# Age-Related Changes in the Dynamics of Memory Encoding Processes Provide a Biomarker of Successful Aging

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Abstract

Memory impairments are among the most troubling aspects of cognitive aging. Can older adults change how they encode events to compensate for impairments? We reveal a novel compensatory effect by examining how neural activity changes across protracted study periods. We examined oscillatory power in electroencephalographic recordings obtained while younger (18-30 years) and older (60+ years) adults studied lists of words. 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 compensate for age-related decline.

# Age-Related Changes in the Dynamics of Memory Encoding Processes Provide a Biomarker of Successful Aging

Our memories define us as individuals, they record our personal histories on an autobiographical timeline. Memory is also central to our intellectual lives, as almost every cognitive task requires finding key information in memory. Sadly, our memory tends to get worse as we grow older, as revealed by both self-reports (Newson & Kemps, 2006; Zacks, Hasher, & Li, 2000) and laboratory studies (Craik & Jennings, 1992; Light, 1991; Salthouse, 1991; Stark, Yassa, & Stark, 2010). Identifying age differences in cognitive processing that contribute to, or compensate for, age-related memory impairments is a critical step in developing effective treatments.

One potential source of age differences that has received little attention in the literature arises when we must encode a series of events or items that unfold over time. 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 participants engage changes from item-to-item as the list is studied. Most cognitive aging theories are silent about the contribution of these dynamics to memory impairments (Benjamin, 2010; Hasher & Zacks, 1988; Naveh-Benjamin, 2000; Salthouse, 1996; Stark et al., 2010). Perhaps because age differences in such encoding dynamics are difficult to detect by examining recall behavior, as such behavior is determined not only by encoding but also by retrieval processes. We argue, however, that there are two general categories of item-to-item changes in cognitive processing that are likely to show age differences—age differences that may be detectable by examining neural activity.

First, some processes likely become less efficient as the list progresses. As one example, early list items may be encoded more strongly than later items due to fatigue of

the neural circuitry underlying encoding (Tulving & Rosenbaum, 2006) and these dynamics may show age differences. Indeed, using a model of memory encoding and search that simulates this "primacy gradient" (Lohnas, Polyn, & Kahana, 2015), we found that older adults' encoding efficiency starts out higher than younger adults' but decays rapidly, dropping below the young adult level within a few items (Healey & Kahana, 2016). The second class of processes likely to show age differences are those that ramp up as the list goes on. As an example, younger adults engage in rehearsal (thinking back to early list items) and elaborative encoding (forming connections between current and earlier items). Engagement of these processes should increase across a list, simply because the number of earlier items to think back to increases across the list. There is reason to suspect age differences in the rate at which these processes increase across the list. For example, asking participants to rehearse aloud reveals that whereas rehearsals increase across early serial positions for younger adults (Ward & Maylor, 2005) older adults rehearse fewer items across a list (Ward & Maylor, 2005).

A natural prediction is that any such age-related changes in the dynamics of encoding processes would exacerbate memory impairment. Some cognitive changes, however, are compensatory (Buckner, 2004; Cabeza, Anderson, Locantore, & McIntosh, 2002; Daselaar et al., 2015; Gutchess et al., 2005; Lighthall, Huettel, & Cabeza, 2014; Zimmerman, Hasher, & Goldstein, 2011). Therefore, we raise the possibility that age differences in encoding dynamics are associated with reduced memory impairments, contributing to successful aging. For example, rehearsal and elaborative encoding require retrieving earlier list items (Laming, 2008). Given that older adults have impaired retrieval processes (Healey & Kahana, 2016), attempts to think back to earlier items will often fail and older adults may be well-served by forgoing rehearsal and elaborative encoding in favor of focusing on encoding the current item.

We will test the hypotheses that there are age differences in the dynamics of cognitive processing across a list and that these processing differences may either contribute to, or

compensate for, age-related memory impairment. We have offered two examples of processes that might show age differences in across-item dynamics (encoding efficiency and rehearsal/elaborative encoding). Many other processes might show age differences as well. Any process that changes across encoding should leave its signature in neural activity. Therefore, our approach is to examine neural recordings taken while participants study lists and test for biomarkers of age that take the form of different gradients of neural activity across an encoding period.

## Method

### Participants

The data reported here are from the Penn Electrophysiology of Encoding and Retrieval Study (PEERS). PEERS aims to assemble a large database on the electrophysiological correlates of memory encoding and retrieval. 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. Sample sizes were determined based on previous work showing these sample sizes to provide adequate power to detect the effects of intrest (Healey & Kahana, 2016). The larger sample in the younger adult group was collected to allow for individual differences analyses, which are reported elsewhere (Healey, Crutchley, & Kahana, 2014; Healey & Kahana, 2014). Participants were recruited through a two–stage process. First, we recruited right-handed native English speakers for a single session to introduce participants to EEG recordings and the free recall task. 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 qualified for, and agreed to participate in, the full study. To ensure we do not confound effects of healthy aging with pathological deficits, older adults were extensively pre-screened for signs of pathology using a detailed medical history and the

Short Blessed Test (Katzman et al., 1983). Participants were consented according the University of Pennsylvania's IRB protocol and were compensated for their participation.

**PEERS Experiment 1**

The analysis 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.

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 word 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 described by (Steyvers, Shiffrin, & Nelson, 2004). WAS similarity values were used to group words into four similarity bins (high similarity: $\cos\theta$ between words $> 0.7$; medium–high similarity, $0.4 < \cos\theta < 0.7$; medium-low similarity, $0.14 < \cos\theta < 0.4$; low similarity, $\cos\theta < 0.14$). 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.

If a session was selected for final free recall, following the immediate free recall test from the last list, participants were shown an instruction screen for final free recall, telling them to recall all the items from the preceding lists. After a 5 s delay, a tone sounded and a row of asterisks appeared. Participants had 5 minutes to recall any item from the preceding lists. Each session ended with a recognition test, which is not considered here (for full details see, Lohnas & Kahana, 2013).

**Electrophysiological recordings and data processing**

We used Netstation to record EEG from Geodesic Sensor Nets (Electrical Geodesics, Inc.) with 129 electrodes. The signal from all electrodes was digitized at 500 Hz by either the Net Amps 200 or 300 amplifier and referenced to Cz. Prior to any data processing, recordings were rereferenced to the average of all electrodes except those with high impedance or poor contact with the scalp. To eliminate electrical line noise, a fourth order 2 Hz stopband butterworth notch filter was applied at 60 Hz. We used independent component analysis (ICA, Bell & Sejnowski, 1995; Onton & Makeig, 2006) to identify and correct for EEG artifacts such as voltage deflections caused by blinks or electrodes with poor contacts.

Manual identification of artifactual Independent components (IC) can be unreliable (Nolan, Whelan, & Reilly, 2010) and would be impractical given the number and length of

sessions in the current study. Therefore we used an automatic artifact correction algorithm based on (Nolan et al., 2010). 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 (Delorme & Makeig, 2004) spherical spline interpolation algorithm. The maximum number of ICs that can be reliably estimated depends on the number of samples recorded for each channel. Following (Nolan et al., 2010) 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, Onton & Makeig, 2006) or the number of non-interpolated channels, which ever was smaller. We then ran EEGLAB's implementation of infomax ICA (Bell & Sejnowski, 1995; Delorme & Makeig, 2004) 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 6 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 analysis were carried out on this corrected EEG data.

To compute spectral power, the corrected EEG 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.

### Data Sharing

All behavioral and electrophysiological data analyzed in this report will be made freely available at http://memory.psych.upenn.edu/Electrophysiological_Data.

## Results

Older adults recalled fewer words ($M = 41.37\%, SD = 9.09\%$) than did younger adults ($M = 60.50\%, SD = 13.21\%$), $t(206) = -8.28$, $p < .001$. Do age differences in the dynamics of encoding contribute to this memory deficit? To test for biomarkers of such age differences, we examined Electroencephalographic (EEG) signals recorded while the participants studied the lists. We analyzed spectral power derived from the EEG signals as

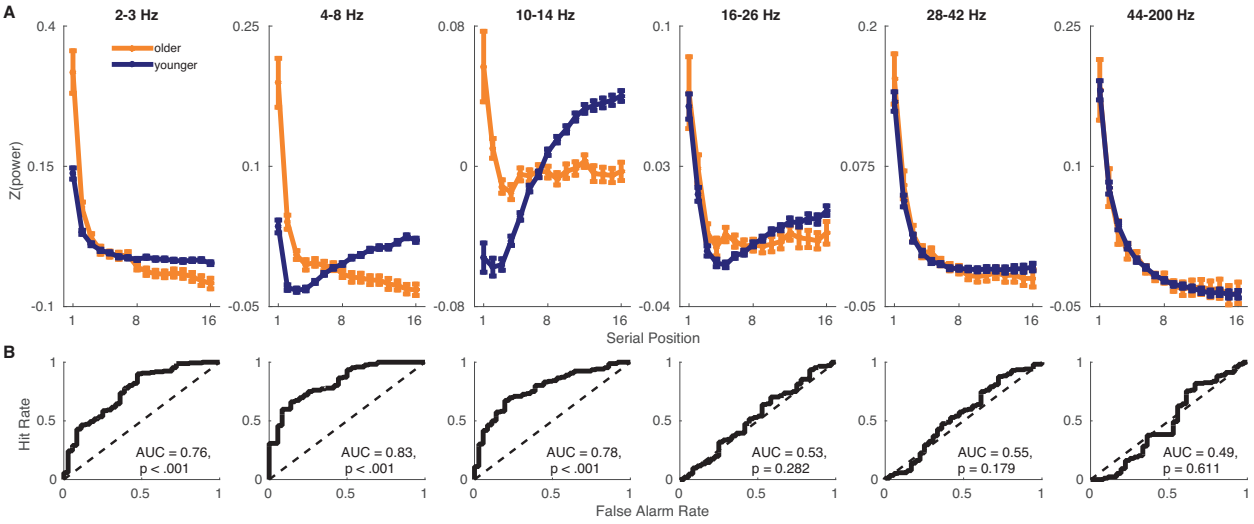


*Figure 1*. **A.** Spectral power in six frequency bands across serial positions for younger adults versus older adults. Error bars are one standard error of the mean. **B.** ROC curves created by varying the threshold value of $\Delta_{EEG}$ (the change from the power level at the first serial position 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 $\Delta_{EEG}$ values across participants 50000 times and running the analysis on each permuted dataset.

past research has shown that effective memory encoding is correlated with spectral power in specific frequency bands (Nyhus & Curran, 2010) and that spectral power shows reliable age differences during memory tasks (Werkle-Bergner, Freunberger, Sander, Lindenberger, & Klimesch, 2012).

Figure 1A shows the gradient of power across serial positions in six frequency bands. For younger adults, these gradients are in close agreement with those found in previous work (Sederberg et al., 2006). 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.

To determine if these neural gradients provide a reliable biomarker of 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:

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

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 we could use $\Delta_{EEG}$ to distinguish older from younger adults by examining receiver operating characteristic (ROC) curves created by varying the criterion value of $\Delta_{EEG}$ 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 robust 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 $\Delta_{EEG}$ across participants 50000 times and computing a ROC for each permuted dataset.

Although striking, these age differences in the dynamics of neural activity are not necessarily related to age differences in recall probability. It is possible that spectral power and memory ability account for non-overlapping portions of the variability in age. Moreover, successful recall is the outcome of many different processes that operate both during encoding and during retrieval, which dilutes the impact of any particular process on behavior. Can we find evidence that age-differences in neural dynamics are related to age-differences in memory despite these challenges?

We conducted mediation analyses to determine if the gradient of spectral power across the encoding period mediates the relationship between age and recall probability. To test for a mediator, one must show that the relationship between the two variables (age

and memory ability), as measured by a regression, is significantly changed by including the proposed mediator ($\Delta_{EEG}$) as a predictor in the equation (Baron & Kenny, 1986). Mediation analyses have been used to test whether various behavioral measures mediate the relationship between age and memory (e.g., Darowski, Helder, Zacks, Hasher, & Hambrick, 2008). To determine if $\Delta_{EEG}$ mediates the age difference in memory ability we ran a mediation analysis at each frequency band. The first step in a mediation analysis is predicting memory ability ($P_{rec}$, i.e., total percent recall across all lists) from age alone:

$$P_{rec} = \beta_0 + \beta_A A_i, \tag{2}$$

where $A$ is a dummy variable coding for age group (younger adult = 0, older adult = 1). This regression provides a coefficient, $\beta_A$, that gives the *total effect* of age on recall probability. The next step is to run a second regression, but this time including $\Delta_{EEG}$ as a predictor in addition to age:

$$P_{rec} = \beta'_0 + \beta'_A A_i + \beta_{\Delta_{EEG}} \Delta_{EEG_i}, \tag{3}$$

where $\beta_{\Delta_{EEG}}$ is the effect of $\Delta_{EEG}$ on recall probability, controlling for age. And $\beta'_A$ is the *direct effect* of age on recall probability controlling for $\Delta_{EEG}$. If $\Delta_{EEG}$ mediates the age deficit, the direct effect, $\beta'_A$, will differ significantly from the total effect, $\beta_A$.

Age-related change in brain activity can be either detrimental or compensatory, as discussed above. That is, the direct effect can either be larger or smaller than the total effect. If the direct effect is more positive than the total effect, it means that statistically controlling for age differences in $\Delta_{EEG}$ decreases the behavioral age deficit, suggesting that age differences in $\Delta_{EEG}$ contribute to the age deficit. It may be, for example, that rapid decline of power is related to rapid fading of encoding efficiency, in which case we would expect more negative values of $\Delta_{EEG}$ (steeper decline) to be related to larger age deficits. By contrast, if the direct effect is more negative than the total effect, it means that

statistically controlling for age differences in $\Delta_{EEG}$ actually increases the behavioral age deficit, suggesting that rapid fading of power has a compensatory effect. It may be, for example, that the increase in power shown by younger adults in the 4–14 Hz range (positive values of $\Delta_{EEG}$) is related to elaborative encoding (Sederberg et al., 2006), and that because older adults are less able to engage these processes (Craik, 2002), those older adults who forgo elaborative encoding (and thus show negative $\Delta_{EEG}$ values), actually do better.

Figure 2 shows the direct effect for each frequency band (i.e., the difference in percent recall between younger and older adults, statistically controlling for $\Delta_{EEG}$ in that band) plotted against the total effect (dotted line). A compensatory effect (direct effect is more negative than the total effect) was observed in the 4–8 Hz and 10–14 Hz bands, suggesting that a rapid change in power across serial positions serves a compensatory function. To determine if this mediation effect was statistically significant, we used a permutation procedure (Taylor & MacKinnon, 2012) to develop a non-parametric distribution under the null hypothesis that $\Delta_{EEG}$ does not mediate the age deficit (specifically, the null is: $\beta'_A = \beta_A$). The gray shaded region in Figure 2 shows the middle 95% of this null distribution of the direct effect, which was formed by permuting the values of $\Delta_{EEG}$ across participants 50000 times and running the mediation analysis on each permuted dataset. Any bars outside the null distribution are significant at $\alpha = .05$, two-tailed. The effect reached significance in both the 4–8 Hz and 10–14 Hz bands.

This mediation effect suggests that neural dynamics may be able to distinguish between older adults who show substantial memory deficits and those who show smaller deficits. We used a median split to divide the older adult sample in to a group of impaired older adults and a group of non-impaired older adults (see the inset in the first panel of Figure 3). As shown in Figure 3, 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

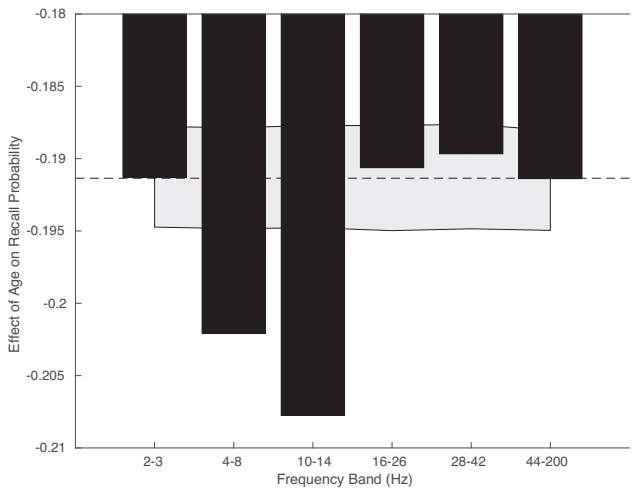


*Figure 2*. The direct effect of age on recall probability after controlling for $\Delta_{EEG}$ independently for each of the six frequency bands. The dotted horizontal line shows the total effect of age on recall probability; values lower than the dotted line indicate that controlling for $\Delta_{EEG}$ *increases* the behavioral age deficit. The gray shaded region shows the middle 95% of a null distribution on the direct effect formed by permuting the values of $\Delta_{EEG}$ across participants 50000 times and running the analysis on each permuted dataset; any bars outside the null distribution are significant at $\alpha = .05$, two-tailed.

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.

Figure 3 is purely descriptive—is deviation from the younger adult pattern a statistically reliable biomarker of degree of age-related memory impairment? ROC analyses on $\Delta_{EEG}$ 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

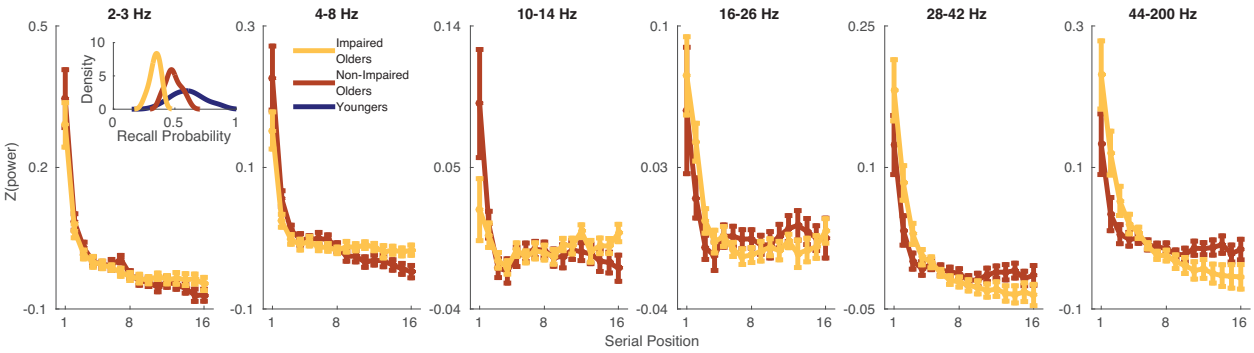


*Figure 3*. Spectral power in 6 frequency bands across serial positions for older adults with recall probabilities above (non-impaired) versus below (impaired) the older adult median. Error bars are one standard error of the mean. The inset in the first panel shows kernel density estimates of the distributions of overall probability of recall values for each group.

the difference between $\Delta_{EEG}$ in each lower frequency band, $F_i$, and the 46–100 Hz band:

$$\Delta_{EEG_{F_i}} - \Delta_{EEG_{44-200Hz}}. \tag{4}$$

Figure 4A 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. To provide a statistical test, 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 robustly discriminated impaired from non-impaired older adults (Figure 4B). That is, larger deviation from the younger adult pattern of neural dynamics across an encoding episode is a biomarker of successful aging.

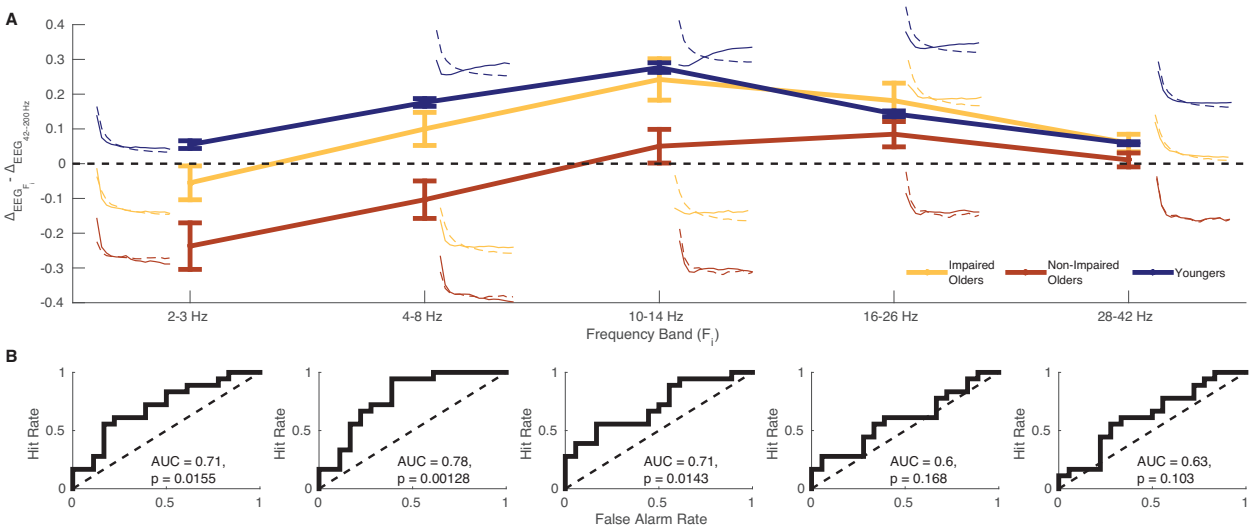


*Figure 4*. **A.** Mean values of $\Delta_{EEG_{F_i}} - \Delta_{EEG_{44-200Hz}}$ for the 2–3 Hz, 4–8 Hz, 10–14 Hz, 16–26 Hz, and 28–42 Hz bands for the younger adults, older adults with recall probabilities above (non-impaired) the older adult median, and older adults below (impaired) the older adult median. Error bars are one standard error of the mean. To ease interpretation 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

There are many episodes in our lives that we want to commit to memory. These episodes, like a grandson's first birthday or a doctor's appointment, are comprised of a series of events that unfold over time. One of the most troubling aspects of growing older is difficulty remembering the details of these important episodes. We found evidence that, regardless of age, neural activity changes in a highly consistent way across a series of events. Oscillatory power at high frequencies (16–200 Hz) was high for early events in an episode and declined rapidly across events (Sederberg et al., 2006). This pattern was virtually identical for younger and older adults. 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 robust 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 (Caplan, Bottomley, Kang, & Dixon, 2015; Roca-Stappung et al., 2012; Sander, Werkle-Bergner, & Lindenberger, 2012; Voytek et al., 2015; Werkle-Bergner et al., 2012; Werkle-Bergner, Müller, Li, & Lindenberger, 2006; Zanto, Hennigan, Östberg, Clapp, & Gazzaley, 2010; Zanto, Toy, & Gazzaley, 2010). But more than providing a biomarker of age per se, we found that age differences in neural dynamics mediated the age-related deficit in recall success.

One might expect those older adults who's neural dynamics deviated most from the young adult pattern to show the largest memory impairments. This was not the case. Instead, deviation from the young adult pattern, particularly in the 4–14 Hz range, predicted better memory. These results suggest that age differences in the dynamics of neural activity across an encoding episode reflect changes in cognitive processing that compensate for age-related decline.

We have tested the general hypothesis that there are age differences in the dynamics of processing across a study list. In doing so we have identified robust biomarkers of age-related memory decline that we hope will be useful both in guiding basic science and in designing assessments to detect signs of memory impairment. An obvious next step is to link these biomarkers to specific underlying cognitive processes. We have suggested two general categories of processes as likely candidates. First, those processes that become less efficient, due to fatigue, as the list progresses. Second, those that ramp up as the list goes on such as rehearsal. 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 (Ward & Maylor, 2005), perhaps because they are impaired at the retrieval processes (Healey & Kahana, 2016) needed to think back to early list items (Laming, 2008). If attempts to rehearse are likely to fail, older adults may be well-served by forgoing rehearsal in favor of focusing on encoding the current item. Indeed, it has been suggested that alpha power (corresponding to the 10–14 Hz band used here) is related to holding more items in mind (Jensen, Gelfand, Kounios, & Lisman, 2002) and that the increases in 10–14 Hz power younger adults show across a list may be an index of elaborative encoding or rehearsal (Sederberg et al., 2006). 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.

## Conclusion

We have shown that the gradient of neural activity across an encoding episode is a reliable biomarker of age. Moreover, those older adults who's neural activity deviated most from the young adult pattern had the smallest age-related memory deficits. Thus, we provide a biomarker of successful aging: a pattern of neural dynamics that identifies older adults who show the smallest memory impairments. This biomarker may be useful in designing assessments to detect signs of memory impairment.

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