Age-related differences in the neural dynamics of memory encoding

M. Karl Healey[1], Michael J. Kahana[2]

1 Department of Psychology, Michigan State University, East Lansing, MI, USA
2 Department of Psychology, University of Pennsylvania, Philadelphia, PA, USA

* khealey@msu.edu

Abstract

We examined oscillatory power in electroencephalographic recordings obtained while younger (18-30 years) and older (60+ years) adults studied lists of words for later recall. Power changed in a highly consistent way from word-to-word across the study period. Above 14 Hz, there were virtually no age differences in these neural gradients. But gradients below 14 Hz reliably discriminated between age groups. Older adults with the best memory performance showed the largest departures from the younger adult pattern of neural activity. These results suggest that age differences in the dynamics of neural activity across an encoding period reflect changes in cognitive processing that may compensate for age-related decline.

Introduction

Memory impairments are among the most common complaints of older adults [1]. Much effort has been devoted to identifying the neurocognitive causes of age related memory decline [2, 3]. But one potential source of age differences has received little attention: the ability to sustain encoding processes across a series of events or items that unfold over time [4]. For example, the people you meet during a job interview, the grocery list your spouse dictates over the phone, or which of your medications you have already taken today.

Researchers have studied this aspect of memory using the free recall task, in which participants study a list of sequentially presented items (e.g., words) and then recall the items in any order. The nature of the encoding processes in which participants engage changes from item-to-item as the list is studied [5]. These changes unfold in the brain without any obvious behavioral correlates—they can only be inferred from which items are subsequently remembered and forgotten. Perhaps for this reason, most cognitive aging theories are silent about the contribution of encoding dynamics to memory impairments [3, 6–8].

We argue, however, that there are two general categories of item-to-item changes in cognitive processing that are likely to show age differences. The first category is processes that become less efficient as the list progresses with time due to fatigue [9]. The second category is processes that ramp up as the list goes on such as rehearsing early items in the list [10]. Although differences in such processes are difficult to detect from behavior, they should leave a signature in how neural activity changes while studying a list.

We seek to provide an initial test of the hypothesis that there are age differences in the dynamics of neural activity across the encoding period of a free recall list and that

these processing differences may either contribute to, or compensate for, age-related memory impairment. Our approach was to examine electroencephalographic (EEG) recordings taken while participants study lists for free recall. We analyzed the data by converting raw EEG into the frequency domain and examining how spectral power changes across time during the study period. We then tested for age differences in these across-time changes in spectral power. Finally, we tested whether the neural age differences could predict behavioral age differences in memory performance.

Materials and methods

The data are from the Penn Electrophysiology of Encoding and Retrieval Study (PEERS), an ongoing project aiming to assemble a large database on memory ability in older and younger adults.

0.1 Participants

The present analyses are based on the 172 younger adults (age 17–30) and 36 older adults (age 61-85 years) who had completed Experiment 1 of PEERS as of September 2015. Participants were recruited through a two–stage process. First, we recruited right-handed native English speakers for a single session. Participants who did not make an excess of eye movements during item presentation epochs of the introductory session and had a recall probability of less than 0.8 were invited to participate in the full study. Approximately half of the subjects recruited for the preliminary session moved on to the full study. Older adults were pre-screened for signs of pathology using a detailed medical history and the Short Blessed Test [11].

0.2 PEERS Experiment

The analyses reported here focus on the free recall data from PEERS Experiment 1, which consisted of 7 sessions each of which included 16 free recall lists. For each list, 16 words were presented one at a time on a computer screen followed by an immediate free recall test. Each session ended with a recognition test. The first session and half of the remaining sessions were randomly chosen to include a final free recall test before recognition, in which participants recalled words from any of the lists from the session. The recognition data are not examined here, but details on these data can be found in prior publications [9].

Each word was accompanied by a cue to perform one of two judgment tasks ("Will this item fit into a shoebox?" or "Does this word refer to something living or not living?") or no encoding task. The current task was indicated by the color and typeface of the presented item. There were three conditions: no-task lists (participants did not have to perform judgments with the presented items), single-task lists (all items were presented with the same task), and task-shift lists (items were presented with either task). The first two lists were task-shift lists, and each list started with a different task. The next fourteen lists contained four no-task lists, six single-task lists (three of each of the task), and four task-shift lists. List and task order were counterbalanced across sessions and participants.

Each stimulus was drawn from a pool of 1638 words. Lists were constructed such that varying degrees of semantic relatedness occurred at both adjacent and distant serial positions. Semantic relatedness was determined using the Word Association Space (WAS) model [12]. WAS similarity values were used to group words into four similarity bins (high similarity: cos θ between words ¿ 0.7; medium-high similarity, 0.4 ¡ cos θ ¡ 0.7; medium-low similarity, 0.14 $<$ cos θ ¡ 0.4; low similarity, cos θ ¡ 0.14). Two pairs of

items from each of the four groups were arranged such that one pair occurred at adjacent serial positions and the other pair was separated by at least two other items. For each list, there was a 1500 ms delay before the first word appeared on the screen. Each item was on the screen for 3000 ms, followed by jittered (i.e., variable) inter-stimulus interval of 800-1200 ms (uniform distribution). If the word was associated with a task, participants indicated their response via a keypress. After the last item in the list, there was a jittered delay of 1200-1400 ms, after which a tone sounded, a row of asterisks appeared, and the participant was given 75 seconds to attempt to recall aloud any of the just-presented items.

0.3 Electrophysiological Recordings and Data Processing

We used Netstation to record EEG from Geodesic Sensor Nets (Electrical Geodesics, Inc.) with 129 electrodes digitized at 500 Hz by either the Net Amps 200 or 300 amplifier and referenced to Cz. Recordings were then rereferenced to the average of all electrodes except those with high impedance or poor scalp contact. To eliminate electrical line noise, a fourth order 2 Hz stopband butterworth notch filter was applied at 60 Hz.

To correct artifacts such as eye blinks or electrodes with poor contacts we used independent component analysis (ICA [13]) and an artifact detection/correction algorithm based on [14]. Manual identification of artifactual independent components (IC) can be unreliable [14] and would be impractical given the number and length of sessions in the current study. Therefore, we used an automatic artifact correction algorithm [14]. The algorithm starts with raw EEG. For each channel, several statistics were used to identify channels with severe artifacts. First, electrodes should be moderately correlated with other electrodes due to volume conduction, thus the mean correlation between the channel and all other channels was calculated, and these means were z-scored across electrodes. Channels with z-scores less than -3 were rejected. Second, electrodes with very high or low variance across a session are likely dominated by noise or have poor contact with the scalp, therefore the variance was calculated for each electrode and z-scored across electrodes. Electrodes with a $|z| >= 3$ were rejected. Finally, we expect many electrical signals to be autocorrelated but signals generated by the brain versus noise likely have different forms of autocorrelation. Therefore, the Hurst exponent, which is a measure of long-range autocorrelation was calculated for each electrode and electrodes with a $|z| >= 3$ were rejected. Electrodes that were marked as bad by this procedure were interpolated using EEGLAB's [15] spherical spline interpolation algorithm. The maximum number of ICs that can be reliably estimated depends on the number of samples recorded for each channel. We extracted $c = floor(\sqrt{L/k})$ ICs where L is the number of samples in the session and k is a constant set to 25 (for a discussion of k, see [14, 16]) or the number of non-interpolated channels, which ever was smaller. We then ran EEGLAB's implementation of infomax ICA [13, 15] on the first c principal components of the EEG matrix to decompose it into ICs.

ICs that capture blinks or saccades should be highly correlated with the raw signal from the EOG electrodes. Therefore, for each IC we computed the absolute value of its correlation with each of the six EOG electrodes, retained the maximum of those values and z-scored the maximum correlations across ICs. ICs with $|z| >= 3$ were rejected. ICs that capture artifacts isolated to single electrodes (e.g., an electrode shifting or "popping off") should have high weights for the implicated electrodes but low weights for other electrodes. To identify such ICs, we calculated the kurtosis of the weights across electrodes and excluded any IC with a z-score above +3. Finally, ICs capturing white noise should have a nearly flat power spectrum (vs. the 1/f spectrum expected for neural signals). Therefore, we calculated the absolute value of the slope of the power

spectrum for the frequencies included in the analyses (2–200 Hz) and rejected ICs with $z >= -3$ (i.e., the ones closest to zero slope). Rejected ICs were removed from the matrix and the remaining IC activation time courses were projected back into electrode space. All subsequent analyses were carried out on this corrected EEG data.

To compute spectral power, the corrected EEG data time series for an entire session was convolved with Morlet wavelets (wave number = 6) at each of 60 frequencies logarithmically spaced between 2 Hz and 200 Hz. The resulting power time series were downsampled to 10 Hz. We then defined encoding events by extracting the time period from -200 ms to 3000 ms relative to each item's presentation. For each frequency, a participant's raw power values were z-scored across encoding events separately for each session and each encoding task (no-task, single-task, and task-shift) to remove the effects of these variables. Z-scored power was then averaged across the -200 ms to 3000 ms encoding interval to provide one power value for each study event.

Results

To test for age differences in the dynamics of encoding, we examined Electroencephalographic (EEG) signals recorded while the participants studied the lists. We analyzed spectral power derived from the EEG signals as past research has shown that effective memory encoding is correlated with spectral power in specific frequency bands [17] and that spectral power shows reliable age differences during memory tasks [2].

Figure 1A shows the gradient of spectral power across serial positions in six frequency bands. For younger adults, these gradients are in close agreement with those found in previous work [18]. In the 16–26 Hz, 28–42 Hz, and 44–200 Hz bands, both younger and older adults show high initial power followed by a rapid decline across serial positions, with little age difference. By contrast, the 2–3 Hz, 4–8 Hz, and 10–14 Hz bands all show clear age differences. Just as at higher frequencies, older adults exhibit a steep decline in power across serial positions at lower frequencies, but younger adults exhibit a shallower decline (in the 2–3 Hz band) or a net increase across serial positions (in the 4–8 Hz and 10–14 Hz bands). That is, older adults show higher power than younger adults early in a study list, but the age difference reverses for late-list items.

Fig 1. Age differences in spectral power gradients. A: Spectral power in six frequency bands across serial positions for younger adults versus older adults. Error bars are one standard error of the mean. B: ROC curves created by varying the threshold value of Δ_{EEG} (the change from the power level at the first serial position to the average power of the last 5 items) used to classify a participant as a younger or older adult. Significance was assessed by comparing the observed AUC value with a null distribution created by permuting Δ_{EEG} values across participants 50000 times and running the analysis on each permuted dataset. Note that the y-axis scale differs across panels.

To determine if these neural gradients reliably predict age, we began by condensing the gradients into a single number for each participant by computing the change from the power level at the first serial position to the average power of the last 5 items:

$$\Delta_{EEG} = \frac{\sum_{i=k}^{LL} SP_i}{LL - k + 1} - SP_1, \quad (1)$$

where SP_i is power during the i^{th} list item, LL is the total number of items in a list (here $LL = 16$), and k is the first item included in the late-item average ($k = 5$ for the

analyses reported here). We then tested whether Δ_{EEG} distinguishes older from younger adults by examining receiver operating characteristic (ROC) curves created by varying the criterion value of Δ_{EEG} used to classify a participant as younger versus older. These curves (Figure 1B) show that the 2–3 Hz, 4–8 Hz, and 10–14 Hz gradients were all highly reliable biomarkers of age group. Significance was assessed by finding where the area under the curve (AUC) for the actual ROC curves lay in a null AUC distribution formed by permuting Δ_{EEG} across participants 50000 times and computing a ROC for each permuted dataset.

How do these age differences in neural dynamics relate to age age differences in memory ability? To explore this question, we conducted a median split analysis comparing the older adults with the highest memory scores to the older adults with the lowest memory scores (see the inset in the first panel of Figure 2). As shown in Figure 2, these subgroups showed distinct neural gradients.

In the 2–3 Hz, 4–8 Hz, and 10–14 Hz bands, the older adults with the largest memory impairments showed neural gradients that were more similar to the younger adult pattern of shallowly decreasing (2–3 Hz) or gradual increasing (4–8 Hz and 10–14 Hz) power across serial positions. That is, the best preforming older adults looked *least* like younger adults at the neural level. A similar situation is observed at higher frequencies. Young adults show a steep decrease in power in the 28–42 Hz and 46–200 Hz bands, as do the impaired older adults. But the non-impaired older adults show a shallower decrease. Again, the non-impaired older adults depart most strikingly from the younger adult pattern of neural dynamics.

Fig 2. Spectral power in 6 frequency bands across serial positions for older adults with recall probabilities above (non-impaired) versus below (impaired) the older adult median. Error bars are one standard error of the mean. The inset in the first panel shows kernel density estimates of the distributions of overall probability of recall values for each group. Note that the y-axis scale differs across panels.

ROC analyses on Δ_{EEG} values, analogous to those reported in Figure 1, revealed that no individual frequency band reliably discriminated impaired from non-impaired older adults ($.06 < p < .20$). However, the younger adult pattern is not fully described by any individual frequency band, instead it is characterized by gradual increases across serial positions at 10–14 Hz and sharp decreases for higher frequencies. To capture this pattern we computed the difference between Δ_{EEG} in each lower frequency band, F_i, and the 46–100 Hz band:

$$\Delta_{EEG_{F_i}} - \Delta_{EEG_{44-200Hz}}. \tag{2}$$

Figure 3A compares this measure among younger adults, impaired older adults, and non-impaired older adults for each of the frequency bands. To ease interpretation the $\Delta_{EEG_{F_i}} - \Delta_{EEG_{44-200Hz}}$ values, the small curves next to each data point show the full gradients across serial positions for the current frequency (F_i, solid lines) and 44–200 Hz (dotted lines). $\Delta_{EEG_{F_i}} - \Delta_{EEG_{44-200Hz}}$ represents the difference in the rate of change of these two gradients. At all frequencies, the impaired older adults are numerically closer to the younger adult pattern than are the non-impaired older adults. We conducted an ROC analysis on the ability of this measure to distinguish the two older adult subgroups. The measure for the 2–3 Hz, 4–8 Hz, and 10–14 Hz bands reliably discriminated impaired from non-impaired older adults (Figure 3B). That is, larger deviation from the younger adult pattern of neural dynamics across an encoding episode is a biomarker of successful aging.

Fig 3. Spectral power distinguishes between impaired and non-impaired older adult. A: Mean values of $\Delta_{EEG_{F_i}} - \Delta_{EEG_{44-200Hz}}$ for the 2–3 Hz, 4–8 Hz, 10–14 Hz, 16–26 Hz, and 28–42 Hz bands for the younger adults, older adults with recall probabilities above (non-impaired) the older adult median, and older adults below (impaired) the older adult median. Error bars are one standard error of the mean. To ease interpretation of the $\Delta_{EEG_{F_i}} - \Delta_{EEG_{44-200Hz}}$ values, the small curves next to each data point show the full gradients across serial positions for the current frequency (F_i, solid lines) and 44–200 Hz (dotted lines). $\Delta_{EEG_{F_i}} - \Delta_{EEG_{44-200Hz}}$ represents the difference in the rate of change of these two gradients. B: ROC curves created by varying the threshold value of $\Delta_{EEG_{F_i}} - \Delta_{EEG_{44-200Hz}}$ used to classify a participant as an impaired versus a non-impaired older adult. Significance was assessed by comparing the observed AUC value with a null distribution created by permuting $\Delta_{EEG_{F_i}} - \Delta_{EEG_{44-200Hz}}$ values across participants 50000 times and running the analysis on each permuted dataset.

Discussion

We found evidence of age differences in how neural activity changes while encoding a series of events. For both older and younger adults, high frequency oscillatory power (16–200 Hz) declined rapidly across events [18]. By contrast, power at lower frequencies showed marked age differences. Whereas older adults exhibited rapid power declines at both high and low frequencies, younger adults exhibited shallower decreases (2–3 Hz) and even rapid increases (10–14 Hz) at low frequencies. The rate and direction of change of the gradient at these low frequencies was a highly reliable biomarker of age, as revealed by ROC analyses. These results add neural dynamics across encoding periods to the growing list of age differences in electrophysiology [2, 19–23]. Intriguingly, older adults who preformed best on the memory task showed the largest deviation from the younger adult pattern, particularly in the 4–14 Hz range. This finding complements previous work that has suggested that some aspects of age-related differences in processing compensates for, rather than contributes to, behavioral impairments [24–28].

Here we provide evidence for the general hypothesis that there are age differences in the neural dynamics of encoding. We hope these preliminary results will be useful both in guiding basic science and in designing assessments to detect signs of memory impairment. To conclude, we highlight two important questions for future work and provide some speculations on promising answers.

The first question is which cognitive processes are linked to the observed age difference in neural dynamics? Two general categories of processes strike us as likely candidates: processes that become less efficient as the list progresses with time due to fatigue [5] and processes that ramp up as the list goes on such as rehearsing early items in the list.

The second question is why would age differences in such processes compensate for, rather than exacerbate, memory impairment? In the case of fading efficiency, if older adults are aware they will fatigue across a list, it might make sense for them to strongly engage encoding processes for early items to ensure that at least some items are well-encoded. In the case of rehearsal, it is known that older adults are less likely to rehearse items [10], perhaps because they are impaired at the retrieval processes [4] needed to think back to early list items [29]. If rehearsal is likely to fail, older adults may be well-served by instead focusing on encoding the current item. Indeed, alpha power (corresponding to the 10–14 Hz band used here) has been linked to holding more items in mind [30] and increases in 10–14 Hz power younger adults show across a list may be an index of elaborative encoding or rehearsal [18]. Therefore, the lack of 10–14 Hz increases in our group of non-impaired older adults may indicate that they are not

attempting to engage in elaborative encoding or rehearsal.

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