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Auswirkung pathologischer und gezielter Schlaffragmentierung auf das Traumverhalten

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Inhaltsverzeichnis

Abkürzungsverzeichnis

tACS	transcranial alternating current stimulation
ASRM	Fragebogen: Altman Self-Rating Mania Scale
BDI-V2	Fragebogen: Becker-Depression-Inventar Version 2
CSD	current source densities
tDCS	transcranial direct current stimulation
EEG	Elektroenzephalogramm
FLei	Fragebogen: Fragebogen zur geistigen Leistungsfähigkeit
fMRI	funktionelles Magnetresonanzimaging
Non-REM-Schlaf	non rapid eye movement Schlaf
NREM-Schlaf	non rapid eye movement Schlaf
POT	power based on scalp potentials
REM-Schlaf	rapid eye movement Schlaf
SOREM-Schlaf	sleep onset rapid eye movement Schlaf
WEC	wake with eyes closed

Publikationsliste

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* equal contribution

1. Einleitung

Die Schlafmedizin hat sich in der Vergangenheit vornehmlich auf die Definition, Identifikation und Behandlung von Schlafproblemen fokussiert. Jüngst rückt sowohl in der wissenschaftlichen als auch in der gesellschaftlichen Diskussion die Schlafgesundheit immer mehr in den Fokus. Empirische Daten zeigen verschiedene Dimensionen von Schlaf in Verbindung mit Gesundheit. Buysse et al. definiert Schlafgesundheit wie folgt: „Schlafgesundheit ist ein multidimensionales Bild von Schlaf-Wach-Mustern, welches an individuelle, soziale und Umweltanforderungen adaptiert ist und körperliches sowie mentales Wohlbefinden fördert. Guter Schlaf ist durch subjektive Zufriedenheit, angemessenes Schlaf-Timing, adäquate Schlafdauer, hohe Schlaf-Effizienz und nachhaltige Wachheit während der Wachstunden charakterisiert“ (1).

Eine pathologische Veränderung der Schlafdauer und des Schlafrhythmus wie beispielsweise beim Krankheitsbild der Narkolepsie senkt in hohem Maße die gesundheitsbezogene Lebensqualität der betroffenen Patienten (2). Gleichwohl gibt es eine stetig wachsende Szene gesunder Individuen, die durch gezielte Veränderung der Schlafdauer und des Schlafrhythmus versucht, den individuellen Tagesablauf im Sinne von weniger Schlafdauer → mehr Wachheit zu optimieren. Dabei sind die Auswirkungen auf die Schlafgesundheit, auf das Traumverhalten und letztendlich auf die Gesamtgesundheit noch wenig erforscht (3).

1.1. Schlaf und Traum

Der physiologische Schlaf ist dynamisch durch zyklisch wechselnde Schlafstadien charakterisiert, die allgemein in rapid eye movement (REM)-Schlaf und non-rapid eye movement (NREM)-Schlaf eingeteilt werden (4) (Abb. 1). Der NREM-Schlaf wird wiederum in 4 Schlafstadien untergliedert, wobei die Schlafstadien 1-2 den Leichtschlaf und die Schlafstadien 3-4 den Tiefschlaf kennzeichnen. Der REM-Schlaf unterscheidet sich wesentlich von den anderen Schlafstadien und ist durch Erschlaffung der Skelettmuskulatur und gleichzeitig schnelle Augenbewegungen gekennzeichnet. In seiner elektrophysiologischen Aktivität ähnelt der REM-Schlaf dem Wachzustand, und geht subjektiv häufig mit intensiven Traumphasen einher (4,5,6).

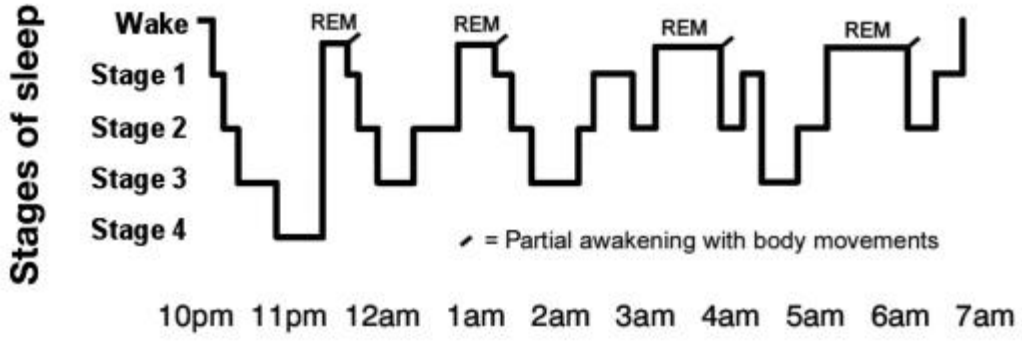

Abbildung 1: Die verschiedenen Schlafstadien über die Nacht verteilt (4)
Dement W, Kleitman N. The relation of eye movements during sleep to dream activity: an objective method for the study of dreaming. J Exp Psychol. 1957;53(5):339-46.
Hinweis: Die Abbildung ist gemeinfrei.

1.2. Schlaffragmentierung

Schlaffragmentierung beschreibt die Unterbrechung des Schlafes durch exogene oder endogen Weckreize und hat folglich Auswirkungen auf die Schlaftiefe, das Traumverhalten und auf das Erwachen. Wie sich ein fragmentierter Schlafrhythmus auf die Schlafgesundheit auswirkt, ist aktuell Gegenstand der Schlafforschung.

Bezogen auf das Traumverhalten zeigen einzelne Forschungsergebnisse bereits, dass fragmentierter Schlaf das Traumverhalten beeinflusst. So wird beispielsweise gezeigt, dass schnelle wiederkehrende Abschnitte von Wach- und Schlafperioden mit einem vermehrten Aufkommen von Luzidträumen einhergehen. Die Luzidität kann beispielsweise durch die Anzahl von nächtlichem Erwachen und die Anwendung der „Schlummerfunktion“ beim Wecksignal während des morgendlichen Schlafes gesteigert werden (7). Experimentelle Studien zeigen darüber hinaus, dass ausgedehnte Wachperioden in den frühen Morgenstunden die Chance auf einen Luzidtraum in anschließenden Schlafperioden erhöht (8,9). Die Technik dazu, dies gezielt zu trainieren, wird als „wake-back-to-bed“ Methode bezeichnet (10).

1.3. Schlaf und Narkolepsie

Narkolepsie ist eine Erkrankung, die durch einen gestörten Schlaf-Wach-Rhythmus gekennzeichnet ist. Der Schlafrhythmus ist ungeordnet und von den Patienten mit Narkolepsie nicht steuer- oder beinflussbar. Neben der pathologischen Schlaffragmentierung der Narkolepsie gibt es auch physiologische sowie künstlich induzierte Änderungen des Schlaf-Wach-Rhythmus. Das Krankheitsbild der Narkolepsie ist gekennzeichnet durch die Tetrade: gestörter Nachtschlaf-Wach-Rhythmus, exzessive Tagesschläfrigkeit, unaufhaltsame Schlafattacken und Kataplexie (11,12), wobei die Kataplexie nicht bei allen Patienten mit Narkolepsie auftaucht (Unterscheidung von Typ 1 mit Kataplexie und Typ 2 ohne Kataplexie). Neben dem Schlafverhalten ist auch das Traumverhalten beeinträchtigt. Patienten mit Narkolepsie weisen eine signifikant höhere Traumerinnerungsrate auf und beschreiben ihre Träume häufig als sehr lebhaft und beunruhigend (13,14). Die Trauminhalte werden als negativ, bizarr und zeitlich sehr ausgedehnt beschrieben (15,16). Daneben ist auch die Alptraumrate nicht nur gegenüber Kontrollgruppen, sondern auch verglichen mit anderen Schlafstörungen signifikant erhöht

(17). Das charakteristische vorzeitige Auftreten des REM-Schlafes (SOREM) führt zu direkten Traumphasen nach dem Einschlafen, welche oft als sehr unangenehm beschrieben werden, da die Unterscheidung zwischen Traum und Realität schwerfällt (18,19). Ein ähnlicher Effekt wird ebenfalls beim Aufwachvorgang erlebt, welcher als „falsches Erwachen“ bezeichnet wird. Hier wird der Aufwachvorgang zwar bewusst aber im Traum erlebt, so dass auch hier eine Unterscheidung zwischen Traum und Realität schwerfällt (20).

1.4. Polyphasischer Schlaf

Wir Menschen sind gewöhnlich monophasische Schläfer mit einer einzigen Schlafperiode in der Nacht. Jedoch findet man bei den meisten Säugetieren einen polyphasischen Schlafrhythmus vor, und manche zeigen sogar einen ultrakurzen Schlaf-Wach-Rhythmus auf, wenn sie in einer gefährlichen Umgebung in der Natur leben. Dennoch zeigen auch wir Menschen polyphasische Schlafrhythmen, bspw. im Säuglingsalter, in Erkrankungsphasen oder bei 24h-Tätigkeiten (21,22). Claudio Stampie geht in der Publikation „why we nap“ der Frage nach, ob der Mensch sich seinen Schlaf zwingend über eine singuläre regelmäßig nächtliche Schlafperiode holen muss, oder ob er auch in mehreren Kurzschlafphasen ausreichend Schlaf erhalten kann (21,22). Diese Frage gewinnt immer mehr an Bedeutung, gerade in unserer immer rastloser werdenden „Rund-um-die-Uhr-Gesellschaft“ (21). Dabei sind die Auswirkungen auf das Traumverhalten noch gänzlich unerforscht.

Polyphasischer Schlaf definiert alle Schlafmuster mit multiplen Schlafepisoden im Gegensatz zum monophasischen Schlaf mit nur einer Schlafepisode pro Tag. Typischerweise kennzeichnet der polyphasische Schlaf eine konstante Schlafphase in der Nacht (Kernschlaf) und kurze Schlafepisoden am Tag (Ankerschlaf), im Schnitt 20-30 min. Diese kurzen Schlafepisoden müssen stets im gleichen Zeitabstand über den Tag verteilt durchgeführt werden. In Tabelle 1 werden die verschiedenen polyphasischen Schlafmuster aufgeführt.

Tabelle 1: Striktes polyphasisches Schlafmuster mit dem typischen Schlafaufbau (23,24)

Schlafplan Name	Anker-schlaf (20 min)	Kern-schlaf (Std)	Gesamt-schlaf (Std)	Benefit (vs. 8 Std)
Monophasisch	0	8	8	0
Biphasisch	1	6	6,3	1,7
Everyman 2	2	4,5	5,2	2,8
Everyman 3	3	3	4	4
Everyman 4	4	1,5	2,8	5,2
Uberman	6	0	2	6

Das Everyman-Schlafmuster kann in seiner Kernschlaufdauer und der Anzahl der Ankerschlafperioden variiert werden. Mit abnehmender Kernschlaufdauer nimmt die Anzahl der Ankerschlafperioden zu.

Das Ubermann-Schlafmuster zeigt keinen Kernschlaf und ist gekennzeichnet durch sechs 20-minütige Ankerschlafperioden im exakt gleichen Abstand zueinander verteilt über 24 Stunden (equihexaphasisch) (24). Daraus ergibt sich eine Gesamtschlafdauer von 2 Stunden auf einen 24-Stunden-Tag verteilt.

1.5. Luzides Träumen

Luzides Träumen, auch Klarträumen genannt, beschreibt das Phänomen, dass der Träumer sich im Traumzustand bewusst ist, dass er träumt (25). Während des luziden Träumens ist der Träumer fähig, die Aktionen im Traum bewusst zu kontrollieren, den Traumverlauf bewusst passiv zu beobachten oder bewusst aus einem Traum aufzuwachen (26,27). Die

Prävalenz in der deutschen Bevölkerung liegt bei ca. 50% und tritt signifikant höher bei Frauen auf. Zudem sinkt das Auftreten von Luzid-Träumen im Alter ab (28). Diese Möglichkeit des Bewusstwerdens in Träumen bietet Menschen, die unter Alpträumen leiden, die Möglichkeit, die Alptraumrate, die Intensität und den resultierenden psychischen Stress zu minimieren (26,29,30).

Luzidität tritt überwiegend während des REM-Schlafs auf, ist aber kein alleiniges Phänomen des REM-Schlafs, sondern zeigt sich auch im Non-REM-Schlaf (31). Die Unterschiede zwischen Luzidträumen während eines REM- und eines Non-REM-Schlafs sind sehr subtil und scheinen während einer Non-REM-Periode weniger emotional und weniger visuell lebhaft aber gleichzeitig mehr gedankenhaft (32) zu sein.

Um einen nicht luziden REM-Schlaf in einen luziden REM-Schlaf zu lenken, kann ein Wechsel in der Gehirnaktivität in Richtung Wachzustand zielführend sein. Studien geben Hinweise auf eine kortikale Aktivität im niedrigen gamma-Frequenz-Bereich (40 Hz), jedoch ist noch unklar, ob dieser Frequenzbereich eine entscheidende Rolle spielt. Die frontale und fronto-laterale Region spielt eine Schlüsselrolle in der Erzeugung von Luzidität im Traum (33,34) (Abb. 2).

Darüber hinaus gibt es Studien, die die Induktion von Luzditräumen mit transkranieller Stimulierung (tACS, tDCS) untersuchten. Die Anwendung der transkraniellen Stimulierung zeigte interessante Effekte auf eine gesteigerte Traumkognition. Eine verlässliche Methode zur Luzidtrauminduktion konnte jedoch noch nicht gezeigt werden (32,35).

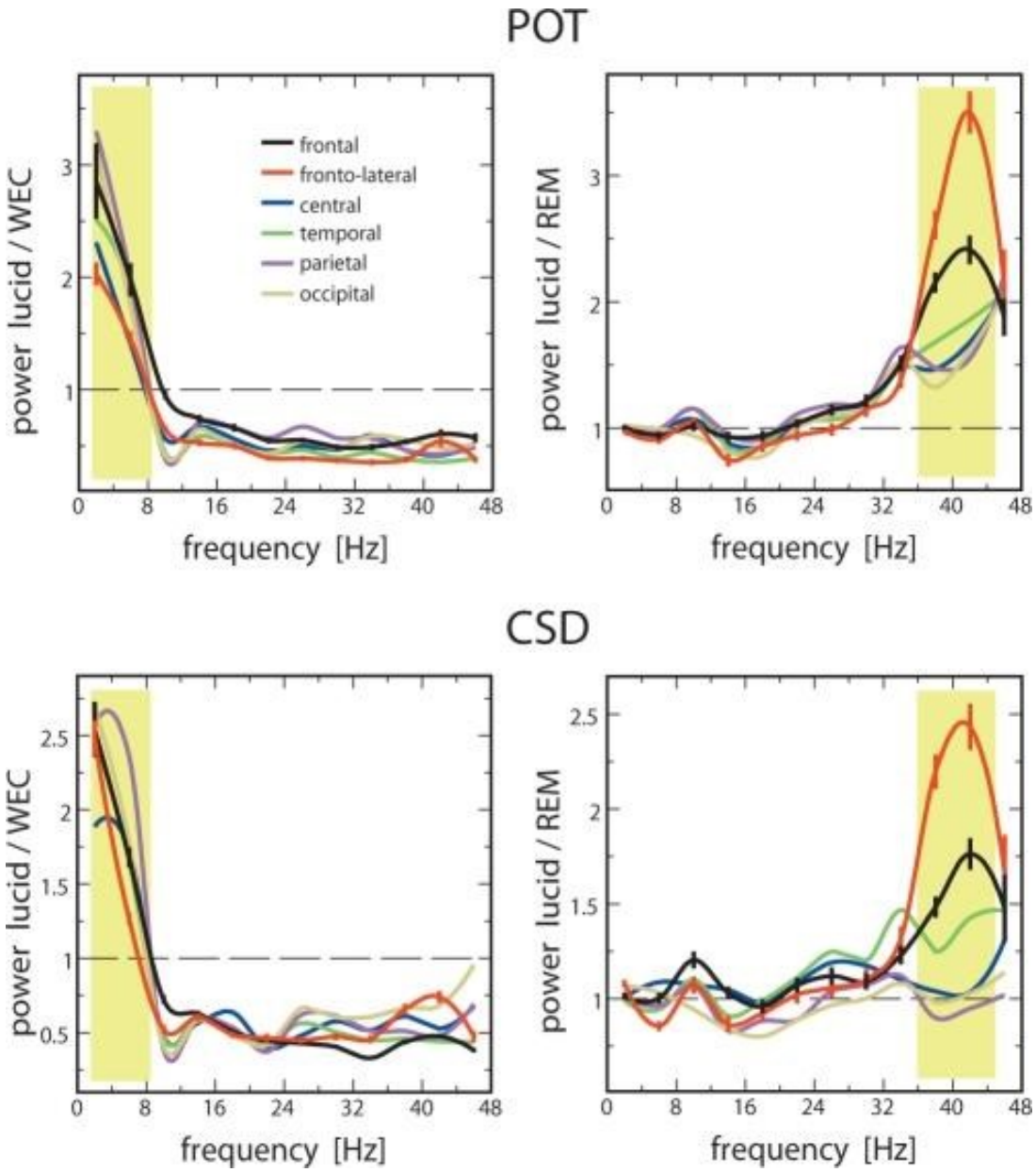

Abbildung 2: Region von Interesse: Komplettes Frequenzspektrum zwischen Luzidtraum und Wachzustand (links) und zwischen Luzidtraum und REM-Schlaf (rechts). Die Analysen wurden in POT (scalp potential) und in CSD (current source density) durchgeführt (33).

1.6. Metakognition im Schlaf

Es gibt zwei wesentliche neuronale Merkmale, die mit Luzidträumen im Zusammenhang stehen. Zum einen wird der „Moment der Luzidität“ beschrieben – ein vorübergehender Moment von Meta-Bewusstsein, in welchem der/die Träumende die metakognitive Einsicht hat, dass er gerade träumt (36). Zum anderen wird der Wechsel des Bewusstseinszustandes bei einem Übergang von nicht luziden zu luziden Träumen beschrieben. Dieser ist gekennzeichnet durch gesteigerten Willen, episodisches Gedächtnis und einen Zugang zur Metakognition (37, 38)

Die praefrontale Hirnregion unterliegt der Metakognition im Wachzustand. Im REM-Schlaf ist die Fähigkeit zur Metakognition heruntergefahren, welches sich in der Traumerinnerung und im Bewusstwerden darüber erst nach dem Erwachen zeigt (39). Der Luzidtraum geht mit einer erhöhten praefrontalen Hirnaktivität und einer erhöhten Metakognition einher (40).

1.7. Übergeordnete Fragestellung

In beiden Publikationen wurde die Auswirkung von pathologischer und künstlich induzierter Schlaffragmentierung auf das Traumverhalten, insbesondere auf das Luzidtraumverhalten untersucht. Ziel war es, mögliche Einflussfaktoren auf die Luzidität zu erfassen. Es zeigte sich ein deutlicher Zusammenhang zwischen Schlaffragmentierung und Luzidträumen, welcher durch eine anhaltend höhere Aktivität des praefrontalen Kortex und dadurch ein schnelles Wiedereintreten in den REM-Schlaf aus einem erwachten Zustand zu erklären ist. Dies führt zu einem förderlichen neuronalen Milieu für Luzidtraumerfahrungen. Jedoch sind die spezifischen neuronalen Mechanismen, die dem zugrunde liegen, noch nicht vollständig erfasst. In weiteren Studien müssen die Wach-

REM-Perioden und ihre Rolle der Potenzierung von neurophysiologischen Mechanismen, die der Metakognition im Traum unterliegen, geprüft werden.

Eine alleinige Aktivierung des praefrontalen Kortex erklärt jedoch noch nicht alleine die reichhaltigeren und lebhafteren Trauminhalte bei Patienten mit Narkolepsie verglichen mit gesunden Probanden in einem polyphasischen Schlafmuster. Es wäre deshalb interessant zu untersuchen, ob die Luzidität bei Patienten mit Narkolepsie mit Wach-REM-Perioden im Schlaf assoziiert ist. Gerade das zusätzliche Auftreten einer erhöhten Alptraumrate stellt für Patienten mit Narkolepsie eine starke Belastung dar, welchem durch ein gezieltes Luzidtraumtraining positiv begegnet werden kann. In weiteren Studien sollte die Theorie zur Alptraum-Ätiologie anhand von EEG/fMRI-Studien überprüft werden, ob der hyperaktive REM-Schlaf in einer erhöhten Aktivierung des Limbischen Systems, insbesondere der Amygdala, begründet ist.

Beide Studien zeigen ein plausibles Bild, demzufolge ein schnelles Wiedereintreten in einen REM-Schlaf aus einem wachen oder erregten Zustand eine Luzidtraumerfahrung erhöht. Dabei wird dieser Effekt durch fragmentierten Schlaf wie bei Narkolepsie oder durch polyphasische Schlafmuster potenziert. Dieser wurde bereits neurophysiologisch mit der Luzidtraum-Induktionstechink „wake-back-to-bed“ Technik untermauert.

1.8. Beitrag des Doktoranden

Die Studien „Increased Lucid Dreaming frequency in Narcolepsy” und Studie 2 „Polyphasic sleep“ der Publikation “Sleep fragmentation and lucid dreaming” wurden von Michael Rak selbständig konzipiert, durchgeführt und ausgewertet und anschließend in den jeweiligen Publikationen beschrieben.

In der ersten Studie wurden die Patientenkontaktdaten von Koautor Pierre Beitinger bereitgestellt. Der Autor erhob selbständig alle Daten des Patientenkollektivs und wertete diese statistisch selbständig aus, wobei er durch Michael Schredl begleitet wurde. Darüber

hinaus standen Martin Dresler und Axel Steiger als Koautoren unterstützend beim Studiendesign und bei der wissenschaftlichen Betreuung zur Seite.

Die zweite Publikation „Sleep fragmentation and lucid dreaming“ umfasst vier zusammenhängende Studien, die an mehreren Instituten durchgeführt und gemeinsam veröffentlicht wurden. Alle Autoren haben sich auf Grund der thematischen Passung und angesichts der Publikationsinflation der letzten Jahre bewusst dazu entschieden alle 4 Studien zu einer Publikation zusammen zu fassen, welche auch jeweils eigenständig hätten publiziert werden können. In Studie 2 „Polyphasic Sleep“ haben die Koautoren Martin Dresler und Axel Steiger den Autor beim Studiendesign und in der wissenschaftlichen Betreuung unterstützt. Die Betreuung der Studienteilnehmer während und nach dem Zeitraum des polyphasischen Schlafrhythmus sowie die Erhebung der Daten führte der Autor selbständig durch. Anschließend wurde die Publikation vom Autor verfasst. Neben den in der Publikation dargestellten Daten zum Zusammenhang zwischen polyphasischem Schlaf und luziden Träumen hat die Studie noch sehr viele weitere Daten ohne Bezug zum Traumverhalten produziert. Es wurde wöchentlich mit Fragebögen (BDI-V2, ASRM, FLei) sowohl der psychische Zustand als auch die geistige Leistungsfähigkeit der 12 Probanden im Schlaflabor evaluiert. Weiterhin wurden während eines 24-Stunden-Monitorings eine Polysomnographie und regelmäßige Blutabnahmen alle 30 min vor und nach der Umstellung in das polyphasische Schlafmuster durchgeführt. Während dieser Zeiträume mit 24-Stunden-Monitoring wurden diverse kognitive Testungen durchgeführt.

Keine der beiden Publikationen, die dieser Dissertation zugrunde liegen, wird von einem anderen Co-Autoren für eine abgeschlossene oder laufende Promotionsarbeit verwendet.

2. Zusammenfassung

2.1. Increased Lucid Dreaming Frequency in Narcolepsy

In der ersten Studie wurde die Auswirkung pathologischer Schlaffragmentierung der Narkolepsie auf das Traumverhalten untersucht. Dazu wurden 60 Patienten mit der gesicherten Diagnose Narkolepsie aus dem Max-Planck-Institut für Psychiatrie in München und 919 Kontrollprobanden mit Hilfe eines strukturierten Telefoninterviews befragt. Zunächst wurde die Traumerinnerungsrate, die Alptraumrate sowie die Luzidtraumrate in beiden Kollektiven ermittelt. Zusätzlich wurde bei den Patienten mit Narkolepsie erfragt, ob es Unterschiede in der Traumerinnerungsrate, der Alptraumrate sowie der Luzidtraumrate während eines früheren medikamentenfreien Intervalls gab. Abschließend wurde eruiert, ob Patienten mit Narkolepsie, die Erfahrung mit Luzidträumen haben, dadurch Erleichterung während Alpträumen erfahren.

Die Auswertung der Interviewdaten durch logistische Regression ergab eine signifikant höhere Traumerinnerungsrate, Alptraumrate sowie Luzidtraumrate bei Patienten mit Narkolepsie verglichen mit den Probanden der Kontrollgruppe. Die spezifischen Symptome der Narkolepsie wie fragmentierter Nachtschlaf, eine kurze Schlaflatenz und „sleep onset REM“-Perioden führten zu den gleichen Effekten wie der „wake-back-to-bed“ Schlafrhythmus, welcher die Luzidtraumrate bei gesunden Probanden erhöht. Auf den ersten Blick wirken die Ergebnisse inkonsistent, wenn man diese mit der Masse älter werdender Personen vergleicht, welche normalerweise ein Abfallen der Traumerinnerungsrate bei gleichzeitigem Anstieg nächtlichen Erwachens aufweisen. Dies kann jedoch durch den Abfall der Gedächtnisfunktion, die reduzierte zirkadiane Modulation des REM-Schlafs und einen Abfall der REM-Schlafzeit erklärt werden.

Eine erhöhte Traumerinnerungsrate bei Patienten mit Narkolepsie kann durch das „arousal-retrieval-model“ erklärt werden: Für die Traumerinnerung ist ein gewisser Wachheitsgrad

und Intensität des Traumes nötig, um diesen ins Kurzzeitgedächtnis zu überführen. Wie auch bei Insomnie, liegt auch bei Patienten mit Narkolepsie ein hohes Aufkommen von Erwachen während der Nacht vor, welches das Erinnern der Träume im Vergleich zu gesunden Probanden begünstigt.

Alpträume, die bei Patienten mit Narkolepsie vermehrt vorkommen, werden auch als länger, komplexer, negativer und lebendiger beschrieben. Das erhöhte Vorkommen der Alpträume kann durch den erhöhten Stresslevel der spezifischen Narkolepsiesymptome über den Tag erklärt werden, welcher sich nachts im Schlaf zu spiegeln scheint.

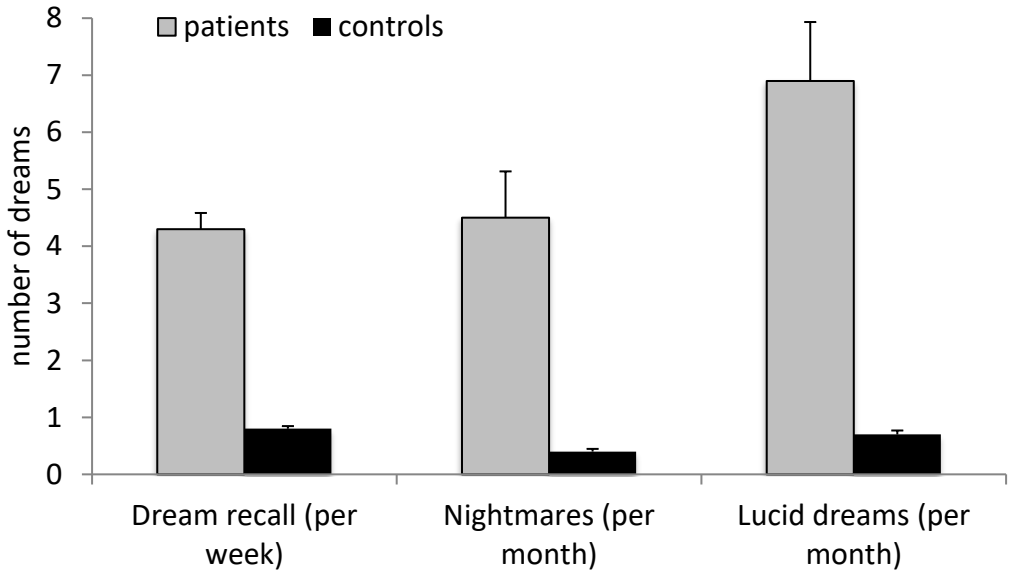

Abbildung 3: Traumerinnerungsrate, Alptraumerinnerungsrate und Luzidtraumrate bei Patienten mit Narkolepsie und Kontrollgruppe. Die Unterschiede sind statistisch signifikant.

70% der Patienten mit Narkolepsie, die Erfahrung mit Luzidträumen haben, können ihre Alpträume positiv beeinflussen und erfahren dadurch Erleichterung, was ein gezieltes Luzidtraumtraining bei Patienten mit Narkolepsie mit hoher Alptraumbelastung empfiehlt. In einem früheren medikamentenfreien Intervall zeigten die Patienten mit Narkolepsie eine signifikant höhere Traumerinnerungsrate und Alptraumrate als unter Medikation.

Interessanterweise ergab die Auswertung keinen signifikanten Unterschied in der Luzidtraumrate im Vergleich zu einem medikamentenfreien Intervall.

2.2. Sleep Fragmentation and Lucid Dreaming

Luzidträume sind mit gesteigerten neuronalen Aktivitäten in präfrontalen Hirnregionen verglichen mit dem Wachzustand assoziiert. Einzelne Studien zur Luzidtrauminduktion haben gezeigt, dass die Wahrscheinlichkeit, einen Luzidtraum zu erleben, durch abwechselnde Perioden von Wachsein und Schlaf steigt.

In der Originalarbeit wird über vier thematisch zusammenhängende Studien berichtet, welche die Beziehung zwischen Schlaffragmentierung und luzidem Träumen untersuchten. Schlaffragmentierung, nächtliches Erwachen, Schlafqualität und polyphasische Schlafrhythmen wurden mit objektiven und subjektiven Messmethoden erfasst. Die Ergebnisse aller 4 Studien zeichnen ein detaillierteres Bild des bisher vermuteten Zusammenhangs zwischen Schlaffragmentierung und Luzidträumens ab.

In der Studie „Polyphasic sleep“ wurden 22 freiwillige Probanden zu ihrer individuellen Luzidtraumrate während eines monophasischen und polyphasischen Schalfrhythmus befragt. 10 der 22 Probanden wechselten in einen radikal polyphasischen Schlafrhythmus – auch Uberman genannt. Dieser bestand aus 6 Schlafperioden von 20 min Dauer über einen 24-Stunden-Zyklus verteilt, während die restlichen 12 Probanden ähnliche polyphasische Schlafmuster zeigten.

Danach erlebten Probanden unter einem radikal polyphasischen verglichen mit einem monophasischen Schlafrhythmus mehr luzide Träume, sowohl absolut als auch relativ zur allgemeinem Traumfrequenz.

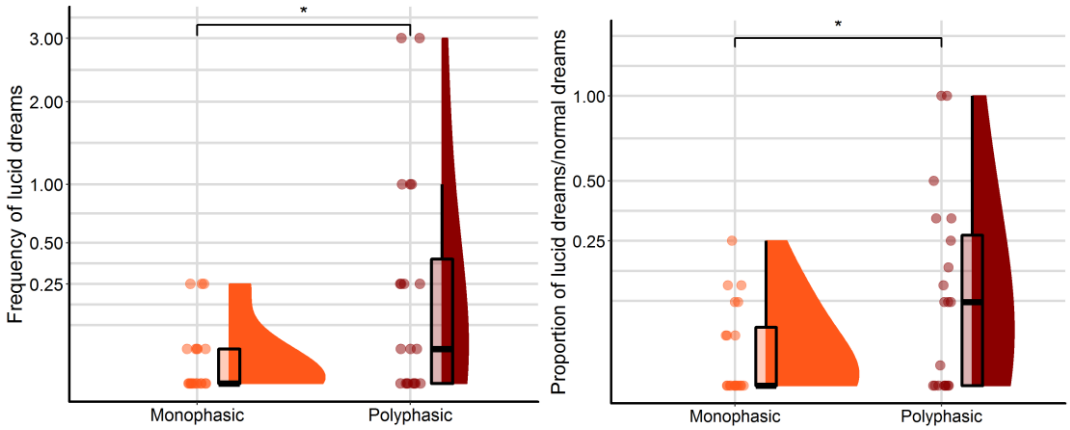

Abbildung 4: Selbsterfasste Luzidtraumrate ist während eines polyphasischen Schlafrhythmus verglichen mit einem monophasischen Schlafrhythmus erhöht. Links: Absolute Anzahl von Luzidträumen pro Tag. Rechts: Anzahl der Luzidträume pro Tag bezogen zur allgemeinen Traumerinnerungsrate.

Während eines monophasischen Schlafmusters können ebenfalls kurze Perioden von Wachheitsphasen auftreten. Vergleicht man diese mit den viel länger andauernden Wachheitsphasen unter einem polyphasischen Schlafmuster, so erhöht sich die Wahrscheinlichkeit für REM-Schlaf-Perioden in den vermehrten Schlafperioden, welche direkt auf die Wachheitsphasen folgen. Diese beiden Aspekte Schlaffragmentierung und das schnelle Wiedereintreten der REM-Schlaf-Phasen nach Phasen der Wachheit erhöht die Wahrscheinlichkeit auf einen immer noch aktivierten praefrontalen Cortex, welcher auf REM-Schlaf-Episoden trifft und dadurch die Chance, eine Luzidtraumerfahrung zu machen.

Darüber hinaus konnte gezeigt werden, dass die Selbsteinschätzung über die Anzahl des nächtlichen Erwachens und physiologisch validierte Sequenzen aus Wachheit und REM-Schlaf mit Luzidträumen in Zusammenhang stehen. Jedoch konnten weder eine selbsterfasste Schlafqualität noch die physiologisch validierte Anzahl von nächtlichem Erwachen mit Luzidträumen in Verbindung gebracht werden.

3. Summary

3.1. Increased Lucid Dreaming Frequency in Narcolepsy

The first study examined the effect of pathological sleep fragmentation in patients with narcolepsy on dream behavior. Therefore, 60 patients with the confirmed diagnosis of narcolepsy from the Max Planck Institute for Psychiatry in Munich and 919 control subjects were questioned using a structured telephone interview. First, the dream recall frequency, the nightmare frequency and the lucid dream frequency were determined in both collectives. In addition, the patients with narcolepsy were asked whether there were differences in the dream recall frequency, the nightmare frequency, and the lucid trauma frequency during an earlier drug-free interval. Finally, it was determined whether patients with narcolepsy who have experience with lucid dreaming experience relief during nightmares.

The evaluation of the interview data using logistical regression revealed a significantly higher dream recall frequency, nightmare frequency and lucid dream frequency in patients with narcolepsy compared to the subjects in the control group. The distinctive symptoms of narcolepsy like fragmented night sleep, short sleep latency and “sleep-onset REM "periods lead to the same effects as the “wake-up-to-bed" sleep rhythm, which increases the lucid dream frequency in healthy control subjects. At first sight a parallel decrease of dream frequency and an increase of nocturnal awakening as seen in normal ageing appears inconsistent with this view. However, this can be explained by the decrease in memory function, reduced circadian modulation of REM sleep, and a decrease in REM sleep time.

An increased dream recall frequency in patients with narcolepsy can be explained by the "arousal-retrieval model": For dream recall, a certain degree of wakefulness and intensity of the dream is necessary to transfer it into short-term memory. As in insomnia, patients with narcolepsy have a high incidence of awakenings during the night, which favors dream recall compared to healthy subjects.

Nightmares, which occur more frequently in patients with narcolepsies, are also described as longer, more complex, more negative, and more vivid. The increased incidence of nightmares may be explained by the increased stress level of the specific narcolepsy symptoms throughout the day, which seems to be mirrored in sleep at night.

70% of patients with narcolepsy who have experience with lucid dreaming can positively influence their nightmares and experience relief, which recommends targeted lucid dream training in patients with narcolepsy with high nightmare burden.

During an earlier medication-free interval, patients with narcolepsy showed significantly higher dream recall and nightmare rates than on medication. Interestingly, the evaluation revealed no significant difference in lucid dream rate compared with a medication-free interval.

3.2. Sleep Fragmentation and Lucid Dreaming

Lucid dreams are associated with increased neural activity in prefrontal brain regions compared to wakefulness. Single studies on lucid dream induction have shown that alternating periods of wakefulness and sleep increase the probability of experiencing a lucid dream.

The original paper reports four thematically related studies that investigated the relationship between sleep fragmentation and lucid dreaming. Sleep fragmentation, nocturnal awakenings, sleep quality, and polyphasic sleep rhythms were assessed using objective and subjective measurement methods. The results of all 4 studies provide a more nuanced picture of the previously suspected relationship between sleep fragmentation and lucid dreaming.

In the study "Polyphasic sleep" 22 volunteers were asked about their individual lucid dream rate during a monophasic and polyphasic sleep rhythm. 10 of the 22 subjects switched to a radical polyphasic sleep rhythm - also called Uberman. This consisted of 6 sleep periods of

each 20 minutes spread over 24 hours, while the remaining 12 subjects showed similar polyphasic sleep patterns.

According to this, subjects under a radically polyphasic compared to a monophasic sleep rhythm experienced more lucid dreams, both in absolute terms and relative to general dream frequency.

Short periods of wakefulness may also occur during a monophasic sleep pattern. Comparing these to the much longer lasting periods of wakefulness under a polyphasic sleep pattern, the propability of REM sleep periods increases in the increased sleep periods that directly follow periods of wakefulness. These two aspects of sleep fragmentation and the rapid re-entry of REM sleep periods after periods of wakefulness increase the propability of still-activated prefrontal cortex encountering REM sleep episodes and thus the chance of having a lucid dream experience.

Furthermore, self-assessment of the number of nocturnal awakenings and physiologically validated sequences of wakefulness and REM sleep were shown to be related to lucid dreams. However, neither self-assessed sleep quality nor physiologically validated number of nocturnal awakenings could be associated with lucid dreams.

4. Veröffentlichungen

4.1. Increased lucid dreaming frequency in narcolepsy

Increased Lucid Dreaming Frequency in Narcolepsy

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Study Objective: Nightmares are a frequent symptom in narcolepsy. Lucid dreaming, i.e., the phenomenon of becoming aware of the dreaming state during dreaming, has been demonstrated to be of therapeutic value for recurrent nightmares. Data on lucid dreaming in narcolepsy patients, however, is sparse. The aim of this study was to evaluate the frequency of recalled dreams (DF), nightmares (NF), and lucid dreams (LDF) in narcolepsy patients compared to healthy controls. In addition, we explored if dream lucidity provides relief during nightmares in narcolepsy patients.

Design: We interviewed patients with narcolepsy and healthy controls.

Setting: Telephone interview.

Patients: 60 patients diagnosed with narcolepsy (23–82 years, 35 females) and 919 control subjects (14–93 years, 497 females)

Interventions: N/A.

Measurements and Results: Logistic regression revealed significant ($P < 0.001$) differences in DF, NF, and LDF between narcolepsy patients and controls after controlling for age and gender, with effect sizes lying in the large range (Cohen's $d > 0.8$). The differences in NF and LDF between patients and controls stayed significant after controlling for DF. Comparison of 35 narcolepsy patients currently under medication with their former drug-free period revealed significant differences in DF and NF ($z < 0.05$, signed-rank test) but not LDF ($z = 0.8$). Irrespective of medication, 70% of narcolepsy patients with experience in lucid dreaming indicated that dream lucidity provides relief during nightmares.

Conclusion: Narcolepsy patients experience a markedly higher lucid dreaming frequency compared to controls, and many patients report a positive impact of dream lucidity on the distress experienced from nightmares.

Keywords: narcolepsy, dreaming, nightmares, lucid dreaming

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INTRODUCTION

Narcolepsy is a disabling and chronic sleep-wake disorder primarily characterized by excessive daytime sleepiness, irresistible sleep attacks, and cataplexy.[1,2] As other sleep disorders,[3] narcolepsy is associated with changes in dream mentation: Narcolepsy patients have a higher dream recall frequency than both healthy controls and patients with other sleep disorders.[4,5] In addition, narcolepsy patients describe their dreams as very vivid and often disturbing,[6–8] and content analytic studies indicate that dreams of narcolepsy patients are more negative, longer, and more bizarre than dreams of healthy controls.[4,9,10] Nightmare disorder is frequently present in narcolepsy patients[11]; even compared to patients with other sleep disorders, nightmare frequency is increased in narcolepsy.[5]

Lucid dreaming, defined as dreaming during which the dreamer is aware that he or she is dreaming, has been demonstrated to be an effective therapy for recurrent nightmares.[12–14] Although dream phenomenology in narcolepsy is well documented, the occurrence of lucid dreaming in narcolepsy and its possible benefit for nightmare relief has been poorly investigated. The aim of the present study was to investigate lucid dreaming in patients with narcolepsy. Since anecdotal reports and studies with very small sample sizes suggest that narcolepsy patients frequently reported that they are aware of their dreaming state while dreaming,[15–17] we expected a higher number of lucid dreams in this patient group compared to a representative control sample of the general population. As narcolepsy can be conceptualized as a disorder of state boundary control both physiologically and psychologically,[18,19] we further hypothesized that higher lucid dreaming frequency cannot be explained by higher overall dream recall frequency. Phenomenological features of lucid dreaming were studied in this patient group, including the question if narcolepsy patients feel relief through dream lucidity during nightmares.

METHODS

Participants

Eighty-three patients who met diagnostic criteria for narcolepsy (with and without cataplexy) were contacted after giving informed consent to be called for research purposes. Patients were diagnosed via sleep history, polysomnographic recordings of 2 nights, and multiple sleep latency test (MSLT). Physicians of the patients were contacted to confirm the diagnosis of narcolepsy for cases not diagnosed and treated in the Max Planck Institute of Psychiatry, Munich. The diagnosis of narcolepsy according to the International Classification of Sleep Disorders, 1st edition (ICSD-1) criteria was verified in all 83 patients. Of the 83 patients initially contacted, 60 patients participated in the present study (mean age 53.8 years [SD 7.8], age range 23–82 years; 35 women). Reasons for dropouts were as follows: outdated contact information (n = 15), declined to participate (n = 3), not available for interview (n = 2), seriously ill (n = 2), and patient died (n = 1). Mean duration since diagnosis was 14.3 years, SD 7.8, range 4–26 years. Fifty-one patients (85%) were diagnosed with narcolepsy with cataplexy; 40 of

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Table 1—Number of narcolepsy patients receiving medication.

	Medicated	If needed
Modafinil	19	3
Methylphenidate	9	4
Amphetamines	1	
Sodium oxybate	10	
Ephedrine	1	
Caffeine	1	
Antidepressants	27	
Benzodiazepines	1	
Neuroleptics	1	
Opiates	2	

these 51 were HLA positive, one patient was HLA negative, and HLA status was unknown for 10 patients. Fifty patients (83.3%) took medications during the study period; 35 of these could answer questions both for drug-free periods and for the period with the current medication. Of 50 medicated patients, 10 could indicate only about their medicated status and 5 took medication only if needed and were drug-free most of the time. Ten patients reported they were currently completely drug-free, with 5 of them being drug-naïve until now and 5 having taken medication previously but were unable to give further details for this period. Medications of narcolepsy patients are documented in Table 1. A sample of 919 subjects (mean age 48.1, SD 18.4, range 14–93 years; 497 females) representative of the general population served as control group. Further information about this sample is documented elsewhere.[20]

Procedures

Both control subjects and patients were assessed with previously validated dream recall frequency scales via telephone interviews (methodological details are documented elsewhere[20–22]). Specifically, to assess dream recall frequency (DF), participants were asked how often they remembered their dreams in the last few months on a 7-point rating scale ranging from 0 (never) to 6 (almost every morning). To obtain units of dreams per week, the scale was recoded using the class mean as follows: 0: never (class mean 0); 1: less than once a month (between 0 and 1 dream in 4 weeks, class mean 0.125); 2: about once a month (1 dream in 4 weeks, class mean 0.25); 3: two or three times a month (2–3 dreams in 4 weeks, class mean 0.625); 4: about once a week (class mean 1.0), 5:several times a week (2–5 dreams per week, class mean = 3.5); 6: almost every morning (6–7 dreams per week, class mean = 6.5).

Nightmare frequency (NF) and lucid dream frequency (LDF) were assessed using an 8-point rating scale. Definitions of nightmares and lucid dreams were provided (*Nightmares are dreams with such strong negative feelings that one wakes up. The storyline of the nightmare can be remembered very well. / During lucid dreaming, one is, <u>while</u> dreaming, aware of the fact that one is dreaming. It is possible to wake up deliberately, to control the dream action or to observe passively the course of the dream with this awareness.*), and before the patient answered, it was double-checked that patients did not confuse lucid dreams with hypnagogic or hypnopompic hallucinations. The answer categories were as follows: 0: never, 1: less than once a year, 2: about once a year, 3: about 2 to 4 times a year, 4: about once a month, 5: two or three times a month, 6: about once a week, 7: several times a week. In order to obtain units in dreams per month, the scales were recoded using the class means (see above, 0 → 0, 1 → 0.042, 2 → 0.083, 3 → 0.25, 4 → 1.0, 5 → 2.5, 6 → 4.0, 7 → 18.0), i.e., if category 3 (about 2 to 4 times a year) was chosen, the value of 0.25 nightmares/lucid dreams per month was assigned.

For narcolepsy patients, additional questions were added after this first part of the interview. First, we asked if the occurrence of dream lucidity provided relief during nightmares. A 5-point rating scale was used: 1: I don´t know, 2: almost never, 3: rarely, 4: yes, often, 5: yes, almost always. Furthermore, patients were asked when lucid dreams occurred most frequently across the 24-hour cycle. The following categories were provided for the night: 1: never, 2: primarily at sleep onset, 3: primarily in the middle of night, 4: primarily at the end of night; and for the day: 1: never, 2: primarily in the morning, 3: primarily during the day, 4: primarily in the evening.

Statistical Analysis

We compared dream frequency data of the 60 narcolepsy patients with the 919 subjects of the control group via ordinal regression, including gender and age as regressors. Furthermore, via Wilcoxon rank-sum test we compared dream recall, nightmare relief, and diurnal dream lucidity distribution of 10 currently drug-free narcolepsy patients with 45 medicated narcolepsy patients regarding dream frequencies, diurnal distribution of lucid dreaming, and potential relief experienced from dream lucidity during nightmares. We compared these variables also for the current status of 35 medicated narcolepsy patients with their former drug-free periods. Finally, we compared these variables in 10 patients receiving sodium oxybate with their former drug-free period. For all tests, we considered an α of $P = 0.05$ or $z = 0.05$, respectively, as statistically significant. All values are given as means ± SEM unless indicated otherwise.

RESULTS

Dream Recall

Compared to controls, narcolepsy patients had a significantly higher DF (4.3 ± 0.3 vs. 0.8 ± 0.1 dreams per week, $d = 2.1$), NF (4.5 ± 0.8 vs. 0.4 ± 0.1 nightmares per month, $d = 0.9$), and LDF (6.9 ± 1.0 vs. 0.7 ± 0.1 lucid dreams per month, $d = 1.1$), with very large effect sizes illustrating strong differences between the groups (Figure 1). While more than 3 of 4 patients experienced lucid dreams at least from time to time, only every second control subject knew the phenomenon from his or her own experience. Furthermore, DF, NF, and LDF turned out to be higher in women and to decrease with age. After controlling for the effects of gender and age via logistic regression, differences in DF, NF, and LDF between the groups were still highly significant. Also when DF was introduced into the regression analysis, differences in NF and LDF between narcolepsy patients and controls remained statistically significant. Hence, increased frequency of nightmares and lucid dreams in narcolepsy patients cannot be fully explained by increased overall dream recall frequency. For statistical details, see Table 2.

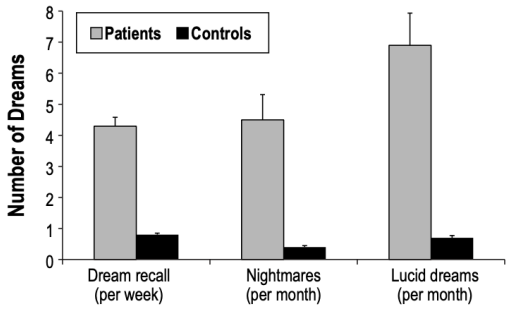

Figure 1—Dream recall frequency, nightmare frequency and lucid dreaming frequency in narcolepsy patients and controls. Shown are means ± SEM; differences in all three comparisons are statistically significant.

Table 2—Logistic regressions comparing narcolepsy patients with controls.

Variable	Standardized Estimate	χ^2	P
Dream frequency			
Diagnosis	0.46	155.2	**0.001**
Age	−0.13	16.0	**0.001**
Sex	0.15	26.6	**0.001**
Nightmare frequency			
Diagnosis	0.40	132.9	**0.001**
Age	−0.15	21.6	**0.001**
Sex	0.08	6.2	**0.013**
Nightmare/dream frequency			
Diagnosis	0.21	32.1	**0.001**
Age	−0.10	8.2	**0.003**
Sex	0.01	0.1	0.778
DF	0.61	225.0	**0.001**
Lucid dream frequency			
Diagnosis	0.32	99.6	**0.001**
Age	−0.09	8.1	**0.004**
Sex	0.11	10.2	**0.002**
Lucid dream/dream frequency			
Diagnosis	0.11	9.1	**0.003**
Age	−0.01	0.0	0.866
Sex	0.03	0.8	0.364
DF	0.75	307.2	**0.001**

Significant effects are indicated by bold print.

Clinical Value and Diurnal Distribution of Lucid Dreaming

Of the 47 patients who experienced lucid dreams at least from time to time, 33 patients felt relieved through dream lucidity during nightmares at least sometimes, with 20 patients profiting from dream lucidity often or almost always. Regarding the diurnal distribution of dream lucidity, 10 patients had lucid dreams primarily at sleep onset, 9 primarily in the middle of the night, and 27 primarily at the end of the night. Furthermore, 2 of 3 patients with lucid dreaming experience also had lucid dreams during daytime. For details, see Table 3.

Medication Effects

DF, NF, and LDF did not differ between 10 drug-naïve and 45 medicated patients ($P > 0.4$ each; 5 narcolepsy patients who took medication infrequently were excluded from this analysis). The diurnal distribution of dream lucidity differed neither during the night nor during the day between subgroups ($P > 0.5$ each). We also found no significant difference between these groups concerning the question whether dream lucidity provided relief during nightmares ($P = 0.68$). We further compared the status of 35 medicated patients with their former drug-free periods and found significant lower DF ($P = 0.018$) and NF ($P < 0.001$) during the medicated status, but no difference for LDF ($P = 0.81$). We also did not find any difference in the diurnal distribution of the occurrence of dream lucidity or experience of relief through dream lucidity during nightmares between medication states ($z > 0.24$ each; Table 3). Finally, we compared the status of 10 patients receiving sodium oxybate with their former drug-free periods and found a significant lower DF ($P = 0.023$) and NF ($P = 0.037$) under sodium oxybate, but again no difference for LDF ($P = 0.14$).

DISCUSSION

We found a strongly increased lucid dreaming frequency in 60 narcolepsy patients as compared to a representative sample of 919 control subjects. This increase stayed significant after statistically controlling for age, gender, and generally increased dream recall frequency in narcolepsy patients. Physiologically, narcolepsy has been conceptualized as disorder of state boundary control,[18] and anatomical changes in the hypothalamus as the key switch of the wake-sleep cycle have repeatedly been documented in narcolepsy.[23,24] Also psychologically, narcolepsy patients sometimes fail to establish the boundaries between real experience and dream mentation during sleep paralysis,[25] or mistake the memory of a dream for a real experience.[19] Our data show that also during dreaming, narcolepsy patients cognitively transgress normal state boundaries and frequently acquire wake-like insight into their current state. An effective strategy to induce lucid dreams in healthy subjects is the so called wake-up-back-to-bed technique, by which subjects are awakened in the early morning hours and go back to sleep after a period of wakefulness.[26,27] In narcolepsy patients, symptoms of fragmented night sleep, a short sleep latency, and sleep onset REM periods might exert effects similar to intentional wake-up-back-to-bed schedules. On first sight a parallel decrease of dream frequency and increase of nocturnal awakening as seen in normal ageing appears inconsistent with this view; however, ageing is also associated with decline in memory functions, reduced circadian modulation of REM sleep, and decrease in REM sleep time,[28,29] which might counteract potential effects of age-related sleep fragmentation on lucid dream frequency.

The finding of higher dream recall frequency in narcolepsy patients is in line with previous studies.[4,5] It might be explained by the arousal-retrieval-model,[30] postulating that dreams can only be retrieved if awakening occurs while a short-term memory trace of the dream is still active. Dream recall frequency is indeed correlated with the number of nocturnal

Table 3—Dream frequency, diurnal distribution of the occurrence of dream lucidity, and experience of relief during nightmares through dream lucidity in all 60 patients with narcolepsy; in 10 drug-naïve and 45 medicated patients; and in 35 medicated patients compared to their former drug-free periods.

	All (n = 60)	Drug-Naïve (n = 10)	Medicated (n = 45)	Drug-Free Phase (n = 35)	Medicated Phase (n = 35)
Dream Frequency					
Dreams per week	4.3 ± 0.3	4.2 ± 0.7	4.3 ± 0.3	**5.3 ± 0.3**	**4.2 ± 0.4**
Nightmares per month	4.5 ± 0.8	5.1 ± 2.2	3.6 ± 0.8	**10.9 ± 1.4**	**3.7 ± 0.9**
Lucid dreams per month	6.9 ± 1.0	7.8 ± 2.8	6.2 ± 1.1	9.1 ± 1.5	7.1 ± 1.4
Dream Lucidity during Nighttime					
Never	14	3	11	10	8
Primarily at sleep onset	10	0	8	5	7
Primarily in the middle of the night	9	2	4	6	3
Primarily at the end of the night	27	5	22	14	17
Dream Lucidity during Daytime					
Never	29	6	22	16	16
Primarily in the morning	2	0	2	0	1
Primarily during the day	28	4	20	19	17
Primarily in the evening	1	0	1	0	1
Nightmare Relief through Lucidity					
Almost always	10	3	6	12	10
Often	10	1	8	3	5
Rarely	13	2	10	7	9
Almost never	8	0	6	7	5
I don't know	19	4	15	6	6

Dream frequencies are given as means ± SEM, diurnal dream lucidity distribution and relief data are given as number of patients. Significant differences between subgroups are indicated by bold print.

awakenings.[31] As in insomnia, in narcolepsy high levels of arousal during both night and day might favor the retention of dream memories, which are forgotten more frequently in healthy subjects.[32] Also, our findings of a negative association between age and dream recall frequency and of a higher dream recall frequency in females compared to males are in line with previous studies.[33–36]

The finding of an increased nightmare frequency (also controlled for general dream recall) in narcolepsy patients confirms many previous studies. Generally, narcolepsy patients experience longer, more complex, more negative, and more vivid dream mentation than healthy subjects,[4,6,8,9,37,38] and nightmares are a frequent symptom in narcolepsy.[5,11] According to a current model of nightmare etiology,[39] two factors might explain increased nightmare frequency in narcolepsy patients. First, daytime stress due to the impairments caused by narcolepsy might be reflected in patients' dreams, as current stressors exert strong effects on nightmare frequency also in healthy controls.[40] Second, brain activation in limbic areas and particularly the amygdala might be higher in narcolepsy patients due to their hyperactive REM sleep system. However, to our knowledge there is no fMRI/EEG study of REM sleep in narcolepsy patients that has tested this hypothesis.

We further found that 70% of the narcolepsy patients in our study who had experience with lucid dreaming indicated that dream lucidity provides relief during nightmares, independent of medication status. Dream lucidity enables dreamers to influence dream content, thereby potentially also altering negative dream mentation.[41,42] Lucid dreaming has indeed repeatedly been shown to be an effective therapy for nightmare disorder.[13,14,43] Anatomical changes of the amygdala,[44] altered amygdala activity related to fear conditioning during wakefulness,[45] and hypermetabolism in further emotion-related brain regions like the anterior cingulate during sleep have been observed in narcolepsy, which might be related to nightmare symptomology.[46] Neurocognitive models of disturbed dreaming emphasize a hyperresponsivity of the amygdala in nightmare generation, coupled with a failure of medial prefrontal regions to dampen this activation.[39] Lateral prefrontal regions have been shown capable to influence amygdala function through connections to the medial prefrontal cortex.[47] The dorsolateral prefrontal cortex activation has been demonstrated to subserve lucid dreaming[48,49] and has been associated with therapeutic effects of lucidity training on recurrent nightmares.[49] Increased lucid dreaming frequency in narcolepsy patients might be considered as an underrecognized opportunity for a systematic therapy of narcolepsy-related nightmare symptoms: patients could be instructed to use dream lucidity to confront fearful dream elements in nightmares and, in turn, change the course of the dream plot. Since lucid dreaming is a skill that can be induced, e.g., by training[50] or electrical brain stimulation,[51] patients without lucid dreaming experience might also profit from such a therapeutic strategy.

Medication status did not affect lucid dreaming frequency or relief experienced through dream lucidity during nightmares. However, patients indicated a significant lower dream recall and nightmare frequency under medication compared to previous drug-free periods. Many medications used in the

treatment of narcolepsy suppress REM sleep, which might explain decreased dream recall and nightmare frequency.[52] Successful treatment reduces distress associated with the disease, which might further reduce nightmare frequency. Furthermore, narcolepsy medications like sodium oxybate improve sleep quality[53,54] and support a regular sleep-wake rhythm.[52,55] As dream recall frequency is correlated with the number of nocturnal awakenings, improved sleep quality might be further factor explaining the reduction of dream recall frequency under medication.[31]

Methodologically, two limitations of the medication data should be mentioned. First, even though coarse medication status for all patients was assessed, it was not possible to obtain specific details about exact medication dosages or times. Second, comparisons between medicated and medication-free periods rely on subjective estimates from memory, as patients were interviewed just once. Future studies on this topic should assess whether lucid dreaming is present before the initial classic manifestations of narcolepsy or whether it emerges concurrently with the classic symptoms or subsequently. It should also be objectively assessed whether lucid dreaming frequency is altered before the start of medication with potentially dream-altering drugs (e.g., antidepressants). Furthermore, more comprehensive information about the narcolepsy-related clinical context should be assessed in order to trace changes in lucid dreaming to their potential causal underpinnings. Finally, a more direct comparison with other sleep disorder patient groups might clarify if changes in lucid dreaming are associated with altered sleep regulation in general or with the specific neurophysiological changes of narcolepsy in particular.

In conclusion, narcolepsy patients reported a markedly increased lucid dreaming frequency compared to a large representative population sample. In line with therapeutic approaches using lucid dreaming training for treatment of nightmare disorder, a majority of patients who had experience with lucid dreaming felt that dream lucidity provides relief during nightmares. Hence, a more systematic use of lucid dreaming has to be considered as a promising approach for treating nightmare symptoms in narcolepsy.

DISCLOSURE STATEMENT

This was not an industry supported study. The authors have indicated no financial conflicts of interest.

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4.2. Sleep fragmentation and lucid dreaming

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Sleep fragmentation and lucid dreaming

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ABSTRACT

Lucid dreaming—the phenomenon of experiencing waking levels of self-reflection within one's dreams—is associated with more wake-like levels of neural activation in prefrontal brain regions. In addition, alternating periods of wakefulness and sleep might increase the likelihood of experiencing a lucid dream. Here we investigate the association between sleep fragmentation and lucid dreaming, with a multi-centre study encompassing four different investigations into subjective and objective measures of sleep fragmentation, nocturnal awakenings, sleep quality and polyphasic sleep schedules. Results across these four studies provide a more nuanced picture into the purported connection between sleep fragmentation and lucid dreaming: While self-assessed numbers of awakenings, polyphasic sleep and physiologically validated wake-REM sleep transitions were associated with lucid dreaming, neither self-assessed sleep quality, nor physiologically validated numbers of awakenings were. We discuss these results, and their underlying neural mechanisms, within the general question of whether sleep fragmentation and lucid dreaming share a causal link.

1. Introduction

Lucid dreaming is a distinct phenomenon whereby waking levels of self-reflection and insight are made available to within one's dreams (Baird et al., 2019). Spontaneous lucid dreaming occurs very infrequently in the general population (Schredl & Erlacher, 2011; Saunders et al., 2016), however its frequency can be enhanced by both intentional strategies and more unintentional mechanisms. Several lines of research suggest that rapidly alternating sequences of wake and sleep periods can be associated with

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increased incidence of lucid dreaming. For example, patients with narcolepsy, who experience fragmented sleep during the night and sleep attacks during day, report significantly increased lucid dreaming frequency (Rak et al., 2015; Dodet et al., 2015). Lucid dreaming frequency has furthermore found to be associated with the self-reported number of nocturnal awakenings, and alarm clock 'snooze' function usage during morning sleep (Smith and Blagrove, 2015). In experimental studies, extended periods of wakefulness during morning hours increases the chance to dream lucidly in subsequent sleep periods (LaBerge et al., 1994; Appel et al., 2020; Erlacher & Stumbrys, 2020); a technique also known as the 'wake-back-to-bed' method (Stumbrys et al., 2012).

While lucid dreaming has been observed during NREM sleep (Stumbrys & Erlacher, 2012), it appears to occur most frequently in REM sleep (Baird et al., 2019). During normal REM sleep, brain regions that have been associated with higher cognitive processing and metacognition in particular, namely the dorsolateral prefrontal and frontopolar cortices, show decreased activation. Lucid dreaming, in contrast, is associated with functional and structural state and trait differences in the dorsolateral prefrontal and frontopolar cortices (Voss et al., 2009; Dresler et al., 2012; Filevich et al., 2015; Baird, Castelnovo, Gosseries, & Tononi, 2018).

A reasonable explanation for the association between lucid dreaming and alternating sequences of wakefulness and sleep would be the assumption that wake-associated prefrontal activation persists into subsequent sleep periods, thereby increasing the occurrence of metacognitive processing. In contrast to continuous sleep, periods of fragmented sleep would thus be expected to be associated with more lucid dreaming – potentially through the promotion a 'hybrid' state between wakefulness and REM sleep (Voss et al, 2009).

Beyond the above mentioned studies, specific investigations into the relation between sleep fragmentation and lucid dreaming have not been conducted. Generally, the association between these two phenomena requires more nuanced investigation before conclusions can be drawn. In particular, whether dream lucidity can best be understood as an artefact of deleterious or disrupted sleep, whether the inverse is true, or whether these phenomenon only incidentally correlate—so far remain unanswered.

Here, we aim to contribute to these questions by investigating the relation between sleep fragmentation and lucid dreaming through four different studies, each targeting different instances of alternating sequences of wake and sleep periods. In *Study 1,* we investigated the association between sleep fragmentation and lucid dreaming using an online survey of 202 participants recruited through internet forums and Twitter. In *Study 2,* dream lucidity was assessed through interviewing 22 volunteers about their dream experiences during and after subsisting on a radically polyphasic sleep schedule. In *Study 3* we assessed the association between self-rated sleep quality and longitudinally assessed lucid dreaming, by administrating clinical diagnostic questionnaires to 42 volunteers. In *Study 4*, we investigated the association between nocturnal arousal features and lucid dreaming, through administration of polysomnography and dream lucidity questionnaires in 30 volunteers.

Though these studies, we explored the association between sleep fragmentation and lucid dreaming using a broad-spectrum analysis of four different aspects of unconventional sleep architecture. Together, these provide a comprehensive portrait of the heterogeneities that occur within sleep architecture; and assess each one independently, in order to determine which (if any) may correlate with or account for aberrant dream metacognition.

2. Study 1: Self-assessed sleep fragmentation and awakenings

2.1. Methods

2.1.1. Participants

202 participants (age 18–63; mean = 28.91; SD = 10.96); 139 (68.81%) male, 60 (29.70%) female, 3 (1.49%) reported their gender as "other". The participants were recruited through internet forums related to lucid dreaming.

2.1.2. Materials and procedures

Dream recall frequency and lucid dreaming frequency were assessed through an online survey (Survey Monkey; https://surveymonkey.com). Beyond biographical data, the survey assessed dream recall frequency and lucid dreaming on a 9-point rating scale (*0, never; 1, less than once a year; 2, about once a year; 3, about 2 to 4 times a year; 4, about once a month; 5, 2 or 3 times a month; 6, about once a week; 7, several times a week; 8, almost every night)* based on a similar scale from Schredl and Erlacher (2004). To obtain units for dream recall and lucid dream frequency per month, the scale was re-coded using the class mean system: 0 → 0, 1 → 0.042, 2 → 0.083, 3 → 0.25,4 → 1.0, 5 → 2.5, 6 → 4.0, 7 → 18.0., 8 → 30.

The following definition of lucid dreaming was provided to ensure participants understood the phenomenon of lucid dreaming: "During lucid dreaming, one is aware of the fact that one is dreaming, while the dream is still ongoing. With this awareness it is possible to control one's dream actions or to observe passively the course of the dream" (Schredl and Erlacher, 2004).

To obtain data on continuity of sleep, participants were asked to rate their sleep on a 5-point scale ranging from: *1, highly continuous; 2, quite continuous; 3, normally continuous; 4, quite fragmented; 5, highly fragmented*. Participants were further asked to self-report: "How many times do you usually wake up at night (give your best estimate)?"

To assess the subjectively perceived link between lucid dreaming and fragmented sleep, participants were finally asked: "Do you have more lucid dreams during: *1, highly continuous; 2, quite continuous; 3, normally continuous; 4, quite fragmented; 5, highly fragmented sleep.*"

2.1.3. Statistical analysis

Two separate partial correlations between lucid dreaming frequency and sleep fragmentation and number of awakenings, respectively, were calculated, each using general dream frequency as a control variable.

Table 1
Questionnaire responses of 202 participants asked for their frequency of dream recall and lucid dreaming.

Response	Dream Recall	Lucid Dreams
never	0	21
less than once a year	1	12
about once a year	0	17
about 2 to 4 times a year	5	45
about once a month	16	47
2 or 3 times a month	23	19
about once a week	33	26
several times a week	61	12
almost every night	63	3

2.2. Results

The number of participants for the different questions differed from the overall sample ($N = 202$), due to missing data. 3 participants skipped the *average sleep continuity* question while one participant skipped the *awakenings per night* question which brought the final number for analysis down to $N = 198$. On average, respondents remembered their dreams 15.8 ± 11.4 times per month; and experienced a lucid dream almost once a week (0.31 ± 0.42) (see Table 1).

The average number of *lucid dreams per month* reported by participants was 2.7 ± 5.7. Most participants rated their sleep as *normally continuous* ($N = 78$) or *quite fragmented* ($N = 51$). The majority of participants reported having 2 or less awakenings per night (N = 147) while (N = 46) reported having between 2 and 5 awakenings, and (N = 5) reported having 6 or more awakenings. Partial correlation analyses revealed a significant association between lucid dreaming frequency and the number of awakenings ($r = 0.25$; $p < 0.001$), but not between lucid dreaming frequency and the amount of sleep fragmentation ($r = 0.05$; $p = 0.244$). However, directly asked for their impression of the association between lucid dreaming and sleep fragmentation, many participants reported having more lucid dreams during *quite fragmented* sleep (see Fig. 1).

2.3. Interim discussion

Lucid dreaming frequency was significantly associated with self-reported number of awakenings per night, as opposed to subjective degrees of fragmentation. Intraindividually, participants did however report having increased lucidity when their own sleep was determined as being fragmented. This paints a relatively mixed picture; tentatively supporting the association between fragmentation and lucidity, but doing little to narrow the focus enough to suggest an underlying physiological mechanism.

The discrepancies in these results could potentially be explained though differences in how participants self-report sleep fragmentation. For example, those whose sleep is notably fragmented (but otherwise satisfactory) could be more likely to accurately report nocturnal awakenings but under-report fragmentation; with those who are dissatisfied with their sleep more likely to exaggerate or over-report degrees of fragmentation relative to actual number of awakenings. It could likewise be the case that the types of fragmentation apt to produce the most severe deleterious sleep quality and subjective dissatisfaction (for instance multiple arousals

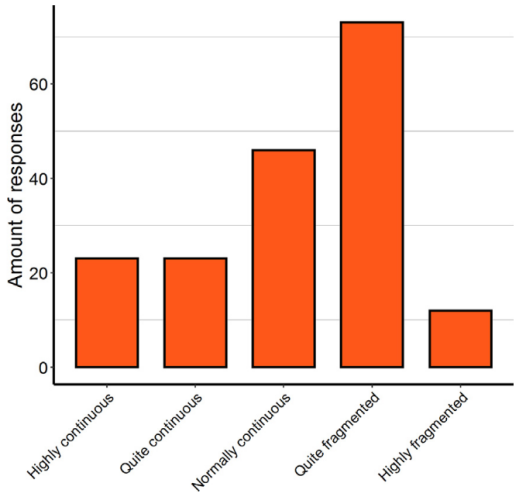

Fig. 1. Levels of sleep fragmentation that survey participants report to be most closely associated with lucid dreaming.

from light sleep) are different from the kinds of fragmentation apt to produce lucidity (arousal and re-entry into REM). It could furthermore be the case that nocturnal arousals from REM are more likely to be remembered the following morning, while multiple arousals from NREM could subjectively be perceived as a single, contiguous period; effectively resulting in under-reporting of nocturnal arousals relative to subjective and objective fragmentation.

This study provided robust indications that some connection between fragmentation and lucidity does exist; however drawing comprehensive conclusions will ultimately necessitate physiological validation, and integration of other research findings.

3. Study 2: Polyphasic sleep

3.1. Methods

3.1.1. Participants

22 volunteers were interviewed on their lucid dream frequency during monophasic vs. polyphasic sleep rhythms. Of these, 10 participants ('in-house group', 1 female, 9 male; age range 21–28, mean 23.9 ± 2.4) participated in a separate study on the cognitive and physiological effects of radically polyphasic sleep, switching to a sleep rhythm of 6 naps of 20 min each, evenly distributed across the 24-h cycle, without any extended night sleep period. We interviewed these participants within 3 months after switching back to a regular monophasic sleep rhythm. In addition, 12 participants ('external group', 1 female, 11 male; age range 18–48, mean 27.8 ± 8.8) who stated to have tried similar polyphasic sleep rhythms previously were interviewed with the same questions via email.

3.1.2. Materials and procedures

All participants of the in-house group underwent 4 weeks of preparation on their normal, monophasic sleep schedule. Sleep timing was secured by sleep diaries and actigraphy. In addition, all subjects and control subjects completed weekly questionnaires including the Altman Self-Rating Mania Scale (ASRM), the simplified Beck Depression Inventory (BDI-V2), and a questionnaire for complains of cognitive disturbance (FLei). A subsample further underwent 24 h of polysomnography (SOMNOwatch; Somnomedics, Randersacker, Germany) and blood sampling every 30 min before changing to the polyphasic sleep schedule. During this 24 h monitoring, participants further underwent tests of declarative and procedural memory, fluid reasoning, and psychomotor vigilance. The polyphasic schedule was planned to last until a second 24 h monitoring after 8 weeks. In case of premature termination, a custom-made questionnaire asked for different reasons for terminating the polyphasic sleep schedule.

Participants of the external group were sampled from independent attempts to switch on a polyphasic sleep schedule, and thus did not share any systematic testing or procedures beyond the lucid dreaming interviews.

All participants were interviewed after termination of the polyphasic sleep rhythm, and were interviewed for their dream experiences during the period of polyphasic sleep and during the subsequent period with a regular monophasic sleep rhythm.

Both frequency of dreaming and frequency of lucid dreaming was assessed with a 6-point-rating scale for the time period with a polyphasic sleep rhythm (0: never, 1: monthly, 2: weekly, 3: after one nap/24 h, 4: after several naps/24 h, 5: after each nap/24 h) and with an 5-point-rating scale during for the time period with a regular monophasic sleep rhythm (0: never, 1: monthly, 2: weekly, 3: daily, 4: several dreams a day). The same definition of lucid dreaming as in study 1 was given. To obtain units for dream recall and lucid dream frequency per day, similarly to study 1 (and Schredl and Erlacher, 2004) the scale was re-coded as follows: 0 → 0, 1 → 0.03, 2 → 0.25, 3 → 1.0, 4 → 3.0, 5 → 6.0 for polyphasic sleep; and 0 → 0, 1 → 0.03, 2 → 0.25, 3 → 1.0, 4 → 3.0 for monophasic sleep.

In addition, participants were asked if their sleep quality (difficulties in falling asleep and difficulties to sleep through) was different during polyphasic compared to monophasic sleep (possible answers 1. much better, 2, better, 3. comparable, 4. worse, 5. much worse). The study was approved by the ethics committee of the Medical Faculty of the Ludwig Maximilian University, Munich.

3.1.3. Statistical analysis

Two participants of the external group had to be excluded due to inconsistent answers (more lucid dreams than dreams overall). We compared the self-reported lucid dreaming frequencies ofthe remaining 20 participants during polyphasic vs. monophasic sleep using a Wilcoxon signed-rank test. As a control, we performed another Wilcoxon signed-rank test for relative lucid dream frequencies, i.e. lucid dream frequency divided by general dream frequency. As another control, we correlated the individual change in lucid dreaming frequency with the reported change in sleep quality.

3.2. Results

Participants of the in-house group kept the polyphasic sleep rhythm for a duration of 3–44 days (mean 16.7 ± 12.9) before switching back to a normal sleep rhythm. Participants of the external group spent 3–220 days (mean 41.9 ± 68.3) on a polyphasic sleep rhythm before switching back to a normal sleep rhythm.

Participants reported 2.64 ± 1.90 dreams per day during and 0.81 ± 1.01 dreams per day after cessation of the polypohasic sleep rhythm. They further reported 0.50 ± 0.92 lucid dreams per day during and 0.05 ± 0.09 lucid dreams per day after cessation of the polypohasic sleep rhythm (see Fig. 2).

Paired t-tests indicated that these differences in both absolute (t_{19} = 2.31p = 0.016) and relative (t_{19} = 2.3, p = 0.016) lucid dreaming frequency between polyphasic and monophasic sleep rhythms were significant. In contrast, a Pearson correlation did not

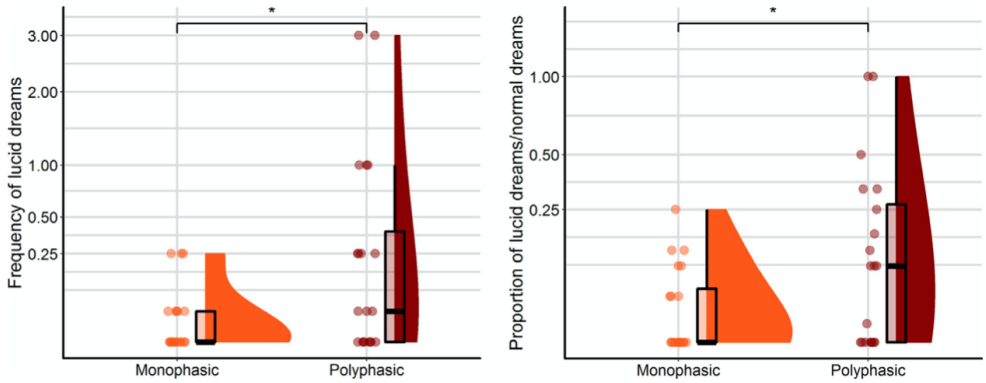

Fig. 2. Self-assessed lucid dreaming frequency is increased during a polyphasic sleep schedule compared to monophasic sleep. Left: absolute number of lucid dreams per day. Right: number of lucid dreams per day relative to general dream recall frequency.

find a significant association between the change in lucid dreaming frequency and the change in sleep quality during vs. after polyphasic sleep ($r = -0.14$, $p = 0.58$). This did not change when the change in lucid dreaming frequency was normalized by general dream frequency ($r = 0.06$, $p = 0.82$).

3.3. Interim discussion

Compared to a single period of extended sleep as experienced in regular monophasic sleep schedules, participants on radically polyphasic sleep schedules with alternating periods of sleep and wakefulness across the 24-hour cycle appear to experience an increased number of lucid dreams. This difference seems to be robust, as it can be demonstrated for both absolute numbers of lucid dreams experienced per day and the proportion of lucid dreams in relation to general dream recall frequency. Compared to rather brief periods of wakefulness that might characterize fragmented sleep during normal sleep patterns, radically polyphasic sleep allows for extended periods of wakefulness preceding an increased number of sleep episodes, and likely for REM periods following wakefulness more closely than in extended monophasic sleep episodes. Both factors might increase the probability of a still activated prefrontal cortex meeting a REM episode, and thereby increasing the chance of experiencing a lucid dream.

4. Study 3: Sleep quality

4.1. Methods

4.1.1. Participants

42 healthy volunteers (29 females, 13 males; age range 18–34, mean 22.8 ± 4.1) were included. The participants were recruited via the participant recruitment system of the University of Amsterdam and at the Radboud University Nijmegen as part of an independent larger project (not further discussed here). Only volunteers who remembered their dreams at least three times a week and who had personal experience with LD were included. Exclusion criteria were: current or history of sleep problems, psychiatric or neurological disorders such as anxiety or depression, night shift work, excessive use of alcohol, cigarettes or other recreational or psychoactive drugs. One participant reported experiencing sleep paralysis during multiple nights and was excluded from the dataset. All participants gave written consent after the procedures had been fully explained and were paid for their participation. The research was approved by the ethics committee of the University of Amsterdam and the CMO Regio Arnhem-Nijmegen.

4.1.2. Materials and procedures

Pittsburgh Sleep Quality index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The 19 questions of the PSQI are related to the sleep habits of the participant during the past month (e.g. When have you usually gone to bed at night? How often during the past month have you had trouble sleeping?). These 19 questions are combined to form seven different component scores: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication and daytime dysfunction. Each component score has a range of 0–3 points, in which a score of '0' indicates no difficulty and a score of '3' indicates severe difficulty. A global score is obtained by adding all seven component scores, leading to a range of 0–21 points, with higher global PSQI scores indicating a worse sleep quality.

Dream Lucidity Questionnaire (DLQ, Stumbrys et al., 2013). The DLQ measures different aspects of lucidity within dreams. It consists of twelve items assessing awareness as the core aspect of lucid dreaming (awareness that dream characters/objects are not real, awareness of dreaming, awareness that the physical body is asleep), and further different types of control (changing dream scenes/characters/events, breaking the physical laws, deliberately choosing an action), and remembrance (of intentions and of waking life). Each item has to be scored on a 5-point scale (0—not at all, 1—just a little, 2—moderately, 3—pretty much, 4—very

much). The total DLQ score was derived as mean of all original items except for item 7 and 12. This was because these questions loaded poorly (< 0.4) in the original factor analysis that produced the DLQ; relating predominantly to recall of "waking facts, episodes or intentions" and not lucidity per se (Stumbrys et al, 2013). As such, all DLQ scores were out of a maximum of 40 points.

Volunteers who were interested in participating in the study were invited to fill out a preliminary survey during the first week of the study. This survey consisted of the demographics questionnaire and the dream frequency/general attitude towards dreams/level of control questionnaire. Based on these questionnaires volunteers were included or excluded in the study. Included participants completed the PSQI once at the beginning of the study, and daily questionnaires on dream lucidity (DLQ), sleep and mood every day for six weeks.

All participants gave written consent after the procedures had been fully explained and were paid for their participation. During the experiment, participants were asked not to read any additional information about lucid dreaming outside what this study provided to them, since this could potentially bias the results. The research was approved by the ethics committee of the University of Amsterdam.

4.1.3. Statistical analysis

The data reported here focuses on the DLQ and PSQI questionnaires. For this study, we focused in particular on question 5b: During the past month, how often have you had trouble sleeping because you wake up in the middle of the night or early morning (possible answers: 0: Not during the past month, 1: Less than once a week, 2: Once or twice a week, 3: Three or more times a week). Pearson correlations were computed to provide insight into the relation between a longitudinal measure of lucid dreaming (mean of the 42 DLQ questionnaires) and PSQI question 5b and the global PSQI score, respectively.

4.2. Results

The mean global PSQI score before the 6-weeks period was 4.48 ± 1.72, indicating some variability in the sample, but was mostly still in the healthy (i.e. not clinically sleep disturbed) range. The mean score for PSQI question 5b was 1.38 ± 0.90, again indicating variability in the sample but no strong complaints about nocturnal awakenings. The mean DLQ score across all subjects and nights was 5.46 ± 6.63. We found no significant correlation between nocturnal awakenings as measured by PSQI question 5b and lucid dreaming as measured by the DLQ score ($r = -0.02$, $p = 0.88$) during the 6-weeks period. We further found no significant correlation between global PSQI scores and the average DLQ score ($r = 0.1$, $p = 0.53$).

4.3. Interim discussion

No correlations were found between nocturnal awakenings or overall sleep quality and dream lucidity. Since the PSQI is designed as a diagnostic tool for clinical purposes, it can therefore be concluded that if a connection between sleep disturbance and lucidity does exist, this particular tool is not apt to measure it; or that such disturbance is reasonably unlikely to be deleterious as per clinical definitions.

5. Study 4: EEG arousals

5.1. Methods

5.1.1. Participants

30 participants (7 males, 23 females; age range 18–27; mean 21.1 ± 2.13) were recruited from the student population of Radboud University in Nijmegen, Netherlands. Inclusion criteria included 'dream recall frequency' of ≥ 3 dreams per week, consistent sleep schedule, and infrequent alcohol intake (*Socially on Weekends* included, *One drink with a meal* excluded). Exclusion criteria were the presence of health or sleep related issues, prescription of psychopharmacological medication, drug use exceeding recreational and legal standards, and ongoing shift work.

5.1.2. Materials and procedures

Lucidity and Consciousness in Dreams Scale (LuCiD; Voss, Schermelleh-Engel, Windt, Frenzel & Hobson, 2013). The LuCiD measures key aspects of dream lucidity in detail and consists of 28 statements (e.g. While dreaming, I was aware of the fact that the things I was experiencing in the dream were not real; While dreaming, I thought about my own actions). Participants had to rate to what extent they agreed or disagreed with the statement on a 5-point scale from strongly disagree (0 points) to strongly agree (5 points), with unlabeled numeric values of 1–4 between these points.

For each participant, a night of sleep was recorded via ambulant 16 channel polysomnography (Somnoscreen plus, Somnomedics) using the 10/20 system, with a ground location on the forehead and the reference location on Cz, 2 EOG channels on the outer canthi, 2 EMG channels on the chin area referenced to a third channel, 2 ECG channels for electrocardiograph activity attached to the right collarbone area and under the contralateral ribs. Polysomnography was applied in the evening at Donders Institute, participants then went home and brought the Somnoscreen back the following morning. The raw EEG data was pre-processed through DOMINO (https://www.dominodatalab.com), with a software-based hypnogram produced. Pre-processed .EDF files were subsequently imported into SpiSOP (https://www.spisop.org) for manual sleep scoring and artefact rejection. Sleep scoring was performed for subsequent 30 s epochs according to standard scoring system (Iber, Ancoli-Israel, Chesson, & Quan, 2017) to obtain sleep

macrostructural variables. Two independent scorers performed a first scoring and a third scorer was used in cases of disagreement on sleep scoring. Arousals were analysed automatically by DOMINO and then manually quantified. Transitions (awakenings) between sleep and wake were counted manually.

Based on the hypothesis that dream lucidity achieved by the wake-back-to-bed strategy (LaBerge et al., 1994; Appel et al., 2020; Erlacher & Stumbrys, 2020) could be explained by waking levels of prefrontal activity persisting into subsequent REM sleep (Dresler et al., 2012), we analysed the number of wake-REM sequences (WREM); defined as the number of times a participant successfully entered or re-entered into REM from an aroused state within 5 min. Specifically, an aroused state was defined by either a continuous wake period that spanned at least two epochs, or a single wake epoch or arousal that resulted in a transition through sleep stages 1 and 2 before returning to REM. When these conditions were met, a 'Wake REM sequence' was recorded. Where discrepancy between two independent raters' scoring of ambiguous arousal/wakefulness events resulted in different numbers of WREM sequences being counted for a single sleep recording, the average of these two was taken.

In addition, we assessed the Number of Awakenings (NOA) and Total Wake Time (TWT), independent of REM sleep transitions. All three measures were assessed manually in addition to automatic analysis using an R script (https://www.r-project.org). We initially assessed only the two last hours before final awakening, given that REM sleep incidence is highest in the morning hours and the assumption that dream recall is most robust for dreams close to awakening and reporting, and the preponderance towards lucid dreams occurring during the hours that precede awakening. In an exploratory analysis, we varied this time window and the timing of the wake-REM transitions.

In the morning after polysomnography, subjects had to fill out the LuCiD immediately after awakening. The research was approved by the responsible ethics committee CMO Regio Arnhem-Nijmegen.

5.1.3. Statistical analysis

Pearson's correlation coefficients were computed to analyze the relation between the main lucidity factor *Insight* and the three sleep parameters. Additional exploratory correlational analyses were performed for the remaining lucidity factors, and for different timings of the sleep parameters.

5.2. Results

Participants experienced on average 1.17 ± 1.07 (range: 0–5) wake-REM sequences in the last two hours of the night. A significant correlation was found between the number of wake-REM sequences and the lucidity factor *Insight* ($r = 0.357 p = 0.026$) as seen in Fig. 3. There was no correlation found between *Insight* and *Number of Awakenings* ($r = 0.011$, $p = 0.478$) or *Total Awake Time* ($r = -0.081$, $p = 0.336$).

Under exploratory analysis, wake-REM sequences did not significantly correlate with any other LuCiD scales (*Realism*: $r = -0.078$, $p = 0.34$; *Control*: $r = 0.258$, $p = 0.084$; *Memory*: $r = -0.077$, $p = 0.343$; *Thought*: $r = -0.219$, $p = 0.123$) however *Control* did come close to significance threshold. LuCiD scales did not correlate with either *Number of Awakenings* or *Total Awake Time*, with the exception of the subscale *Realism*, which anti-correlated *Number of Awakenings* ($r = -0.341$, $p = 0.033$).

For further exploratory analysis, we varied the temporal parameters (last 2 h of sleep, 5 min wake-REM sequences) to determine whether these were adequate, and justifiable for re-employment in future analyses. Since both of these variables were chosen somewhat arbitrarily, it was pertinent to investigate whether lucidity from previous REM epochs (beyond two hours) could have been

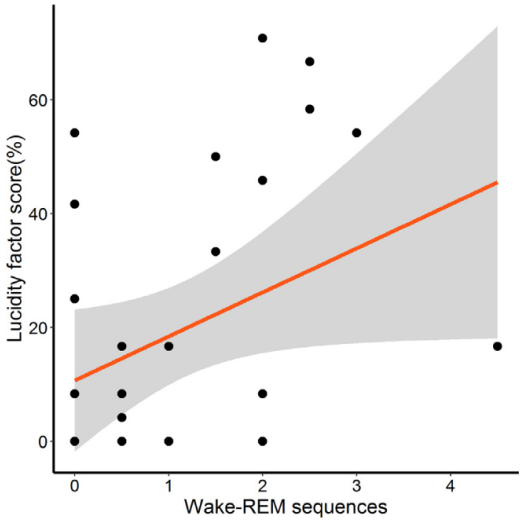

Fig. 3. Regression analysis of Lucidity factor *Insight* and wake-REM sequences ($r = 0.357$ $p = 0.026$, $n = 30$).

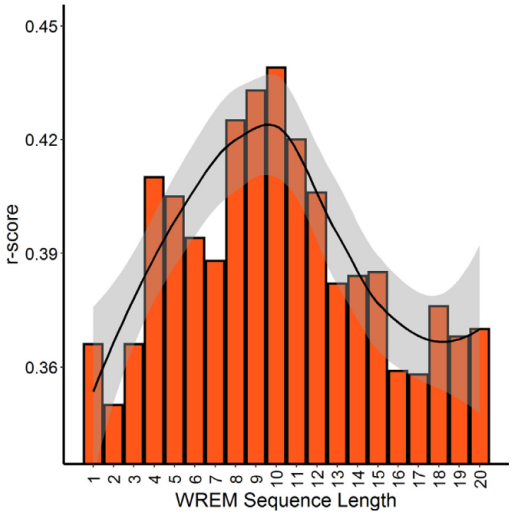

Fig. 4. R values of the lucidity factor *Insight* using varied wake-REM sequence lengths between 1 and 20 min, over the last 4 h of sleep (n = 30).

driving some of the questionnaire answers; and indeed whether the 5-minute wake-REM sequence was optimal for capturing the observable phenomena in question, at its maximal statistical power. The analysis of the association between wake-REM transitions and dream lucidity came out much stronger when applied to the last 4 h of sleep (including 3.57 ± 2.29 wake-REM sequences, range 0–8), and also appeared to maximise at this threshold. The chosen length of the wake-REM sequence did not change the effects substantially when varied between 1 and 20 min, but appeared to maximise between 5 and 10 min, diminishing above and below this window. The highest r value was at 10 min (r = 0.439) while the lowest was at 2 min (r = 0.35), as seen in Fig. 4.

5.3. Interim discussion

While no correlation was found between self-reported lucidity and either *Number of Awakenings* or *Total Wake Time*, the correlation between our novel variable w*ake-REM sequence* did indicate a potential connection between dream lucidity, sleep architecture and underlying physiological processes. Moreover, this result provided a clear and concise avenue by which to further investigate and quantify WBTB-based lucidity induction. While this variable provided preliminarily promising results, fully understanding its role within dream lucidity will require further analysis beyond that carried out though this investigation.

The somewhat arbitrarily chosen parameters of 5 min for time taken between arousal and REM re-entry appeared, under exploratory analysis, to be reasonably well chosen. However, in retrospect, analysis of the final 4 h of sleep was more desirable (particularly with wake-REM sequences set at 10 min) as the observed effects were considerably stronger at these points. Even though lucid dreams tend to occur more often in later REM periods (LaBerge et al., 1986), in principle they can be remembered from any nocturnal REM epoch. It thus seems plausible that taking a longer view into the hours preceding waking simply captures more overlap between the empirical and subjective data. An ideal timing of wake-REM to support lucid dreaming remains to be established though.

Results of the last study tentatively support a link between sleep fragmentation and dream lucidity, at least for the case of wake-REM sequences. Future analysis might explicitly investigate neurophysiological correlates of these sequences; attempting to expand this investigation to determine where and how given brain structures retain waking levels of activity as a deeper causal substrate for maintenance of metacognition during dream re-entry.

6. General discussion

The four studies presented here paint a rather nuanced picture of the hypothesized connection between sleep fragmentation and lucid dreaming: self-assessed numbers of awakenings, polyphasic sleep and physiologically validated wake-REM sleep transitions were associated with lucid dreaming; in contrast self-assessed sleep quality and physiologically validated numbers of awakenings were not.

At the neurophysiological level, it is tempting to speculate that sleep interruptions causally increase the likelihood of elevated activity in the prefrontal cortex persisting into REM sleep, and that such activity can explain the neurobiological mechanisms behind the results observed. Sleep onset REM episodes also occur in healthy subjects, particularly after periods of sleep interruption during the night (Fukuda et al., 1987; Miyasita et al., 1989; for a review see Takeuchi et al., 2002) and during daytime naps (Bishop et al., 1996; Singh et al., 2006). A common feature of sleep fragmentation seen in both pathological conditions such as narcolepsy and intentional sleep disruptions that promote lucid dreaming are thus REM periods rapidly following wake periods characterized by

more activated metacognition-related prefrontal brain regions. In such a physiological (or 'bottom up') model—activity in the prefrontal cortex would normally remain active during the return to REM, through a process of inertia (Hobson and McCarley, 1977) with higher numbers of wake-REM sequences comparatively maximising the likelihood of this happening. This would appear to be supported by research into narcolepsy, a pathological condition involving hypothalamic abnormalities in Orexin projecting neurons, which predicts dream lucidity significantly (Rak et al., 2015). However, the direction of causality becomes complicated with this explanation, and as such, this conclusion would be premature to draw. Narcolepsy is associated with richer than average dream content (Schredl, 1998; Fosse, 2000; Cipolli et al., 2008) indicating some degree of *heightened* cognitive function associated with its pathological presentation. Therefore, it can only be said that sleep disruptions may increase the chance of wake-REM sequences; with the predilection for vivid dream content (in these circumstances) requiring an alternate explanation. It would therefore be interesting to see whether lucidity correlates specifically with wake-REM sequences in sleep of Narcolepsy patients too.

A psychological (or 'top down') interpretation would also be consistent with the observations of this study. This would suggest that spontaneous metacognitive activity produces lucidity in reported cases, with such activity also coming at a cost—sleep is more likely to be disrupted, through 'failed' lucidity attempts that activate and arouse the entire brain as an unintended consequence. This would be difficult to refute directly, since other studies involving alarm snooze button use (Smith and Blagrove, 2015) and late night gaming (Gackenbach, 2009) as mechanisms to induce dream lucidity could not rule out lucidity as the primary goal of the behaviour; with voluntary 'disruption of sleep' serving as a crude means of achieving this outcome. To determine the value of this explanation, it would be interesting to investigate whether failed lucidity attempts (that instead result in full arousal) are particularly likely to result in a rapid return to REM. However, this could only be determined through empirical methods, which would in turn require a deeper understanding of the underlying neurophysiology and electrophysiology that underpin these phenomena, in order to capture these attempts outside of subjective reports.

These competing explanations could potentially be balanced by a third option; one that involves both psychological and physiological processes. Several waking studies have suggested that diminished cognitive performance through sleep fragmentation and deprivation can result in functional 'independence' of certain key hubs of the default mode network, including the precuneus (Chee and Chuah, 2007) which also share crucial importance with lucid dreaming (Dresler et al, 2012; Dresler et al, 2015), virtual representation (Cavanna and Trimble, 2006; Utevsky et al., 2014) and mind wandering (Mason et al, 2007; Christoff et al, 2009; Schooler et al, 2011). Together, this indicates that it is plausible for high level cognitive content to emerge once the physiological processes that constrain their manifestation become voluntarily or involuntarily disrupted. Such an interpretation coheres with theoretical models of brain function that view the mind in terms of primary and secondary processes (Dresler et al., 2009; Hobson, 2009; Hobson and Voss, 2010; Hobson and Voss, 2011), and indeed, many models of psychosis (Limosani et al., 2011; Dresler et al., 2015; Scarone et al., 2007) which also suggest that sleep-based stressors can cause certain types of brain function to experience heightened activation, through brain function as a global phenomenon becoming negatively impacted. As such, one cannot conclusively determine how the wake-REM sequence manifests or functions, at either the physiological or psychological level; but future investigations would be well placed to explicitly examine the neurophysiological correlates of such phenomena, and consider these theoretical discussion points, to help answer this question.

7. Concluding remarks

Our studies represent a diverse assemblage of the variety of ways that sleep fragmentation might contribute towards incidences of metacognition during sleep. The concluding picture that emerges from these studies is that *deleterious sleep* does not seem to have a substantive effect on dream lucidity. For subjective and objective sleep fragmentation the picture is mixed, with dream lucidity being associated with self-reported number of awakenings per night and polyphasic sleep, but not subjective degrees of fragmentation or dissatisfactory sleep; even though participants had the subjective impression that lucid dreaming occured most often in sleep that was quite fragmented. What does appear plausible is that rapid re-entry into REM sleep from a waking or aroused state does increase the chance of experiencing a lucid dream—and that fragmented or polyphasic sleep might indeed potentiate such occurrences. Our preliminarily hypothesis that rapid re-entry into REM sleep from a waking state supports increased incidences of dream lucidity can therefore be supported, laying some deeper context to the neurophysiological processes potentially responsible for the efficacy of the wake-back-to-bed strategy of lucid dream induction. Our findings thus indicate that the presence of a relatively specific sleep architectural phenomenon might be the causal instigator of dream lucidity, which may manifest as a consequence poor sleep, but does not require it. Given recent concerns about potential detrimental effects of lucid dreaming on sleep quality and/or sleep function (Vallat & Ruby, 2019; Soffer-Dudek, 2020), this distinction is an important one—lending further support to the findings of a recent study (Schadow et al., 2018) which showed no correlation between poor sleep and lucidity once nightmare content was controlled for.

Future investigations into lucid dreaming would do well to consider the wake-REM sequences and their role in potentiating the neurophysiological mechanisms that might underlie dream metacognition; and should consider this as a distinct variable from fragmentation per se. The efficacy of the wake-back-to-bed technique, together with unusual sleep scheduling (including morning REM naps) might be reconciled within the context of sleep fragmentation; with a meaningful distinction now available between deleterious and potentially more benign categories of sleep fragmentation.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.concog.2020.102988.

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