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Reduced Gamma Range Activity at REM Sleep Onset and Termination in Fear-Conditioned Wistar-Kyoto Rats

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Abstract

Recent investigations of rapid eye movement sleep (REMS) continuity have emphasized the importance of transitions both into and out of REMS. We have previously reported that, compared to Wistar rats (WIS), Wistar-Kyoto rats (WKY) responded to fear conditioning (FC) with more fragmented REMS. Gamma oscillations in the electroencephalogram (EEG) are synchronized throughout the brain in periods of focused attention, and such synchronization of cell assemblies in the brain may represent a temporal binding mechanism. Therefore, we examined the effects of FC on EEG gamma range activity (30–50 Hz) at REMS transitions in WKY compared to WIS. Relative power in the gamma range (measured as a percent of total power) at baseline and upon re-exposure to the fear-inducing conditioning stimulus was measured 35 s before REMS onset to 105 s after REMS onset (ARO) and 85 s before REMS termination (BRT) to 35 s after REMS termination. After baseline recording, rats received ten tones, each co-terminating with an electric foot shock. On Days 1 and 14 post-conditioning, rats were re-exposed to three tones. Fast-Fourier transforms created power spectral data in the gamma frequency domain. Relative power was extracted from an average of 4–5 REMS transitions. Relative gamma power was always higher in WIS. On Day 14, at 15 s and 25 s ARO, WKY had significant increases in relative gamma power from Baseline. WIS had a significant increase on Day 1 at 25 s ARO. Despite the increases in relative gamma power, WKY never achieved levels attained by WIS. Moreover, at 5 s BRT, only WKY had a significant decrease in relative gamma power from Baseline to Day 14. Gamma range activity may indicate neural activity underlying maintenance of REMS continuity. Low relative gamma power at REMS transitions may be associated with increased REMS fragmentation in WKY after FC.

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Keywords

Gamma Power; Rapid Eye Movement Sleep; Fear Conditioning; Posttraumatic Stress Disorder

INTRODUCTION

Stress has significant effects on sleep in humans as well as rodents [28]. Rapid eye movement sleep (REMS), in particular, changes dramatically after fear conditioning (FC) [28]. The effects of FC on REMS have generally been assessed using standard measures of REMS macroarchitecture, i.e., number and average duration of REMS episodes and total amount of REMS time. However, the results of several studies demonstrate the importance of investigating REMS microarchitecture as well, by partitioning total REMS time into sequential REMS (seq-REMS, inter-REMS episode interval ≤ 3 min) and single REMS (si-REMS, inter-REMS episode interval > 3 min) [2,35]. Episodes of seq-REMS, which are on average of shorter duration than those of si-REMS, occur in clusters. Studies have demonstrated that various stressors affect seq-REMS specifically [2,35]. For example, Amici et al. [2] found an increase in the number of seq-REMS episodes in the recovery sleep of rats after exposure to cold stress. Studying REMS microarchitectural changes following FC in rats can yield important new insights into the mechanisms of stress-induced sleep disturbances [7].

The REMS response to stress differs among rat strains. Wistar-Kyoto rats (WKY), known according to a range of criteria to be stress-sensitive [22–27,31], respond to FC with fragmentation of REMS, defined as a shift in the distribution of seq-REMS and si-REMS episodes towards seq-REMS [7]. This response is in contradistinction to the responses shown by Sprague-Dawley and Wistar rats (WIS) [7,17], in which a preponderance of longer duration si-REMS episodes following FC suggests that FC is less disruptive to REMS.

Mechanisms of REMS fragmentation, i.e., the neurophysiological substrates of a form of REMS that occurs in short duration episodes separated by short duration intervals, require explanation. Electroencephalographic activity in the gamma frequency range, which some suggest to be associated with focused attention during waking (W) [4,6,15,18], also is prominent during REMS [4,18], a state of hyperalerting to stimuli of endogenous origin [21]. In an initial attempt to explore the relationship of gamma power to REMS [14], we demonstrated that relative gamma power (measured as percent of total power) during REMS was lower in WKY compared to WIS, both before and after a FC procedure. However, there was no effect of FC on gamma power in REMS, an effect that would have been expected if gamma power signals brain mechanisms serving to maintain continuous, focused REMS, and the specific neural interactions that characterize the beginning and ending of a REMS episode [10]. Bassi et al. [3] have emphasized the importance of looking at the transitions into and out of REMS, suggesting that at these time points REMS is most likely to fail initially to consolidate properly or to terminate early, respectively. Therefore, we examined gamma power changes at the transitions into and out of REMS.

Animals from our previous study [7] provided data with which to explore the effects of FC on gamma range activity during REMS transitions in WKY compared to WIS. WKY and WIS underwent a cued FC procedure in that study. They were presented with ten tones (800 Hz, 90dB, 5 s duration), each co-terminating with a mild electric foot shock (1.0 mA, 0.5 s duration) at 30 s intervals. The effects of FC on sleep were examined by analyzing electroencephalographic and electromyographic traces obtained in the light phase (11 AM to 3 PM) after animals were re-exposed to three tones, without shock, one day, and again 14

days, after FC. Only WKY demonstrated an increase in REMS fragmentation, defined as a shift in the distribution of seq-REMS and si-REMS toward seq-REMS [7]. Also, WKY continued to freeze to tone presentations until the end of the test period, 14 days, whereas WIS did not. To determine that the alterations in REMS microarchitecture were specific to FC and not due to a lasting effect of shock stress (SS) alone, the effects of foot shock alone were studied in an additional group of animals. DaSilva et al. [7] reported that SS had no effect on REMS microarchitecture in either strain, suggesting that the changes in REMS microarchitecture were due to the FC procedure and not a residual effect of SS.

To understand better the mechanisms of REMS fragmentation in the WKY strain, we examined, in the present study, changes in relative gamma power in the electroencephalogram (EEG) during those times when REMS was beginning (35 s before to 105 s after REMS onset (ARO)) and terminating (85 s before REMS termination (BRT) to 35 s later). We hypothesized that relative gamma power at REMS onset and termination would be differentially altered in WKY compared to WIS following FC, with decreases in relative gamma power both ARO and BRT in WKY only. This would suggest different alterations of neural activity in REMS after FC in WKY compared to WIS. Changes in relative gamma power could be a useful measure of changes in the neural activity underlying REMS.

METHODS

Subjects

The animals used in the report by DaSilva et al. [7] provided data from which to explore further the effect of FC on relative gamma range activity during REMS in WKY and WIS. Male WKY (300–350 g, 8 weeks of age; $n = 6$ for FC, $n = 4$ for SS) and male WIS (300–350 g, 8 weeks of age; $n = 4$ for FC, $n = 4$ for SS) were obtained from Charles River Laboratories. All experiments were conducted in accordance with a protocol approved by the Institutional Animal Care and Use Committee (IACUC) of the University of Pennsylvania. Sleep recordings were analyzed in the light phase (11 AM–3 PM) on the Baseline day, and one day (Day 1) and 14 days (Day 14) after FC. Detailed descriptions of the housing, surgical and fear conditioning procedures are provided in DaSilva et al. [7] and above.

Sleep Parameters and Power Spectral Analysis

Computerized electroencephalographic and electromyographic traces were visually displayed, divided into 10 s epochs and manually scored using commercial software (Somnologica), employing standard criteria for rats: Waking (W)—low voltage, high frequency EEG, with increased motor activity; Slow wave sleep (SWS)—high voltage, low frequency EEG and decreased motor activity; REMS—low voltage, high frequency EEG, with a prominent theta peak, and nuchal muscle atonia. Theta range activity, which clearly defines REMS in the rat, was useful for accurately defining REMS transitions, using Pearson's correlations to ensure that theta range activity changes corresponded to gamma range activity changes. All transitions showed strong positive correlations between theta and gamma power ($p < 0.001$). Determination of transitions was aided by using EMG muscle atonia in addition to EEG changes. All scoring was blinded, achieved by having a non-scoring individual randomly assign code names to all data files.

During the period analyzed, the EEG was subjected to Fast-Fourier Transform analysis in successive 10 s intervals, creating power spectral data in the frequency domain. Times reported are the relative power values at stated time points. Relative power (measured as a percent of the total power from 0.2–50 Hz) was obtained to account for amplification

differences and allow for comparisons. Gamma power (30–50 Hz) and theta power (5–9 Hz) were extracted from an average of 4–5 transitions for each animal and averaged, on each day, in 10 s intervals, from 35 s before REMS onset to 105 s after REMS onset (ARO) and 85 s before REMS termination (BRT) to 35 s after REMS termination. REMS onset transitions and REMS termination transitions were extracted separately: 35 s of uninterrupted SWS had to precede the REMS episode and 35 s of uninterrupted W had to follow it in order for the episode to be included in the analyses. Power was taken from both si-REMS and seq-REMS episodes, without discrimination of type, in the last two hours of the sleep recording, where there was less artifact and transitions were more clearly definable. All REMS episodes fitting the above constraints were analyzed.

Data Analysis

Data were analyzed for statistical significance using SigmaStat (Systat Software, Inc.). Baseline, Day 1, and Day 14 data for each strain were compared using a repeated measures with between subjects factor analysis of variance (ANOVA) model (between subjects factor: Strain; within subjects factor: Day). As noted above, correlations between theta and gamma range activity at REMS transitions were assessed with Pearson's correlation test. Comparisons were considered significant at $P < 0.05$. Effect size was evaluated using Cohen's d test.

RESULTS

Shock Stress

There were no significant differences in relative gamma power ARO or BRT, either between strains or between conditions.

Fear Conditioning

REMS Onset—Relative gamma power was higher in WIS on all days and at all time points analyzed. At 15 s ARO, WKY had a significant increase in relative gamma power from Baseline to Day 14 (0.08 ± 0.01 vs. 0.11 ± 0.01 [$p=0.008$]) (Figure 1) and no significant change in relative gamma power from Baseline to Day 1. WIS had no increase in relative gamma power at 15 s ARO on either day. At 25 s ARO, WKY had a significant increase in relative gamma power from Baseline on Day 14 only [0.09 ± 0.01 vs. 0.11 ± 0.01 ; $p=0.001$] (Figure 1). At 25 s ARO, WIS had a significant increase in relative gamma power from Baseline on Day 1 only [0.17 ± 0.02 vs. 0.22 ± 0.02 ; $p=0.01$] (Figure 2). There was a significant Day $\times$ Strain interaction for relative gamma power at 25 s ARO [$DF=2$, $F=8.288$, $p=0.003$].

REMS Termination—Relative gamma power was higher in WIS at all time points analyzed. At 5 s BRT, WKY had a significant decrease in relative gamma power from Baseline to Day 14 [0.11 ± 0.01 vs. 0.06 ± 0.01 ; $p=0.016$] (Figure 3). A significant decrease in relative gamma power was not observed in WIS at 5 s BRT or at any other time.

Effect Size—With Cohen's d test, all significant results showed a large effect size ($d > 2$).

DISCUSSION

DaSilva et al. [7] have shown an increase in REMS fragmentation (defined as a shift in the distribution of si-REMS and seq-REMS episodes towards seq-REMS) in WKY compared to WIS after FC. Increases in REMS fragmentation reduce average REMS episode duration, as seq-REMS episodes are, on average, shorter in duration. Gamma oscillations in the EEG are synchronized throughout the brain in periods of focused attention [11,12,18]. Such

synchronization of cell assemblies in the brain may represent a temporal binding mechanism, facilitating the association of various percepts to create conscious attention [6]. Gamma range activity is commonly associated with focused attention during W. However, REMS is also a state of hyper-attentiveness, albeit to stimuli of endogenous origin [21]. Thus gamma range activity could be associated with the maintenance of continuous REMS. Recognizing that changes in relative gamma power after FC may signal an alteration in the underlying mechanisms of REMS continuity, we investigated relative gamma power at REMS transitions into and out of REMS.

As in our previous examination of gamma range activity [14], WIS had higher relative gamma power ARO and BRT in every test condition. This finding could relate to the greater ability of WIS to maintain REMS continuity after FC. Neural activity associated with low relative gamma power does not likely cause REMS fragmentation, because, at Baseline, WKY had lower relative gamma power during REMS than did WIS and yet showed equivalent REMS continuity [7]. However, low relative gamma power ARO and BRT may be associated with a predisposition in WKY to increased REMS fragmentation after FC. In support of this hypothesis, Bassi et al. [3] proposed that the distribution of REMS episodes of various durations is affected by an unknown underlying process, and that the probability of a REMS episode terminating is highest at both the beginning of the episode, when the new state attempts to consolidate, and at the end of the episode, when other processes can undermine the permanence of the episode or there is increasing pressure to terminate REMS. FC likely impairs the specific neural interactions that characterize the beginning and ending of a REMS episode [10].

After FC, WKY had a small increase in relative gamma power ARO from Baseline to Day 14, but did not attain the level exhibited by WIS, which, with already higher relative gamma power at Baseline, had a small increase ARO on Day 1. After a fearful experience, the normal adaptive response may be to maintain and “strengthen” the REMS state. This strengthening may be manifested by the increased relative gamma power in WIS shortly after FC, on Day 1. The neural organization expressed by an increase in relative gamma power observed in WKY ARO on Day 14 may be insufficient and too delayed to prevent REMS fragmentation, and a “full” REMS state may not be attained. The findings BRT in the present study support this interpretation. While it is normal for relative gamma power to decrease upon exit from REMS into a quiet, unfocused W period, there was a sharper and earlier decrease in relative gamma power BRT in WKY on Day 14 than in WIS. Additionally, the lack of significant power changes among days in the SS rats suggests that the passage of time alone did not account for the changes in relative gamma power seen, and that with an intervention in which REMS microarchitecture was not altered, neither was the proportion of gamma power. Thus, gamma power may be a manifestation of Bassi et al.'s [3] unknown underlying processes that make a REMS state more or less likely to consolidate or fragment.

The results of this study may provide insight into the neural mechanisms of the REMS sleep abnormality that has been described in posttraumatic stress disorder (PTSD), an anxiety disorder that develops in a significant proportion of individuals who have experienced a traumatic event [1,28,29]. The majority of individuals diagnosed with PTSD exhibit disturbances of sleep that involve re-experiencing the traumatic event with repetitive, stereotypical nightmares and insomnia [29]. Interestingly, REMS discontinuity, both shortly after a trauma and in chronic PTSD, has emerged as a polysomnographic abnormality likely related to the subjective sleep disturbance [5,13,20]. Following a traumatic experience, the continuity of REMS episodes may be important in adaptive processing of fearful stimuli received during associative learning [8,19,33], and it has been hypothesized that REMS fragmentation in the early aftermath of a trauma may predict later development of PTSD

[20]. Thus, FC in WKY compared to other rat strains may be a good animal model for understanding PTSD in humans. Indeed, PTSD develops in only a subset of individuals exposed to a potentially traumatic event, and genetic factors are thought to be important in explaining such differences in susceptibility [9,16,30,32,34].

CONCLUSION

Relative gamma activity may indicate neural activity underlying maintenance of REMS continuity. Low relative gamma power at REMS transitions may be associated with increased REMS fragmentation in WKY after FC.

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Abbreviations

ARO	after REMS onset
BRT	before REMS termination
EEG	electroencephalogram
EMG	electromyogram
FC	fear conditioning
PTSD	posttraumatic stress disorder
REMS	rapid eye movement sleep
seq-REMS	sequential rapid eye movement sleep
si-REMS	single rapid eye movement sleep
SS	shock stress
SWS	slow wave sleep
W	waking
WIS	Wistar rats
WKY	Wistar-Kyoto rats

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Figure 1.

Comparison of relative gamma power changes at REMS onset from Baseline to Day 14 after FC in WKY. Gamma power is shown at 10 s intervals. Time zero ($t = 0$) is the point of REMS onset. Negative times indicate gamma power during SWS. Positive times indicate gamma power during REMS. All gamma power is expressed as relative power (as percent of total power—0.2–50 Hz). Data are expressed as mean $\pm$ S.E.M. Significant differences within subjects: * $p < 0.05$. WKY showed increases in relative gamma range activity at 15 s and 25 s ARO on Day 14 as compared to Baseline. WIS had higher relative gamma power than WKY at all time points ARO.

Figure 2.

Comparison of relative gamma power changes at REMS onset from Baseline to Day 1 after FC in WIS. Gamma power is shown at 10 s intervals. Time zero ($t = 0$) is the point of REMS onset. Negative times indicate gamma power during SWS. Positive times indicate gamma power during REMS. All gamma power is expressed as relative power (as percent of total power—0.2–50 Hz). Data are expressed as mean $\pm$ S.E.M. Significant differences within subjects: * $p < 0.05$. WIS showed an increase in relative gamma range activity at 25 s ARO on Day 1 as compared to Baseline. WIS had higher relative gamma power than WKY at all time points ARO.

Figure 3.

Comparison of relative gamma power changes at REMS termination from Baseline to Day 14 after FC in WKY. Gamma power is shown at 10 s intervals. Time zero ($t = 0$) is the point of REMS termination. Negative times indicate gamma power during REMS. Positive times indicate gamma power during W. All gamma power is expressed as relative power (as percent of total power—0.2–50 Hz). Data are expressed as mean $\pm$ S.E.M. Significant differences within subjects: * $p < 0.05$. WKY showed a decrease in relative gamma range activity at 5 s BRT on Day 14 compared to Baseline. WIS had higher relative gamma power than WKY at all time points BRT.