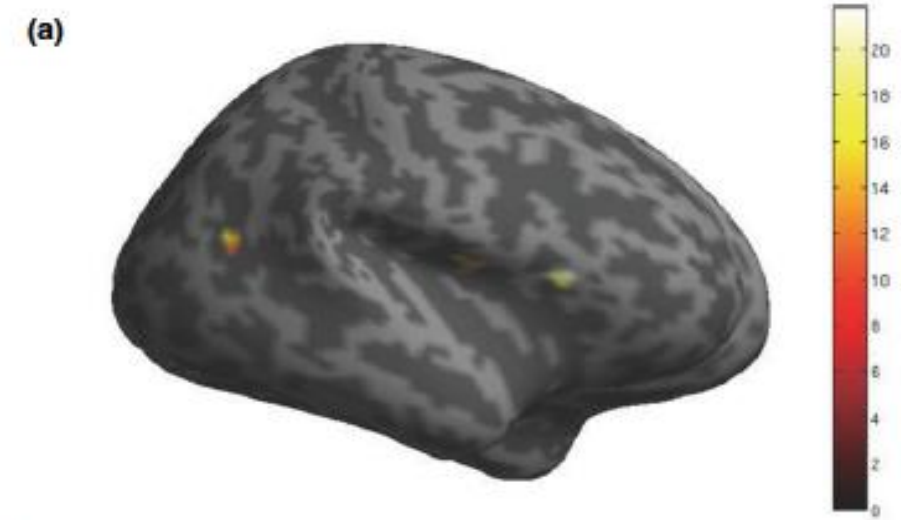
is it possible to identify a model of brain activation that reflects differences in bizarreness across dreams and fantasies?



Right hemisphere neural activations in the recall of waking fantasies and of dreams

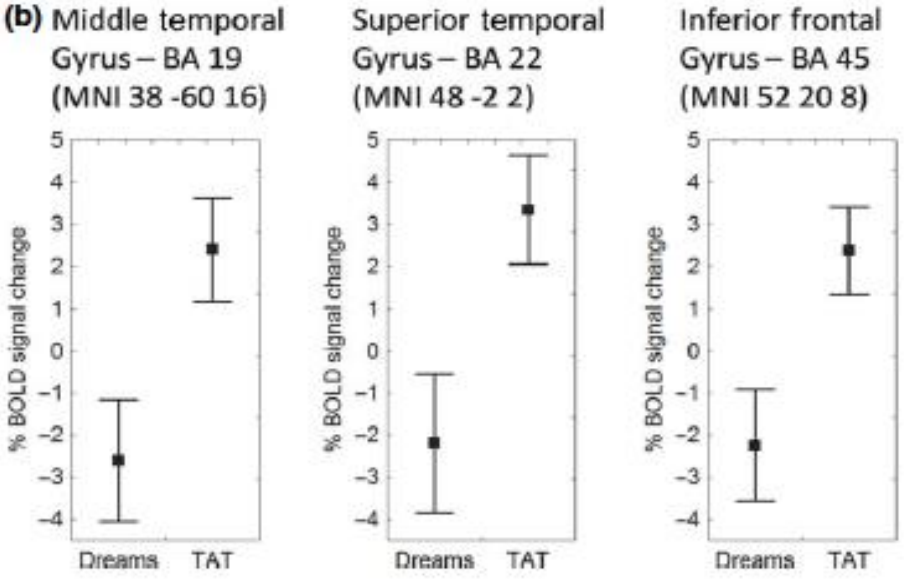
FRANCESCO BENEDETTI[1,2], SARA POLETTI[1,2], DANIELE RADAELLI[1,2], REBECCA RANIERI[3], VALERIA GENDUSO[1,2], SIMONE CAVALLOTTI[3], ANNA CASTELNOVO[3], ENRICO SMERALDI[1,2], SILVIO SCARONE[3] and ARMANDO D'AGOSTINO[3]

Journal of Sleep Research



Differentially activated grey matter areas during dream and fantasy (TAT) conditions, plotted on an inflated cortical surface of the human brain reconstructed from MRI scans **(a)**.

The BOLD signal change zero line marks the higher activation observed for both imaginative conditions (dreams and fantasies) compared to non-imaginative control conditions (narrative reports of daytime activities or news) **(b).**



implications?

- first experimental support to Kraepelin, who postulated that language networks and frontal structures associated with abstract reasoning must be shut down during dreaming

- Right IFG, STG, and MTG neuronal loss observed in schizophrenia could underlie illogicity in patients' wakefulness

Commentary on "Investigating the diagnostic utility of speech patterns in schizophrenia and their symptom associations": The current need for the harmonization of speech elicitation protocols in basic and applied science

Natália Bezerra Mota *

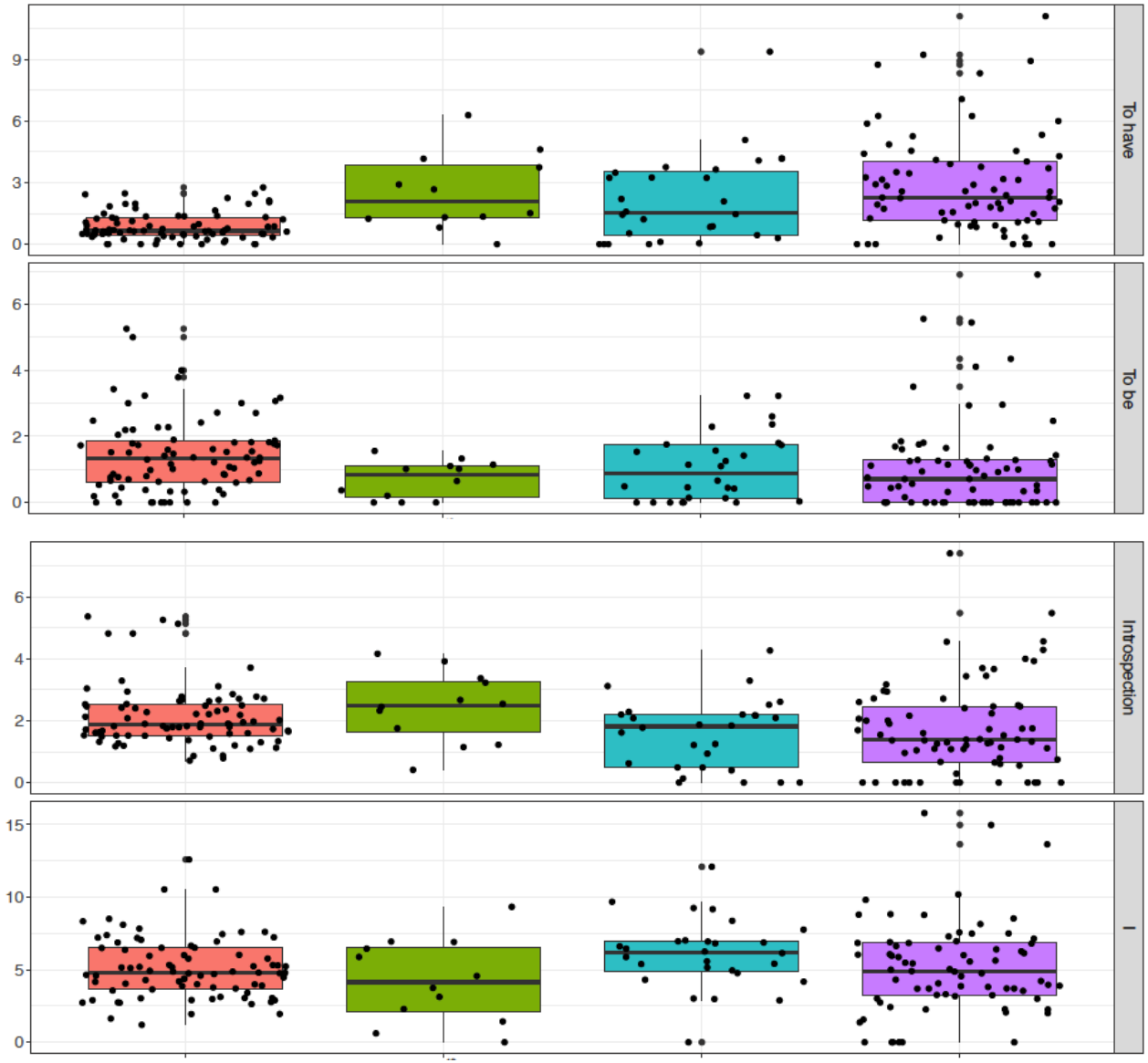
DI
SS

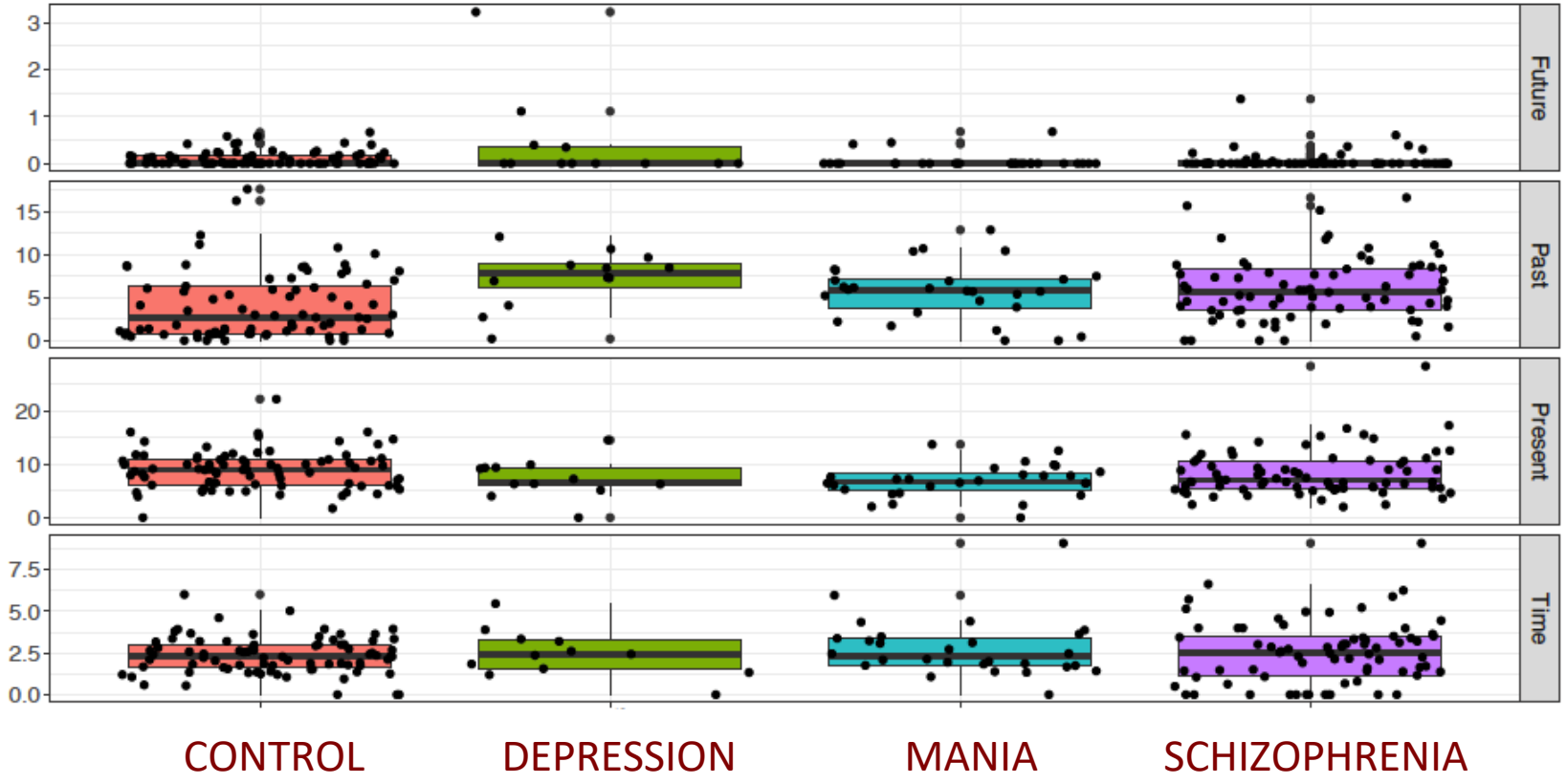
- Predictive value and negative symptom correlation of automated connectedness measures in dream reports but not previous day report (Mota et al., 2014).

- Decreasing rank of diagnostic value for schizophrenia from dream reports to story-telling based on emotional scenes and neutral scenes (Mota et al., 2017).

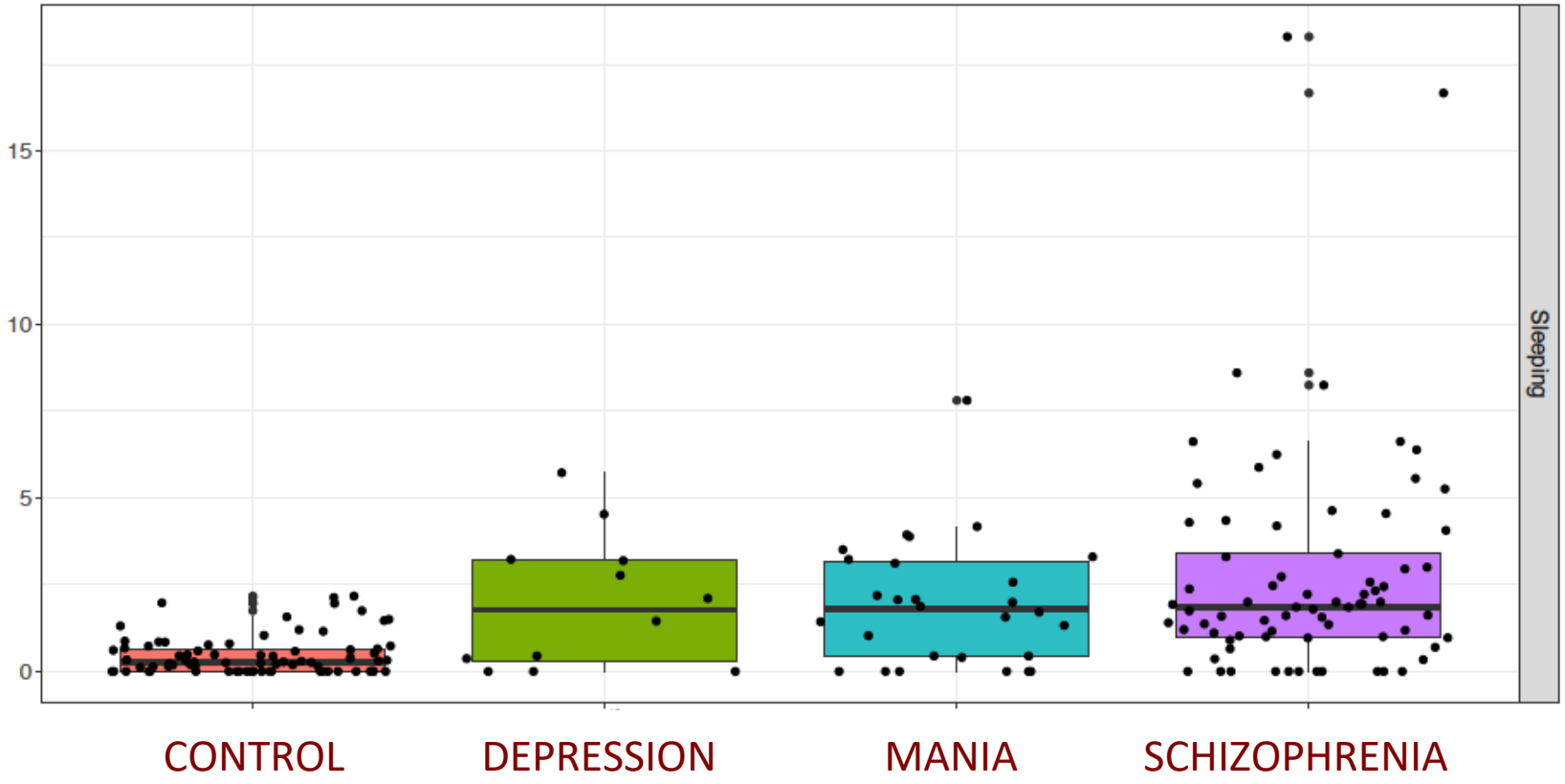
DISCOURSE
IN PSYCHOSIS

	PSYCHOSIS (115)	CONTROL (80)
Mean age (±SD)	44.9 ± 14.0	44.2 ± 17.4
Female	43,5%	58,8%
Mean dreams (n)	4,2	4,5
Diagnoses (n)		/
Schizophrenia	67	
Mania	28	
Depression	12	
Delusional Disorder	8	


To have
To be
Introspection
I
CONTROL
DEPRESSION
MANIA
SCHIZOPHRENIA


Future
Past
Present
Time
CONTROL
DEPRESSION
MANIA
SCHIZOPHRENIA

LIWC



LIWC

- Some **overlap** exists between the physiological experience of **dreaming** and the pathological experience of **psychosis**

- **Brain** regions that are relevant for **language processing** may **lower activity** during dreaming, as predicted by Kraepelin

- **Automated language analysis** may be a privileged way to explore similarities and differences which may enhance our understanding of psychosis

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In memoriam J. Allan Hobson, M.D. Professor of Psychiatry , Emeritus

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…hence we may say that we are working towards the explanation of the psychoses when we endeavour to elucidate the mystery of dreams