Phase-Amplitude Coupling of Theta and Gamma Rhythms During Rapid Eye Movement Sleep Impacting Memory Across the Lifespan

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ABSTRACT

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Phase-amplitude coupling (PAC) between brain oscillations is thought to be an underlying neural mechanism of memory consolidation. Oscillation coupling may become weaker with greater age, possibly explaining natural memory decline across the lifespan, as suggested by studies of PAC during non-rapid-eye-movement sleep. Theta-gamma PAC (TGC) during wake is correlated with stronger encoding and better recall. However, it is unclear how TGC during rapid-eye-movement (REM) sleep correlates with memory or changes with age. I aimed to find TGC during REM sleep (REM TGC) affecting sleep-dependent memory consolidation that changes with age-related memory decline. We recorded scalp electroencephalography of good sleeping younger and older adults. Oscillatory data was extracted from filtered electroencephalography signals. Before sleep, participants learned a declarative memory or non-memory control task, then retested the respective task after sleep to measure memory consolidation. Memory consolidation was better in younger, compared to older, adults. REM TGC strength, measured by a modulation index, was not different between age groups nor task nights. Faster gamma coupling in a frontal channel was positively correlated with and predicts improvements in memory consolidation in younger adults. Slower gamma coupling in a central channel was positively correlated with memory consolidation in older adults. Our results suggest REM TGC strength is stable across the lifespan. However, the strength of faster TGC in younger and of slower TGC in older adults may improve memory consolidation. These results uncover more about how REM sleep and REM TGC changes across the lifespan, in relation to memory.

Keywords: rapid eye movement sleep, theta-gamma phase-amplitude coupling, sleep-dependent memory consolidation, aging

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Contribution of Authors

Study designed created by Dr. Dang-Vu and Oren Weiner. SOG completed minority of the recruitment, data collection and sleep staging, with help from the Sleep, Neuroimaging, and Cognition Laboratory. SOG completed all analysis, reporting and writing.

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LIST OF ABBREVIATIONS

Abbreviation	Definition
AASM	American Academy of Sleep Medicine
AHI	Apnea-Hypopnea Index
ANOVA	Analysis of Variance
ANVOCA	Analysis of Covariance
aTGCf	Adapted Theta-Faster Gamma Coupling
aTGCs	Adapted Theta-Slower Gamma Coupling
BDNF	Brain-Derived Neurotropic Factor
CFC	Cross-Frequency Coupling
CP	Coupling Phase
DR	Delayed Recall
ECG	Electrocardiogram
EEG	Electroencephalogram
EMG	Electromyogram
ESS	Epworth Sleepiness Scale
GNG task	Go-No-Go Task
IR	Immediate Recall
MCI	Mild Cognitive Impairment
MI	Modulation Index
MMSE	Mini-Mental State Examination
MoCA	Montreal Cognitive Assessment
NREM Sleep	Non-rapid eye movement sleep
PAC	Phase-Amplitude Coupling
PGO waves	Pontine-Geniculo-Occipital
PLMI	Periodic Leg Movement Index
PSD	Power Spectral Density
PSG	Polysomnography
REM Sleep	Rapid eye movement sleep
rmANCOVA	Repeated Measures Analysis of Covariance
SD	Standard Deviation
SE	Sleep Efficiency
SFI	Sleep Fragmentation Index
SO	Slow Oscillations
SOL	Sleep Onset Latency
SSI	Sleep Switch Index
SWS	Slow-wave sleep
TGC	Theta-Gamma Coupling
TGCf	Fixed Theta-Faster Gamma Coupling
TGCs	Fixed Theta-Slower Gamma Coupling
TiB	Time in Bed
TST	Total Sleep Time
WASO	Wake After Sleep Onset
WPA Task	Word-Pair Association Task

INTRODUCTION

Sleep is virtually universal in the animal kingdom, and occurs every day without fail or great effort (Siegel, 2008). It affects everything we and our bodies do, such as regulating metabolism and hormones, promoting musculoskeletal growth, and supporting cognition (Chen et al., 2017; Diekelmann & Born, 2010; Luyster et al., 2012). An abundance of relatively new research shows that sleep is essential in the daily functioning of cognition, and this is especially the case for the maintenance of memory (Diekelmann & Born, 2010). Despite a growing pool of knowledge on the positive relationship between sleep and memory, there is a poorly understood age-related decline in both sleep and memory (Lavoie et al., 2018). The responsible underlying mechanisms of which are not well known, but can be investigated by monitoring brain activity. My project is on how brain waves during rapid eye movement (REM) sleep relate to memory, and how those brain waves change with age, because little is known about the specific impact of REM sleep on memory, but especially in aging. To investigate this, memory must be examined, and this starts with its acquisition during learning and encoding. The aim of the following review is to summarize what we know about the psychology and neuroscience of learning, memory, and the roles of sleep.

Encoding

Encoding is considered the neural representation of learning. It is a process whereby incoming sensory information to the brain, transduced to the form of electrical impulses, are collected together in a unique network of neurons to form a memory trace in the hippocampus (Takeuchi et al., 2014). The encoding of memory traces is uniquely shaped based on what we experience moment to moment (Poo et al., 2016). What we experience is influenced by attention to incoming sensory information. Information that is not attended to enough is either not made into a lasting memory trace or made into a weak one (Aly & Turk-Browne, 2016; Muzzio et al., 2009). Various brain regions contribute to the encoding. Declarative memory relies on the hippocampus, a short-term memory storage, where memory is more malleable and subject to change. When transferred to a long-term storage, thought to be the cortex, memory is less likely to be altered (Takeuchi et al., 2014). Memory would need to be maintained in long-term storage and especially from use in the short-term storage (Goshen et al., 2011).

Consolidation

Memory consolidation is the process where memory is transferred between regions of the brain, consequently protecting them from alterations (Diekelmann & Born, 2010). There are two overarching ways maintenance of memory is thought to happen at the neuronal level: active systems consolidation and synaptic homeostasis (Klinzing et al., 2019; Tononi & Cirelli, 2014). Through active systems consolidation, important connections between neurons are reactivated, strengthening, and improving memory (Klinzing et al., 2019). An example of this can be seen when an odor cue, associated to a memory task, is presented during subsequent slow-wave sleep (SWS). There was an odor-cued reactivation of brain areas related to the task, which was correlated with improvement on the memory task (Rasch et al., 2007). Active systems consolidation hypothesis states that reactivation or related memory representations occur in the hippocampus and neocortex, integrating newly encoded information from short-term to long-term memory without overriding existing memory (Rasch & Born, 2013). Dendrites of neurons are upscaled, meaning more synapses are made or enlarged, thought to protect memories prone

to be forgotten. In this way, there is an additive effect to the existing memory, according to the active systems consolidation hypothesis. A challenge to the active systems consolidation hypothesis is that the brain is in the skull, a finite space. Too many connections being enlarged to strengthen memory traces is not feasible. Therefore, a balance is required to maintain space at minimal expense to memory accuracy. The synaptic homeostasis hypothesis states that dendrites are downscaled, meaning there are fewer and smaller synapses. Unimportant connections made between neurons in a memory trace are either reduced or lost, to make memory more efficient and effective representations of important memory (Tononi & Cirelli, 2016). Both models working together promote accurate, efficient, and secured memory, which is observed in declarative, long-term memory tasks (Diekelmann & Born, 2010; Wang et al., 2017). However, in long-term storage, where memory is in the cortex, it may not be as easily accessible.

Retrieval

For memory to be brought into awareness for use, the memory trace must be reactivated and brought out of the long-term storage, and back to short-term storage, the hippocampus, through the process of retrieval (Tanaka et al., 2014; Wiltgen et al., 2010). Reactivation can be cued through presenting stimuli related to the memory or, for example, by being asked a question to recall a certain fact (Trelle et al., 2020). Memory is retrieved into conscious awareness so it can be recalled and manipulated. Retrieval is the utility and purpose of memory, but consequently, this makes the memories more susceptible to change (Dudai & Eisenberg, 2004; Hardt et al., 2013).

Introduction to Role of Sleep

Sleep plays a critical role in protecting memory from drastic changes, meaning sleep maintains it. In general, the less sleep one gets, the worse memory performance is. Different types of sleep are thought to influence different types of memory, such as rapid eye movement (REM) sleep supporting emotional memory and non-REM (NREM) sleep supporting semantic memory (Diekelmann & Born, 2010). The exact underlying mechanisms have been investigated more in NREM sleep, than REM, in the past two decades. Therefore, more is known about the role of NREM sleep in relation to memory (Diekelmann & Born, 2010). Although, some work has shown that REM sleep influences more types of memory than previously thought, including those NREM sleep was thought to be solely responsible for (Diekelmann & Born, 2010; Fogel et al., 2007; Wagner et al., 2001). The roles of REM and NREM sleep will be discussed further in the next sections.

Role of NREM Sleep

The impact of NREM sleep on healthy, but especially memory is relatively well established, as most sleep and memory research over the past decade has focused on this stage (Muehlroth et al., 2020; Rasch & Born, 2013). NREM sleep has three substages: N1, N2, and N3 (or SWS), which tend to occur cyclically and sequentially as we enter sleep (Berry et al., 2012). Out of all substages, N1 has not been as thoroughly investigated in general as other stages of sleep. It is widely considered as a transition state from wake to sleep, but there is some correlative evidence with N1 and improvements on a navigation task (Manoach & Stickgold, 2019; Wamsley et al., 2010). N2 is well documented to be related to memory consolidation, even during development from adolescences to teens (Hahn et al., 2019). SWS is associated with clearing cellular waste and toxins via the glymphatic system (Reddy & van der Werf, 2020). The

brain is operating during NREM sleep to ensure it removes waste, restores itself and the body, and improves memory.

Role of REM Sleep

The role of REM sleep in general, but especially memory is less clear than NREM sleep, as there is not enough evidence to definitively argue one role. Research has shown that REM sleep is associated with various functions, including homeostatically restoring aminergic reserves (Siegel & Rogawski, 1988), facilitating creativity and cortical plasticity (Cai et al., 2009; Sterpenich et al., 2014; Wagner et al., 2001), preparing the brain for wakefulness (Brooks & Peever, 2016), and especially supporting memory consolidation (Diekelmann & Born, 2010). Evidence for both synaptic homeostasis and active systems consolidation were seen in one study in mice. Where there was a REM sleep-dependent pruning of new pyramidal dendritic spines, as well as a strengthening and maintenance of new spines related to a novel motor task (Li et al., 2017). Pruning of dendritic spines supports synaptic downscaling, which is involved with the synaptic homeostasis hypothesis, and strengthening of new spines is related to active systems consolidation (Tononi & Cirelli, 2016; Rasch & Born, 2013). Further, for active systems consolidation, a PET study demonstrated that brain regions activated during a serial reaction time task during wakefulness were subsequently reactivated during REM sleep, providing evidence in support of active systems consolidation (Maquet et al., 2000). In theory, both models of consolidation influence every type of memory. Types of memory thought to be processed during REM sleep, include declarative and emotional memory (Fogel et al., 2007; Wagner et al., 2001). One study found an increase in theta power during REM sleep, after a word-pair association task was learned, and this related to an improvement on the memory task (Fogel et al., 2007). Other studies found greater REM sleep times to be correlated with the formation, improvement, and maintenance of emotional text material, compared to neutral text (Nishida et al., 2009; Wagner et al., 2001). Studies have shown several roles for REM sleep, most of which indicate a significant role with memory. NREM sleep has different and shared roles as REM sleep, such as impacting declarative and procedural memory (Fogel et al., 2007).

Oscillations in Encoding

Brain activity during sleep, and wake, generates different spectra of rhythms respective to the behavioral state, such as encoding. Theta (4-8Hz) and gamma (30Hz+) oscillations are observed during REM sleep in animals, and encoding in humans and animals. Results from several studies suggest that increases in theta and gamma power and decreases in alpha and beta power during encoding predicts successful memory recall in humans (Fell et al., 2001; Fellner et al., 2013; Hanslmayr et al., 2009; Klimesch et al., 1997; Sauseng et al., 2002; Sederberg et al., 2003). The four general phases of brain rhythms, either when they oscillate 1) up to a 2) peak or oscillate 3) down to a 4) trough, is thought to be a contributing factor to encoding. Theta and alpha (8-12Hz) phase variability in relation to a stimulus onset, termed phase-locking, is higher during encoding of a visual recognition task compared to a pre-stimulus baseline, and the same was observed with alpha and gamma (80-150Hz) phase-amplitude coupling (PAC), where there is an amplitude increase in gamma power during the trough phase of alpha waves (Klimesch et al., 2004; Voytek et al., 2010). Increasing frontal theta and parietal gamma power, and cortical and hippocampal theta-gamma coupling are associated with successful declarative memory encoding (Friese et al., 2013; Lega et al., 2016). Theta phase preference is unique to each task, so coupling resets and changes upon task switching, indicating theta-gamma coupling is task-

dependent (Canolty et al., 2006; Mormann et al., 2005). Theta and alpha phase and power, and gamma power all seem to reflect proper encoding. There is a relatively large pool of information on oscillatory brain activity related to encoding, in addition to oscillations during NREM sleep.

Oscillations during NREM Sleep

Delta and sigma occupy a majority of the oscillatory framework of NREM sleep. Oscillations known to be related to memory during NREM sleep primarily contains slow oscillations (SO) (0.5-2Hz) and sleep spindles. As NREM sleep progresses, neurons become increasingly synchronized from theta, alpha, sigma (sleep spindles) and K-complexes, to delta. N2, seen as a lighter stage of sleep, primarily contains sleep spindles (9-15Hz) and k-complexes which positively impact memory (Fogel et al., 2012; Rasch & Born, 2013). SOs alone during SWS are associated with clearing cellular waste and toxins via the glymphatic system, which relates to a general reduction in space to improve efficiency as a part of the synaptic homeostasis hypothesis (Reddy & van der Werf, 2020). An abundance of recent work supporting active systems consolidation, found the interaction between SOs and sleep spindles during N2 and SWS sleep greatly supports declarative memory formation (Maingret et al., 2016; Naji et al., 2019). Evidence on oscillatory processes of NREM sleep has accumulated more relative to REM sleep.

Oscillations during REM Sleep

Few papers specifically aimed to investigate oscillations during REM sleep, especially memory-related rhythms. It is known that REM sleep primarily consists of theta and gamma rhythms, but what they relate to is not known (Cantero et al., 2003; Montgomery et al., 2007; Rasch & Born, 2013). One study did find prominent beta and theta oscillations in anterior cingulate and dorsolateral prefrontal cortices during REM sleep in humans but could only inference the purpose to mediate memory consolidation (Vijayan et al., 2017). Theta power increase during REM sleep is, however, thought to be related to memory. This increase is seen in the cortex after a declarative memory task and is correlated with emotional recognition memory and hippocampal theta rhythms in humans (Cantero et al., 2003; Fogel et al., 2007; Nishida et al., 2009). After optogenetically inhibiting medial septum inputs to the hippocampus during REM sleep, a reduction in hippocampal theta power and spatial and contextual memory accuracy was found in rats (Boyce et al., 2016). This showed hippocampal theta was necessary for spatial and contextual memory consolidation (Boyce et al., 2016). Knowledge on the general framework of REM sleep oscillations show prominent theta and somewhat gamma waves, and some evidence on other rhythms. The interaction between oscillations is a complicated topic, which shows some implications for memory improvement and is studied in both sleep and wake.

Cross-Frequency Coupling

A well-studied concept on relations between oscillations is cross-frequency coupling (CFC). CFC is where two frequencies synchronously occur in the brain, for example SO-spindle coupling. This is thought to take place in many behavioral states, from sleep, to encoding and retrieval during wake (Köster et al., 2014; Lega et al., 2016; Rasch & Born, 2013). A study found temporal precision of sleep spindles coupled to the up-state of SOs improves declarative memory consolidation in younger and older adults (Muehlroth et al., 2019). In general, SO-spindle coupling during SWS has been sufficiently documented in memory research (Mölle et al., 2002; Staresina et al., 2015). Though not as well studied as SOs and spindles, theta and gamma rhythms are known to dominate REM sleep (Montgomery et al., 2007). As gamma

synchronization occurs, the theta wave may enter the trough, and the phase of both rhythms are coupled, called phase-phase coupling (Belluscio et al., 2012). This coupling was observed in pyramidal cells of the CA1 of the hippocampus of rats performing an elevated linear track but was seen more so during subsequent REM sleep (Belluscio et al., 2012). Similar results were found where the phase of a slower oscillation is synchronously coupled to the amplitude of a faster oscillation (PAC), in this case, theta-gamma coupling was observed in the neocortex and CA1 of the hippocampus in rats after a novel open field task (Scheffzük et al., 2011). The modulation index, a measure of PAC, was 9-fold greater during REM sleep than wakefulness, indicating a functional role of theta-gamma PAC during REM sleep in animals (Scheffzük et al., 2011; Tort et al., 2010). Currently, there is no evidence of theta-gamma PAC during REM in humans. Similar to SO-spindle coupling in NREM, based off of speculation, the functional role of PAC and memory in REM sleep may also be influenced by age. However, to the best of our knowledge, published evidence for theta-gamma PAC and aging is completely lacking, with the exception to one study on coupling phase during encoding (Karlsson et al., 2022).

Aging Effects on NREM Sleep

Despite a lack of evidence of age-related PAC changes during REM sleep, there is more evidence on NREM sleep throughout the lifespan. N1 and N2 occur more, and SWS occurs significantly less starting from middle age, in concurrence to receiving fewer hours of total sleep time per night (Muehlroth et al., 2020). Older adults often have a longer sleep onset latency (SOL) and have more fragmented sleep, accordingly they wake up more often during the night, and as a result, have a longer wake after sleep onset (WASO) and nap more frequently (Li et al., 2018). With age, internal clocks, or circadian rhythms, become more advanced, meaning time to sleep and wake up are earlier (Hood & Amir, 2017). Because of this and napping, older adult sleep is less efficient, as more time is spent awake in bed compared to younger adults (Landolt & Borbély, 2001; Scullin & Gao, 2018). Nap frequency increase may also occur from age-related deterioration of wake-promoting and -stabilizing neurons in the lateral hypothalamic area (HLA) (Mander, Winer & Walker, 2017). These HLA neurons have connections to noradrenergic cells of the locus coeruleus (LC). LC neurons impact sleep-wake transitions, so an age-related reduction in HLA neuronal inputs impairs the function of these LC cells to control sleep and wake (Downs et al., 2007). There is an association between age-related atrophy of medial prefrontal cortical (PFC) grey matter with reductions in NREM sleep slow-wave activity and SO-spindle coupling (Mander et al., 2013; Helfrich et al., 2018). Gray matter reduction in the lateral PFC and superior temporal cortices were associated with shorter sleep duration in older adults (Lim et al., 2016; Mander, Winer & Walker, 2017). SOL, sleep fragmentation, WASO, and fewer hours of total sleep time, likely resulting from age-related anatomical and physiological changes in the brain, contribute to worse sleep efficiency. This consequently affects NREM sleep oscillations.

Aging Effects on NREM Sleep Oscillations

Aging differentially affects NREM sleep oscillations. Lower SO density and amplitude are concurrently seen with the reduction of sleep efficiency, and in men, this is particularly observed in prefrontal/frontal regions (Landolt & Borbély, 2001). One study suggested more time is needed SOs to synchronize older adults, and this was mostly seen in the first half of the night, when homeostatic pressure is the highest, which the authors concluded was a weakened homeostatic pressure to sleep in older adults (Carrier et al., 2011). Likewise, sleep spindle

density, duration, and number, and K-complex duration and number were significantly lower in older compared to younger adults, and this large reduction was predictive of age (Crowley et al., 2002). As such, an age-related decline in sleep time is accompanied by less SO and spindle events during NREM sleep. As we get older, NREM sleep is negatively impacted, and this can be seen in studies investigating its oscillations and architecture.

Aging Effects on REM Sleep

Unlike NREM sleep, most studies investigating REM sleep architecture and age have concluded a small decline in REM sleep percentage and time. A small REM sleep percentage decline has been observed up until 60 years of age, when declines were no longer observed (Floyd et al., 2007; Ohayon et al., 2004). Whereas one study observed a continuing, but lesser decline after 60 years (Redline et al., 2004). Similar declines results were separately reported for total REM sleep time, as well as a percentage decline in older females, but not males (Dorffner et al., 2015; Moraes et al., 2014; Van Cauter et al., 2000). It has been noted that REM sleep cycles occur more in the first half of the night in older adults compared to young adults, suggesting a lack of temporal organization due to changes in the circadian rhythm (Sonni & Spencer, 2015). Overall, there not many known large changes in REM sleep architecture.

Aging Effects on REM Sleep Oscillations

An unclear impact of aging on REM sleep is likely due to a lack of investigations into age-related alterations in REM sleep oscillations. An early study found age-related power reduction in the 0.75Hz-10Hz range in REM sleep in older adults (Landolt & Borbély, 2001). These results were corroborated by others that found a decrease in delta power but increase in higher frequencies such as alpha and beta power during REM sleep (Bruce et al., 2009; Luca et al., 2015). However, other studies found no change in delta power in REM sleep, despite increases in theta, alpha, and beta power (Mann & Röschke, 2004). A more recent study provided correlative evidence in older adults, showing lesser theta power in older, compared to younger adults, but that the specific oscillatory theta is related to better cognition (Scarpelli et al., 2019). Given gamma's known interactions with theta and alpha during wake related to cognitive function, but a lack of evidence for its implications during REM sleep, a direction this research may approach are the effects of gamma in an aging population to help bridge a gap in our knowledge (Pace-Schott & Spencer, 2011).

Aging Effects on Coupling during Learning

A direction aging research is going to is changes in coupled oscillations during learning across the lifespan. One study comparing across ages found theta-gamma rhythms to be more uncoupled in older adults during, and related to, a change-detection memory task (Reinhart & Nguyen, 2019). In terms of coupling phase (CP), the temporal precision of gamma amplitude changes coupled with theta phase is less precise in older adults during learning, and this was reflected in poorer age-related memory performance (Karlsson et., 2022). A study investigating cognition-related oscillations and their PAC in older rats exploring a novel environment found a reduction of theta power but no relation with gamma power (Jacobson et al., 2013). The issue is that older rats showed an age-related baseline reduction to theta-gamma PAC unrelated to novel environment exploration, meaning their findings on age-related changes in oscillations could not be associated with cognition. That said, aging in animals, especially rodents, may not effectively represent that of humans. In older adults conducting a delayed match-to-sample (DMTS) task,

theta-gamma PAC in the parietal cortex was positively correlated with successful memory performance (Park et al., 2011). Similar results were observed in humans during a 2-Back task (Goodman et al., 2018). However, both studies did not compare coupling or memory performance to younger adults. Compared to healthy controls, people with mild cognitive impairment (MCI) showed significantly lower levels of theta-gamma PAC during a 2-Back task, and those with Alzheimer's disease showed even lower levels (Goodman et al., 2018). The DMTS, 2-Back, and change-detection tasks involve retaining information that will be used seconds later, reflecting the ability of working memory. An advantage to studying declarative memory may be assessing retention of information on a larger time scale, such as minutes to hours. Larger intervals between information acquisition to testing allows for the assessment of long-term memory. A delayed recall from a word-pair association task, for example, considers this and is a good future direction to study aging effects on learning, in relation to coupling.

Aging Effects on Coupling during Sleep

As for age-related changes in CFC during sleep, there are known age-related negative impacts on sleep spindles and SOs during NREM sleep. Sleep spindle amplitude increase is slower, occurring shortly before SOs peak in older, compared to younger adults. With increasing age, there is less precision in the SO-spindle coupling during N2/SWS (Muehlroth et al., 2019). In addition, there is a reduction in SO-spindle coupling amount in fronto-central regions (Helfrich et al., 2018). A conclusion may be that a reduction in SO-spindle coupling would result in memory impairments, as SO-spindle coupling is positively correlated with memory consolidation and improvement (Muehlroth et al., 2019. Like the few studies providing insight into age-related coupling changes during sleep, these only researched NREM sleep. Therefore, to the best of our knowledge, no studies investigate CFC during REM sleep with age, specifically theta-gamma PAC.

Objectives, Hypothesis and Significance

TGC has been observed in rodent REM sleep, and in human EEG during encoding, working memory, and retrieval tasks (Scheffzük et al., 2011; Montgomery et al., 2007; Belluscio et al., 2012; Bandarabadi et al., 2019; Scheffer-Teixeira et al., 2012; Del Vechio Koike et al., 2017; Friese et al., 2013; Lega et al., 2016; Karlsson et al., 2022; Canolty et al., 2006; Mormann et al., 2005; Goodman et al., 2018; Park et al., 2013; Köster et al., 2014). Only one study compared differences in TGC coupling phase (CP) during encoding between younger and older adults (Karlsson et al., 2022). To the best of our knowledge, a gap in our knowledge is a link between TGC during REM sleep (REM TGC) and long-term memory formation, and whether REM TGC declines with memory across the lifespan. The objectives of my project are to 1) establish the presence of TGC in EEG during REM sleep in humans, 2) to investigate whether TGC is related to overnight sleep-dependent memory consolidation, and 3) to examine age-related differences in TGC during REM sleep and whether those differences are associated with differences in memory consolidation between older and younger age-groups. I hypothesize 1) TGC in EEG during REM sleep in humans is correlated with and can predict improvements in sleep-dependent declarative memory consolidation, and 2) TGC strength would become worse as memory becomes worse with age. It is also hypothesized that memory consolidation will be greater in adults who slept overnight, compared to adults who were awake over an equivalent period of time during the day, to control for effects of the passage of time. Results from this study would further our understanding of how a poorly understood neural mechanism during

REM sleep changes across the lifespan and how those mechanisms may underlie sleep dependent memory consolidation processes and possibly lead to age-related declines in memory.

METHODS

Participant Recruitment and Screening

Younger (18-30yr) and older (55-85yr) healthy, good sleeping adults were recruited using flyers sent via email and posted around the PERFORM Centre, through the CRIUGM (Centre du Research du Institut Universitaire de Gériatrie de Montréal) participant pool database, and from direct email contact through the Sleep, Cognition, and Neuroimaging Laboratory website. Younger and older adults came from an independent sample from the general population who completed the complementary 3-night or 2-visit daytime protocol. Interested participants completed a brief phone interview. This was followed by a semi-structured interview to further assess sleep, mood, medical history, and daily habits, and to complete cognitive screening tests (Mini Mental State Examination and Montreal Cognitive Assessment). Participants were excluded if they meet any of the following a priori criteria: chronic medical or psychiatric conditions, sleep or neurological disorders, including periodic leg movements, PLMI >15, and sleep apnea as defined by AHI >5 in younger or >15 in older adults, currently working night shifts, using elicit substances, have traveled outside of the time zone for more than four weeks, using sedative/hypnotic or other psychotropic substance altering alertness or sleep, have inconsistent sleep/wake schedules, consume cannabis more than once per month, and have more than 10 alcoholic drinks per week. Additionally, participants filled the following self-report questionnaires, and were excluded if they exceeded a priori threshold scores listed beside each questionnaire if applicable: Insomnia Severity Index, 15 (Morin et al., 2011), Pittsburgh Sleep Quality Index, 5 (Buysse et al., 1989), Epworth Sleepiness Scale, 10 (Johns, 1991), STOP-Bang, 5 (Chung et al., 2012), Ullanlinna Narcolepsy Scale, 14 (Hublin et al., 1994), Morningness-Eveningness Questionnaire Self-Assessment Version (Horne & Ostberg, 1976), Edinburgh Handedness Inventory (Ransil & Schachter, 1994), and Beck Depression Inventory II 10 (Jackson-Koku, 2016), and Beck Anxiety Inventory, 10 (Creamer et al., 1995), for younger adults or Geriatric Depression Inventory, 15 (Sheik & Yesavage, 1986), and Geriatric Anxiety Inventory, 10 (Pachana et al., 2007), for older adults.

Electrophysiological Recordings

Eligible participants for the three-night study completed a PSG sleep recording in a private research bedroom in our laboratory. PSG is the first of the three overnight studies, and the final screen for sleep disorders, as it is the gold-standard for examining sleep. Between the three overnight sleep recordings participants wore an actigraphy watch and filled out a daily sleep diary to provide objective and subjective measures of sleep-wake rhythms and sleep quality, respectively. On the second and third nights, actigraphy and sleep diary data were reviewed to confirm a consistent sleep/wake schedule is maintained across the preceding weeks. Afterwards, participants completed an overnight EEG study, which included pre- and post-sleep computerized cognitive testing (word-pair association (WPA) task or letter-shape discrimination task). Participants were tested in either English or French, depending on their mother tongue. The WPA and letter-shape discrimination tasks were counterbalanced across subjects between the second and third overnights (Figure 1).

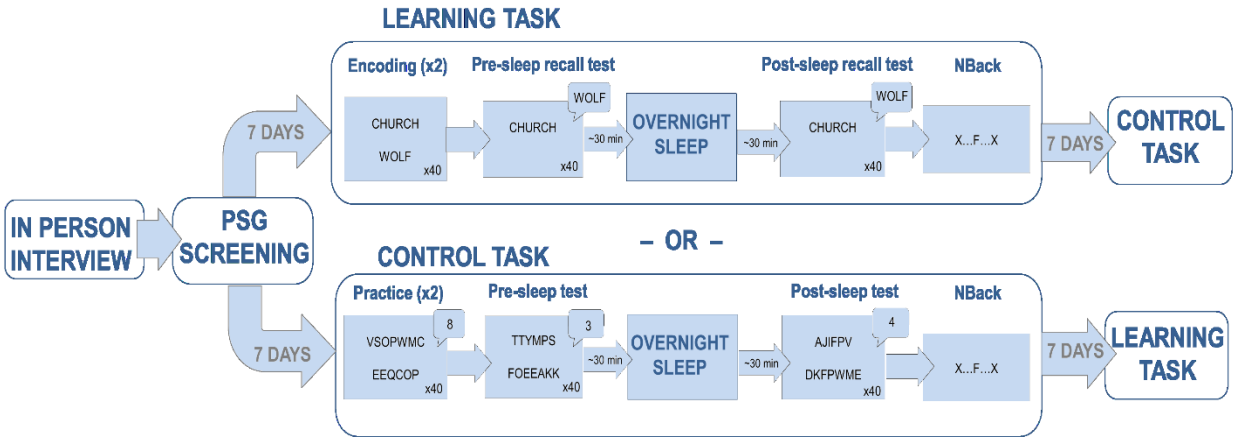

Figure 1. Overview of three-night study procedure. Series of events involved with the three-night sleep study. In order, participants undergo polysomnography (PSG), followed by at least seven days and two overnight electroencephalographrams with memory (LEARNING TASK), or non-memory control task (CONTROL TASK), each separated by seven days. Figure adapted from Mameri-Arab et al. (2019), Figure 2.

Computerized Cognitive Testing

Cognitive tasks given to participants before and after bed were either a declarative memory, WPA, or non-memory control, letter-shape discrimination, task with EEG. The WPA task is often used in memory research as a measure of declarative memory, especially in sleep research with REM and NREM sleep (Marshall et al., 2011; Fogel, Smith, & Cote, 2007). In children and young adults, a WPA task was found to be benefitted after nighttime sleep, compared to daytime wake (Wang et al., 2017). Before all tasks were performed, participants watched a resting baseline video, involving a dynamic nature scene with calming music for up to 10 minutes, to obtain at least 5 minutes of resting, wake brain activity with limited artifacts in the EEG. During the video, participants were instructed to minimize movement, especially of the head, stay awake, and not look at or touch their cellphone. For the WPA task, participants were shown a blank screen for 2-5 seconds, followed a 1 second fixation cross in the middle of the screen, then a pair of words. There was one block of forty pairs of primarily unrelated nouns and participants were tasked to visualize a scene combining the two items. They were shown the block twice, with two-minute breaks after each block, before being tested on the recall portion of the task. The word-pair list was shuffled between the first and second presentation, to minimize learning-order effects. For the test, the first word from each pair was cued, and participants were tasked to recall the paired word from each forty-word pairs aloud to a researcher in the room, who scored the response. The WPA task recall test was repeated the following morning after participants have been awake for at least thirty minutes. Performance on the WPA task is expressed as the absolute correct pre- and post-sleep responses. A threshold of ten correct responses was placed to exclude participants with poor declarative memory. The measure of memory stability is the ratio of post- and pre-sleep responses (AM/PM), and memory consolidation is measured as the total amount of correct responses for each individual pair recalled post- and pre-sleep.

The letter-shape discrimination task was done on nights the WPA task was not. This non-memory control task followed the same order of events as the WPA task, including the 2-5 second of blank screen followed by a 1 second fixation cross. Participants were shown pairs of

non-sense words and tasked to count the number of letters that have curves in their shape (e.g., C, O, and D, but not K, X, or Z), and verbally reported this number to a researcher in the room. 120 pairs of non-sense words equally divided in to three blocks of forty pairs were shown to participants, with two-minute breaks in between each block. In the evening, the first and second blocks were for participants to practice the task. A researcher was in the room for the third and final block to record the participants count for each pair; this was repeated in the morning after the participant is awake. This task does not require memory or previous experience to complete or improve on, which considers possible non-memory related mental effort on brain activity. All participants were connected to EEG equipment while watching a resting baseline video and performing all computerized cognitive testing.

Daytime Study Procedure

To control for the effects of sleep and the passage of time alone on memory consolidation, a similar protocol as the WPA task EEG overnight was done, except during the day without a sleep period. Participants in the two-visit daytime control study completed the same screening as those in the 3-night study, except without undergoing an initial PSG. Eligible participants arrived at the lab in the morning between 7:00hr-9:00hr where they were connected to EEG equipment and repeated the exact learning session for the WPA task as described above. After completing the test during the learning session, EEG equipment were disconnected, and participants were given a list of acceptable activities to do and not do, and an actigraphy watch to objectively verify that participants did not nap or engage in strenuous physical activity during the delay period. Actigraphy data was scored to ensure the approved activities between testing times involve minimal mental and physical exertion was followed. Unapproved activities include moderate to intense physical exercise, psychologically intense movies or television, napping, high-stress activities, and consuming alcohol and recreational drugs. Participants returned in the evening, approximately eight hours after leaving in the morning; an amount of time comparable to the sleep opportunity during the three-night study. During the evening visit, participants were re-connected to the EEG equipment, then completed the WPA long-delay recall test.

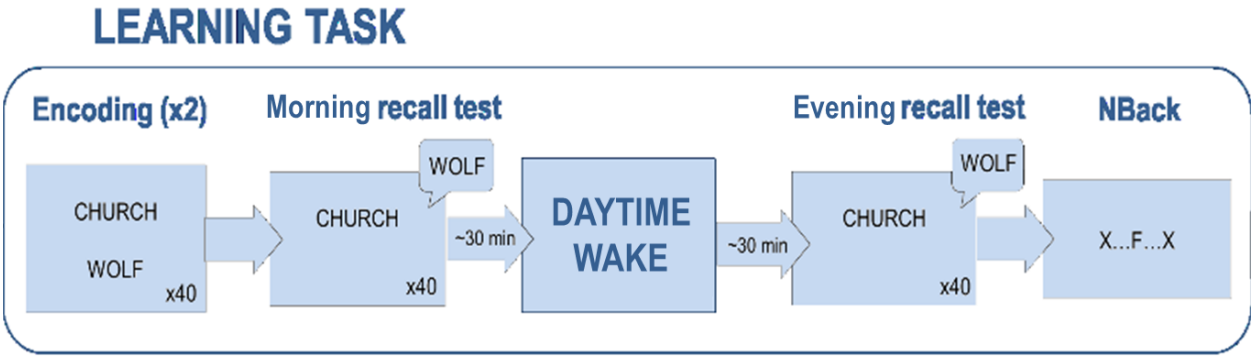

Figure 2. Overview of daytime study procedure. In order, participants 1) conduct learning and immediate recall for WPA task in the morning with EEG, 2) leave the lab to conduct preapproved, calm activities during the day, then 3) return to the lab to conduct delayed recall of WPA task, 1- and 2-Back, and Cued GNG tasks in the evening with EEG. Figure adapted from Mameri-Arab et al. (2019), Figure 2.

Electrophysiological Analysis

All PSG and EEG signals were recorded using Domino equipment and software (bandpass filter 0.2-128Hz, EEG sampling rate 512 Hz: SOMNOmedics, GmbH, Randersaker, Germany). The PSG setup included EEG, electrooculogram (EOG), chin electromyogram (EMG), electrocardiogram (ECG), transcutaneous finger pulse oximeter (SpO2), respiratory measures (oronasal thermocouple, nasal pressure cannula, thoracic and abdominal piezo-electric belts), and leg EMG. The EEG used for the PSG screening night included a 12-channel montage following the International 10-20 System for electrode placement according to the American Academy of Sleep Medicine (AASM) (Fz, F3, F4, Cz, C3, C4, O1, O2, M1, M2, Pz for reference, Fpz for ground). This setup allowed for the objective detection of sleep disorders (e.g., sleep apnea, restless leg syndrome, insomnia). The second and third overnight recordings included only ECG, EOG, chin EMG, and EEG with an eighteen-channel montage (Fz, F3, F4, F7, F8 Cz, C3, C4, T3, T4, T5, T6, P3, P4, O1, O2, M1, M2, Pz for reference, Fpz for ground).

Sleep Staging and Architecture

Sleep and daytime study recordings were collected using Domino and processed and scored using a custom-built software package ("Wonambi") run using Python. Sleep recordings are scored in 30 second epochs as either N1, N2, N3, or REM sleep by an experienced sleep scorer. Artifacts, poor signal quality, and arousals were removed from EEG analysis. Artifacts include heart, eye and muscle activity, and brief transient electrode activity. Arousals are sudden increases in EEG activity, typically lasting 3 seconds and accompanied by an increase in chin EMG, followed by at least 10 seconds of sleeping EEG activity. The following was calculated for each participant in each group: total sleep time (TST), sleep efficiency percentage (SE%), sleep onset latency (SOL), wake after sleep onset (WASO), sleep fragmentation index (SFI), the number of awakenings per hour, sleep switch index (SSI), the number of sleep stage transitions per hour, Apnea-Hypopnea Index (AHI), and TST and TST% for N1, N2, N3, and REM sleep.

Spectral Analysis

Power spectral density (PSD) analysis was performed on EEG recording signals during REM sleep using a Welch method with overlapping segments (50% overlap), and a four second Hanning window filter. This was done for fixed and adapted bands for their comparison. Adapted bands are frequency bands based on spectral peaks of each individual subject. An advantage of adapted bands allows for the personalization of peak frequencies unique to individuals, which considers between-subject variance. However, analysis with adapted bands is in the *supplementary* section, due to several subjects lacking adapted theta peaks, especially in older adults. Therefore, an average peak per age group per night was used for subjects without identifiable peaks. Spectral peaks were derived using the FOOOF algorithm, where frequency and amplitudes of spectral peaks are determined from modeling 1/f background activity (Donoghue et al., 2020). If more than one theta peak was detected, the frequency of the largest amplitude was used. Fixed theta frequency band was from 4-8Hz, adapted theta was calculated as individual subject peak frequencies within 4-8Hz +/- 2Hz, and slower and faster gamma frequency band was 35-64.75Hz and 65-100Hz respectively (Köster et al., 2014; Mameri-Arab et al., 2019). Primary analysis was done within frontal electrode, Fz, and parietal electrode, Pz, given the majority of cortical TGC research was performed on frontal regions and REM TGC in parietal regions (Bandarabadi et al., 2019; Scheffzuk et al., 2011). Secondary analysis was done with central electrode, Cz, and temporal electrodes, T3 and T4, which are listed in the

supplementary section. Adapted theta analysis was done only on Cz and Fz electrodes. Spectral analysis data was derived as a grand mean value across all REM sleep cycles (Bandarabadi et al., 2019). Theta and gamma PSD were detected using Wonambi. Prior to coupling analysis, differences between memory and control task nights in individual fixed and adapted theta band and slower and faster gamma band power were compared with overnight memory consolidation and contrasted between groups.

Coupling Analysis

TGC from Fz, Pz, Cz, T3, and T4 were computed using specialized programming scripts as a mean value for each participant on each of the two experimental nights. Gamma bands were filtered using a Laplacian filter to remove gamma-related muscle artifact (Machado et al., 2010). A Modulation Index (MI; Tort et al., 2010) and coupling phase (CP) were computed from this data. Using the EEG signal during REM sleep, MI and CP were examined as 1) fixed theta-slower gamma coupling (TGCs), 2) fixed theta-faster gamma coupling (TGCf), 3) adapted theta-slower gamma coupling (aTGCs), and 4) adapted theta-faster gamma coupling (aTGCf).

EEG data was filtered using a Hilbert transform by extracting the instantaneous phase time series of theta and amplitude time series of slower and faster gamma. MI was calculated by comparing the differences between the distributions of 1) instantaneous gamma amplitude changes across 18 bins (20° each) of theta phase to 2) the same variables with a null hypothesis assuming there will be no change in gamma amplitude in any theta phase bin, meaning there is an equal, flat distribution for each phase bin. MI is a measure of coupling strength, where differences between the calculated and the null distributions is expressed as a value from 0-1. MI values were log-transformed to quantify increases in frequency as a linear progression (Buzsáki & Draguhn, 2004). Log-transformed MI values that are less negative indicate that the calculated and null distributions lesser coupling strength; in turn, more negative values signify greater coupling strength.

Preferred CP is the phase of theta accompanied by an increase in gamma amplitude (Takahashi et al., 2014; Belluscio et al., 2012). A circular mean direction, which is the average direction gamma couples in, as circular degrees of theta was extracted. To compute CP, a mean PAC time series is created by the extracted circular mean direction (Takahashi et al., 2014; Belluscio et al., 2012). CP value changes between nights indicate a shift in the theta phase preference of gamma amplitude, possibly due to dependence upon the task night, CP was calculated as a grand average across all REM cycles.

MI and CP, both measures of TGC, compared, 1) between learning and non-learning nights to test whether increases or changes in TGC and PSD during REM sleep are dependent upon whether a memory task was done prior to sleep, and 2) younger and older adults to test whether there is an age-related weakening of TGC and PSD during REM sleep. Memory consolidation between overnight sleep and daytime wakefulness, and older and younger adults were compared to assess sleep-dependency and age-related differences on memory consolidation.

Statistical Analysis

To first screen data, we assumed a random sample, and every participant was independent of each other, i.e., not related. Everyone in a population of healthy, good sleeping younger and older adults in Montreal, Quebec, Canada had an equal chance of being in the study. All MI and PSD tests were each done primarily for channels Fz, and Pz. Whereas the same tests

were done for Cz, T3, and T4, as supplementary analysis. The frequency of means of self-report questionnaires, MMSE, MoCA, sleep architecture values, sleep diary and actigraphy data, WPA task scores, and MI and PSD values were plotted to determine normality of the sampled dataset, and the presence, or lack of, left- or right-skewed tails. Using the same variables and values, maximum and minimum of z-score transformation values were calculated using a descriptive statistics test to find +/-3 standard deviations to identify outliers. The standard deviation (SD) of MI and PSD values of younger and older adults were compared using a Levene's test, confirming SDs are equal between age groups to find the homogeneity of variance. MI and PSD values were plotted against memory consolidation, respectively, to determine whether there was a linear relationship between variables, as assumed for Pearson's correlation and multiple linear regression. To assume normal distribution of the residuals, first, for each age group, respective MI and PSD values were plotted in a scatter plot. Correlations between independent variables and covariates were calculated to ensure the multicollinearity assumption was not violated for multiple linear regression.

For between-night comparisons, the primary outcome variables related to the objectives are MI and CP, and PSD as a secondary outcome. Likewise, with age and biological sex being covariates, repeated measures analysis of covariance (rmANCOVA) was performed for MI and PSD values during REM sleep to determine whether coupling is sufficiently different between task nights. Analysis of variance (ANOVA) for 3-night and daytime study memory consolidation scores were conducted separately for both age groups to determine whether memory consolidation was better after overnight sleep as to opposed daytime wake. Ensuring age groups are significantly different, analyses of covariance (ANCOVAs) were performed for MI, PSD, and 3-night and daytime study memory consolidation, respectively. To test associations between all MI and PSD values with memory consolidation, 1) hierarchical multiple linear regression, controlling for age and biological sex, and 2) Pearson's r correlations were performed separately for both age groups. For hierarchical multiple linear regressions, the first (baseline) model included just age and biological sex to predict memory, and in the second step the PSD or MI value of interest was added to examine its influence while controlling for the covariates. Pearson's r correlations with either a PSD or MI value with memory consolidation. No corrections for multiple comparisons were done, given the exploratory nature of this study. Lastly, to consider the effects of sleep disordered breathing, measured by AHI, supplementary ANCOVAs, rmANCOVAs and hierarchical multiple linear regressions tests were performed using AHI, in addition to age and biological sex as covariates.

Ethics Statement

This research project is evaluated and approved by the Concordia University Human Research Ethics Committee and Centre de Recherche de Institut Universitaire de Gériatrie de Montréal (IUGM).

RESULTS

Participant Characteristics

A final sample of 37 participants were recorded for two age groups, younger (N = 21) and older (N = 16) adults. The younger adult sample consists of 13 female and 7 French speaking adults, and the older adult sample consists of 11 female and 5 French speaking adults (Table 1).

Biological sex and mother tongue (French or English) were not significantly different between groups. MMSE and MoCA scores were within healthy ranges for cognition and not significantly different between age groups. All self-report questionnaires scores indicated most sampled participants did not exhibit exclusion criteria, as there were two older adults with scores above 10 for the Epworth Sleepiness Scale (ESS), one of whom scored above 10 on the Geriatric Depression Scale, indicating excessive daytime sleepiness and depression. However, those participants met the criteria of healthy, good sleepers based on PSG and overnight EEG studies. Questionnaire scores were not significantly different between age groups, with the three exceptions. Older adults scored higher on the Insomnia Severity Index ($p = 0.015$), higher depression scores (Beck Depression Inventory II for younger adults, and Geriatric Depression Inventory for older adults; $p = 0.003$), and more right-handedness on the Edinburgh Handedness Inventory ($p = 0.036$).

Actigraphy and Sleep Diary Parameters Between Overnights

To control for a consistent sleep/wake schedule, participants wore actigraphy watches and filled out sleep diaries daily starting from the first to the third/last EEG overnight study. All measures for actigraphy data were not significantly different between age groups, including total sleep time (TST), sleep efficiency (SE), sleep onset latency (SOL), wake after sleep onset (WASO), number of awakenings (AW), time in bed (TiB), and number of days recorded (Days). For the sleep diary entries, average TST ($p = 0.001$), SE ($p < 0.001$), and Days ($p = 0.026$) were greater in younger adults, and WASO ($p < 0.001$) was greater in older adults (Table 2). According to both actigraphy watches and sleep diary entries, healthy, average sleep habits according to the national sleep foundation, were maintained by participants throughout the study (Ohayon et al., 2017). Interestingly, subjective but not objective sleep measures were different between older and younger adults as only sleep diary, but not actigraphy measures were significantly different between age groups.

Healthy Sleep Architecture Among all 3 Overnight Sleep Studies

The first of three overnight sleep studies was a polysomnogram (PSG) to objectively identify sleep disorders and help participants acclimate to sleeping in a lab setting. All sleep architecture parameters, including TST, SE, SOL, percent of TST (%) of N1, N2, N3, and REM sleep, WASO, stage switching index (SSI), and apnea-hypopnea index (AHI), were below exclusion criteria of healthy, good sleepers described in methods, respective for average younger and older adults (Ohayon et al., 2017). AHI is only measured and calculated in the PSG overnight and for younger adults was 2.72 ($SD = 2.20$) and for older adults was 8.04 ($SD = 3.46$). SE was significantly greater in younger adults, and % N1, WASO, SSI and AHI were significantly greater in older adults (Table 3). For the latter two overnight sleep studies (EEGm, memory task night, and EEGc, control task night), all sleep architecture parameters were within healthy ranges respective to age groups on both nights according to the national sleep foundation (Ohayon et al., 2017). Additional sleep measures calculated in EEGm and EEGc but not in PSG are the number of cycles (Cycles) and sleep fragmentation index (SFI). SE, % N3 and % REM were significantly greater in younger adults, and % N1, % N2, WASO, SSI, and SFI were significantly greater in older adults (Table 4). In general, younger adults slept better and got greater proportions of N3 and REM sleep, which is expected.

Learning Task

The learning task performed during the memory task night (EEGm) and daytime study was a word-pair association (WPA) task measuring declarative memory before (immediate) and after (delayed) sleep, and morning (immediate) and evening (delayed), respectively. Younger adults recalled an average of 37.48 words out of 40 for immediate recall (IR) and 37.67 for delayed recall (DR) (IR: $SD = 2.44$; DR: $SD = 2.85$). Older adults recalled an average of 33.38 out of 40 words for IR, and 31.44 for DR (IR: $SD = 5.49$; DR: $SD = 5.93$). Younger adults recalled significantly more words in the IR and DR (IR: F (1, 36) = 9.032, $p = 0.005$; DR: F (1, 36) = 17,456, $p < 0.001$). In the daytime study, younger adults scored 34.09 for IR and 33.00 for DR (IR: $SD = 6.44$; DR: $SD = 6.48$). Older adults scored an average of 25.13 for IR and 21.88 for DR (IR: $SD = 10.53$; DR: $SD = 12.14$). All adults during EEGm, compared to the daytime study, recalled significantly more words during IR and DR (IR: F (1, 54) = 8.467, $p = 0.005$; DR: F (1, 54) = 9.688, $p = 0.003$) (Table 5).

Memory consolidation was calculated as total amount of paired words recalled in both IR and DR. Average EEGm memory consolidation scores were 37.10 for younger adults, and 32.35 for older adults (younger: $SD = 2.88$; older: $SD = 7.79$). Average daytime study memory consolidation scores were 32.35 for younger and 20.63 for older adults (younger: $SD = 7.79$; older: $SD = 12.50$). IR, DR and memory consolidation scores were all significantly greater in EEGm than daytime studies (EEGm: $p = 0.005$; $p = 0.003$; daytime: $p = 0.012$; $p = 0.006$). IR, DR, and memory consolidation scores were all significantly greater in younger than older age groups (IR: $p = 0.005$; DR: $p < 0.001$; memory stability: $p < 0.001$; memory consolidation: $p < 0.001$) (Table 5). Compared to older adults, younger adults had higher IR, DR, and consolidation scores. Likewise, compared to adults who were awake during the day, adults who slept overnight had higher IR, DR, and consolidation scores.

PSD and TGC MI Parameters Between Age Group and Night Types

All following analyses in remaining *result* sections below were done in channels Fz and Pz. Between memory (EEGm) and control (EEGc) task nights and between age groups, controlling for biological sex and age, all PSD and MI analyses were not significantly different (Tables 6-7).

TGC Coupling Phase Between Age Group and Night Types

To calculate coupling phase (CP), circular plots were made for different types of TGC between EEGm and EEGc, separately for younger and older adults. All results for CP were purely based off visual inspection, and no statistical analysis was performed, therefore interpretation was cautious. Vectors in circular plots represent greater amounts of gamma waves coupling in that direction of degrees of theta. In terms of CP for these results, precision is defined as how clustered together large vectors are in circular plots. In Fz, TGC seemed more precise in EEGc (Figure 3). Slower gamma coupling (TGCs) seemed to prefer the trough in younger, and trough-to-peak state in older adults. Faster gamma coupling (TGCf) in older adults somewhat preferred the peak and trough-to-peak state. Speaking about Fz overall, TGCs seemed to prefer the trough in younger and peak in older adults in EEGm but preferred the trough in EEGc in younger adults. In Pz, TGC precision does not seem remarkably different between age groups or nights (Figure 4). Directionality between age groups in Pz show TGCs possibly preferring the peak in younger adults and trough-to-peak state and peak in older adults, but no visually meaningful differences for TGCf. There are no remarkable differences in directionality

between night types in Pz. Altogether, there seemed to be an overall decrease in precision in older adults, regardless of channel. There is a possible difference in directionality between age groups for TGCs, but not TGCf, in Fz and Pz, but whether the directionality in younger adults is meaningful in terms of memory is questionable, based on differences between night types.

TGC Mean Amplitudes Between Age Group and Night Types

To visualize instantaneous gamma amplitude change in relation to theta phase, plots of mean gamma amplitude among 18 20° discrete phase bins of theta were made. All results for mean gamma amplitude plots were purely based off visual inspection, and no statistical analysis was performed, therefore interpretation was cautious. There were no remarkable differences in slower gamma (TGCs) mean amplitude between age groups and night types in Fz (Figures 5). Between age groups in EEGm in Fz, faster gamma (TGCf) mean amplitude may show a very small decrease near the peak in younger adults and near the trough in older adults. In both age groups, these decreases are not shown in EEGc. However, there may be a slight increase in the transition state in EEGc in younger adults. In Pz, between age groups, there are no differences in TGCs or TGCf in EEGm (Figure 6). Like in Fz, there are no noticeable differences between night types in TGCs in Pz in younger adults. Interestingly, during EEGc in older adults, sequentially as theta is in the trough there may be a gamma amplitude decrease, in the transition state there may be a sharp increase and, at the peak, there may be a subsequent decrease. A similar, much weaker pattern can be seen in faster gamma mean amplitude in younger adults, but in both EEGm and EEGc. Essentially, there were minor, and possibly negligible, differences in mean gamma amplitude in EEGm between age groups in both channels. Although, Pz showed possible amplitude increases in EEGc in older adults, that were not reflected in EEGm, nor younger adults.

Relationship between PSD and TGC MI with Memory Consolidation

Covariates biological sex and age were introduced in the first of two steps in each regression analysis. Biological sex and age with the TGC MI or PSD value of interest were introduced in the second of two steps. In Fz, TGCf MI in younger adults was positively correlated with memory consolidation ($r = 0.558$, $p = 0.009$) (Table 8; Figure 7). Supporting the significant correlation, model 2 TGCf MI explained 47%, and alone an additional 34% of the variance in memory consolidation (model 2: $Adj.\ R^2 = 0.47$, $\Delta R^2 = 0.34$, $\beta = 0.59$, $F\ (3, 17) = 6.903$, $p = 0.002$) (Tables 9; Figure 7). In Pz in younger adults, fixed theta PSD in model 2 explained 28%, and alone an additional 18% of the variance in memory consolidation, despite not being correlated with memory consolidation (model 2: $Adj.\ R^2 = 0.28$, $\Delta R^2 = 0.18$, $\beta = 0.42$, $F\ (3, 17) = 3.566$, $p = 0.042$) (Table 9-10; Figure 8). No other significant correlations or regressions were found for other PSD and TGC MI values in any channel in either age group (Tables 9-10).

Summary

All adults exhibited healthy, good sleep, based on averages of the respective age group, as expected. Likewise, all measures of memory were better in younger adults and for all adults who slept overnight. PSD and TGC MI measures were not significantly different between age groups and night types. CP seemed less precise (i.e., more varied) in older adults, which was expected, and there were questionable age-related differences in the directionality of coupling for TGCs. Similarly, there were minor, questionably negligible differences in mean gamma

amplitude between age groups during EEGm. However, there may be increases in gamma amplitude in Pz in older adults in EEGc. Despite this, there were no significant correlations or regressions between TGC MI and CP with memory consolidation in older adults. Conversely, TGCf and theta PSD were positively correlated with and TGCf predicted improved memory consolidation in younger adults.

Summary of Supplementary Analyses

TGC MI and theta and gamma PSD were not significantly different between age groups and night types in all channels (Tables S1-S4). Gamma amplitude coupling to adapted theta phase (aTGCs and aTGCf) in Fz in younger adults may show a preference in the theta peak-to-trough transition state and possibly appeared to have less variability in the temporal synchronization in aTGCf (Figure S1). Likewise, there was likely less CP precision in older (Figures S1-S3).

Regarding the association with memory consolidation in younger adults, aTGCf in Fz showed positive correlation and regression results, and TGCs showed a positive trend with memory consolidation in T4 (Tables S5-S7; Figure S7, and S13). In younger adults, fixed theta PSD showed a positive trend to predict changes in memory consolidation in Cz and T3, and a positive trend correlating it with memory consolidation (Tables S6, S8-S9; Figures S11-S12). Likewise, in older adults, TGCs in Cz was positively correlated with memory consolidation (Tables S5; Figures S8). As for PSD, faster gamma PSD in Cz and T3 in older adults showed a respective positive trend and significant correlation with memory consolidation (Tables S5 -S6; Figures S9-S10). In terms of CP, results show possible less precise TGC synchronization in older adults. Overall, in younger adults there is evidence relating aTGCf in Fz, TGCs in T4, and fixed theta PSD in Cz and T3 to memory consolidation. In older adults, TGCs in Cz and faster gamma PSD in Cz and T3 were positively related to memory consolidation. When using age and sex in addition to AHI as covariates, there were no differences in TGC MI and PSD between night types and age groups, and when used in the second step of multiple linear regressions, TGC and PSD were not predictive of memory consolidation (Tables S11-S20).

DISCUSSION

Summary

The goal of this study was to find memory-related theta-gamma coupling (TGC) during REM (REM TGC) that may change with age. My first aim was to establish REM TGC in humans and my second aim was to examine whether REM TGC is related to overnight sleep-dependent memory consolidation. My first hypothesis that REM TGC in humans is correlated with and can predict overnight declarative memory consolidation was partially supported. Only theta-faster gamma coupling (TGCf) was positively correlated with and predicted increases in memory consolidation in Fz in younger adults (Tables 8-9; Figure 7). My third aim was to investigate age-related differences in REM TGC and whether those differences are associated with age-related declines in sleep-dependent memory consolidation. I hypothesized that REM TGC strength would become weaker with age-related memory decline. Evidence from the primary analysis did not fully support my second hypothesis. REM TGCf related to memory consolidation was only found in younger adults and there were no significant differences in REM TGC MI and mean gamma amplitude between age groups (Table 7; Figures 5-6). It was expected

that CP be less precise in older adults but without statistical analysis comparing groups, it cannot support for my second hypothesis (Figures 3-4). My third, and last hypothesis was that memory consolidation would be greater in adults who slept overnight in the three-night study than those who were awake during the day for a similar amount of time in the daytime study. This considers the effects of the passage of time alone on memory, rather than just sleep. Evidence from my main analysis fully supports my third hypothesis. All adults who slept overnight had greater memory consolidation than those awake during the day for a similar amount of time, suggesting the passage of time alone is not the cause of memory consolidation improvement (Table 5).

Supplementary analysis enables a slightly different interpretation of the association between REM TGC and memory, as well as between age groups. In support of my first hypothesis stating REM TGC is related to memory, aTGCf in Fz and TGCs in T4 in younger adults and TGCs in Cz in older adults were positively related to memory consolidation (Tables S5-S7; Figures S7-S8 and S13). Considering age-related changes in REM TGC, however, my second hypothesis, was not wholly supported as all REM TGC MI and PSD measures are not significantly different between age groups and night types in Cz, T3, and T4 (Tables S1-S4). Like my primary analysis in Fz and Pz, CP possibly shows less precision in the synchronicity of TGC in older adults, but this is not sufficient without statistical analysis to support my second hypothesis (Figures S1-S3). Our supplementary analysis found an association with REM TGC with memory in older adults which was not found in the primary analysis. Likewise, gamma PSD has a positive correlation trend effect in older adults, that was only observed in the supplementary analysis. Both analyses show REM TGC MI is not different between age groups. Interestingly, when controlling for AHI, there was no difference in TGC and PSD between age groups, and TGC and PSD did not predict changes in memory consolidation (Tables S11-S20).

REM TGC in Relation to Memory

Ours was the first study to investigate REM TGC in humans, and the effect of REM TGC on declarative memory. Faster gamma coupling (TGCf and aTGCf) in the frontal channel in younger adults is positively correlated with and predicted positive changes in memory consolidation (Figure 7, Figure S7). TGCs in Cz in older adults was positively correlated with memory consolidation (Figure S8). Finding slower gamma coupling to be correlated to memory consolidation in older, but not younger adults, and vice versa for faster gamma coupling, may reflect the natural slowing of oscillations with increasing age (Zhang et al., 2022). It is known that REM sleep mostly consists of theta and gamma waves, and there is some evidence of prominent beta waves (Cantero et al., 2003; Montgomery et al., 2007; Rasch & Born, 2013; Vijayan et al., 2017). The functional roles of gamma and beta waves during REM sleep are not well known. However, accumulating evidence suggests cortical and hippocampal theta during REM sleep positively impacts semantic and emotional memory (Vijayan et al., 2017; Cantero et al., 2003; Fogel et al., 2007; Nishida et al., 2009; Boyce et al., 2016). It is not entirely understood how these oscillations interact with one another, and our study attempted to investigate those interactions, mainly through TGC.

Our study found that REM sleep faster and slower gamma coupling in younger adults and slower gamma coupling in older adults were somewhat associated with memory consolidation, as measured by MI. To the best of our knowledge, no other studies linked REM TGC to memory consolidation. Most human TGC studies focused on encoding to predict successful retrieval, using a modulation index (MI) to quantify coupling strength (Tort et al., 2010; Lega et al., 2016; Canolty et al., 2006; Friese et al. 2013; Schomburg et al., 2014; Colgin et al., 2015). The few

studies that do not use MI, either looked at coupling phase (CP) or used their own method for quantifying synchronization between theta and gamma rhythms (Karlsson et al., 2022; Park et al., 2013; Mormann et al., 2005). In human research not all studies agree what theta phase gamma prefers to couple in, during encoding. To the best of our knowledge, all invasive imaging studies reporting TGC CP and MI in humans, and all animal studies, in either the cortex or hippocampus, suggest TGC in the trough is most related to successful encoding (Lega et al., 2016; Canolty et al., 2006). Only one paper, which only reported CP and used non-invasive, posterior cortical EEG, found TGC (gamma: 30-100Hz) in the peak was the most predictive of successful encoding of semantic memory in humans (Karlsson et al., 2022). It is difficult to draw conclusions between TGC CP and memory during encoding, retrieval, or REM sleep. This is because there is only one known study examining that relationship with encoding, and, in our study, statistics were not calculated to identify chance levels nor to investigate TGC CP to corroborate the aforementioned study (Karlsson et al., 2022).

Interestingly, one study examining both working memory and REM sleep in the rat hippocampus (CA1) showed that greater TGC MI was related to working memory, but that a slower gamma coupling (30-45Hz) preferred the peak, and a faster gamma coupling (60-90Hz) preferred the trough (Takahashi et al., 2014). This same CP was found during REM sleep in the same rodents, and those results were corroborated by another study (Takahashi et al., 2014; Belluscio et al., 2012). Of the handful of studies examining REM TGC, all are in rodents, analyze TGC with different speeds of gamma, (slow, middle, and fast for example), and report different preferred CP (Scheffzuk et al., 2011; Belluscio et al., 2012; Montgomery et al., 2007; Bandarabadi et al., 2019; Scheffer-Teixeira et al., 2012; Del Vechio Koike et al., 2017). In the rat parietal cortex, gamma coupled the peak in one study and the trough in another, regardless of the speed of gamma (Scheffzuk et al., 2011; Bandarabadi et al., 2019). Summarizing this previous work in rodent models, in the hippocampus, a slower gamma coupled in the peak, a faster gamma coupled in the trough, whereas it is unclear whether gamma prefers the peak or trough in the parietal cortex (Takahashi et al., 2014; Belluscio et al., 2012; Scheffzuk et al., 2011; Montgomery et al., 2007; Bandarabadi et al., 2019; Scheffer-Teixeira et al., 2012; Del Vechio Koike et al., 2017). In our study however, we found that slower gamma possibly prefers to couple in the peak and faster gamma possibly prefers the trough-to-peak transition in Pz. Our results do not provide additional clarity to previous studies. If TGC CP in the hippocampus is like that of in the parietal cortex, our results may be consistent with what was found in the rodent hippocampus, but that is speculative. The relevance of REM TGC CP is unclear in terms of memory consolidation, as our and previous studies did not use circular statistics to analyse this.

It is thought that cross-frequency coupling (CFC) represents an underlying mechanism of information transfer between and within regions of the brain, which is memory consolidation (Rasch & Born, 2013). During NREM sleep, better temporal precision in SO-spindle CFC improves declarative memory consolidation (Muehlroth et al., 2019). Such CFC associations have only been previously found during NREM sleep. Making comparisons with previous literature is difficult, as no animal study investigated cortical frontal, central or temporal REM TGC, to the best of our knowledge. However, one study positively cross-correlated TGC occurring in the CA1 and subiculum of the hippocampus with TGC occurring in the parietal cortex of rodents, but no memory measures were performed (Bandarabadi et al., 2019). REM TGC co-occurring in the hippocampus and the parietal cortex may be an underlying mechanism of memory consolidation. Under that assumption, REM TGC in the hippocampus may also be observed in other cortical regions than just parietal, such as frontal and temporal (Bandarabadi et

al., 2019; Rasch & Born, 2013). This process supports the multiple trace theory of hippocampal dependent memory, supported by active systems consolidation (Rasch & Born, 2013; Diekelmann & Born, 2010; Wang et al., 2017).

Memory traces in the hippocampus would be reactivated when retrieving declarative memory, reflecting hippocampal TGC related to encoding and possibly REM TGC (Adamantidis et al., 2019; Rasch & Born, 2013; Diekelmann & Born, 2010). The formation and strengthening of memory traces is supported by long-term potentiation (LTP) of synapses which is known to be driven by brain-derived neurotrophic factor (BDNF) (Leel et al., 2017; Panja & Bramham, 2014; Gott et al., 2017; Ulloor et al., 2005). Hippocampal LTP and memory trace reactivation may lead to subsequent reactivation in the cortex, which could be reflected by known cortical TGC related to successful encoding (Rasch & Born, 2013; Diekelmann & Born, 2010; Friese et al., 2013; Karlsson et al., 2022; Lega et al., 2016; Mormann et al., 2005; Canolty et al., 2006). In this regard, REM TGC occurring in other cortices may be the result of information transfer from the hippocampus, which can possibly explain why faster gamma coupling in Fz and slower in T4 in younger adults, and slower gamma coupling in Cz in older adults were related to successful declarative memory consolidation (Figures 7, S7-S8, and S13). In one study, 1) hippocampal TGC strength and synchrony, 2) theta phase coherence, and 3) synchrony of theta-driven hippocampal connections with the prelimbic cortex were all reduced with BDNF suppression in the hippocampus of mice during memory retrieval (Hallock et al., 2019). Likewise, the loss of BDNF function in mice resulted in reduction in synchrony in theta between the hippocampus and medial prefrontal cortex, which was related to a reduction in fear memory retention in mice (Hill et al., 2016). Results from both studies provide causal evidence of the relationship between a synchronization of TGC and theta oscillations with improvement of memory traces through a BDNF-driven mechanism.

Hippocampal TGC and theta synchrony may be influenced by pontine-geniculo-occipital (PGO) waves which originate in the brainstem (Adamantidis et al., 2019). Most previous work on PGO waves and hippocampal oscillations examined them in relation to emotional memory and connections between the hippocampus and amygdala (Adamantidis et al., 2019; Abe et al., 2008; Gott et al., 2017). TGC driven communication between the hippocampus and other regions may be the result of PGO waves (Adamantidis et al., 2019). Considering that, a recent study showed REM sleep PGO waves synchronously coupled with hippocampal theta waves in mice, which directly led to the modulation of the excitability of the CA1 (Tsunematsu et al., 2022). This provides support for deeper brain structures, such as the pons and geniculate nuclei, to regulate hippocampal connections with other brain regions, driven by theta wave synchrony (Adamantidis et al., 2019; Abe et al., 2008; Gott et al., 2017). Conversely, during NREM sleep, hippocampal sharp-wave ripples (SWRs) couple with cortical oscillations and precede PGO waves. This suggests a functional communication from the cortex to the hippocampus, which may influence PGO waves through SWRs (Ngo et al., 2020; Tsunematsu et al., 2020). Given hippocampal theta oscillations and TGC have been observed in tandem with theta and TGC in cortical regions, it is possible that PGO waves drive the precision in TGC that leads to successful sleep-dependent memory consolidation (Bandarabadi et al., 2019; Adamantidis et al., 2019). This is likely due to PGO waves being a means of the ascending reticular activating system to generate and coordinate essential sleep-dependent neural activity throughout the brain, including the hippocampus and cortex, and vice versa, depending on whether it is NREM or REM sleep (Ngo et al., 2020; Tsunematsu et al., 2020; Datta et al., 1998; Tsunematsu et al., 2020; Yeo et al., 2013).

REM TGC Across the Lifespan

Synchronization and power of oscillations is thought to play a role in memory performance (Muehlroth et al., 2019; Helfrich et al., 2018; Scarpelli et al., 2019; Jacobson et al., 2013). One study found that during NREM sleep, phase durations of slow oscillations (SO) are longer, suggesting more time needed for neurons to synchronize. The authors indicated an age-related reduction in synchronization (Carrier et al., 2011). Likewise, there is an age-related lack of precision and overall reduction in SO-spindle coupling during N2 and N3 (Muehlroth et al., 2019; Helfrich et al., 2018). There may be a relatively well-recognized impact on the dynamics of SO-spindle CFC. The same age-related impact may exist for other oscillations, such as theta and gamma, but this is not as clear as oscillations during NREM sleep.

REM sleep oscillations show increases in alpha and beta waves, and mixed reporting of decreased delta waves across the lifespan (Bruce et al., 2009; Luca et al., 2015; Mann & Röschke, 2004). More pertinent to our study is an age-related decrease in theta power, observed in rats and older adults, but this reduction was not measured during sleep or related to cognition (Scarpelli et al., 2019; Jacobson et al., 2013). To the best of our knowledge, there is no previously documented age-related impact on TGC MI. We found theta and gamma PSD and TGC MI were not significantly different between age groups (Tables 7, and S3-S4). This suggests that REM TGC strength is stable across the lifespan, going against my second hypothesis which states it declines with age.

Relating MI with CP, larger REM TGC MI values tend to be related to greater synchronization of theta phase-locking to gamma amplitude (Scheffzuk et al., 2011; Bandarabadi et al., 2019; Del Vechio Koike et al., 2017). Despite no reported age-related reduction in TGC MI, it has been observed that the precision in synchronization of TGC declines with age in humans, and that this lack of synchronization likely reflects poorer cognition (Reinhart & Nguyen, 2019; Karlsson et al., 2022). This lack of synchronization reflected low-performance on a memory task, compared to those who were high-performing, further supporting the influence of synchronicity on cognition (Karlsson et al., 2022). We found possible worse precision in the synchronization of TGC in older adults (Figures 3-4, and S1-S3). These need further investigation, as we were unable to employ statistical analysis for CP and mean gamma amplitude increases. Therefore, despite possible age-related reductions in synchrony, it cannot be concluded that this evidence supports my second hypothesis.

Although the findings for synchronicity in our study are inconclusive, there is some evidence suggesting an age-related reduction in synchronicity of REM TGC and TGC during learning (Karlsson et la., 2022; Reinhart & Nguyen, 2019). Theta and gamma rhythms become more uncoupled with increasing age in humans, reflected by poorer performance on a change-detection task (Reinhart & Nguyen, 2019). Studies of healthy aging and memory provide some evidence that non-rhythmic, or non-oscillatory theta during REM sleep may be a factor for memory (Karlsson et al., 2022; Caplan et al; 2015). Uncoupled, and less synchronization in dynamic network communication is suggested to result from natural aging (Voytek & Knight, 2015; Pinal et al., 2015). Further research examining non-oscillatory bandwidths during sleep, and their impacts on memory are needed (Scarpelli et al., 2019).

An interesting finding in our study was a possible gamma amplitude increase during the trough-to-peak transition in older adults in Pz, during only one of the two task nights, EEGc. Of the few studies examining TGC in parietal regions, REM TGC in animals show gamma amplitude increase in the peak (Scheffzuk et al., 2011; Bandarabadi et al., 2019). One study

showed that during successful encoding in humans, gamma preferred to couple in the peak for parietal TGC, and another study exhibited frontal theta-parietal gamma coupling MI but made no mention of theta phase (Karlsson et al., 2022; Friese et al., 2013). Previous studies show gamma preferring the peak for TGC in parietal regions, which corroborates what we observed, but those studies did not incorporate or find differences in younger age groups. The reason for the increase occurring just in older adults and on one, not both task nights is unclear, and future research on TGC should investigate this further.

REM Sleep Theta and Gamma PSD Related to Cognition Across the Lifespan

Theta PSD has a positive relationship with TGC MI, and both have a positive impact on memory (Scheffzuk et al., 2011; Bandarabadi et al., 2019; Canolty et al., 2006). Cortical theta PSD during REM sleep is known to be related to cognition (Rasch & Born, 2013; Diekelmann & Born, 2010). We observed a positive relationship with REM sleep theta PSD in Pz, T3, and Cz with memory consolidation in younger adults (Figures 8 and S11-S12). Interestingly, theta PSD was not related to memory consolidation in older adults (Tables 8-10 and S5-S10). Although this may explain why we observed worse memory consolidation in older adults, fixed and adapted theta PSD was found to not be significantly different between age groups (Tables 7, and S3-S4). We found no positive results for adapted theta in terms of memory consolidation, suggesting individualized theta PSD may not necessarily reflect cognition-related or rhythmic theta bands (Tables S5-S10). In a similar nature, non-rhythmic theta in older adults may explain why no difference in theta PSD was observed between age groups, but only theta PSD in younger adults was positively related to memory consolidation (Karlsson et al., 2022; Caplan et al; 2015; Nyberg & Pudas, 2019; Nyberg et al., 2012). Nonrhythmic theta waves, as compared to rhythmic, are less synchronized, and there is an age-related reduction in precision of the synchronization of various oscillations during sleep (Karlsson et al., 2022; Caplan et al; 2015). This lack of synchronization may be separate from the PSD of an oscillation, which would explain why we observed no differences in theta PSD between age groups, but a positive relationship between theta PSD and memory consolidation in younger, but not older adults.

Aside from theta PSD, a novel finding in our study was a positive relationship between REM sleep gamma PSD with memory consolidation in older adults (Figures S9-S10). To the best of our knowledge, previous studies have not investigated REM sleep gamma in terms of aging or memory. During encoding and working memory in previous studies, gamma PSD was not significantly different between younger and older adults or between patients with mild-cognitive impairment or Alzheimer's disease and healthy controls (Karlsson et al., 2022; Goodman et al., 2018). This somewhat aligns with our results, as we found no significant differences in gamma PSD between age groups (Tables 7 and S3-S4). However, one study with a very large sample found reductions in wake gamma PSD with increasing age but did not investigate in terms of memory (Murty et al., 2020). Some evidence points to gamma PSD being unrelated to TGC MI during encoding, and, interestingly, this gamma PSD is topographically, evenly distributed (Canolty et al., 2006; Park et al., 2013). Our results do not align with an even distribution. Even though we did not compare gamma PSD between channels, we did find that gamma PSD was not significantly different between age groups. The only significant relationship with memory consolidation we found for gamma PSD were in Cz and T3 in older adults, and we did observe a possible increase in mean gamma amplitude in just Pz in older adults. Altogether, there are mixed results on gamma PSD in terms of aging, memory, and REM sleep, in our and other studies. One factor that clouded interpretation of all gamma PSD analyses

in our study was the lack of an applied Laplacian filter when calculating power spectral analysis. Any significant result we found for gamma PSD may, therefore, be confounded by gamma activity from muscle artifact or noise.

Strengths

To the best of our knowledge, this is the first study to investigate TGC during REM sleep (REM TGC) in humans. This is the first study to associate REM TGC with memory consolidation and examine differences in REM TGC across the lifespan. This is also one of the few studies to examine theta and gamma PSD during REM sleep between age groups. Our analysis found evidence for a positive impact of REM TGC on memory consolidation and significantly better memory in younger adults and in adults who slept regularly overnight, compared to those in the daytime study. In terms of novelty, we also found that biological sex was a significant predictor of memory consolidation. Being female was positively related to memory consolidation, especially in older adults, which is a novel finding, and will be discussed in further in *next steps* (Tables S21-S28).

The screening process to identify criteria of healthy, good sleeping adults was rigorous. It included nine different standardized self-report questionnaires apart of an in-person interview to subjectively assess eligibility. The PSG night not only monitored sleep disorders, but also acted as an adaptation night for participants to get used to sleeping in a novel environment wearing PSG equipment. This ensure all participants are healthy, good sleepers so we can compare their sleep and memory to several control groups. We controlled for sleeping brain activity that may reflect cognitive effort, but not memory by comparing the brain activity of a non-memory control task EEG night to that of a declarative memory WPA task night. Not all sleep studies control for the passage of time alone, which is known to change memory. We incorporated a daytime study to compare how memory consolidation is different without sleep during the day to with sleep overnight. In analyzing our spectral data, we applied a Laplacian filter when running all TGC analyses, to ensure all gamma coupling with theta is without gamma from muscle artefact, therefore reflecting brain activity.

Limitations

Our study has five limitations. First, our small sample size limits statistical power, particularly in the older adult sample (N = 16), which was smaller than that of the younger adults (N = 21). Secondly, since our study was exploratory, there were no corrections for multiple comparisons. Thirdly, memory consolidation scores were not normally distributed, likely reflecting the WPA task was not difficult enough for participants to perform just as poorly as they did well, on average. This violated the assumptions for parametric tests, and their results should be interpreted cautiously. Fourthly, gamma PSD values used for the aforementioned tests were calculated using power spectral analyses without a Laplacian filter, which, when applied, filters out gamma that is from noise and artifacts, particularly from muscle artifact (Machado et al., 2010). However, a Laplacian filter was applied for TGC analysis. Lastly, after controlling for AHI, there were no significant regression models that were not impacted by AHI, indicating sleep disordered breathing may have been a factor in our sample of healthy, good sleepers.

Next Steps

An interesting and unexpected finding was that biological sex significantly predicts improvements in memory consolidation, particularly in older adults (Tables S23-S28). Little can

be confidently asserted as to why this is the case, given the lack of previous literature on biological sex differences for REM sleep (Dorsey et al., 2021). It is known that hormonal fluctuations, the menstrual cycle, and menopausal status impacts sleep, particularly reducing sleep quality, which would lead to poorer memory performance (Dorsey et al., 2021). In our study, being female significantly predicted positive changes in memory consolidation. With what little is currently known, future directions should examine biological sex with coupling and oscillations during REM sleep.

In terms of memory consolidation, previous work with TGC during encoding in younger and older adults found that there are significant TGC differences between age groups, but with a particular focus on high- and low-performing adults within age groups (Karlsson et al., 2022; Nyberg & Pudas, 2019; Nyberg et al., 2012). This suggests TGC during wake is capable of being preserved across the lifespan, reflected by maintained short-term memory performance. This begs the question of whether the same logic can be applied to REM TGC and memory consolidation. Future studies with REM TGC and memory consolidation should separate high- and low-performing younger and older adults and conduct REM TGC MI and CP analyses in respective performing groups.

Aside from MI and CP, after inferring based on visual inspection, there may be a possible mean gamma amplitude increase in Pz in older adults, in just the control EEG night (EEGc) (Figure 6). At first glance there does not seem to be a clear explanation. Future research could run Whatson-William tests to compare CP between age groups and night types. This would provide statistical evidence regarding the phase gamma amplitude prefers to couple in. In a similar nature, circular correlations and regressions should be performed to consider the relationship between CP and memory consolidation.

Lastly, one of the prevailing distinctions in REM sleep are the subtypes, phasic and tonic REM sleep. Phasic and tonic REM sleep were not considered in our study, given a lack of a validated, objective metric to identify the stages in human sleep (Simor et al., 2021). In general, research on human phasic and tonic REM sleep has used non-validated, and relatively arbitrary metrics (De Carli et al., 2016; Waterman et al., 1993). Phasic REM sleep includes more sawtooth waves in cortical EEG, which is thought to reflect PGO waves and hippocampal theta synchronicity (Simor et al., 2021; Adamantidis et el., 2019). Even though one study in rodents found no significant correlations between REM TGC and eye movements, indicating phasic REM sleep, with a sample of 4 rodents for that correlation, future research should explore a validated metric of phasic and tonic REM sleep in humans, in relation to memory (Bandarabadi et al., 2019; Simor et al., 2021).

Concluding Remarks

We found that TGC MI is maintained throughout the lifespan, despite an age-related decline in memory consolidation. To the best of our knowledge, this is the first study to find this. This suggests the REM TGC strength is not a concern to the sleeping, aging brain. Although not every type of REM TGC is positively related to memory consolidation, faster gamma coupling (TGCf and aTGCf) likely plays a role for younger adults, and same for slower gamma coupling (TGCs) for older adults. This difference in faster and slower gamma coupling between age groups may be due to the natural slowing of oscillations with age (Zhang et al., 2022). Based on a previous study with TGC during encoding, what may be responsible for age-related memory decline is the desynchronization of TGC, and if desynchronization occurs during REM, it may be driven by reduced synchronization in PGO waves (Karlson et al., 2022; Adamantidis et al.,

2019). We possibly found a reduction in temporal precision of REM TGC, which aligns with an age-related reduction in synchronization in oscillations and an uncoupling of CFC (Adamantidis et al., 2019; Voytek & Knight, 2015; Pinal et al., 2015; Muehlroth et al., 2019; Helfrich et al., 2018; Karlsson et la., 2022; Reinhart & Nguyen, 2019). Our study may fill gaps in our understanding of the underlying mechanisms of memory decline across the lifespan. Using what little is known on it, we found REM TGC may improve sleep-dependent memory consolidation, the first study to do so.

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TABLES

Table 1
Demographics of Sample Population with Comparisons Between Younger (N = 21) and Older (N = 16) Age Groups

	YA (*M* (*SD*)) [*min-max*]	**OA (*M* (*SD*)) [*min-max*]**	***F* (1, 37)**	***P***
Age	**24.24 (2.96) [20-29]**	**67.44 (6.77) [56-77]**	**686.712**	**<0.001**
Sex	13 Female	11 Female	0.187	0.666
Language	7 French	5 French	0.018	0.893
Education (yr)	16.81 (4.17) [9-25]	17.17 (2.12) [14.5-21]	0.114	0.738
MMSE	29.05 (1.16) [27-30]	28.88 (1.20) [26-30]	0.129	0.722
MoCA	28.33 (1.32) [25-30]	27.13 (2.06) [22-30]	1.828	0.185
ISI	**1.55 (1.79) [0-7]**	**3.38 (2.87) [0-9]**	**6.581**	**0.015**
PSQI	2.38 (1.24) [0-4]	3.13 (1.20) [0-5]	0.345	0.561
ESS	5.10 (2.77) [1-9]	4.94 (4.22) [0-14]	2.380	0.132
STOP-BANG	0.48 (0.51) [0-1]	1.56 (0.63) [1-3]	1.451	0.236
UNS	4.80 (3.08) [0-10]	8.43 (4.04) [3-13]	0.561	0.465
MEQ-SA	55.52 (8.87) [40-70]	64.25 (9.49) [40-80]	0.022	0.883
Depression	**2.00 (2.37) [0-7]**	**4.63 (4.01) [0-12]**	**9.917**	**0.003**
Anxiety	1.57 (2.06) [0-8]	1.94 (2.98) [0-10]	1.330	0.257
EHI	**16 Right**	**16 Right**	**4.406**	**0.036**

Note. Depression scores measured using BDI, Beck Depression Inventory II (younger adults), and GDI, Geriatric Depression Scale (older adults). Anxiety scores measured using BAI, Beck Anxiety Inventory (younger adults), GAI, Geriatric Anxiety Inventory (older adults). YA, Younger Adults; OA, Older Adults; MMSE, Mini-Mental State Examination; MoCA, Montreal Cognitive Assessment; ISI, Insomnia Severity Index; PSQI, Pittsburgh Sleep Quality Index; ESS, Epworth Sleepiness Scale; UNS, Ullanlinna Narcolepsy Scale, MEQ-SA; Morningness-Eveningness Questionnaire Self-Assessment; EHI, Edinburgh Handedness Inventory; M, mean; SD, standard deviation; yr, years of education.

Table 2

Summary of Actigraphy and Sleep Diary Data with Comparisons Between Younger (N = 21) and Older (N = 16) Age Groups

	Actigraphy (*M* (*SD*))				**Sleep Diary (*M* (*SD*))**			
	YA	**OA**	***F* (1, 33)**	***P***	**YA**	**OA**	***F* (1, 35)**	***P***
TST (min)	440.64 (51.07)	428.71 (22.51)	0.709	0.406	**457.38 (49.27)**	**405.77 (32.56)**	**13.134**	**0.001**
SE (%)	90.83 (4.20)	91.51 (2.70)	0.300	0.588	**90.96 (3.31)**	**84.42 (5.04)**	**22.608**	**<0.001**
SOL (min)	20.44 (11.70)	18.25 (9.82)	0.342	0.563	11.35 (7.43)	12.63 (6.78)	0.294	0.591
WASO (min)	7.49 (8.81)	7.89 (5.17)	0.025	0.874	**5.50 (5.33)**	**21.32 (16.85)**	**17.169**	**<0.001**
AW (#)	9.80 (7.80)	8.54 (4.95)	0.300	0.588	1.18 (1.37)	1.99 (1.06)	3.862	0.057
TiB (min)	485.21 (46.74)	469.53 (33.31)	1.219	0.278	503.80 (56.33)	481.21 (38.66)	1.890	0.178
Days (#)	15.30 (4.37)	13.80 (3.57)	1.177	0.286	**16.81 (3.53)**	**14.63 (1.41)**	**5.437**	**0.026**
Sleep Quality Rating	-	-	-	-	**4.11 (0.42)**	**3.77 (0.53)**	**4.557**	**0.040**

Note. YA, Younger Adults, OA, Older Adults, TST, total sleep time, SE, sleep efficiency, SOL, sleep onset latency, WASO, wake after sleep onset, AW, number of awakenings after sleep onset, TiB, time in bed, Days, number of days recorded, M, mean, SD, standard deviation.

Table 3

Summary of Sleep Architecture of Younger (N= 21) and Older (N = 16) Age Groups during Polysomnography Overnight, with Between Age Group Comparisons

	YA (*M* (*SD*)) [*min-max*]	**OA (*M* (*SD*)) [*min-max*]**	***F* (1, 36)**	***P***
TST (min)	410.38 (50.57) [308.78-527.48]	384.46 (42.79) [319.50-456.48]	2.716	0.108
SE (%)	**88.60 (5.07) [78.30-94.80]**	**83.13 (5.72) [72.20-90.60]**	**9.499**	**0.004**
SOL (min)	18.78 (19.05) [2.70-79.50]	10.20 (9.18) [1.50-31.20]	2.743	0.107
% N1	**9.37 (5.92) [2.60-24.30]**	**13.93 (4.32) [8.00-21.30]**	**6.741**	**0.014**
% N2	51.30 (10.82) [16.40-64.80]	52.41 (7.89) [36.30-67.50]	0.119	0.732
% N3	20.90 (11.31) [3.90-58.90]	16.46 (7.58) [0.50-37.40]	1.826	0.185
% REM	18.42 (4.68) [6.30-26.40]	17.22 (4.42) [8.90-24.00]	0.631	0.432
WASO (min)	**53.81 (27.31) [18.55-102.95]**	**77.74 (26.71) [39.25-128.33]**	**7.107**	**0.012**
SSI (#/hr)	**14.40 (5.01) [6.60-24.40]**	**20.47 (6.13) [6.60-28.00]**	**10.975**	**0.002**
AHI (#/hr)	**2.72 (2.20) [0.20-8.00]**	**8.04 (3.46) [2.50-14.70]**	**32.571**	**<0.001**

Note. YA, Younger Adults, OA, Older Adults, TST, total sleep time, SE, sleep efficiency, SOL, sleep onset latency, % N1, percentage of NREM 1 sleep, % N2, percentage of NREM 2 sleep, % N3, percentage of NREM 3 sleep, % REM, percentage of REM sleep, WASO, wake after sleep onset, SSI, stage switching index, AHI, apnea-hypopnea index, M, mean, SD, standard deviation.

Table 4

Summary of Sleep Architecture Between Memory (EEGm) and Control (EEGc) EEG Overnight Studies in Younger (N = 21) and Older (N = 16) Age Groups with Between Age Group and Overnight Type Comparisons

	YA (*M* (*SD*))		**OA (*M* (*SD*))**		**YA vs. OA**		**EEGm vs. EEGc**	
	EEGm	**EEG**	**EEGm**	**EEGc**	***F* (1, 72)**	***P***	***F* (1, 72)**	***P***
TST (min)	419.12 (39.58)	404.95 (49.00)	402.44 (37.14)	393.50 (44.98)	1.943	0.168	1.408	0.239
SE (%)	93.64 (3.27)	91.51 (5.31)	88.48 (6.27)	86.77 (6.30)	**15.763**	**<0.001**	2.115	0.150
SOL (min)	12.96 (9.46)	11.67 (9.08)	10.35 (8.72)	8.94 (7.97)	1.684	0.199	0.426	0.516
Cycles (#)	4.67 (0.80)	4.52 (0.87)	4.81 (0.83)	4.94 (0.77)	2.149	0.147	0.020	0.888
% N1	6.43 (3.76)	6.61 (4.07)	11.24 (5.42)	11.13 (7.10)	**15.708**	**<0.001**	0.002	0.966
% N2	52.41 (7.02)	52.95 (7.89)	59.97 (6.79)	59.03 (6.72)	**16.866**	**<0.001**	0.003	0.958
% N3	20.47 (7.83)	20.04 (7.48)	11.28 (5.41)	12.25 (8.02)	**25.101**	**<0.001**	0.008	0.928
% REM	20.69 (4.83)	20.39 (4.44)	17.51 (4.47)	17.59 (4.91)	**7.668**	**0.007**	0.015	0.904
WASO (min)	19.86 (28.81)	18.69 (14.71)	39.28 (27.08)	41.53 (29.22)	**13.036**	**0.001**	0.002	0.961
SSI (#/hr)	14.48 (5.42)	14.06 (5.90)	23.17 (5.13)	22.32 (5.52)	**43.378**	**<0.001**	0.161	0.690
SFI (#/hr)	6.37 (3.00)	6.08 (2.91)	11.09 (3.02)	11.01 (3.47)	**45.589**	**<0.001**	0.047	0.828

Note. YA, Younger Adults; OA, Older Adults; EEGm, memory task night; EEGc, control task night; TST, total sleep time' SE, sleep efficiency; SOL, sleep onset latency; % N1, percentage of NREM 1 sleep; % N2, percentage of NREM 2 sleep; % N3, percentage of NREM 3 sleep; % REM, percentage of REM sleep; WASO, wake after sleep onset; SSI, stage switching index, SFI, sleep fragmentation index; M, mean; SD, standard deviation.

Table 5

Results of Word-Pair Association Task in Younger and Older Age Groups with Between Age Group and Nighttime and Daytime Study Comparisons

	YA (*M* (*SD*)) [*min-max*]		**OA (*M* (*SD*)) [*min-max*]**		**YA vs. OA**		**Night vs. Day**	
	Night (N = 21)	**Day (N = 11)**	**Night (N = 16)**	**Day (N = 8)**	***F* (1, 36)**	***P***	***F* (1, 54)**	***P***
IR	**37.48 (2.44) [33-40]**	**34.09 (6.44) [22-40]**	**33.38 (5.49) [17-40]**	**25.13 (10.53) [10-40]**	**9.363**	**0.004**	**8.598**	**0.005**
DR	**37.67 (2.85) [33-40]**	**33.00 (6.48) [20-40]**	**31.44 (5.93) [14-40]**	**21.88 (12.14) [9-40]**	**17.858**	**<0.001**	**9.795**	**0.003**
IR/DR	**1.01 (0.03) [0.94-1.11]**	**0.97 (0.07) [0.83-1.07]**	**0.94 (0.05) [0.82-1.00]**	**0.84 (0.15) [0.63-1.00]**	**24.118**	**<0.001**	**6.801**	**0.012**
MC	**37.10 (2.88) [33-40]**	**32.35 (7.79) [17-40]**	**30.13 (6.56) [14-40]**	**20.63 (12.50) [7-40]**	**19.027**	**<0.001**	**8.217**	**0.006**

Note. Immediate Recall is pre-sleep recall test score for nighttime study, and morning recall test score for daytime study. Delayed Recall is post-sleep recall test score for nighttime study, and evening recall test score for daytime study. Immediate Recall/Delayed Recall is the ratio of delayed/immediate recall test scores. Memory Consolidation is calculated as total same paired word recalled in both pre- and post-sleep. YA, Younger Adults; OA, Older Adults; IR, Immediate Recall; DR, Delayed Recall; MC, Memory Consolidation; M, mean; SD, standard deviation.

Table 6

Results rmANCOVA Comparing TGC MI and Theta and Gamma PSD in Fz and Pz Between Control and Memory EEG Overnights in Younger (N = 21) and Older (N = 16) Age Groups

Parameters	**YA**				**OA**			
	EEGm ***M* (*SD*)**	**EEGc** ***M* (*SD*)**	***F*** **(1, 18)**	***P***	**EEGm** ***M* (*SD*)**	**EEGc** ***M* (*SD*)**	***F*** **(1, 13)**	***P***
				Fz				
TGCs	-13.30 (1.08)	-13.14 (0.92)	1.760	0.201	-13.07 (1.26)	-13.00 (1.45)	0.212	0.653
TGCf	-13.28 (0.79)	-13.32 (0.75)	0.010	0.920	-12.96 (0.86)	-13.05 (1.49)	0.006	0.939
PSD								
Fixed θ	-3.71E-3 (1.13E-3)	-3.50E-3 (1.02E-3)	0.293	0.595	-2.30E-3 (1.66E-3)	-2.26E-3 (1.72E-3)	3.734	0.075
Slower γ	0.1073 (0.0121)	0.1073 (0.0140)	0.949	0.343	0.108 (0.011)	0.108 (0.012)	0.149	0.706
Faster γ	0.1415 (2.62E-3)	0.1412 (2.33E-3)	0.858	0.367	0.142 (2.66E-3)	0.141 (2.39E-3)	0.018	0.895
				Pz				
TGCs	-12.56 (1.25)	-12.91 (0.85)	0.321	0.578	-12.83 (1.33)	-12.44 (2.13)	0.124	0.731
TGCf	-12.87 (1.13)	-12.93 (0.87)	1.569	0.226	-13.20 (0.73)	-12.79 (2.29)	0.135	0.719
PSD								
Fixed θ	-2.12E-3 (9.72E-3)	-2.14 (1.05E-3)	0.082	0.777	-1.10E-3 (1.70E-3)	-1.02E-3 (1.88E-3)	4.047	0.065
Slower γ	0.10 (0.012)	0.10 (0.011)	0.100	0.756	0.10 (0.011)	0.10 (0.012)	0.010	0.923
Faster γ	0.14 (3.67E-3)	0.14 (2.12E-3)	1.163	0.295	0.14 (2.78E-3)	0.14 (2.33E-3)	0.036	0.853

Note. ANCOVA type III test. Sphericity assumed. *F*-statistic and *p*-value are for main effect in ANCOVA, being TGC and PSD, with memory and control nights are repeated measures, and age and sex as covariates. YA, Younger Adults; OA, Older Adults; EEGm, memory task night; EEGc, control task night; M, mean; SD, standard deviation; TGCs, fixed theta and slower gamma coupling; TGCf, fixed theta and faster gamma coupling; θ, theta, γ, gamma PSD, power spectral density.

Table 7

Results for ANCOVA Comparing TGC MI and Theta and Gamma PSD in Fz and Pz Between Younger (N = 21) and Older (N = 16) Age Groups during Memory EEG Overnight

Measures	**OA (*M* (*SD*))**	**YA (*M* (*SD*))**	***F* (1, 36)**	***P***
		Fz		
TGCs	-13.07 (1.26)	-13.30 (1.08)	1.536	0.224
TGCf	-12.96 (0.86)	-13.28 (0.79)	0.086	0.771
PSD				
Fixed θ	-2.30E-3 (1.66E-3)	-3.71E-3 (1.13E-3)	0.266	0.610
Slower γ	0.11 (0.01)	0.11 (0.01)	0.911	0.347
Faster γ	0.14 (2.66E-3)	0.14 (2.62E-3)	0.003	0.955
		Pz		
TGCs	-12.83 (1.33)	-12.56 (1.25)	0.131	0.720
TGCf	-13.20 (0.73)	-12.87 (1.13)	0.063	0.803
PSD				
Fixed θ	-1.10E-3 (1.70E-3)	-2.12E-3 (9.72E-3)	0.512	0.479
Slower γ	0.10 (0.01)	0.10 (0.01)	1.438	0.239
Faster γ	0.14 (2.78E-3)	0.14 (3.67E-3)	1.220	0.277

Note. ANCOVA type III test. *F*-statistic and *p*-value are for main effect in ANCOVA, being TGC and PSD, with age and sex as covariates. YA, younger adults; OA, older adults; M, mean; SD, standard deviation; TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

Table 8
Results for Pearson's r Correlations between TGC MI and Theta and Gamma PSD in Fz during Memory EEG Overnight with Memory Consolidation

Age group	Measure	*M* (*SD*)	Correlation	*P*
		Fz		
YA	TGCs	-13.30 (1.08)	0.192	0.405
(N = 21)	TGCf	**-13.28 (0.79)**	**0.558**	**0.009**
	Fixed θ PSD	-3.71E-3 (1.13E-3)	0.332	0.141
	Slower γ PSD	0.1073 (0.0121)	0.347	0.123
	Faster γ PSD	0.1415 (2.62E-3)	0.122	0.597
OA	TGCs	-13.07 (1.26)	0.004	0.987
(N =16)	TGCf	-12.96 (0.86)	0.139	0.609
	Fixed θ PSD	-2.30E-3 (1.66E-3)	-0.393	0.132
	Slower γ PSD	0.108 (0.011)	0.110	0.686
	Faster γ PSD	0.142 (2.66E-3)	0.307	0.247
		Pz		
YA	TGCs	-12.56 (1.25)	0.100	0.667
(N = 21)	TGCf	-12.87 (1.13)	0.044	0.848
	Fixed θ PSD	-2.12E-3 (9.72E-3)	-0.358	0.111
	Slower γ PSD	0.10 (0.012)	0.282	0.215
	Faster γ PSD	0.14 (3.67E-3)	0.197	0.393
OA	TGCs	-12.83 (1.33)	0.043	0.874
(N = 16)	TGCf	-13.20 (0.73)	-0.066	0.809
	Fixed θ PSD	-1.10E-3 (1.70E-3)	-0.357	0.175
	Slower γ PSD	0.10 (0.011)	0.038	0.888
	Faster γ PSD	0.14 (2.78E-3)	0.105	0.700

Note. Pearson's r Correlation is significant at the 0.05 level (2-tailed). Each test includes either a TGC or PSD value as one variable with the other variable being memory consolidation. Memory consolidation was calculated as total same paired word recalled in both pre- and post-sleep. YA, younger adults; OA, older adults; M, mean; SD, standard deviation; TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

Table 9
Results for TGC MI and Theta and Gamma PSD Predicting Memory Consolidation in Fz Between Age Groups with Covariates

Group	**Measure**	***F***	***Adj. R²***	***Δ R²***	***B* (*SE*)**	***β***	***P***
YA (N = 21)	TGCs MI	2.286	0.16	0.08	0.76 (0.56)	0.28	0.195
	TGCf MI	**6.903**	**0.47**	**0.34**	**2.14 (0.60)**	**0.59**	**0.002**
	Fixed θ PSD	3.154	0.24	0.15	984.62 (500.09)	0.39	0.065
	Slower γ PSD	2.086	0.14	0.06	58.94 (50.79)	0.25	0.262
	Faster γ PSD	1.530	0.07	<0.01	-46.16 (253.69)	-0.04	0.858
OA (N = 16)	TGCs MI	5.120	0.45	0.02	-0.65 (1.03)	-0.13	0.537
	TGCf MI	4.845	0.44	<0.01	0.26 (1.49)	0.03	0.867
	Fixed θ PSD	4.988	0.44	0.01	489.45 (1033.20)	0.12	0.644
	Slower γ PSD	6.169	0.51	0.06	161.14 (119.10)	0.27	0.201
	Faster γ PSD	4.823	0.43	<0.01	6.64 (534.35)	<0.01	0.990

Note. *F*-statistic, R^2, *Adj.* R^2, and $R^2\Delta$ are for the regression model with TGC and PSD types, age, and sex as IVs. *B (SE)*, *β*, and *p*-value are for the TGC or PSD predictor in model 2. DV is memory consolidation, calculated as total same paired word recalled in both pre- and post-sleep. F (3, 17) for YA. F (3, 12) for OA. R^2, proportion of explained variance, Adj. R^2, proportion of explained variance considering error inflation, ΔR^2, change in proportion of explained variance by IV alone, YA, younger adults; OA, older adults; B, unstandardized coefficient; SE, standard error; β, standardized coefficient; MI, modulation index TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

Table 10
Results for TGC MI and Theta and Gamma PSD Predicting Memory Consolidation in Pz Between Age Groups with Covariates

Group	**Measure**	***F***	***Adj. R^2***	***Δ R^2***	***B* (*SE*)**	***β***	***P***
YA (N = 21)	TGCs MI	1.764	0.10	0.03	0.38 (0.49)	0.16	0.455
	TGCf MI	1.548	0.08	<0.01	0.15 (0.55)	0.06	0.789
	Fixed θ PSD	**3.566**	**0.28**	**0.18**	**1250.29 (567.69)**	**0.42**	**0.042**
	Slower γ PSD	1.890	0.12	0.04	48.54 (51.65)	0.20	0.361
	Faster γ PSD	1.57	0.08	<0.01	54.97 (176.49)	0.07	0.759
OA (N = 16)	TGCs MI	4.940	0.44	<0.01	0.38 (0.95)	0.08	0.697
	TGCf MI	4.928	0.44	0.01	0.67 (1.77)	0.07	0.712
	Fixed θ PSD	4.837	0.43	<0.01	129.94 (932.76)	0.03	0.892
	Slower γ PSD	5.497	0.47	0.03	124.52 (130.03)	0.21	0.357
	Faster γ PSD	4.828	0.43	<0.01	-40.72 (470.45)	-0.02	0.932

Note. *F*-statistic, R^2, *Adj.* R^2, and $R^2\Delta$ are for the regression model with TGC and PSD types, age, and sex as IVs. *B (SE)*, *β*, and *p*-value are for the TGC or PSD predictor in model 2. DV is memory consolidation, calculated as total same paired word recalled in both pre- and post-sleep. F (3, 17) for YA. F (3, 12) for OA. R^2, proportion of explained variance, Adj. R^2, proportion of explained variance considering error inflation, Δ R^2, change in proportion of explained variance by IV alone, YA, younger adults; OA, older adults; B, unstandardized coefficient; SE, standard error; β, standardized coefficient; MI, modulation index TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

FIGURES

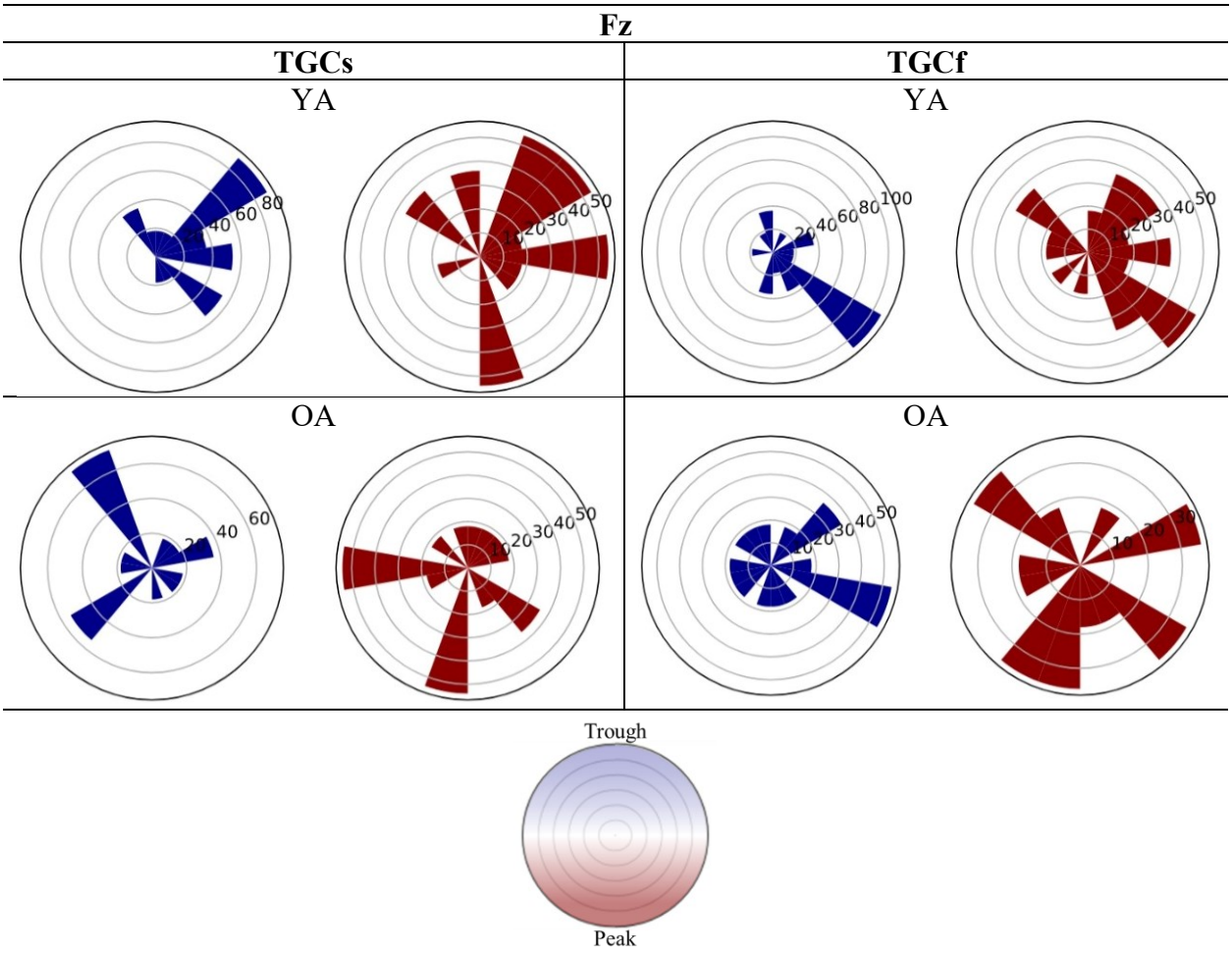

Figure 3. In channel Fz in YAs (top) and OAs (bottom), polar plots for TGCs and TGCf in both EEGc (blue) and EEGm (red). Vector length in axis rings reflect gamma amplitude strength, and direction reflects degrees of theta cycle. The top and bottom of each plot corresponds to theta trough and peak, respectively (bottom). YA, younger adult; OA, older adult; TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; EEGc, control task EEG night; EEGm, memory task EEG night.

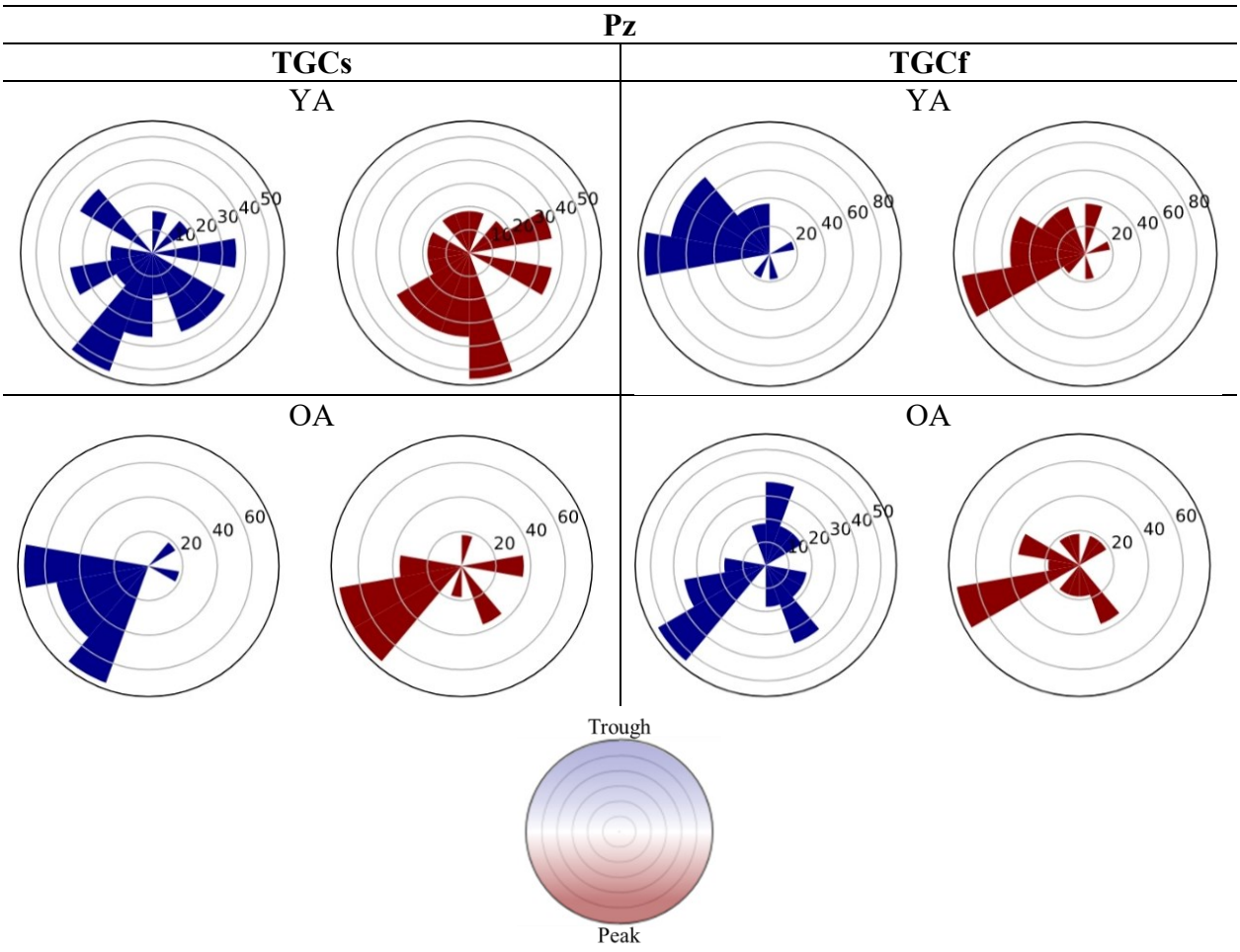

Figure 4. In channel Pz in YAs (top) and OAs (bottom), polar plots for TGCs and TGCf in both EEGc (blue) and EEGm (red). Vector length in axis rings reflect gamma amplitude strength, and direction reflects degrees of theta cycle. The top and bottom of each plot corresponds to theta trough and peak, respectively (bottom). YA, younger adult; OA, older adult; TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; EEGc, control task EEG night; EEGm, memory task EEG night.

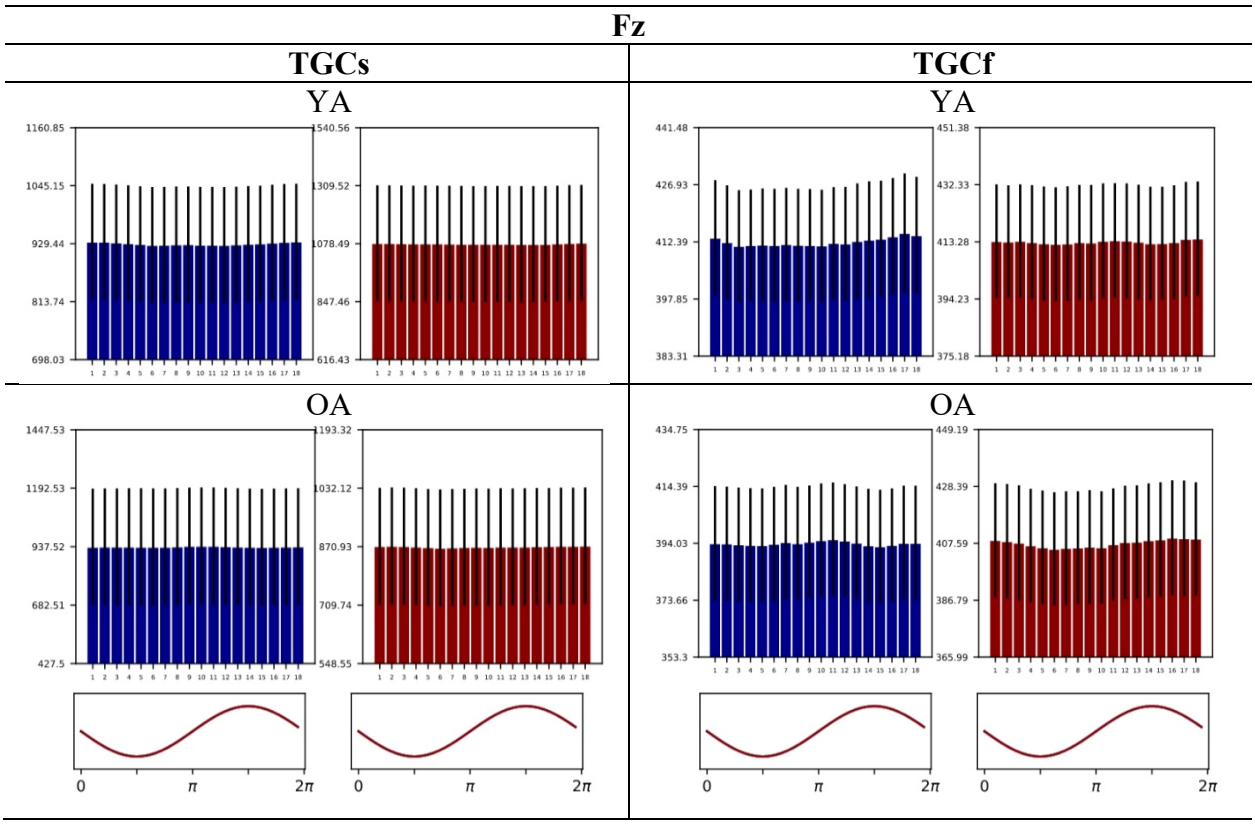

Figure 5. In channel Fz in YAs (top) and OAs (bottom) mean gamma amplitude plots with theta phase bins for both EEGc (blue) and EEGm (red). 18 phase bins in total, corresponding to sinusoidal theta wave (bottom). YA, younger adult; OA, older adult; TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; EEGc, control task EEG night; EEGm, memory task EEG night.

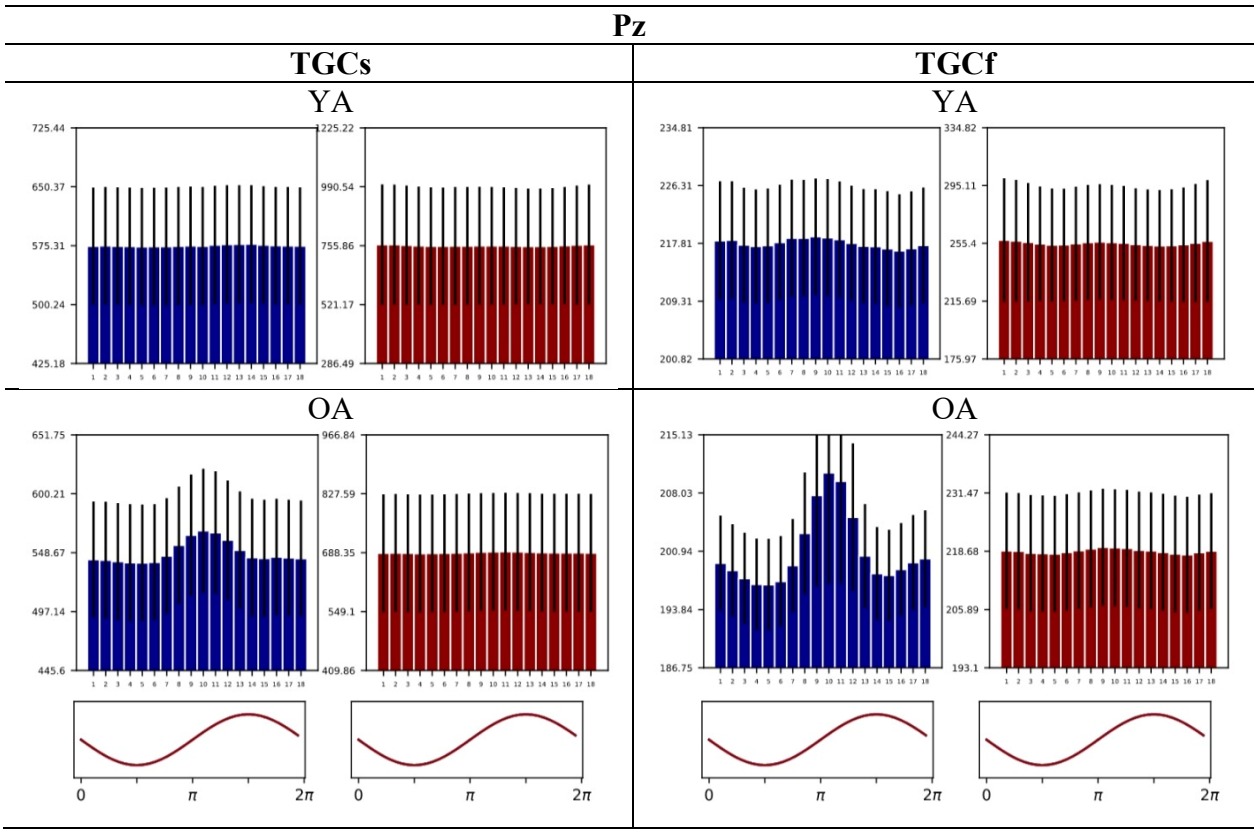

Figure 6. In channel Pz in YAs (top) and OAs (bottom) mean gamma amplitude plots with theta phase bins for both EEGc (blue) and EEGm (red). 18 phase bins in total, corresponding to sinusoidal theta wave (bottom). YA, younger adult; OA, older adult; TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; EEGc, control task EEG night; EEGm, memory task EEG night.

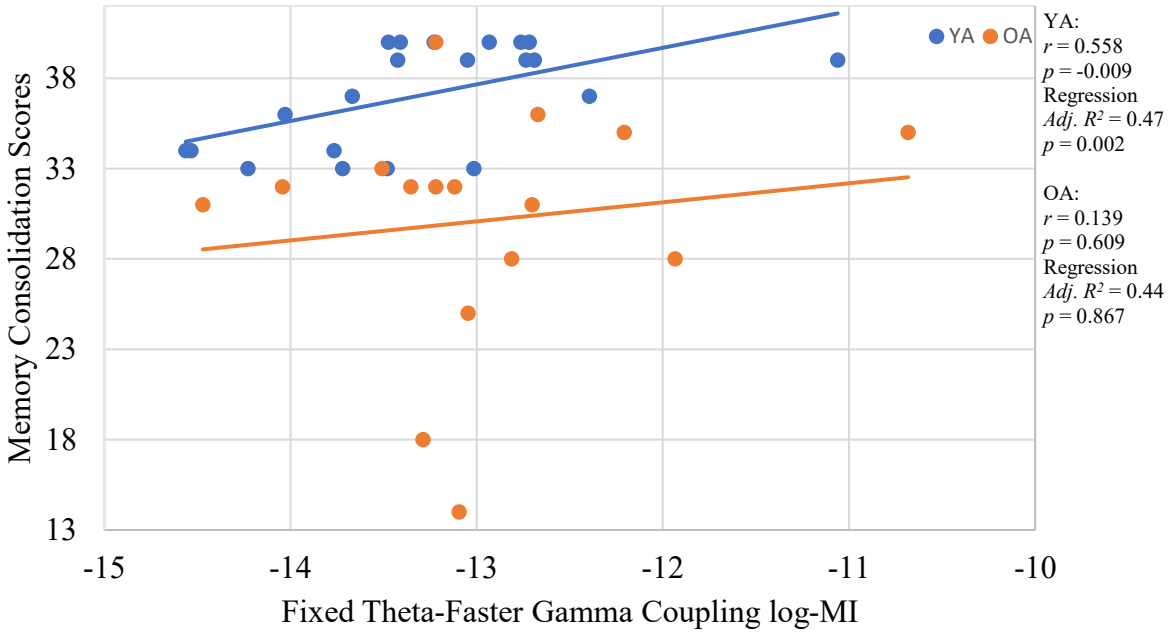

Figure 7. In Fz, correlations between fixed theta-faster gramma coupling (TGCf) log-MI and memory consolidation in YAs (N = 21) (r = 0.558, p = 0.009) and OAs (N = 16) (r = 0.139, p = 0.609). Model 2 of hierarchical multiple linear regression shows a significant prediction in YAs (*Adj.* R^2 = 0.47, ΔR^2 = 0.34, β = 0.59, F (3, 17) = 6.903, p = 0.002), but not OAs (*Adj.* R^2 = 0.44, ΔR^2 = <0.01, β = 0.03, F (3, 12) = 4.845, p = 0.867). Top right legend shows r and p values for Pearson's r correlation, and *Adj.* R^2 and p values below, for model 2 of multiple linear regression, for both YA and OA. Log-MI, log-transformed modulation index; Adj. R^2, adjusted R^2; YA, younger adult; OA, older adults.

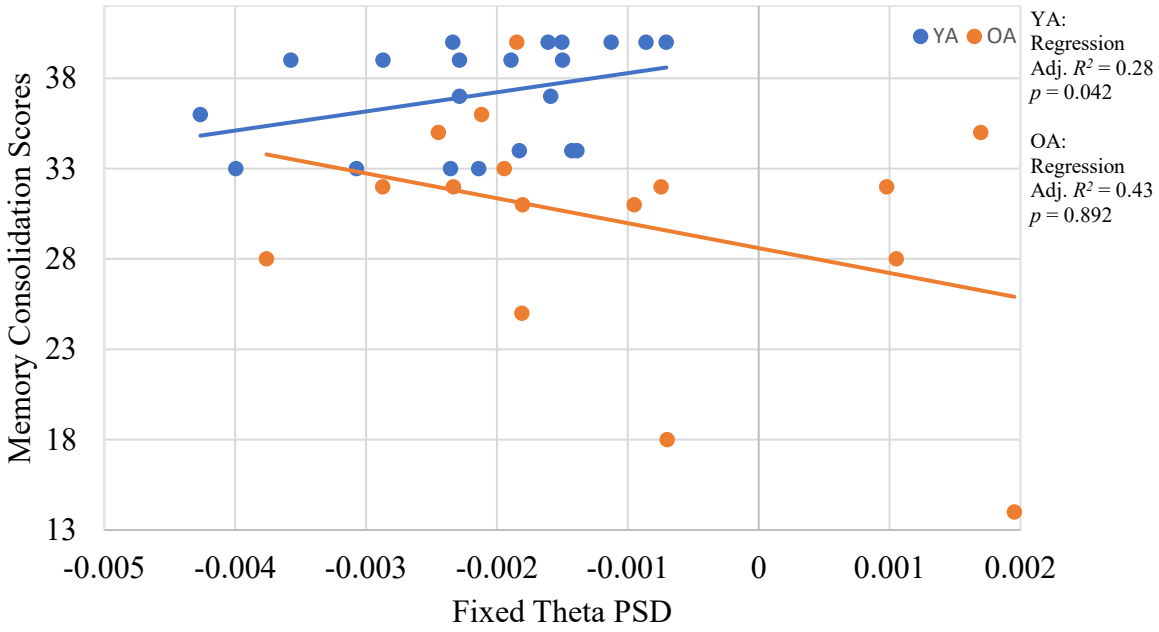

Figure 8. In Pz, model 2 of hierarchical multiple linear regression for fixed theta PSD shows a significant prediction in YAs (*Adj.* $R^2 = 0.28$, $\Delta R^2 = 0.18$, $\beta = 0.42$, F (3, 17) = 3.566, $p = 0.042$), but not OAs (*Adj.* $R^2 = 0.43$, $\Delta R^2 = <0.01$, $\beta = 0.03$, F (3, 12) = 4.837, $p = 0.892$). Top right legend shows *Adj.* R^2 and p values for model 2 of multiple linear regression, for both YA and OA. PSD, power spectral density; Adj. R^2, adjusted R^2; YA, younger adult; OA, older adults.

SUPPLEMENTARY

Table S1

Results rmANCOVA Comparing Adapted TGC MI and Theta and Gamma PSD in Fz and all Parameters Cz Between Control and Memory EEG Overnights in Younger (N = 21) and Older (N = 16) Age Groups

Parameters	**YA**				**OA**			
	EEGm ***M* (*SD*)**	**EEGc** ***M* (*SD*)**	***F*** **(1, 18)**	***P***	**EEGm** ***M* (*SD*)**	**EEGc** ***M* (*SD*)**	***F*** **(1, 13)**	***P***
				Fz				
aTGCs	-13.41 (1.29)	-13.44 (0.98)	2.959	0.103	-13.39 (1.25)	-13.18 (1.83)	1.723	0.212
aTGCf	-13.27 (0.73)	-13.32 (0.91)	0.130	0.722	-13.03 (0.77)	-13.09 (1.47)	0.742	0.405
PSD								
Adapted θ*	**-3.32E-3 (1.27E-3)**	**-2.79E-3 (1.21E-3)**	**6.974**	**0.017**	-1.92E-3 (1.73E-3)	-1.73E-3 (1.55E-3)	0.860	0.371
				Cz				
TGCs	-13.09 (1.03)	-13.01 (1.22)	2.175	0.158	-12.53 (0.72)	-12.84 (0.69)	0.278	0.607
TGCf	-13.18 (0.88)	-13.24 (0.48)	0.011	0.919	-13.09 (0.81)	-13.30 (0.68)	0.001	0.982
aTGCs	-12.99 (0.98)	-13.15 (1.32)	3.226	0.089	-12.72 (1.05)	-13.24 (0.82)	0.535	0.477
aTGCf	-13.25 (0.94)	-13.29 (0.77)	0.050	0.826	-13.15 (0.89)	-13.26 (0.49)	0.123	0.732
PSD								
Fixed θ**	-4.05E-3 (1.16E-3)	-4.00E-3 (1.26E-3)	1.520	0.233	**-2.47E-3 (1.74E-3)**	**-2.45E-3 (1.89E-3)**	**6.347**	**0.026**
Adapted θ	-3.96E-3 (1.57E-3)	-3.82E-3 (1.50E-3)	2.968	0.102	-2.26E-3 (1.78E-3)	-2.24E-3 (1.68E-3)	0.127	0.728
Slower γ	0.105 (0.014)	0.103 (0.014)	0.571	0.459	0.10 (0.013)	0.11 (0.014)	0.126	0.729
Faster γ	0.140 (2.74E-3)	0.140 (2.13E-3)	2.640	0.122	0.14 (2.38E-3)	0.14 (2.92E-3)	0.353	0.563

Note. ANCOVA type III test. Sphericity assumed. *F*-statistic and *p*-value are for main effect in ANCOVA, being TGC and PSD, with memory and control nights are repeated measures, and age and sex as covariates. YA, Younger Adults; OA, Older Adults; EEGm, memory task night; EEGc, control task night; M, mean; SD, standard deviation; TGCs, fixed theta and slower gamma coupling; TGCf, fixed theta and faster gamma coupling; aTGCs, adapted theta and slower gamma coupling, aTGCf, adapted theta and faster gamma coupling, θ, theta, γ, gamma PSD, power spectral density.

* YA: Night type interaction with age covariate (F (1, 18) = 5.073, p = 0.038)
**OA: Night type interaction with age covariate (F (1, 13) = 7.837, p = 0.015)

Table S2

Results rmANCOVA Comparing TGC MI and Theta and Gamma PSD in T3 and T4 Between Control and Memory EEG Overnights in Younger (N = 21) and Older (N = 16) Age Groups

Parameters	**YA**				**OA**			
	EEGm ***M* (*SD*)**	**EEGc** ***M* (*SD*)**	***F*** **(1, 18)**	***P***	**EEGm** ***M* (*SD*)**	**EEGc** ***M* (*SD*)**	***F*** **(1, 13)**	***P***
				T3				
TGCs	-12.76 (0.98)	-12.92 (0.95)	2.498	0.131	-12.80 (1.45)	-12.57 (1.72)	0.011	0.916
TGCf	-12.82 (1.20)	-12.70 (0.84)	0.362	0.555	-12.65 (0.78)	-12.60 (1.58)	0.320	0.581
PSD								
Fixed θ	1.46E-3 (1.09E-3)	1.66E-3 (1.13E-3)	0.001	0.978	1.81E-3 (1.65E-3)	2.01E-3 (1.74E-3)	0.348	0.565
Slower γ	0.12 (0.011)	0.12 (0.011)	0.155	0.698	0.11 (0.011)	0.11 (0.011)	0.756	0.400
Faster γ	0.14 (2.67E-3)	0.14 (2.56E-3)	0.245	0.627	0.14 (2.55E-3)	0.14 (2.82E-3)	0.208	0.656
				T4				
TGCs	-12.99 (1.10)	-12.88 (0.94)	0.015	0.903	-12.95 (0.79)	-12.45 (1.18)	1.103	0.313
TGCf	-12.80 (0.90)	-13.08 (0.85)	1.519	0.234	-12.78 (0.91)	-12.80 (1.03)	0.075	0.788
PSD								
Fixed θ	1.54E-3 (1.15E-3)	1.57E-3 (1.16E-3)	0.017	0.898	1.77E-3 (1.61E-3)	1.80E-3 (1.45E-3)	1.088	0.316
Slower γ	0.12 (0.011)	0.11 (0.010)	2.307	0.146	0.11 (0.010)	0.11 (0.011)	0.487	0.498
Faster γ	0.14 (2.09E-3)	0.14 (2.05E-3)	0.584	0.455	0.14 (2.68E-3)	0.14 (2.85E-3)	0.227	0.642

Note. ANCOVA type III test. Sphericity assumed. *F*-statistic and *p*-value are for main effect in ANCOVA, being TGC and PSD, with memory and control nights are repeated measures, and age and sex as covariates. YA, Younger Adults; OA, Older Adults; EEGm, memory task night; EEGc, control task night; M, mean; SD, standard deviation; TGCs, fixed theta and slower gamma coupling; TGCf, fixed theta and faster gamma coupling; θ, theta, γ, gamma PSD, power spectral density.

Table S3

Results for ANCOVA Comparing Adapted TGC MI and Theta and Gamma PSD in Fz and all Measures Cz Between Younger (N = 21) and Older (N = 16) Age Groups during Memory EEG Overnight

Measures	**OA (*M* (*SD*))**	**YA (*M* (*SD*))**	***F* (1, 36)**	***P***
		Fz		
aTGCs	-13.39 (1.25)	-13.41 (1.29)	0.481	0.493
aTGCf	-13.03 (0.77)	-13.27 (0.73)	0.891	0.352
PSD				
Adapted θ	-1.92E-3 (1.73E-3)	-3.32E-3 (1.27E-3)	0.090	0.766
		Cz		
TGCs	-12.53 (0.72)	-13.09 (1.03)	1.389	0.247
TGCf	-13.09 (0.81)	-13.18 (0.88)	0.719	0.403
aTGCs	-12.72 (1.05)	-12.99 (0.98)	1.716	0.199
aTGCf	-13.15 (0.89)	-13.25 (0.94)	0.178	0.676
PSD				
Fixed θ	-2.47E-3 (1.74E-3)	-4.05E-3 (1.16E-3)	0.222	0.640
Adapted θ	-2.26E-3 (1.78E-3)	-3.96E-3 (1.57E-3)	0.418	0.522
Slower γ	0.10 (0.013)	0.105 (0.014)	1.347	0.254
Faster γ	0.14 (2.38E-3)	0.140 (2.74E-3)	0.499	0.485

Note. ANCOVA type III test. *F*-statistic and *p*-value are for main effect in ANCOVA, being TGC and PSD, with age and sex as covariates. YA, younger adults; OA, older adults; M, mean; SD, standard deviation; TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; aTGCs, adapted theta-slower gamma coupling, aTGCf, adapted theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

Table S4

Results for ANCOVA Comparing TGC MI and Theta and Gamma PSD in T3 and T4 Between Younger (N = 21) and Older (N = 16) Age Groups during Memory EEG Overnight

Measures	**OA (*M* (*SD*))**	**YA (*M* (*SD*))**	***F* (1, 36)**	***P***
		T3		
TGCs	-12.80 (1.45)	-12.76 (0.98)	0.385	0.539
TGCf	-12.65 (0.78)	-12.82 (1.20)	0.014	0.907
PSD				
Fixed θ	1.81E-3 (1.65E-3)	1.46E-3 (1.09E-3)	0.006	0.940
Slower γ	0.11 (0.011)	0.12 (0.011)	0.803	0.377
Faster γ	0.14 (2.55E-3)	0.14 (2.67E-3)	2.855	0.100
		T4		
TGCs	-12.95 (0.79)	-12.99 (1.10)	0.035	0.854
TGCf	-12.78 (0.91)	-12.80 (0.90)	0.163	0.689
PSD				
Fixed θ	1.77E-3 (1.61E-3)	1.54E-3 (1.15E-3)	0.103	0.750
Slower γ	0.11 (0.010)	0.12 (0.011)	0.940	0.339
Faster γ	0.14 (2.68E-3)	0.14 (2.09E-3)	2.251	0.143

Note. ANCOVA type III test. *F*-statistic and *p*-value are for main effect in ANCOVA, being TGC and PSD, with age and sex as covariates. YA, younger adults; OA, older adults; M, mean; SD, standard deviation; TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

Table S5

Results for Pearson's r Correlations between Adapted TGC MI and Theta PSD in Fz and all Measures in Cz during Memory EEG Overnight with Memory Consolidation

Age group	Measure	*M* (*SD*)	Correlation	*P*
		Fz		
YA	aTGCs	-13.41 (1.29)	0.258	0.258
(N = 21)	aTGCf	**-13.27 (0.73)**	**0.475**	**0.030**
	Adapted θ PSD	-3.32E-3 (1.27E-3)	0.144	0.532
OA	aTGCs	-13.39 (1.25)	-0.004	0.989
(N = 16)	aTGCf	-13.03 (0.77)	-0.036	0.895
	Adapted θ PSD	-1.92E-3 (1.73E-3)	-0.316	0.234
		Cz		
YA	TGCs	-13.09 (1.03)	0.010	0.966
(N = 21)	TGCf	-13.18 (0.88)	0.018	0.940
	aTGCs	-12.99 (0.98)	-0.083	0.722
	aTGCf	-13.25 (0.94)	-0.020	0.931
	Fixed θ PSD	-4.05E-3 (1.16E-3)	0.355	0.114
	Adapted θ PSD	-3.96E-3 (1.57E-3)	0.203	0.378
	Slower γ PSD	0.105 (0.014)	0.355	0.115
	Faster γ PSD	0.140 (2.74E-3)	0.278	0.222
OA	TGCs	**-12.53 (0.72)**	**0.617**	**0.011**
(N =16)	TGCf	-13.09 (0.81)	0.078	0.774
	aTGCs	-12.72 (1.05)	0.352	0.181
	aTGCf	-13.15 (0.89)	0.072	0.791
	Fixed θ PSD	-2.47E-3 (1.74E-3)	-0.478	0.061
	Adapted θ PSD	-2.26E-3 (1.78E-3)	-0.393	0.132
	Slower γ PSD	0.10 (0.013)	0.109	0.688
	Faster γ PSD	*0.14 (2.38E-3)*	*0.496*	*0.051*

Note. Pearson's r Correlation is significant at the 0.05 level (2-tailed). Each test includes either a TGC or PSD value as one variable with the other variable being memory consolidation. Memory consolidation was calculated as total same paired word recalled in both pre- and post-sleep. YA, younger adults; OA, older adults; M, mean; SD, standard deviation; aTGCs, adapted theta-slower gamma coupling, aTGCf, adapted theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

Table S6

Results for Pearson's r Correlations between TGC MI and Theta and Gamma PSD in T3 during Memory EEG Overnight with Memory Consolidation

Age group	**Measure**	***M* (*SD*)**	**Correlation**	***P***
		T3		
YA	TGCs	-12.76 (0.98)	0.163	0.481
(N = 21)	TGCf	-12.82 (1.20)	-0.091	0.696
	Fixed θ PSD	*1.46E-3 (1.09E-3)*	*0.426*	*0.054*
	Slower γ PSD	0.12 (0.011)	0.339	0.133
	Faster γ PSD	0.14 (2.67E-3)	0.315	0.164
OA	TGCs	-12.80 (1.45)	0.077	0.776
(N =16)	TGCf	-12.65 (0.78)	-0.176	0.515
	Fixed θ PSD	1.81E-3 (1.65E-3)	-0.338	0.200
	Slower γ PSD	0.11 (0.011)	0.253	0.345
	Faster γ PSD	**0.14 (2.55E-3)**	**0.531**	**0.034**
		T4		
YA	TGCs	-12.99 (1.10)	0.394	0.077
(N = 21)	TGCf	-12.80 (0.90)	0.289	0.204
	Fixed θ PSD	1.54E-3 (1.15E-3)	0.344	0.127
	Slower γ PSD	0.12 (0.011)	0.327	0.147
	Faster γ PSD	0.14 (2.09E-3)	0.315	0.165
OA	TGCs	-12.95 (0.79)	-0.167	0.537
(N = 16)	TGCf	-12.78 (0.91)	0.060	0.825
	Fixed θ PSD	1.77E-3 (1.61E-3)	-0.377	0.150
	Slower γ PSD	0.11 (0.010)	0.206	0.445
	Faster γ PSD	0.14 (2.68E-3)	0.359	0.173

Note. Pearson's r Correlation is significant at the 0.05 level (2-tailed). Each test includes either a TGC or PSD value as one variable with the other variable being memory consolidation. Memory consolidation was calculated as total same paired word recalled in both pre- and post-sleep. YA, younger adults; OA, older adults; M, mean; SD, standard deviation; TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

Table S7

Results for Adapted TGC MI and Theta PSD Predicting Memory Consolidation in Fz Between Age Groups with Covariates

Group	**Measure**	***F***	***Adj. R²***	***Δ R²***	***B* (*SE*)**	***β***	***P***
YA	aTGCs MI	2.581	0.19	0.10	0.74 (0.47)	0.33	0.131
(N = 21)	aTGCf MI	**4.768**	**0.36**	**0.25**	**1.97 (0.71)**	**0.50**	**0.013**
	Adapted θ PSD	2.095	0.14	0.06	562.44 (480.82)	0.25	0.258
OA	aTGCs MI	4.823	0.43	<0.01	-0.01 (1.03)	<-0.01	0.992
(N = 16)	aTGCf MI	4.970	0.43	0.01	-0.77 (1.72)	-0.09	0.662
	Adapted θ PSD	5.051	0.45	0.01	498.39 (894.00)	0.13	0.587

Note. *F*-statistic, R^2, *Adj.* R^2, and $R^2\Delta$ are for the regression model with TGC and PSD types, age, and sex as IVs. *B (SE)*, *β*, and *p*-value are for the TGC or PSD predictor in model 2. DV is memory consolidation, calculated as total same paired word recalled in both pre- and post-sleep. F (3, 17) for YA. F (3, 12) for OA. R^2, proportion of explained variance, Adj. R^2, proportion of explained variance considering error inflation, ΔR^2, change in proportion of explained variance by IV alone, YA, younger adults; OA, older adults; B, unstandardized coefficient; SE, standard error; β, standardized coefficient; MI, modulation index; aTGCs, adapted theta-slower gamma coupling, aTGCf, adapted theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

Table S8

Results for TGC MI and Theta and Gamma PSD Predicting Memory Consolidation in Cz Between Age Groups with Covariates

Group	**Measure**	***F***	***Adj. R²***	***Δ R²***	***B* (*SE*)**	***β***	***P***
YA (N = 21)	TGCs MI	1.785	0.11	0.03	-0.51 (0.63)	-0.18	0.436
	TGCf MI	1.578	0.22	0.01	0.27 (0.71)	0.08	0.708
	aTGCs MI	1.958	0.13	0.05	-0.65 (0.64)	-0.22	0.321
	aTGCf MI	1.553	0.08	<0.01	0.20 (0.67)	0.07	0.772
	Fixed θ PSD	*3.360*	*0.26*	*0.16*	*1007.10 (482.13)*	*0.41*	*0.052*
	Adapted θ PSD	1.913	0.12	0.04	381.61 (393.74)	0.21	0.346
	Slower γ PSD	2.182	0.15	0.07	54.62 (43.52)	0.27	0.226
	Faster γ PSD	0.577	-0.07	0.02	161.53 (278.89)	0.16	0.570
OA (N = 16)	TGCs MI	5.739	0.49	0.55	2.39 (2.14)	0.26	0.286
	TGCf MI	6.750	0.54	0.08	2.43 (1.50)	0.30	0.131
	aTGCs MI	4.846	0.44	<0.01	0.24 (1.38)	0.04	0.862
	aTGCf MI	5.944	0.50	0.05	1.70 (1.40)	0.23	0.240
	Fixed θ PSD	4.834	0.43	<0.01	-123.43 (997.76)	-0.03	0.904
	Adapted θ PSD	4.816	0.44	<0.01	202.96 (892.98)	0.06	0.824
	Slower γ PSD	5.829	0.49	0.05	128.15 (109.55)	0.25	0.265
	Faster γ PSD	5.796	0.49	0.05	643.84 (559.60)	0.23	0.272

Note. *F*-statistic, R^2, *Adj.* R^2, and $R^2\Delta$ are for the regression model with TGC and PSD types, age, and sex as IVs. *B (SE)*, *β*, and *p*-value are for the TGC or PSD predictor in model 2. DV is memory consolidation, calculated as total same paired word recalled in both pre- and post-sleep. F (3, 17) for YA. F (3, 12) for OA. R^2, proportion of explained variance, Adj. R^2, proportion of explained variance considering error inflation, ΔR^2, change in proportion of explained variance by IV alone, YA, younger adults; OA, older adults; B, unstandardized coefficient; SE, standard error; β, standardized coefficient; MI, modulation index TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; aTGCs, adapted theta-slower gamma coupling, aTGCf, adapted theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

Table S9

Results for TGC MI and Theta and Gamma PSD Predicting Memory Consolidation in T3 Between Age Groups with Covariates

Group	**Measure**	***F***	***Adj. R²***	***Δ R²***	***B* (*SE*)**	***β***	***P***
YA (N = 21)	TGCs MI	1.760	0.10	0.03	0.48 (0.63)	0.16	0.458
	TGCf MI	1.552	0.08	0.09	-0.15 (0.52)	-0.06	0.774
	Fixed θ PSD	*3.384*	*0.26*	*0.16*	*1070.16 (509.07)*	*0.40*	*0.051*
	Slower γ PSD	1.966	0.13	0.05	57.97 (56.16)	0.23	0.317
	Faster γ PSD	0.457	-0.09	<0.01	-12.56 (276.97)	-0.01	0.964
OA (N = 16)	TGCs MI	4.835	0.43	<0.01	-0.11 (0.89)	-0.03	0.901
	TGCf MI	5.018	0.45	0.01	-0.84 (1.64)	-0.10	0.616
	Fixed θ PSD	5.097	0.45	0.01	685.68 (959.42)	0.15	0.553
	Slower γ PSD	6.186	0.51	0.06	157.08 (115.37)	0.26	0.198
	Faster γ PSD	5.589	0.48	0.04	581.81 (569.93)	0.23	0.327

Note. *F*-statistic, R^2, *Adj.* R^2, and $R^2\Delta$ are for the regression model with TGC and PSD types, age, and sex as IVs. *B (SE)*, *β*, and *p*-value are for TGC type. DV is memory consolidation, calculated as total same paired word recalled in both pre- and post-sleep. F (3, 17) for YA. F (3, 12) for OA. R^2, proportion of explained variance, Adj. R^2, proportion of explained variance considering error inflation, Δ R^2, change in proportion of explained variance by IV alone, YA, younger adults; OA, older adults; B, unstandardized coefficient; SE, standard error; β, standardized coefficient; MI, modulation index TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; aTGCs, adapted theta-slower gamma coupling, aTGCf, adapted theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

Table S10

Results for TGC MI and Theta and Gamma PSD Predicting Memory Consolidation in T4 Between Age Groups with Covariates

Group	**Measure**	***F***	***Adj. R^2***	***Δ R^2***	***B* (*SE*)**	***β***	***P***
YA (N = 21)	TGCs MI	*3.248*	*0.25*	*0.15*	*1.04 (0.51)*	*0.40*	*0.059*
	TGCf MI	2.347	0.17	0.08	0.93 (0.66)	0.29	0.179
	Fixed θ PSD	2.044	0.14	0.05	602.16 (538.68)	0.24	0.279
	Slower γ PSD	0.460	-0.09	<0.01	6.77 (68.14)	0.03	0.922
	Faster γ PSD	0.505	-0.08	<0.01	-127.31 (345.58)	-0.10	0.717
OA (N = 16)	TGCs MI	4.849	0.44	<0.01	-0.33 (1.71)	-0.04	0.853
	TGCf MI	4.824	0.43	<0.01	0.05 (1.40)	0.01	0.971
	Fixed θ PSD	4.853	0.44	<0.01	194.01 (962.76)	0.05	0.844
	Slower γ PSD	6.011	0.50	0.05	161.40 (126.96)	0.25	0.228
	Faster γ PSD	1.868	0.12	0.04	54.07 (59.31)	0.20	0.375

Note. *F*-statistic, R^2, *Adj.* R^2, and $R^2\Delta$ are for the regression model with TGC and PSD types, age, and sex as IVs. *B (SE)*, *β*, and *p*-value are for the TGC or PSD predictor in model 2. DV is memory consolidation, calculated as total same paired word recalled in both pre- and post-sleep. F (3, 17) for YA. F (3, 12) for OA. R^2, proportion of explained variance, Adj. R^2, proportion of explained variance considering error inflation, Δ R^2, change in proportion of explained variance by IV alone, YA, younger adults; OA, older adults; B, unstandardized coefficient; SE, standard error; β, standardized coefficient; MI, modulation index TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; aTGCs, adapted theta-slower gamma coupling, aTGCf, adapted theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

Table S11

Results for rmANCOVA Comparing TGC MI and PSD in Fz and Pz Between Control and Memory EEG Overnights in Younger (N = 21) and Older (N = 16) Adults with Covariates Age, Sex, & AHI

Parameters	**YA**				**OA**			
	EEGm ***M* (*SD*)**	**EEGc** ***M* (*SD*)**	***F*** **(1, 17)**	***P***	**EEGm** ***M* (*SD*)**	**EEGc** ***M* (*SD*)**	***F*** **(1, 12)**	***P***
				Fz				
TGCs	-13.30 (1.08)	-13.14 (0.92)	1.734	0.205	-13.07 (1.26)	-13.00 (1.45)	0.230	0.640
TGCf	-13.28 (0.79)	-13.32 (0.75)	0.009	0.924	-12.96 (0.86)	-13.05 (1.49)	0.029	0.867
aTGCs	-13.41 (1.29)	-13.44 (0.98)	2.985	0.102	-13.39 (1.25)	-13.18 (1.83)	1.685	0.219
aTGCf	-13.27 (0.73)	-13.32 (0.91)	0.133	0.720	-13.03 (0.77)	-13.09 (1.47)	0.678	0.426
PSD								
Fixed θ	-3.71E-3 (1.13E-3)	-3.50E-3 (1.02E-3)	0.275	0.607	-2.30E-3 (1.66E-3)	-2.26E-3 (1.72E-3)	3.257	0.096
Adapted θ*	**-3.32E-3 (1.27E-3)**	**-2.79E-3 (1.21E-3)**	**6.617**	**0.020**	-1.92E-3 (1.73E-3)	-1.73E-3 (1.55E-3)	0.936	0.352
Slower γ	0.1073 (0.0121)	0.1073 (0.0140)	0.897	0.357	0.108 (0.011)	0.108 (0.012)	0.223	0.646
Faster γ	0.1415 (2.62E-3)	0.1412 (2.33E-3)	0.820	0.378	0.142 (2.66E-3)	0.141 (2.39E-3)	0.032	0.861
				Pz				
TGCs**	-12.56 (1.25)	-12.91 (0.85)	0.384	0.543	-12.83 (1.33)	-12.44 (2.13)	0.064	0.804
TGCf	-12.87 (1.13)	-12.93 (0.87)	1.481	0.240	-13.20 (0.73)	-12.79 (2.29)	0.206	0.658
PSD								
Fixed θ	-2.12E-3 (9.72E-3)	-2.14 (1.05E-3)	0.081	0.779	-1.10E-3 (1.70E-3)	-1.02E-3 (1.88E-3)	3.602	0.082
Slower γ	0.10 (0.012)	0.10 (0.011)	0.095	0.761	0.10 (0.011)	0.10 (0.012)	0.020	0.889
Faster γ	0.14 (3.67E-3)	0.14 (2.12E-3)	1.104	0.308	0.14 (2.78E-3)	0.14 (2.33E-3)	0.032	0.862

Note. ANCOVA type III test. Sphericity assumed. *F*-statistic and *p*-value are for main effect in ANCOVA, being TGC and PSD, with memory and control nights are repeated measures, and age, sex, and AHI as covariates. AHI, apnea-hypopnea index; YA, Younger Adults; OA, Older Adults; EEGm, memory task night; EEGc, control task night; M, mean; SD, standard deviation; TGCs, fixed theta and slower gamma coupling; TGCf, fixed theta and faster gamma coupling; θ, theta, γ, gamma PSD, power spectral density.

* Night Type – Age interaction for YA: F (1, 17) = 4.782, p = 0.043

** Night Type – AHI interaction for YA: F (1, 17) = 6.572, p = 0.020

Table S12

Results for rmANCOVA Comparing TGC MI and PSD in Cz Between Control and Memory EEG Overnights in Younger (N = 21) and Older (N = 16) Adults with Covariates Age, Sex, & AHI

Parameters	**YA**				**OA**			
	EEGm ***M* (*SD*)**	**EEGc** ***M* (*SD*)**	***F*** **(1, 17)**	***P***	**EEGm** ***M* (*SD*)**	**EEGc** ***M* (*SD*)**	***F*** **(1, 12)**	***P***
				Cz				
TGCs	-13.09 (1.03)	-13.01 (1.22)	2.091	0.166	-12.53 (0.72)	-12.84 (0.69)	0.158	0.698
TGCf	-13.18 (0.88)	-13.24 (0.48)	0.009	0.926	-13.09 (0.81)	-13.30 (0.68)	0.029	0.867
aTGCs	-12.99 (0.98)	-13.15 (1.32)	3.105	0.096	-12.72 (1.05)	-13.24 (0.82)	0.400	0.539
aTGCf	-13.25 (0.94)	-13.29 (0.77)	0.050	0.826	-13.15 (0.89)	-13.26 (0.49)	0.221	0.647
PSD								
Fixed θ* **	-4.05E-3 (1.16E-3)	-4.00E-3 (1.26E-3)	1.435	0.247	**-2.47E-3 (1.74E-3)**	**-2.45E-3 (1.89E-3)**	**5.701**	**0.034**
Adapted θ	-3.96E-3 (1.57E-3)	-3.82E-3 (1.50E-3)	2.841	0.110	-2.26E-3 (1.78E-3)	-2.24E-3 (1.68E-3)	0.180	0.679
Slower γ	0.105 (0.014)	0.103 (0.014)	0.539	0.473	0.10 (0.013)	0.11 (0.014)	0.202	0.661
Faster γ	0.140 (2.74E-3)	0.140 (2.13E-3)	2.491	0.133	0.14 (2.38E-3)	0.14 (2.92E-3)	0.405	0.536

Note. ANCOVA type III test. Sphericity assumed. *F*-statistic and *p*-value are for main effect in ANCOVA, being TGC and PSD, with memory and control nights are repeated measures, and age, sex, and AHI as covariates. AHI, apnea-hypopnea index; YA, Younger Adults; OA, Older Adults; EEGm, memory task night; EEGc, control task night; M, mean; SD, standard deviation; TGCs, fixed theta and slower gamma coupling; TGCf, fixed theta and faster gamma coupling; aTGCs, adapted theta and slower gamma coupling, aTGCf, adapted theta and faster gamma coupling, θ, theta, γ, gamma PSD, power spectral density.

* Night Type – Age interaction for OA: F (1, 12) = 5.379, p = 0.039

** Night Type – Sex interaction for YA: F (1, 17) = 5.209, p = 0.036

Table S13

Results for rmANCOVA Comparing TGC MI and PSD in T3 and T4 Between Control and Memory EEG Overnights in Younger (N = 21) and Older (N = 16) Adults with Covariates Age, Sex, & AHI

Parameters	**YA**				**OA**			
	EEGm ***M* (*SD*)**	**EEGc** ***M* (*SD*)**	***F*** **(1, 17)**	***P***	**EEGm** ***M* (*SD*)**	**EEGc** ***M* (*SD*)**	***F*** **(1, 12)**	***P***
				T3				
TGCs*	-12.76 (0.98)	-12.92 (0.95)	2.396	0.140	-12.80 (1.45)	-12.57 (1.72)	0.005	0.946
TGCf	-12.82 (1.20)	-12.70 (0.84)	0.346	0.564	-12.65 (0.78)	-12.60 (1.58)	0.329	0.577
PSD								
Fixed θ	1.46E-3 (1.09E-3)	1.66E-3 (1.13E-3)	0.001	0.982	1.81E-3 (1.65E-3)	2.01E-3 (1.74E-3)	0.213	0.653
Slower γ	0.12 (0.011)	0.12 (0.011)	0.150	0.704	0.11 (0.011)	0.11 (0.011)	1.056	0.324
Faster γ	0.14 (2.67E-3)	0.14 (2.56E-3)	0.239	0.631	0.14 (2.55E-3)	0.14 (2.82E-3)	0.145	0.710
				T4				
TGCs**	-12.99 (1.10)	-12.88 (0.94)	0.031	0.863	-12.95 (0.79)	-12.45 (1.18)	1.102	0.314
TGCf	-12.80 (0.90)	-13.08 (0.85)	1.621	0.220	-12.78 (0.91)	-12.80 (1.03)	0.057	0.816
PSD								
Fixed θ	1.54E-3 (1.15E-3)	1.57E-3 (1.16E-3)	0.014	0.906	1.77E-3 (1.61E-3)	1.80E-3 (1.45E-3)	0.853	0.374
Slower γ	0.12 (0.011)	0.11 (0.010)	2.185	0.158	0.11 (0.010)	0.11 (0.011)	0.709	0.416
Faster γ	0.14 (2.09E-3)	0.14 (2.05E-3)	0.614	0.444	0.14 (2.68E-3)	0.14 (2.85E-3)	0.158	0.698

Note. ANCOVA type III test. Sphericity assumed. *F*-statistic and *p*-value are for main effect in ANCOVA, being TGC and PSD, with memory and control nights are repeated measures, and age, sex, and AHI as covariates. AHI, apnea-hypopnea index; YA, Younger Adults; OA, Older Adults; EEGm, memory task night; EEGc, control task night; M, mean; SD, standard deviation; TGCs, fixed theta and slower gamma coupling; TGCf, fixed theta and faster gamma coupling; θ, theta, γ, gamma PSD, power spectral density.

* YA: Night Type – Sex interaction for YA: F (1, 17) = 6.483, p = 0.021

** YA: Night Type – AHI interaction for YA: F (1, 17) = 7.624, p = 0.013

Table S14

Results for ANCOVA Comparing TGC MI and Theta and Gamma PSD in Fz, Pz and Cz Between Younger (N = 21) and Older (N = 16) Age Groups during Memory EEG Overnight with Covariates Age, Sex, and AHI

Measures	**OA (*M* (*SD*))**	**YA (*M* (*SD*))**	***F* (1, 36)**	***P***
		Fz		
TGCs	-13.07 (1.26)	-13.30 (1.08)	1.528	0.225
TGCf	-12.96 (0.86)	-13.28 (0.79)	0.125	0.726
aTGCs	-13.39 (1.25)	-13.41 (1.29)	0.528	0.473
aTGCf	-13.03 (0.77)	-13.27 (0.73)	1.116	0.299
PSD				
Fixed θ	-2.30E-3 (1.66E-3)	-3.71E-3 (1.13E-3)	0.269	0.607
Adapted θ	-1.92E-3 (1.73E-3)	-3.32E-3 (1.27E-3)	0.094	0.761
Slower γ*	0.108 (0.011)	0.1073 (0.0121)	0.864	0.359
Faster γ	0.142 (2.66E-3)	0.1415 (2.62E-3)	<0.001	0.997
		Pz		
TGCs	-12.56 (1.25)	-12.83 (1.33)	0.132	0.718
TGCf	-12.87 (1.13)	-13.20 (0.73)	0.065	0.800
PSD				
Fixed θ	-2.12E-3 (9.72E-3)	-1.10E-3 (1.70E-3)	0.548	0.464
Slower γ**	0.10 (0.012)	0.10 (0.011)	1.390	0.247
Faster γ	0.14 (3.67E-3)	0.14 (2.78E-3)	1.098	0.303
		Cz		
TGCs	-12.53 (0.72)	-13.09 (1.03)	1.300	0.263
TGCf	-13.09 (0.81)	-13.18 (0.88)	0.668	0.420
aTGCs	-12.72 (1.05)	-12.99 (0.98)	1.691	0.203
aTGCf	-13.15 (0.89)	-13.25 (0.94)	0.131	0.720
PSD				
Fixed θ	-2.47E-3 (1.74E-3)	-4.05E-3 (1.16E-3)	0.241	0.627
Adapted θ	-2.26E-3 (1.78E-3)	-3.96E-3 (1.57E-3)	0.399	0.532
Slower γ***	0.10 (0.013)	0.105 (0.014)	1.326	0.258
Faster γ****	0.14 (2.38E-3)	0.140 (2.74E-3)	0.398	0.532

Note. ANCOVA type III test. *F*-statistic and *p*-value are for main effect in ANCOVA, being TGC and PSD, with age, sex, and AHI as covariates. AHI, apnea-hypopnea index; YA, younger adults; OA, older adults; M, mean; SD, standard deviation; TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; aTGCs, adapted theta-slower gamma coupling, aTGCf, adapted theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

* Age Group – AHI interaction F (1, 36) = 13.865, p = 0.001
** Age Group – AHI interaction F (1, 36) = 9.021, p = 0.005
*** Age Group – AHI interaction F (1, 36) = 11.161, p = 0.002
**** Age Group – AHI interaction F (1, 36) = 4.760, p = 0.037

Table S15

Results for ANCOVA Comparing TGC MI and Theta and Gamma PSD in T3 and T4 Between Younger (N = 21) and Older (N = 16) Age Groups during Memory EEG Overnight with Covariates Age, Sex and AHI

Measures	**OA (*M* (*SD*))**	**YA (*M* (*SD*))**	***F* (1, 36)**	***P***
		T3		
TGCs	-12.80 (1.45)	-12.76 (0.98)	0.563	0.459
TGCf	-12.65 (0.78)	-12.82 (1.20)	0.001	0.976
PSD				
Fixed θ	1.81E-3 (1.65E-3)	1.46E-3 (1.09E-3)	0.004	0.948
Slower γ*	0.11 (0.011)	0.12 (0.011)	0.731	0.399
Faster γ**	0.14 (2.55E-3)	0.14 (2.67E-3)	2.813	0.103
		T4		
TGCs	-12.95 (0.79)	-12.99 (1.10)	0.032	0.860
TGCf	-12.78 (0.91)	-12.80 (0.90)	0.174	0.679
PSD				
Fixed θ	1.77E-3 (1.61E-3)	1.54E-3 (1.15E-3)	0.101	0.752
Slower γ***	0.11 (0.010)	0.12 (0.011)	0.891	0.352
Faster γ	0.14 (2.68E-3)	0.14 (2.09E-3)	2.094	0.158

Note. ANCOVA type III test. *F*-statistic and *p*-value are for main effect in ANCOVA, being TGC and PSD, with age, sex, and AHI as covariates. AHI, apnea-hypopnea index; YA, younger adults; OA, older adults; M, mean; SD, standard deviation; TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

* Age Group – AHI interaction F (1, 36) = 12.455, p = 0.001
** Age Group – AHI interaction F (1, 36) = 5.523, p = 0.025
*** Age Group – AHI interaction F (1, 36) = 13.168, p = 0.001

Table S16

Results for TGC MI and Theta and Gamma PSD Predicting Memory Consolidation in Fz Between Age Groups with Covariates Age, Sex and AHI

Group	Measure	*F*	*Adj. R^2*	*Δ R^2*	*B (SE)*	*β*	*P*
YA	TGCs MI	1.623	0.11	<0.01	0.15 (0.56)	0.06	0.798
	TGCf MI	1.615	0.11	<0.01	-0.16 (0.76)	-0.05	0.834
	aTGCs MI	1.606	0.11	<0.01	0.07 (0.48)	0.03	0.889
	aTGCf MI	1.811	0.14	0.03	-0.67 (0.87)	-0.18	0.448
*	Fixed θ PSD	**4.201**	**0.39**	**0.23**	**-1211.97 (444.41)**	**-0.49**	**0.015**
**	Adapted θ PSD	**4.175**	**0.39**	**0.23**	**-1138.35 (419.54)**	**-0.52**	**0.015**
	Slower γ PSD	1.765	0.13	0.02	-43.09 (62.48)	-0.19	0.500
	Faster γ PSD	2.026	0.17	0.05	-296.29 (268.29)	-0.28	0.286
OA	TGCs MI	5.462	0.54	0.01	-0.46 (0.95)	-0.09	0.636
	TGCf MI	5.684	0.56	0.02	1.01 (1.37)	0.13	0.478
	aTGCs MI	5.572	0.55	0.01	0.60 (0.96)	0.11	0.547
	aTGCf MI	5.289	0.53	<0.01	0.01 (1.63)	<0.01	0.993
***	Fixed θ PSD	6.503	0.59	0.04	1202.63 (933.08)	0.30	0.224
****	Adapted θ PSD	7.289	0.63	0.07	1325.24 (801.14)	0.35	0.126
	Slower γ PSD	5.459	0.54	0.01	64.70 (134.29)	0.11	0.639
	Faster γ PSD	5.289	0.53	<0.01	9.14 (484.79)	<0.01	0.985

Note. *F*-statistic, R^2, *Adj.* R^2, and $R^2\Delta$ are for the regression model with TGC and PSD types, age sex, and AHI as IVs. *B (SE)*, *β*, and *p*-value are for TGC type. DV is memory consolidation, calculated as total same paired word recalled in both pre- and post-sleep. F (3, 17) for YA (N = 21). F (3, 12) for OA (N = 16). AHI, apnea-hypopnea index; R^2, proportion of explained variance, Adj. R^2, proportion of explained variance considering error inflation, Δ R^2, change in proportion of explained variance by IV alone, YA, younger adults; OA, older adults; B, unstandardized coefficient; SE, standard error; β, standardized coefficient; MI, modulation index TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; aTGCs, adapted theta-slower gamma coupling, aTGCf, adapted theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

* AHI effect in model 2: *B (SE)* = -0.56 (0.24), *β* = -0.44, *p* = 0.032
** AHI effect in model 2: *B (SE)* = -0.68 (0.25), *β* = -0.53, *p* = 0.015
*** AHI effect in model 2: *B (SE)* = -0.81 (0.35), *β* = -0.43, *p* = 0.039
**** AHI effect in model 2: *B (SE)* = -0.89 (0.34), *β* = -0.47, *p* = 0.025

Table S17

Results for TGC MI and Theta and Gamma PSD Predicting Memory Consolidation in Pz Between Age Groups with Covariates Age, Sex and AHI

Group		Measure	*F*	*Adj. R^2*	*Δ R^2*	*B* (*SE*)	*β*	*P*
YA		TGCs MI	1.817	0.14	0.03	0.41 (0.52)	0.18	0.442
		TGCf MI	1.627	0.11	<0.01	0.15 (0.53)	0.06	0.781
	*	Fixed θ PSD	**4.257**	**0.39**	**0.23**	**-1413.50 (512.87)**	**-0.49**	**0.014**
		Slower γ PSD	1.791	0.14	0.02	-43.79 (59.04)	-0.19	0.469
		Faster γ PSD	1.678	0.12	0.01	-87.79 (184.88)	-0.11	0.641
OA		TGCs MI	6.087	0.58	0.03	0.90 (0.87)	0.18	0.318
		TGCf MI	5.292	0.53	<0.01	0.11 (1.65)	0.01	0.948
		Fixed θ PSD	5.981	0.57	0.03	853.34 (877.13)	0.22	0.352
		Slower γ PSD	5.338	0.54	<0.01	34.74 (134.01)	0.06	0.800
		Faster γ PSD	5.293	0.53	<0.01	-29.94 (426.90)	-0.01	0.945

Note. *F*-statistic, R^2, *Adj.* R^2, and $R^2\Delta$ are for the regression model with TGC and PSD types, age sex, and AHI as IVs. *B (SE)*, *β*, and *p*-value are for TGC type. DV is memory consolidation, calculated as total same paired word recalled in both pre- and post-sleep. F (3, 17) for YA (N = 21). F (3, 12) for OA (N = 16). AHI, apnea-hypopnea index; R^2, proportion of explained variance, Adj. R^2, proportion of explained variance considering error inflation, Δ R^2, change in proportion of explained variance by IV alone, YA, younger adults; OA, older adults; B, unstandardized coefficient; SE, standard error; β, standardized coefficient; MI, modulation index TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

* AHI effect in model 2: *B (SE)* = -0.55 (0.24), *β* = -0.43, *p* = 0.034

Table S18

Results for TGC MI and Theta and Gamma PSD Predicting Memory Consolidation in Cz Between Age Groups with Covariates Age, Sex and AHI

Group	Measure	*F*	*Adj. R^2*	*Δ R^2*	*B* (*SE*)	*β*	*P*
YA	TGCs MI	1.631	0.11	<0.01	0.19 (0.62)	0.07	0.767
	TGCf MI	1.607	0.11	<0.01	-0.10 (0.68)	-0.03	0.882
	aTGCs MI	1.609	0.11	<0.01	0.11 (0.65)	0.04	0.867
	aTGCf MI	1.674	0.12	0.01	-0.31 (0.66)	-0.10	0.650
*	Fixed θ PSD	**5.605**	**0.48**	**0.30**	**-1341.50 (396.50)**	**-0.55**	**0.004**
**	Adapted θ PSD	**3.668**	**0.35**	**0.19**	**-822.99 (338.45)**	**-0.46**	**0.027**
	Slower γ PSD	1.856	0.15	0.03	-42.71 (49.80)	-0.21	0.404
	Faster γ PSD	1.628	0.11	<0.01	-84.99 (295.53)	-0.08	0.777
OA	TGCs MI	5.767	0.56	0.02	1.64 (2.03)	0.18	0.436
***	TGCf MI	8.856	0.68	0.11	2.78 (1.26)	0.34	0.049
****	aTGCs MI	5.291	0.53	<0.01	-0.07 (1.26)	-0.01	0.959
	aTGCf MI	7.943	0.65	0.08	2.23 (1.17)	0.30	0.083
	Fixed θ PSD	5.560	0.55	0.01	583.11 (957.59)	0.15	0.555
	Adapted θ PSD	5.730	0.56	0.02	635.02 (817.44)	0.17	0.454
	Slower γ PSD	5.345	0.54	<0.01	33.99 (122.47)	0.06	0.786
	Faster γ PSD	5.853	0.56	0.02	463.41 (527.35)	0.17	0.398

Note. *F*-statistic, R^2, *Adj.* R^2, and $R^2\Delta$ are for the regression model with TGC and PSD types, age sex, and AHI as IVs. *B (SE)*, *β*, and *p*-value are for TGC type. DV is memory consolidation, calculated as total same paired word recalled in both pre- and post-sleep. F (3, 17) for YA (N = 21). F (3, 12) for OA (N = 16). AHI, apnea-hypopnea index; R^2, proportion of explained variance, Adj. R^2, proportion of explained variance considering error inflation, Δ R^2, change in proportion of explained variance by IV alone, YA, younger adults; OA, older adults; B, unstandardized coefficient; SE, standard error; β, standardized coefficient; MI, modulation index TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; aTGCs, adapted theta-slower gamma coupling, aTGCf, adapted theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

* AHI effect in model 2: *B (SE)* = -0.52 (0.22), *β* = -0.41, *p* = 0.031

** AHI effect in model 2: *B (SE)* = -0.59 (0.25), *β* = -0.46, *p* = 0.033

*** AHI effect in model 2: *B (SE)* = -0.74 (0.30), *β* = -0.39, *p* = 0.029

**** AHI effect in model 2: *B (SE)* = -0.77 (0.31), *β* = -0.41, *p* = 0.030

Table S19

Results for TGC MI and Theta and Gamma PSD Predicting Memory Consolidation in T3 Between Age Groups with Covariates Age, Sex and AHI

Group		Measure	*F*	*Adj. R^2*	*Δ R^2*	*B* (*SE*)	*β*	*P*
YA		TGCs MI	1.611	0.11	<0.01	-0.11 (0.61)	-0.04	0.856
		TGCf MI	1.716	0.13	0.01	0.29 (0.50)	0.12	0.571
		Fixed θ PSD	2.885	0.27	0.13	-961.66 (501.53)	-0.37	0.073
		Slower γ PSD	1.816	0.14	0.03	-53.39 (67.72)	-0.21	0.442
		Faster γ PSD	1.904	0.15	0.04	-251.07 (268.92)	-0.24	0.364
OA	*	TGCs MI	6.022	0.57	0.03	0.90 (0.90)	0.20	0.338
		TGCf MI	5.601	0.55	0.01	1.18 (1.80)	0.14	0.527
	**	Fixed θ PSD	6.704	0.60	0.05	1187.81 (853.65)	0.30	0.192
		Slower γ PSD	5.531	0.55	0.01	72.48 (125.87)	0.12	0.576
		Faster γ PSD	5.500	0.55	0.01	300.09 (558.44)	0.12	0.602

Note. *F*-statistic, R^2, *Adj.* R^2, and $R^2\Delta$ are for the regression model with TGC and PSD types, age sex, and AHI as IVs. *B (SE)*, *β*, and *p*-value are for TGC type. DV is memory consolidation, calculated as total same paired word recalled in both pre- and post-sleep. F (3, 17) for YA (N = 21). F (3, 12) for OA (N = 16). AHI, apnea-hypopnea index; R^2, proportion of explained variance, Adj. R^2, proportion of explained variance considering error inflation, Δ R^2, change in proportion of explained variance by IV alone, YA, younger adults; OA, older adults; B, unstandardized coefficient; SE, standard error; β, standardized coefficient; MI, modulation index TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

* AHI effect in model 2: *B (SE)* = -0.87 (0.39), *β* = -0.46, *p* = 0.049
** AHI effect in model 2: *B (SE)* = -0.81 (0.34), *β* = -0.43, *p* = 0.037

Table S20

Results for TGC MI and Theta and Gamma PSD Predicting Memory Consolidation in T4 Between Age Groups with Covariates Age, Sex and AHI

Group	Measure	*F*	*Adj. R^2*	*Δ R^2*	*B (SE)*	*β*	*P*
YA	TGCs MI	1.602	0.11	<0.01	-0.05 (0.55)	-0.02	0.927
	TGCf MI	1.840	0.14	0.03	0.55 (0.66)	0.18	0.419
	Fixed θ PSD	2.904	0.28	0.14	-946.08 (489.96)	-0.39	0.071
	Slower γ PSD	1.933	0.16	0.04	-68.55 (70.13)	-0.26	0.343
*	Faster γ PSD	2.390	0.22	0.09	-483.25 (321.39)	-0.36	0.152
OA	TGCs MI	5.329	0.54	<0.01	0.37 (1.60)	0.04	0.820
	TGCf MI	5.289	0.53	<0.01	<0.01 (1.27)	<0.01	0.999
	Fixed θ PSD	5.658	0.55	0.01	625.78 (881.14)	0.15	0.492
	Slower γ PSD	5.425	0.54	0.01	60.64 (140.42)	0.09	0.674
	Faster γ PSD	5.485	0.54	0.01	245.49 (473.50)	0.10	0.614

Note. *F*-statistic, R^2, *Adj.* R^2, and $R^2\Delta$ are for the regression model with TGC and PSD types, age sex, and AHI as IVs. *B (SE)*, *β*, and *p*-value are for TGC type. DV is memory consolidation, calculated as total same paired word recalled in both pre- and post-sleep. F (3, 17) for YA (N = 21). F (3, 12) for OA (N = 16). AHI, apnea-hypopnea index; R^2, proportion of explained variance, Adj. R^2, proportion of explained variance considering error inflation, Δ R^2, change in proportion of explained variance by IV alone, YA, younger adults; OA, older adults; B, unstandardized coefficient; SE, standard error; β, standardized coefficient; MI, modulation index TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

* AHI effect in model 2: *B (SE)* = -0.69 (0.31), *β* = -0.54, *p* = 0.040

Table S21

Results for Sex Covariate Interaction in rmANCOVA Comparing TGC MI and Theta and Gamma PSD Fz, Cz, and T3 Between Control and Memory EEG Overnights in Younger Adults (N = 21)

Parameters	**YA**			
	EEGm *M* (*SD*)	**EEGc *M* (*SD*)**	***F* (1, 18)**	***P***
		Fz		
TGCs	-13.30 (1.08)	-13.14 (0.92)	2.963	0.102
TGCf	-13.28 (0.79)	-13.32 (0.75)	1.609	0.221
aTGCs	-13.41 (1.29)	-13.44 (0.98)	3.245	0.088
aTGCf	**-13.27 (0.73)**	**-13.32 (0.91)**	**5.241**	**0.034**
PSD				
Fixed θ	-3.71E-3 (1.13E-3)	-3.50E-3 (1.02E-3)	1.545	0.230
Adapted θ	-3.32E-3 (1.27E-3)	-2.79E-3 (1.21E-3)	0.151	0.702
Slower γ	0.1073 (0.0121)	0.1073 (0.0140)	0.162	0.692
Faster γ	0.1415 (2.62E-3)	0.1412 (2.33E-3)	0.547	0.469
		Cz		
TGCs	-13.09 (1.03)	-13.01 (1.22)	2.186	0.157
TGCf	-13.18 (0.88)	-13.24 (0.48)	1.505	0.236
aTGCs	-12.99 (0.98)	-13.15 (1.32)	0.316	0.581
aTGCf	-13.25 (0.94)	-13.29 (0.77)	1.368	0.257
PSD				
Fixed θ	-4.05E-3 (1.16E-3)	-4.00E-3 (1.26E-3)	0.189	0.669
Adapted θ	**-3.96E-3 (1.57E-3)**	**-3.82E-3 (1.50E-3)**	**6.083**	**0.024**
Slower γ	0.105 (0.014)	0.103 (0.014)	0.438	0.516
Faster γ	0.140 (2.74E-3)	0.140 (2.13E-3)	0.078	0.783
		T3		
TGCs	**-12.76 (0.98)**	**-12.92 (0.95)**	**6.279**	**0.022**
TGCf	-12.82 (1.20)	-12.70 (0.84)	0.304	0.588
PSD				
Fixed θ	1.46E-3 (1.09E-3)	1.66E-3 (1.13E-3)	0.041	0.842
Slower γ	0.12 (0.011)	0.12 (0.011)	0.986	0.334
Faster γ	0.14 (2.67E-3)	0.14 (2.56E-3)	0.048	0.828

Note. ANCOVA type III test. Sphericity assumed. *F*-statistic and *p*-value are for main effect in ANCOVA, being TGC and PSD, with memory and control nights are repeated measures, and age and sex as covariates. YA, Younger Adults; OA, Older Adults; EEGm, memory task night; EEGc, control task night; M, mean; SD, standard deviation; TGCs, fixed theta and slower gamma coupling; TGCf, fixed theta and faster gamma coupling; θ, theta, γ, gamma PSD, power spectral density.

Table S22

Results for Sex Covariate Interaction in ANCOVA Comparing Adapted TGC MI and Theta and Gamma PSD in Fz, Pz and Cz Between Younger (N = 21) and Older (N = 16) Age Groups during Memory EEG Overnight

Measures	**OA (*M* (*SD*))**	**YA (*M* (*SD*))**	***F* (1, 36)**	***P***
		Fz		
TGCs	-13.07 (1.26)	-13.30 (1.08)	0.538	0.469
TGCf	-12.96 (0.86)	-13.28 (0.79)	0.073	0.789
aTGCs	-13.39 (1.25)	-13.41 (1.29)	0.666	0.420
aTGCf	-13.03 (0.77)	-13.27 (0.73)	0.108	0.744
PSD				
Fixed θ	**-2.30E-3 (1.66E-3)**	**-3.71E-3 (1.13E-3)**	**4.500**	**0.041**
Adapted θ	*-1.92E-3 (1.73E-3)*	*-3.32E-3 (1.27E-3)*	*3.895*	*0.057*
Slower γ	0.11 (0.01)	0.11 (0.01)	0.005	0.942
Faster γ	**0.14 (2.66E-3)**	**0.14 (2.62E-3)**	**5.472**	**0.026**
		Pz		
TGCs	-12.83 (1.33)	-12.56 (1.25)	0.112	0.740
TGCf	-13.20 (0.73)	-12.87 (1.13)	0.210	0.650
PSD				
Fixed θ	**-1.10E-3 (1.70E-3)**	**-2.12E-3 (9.72E-3)**	**4.335**	**0.045**
Slower γ	0.10 (0.01)	0.10 (0.01)	0.247	0.623
Faster γ	0.14 (2.78E-3)	0.14 (3.67E-3)	0.793	0.380
		Cz		
TGCs	**-12.53 (0.72)**	**-13.09 (1.03)**	**5.012**	**0.032**
TGCf	-13.09 (0.81)	-13.18 (0.88)	0.261	0.613
aTGCs	-12.72 (1.05)	-12.99 (0.98)	1.827	0.186
aTGCf	-13.15 (0.89)	-13.25 (0.94)	0.500	0.485
PSD				
Fixed θ	*-2.47E-3 (1.74E-3)*	*-4.05E-3 (1.16E-3)*	*4.039*	*0.053*
Adapted θ	-2.26E-3 (1.78E-3)	-3.96E-3 (1.57E-3)	2.607	0.116
Slower γ	0.10 (0.013)	0.105 (0.014)	0.048	0.827
Faster γ	**0.14 (2.38E-3)**	**0.140 (2.74E-3)**	**5.049**	**0.031**

Note. ANCOVA type III test. *F*-statistic and *p*-value are for main effect in ANCOVA, being TGC and PSD, with age and sex as covariates. YA, younger adults; OA, older adults; M, mean; SD, standard deviation; TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; aTGCs, adapted theta-slower gamma coupling, aTGCf, adapted theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

Table S23

Results for Sex Covariate of TGC MI and Theta and Gamma PSD Predicting Memory Consolidation in the First Model of Hierarchal Regression Between Age Groups with Covariates

Group	***F***	***Adj. R^2***	***R^2***	***B* (*SE*)**	***β***	***P***
YA	2.409	0.12	0.21	2.31 (1.31)	0.40	0.094
OA	**7.837**	**0.45**	**0.55**	**8.55 (2.83)**	**0.62**	**0.010**

Note. *F*-statistic, R^2, *Adj.* R^2, and $R^2\Delta$ are for the regression model age and sex as IVs. *B (SE)*, *β*, and *p*-value are for the sex covariate predictor in model 1. DV is memory consolidation, calculated as total same paired word recalled in both pre- and post-sleep. F (3, 17) for YA (N = 21). F (3, 12) for OA (N = 16). Adj. R^2, proportion of explained variance considering error inflation; R^2, proportion of explained variance; B, unstandardized coefficient; SE, standard error; β, standardized coefficient; YA, younger adults; OA, older adults.

Table S24

Results for Sex Covariate Predicting Memory Consolidation in Fz Between Age Groups with Covariates when factoring TGC MI and Theta and Gamma PSD

Group	Measure	*F*	*Adj. R^2*	*Δ R^2*	*B* (*SE*)	*β*	*P*
YA	TGCs MI	*2.286*	*0.16*	*0.08*	*2.71 (1.31)*	*0.47*	*0.054*
	TGCf MI	*6.903*	*0.47*	*0.34*	*2.12 (1.02)*	*0.37*	*0.053*
	aTGCs MI	**2.581**	**0.19**	**0.10**	**2.80 (1.29)**	**0.48**	**0.045**
	aTGCf MI	4.768	0.36	0.25	2.15 (1.12)	0.37	0.071
	Fixed θ PSD	*3.154*	*0.24*	*0.15*	*2.46 (1.22)*	*0.42*	*0.059*
	Adapted θ PSD	2.095	0.14	0.06	2.46 (1.30)	0.43	0.075
	Slower γ PSD	2.086	0.14	0.06	1.98 (1.33)	0.34	0.154
	Faster γ PSD	1.530	0.07	<0.01	2.40 (1.43)	0.41	0.111
OA	TGCs MI	**5.120**	**0.45**	**0.02**	**8.57 (2.89)**	**0.63**	**0.012**
	TGCf MI	**4.845**	**0.44**	**<0.01**	**8.50 (2.95)**	**0.62**	**0.014**
	aTGCs MI	**4.823**	**0.43**	**<0.01**	**8.54 (2.94)**	**0.62**	**0.013**
	aTGCf MI	**4.970**	**0.43**	**0.01**	**8.34 (2.95)**	**0.61**	**0.015**
	Fixed θ PSD	**4.988**	**0.44**	**0.01**	**9.75 (3.86)**	**0.71**	**0.027**
	Adapted θ PSD	**5.051**	**0.45**	**0.01**	**9.64 (3.51)**	**0.70**	**0.018**
	Slower γ PSD	**6.169**	**0.51**	**0.06**	**10.10 (2.97)**	**0.74**	**0.005**
	Faster γ PSD	**4.823**	**0.43**	**<0.01**	**8.53 (3.25)**	**0.62**	**0.022**

Note. *F*-statistic, R^2, *Adj.* R^2, and $R^2\Delta$ are for the regression model with TGC and PSD types, age, and sex as IVs. *B (SE)*, *β*, and *p*-value are for the sex covariate predictor of model 2. DV is memory consolidation, calculated as total same paired word recalled in both pre- and post-sleep. F (3, 17) for YA (N = 21). F (3, 12) for OA (N = 16). R^2, proportion of explained variance, Adj. R^2, proportion of explained variance considering error inflation, Δ R^2, change in proportion of explained variance by IV alone, YA, younger adults; OA, older adults; B, unstandardized coefficient; SE, standard error; β, standardized coefficient; MI, modulation index TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; aTGCs, adapted theta-slower gamma coupling, aTGCf, adapted theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

Table S25

Results for Sex Covariate Predicting Memory Consolidation in Pz Between Age Groups with Covariates when factoring TGC MI and Theta and Gamma PSD

Group	**Measure**	***F***	***Adj. R²***	***Δ R²***	***B* (*SE*)**	***β***	***P***
YA	TGCs MI	1.764	0.10	0.03	2.38 (1.33)	0.41	0.090
	TGCf MI	1.548	0.08	<0.01	2.35 (1.35)	0.41	0.100
	Fixed θ PSD	*3.566*	*0.28*	*0.18*	*2.51 (1.19)*	*0.43*	*0.050*
	Slower γ PSD	1.890	0.12	0.04	2.12 (1.33)	0.37	0.128
	Faster γ PSD	1.57	0.08	<0.01	2.23 (1.37)	0.38	0.122
OA	TGCs MI	**4.940**	**0.44**	**<0.01**	**8.57 (2.92)**	**0.63**	**0.013**
	TGCf MI	**4.928**	**0.44**	**0.01**	**8.66 (2.94)**	**0.63**	**0.012**
	Fixed θ PSD	**4.837**	**0.43**	**<0.01**	**8.84 (3.63)**	**0.65**	**0.032**
	Slower γ PSD	**5.497**	**0.47**	**0.03**	**9.96 (3.20)**	**0.73**	**0.009**
	Faster γ PSD	**4.828**	**0.43**	**<0.01**	**8.55 (2.94)**	**0.62**	**0.013**

Note. *F*-statistic, R^2, *Adj.* R^2, and $R^2\Delta$ are for the regression model with TGC and PSD types, age, and sex as IVs. *B (SE)*, *β*, and *p*-value are for the sex covariate predictor of model 2. DV is memory consolidation, calculated as total same paired word recalled in both pre- and post-sleep. F (3, 17) for YA (N = 21). F (3, 12) for OA (N = 16). R^2, proportion of explained variance, Adj. R^2, proportion of explained variance considering error inflation, Δ R^2, change in proportion of explained variance by IV alone, YA, younger adults; OA, older adults; B, unstandardized coefficient; SE, standard error; β, standardized coefficient; MI, modulation index TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

Table S26

Results for Sex Covariate Predicting Memory Consolidation in Cz Between Age Groups with Covariates when factoring TGC MI and Theta and Gamma PSD

Group	Measure	*F*	*Adj. R^2*	*Δ R^2*	*B* (*SE*)	*β*	*P*
YA	TGCs MI	1.785	0.11	0.03	2.65 (1.39)	0.46	0.073
	TGCf MI	1.578	0.22	0.01	2.36 (1.35)	0.41	0.097
	aTGCs MI	1.958	0.13	0.05	2.63 (1.34)	0.46	0.066
	aTGCf MI	1.553	0.08	<0.01	2.34 (1.35)	0.40	0.100
	Fixed θ PSD	3.360	0.26	0.16	2.31 (1.20)	0.40	0.071
	Adapted θ PSD	1.913	0.12	0.04	2.46 (1.32)	0.42	0.080
	Slower γ PSD	2.182	0.15	0.07	2.02 (1.31)	0.35	0.140
	Faster γ PSD	0.577	-0.07	0.02	2.13 (1.42)	0.37	0.153
OA	TGCs MI	*5.739*	*0.49*	*0.55*	*6.80 (3.21)*	*0.50*	*0.056*
	TGCf MI	**6.750**	**0.54**	**0.08**	**8.89 (2.67)**	**0.65**	**0.006**
	aTGCs MI	**4.846**	**0.44**	**<0.01**	**8.42 (3.03)**	**0.61**	**0.017**
	aTGCf MI	**5.944**	**0.50**	**0.05**	**9.08 (2.80)**	**0.66**	**0.007**
	Fixed θ PSD	*4.834*	*0.43*	*<0.01*	*8.22 (3.93)*	*0.60*	*0.058*
	Adapted θ PSD	**4.816**	**0.44**	**<0.01**	**8.99 (3.53)**	**0.66**	**0.026**
	Slower γ PSD	**5.829**	**0.49**	**0.05**	**9.96 (3.04)**	**0.73**	**0.007**
	Faster γ PSD	**5.796**	**0.49**	**0.05**	**7.27 (3.00)**	**0.53**	**0.032**

Note. *F*-statistic, R^2, *Adj.* R^2, and $R^2\Delta$ are for the regression model with TGC and PSD types, age, and sex as IVs. *B (SE)*, *β*, and *p*-value are for the sex covariate predictor of model 2. DV is memory consolidation, calculated as total same paired word recalled in both pre- and post-sleep. F (3, 17) for YA (N = 21). F (3, 12) for OA (N = 16). R^2, proportion of explained variance, Adj. R^2, proportion of explained variance considering error inflation, Δ R^2, change in proportion of explained variance by IV alone, YA, younger adults; OA, older adults; B, unstandardized coefficient; SE, standard error; β, standardized coefficient; MI, modulation index TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; aTGCs, adapted theta-slower gamma coupling, aTGCf, adapted theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

Table S27

Results for Sex Covariate Predicting Memory Consolidation in T3 Between Age Groups with Covariates when factoring TGC MI and Theta and Gamma PSD

Group	Measure	*F*	*Adj. R^2*	*Δ R^2*	*B* (*SE*)	*β*	*P*
YA	TGCs MI	1.760	0.10	0.03	2.33 (1.32)	0.40	0.096
	TGCf MI	1.552	0.08	0.09	2.33 (1.34)	0.40	0.101
	Fixed θ PSD	3.384	0.26	0.16	2.28 (1.20)	0.39	0.074
	Slower γ PSD	1.966	0.13	0.05	2.00 (1.34)	0.35	0.153
	Faster γ PSD	0.457	-0.09	<0.01	2.01 (1.37)	0.35	0.162
OA	TGCs MI	**4.835**	**0.43**	**<0.01**	**8.56 (2.94)**	**0.62**	**0.013**
	TGCf MI	**5.018**	**0.45**	**0.01**	**8.30 (2.95)**	**0.61**	**0.016**
	Fixed θ PSD	**5.097**	**0.45**	**0.01**	**9.80 (3.55)**	**0.71**	**0.017**
	Slower γ PSD	**6.186**	**0.51**	**0.06**	**9.27 (2.79)**	**0.68**	**0.006**
	Faster γ PSD	**5.589**	**0.48**	**0.04**	**7.67 (2.95)**	**0.56**	**0.023**

Note. *F*-statistic, R^2, *Adj.* R^2, and $R^2\Delta$ are for the regression model with TGC and PSD types, age, and sex as IVs. *B (SE)*, *β*, and *p*-value are for the sex covariate predictor of model 2. DV is memory consolidation, calculated as total same paired word recalled in both pre- and post-sleep. F (3, 17) for YA (N = 21). F (3, 12) for OA (N = 16). R^2, proportion of explained variance, Adj. R^2, proportion of explained variance considering error inflation, Δ R^2, change in proportion of explained variance by IV alone, YA, younger adults; OA, older adults; B, unstandardized coefficient; SE, standard error; β, standardized coefficient; MI, modulation index TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

Table S28

Results for Sex Covariate Predicting Memory Consolidation in T4 Between Age Groups with Covariates when factoring TGC MI and Theta and Gamma PSD

Group	**Measure**	***F***	***Adj. R²***	***Δ R²***	***B* (*SE*)**	***β***	***P***
YA	TGCs MI	3.248	0.25	0.15	2.06 (1.21)	0.36	0.108
	TGCf MI	2.347	0.17	0.08	2.09 (1.28)	0.36	0.121
	Fixed θ PSD	2.044	0.14	0.05	2.00 (1.33)	0.35	0.150
	Slower γ PSD	0.460	-0.09	<0.01	2.00 (1.36)	0.35	0.160
	Faster γ PSD	0.505	-0.08	<0.01	2.00 (1.36)	0.35	0.159
OA	TGCs MI	**4.849**	**0.44**	**<0.01**	**8.35 (3.11)**	**0.61**	**0.020**
	TGCf MI	**4.824**	**0.43**	**<0.01**	**8.54 (2.95)**	**0.62**	**0.014**
	Fixed θ PSD	**4.853**	**0.44**	**<0.01**	**8.91 (3.45)**	**0.65**	**0.024**
	Slower γ PSD	**6.011**	**0.50**	**0.05**	**9.50 (2.86)**	**0.69**	**0.006**
	Faster γ PSD	**1.868**	**0.12**	**0.04**	**8.31 (3.00)**	**0.61**	**0.017**

Note. *F*-statistic, R^2, *Adj.* R^2, and $R^2\Delta$ are for the regression model with TGC and PSD types, age, and sex as IVs. *B (SE)*, *β*, and *p*-value are for the sex covariate predictor of model 2. DV is memory consolidation, calculated as total same paired word recalled in both pre- and post-sleep. F (3, 17) for YA (N = 21). F (3, 12) for OA (N = 16). R^2, proportion of explained variance, Adj. R^2, proportion of explained variance considering error inflation, Δ R^2, change in proportion of explained variance by IV alone, YA, younger adults; OA, older adults; B, unstandardized coefficient; SE, standard error; β, standardized coefficient; MI, modulation index TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; θ, theta; γ, gamma; PSD, power spectral density.

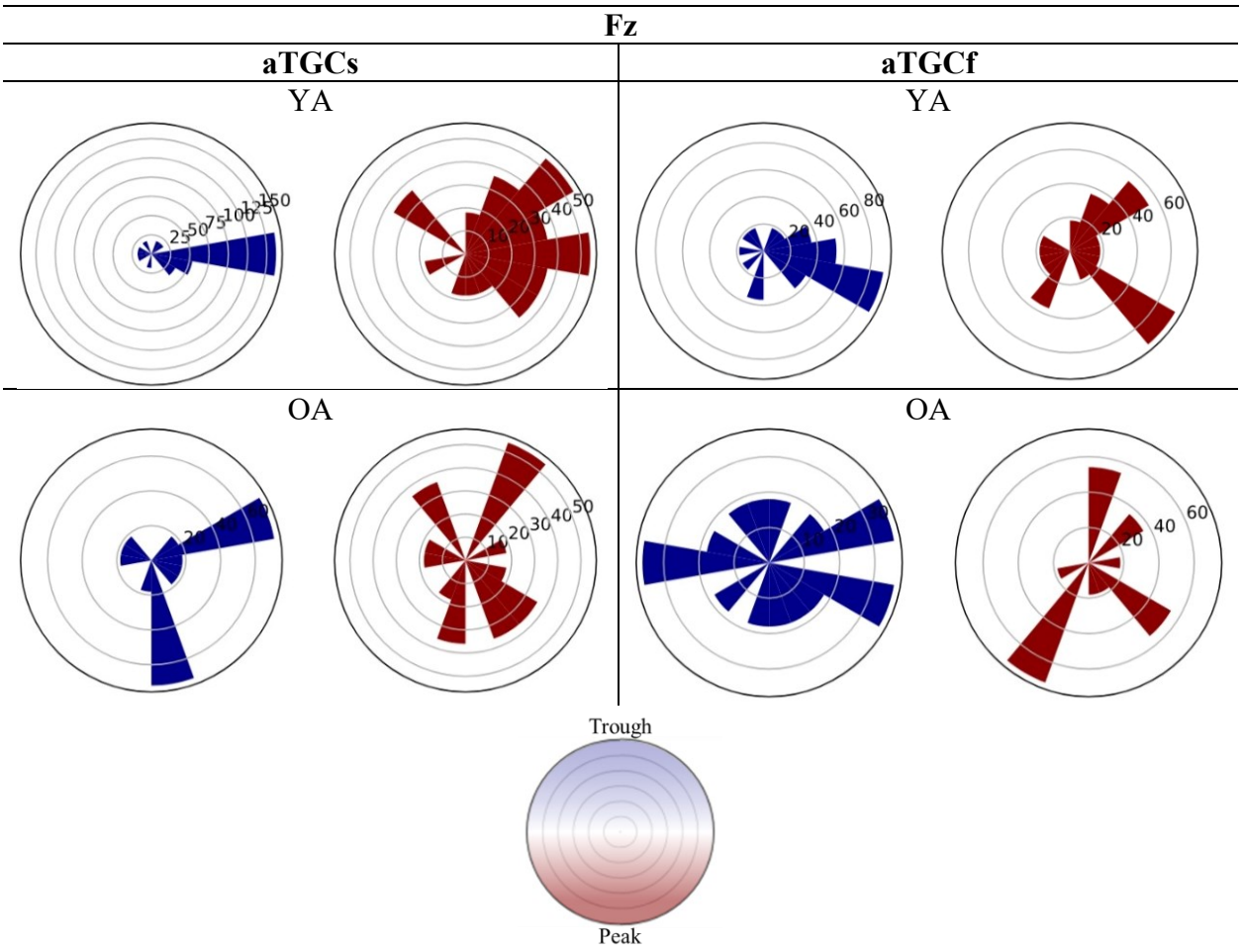

Figure S1. In channel Fz in YAs (top) and OAs (bottom), polar plots for aTGCs and aTGCf in both EEGc (blue) and EEGm (red). Vector length in axis rings reflect gamma amplitude strength, and direction reflects degrees of theta cycle. The top and bottom of each plot corresponds to theta trough and peak, respectively (bottom). YA, younger adult; OA, older adult; aTGCs, adapted theta-slower gamma coupling; aTGCf, adapted theta-faster gamma coupling; EEGc, control task EEG night; EEGm, memory task EEG night.

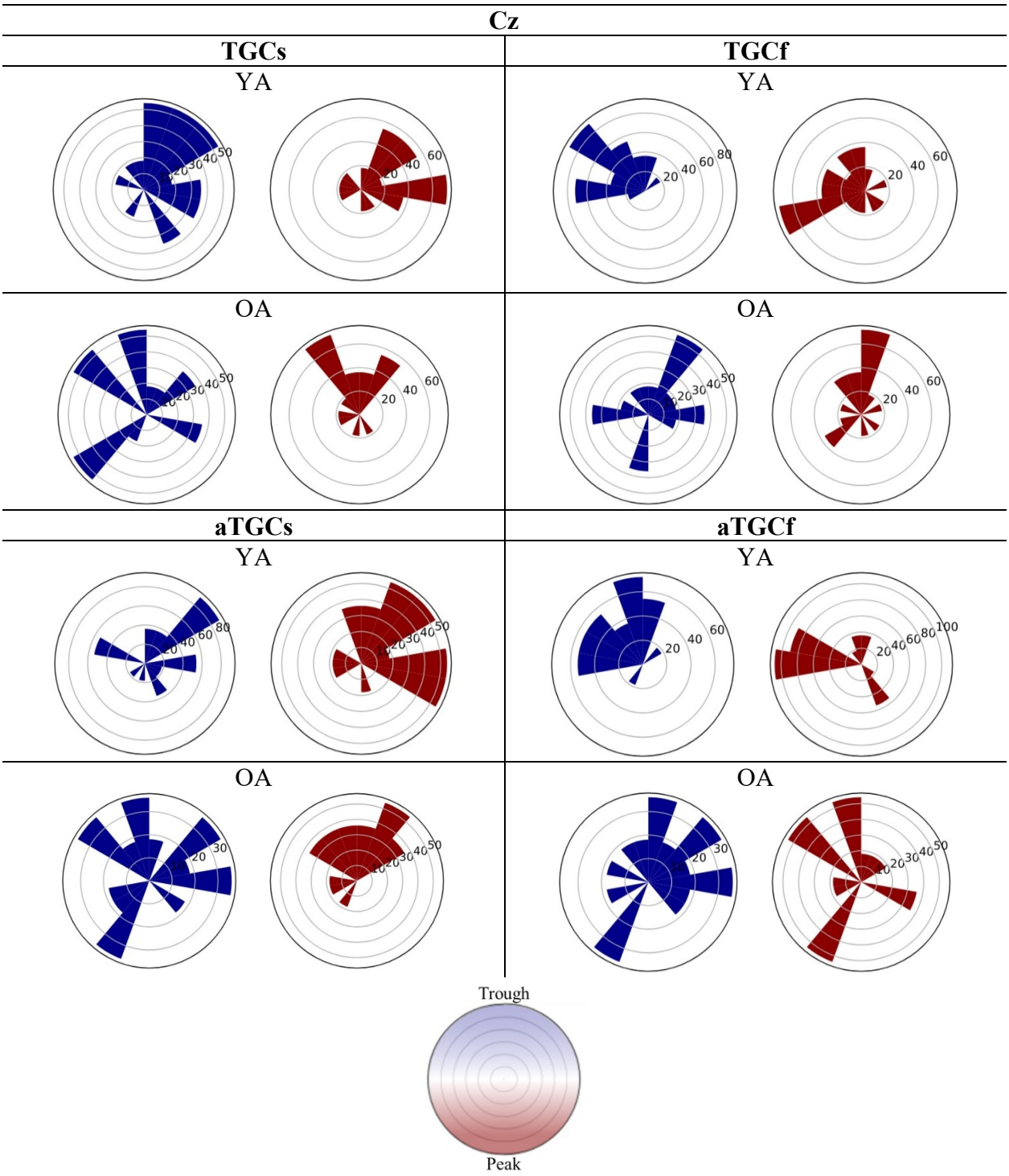

Figure S2. In channel Cz in YAs (top of each TGC type) and OAs (bottom of each TGC type), polar plots for TGCs, TGCf, aTGCs and aTGCf in both EEGc (blue) and EEGm (red). Vector length in axis rings reflect gamma amplitude strength, and direction reflects degrees of theta cycle. The top and bottom of each plot corresponds to theta trough and peak, respectively (bottom). YA, younger adult; OA, older adult; TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; aTGCs, adapted theta-slower gamma coupling; aTGCf, adapted theta-faster gamma coupling; EEGc, control task EEG night; EEGm, memory task EEG night.

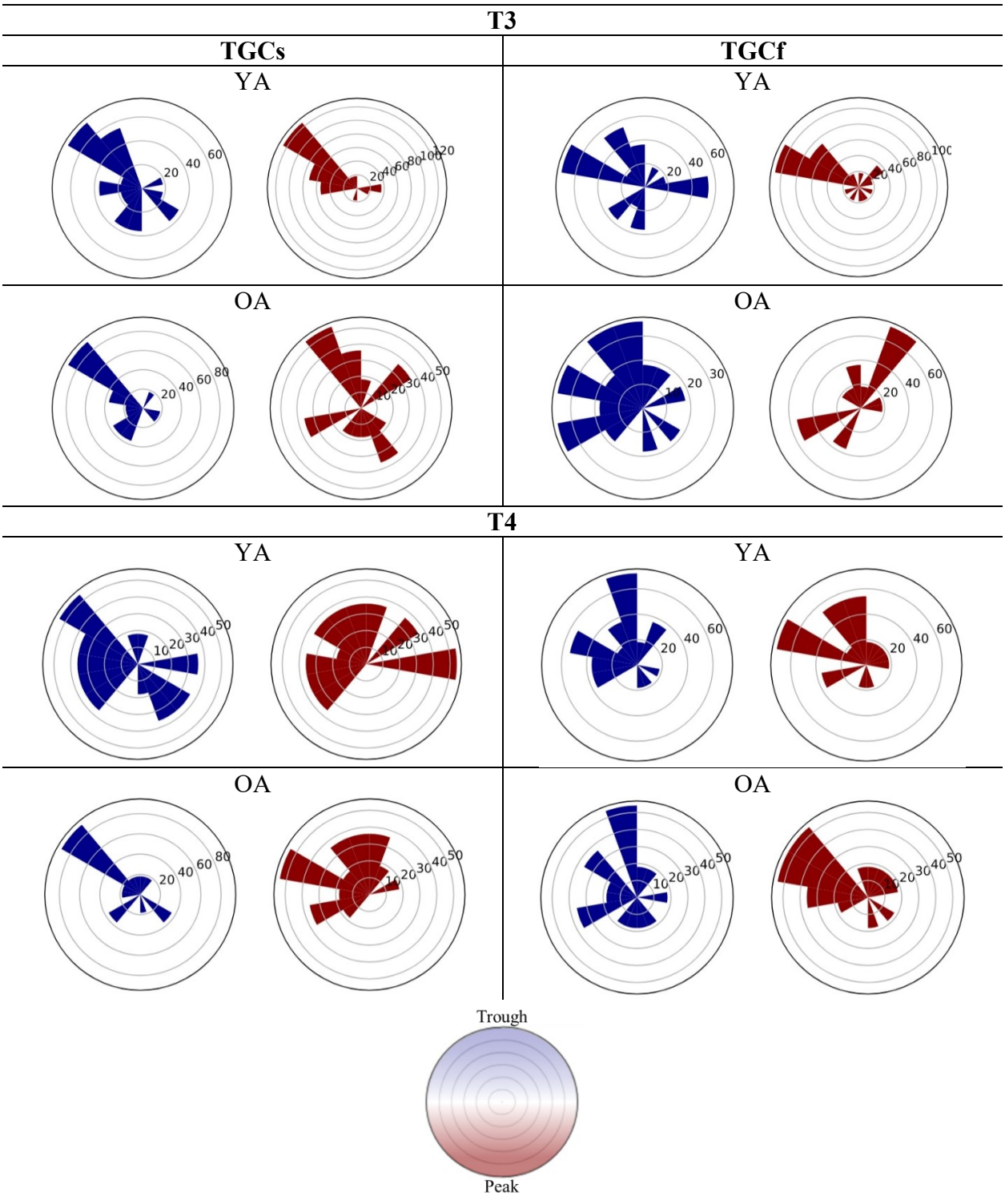

Figure S3. In channel T3s and T4 in YAs (top of each TGC type) and OAs (bottom of each TGC type), polar plots for TGCs and TGCf in both EEGc (blue) and EEGm (red). Vector length in axis rings reflect gamma amplitude strength, and direction reflects degrees of theta cycle. The top and bottom of each plot corresponds to theta trough and peak, respectively (bottom). YA, younger adult; OA, older adult; TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; EEGc, control task EEG night; EEGm, memory task EEG night.

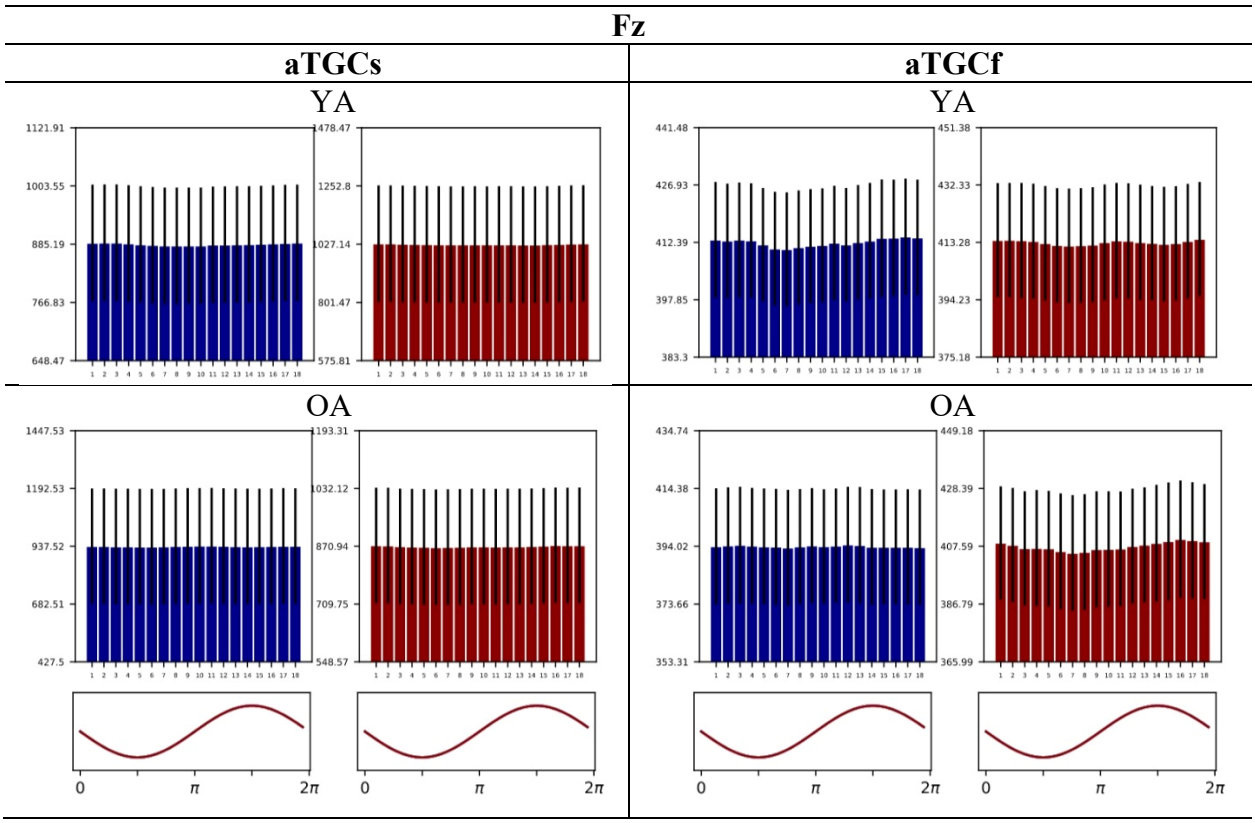

Figure S4. In channel Fz in YAs (top of each TGC type) and OAs (bottom of each TGC type) mean gamma amplitude plots with theta phase bins for both EEGc (blue) and EEGm (red). 18 phase bins in total, corresponding to sinusoidal theta wave (bottom). YA, younger adult; OA, older adult; aTGCs, adapted theta-slower gamma coupling; aTGCf, adapted theta-faster gamma coupling EEGc, control task EEG night; EEGm, memory task EEG night.

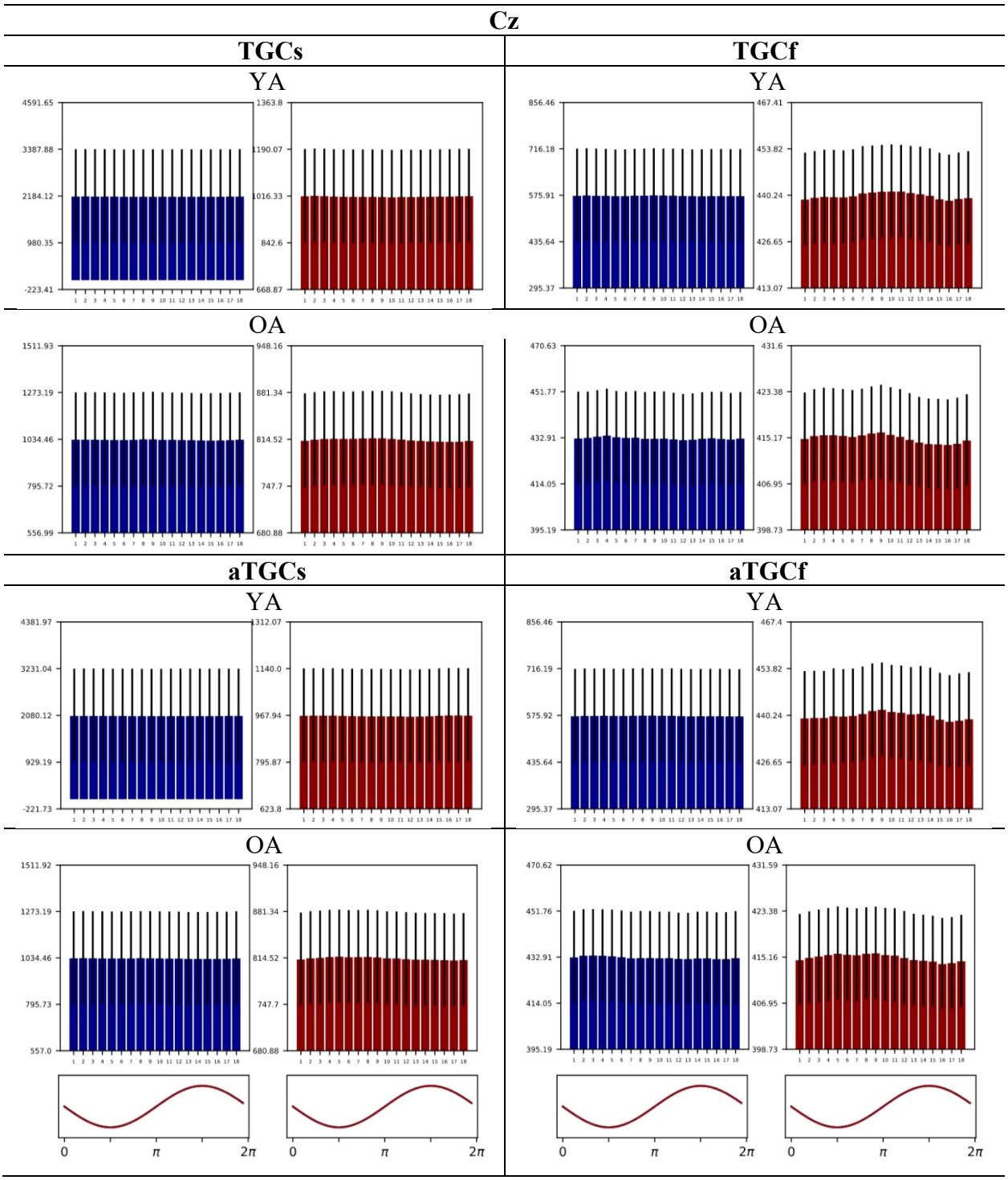

Figure S5. In channel Cz in YAs (top of each TGC type) and OAs (bottom of each TGC type) mean gamma amplitude plots with theta phase bins for both EEGc (blue) and EEGm (red). 18 phase bins in total, corresponding to sinusoidal theta wave (bottom). YA, younger adult; OA, older adult; aTGCs, adapted theta-slower gamma coupling; aTGCf, adapted theta-faster gamma coupling EEGc, control task EEG night; EEGm, memory task EEG night.

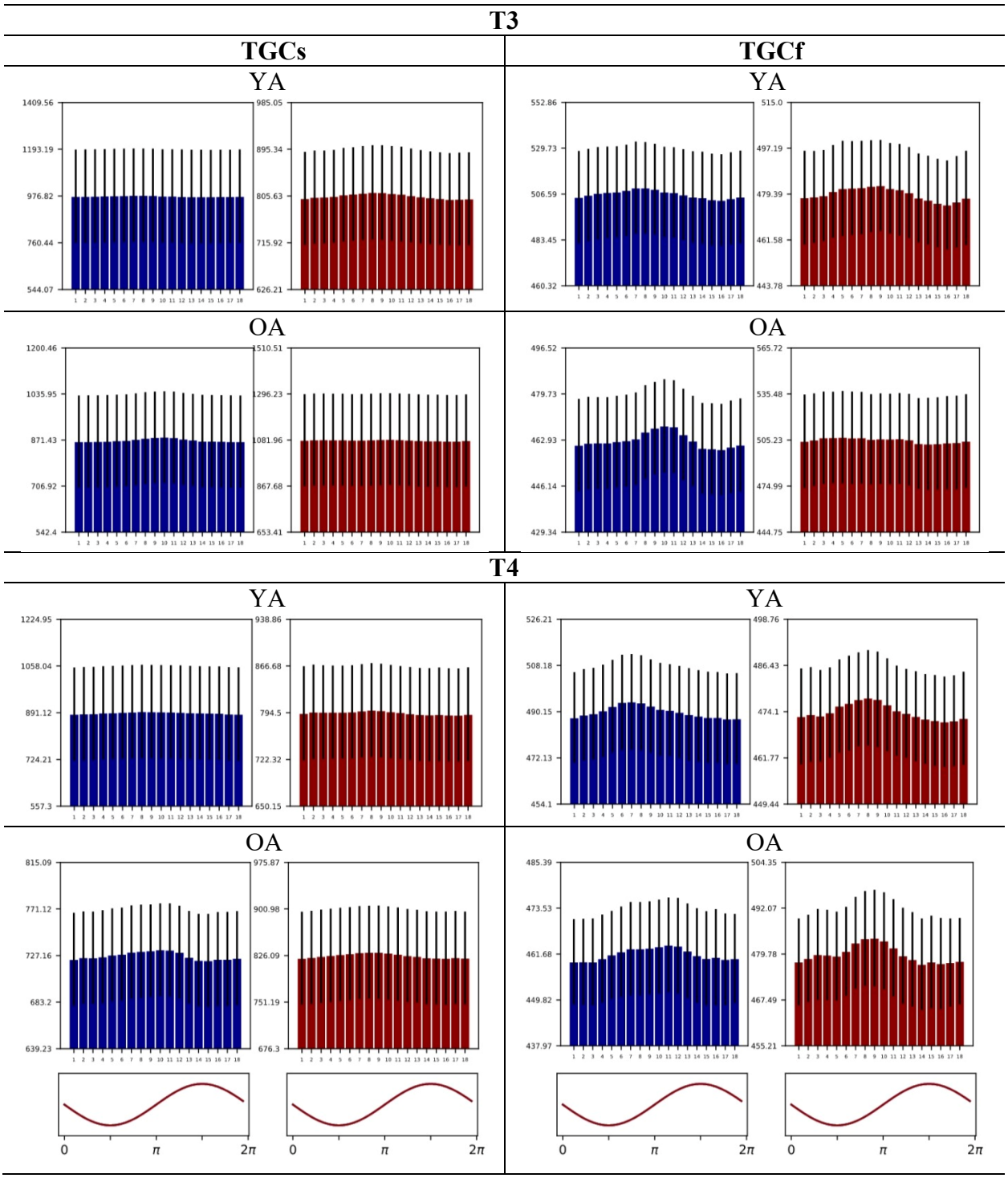

Figure S6. In channels T3 and T4 in YAs (top of each TGC type) and OAs (bottom of each TGC type) mean gamma amplitude plots with theta phase bins for both EEGc (blue) and EEGm (red). 18 phase bins in total, corresponding to sinusoidal theta wave (bottom). YA, younger adult; OA, older adult; TGCs, fixed theta-slower gamma coupling; TGCf, fixed theta-faster gamma coupling; EEGc, control task EEG night; EEGm, memory task EEG night.

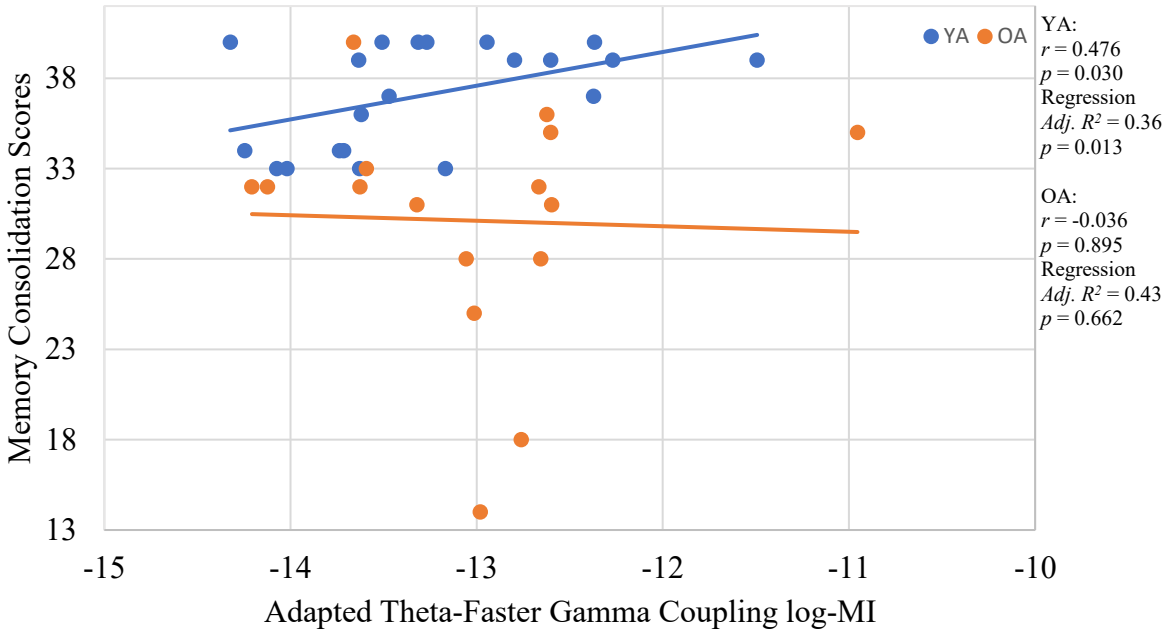

Figure S7. In Fz, correlations between adapted theta-faster gramma coupling (aTGCs) log-MI and memory consolidation that is significant in YAs (N = 21) (r = 0.476, p = 0.030) but not OAs (N = 16) (r = -0.036, p = 0.895). Model 2 of hierarchical multiple linear regression shows a significant prediction in YAs (*Adj.* R^2 = 0.36, ΔR^2 = 0.25, β = 0.50, F (3, 17) = 4.768, p = 0.013), but not OAs (*Adj.* R^2 = 0.43, ΔR^2 = 0.01, β = -0.09, F (3, 12) = 4.970, p = 0.662). Top right legend shows r and p values for Pearson's r correlation, and *Adj.* R^2 and p values below, for model 2 of multiple linear regression, for both YA and OA. Log-MI, log-transformed modulation index; Adj. R^2, adjusted R^2; YA, younger adult; OA, older adults.

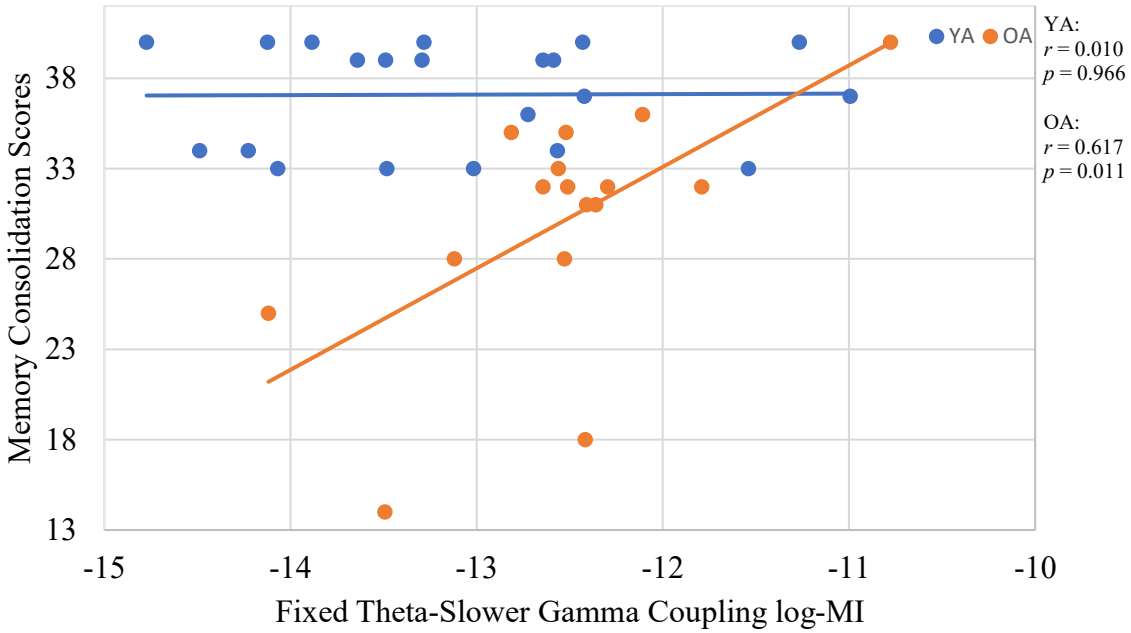

Figure S8. In Cz, correlations between fixed theta-slower gramma coupling (TGCs) log-MI and memory consolidation that is significant in OAs (N = 16) ($r = 0.617$, $p = 0.011$) but not YAs (N = 21) ($r = 0.010$, $p = 0.966$). Top right legend shows r and p values for Pearson's r correlation for both YA and OA. Log-MI, log-transformed modulation index; YA, younger adult; OA, older adults.

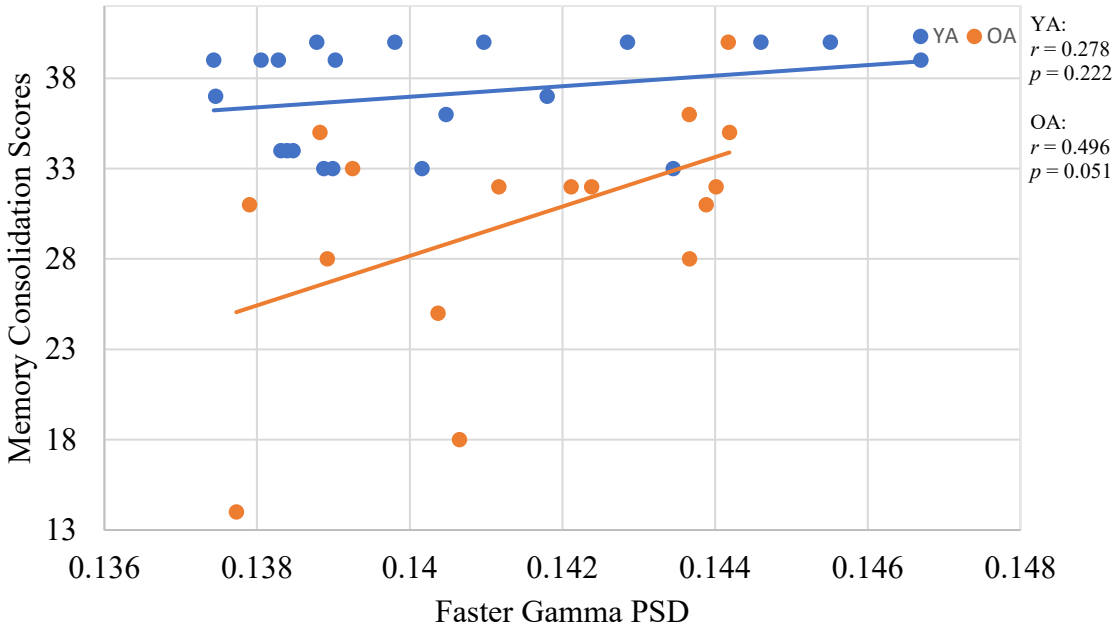

Figure S9. In Cz, correlations between faster gamma PSD and memory consolidation that shows a trend effect in OAs (N = 16) (r = 0.496, p = 0.051) but is not significant in YAs (N = 21) (r = 0.278, p = 0.222). Top right legend shows r and p values for Pearson's r correlation for both YA and OA. PSD, power spectral density; YA, younger adult; OA, older adults.

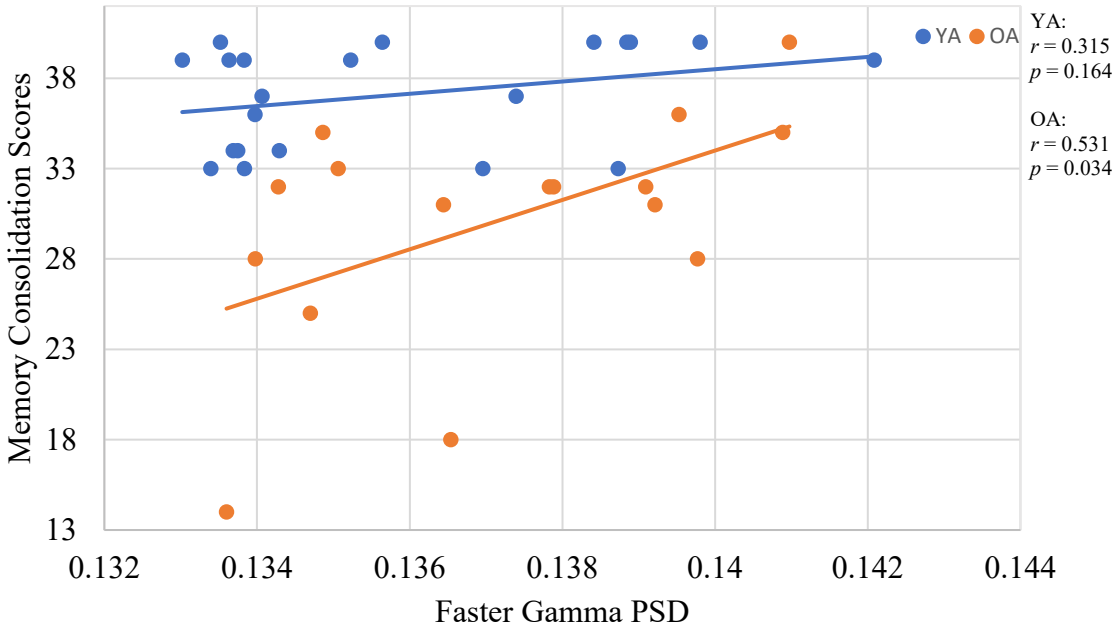

Figure S10. In T3, correlations between faster gamma PSD and memory consolidation that is significant in OAs (N = 16) (r = 0.531, p = 0.034) but not YAs (N = 16) (r = 0.315, p = 0.164). Top right legend shows r and p values for Pearson's r correlation for both YA and OA. Power spectral density, PSD; YA, younger adult; OA, older adults.

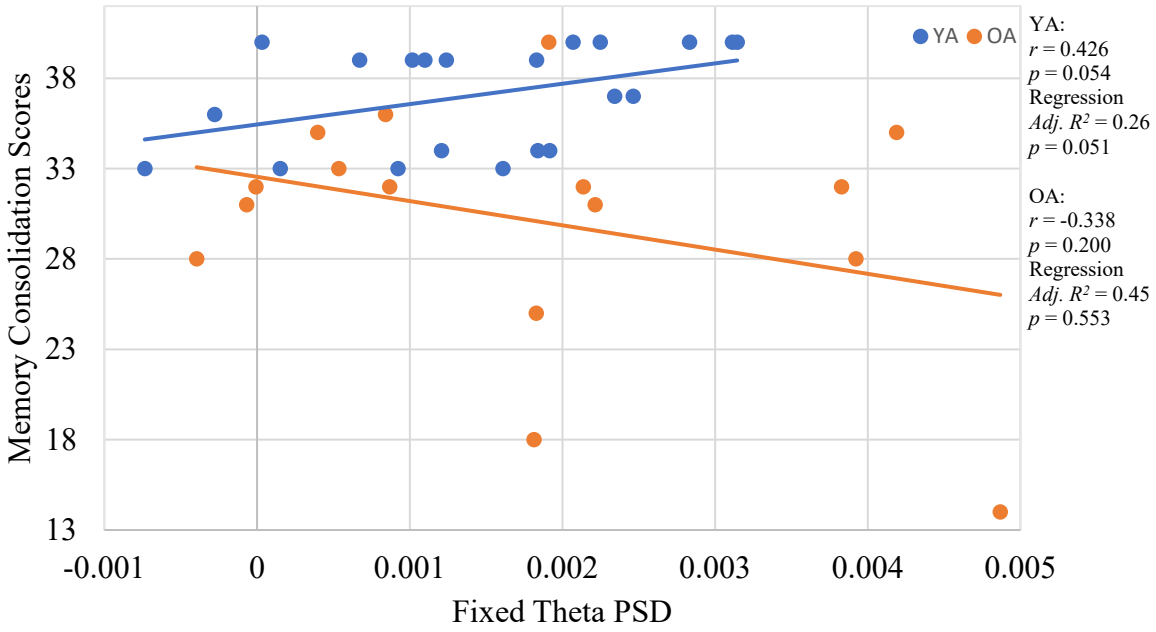

Figure S11. In T3, correlations between fixed theta PSD and memory consolidation that shows a trend effect in YAs (N = 21) (r = 0.426, p = 0.054) but is not significant in OAs (N = 16) (r = -0.338, p = 0.200). Model 2 of hierarchical multiple linear regression shows a trend to predict memory consolidation in YAs (*Adj.* R^2 = 0.26, ΔR^2 = 0.16, β = 0.40, F (3, 17) = 3.384, p = 0.051), but does not significantly predict in OAs (*Adj.* R^2 = 0.45, ΔR^2 = 0.01, β = 0.15, F (3, 12) = 5.097, p = 0.553). Top right legend shows r and p values for Pearson's r correlation, and *Adj.* R^2 and p values below, for model 2 of multiple linear regression, for both YA and OA. PSD, power spectral density; Adj. R^2, adjusted R^2; YA, younger adult; OA, older adults.

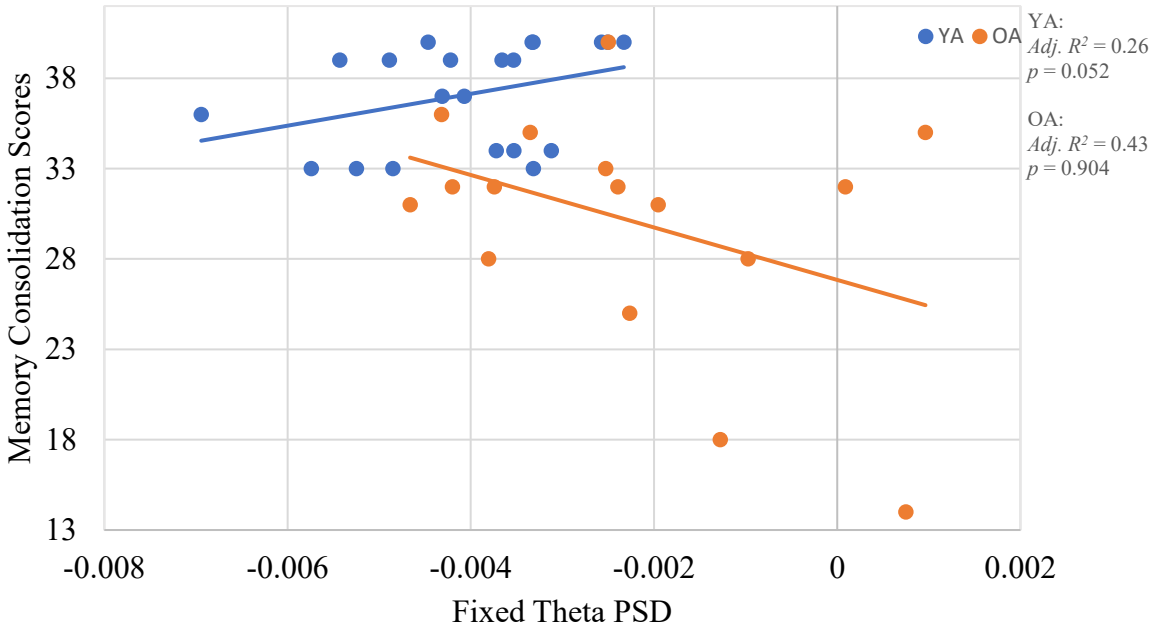

Figure S12. In Cz, model 2 of hierarchical multiple linear regression for fixed theta PSD predicting memory consolidation scores shows a trend in YAs (N = 21) (*Adj.* R^2 = 0.26, ΔR^2 = 0.16, β = 0.41, F (3, 17) = 3.360, p = 0.052), but does not significantly predict memory consolidation in OAs (N = 16) (*Adj.* R^2 = 0.43, ΔR^2 = <0.01, β = -0.03, F (3, 12) = 4.834, p = 0.904). Top right legend shows *Adj.* R^2 and p values for model 2 of multiple linear regression, for both YA and OA. PSD, power spectral density; YA, younger adult; OA, older adults.

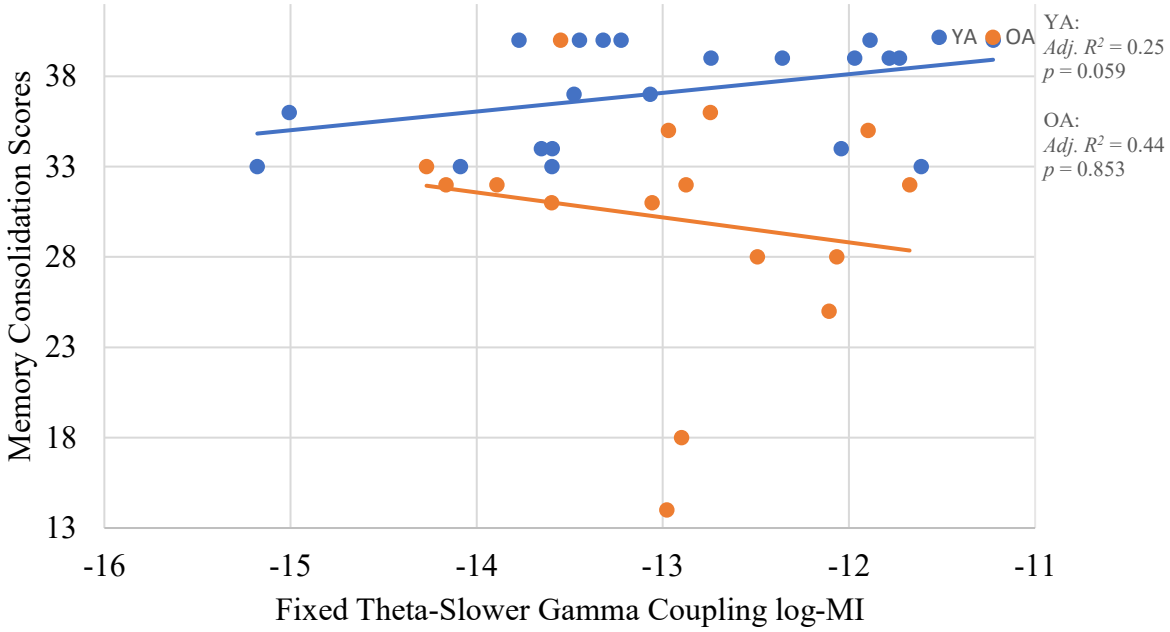

Figure S13. In T4, model 2 of hierarchical multiple linear regression for fixed theta-slower gamma coupling (TGCs) predicting memory consolidation scores shows a trend in YAs (N = 21) (*Adj.* R^2 = 0.25, ΔR^2 = 0.15, β = 0.40, F (3, 17) = 3.248, p = 0.059), but does not significantly predict in OAs (N = 16) (*Adj.* R^2 = 0.44, ΔR^2 = <0.01, β = -0.04, F (3, 12) = 4.949, p = 0.853). Top right legend shows *Adj.* R^2 and p values for model 2 of multiple linear regression, for both YA and OA. Log-MI, log-transformed modulation index; YA, younger adult; OA, older adults.