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Hippocampal-occipital connectivity reflects autobiographical memory deficits in aphantasia

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Abstract

Aphantasia refers to reduced or absent visual imagery. While most of us can readily recall decade-old personal experiences (autobiographical memories, AM) with vivid mental images, there is a dearth of information about whether the loss of visual imagery in aphantasics affects their AM retrieval. The hippocampus is thought to be a crucial hub in a brain-wide network underlying AM. One important question is whether this network, especially the connectivity of the hippocampus, is altered in aphantasia. In the current study, we tested 14 congenital aphantasics and 16 demographically matched controls in an AM fMRI task to investigate how key brain regions (i.e., hippocampus and visual-perceptual cortices) interact with each other during AM re-experiencing. All participants were interviewed regarding their autobiographical memory to examine their episodic and semantic recall of specific events. Aphantasics reported more difficulties in recalling AM, were less confident about their memories, and described less internal and emotional details than controls. Neurally, aphantasics displayed decreased hippocampal and increased visual-perceptual cortex activation during AM retrieval compared to controls. In addition, controls showed strong negative functional connectivity between the hippocampus and the visual cortex during AM and resting-state functional connectivity between these two brain structures predicted better visualization skills. Our results indicate that visual mental imagery plays an important role in detail-rich vivid AM, and that this type of cognitive function is supported by the functional connection between the hippocampus and the visual-perceptual cortex.

eLife assessment

This **important** work substantially advances our understanding of episodic memory in individuals with aphantasia, and sheds light on the neural underpinnings of episodic memory and mental imagery. The evidence supporting the conclusions is **convincing**, including evidence from a well-established interview paradigm complemented with fMRI to assess neural activation during memory recall. The work will be of broad interest to memory researchers and mental imagery researchers alike.

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Introduction

Our unique and personal memories are stored in autobiographical memories (AM) providing stability and continuity of our self (Svoboda et al., 2006). For most of us, travelling mentally back in time and re-visiting such unique personal events is associated with vivid, detail-rich mental imagery (D'Argembeau & Van der Linden, 2006; Greenberg & Knowlton, 2014). This vivid mental imagery during the re-experiencing of AMs has become a hallmark of autonoetic, episodic AM retrieval. However, up to date, it remains unclear to what extent episodic AM retrieval depends on visual mental imagery and what neural consequences a lack of mental imagery has on episodic AM retrieval. This knowledge gap exists because separating AM retrieval from mental imagery is a complex and challenging task.

One way to address this conundrum is to study people with aphantasia (Zeman et al., 2015). Recent research defines aphantasia as a neuropsychological difference in which people experience a marked reduction or complete lack of voluntary sensory imagery (Monzel et al., 2022). This state is associated with psychophysiological alterations, such as reduced imagery-induced pupil contraction (Kay et al., 2022) and reduced imagery-induced priming effects (Keogh & Pearson, 2018; Monzel, Keidel, et al., 2021). Thus, aphantasics offer the unique opportunity to examine the consequences for episodic AM retrieval in the absence or marked reduction of voluntary imagery. Indeed, a handful of previous studies report convergent evidence that aphantasics report less sensory AM details than controls (Dawes et al., 2020, 2022; Milton et al., 2020; Zeman et al., 2020), which may also be less emotional (Monzel et al., 2023; Wicken et al., 2021). Spatial accuracy, on the other hand, was not found to be impaired (Bainbridge et al., 2021). Yet, task-based functional activity has not been fully explored.

Neurally, the hippocampus has been established as a central brain structure to support the detail-rich episodic AM retrieval in the healthy brain (Bauer et al., 2017; Brown et al., 2018; Burianova et al., 2010; McCormick et al., 2020; Moscovitch et al., 2005), albeit some studies differentiate between hippocampal support for remote and recent autobiographical memories (see Bayley et al. 2016). In fact, hippocampal activity correlates with the vividness of AM recollection (Addis et al., 2004; Sheldon & Levine, 2013) and patients with hippocampal damage show marked deficits in detailed episodic AM retrieval (Miller et al., 2020; Rosenbaum et al., 2008). In addition, neuroimaging studies illuminate that the hippocampus is almost always co-activated with a wider set of brain regions, including the ventromedial prefrontal cortex (vmPFC), lateral and medial parietal cortices, as well as visual-perceptual cortices (Svoboda et al., 2006). Interestingly, especially during the elaboration phase of AM retrieval, when people

engage in the active retrieval of episodic details to a specific AM, the hippocampus exhibits a strong functional connection to the visual-perceptual cortices, suggesting a crucial role of this connection for the embedding of visual-perceptual details into AMs (McCormick et al., 2015).

Yet, not many studies have examined the neural correlates of aphantasia, and none during AM retrieval. Of the little evidence there is, reports converge on a potential hyperactivity of the visual-perceptual cortices in aphantasia (Fulford et al., 2018; Keogh et al., 2020). A prominent theory posits that because of this hyperactivity, small signals elicited during the construction of mental imagery may not be detected (Pearson, 2019, Keogh et al., 2020). Pearson further speculates that since spatial abilities seem to be spared, the hippocampus may not be the underlying cause of aphantasia. In agreement, Bergmann and Ortiz-Tudela (2023) speculate that individuals with aphantasia might lack the ability to reinstate visually precise episodic elements from memory due to altered feedback from the visual cortex. In the same vein, Blomkvist (2022) proposes the extended constructive episodic simulation hypotheses (CESH+) that suggests that imagination and memory rely on similar neural structures, since both represent simulated recombinations of previous impressions. This hypothesis has been supported by shared representations for memory and mental imagery in early visual cortex (Albers et al., 2013; see also Zeidman & Maguire, 2016). Within this framework, the hippocampus is supposed to initiate sensory retrieval processes (e.g., in the visual-perceptual cortices; Danker & Anderson, 2010), comparable to its role in the hippocampal memory indexing theory (Langille & Gallistel, 2020). Blomkvist (2022) speculates that in aphantasics, either the hippocampal memory index or the retrieval processes may be impaired.

Thus, the main goal of our study was to examine the neural correlates of AM deficits associated with aphantasia. We hypothesized that the deficits in AM seen in aphantasia rely on altered involvement of the hippocampus, visual-perceptual cortices and their functional connectivity.

Materials and methods

Participants

In total, 31 healthy individuals with no previous psychiatric or neurological condition participated in this study. 15 congenital aphantasics and 16 matched controls were recruited from the database of the *Aphantasia Research Project Bonn* (Monzel, Keidel, et al., 2021; Monzel, Vetterlein, et al., 2021). Due to technical issues during MRI scanning, one participant (with aphantasia) had to be excluded from the analyses. Groups were matched for basic demographic data, that is, sex, age, and education, as well as intelligence assessed with a short intelligence screening (Baudson & Preckel, 2015) (see **Table 1**). Oral and written informed consent was obtained from all participants prior to the commencement of experimental procedure in accordance with the Declaration of Helsinki (World Medical Association, 2013) and the local ethics board of the University Hospital Bonn.

Vividness of visual imagery questionnaire

Aphantasia is typically assessed with the Vividness of Visual Imagery Questionnaire (VVIQ; Marks, 1973, 1995), a subjective self-report questionnaire that measures how vivid mental scenes can be constructed by an individual. For example, individuals are asked to visualize a sunset with as much details as possible and rate their mental scene based on a 5-point Likert scale (ranging from 'no image at all, you only "know" that you are thinking of the object' to 'perfectly clear and as vivid as normal vision'). Since there are 16 items, the highest score of the VVIQ is 80 indicating the ability to visualize mental images with such vividness as if the event were happening right there and then. The minimum number of points is 16 indicating that an individual reported no mental

	Total (n = 30)	Aphantasics (n = 14)	Controls (n = 16)	Test statistic	*p*	BF_{01}
Age				0.80[a]	.431	2.30
M	29.77	31.47	28.19			
SD	11.36	10.45	12.27			
IQ						
M	93.77	91.73	95.69	0.81[a]	.425	2.29

Table 1a.

Demographic data for aphantasics, controls and the total sample.

SD	13.53	16.61	10.02			
Sex				2.76[b]	.097	0.69
Male (%)	32.3	53.3	81.3			
Female (%)	67.7	46.7	18.8			
Education				1.59[b]	.662	7.90
Secondary school (%)	6.5	6.7	6.3			
A-levels (%)	35.5	40.0	31.3			
University degree (%)	54.8	46.7	62.5			
Doctoral degree (%)	3.2	6.7	0.0			

Note. BF_{01} = Bayes Factor, indicates how much more likely H0 is compared to H1. [a] *t*-test, [b] χ^2-test.

Table 1a. (continued)

image for any of the items at all. Aphantasia is at the lower end of the spectrum of imagery-abilities and usually identified with a VVIQ-score between 16 and 32 (e.g., Dawes et al., 2020, 2022).

Binocular rivalry task

Since self-report questionnaires such as the VVIQ are associated with several drawbacks, such as their reliance on introspection (Schwitzgebel, 2002), we administered a mental imagery priming-based binocular rivalry task to assess mental imagery more objectively (for more details, see Keogh & Pearson, 2018; Pearson et al., 2008). In short, after imagining either red-horizontal or blue-vertical Gabor patterns, participants were presented with a red-horizontal Gabor pattern to one eye and a blue-vertical Gabor pattern to the other eye. Subsequently, participants were asked to indicate which type of Gabor pattern they predominantly observed.

Usually, successful mental imagery leads subjects to select the Gabor pattern which they had just visualized. This selection bias can be transferred into a priming score representing visual imagery strength[1]. Mock stimuli consisting of only red-horizontal or blue-vertical Gabor patterns were displayed in 12.5 % of the trials to be able to detect decisional biases. Previous studies have shown that the binocular rivalry task validly correlated with mental imagery strength (Pearson et al., 2011; Wagner & Monzel, 2023).

Autobiographical interview

Detailed behavioral AM measures were obtained in blinded semi-structured interviews either in-person or online via Zoom (Zoom Video Communications Inc., 2016) using the Autobiographical Interview (AI; Levine et al., 2002). All interviews were conducted in German. During the AI, the interviewer asks the participant to recall five episodic AMs from different life periods: early childhood (up to age 11), adolescent years (ages 11–17), early adulthood (ages 18–35), middle age (35–55), and the previous year. In order to acquire five AMs in every participant, the middle age memory was replaced by another early adulthood memory for participants who were younger than 34 years old (see Levine et al., 2002). Hence, all participants provided the last time period with memories from their previous year. Memories from the first four periods were considered remote, whereas the memory from the previous year was considered recent. The interview is structured so that each memory recollection consists of three parts: free recall, general probe, and specific probe. During free recall, the participants were asked to recall as many details as possible for a memory of their choice that is specific in time and place within the given time period. When the participant came to a natural ending, the free recall was followed by the general and specific probes. During the general probe, the interviewer asked the participant encouragingly to provide any additional details. During the specific probe, specific questions were asked for details about the time, place, perception, and emotion/thoughts of each memory. Then, participants were instructed to rate their recall in terms of their ability to visualize the event on a 6-point Likert scale (ranging from ‘not at all’ to ‘very much’). The interview was audiotaped, and afterwards transcribed and then scored by two independent raters according to the standard protocol (Levine et al., 2002). The interviews were scored after all data had been collected, in random order, and scorers were blind to the group membership of the participant.

For scoring, the memory details were assigned to two broad categories, that is, internal and external details. There were the following subcategories of internal details: internal events (happenings, weather conditions, people present, actions), place (country, city, building, part of room), time (year, month, day, time of the day), perceptual details (visual, auditory, gustatory, tactile, smell, body position), and emotion/thought (emotional state, thoughts). The subcategories for external details were semantic details (factual or general knowledge), external events (other specific events in time and place but different to the main event), repetition (repeated identical information without request), and other details (metacognitive statements, editorializing). In addition, following the standard procedure, an episodic richness score was given for each memory

by the rater on a 7-point Likert scale (ranging from 'not at all' to 'perfect'). Furthermore, we added a novel rating score of confidence to the protocol since many participants indicated very strong belief in the details they provided, while others were insecure about the correctness of their own memories. Confidence scores were again rated on a 7-point Likert scale (ranging from 'not at all' to 'perfect').

Debriefing questions

Following the AI, we asked participants three general questions. These were thought of as open questions to get people to talk about their personal perspective on AM, spatial cognition, and imagination.

1. Typically, how difficult is it for you to recall autobiographical memories?
2. Typically, how difficult is it for you to orient yourself spatially?
3. Typically, how difficult is it for you to use your imagination?

After a free report, participants were asked to rate the difficulty on a Likert-scale from 1 ('very easy') to 6 ('very difficult').

Autobiographical memory fMRI task

The experimental fMRI task was adapted from a previous protocol by McCormick et al. (2015). Two conditions, an AM retrieval task and a simple math task (MA), each consisting of 20 randomized trials, were included in this experiment. During AM trials, cue words, such as 'a party', were presented on the screen for 12 s and participants were instructed to recall a personal event related to the word cue which was specific in time and place (e.g., their 20^{th} birthday party). Participants were asked to press a response button once an AM was retrieved to indicate the time point by which they would start to engage in the AM elaboration phase. For the rest of the trial duration, participants were asked to re-experience the chosen AM and try to recall as many details as possible without speaking out loud. After each AM trial, participants were instructed to rate via button presses whether their retrieval had been vivid or faint. We chose a simple two-button response in order to keep the task as easy as possible. During MA trials, simple addition or subtraction problems, for example, 47 + 19, were presented on the screen for 12 s. Here, participants were instructed to press a response button once the problems were solved and asked to engage in adding 3s to the solutions, for example, (47 + 19) + 3 + + 3, until the trial ended. The MA trials were followed by a rating whether the MA problems had been easy or difficult to solve. For both AM and MA, each trial lasted for 12 s, the maximum time for rating of 3 s, and a jittered inter-stimulus interval (ISI) between 1 to 4 s. Since we were especially interested in the elaboration phase of AM retrieval, for the fMRI analyses, we modelled the last 8 s of each AM and MA trial just before the rating screen appeared.

MRI data acquisition

Anatomical and functional data were acquired at the German Center for Neurodegenerative Diseases (DZNE), Bonn, Germany, using a 3 Tesla MAGNETOM Skyra MRI scanner (Siemens Healthineers, Erlangen, Germany). A mirror was mounted on the 32 channel receiver head coil and was placed in the scanner for the participants to view the stimuli shown on an MRI conditional 30-inch TFT monitor (Medres medical research, Cologne, Germany) placed at the scanner's rear end. The MRI protocol consisted of anatomical, resting-state, and AM task-based fMRI scanning sessions. In addition, we acquired further experimental fMRI and DTI data which are not part of this manuscript. Of note, the resting-state scans were acquired before participants engaged in the AM task in order to prevent reminiscing about personal memories during the resting-state. For the anatomical scans, an in-house developed 0.8 mm isotropic whole-brain T1-weighted sagittal oriented multi-echo magnetization prepared rapid acquisition gradient echo (MEMPRAGE; Brenner et al., 2014) was employed with the following parameters: TR = 2.56 s, TEs

= 1.72/3.44/5.16/6.88 ms, TA = 6:48, matrix = 320 x 320 x 224, readout pixel bandwidth = 680 Hz/Pixel, CAIPIRINHA mode. Resting-state (190 volumes, TA = 7 min) and AM task-based fMRI scans (460 volumes, TA = 15 min) were acquired using an interleaved multi-slice 3.5 mm isotropic echo planar imaging (EPI) sequence with TR = 2 s, TE = 30 ms, matrix = 64 x 64 x 39, readout pixel bandwidth = 2112 Hz/Pixel (see Jessen et al., 2018). The images were obtained in an oblique-axial slice orientation along the anterior-posterior commissure line. During resting-state, the participants were asked to close their eyes and to think about nothing at all. The first 5 frames of each functional session were excluded for the scanner to reach equilibrium. Before each functional session an optimal B0 shim was determined and individually mapped by 2-echo gradient echo (GRE) with same voxel resolution and positioning for later post-processing.

Manual segmentation of the hippocampus

Since our main goal was to assess hippocampal involvement during AM retrieval in aphantasia, we sought to examine in depth whether there were any group differences in hippocampal activation in respect to the hemispheric laterality or along its long-axis. For this reason, we segmented the hippocampus based on the T1 structural images using ITKSnap (*www.itksnap.org*, Version 3.8). Although we did not segment specific hippocampal subfields, our masks included the dentate gyrus, CA1-4, subiculum and pre- and parasubiculum. Whole masks of the left and right hippocampus were segmented manually in their respective native space and were divided afterwards into anterior and posterior portions, using the location of the uncus as boundary.

fMRI preprocessing

SPM12 (Statistical Parametric Mapping 12) software package (*www.fil.ion.ucl.ac.uk/spm/*) on MATLAB v19a (MathWorks) computing platform (*https://matlab.mathworks.com/*) was used to perform resting-state and AM task-based fMRI data preprocessing. The anatomical T1w RMS of all MEMPRAGE's echoes and functional 2D-EPI images were reoriented along the anterior-posterior commissure axis. The phase and magnitude images within the field maps were applied to calculate the voxel displacement maps (VDM) for geometrical correction of the distorted EPI images. The echo times were set to 4.92 ms (short) and 7.38 ms (long). The total EPI readout time was 34.56 ms. The calculated EPI and VDMs were applied to the functional scans for realignment and unwarping. The functional scans were then co-registered to the segmented bias corrected T1 scans.

Whole-brain differences between groups were evaluated. Thus, co-registered scans were normalized to the Montreal Neurological Institute (MNI) space and a Gaussian smoothing kernel of 8 mm FWHM was applied. In addition, for functional connectivity analyses, denoising was applied using a linear regression model of potential confounding effects (global white matter signal, CSF signal, and ART scrubbing parameters) in the BOLD signal using CONN software package v20.b (*www.nitrc.org/projects/conn/*). Temporal band pass filter was set from 0.01 to infinite to further minimize artifacts.

Statistical analyses

Behavioral analyses

Two samples t-tests were calculated to assess differences in the priming scores of aphantasics and controls in the binocular rivalry task. One sample t-tests were used to distinguish the performances of both groups from chance. To assess differences of Autobiographical Interview scores between aphantasics and controls, a 2×2×2 ANOVA with post-hoc t-tests were calculated with type of memory details (internal vs. external) and memory recency (remote vs. recent) as within-subject factors and group (aphantasics vs. controls) as between-subject factor. Afterwards, Bonferroni-corrected t-tests were conducted for specific internal (time, place, internal event,

perception, emotion) and external (external event, semantic, repetition, other) memory details. Differences in memory ratings (confidence, episodic richness), self-reported visualization scores, debriefing questions, and behavioral responses during fMRI scanning were also assessed via two sample t-tests.

Hippocampal activity associated with autobiographical memory in native space

In order to examine hippocampal activity associated with autobiographical memory, we extracted signal intensity values for both AM and MA trials for each participant for our manually segmented anatomical masks of the left and right, anterior and posterior portions of the hippocampus using the MATLAB-based Response Exploration toolbox (REX; *www.nitrc.org/projects/rex/*). We then calculated for each participant for each anatomical mask the difference between AM and MA signal intensities. Afterwards, group differences between aphantasics and controls with respect to the laterality effects and effects between the anterior and posterior hippocampus were assessed using a 2-way ANOVA with a post-hoc Tukey's multiple comparison test, applying a significance threshold of $\alpha = .05$.

Whole-brain fMRI activation analyses

After focusing on the hippocampus, we examined group differences of whole-brain activation associated with AM and MA following the standard GLM procedure in SPM12. Owing to the prominence of mental imagery during the elaboration phase of AM retrieval, we analyzed the last 8 s of the AM and MA trials prior to the display of the vividness rating. These trials were modelled as mini blocks in the GLM with motion correction regressors included as covariate of no interest. We specified our main contrast of interest, i.e., AM versus MA on the first level, which was then brought to the second group level using a one-sample t-test. Finally, the activation maps of the two groups were compared using a two-sample t-test. For whole-brain analysis, we applied a significance threshold of $p < .001$, and voxel cluster size of 10 adjacent voxels, uncorrected.

ROI-to-ROI functional connectivity analyses

One of our main a priori hypotheses stated that the hippocampus and the visual-perceptual cortex show differential engagement during AM retrieval associated with aphantasia which was confirmed by our whole-brain activation analyses. Based on these results, we sought to examine the functional connectivity between those two areas. Towards this end, we created regions of interest (ROIs, spheres with a diameter of 10 mm consisting of 536 voxels) around the three peaks of the activation differences, following the whole-brain fMRI activation analyses, using the MarsBaR HBM toolbox (Brett et al., 2002). The ROIs comprised (1) the right hippocampus, MNI: x = 39, y = -31, z = -13, (2) the right visual cortex, MNI: x = 12, y = -79, z = 5, and (3) the left visual cortex, MNI: x = -9, y = -76, z = 29. Using CONN, we examined functional connectivity (i.e., Generalized Psycho-Physiological Interactions, weighted general linear model with bivariate correlation) between the hippocampal ROI and the ROIs situated in the visual-perceptual cortex during AM task-based fMRI and during resting-state. Furthermore, in order to examine how well functional connectivity between hippocampus and the visual cortex reflected an individuals' ability to visualize mental events, we examined a regression model with the visualization scores of the AI as criterion and resting state connectivity values, group allocation and the interaction term of the connectivity values and group allocation as predictors. For these a priori driven analyses, we applied a significance threshold of $p < .05$, small volume corrected, and a voxel cluster threshold of 10 adjacent voxels.

Results

VVIQ and binocular rivalry task

Aphantasics (M = 16.57, SD = 1.02), scored significantly lower on the VVIQ than controls (M = 62.94, SD = 8.71), $t(15.47) = 21.12$, $p < .001$, $d = 7.23$. Furthermore, aphantasics and controls differed in the priming score of the binocular rivalry task, $t(18.04) = 2.41$, $p = .027$, $d = 0.87$. While controls were primed by their own mental imagery in 61.3 % (SD = 13.1 %) of the trials, aphantasics were only primed 52.6 % (SD = 4.9 %) of the time. In fact, the performance of controls differed significantly from chance, $t(14) = 3.34$, $p = .005$, $d = 0.86$, whereas performance of aphantasics did not, $t(13) = 1.96$, $p = .072$. Moreover, the VVIQ scores correlated positively with the performance on the binocular rivalry task, $r(28) = .43$, $p = .022$. For the mock trials, no significant differences in priming scores were found between groups, $t(28) = 0.86$, $p = .396$, or related to chance (aphantasics: $t(13) = 0.74$, $p = .475$; controls: $t(15) = 0.42$, $p = .682$). These findings validate our groups by indicating that visual imagery strength was diminished in aphantasics.

Autobiographical Interview

Regarding the AI, we found significant main effects of memory period, $F(1, 27) = 11.88$, $p = .002$, $\eta_p^2 = .31$, type of memory details, $F(1, 27) = 189.03$, $p < .001$, $\eta_p^2 = .88$, and group, $F(1, 27) = 9.98$, $p = .004$, $\eta_p^2 = .27$. When the other conditions were collapsed, aphantasics (M = 26.29, SD = 9.58) described less memory details than controls (M = 38.36, SD = 10.99). For aphantasics and controls combined, more details were reported for recent (M = 35.17, SD = 14.19) than remote memories (M = 29.06, SD = 11.12), and internal details (M = 43.59, SD = 17.91) were reported more often than external details (M = 20.64, SD = 8.94). More importantly, a 2-way interaction was found between type of memory details and group, $F(1, 27) = 54.09$, $p < .001$, $\eta_p^2 = .67$, indicating that aphantasics reported significantly less internal memory details, $t(27) = 5.07$, $p < .001$, $d = 1.83$, but not significantly less external memory details, $t(27) = 0.13$, $p = .898$, compared to controls (see **Figure 1b**). No 2-way interaction between memory period and group, $F(1, 27) = 0.62$, $p = .439$, and no 3-way interaction between memory period, group and type of memory details was found, $F(1, 27) = 3.87$, $p = .060$.

Based on the interaction effect between group and type of memory details, we compared specific categories of internal and external memory details between the groups. For internal details and in comparison to controls, aphantasics reported less internal events, $t(27) = 3.22$, $p = .016$, $d = 1.17$, less emotional details, $t(27) = 4.40$, $p < .001$, $d = 1.59$, less perceptual details, $t(27) = 4.95$, $p < .001$, $d = 1.79$, and less details regarding time, $t(27) = 5.27$, $p < .001$, $d = 1.90$, and place, $t(27) = 3.31$, $p < .013$, $d = 1.20$ (see **Figure 1c**). On the other hand, no significant differences were found for external details, including external events, $t(27) = 0.71$, $p > .999$, semantic details, $t(27) = 0.02$, $p > .999$, repetition, $t(27) = 0.46$, $p > .999$, and other details, $t(27) = 0.45$, $p > .999$ (see **Figure 1C**). Regarding the rating scales, we found that aphantasics showed less episodic richness, $t(27) = 7.50$, $p < .001$, $d = 2.71$, and less memory confidence, $t(27) = 5.85$, $p < .001$, $d = 2.11$ (see **Figure 1a**) as well as lower self-reported visualization scores, $t(27) = 11.92$, $p < .001$, $d = 4.30$, than controls.

Debriefing questions

The debriefing questions were employed as a way for participants to reflect on their own cognitive abilities. Of note, these were not meant to represent or replace necessary future experiments. There were stark differences between the groups in how they answered our debriefing questions. While aphantasics reported that they typically have greater difficulty to recall autobiographical memories, $t(27) = 6.20$, $p < .001$, $d = 2.31$, and to use their imagination in daily life, $t(24) = 10.18$, $p < .001$, $d = 3.93$, they did not report difficulties in spatial orientation, $t(27) = 0.62$, $p = .541$, $d = 0.23$.

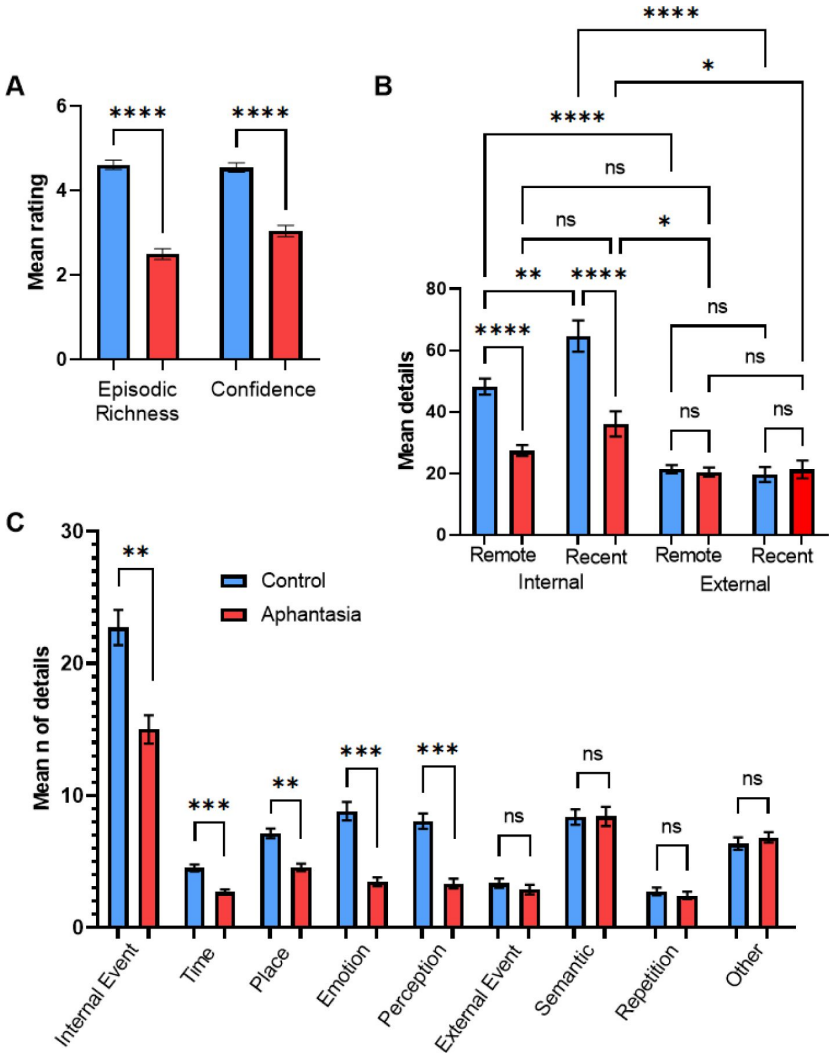

Figure 1.

AM deficits associated with aphantasia.

(A) Mean amount (± SEM) of episodic richness and confidence in the Autobiographical Interview for controls and aphantasics. (B) Mean amount (± SEM) of internal details and external details for recent and remote memories. (C) Mean amount (± SEM) of specific internal and external memory details for aphantasics and controls. * p < .05, ** p < .01, *** p < .001, **** p < .0001, n.s. = non-significant.

Behavioral results of the fMRI AM task

We found stark differences for the vividness response between groups, $t(28) = 5.29$, $p < .001$. While controls reported in 86 % (*SD* = 26 %) of trials that their AM retrieval had been vivid, aphantasics indicated only in 20 % (*SD* = 20 %) of trials that their AM retrieval had been vivid. Moreover, aphantasics responded slower (*M* = 1.34 s, *SD* = 0.38 s) than controls (*M* = 1.00 s, *SD* = 0.29 s) when they were asked whether their retrieved memories were vivid or faint, $t(28) = 2.78$, $p = .009$, possibly reflecting uncertainty in their response. In contrast, there were no significant differences between groups during the MA trials, neither on the easy/hard response, $t(28) = 1.16$, $p = .255$, nor on the reaction times, $t(28) = 0.58$, $p = .567$.

In addition, aphantasics and controls did not differ significantly in their time searching for a memory in AM trials, $t(19) = 1.03$, $p = .315$. On average, aphantasics spent 3.42 s (*SD* = 0.74 s) and controls spent 3.15 s (*SD* = 0.48 s). Furthermore, both groups did not differ in their speed to solve the math problems, $t(23) = 0.09$, $p = .926$. Aphantasics spent 3.87 s (*SD* = 0.97 s) to solve a math problem and controls spent 3.90 s (*SD* = 0.72 s). For the button press, there were 9 % missing values in AM trials and 7 % missing values in MA trials with no significant differences of missing values between groups, neither for AM trials, $t(19.98) = 1.11$, $p = .281$, nor for MA trials, $t(18.13) = 0.52$, $p = .609$.

Native space differences in hippocampal activation during AM retrieval

First, we sought to examine the hippocampal activation associated with AM in aphantasics and controls (see **Figure 2**). The extracted fMRI signals illustrated stark group differences in hippocampal AM-associated activation, $F(17, 252) = 3.03$, $p < .001$. Aphantasics showed reduced activation of bilateral hippocampi, including the left anterior ($p = .033$), left posterior ($p = .027$), right anterior ($p = .047$), and right posterior hippocampus ($p = .025$). There was no laterality effect nor differences along the pattern of activation down the anterior-posterior axis between the groups (all $p > .05$). These findings indicate that the behavioral AM deficit associated with aphantasia is reflected neurally by a reduced bilateral hippocampal activation.

Activation patterns associated with AM retrieval

Second, we examined whole-brain activation during AM retrieval of both groups and the results are displayed in **Figure 3a** and b. Additionally, the peak coordinates of AM and MA activation for aphantasics and controls are shown in **Table 1** and **2**, respectively.

Overall, both groups showed greater activation in all areas typically associated with AM, including bilateral hippocampus, vmPFC, and medial/lateral parietal regions, during AM retrieval. When examining the group differences, aphantasics displayed greater activation in bilateral visual-perceptual regions (maximum in lingual gyrus) in the occipital lobe than controls, $t(28) = 4.41$, $p < .001$ (MNI: right visual cortex: x = 12, y = -79, z = 5; left visual cortex: x = -9, y = -76, z = 29, see **Figure 3c**, d, and e). In contrast, controls showed greater activation in the right hippocampus than aphantasics, $t(28) = 3.77$, $p < .001$ (MNI: x = 39, y = -31, z = -13). An additional correlational analysis revealed that those participants with higher visual-perceptual cortex activation had less hippocampal activation, $r(28) = -.39$, $p = .041$, indicating that there was a trade-off between increased visual-perceptual cortex activation and decreased hippocampal activation.

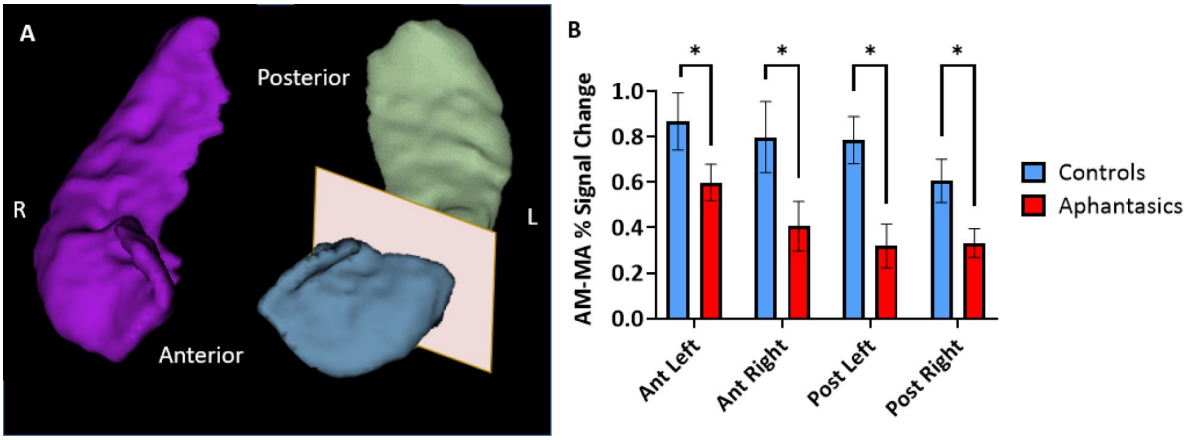

Figure 2.

Reduced hippocampal activity during AM retrieval associated with aphantasia.

The signal intensities during AM and MA were extracted from anatomical hippocampal masks created from each individual participant. (A) An example of a 3D reconstruction of the hippocampus, separated into anterior and posterior portions for the left hippocampus. (B) The comparison between the percentage of signal change during the AM and MA tasks in the hippocampus of aphantasics and controls. Aphantasics show reduced differentiation between AM and MA than controls in all portions of the hippocampus. * $p < .05$.

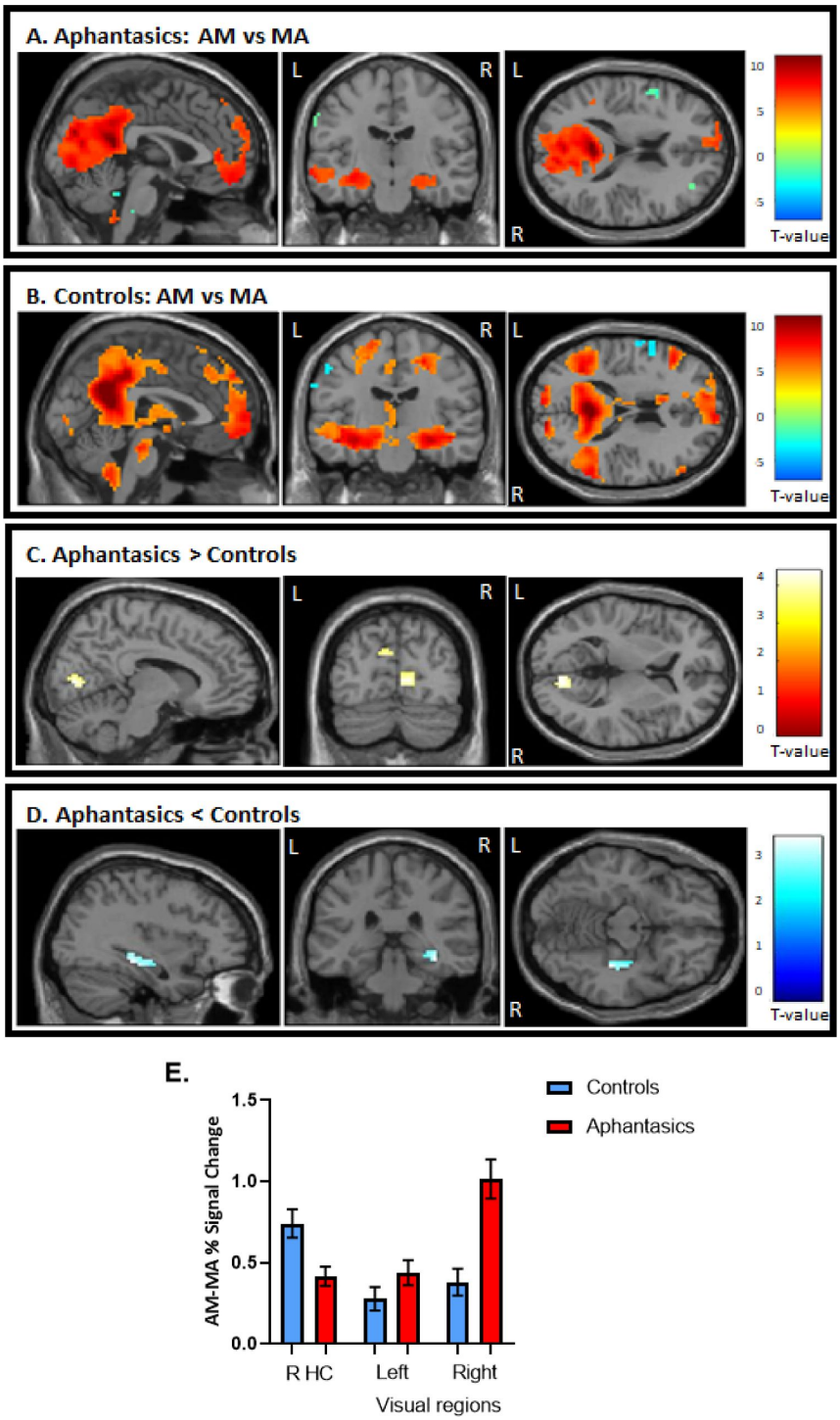

Figure 3.

Activation during AM retrieval task.

(A) Stronger activated cortical regions during AM retrieval (in warm colors) in comparison to math problem solving (in cool colors) in aphantasics and (B) controls. (C) Aphantasics showed greater activation in visual-perceptual cortices than controls, and (D) controls showed stronger activation in the right posterior hippocampus than aphantasics. Images are thresholded at $p < .001$, cluster size 10, uncorrected, except (D) which is thresholded at $p < .01$, cluster size 10, for display purposes only (i.e., the peak voxel and adjacent 10 voxels also survived $p < 0{,}001$, uncorrected). (E) The percentage of signal change for the contrast AM versus MA were extracted from the peaks of activated voxels, each with 1 mm sphere for display purposes.

Region	Hemisphere	MNI Coordinates			Voxels	T-value
		X	Y	Z		
Activation AM > MA						
Posterior Cingulate Gyrus	Right	18	-57	11	4657	11.00
Parahippocampual Gyrus*	Left	-21	-31	-13		9.06
Hippocampus	Left	-27	-17	-19	205	8.40
Superior Frontal Gyrus	Left	-12	47	50	926	8.38
Angular Gyrus	Left	-42	-55	23	165	7.88
Lateral Orbitofrontal Cortex	Left	-42	38	-16	208	7.58
Hippocampus	Right	18	-37	-1	199	6.89
Cerebellum	Right	15	-79	-37	109	6.33
Brainstem	Right	3	-46	-52	43	6.03
Parahippocampual Gyrus*	Right	24	-31	-13		6.02
Middle Temporal Gyrus	Right	60	2	--19	76	5.27
Supramarginal Gyrus	Right	54	-58	32	26	4.99
Middle Frontal Gyrus	Left	-39	20	50	12	4.52
Activation MA > AM						
Precuneus	Left	-18	-58	41	594	-3.85
Inferior Temporal Gyrus	Right	51	-46	-13	123	-3.85
Precuneus	Right	24	-49	53	718	-3.85
Insula	Left	-30	23	11	48	-3.85
Inferior Temporal Gyrus	Left	-51	-49	-13	67	-3.86
Cerebellum	Right	30	-67	-52	27	-3.87
Middle Frontal Gyrus	Right	33	41	17	34	-3.87
Superior Frontal Gyrus	Right	30	5	59	52	-3.87
Inferior Frontal Gyrus	Right	54	14	29	35	-3.88
Insula	Right	39	11	8	51	-3.88
Inferior Frontal Gyrus	Left	-57	11	26	182	-3.88
Lateral Globus Pallidus	Right	23	-7	14	14	-3.92
Cerebellum	Left	-24	-64	-46	16	-3.93

*Sub-cluster level, Cluster size = 10 voxels, p-value = 0.001

Table 1.

Peak coordinates of the AM and MA activation for Aphantasia.

Region	Hemisphere	MNI Coordinates			Voxels	T-value
		X	Y	Z		
Activation AM > MA						
Parahippocampal Gyrus	Right	27	-28	-19	11319	12.41
Parahippocampal Gyrus*	Left	-24	-25	-16		9.01
Cerebellum	Left	-18	-76	-37	108	7.67
Anterior Cingulate	Right	9	35	11	13	7.01
Medial Frontal Gyrus	Right	18	32	29	233	6.93
Inferior Frontal Gyrus	Right	60	32	11	53	5.94
Hippocampus	Left	-36	-22	-16	252	5.64
Hippocampus	Right	27	-22	-16	233	5.28
Hypothalamus	Right	3	-4	-10	16	4.93
Activation MA > AM						
Post Central Gyrus	Left	-33	-43	62	643	-3.73
Precuneus	Right	21	-52	53	483	-3.74
Inferior Frontal Gyrus	Right	51	8	26	16	-3.74
Middle Occipital Gyrus	Right	33	-82	2	26	-3.75
Middle Temporal Gyrus	Left	-51	-58	-1	18	-3.76

*Sub-cluster level, Cluster size = 10 voxels, p-value = 0.001

Table 2.

Peak coordinates of the AM and MA activation for healthy controls.

Exploring functional connectivity of hippocampus and visual-perceptual cortices during AM

The whole-brain analyses strengthened our hypothesis that a core difference between aphantasics and controls lies in the interplay between the visual-perceptual cortex and the hippocampus. To test this interplay, in a third step, we examined functional connectivity between the peak differences of the hippocampus and the visual-perceptual cortex during AM retrieval (see **Figure 4**). We found a group difference in functional connectivity between the right hippocampus and left visual-perceptual cortices, $t(28) = 2.65$, $p = .006$. Interestingly, while aphantasics show almost no functional connectivity between those two ROIs, control displayed a strong negative connectivity between the two locations, $p = .013$.

Exploring functional connectivity of hippocampus and visual-perceptual cortices during resting-state

Lastly, we examined resting-state connectivity between our identified ROIs in the right posterior hippocampus and left and right visual-perceptual cortex. There was no group difference during resting-state between these ROIs. In order to examine whether resting-state functional connectivity carried information about one's ability to visualize AMs, we added the visualization scores as a regressor of interest in our model. While connectivity alone did not predict the visualization scores in the interview, $F(14, 40) = 1.651$, $p = .391$, $\beta = –.06$, we found a main effect of group, $F(3, 40) = 353.2$, $p < .001$, $\beta = .92$, and an interaction between group and connectivity, $F(3, 54) = 305.1$, $p < .001$, $\beta = .26$. Interestingly, for controls, we found a positive correlation between the resting-state connectivity of the right hippocampus and the visual cortex and the visualization scores from the interview, $r(13) = .65$, $p = .011$ (see **Figure 5**). On the other hand, for aphantasics, we found a negative correlation between the resting-state connectivity of the right hippocampus and the visual cortex and the visualization scores from the interview, $r(14) = -.57$, $p = .027$.

In sum, our fMRI results indicate that the impaired AM retrieval associated with aphantasia is reflected by functional alterations of the hippocampus and visual-perceptual cortex, as well as the interaction between them.

Discussion

In this study, we set out to examine the neural correlates of episodic AM retrieval in aphantasia as a way to examine the influence of visual imagery on episodic AM. In line with previous reports, we found that aphantasics reported less sensory details during AM retrieval regardless of the recency of memory (cf. Dawes et al., 2020; 2022; Milton et al., 2020; Zeman et al., 2020). Strikingly, the deficit in constructing visual imagery associated with aphantasia did not only lead to a reduced retrieval of visual-perceptual details but to a broader impairment in retrieving episodic AMs, including reduced emotions and confidence attached to the memories. Thus, in agreement with a recent account of aphantasia (Blomkvist, 2022), our results support the idea that a diminished construction of visual details during AM retrieval leads to a more general episodic memory deficit. We expand the current knowledge by adding that this AM deficit is reflected neurally by an altered activation and connectivity pattern between the hippocampus and visual-perceptual cortices. Our findings provide novel insights into three current debates: 1) the mechanisms of aphantasia-related AM deficits, 2) the similarities and differences between aphantasics and individuals with hippocampal damage, and 3) the neural models of AM.

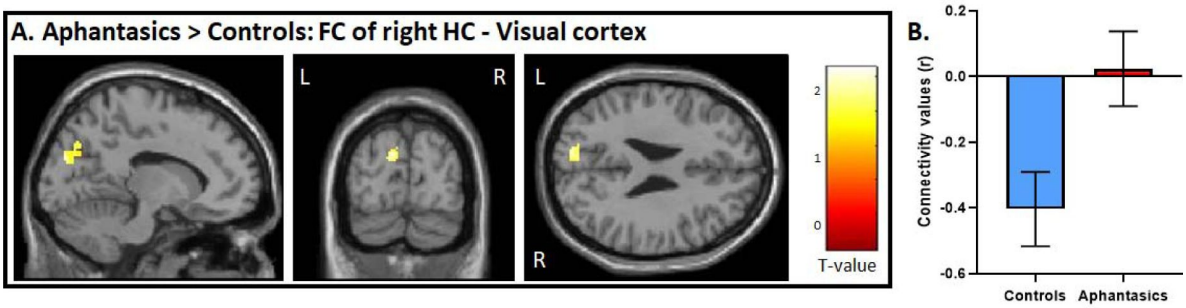

Figure 4.

Functional connectivity between the visual-perceptual cortex and hippocampus during AM retrieval.

(A) During AM retrieval, group differences in functional connectivity amongst the ROIs were only found between the right hippocampus, and left visual-perceptual cortices. (B) Controls displayed a stark negative correlation, whereas aphantasics did not. Image is displayed at $p < .05$, small volume corrected, and a voxel cluster threshold of 10 adjacent voxels.

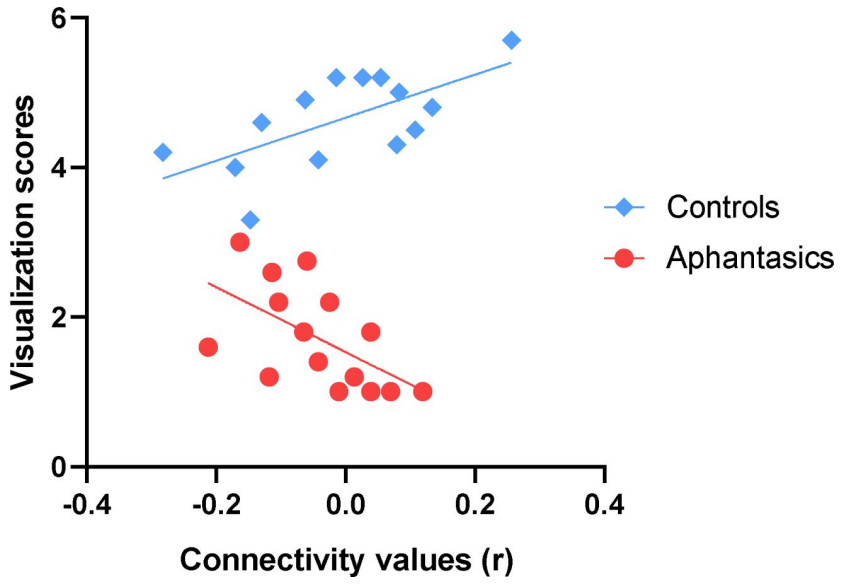

Figure 5.

Functional connectivity between the visual-perceptual cortex and hippocampus during resting-state explains visualization abilities.

Resting-state functional connectivity between the right hippocampus and the right visual-perceptual cortex correlates with visualization abilities. Fitted straight lines indicate a negative correlation for aphantasics (red) and a positive correlation for controls (blue).

Potential mechanisms underlying aphantasia-related AM deficits

We report that aphantasics show increased activation of bilateral visual-perceptual cortices as well as decreased hippocampal activation during AM retrieval in comparison to controls. Increased activity in the visual-perceptual cortices in aphantasics has been reported previously, albeit not associated with AM (Fulford et al., 2018; Keogh et al., 2020). In a prominent review, Pearson synthesizes evidence about the neural mechanism of imagery strength (Pearson, 2019). Indeed, activity metrics in the visual cortex predict imagery strength (Cui et al., 2007; Dijkstra et al., 2017). Interestingly, lower resting activity and excitability result in stronger imagery, and reducing cortical activity in the visual cortex via transcranial direct current stimulation (tDCS) increases visual imagery strength (Keogh et al., 2020). Thus, one potential mechanism of aphantasia-related AM deficits is that the heightened activity of the visual-perceptual cortices observed in our and previous work hinders aphantasics to detect weaker imagery-related signals.

Further, we had the a priori hypothesis that hippocampal activation will be decreased in aphantasics; a hypothesis which we could confirm in our native space hippocampal analysis. On the whole-brain level, perhaps due to our small sample size, only the cluster of activation group differences in the right posterior hippocampus survived the statistical threshold. Given the low power, further studies are needed to confirm this effect; however, the right posterior hippocampus interacts in the healthy brain heavily with the visual-perceptual cortex only during the elaboration phase of AM retrieval (McCormick et al. 2015).

In addition, controls exhibited a strong functional connection between both brain structures during AM retrieval and this functional connectivity predicted better visualization skills. At first glance, it is surprising that this functional connectivity was negative. However, negative visual-perceptual cortex activation during perceiving and imagining scenes has been reported before (McCormick et al. 2021). One possible explanation might be that signals from the hippocampus selectively inhibit imagery-irrelevant activation in the visual-perceptual cortices (e.g., sensory noise) to carve out imagery-related signals (cf. Pace et al., 2023). This would be in line with the hypothesis stated above, that a bad signal-to-noise ratio in the visual cortex hiders aphantasics to create mental imagery. Either way, the described functional connectivity between the hippocampus and the visual-perceptual cortex fits well to previous neuroimaging studies pointing towards a central role of the dynamic interplay between these brain structures during AM retrieval (McCormick, et al. 2015). This interplay seems to be especially important during the elaboration stage of AM retrieval, a period when specific visual-perceptual details are being actively brought back into the mind's eye. At this point, however, it remains unclear whether the disruption of AM elaboration associated with aphantasia takes place during the encoding, storage, or retrieval process.

From a theoretical point of view, the extended constructive episodic simulation hypothesis proposes a top-down hierarchy during mental imagery (Blomkvist, 2022). In this model, the hippocampus initiates retrieval processes in primary sensory brain regions, such as the visual-perceptual cortex in order to retrieve visual-perceptual details associated with a specific AM. Evidence for such top-down hierarchies during mental imagery have been observed in fronto-parietal and occipital networks via effective connectivity analyses, such as Granger Causality and Dynamic Causal Modelling (Dentico et al., 2014; Dijkstra et al., 2017; Mechelli et al., 2004). For example, intracranial and high-density scalp electroencephalography (EEG) provided evidence of high frequency hippocampal signaling during the recall of perceptual cues in patients with epilepsy, indicating that the hippocampus drives the switch from perception to memory recalling (Treder et al., 2021). In aphantasia, it is hypothesized that this top-down hierarchy is disrupted and therefore, the hippocampus can no longer initiate the retrieval and incorporation of visual-perceptual details in one coherent mental event. Because of the slow temporal resolution of our fMRI sequence, our data cannot directly speak to the question of temporal directionality between the hippocampus and visual-perceptual cortex. Nonetheless, our findings suggest that the

bidirectional connectivity between both brain structures is crucial for the re-experience of episodic AMs. As such, hippocampal processes may be needed to retrieve specific details and if these details are not provided by the visual-perceptual cortices, the entire episodic AM retrieval seems to fail.

Similarities and differences between aphantasics and individuals with hippocampal lesions

At face value, the episodic AM deficits in aphantasia in our data and reported previously (Dawes et al., 2020; 2022; Milton et al., 2020; Zeman et al., 2020), as well as the decreased hippocampal activation during AM retrieval suggest that aphantasia is a selective episodic memory condition (see Blomkvist, 2022), similar to AM amnesia known from individuals with hippocampal damage. In fact, previous and the current study show that aphantasics and individuals with hippocampal damage report less internal details across several memory detail subcategories, such as emotional details and temporal details (Rosenbaum et al., 2008; St-Laurent et al., 2009; Steinvorth et al., 2005), and these deficits can be observed regardless of the recency of the memory (Miller et al., 2020). These similarities suggest that aphantasics are not merely missing the visual-perceptual details to specific AM, but they have a profound deficit associated with the retrieval of AM.

Nonetheless, there are also stark differences between aphantasics and individuals with hippocampal damage. Foremost, aphantasics seem not to have difficulties to retrieve spatial information (Bainbridge et al., 2021), which is another inherent function of the hippocampus (Burgess et al., 2002; O'Keefe, 1991). In the current study, we did not set out to examine spatial cognition in aphantasics, however, parts of our data speak to this aspect. While in our study aphantasics reported less amount of spatial details during the AI, this standard scoring procedure only counts place details when the exact place is recalled and is not meant to assess the recall of spatial layout (Levine et al., 2002). Thus, this place score may not represent spatial cognition per se. In fact, when asking aphantasics about their experience, they point out difficulties in recalling AM and using imagination in daily life, however they report no difficulties in spatial orientation. Indeed, often during the interview, aphantasics would explain that they know how the space around them felt, they just cannot see it in front of their mind's eye. One aphantasic put her finger on it, describing it as: "I can put my consciousness in my kitchen at home and feel all around but there is no visual image attached to this feeling." These observations support the idea that some hippocampal processes, at least regarding spatial navigation, may be intact in people with aphantasia (Bainbridge et al., 2021). However, spatial cognition should be formally addressed in future studies. One way to assess this hippocampal function would be to examine tasks, which rely on scene construction. The scene construction theory states that the hippocampus is crucially needed for the construction of spatially coherent mental models of scenes (Maguire & Mullally, 2013). For example, patients with hippocampal damage cannot imagine the spatial layout of fictitious scenes (Hassabis et al., 2007), they detect less errors in spatially-incoherent scenes than controls (McCormick et al., 2017), and they show less scene-dependent mind-wandering episodes (McCormick, Rosenthal, et al., 2018). In contrast, we would predict that aphantasics have diminutive deficits in tasks that depend on hippocampal scene construction processes.

What could be impaired in aphantasics are all cognitive functions which rely on the population of the constructed scenes with visual-perceptual details, such as episodic AM retrieval, episodic future thinking, complex decision-making, and complex empathy tasks.

Towards a novel neural model of autobiographical memory

While more research is required exploring the cognitive landscape associated with aphantasia, such as spatial cognition and scene construction, our data contribute to an old debate of how AM retrieval and visual imagery are intertwined. We propose that the hippocampus is embedded in a

brain-wide network, comprising the vmPFC and visual-perceptual cortices, in which each of these nodes contributes specific processes to the re-construction of extended detail-rich mental events (see also Ciaramelli et al., 2019; McCormick, Ciaramelli, et al., 2018). Within this model, the vmPFC initiates and oversees the scene construction process which takes place in the hippocampus. Further, the visual-perceptual cortex provides the visual details which are essential to populate the hippocampally constructed scenes. This model is backed up by a previous MEG study revealing that the vmPFC directs hippocampal activity during the initiation of AM retrieval (McCormick et al., 2020). This finding has been replicated and extended by Chen et al. (2021), showing that the vmPFC leads hippocampal involvement during scene construction and other scene-based processes (Monk et al., 2021). Moreover, the connection between the hippocampus and the visual-perceptual cortex seems equally crucial. There are a few case reports of damage to the occipital cortex causing AM amnesia (Greenberg et al., 2005), potentially by preventing the population of the hippocampally constructed scenes. Furthermore, our current study suggests that a reliable connectivity between the hippocampus and the visual-perceptual cortices is important to provide the visual details necessary for successful vivid, detail-rich AM retrieval.

Conclusion

Aphantasia provides a natural knock-out model for the influence of visual imagery on different cognitive functions. We here report a tight link between visual imagery and our ability to retrieve vivid and detail-rich personal past events, as aphantasics do not only report fewer visual-perceptual details during episodic AM retrieval but also show decreased confidence and emotionality associated with these memories. In this context, we highlight the central role of the functional connectivity between the hippocampus and occipital cortex to assemble visual-perceptual details into one coherent extended mental event. Exciting novel research avenues will be to examine hippocampal-dependent spatial cognition in aphantasics and to investigate whether neuroscientific interventions can be used to enhance AM retrieval by enhancing visual imagery.

Conflict of interest statement

The authors declare no competing financial interest.

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Editors

Reviewer #1 (Public Review):

Summary:

In this article, the authors investigate whether the connectivity of the hippocampus is altered in individuals with aphantasia ¬- people who have reduced mental imagery abilities and where some describe having no imagery, and others describe having vague and dim imagery. The study investigated this question using a fMRI paradigm, where 14 people with aphantasia and 14 controls were tested, and the researchers were particularly interested in the key regions of the hippocampus and the visual-perceptual cortices. Participants were interviewed using the Autobiographical Interview regarding their autobiographical memories (AMs), and internal and external details were scored. In addition, participants were queried on their perceived difficulty in recalling memories, imagining, and spatial navigation, and their confidence regarding autobiographical memories was also measured. Results showed that participants with aphantasia reported significantly fewer internal details (but not external details) compared to controls; that they had lower confidence in their AMs; and that they reported finding remembering and imagining in general more difficult than controls. Results from the fMRI section showed that people with aphantasia displayed decreased hippocampal and increased visual-perceptual cortex activation during AM retrieval compared to controls. In contrast, controls showed strong negative functional connectivity between hippocampus and the visual cortex. Moreover, resting state connectivity between the hippocampus and visual cortex predicted better visualisation skills. The authors conclude that their study provides evidence for the important role of visual imagery in detail-rich vivid AM, and that this function is supported by the connectivity between the hippocampus and visual cortex. This study extends previous findings of reduced episodic memory details in people with aphantasia, and enables us to start theorising about the neural underpinnings of this finding.

The data provided good support for the conclusion that the authors draw, namely that there is a 'tight link between visual imagery and our ability to retrieve vivid and detail-rich personal past events'. However, as the authors also point out, the exact nature of this relationship is difficult to infer from this study alone, as the slow temporal resolution of fMRI cannot establish the directionality between the hippocampus and the visual-perceptual cortex. This is an exciting future avenue to explore.

Strengths:

A great strength of this study is that it introduces a fMRI paradigm in addition to the autobiographical interview, paralleling work done on episodic memory in cognitive science (e.g. Addis and Schacter, 2007, https://doi.org/10.1016%2Fj.neuropsychologia.2006.10.016), which has examined episodic and semantic memory in relation to imagination (future simulation) in non-aphantasic participants as well as clinical populations. Future work could build on this study, and for example use the recombination paradigm (Addis et al. 2009, 10.1016/j.neuropsychologia.2008.10.026), which would shed further light on the ability of people with aphantasia to both remember and imagine events. Future work could also build on the interesting findings regarding spatial navigation, which together with previous findings in aphantasia (e.g. Bainbridge et al., 2021, https://doi.org/10.1016/j.cortex.2020.11.014) strongly suggests that spatial abilities in people with aphantasia are unaffected. This can shed further light on the different neural pathways of spatial and object memory in general. In general, this study opens up a multitude of new avenues to explore and is likely to have a great impact on the field of aphantasia research.

Weaknesses:

A weakness of the study is that some of the questions used are a bit vague, and no objective measure is used, which could have been more informative. For example, the spatial navigation question (reported as 'How difficult is it typically for you to orient you spatially?' could have been more nuanced to tap into whether participants relied mostly on cognitive maps (likely supported by the hippocampus) or landmarks. It would also have been interesting to conduct a spatial navigation task, as participants do not necessarily have

insight to their spatial navigation abilities (they could have been overconfident or underconfident in their abilities). Secondly, the question 'how difficult is it typically for you to use your imagination?' could also be more nuanced, as imagination is used in a variety of ways, and we only have reason to hypothesise that people with aphantasia might have difficulties in some cases (i.e. sensory imagination involving perceptual details). It is unlikely that people with aphantasia would have more difficulty than controls to use their imagination to imagine counterfactual situations and engage in counterfactual thought (de Brigard et al., 2013, https://doi.org/10.1016%2Fj.neuropsychologia.2013.01.015) due to its non-sensory nature, but the question used does not distinguish between these types of imagination. Again, this is a ripe area for future research. The general phrasing of 'how difficult is [x]' could also potentially bias participants towards more negative answers, something which ought to be controlled for in future research.

https://doi.org/10.7554/eLife.94916.2.sa1

Reviewer #2 (Public Review):

Summary:

This study investigates to what extent neural processing of autobiographical memory retrieval is altered in people who are unable to generate mental images ('aphantasia'). Self-report as well as objective measures were used to establish that the aphantasia group indeed had lower imagery vividness than the control group. The aphantasia group also reported fewer sensory and emotional details of autobiographical memories. In terms of brain activity, compared to controls, aphantasics had a reduction in activity in the hippocampus and an increase in the activity in visual cortex during autobiographical memory retrieval. For controls, these two regions were also functionally connected during autobiographical memory retrieval, which did not seem to be the case for aphantasics. Finally, resting-state connectivity between visual cortex and hippocampus was positively related to autobiographical vividness in the control group but negatively in the aphantasia group. The results are in line with the idea that aphantasia is caused by an increase in noise within the visual system combined with a decrease in top-down communication from the hippocampus.

Recent years have seen a lot of interest in the influence of aphantasia on other cognitive functions and one of the most consistent findings is deficits in autobiographical memory. This is one of the first studies to investigate the neural correlates underlying this difference, thereby substantially increasing our understanding of aphantasia and the relationship between mental imagery and autobiographical memory.

Strengths:

One of the major strengths of this study is the use of both self-report as well as objective measures to quantify imagery ability. Furthermore, the fMRI analyses are hypothesis-driven and reveal unambiguous results, with alterations in hippocampal and visual cortex processing seeming to underlie the deficits in autobiographical memory.

Weaknesses:

In terms of weaknesses, the control task, doing mathematical sums, also differs from the autobiographical memory task in aspects that are unrelated to imagery or memory, such as self-relevance and emotional salience, which makes it hard to conclude that the differences in activity are reflecting only the cognitive processes under investigation. However, given that the most important comparisons are between groups of participants, this does not diminish the main conclusions about aphantasia.

Overall, I believe that this is a timely and important contribution to the field and will inspire novel avenues for further investigation.

https://doi.org/10.7554/eLife.94916.2.sa0

Author response:

The following is the authors' response to the original reviews.

> ***Public Reviews:***
>
> ***Reviewer #1 (Public Review):***
>
> *Summary:*
>
> *In this article, the authors investigate whether the connectivity of the hippocampus is altered in individuals with aphantasia ¬- people who have reduced mental imagery abilities and where some describe having no imagery, and others describe having vague and dim imagery. The study investigated this question using a fMRI paradigm, where 14 people with aphantasia and 14 controls were tested, and the researchers were particularly interested in the key regions of the hippocampus and the visual-perceptual cortices. Participants were interviewed using the Autobiographical Interview regarding their autobiographical memories (AMs), and internal and external details were scored. In addition, participants were queried on their perceived difficulty in recalling memories, imagining, and spatial navigation, and their confidence regarding autobiographical memories was also measured. Results showed that participants with aphantasia reported significantly fewer internal details (but not external details) compared to controls; that they had lower confidence in their AMs; and that they reported finding remembering and imagining in general more difficult than controls. Results from the fMRI section showed that people with aphantasia displayed decreased hippocampal and increased visual-perceptual cortex activation during AM retrieval compared to controls. In contrast, controls showed strong negative functional connectivity between the hippocampus and the visual cortex. Moreover, resting state connectivity between the hippocampus and visual cortex predicted better visualisation skills. The authors conclude that their study provides evidence for the important role of visual imagery in detail-rich vivid AM, and that this function is supported by the connectivity between the hippocampus and visual cortex. This study extends previous findings of reduced episodic memory details in people with aphantasia, and enables us to start theorising about the neural underpinnings of this finding.*
>
> *The data provided good support for the conclusion that the authors draw, namely that there is a 'tight link between visual imagery and our ability to retrieve vivid and detail-rich personal past events'. However, as the authors also point out, the exact nature of this relationship is difficult to infer from this study alone, as the slow temporal resolution of fMRI cannot establish the directionality between the hippocampus and the visual-perceptual cortex. This is an exciting future avenue to explore.*

We thank the reviewer for highlighting our contributions and suggesting that the relationship between visual imagery and autobiographical memory recall is an exciting future avenue.

> *Weaknesses:*
>
> *A weakness of the study is that some of the questions used are a bit vague, and no objective measure is used, which could have been more informative. For example, the*

> *spatial navigation question (reported as 'How difficult is it typically for you to orient you spatially?' - a question which is ungrammatical, but potentially reflects a typo in the manuscript) could have been more nuanced to tap into whether participants relied mostly on cognitive maps (likely supported by the hippocampus) or landmarks. It would also have been interesting to conduct a spatial navigation task, as participants do not necessarily have insight into their spatial navigation abilities (they could have been overconfident or underconfident in their abilities).*
>
> *Secondly, the question 'how difficult is it typically for you to use your imagination?' could also be more nuanced, as imagination is used in a variety of ways, and we only have reason to hypothesise that people with aphantasia might have difficulties in some cases (i.e. sensory imagination involving perceptual details). It is unlikely that people with aphantasia would have more difficulty than controls in using their imagination to imagine counterfactual situations and engage in counterfactual thought (de Brigard et al., 2013, https://doi.org/10.1016%2Fj.neuropsychologia.2013.01.015) due to its non-sensory nature, but the question used does not distinguish between these types of imagination. Again, this is a ripe area for future research. The general phrasing of 'how difficult is [x]' could also potentially bias participants towards more negative answers, something which ought to be controlled for in future research.*

The main goal of our study was to examine autobiographical memory recall. Therefore, we used the gold standard Autobiographical Interview, or AI (Levine et al. 2002) and an fMRI paradigm to explore autobiographical memory recall as standardised, precisely, and objectively as possible.

In addition to these experimentally rigorous tasks, we employed some loosely formulated questions with the intention for people to reflect on how they perceive their own abilities to recall autobiographical memories, navigate spatially, and use their imagination. We agree with the reviewer that these questions are vague and did not have the experimental standard for an investigation into spatial cognition or imagination associated with aphantasia. Nonetheless, we believe that these questions provide important additional insights into what participants think about their own cognitive abilities. In order to set these questions into perspective, we argue in the discussion that spatial cognition and other cognitive functions should be investigated in more depth in individuals with aphantasia in the future.

As an additional note, all tasks were conducted in German. Thus, we were able to correct the wording of the debriefing question in our revision. We thank the reviewer for bringing this to our attention.

> *Strengths:*
>
> *A great strength of this study is that it introduces a fMRI paradigm in addition to the autobiographical interview, paralleling work done on episodic memory in cognitive science (e.g. Addis and Schacter, 2007, https://doi.org/10.1016%2Fj.neuropsychologia.2006.10.016), which has examined episodic and semantic memory in relation to imagination (future simulation) in non-aphantasic participants as well as clinical populations. Future work could build on this study, and for example use the recombination paradigm (Addis et al. 2009, 10.1016/j.neuropsychologia.2008.10.026), which would shed further light on the ability of people with aphantasia to both remember and imagine events. Future work could also build on the interesting findings regarding spatial navigation, which together with previous findings in aphantasia (e.g. Bainbridge et al., 2021, https://doi.org/10.1016/j.cortex.2020.11.014) strongly suggests that spatial abilities in people with aphantasia are unaffected. This can shed further light on the different neural pathways of spatial and object memory in general. In general,*

> *this study opens up a multitude of new avenues to explore and is likely to have a great impact on the field of aphantasia research.*

We much appreciate the acknowledgment of our work into autobiographical memory employing both the autobiographical interview and fMRI. Furthermore, we hope that our work inspires future research in the way the reviewer outlines and in the way we describe in our manuscript.

> ***Reviewer #2 (Public Review):***
>
> *Summary:*
>
> *This study investigates to what extent neural processing of autobiographical memory retrieval is altered in people who are unable to generate mental images ('aphantasia'). Self-report as well as objective measures were used to establish that the aphantasia group indeed had lower imagery vividness than the control group. The aphantasia group also reported fewer sensory and emotional details of autobiographical memories. In terms of brain activity, compared to controls, aphantasics had a reduction in activity in the hippocampus and an increase in activity in the visual cortex during autobiographical memory retrieval. For controls, these two regions were also functionally connected during autobiographical memory retrieval, which did not seem to be the case for aphantasics. Finally, resting-state connectivity between the visual cortex and hippocampus was positively related to autobiographical vividness in the control group but negatively in the aphantasia group. The results are in line with the idea that aphantasia is caused by an increase in noise within the visual system combined with a decrease in top-down communication from the hippocampus.*
>
> *Recent years have seen a lot of interest in the influence of aphantasia on other cognitive functions and one of the most consistent findings is deficits in autobiographical memory. This is one of the first studies to investigate the neural correlates underlying this difference, thereby substantially increasing our understanding of aphantasia and the relationship between mental imagery and autobiographical memory.*

We thank the reviewer for highlighting the importance of our findings.

> *Strengths:*
>
> *One of the major strengths of this study is the use of both self-report as well as objective measures to quantify imagery ability. Furthermore, the fMRI analyses are hypothesis-driven and reveal unambiguous results, with alterations in hippocampal and visual cortex processing seeming to underlie the deficits in autobiographical memory.*

Once again, we thank the reviewer for highlighting the quality of our methods and our results.

> *Weaknesses:*
>
> *In terms of weaknesses, the control task, doing mathematical sums, also differs from the autobiographical memory task in aspects that are unrelated to imagery or memory, such as self-relevance and emotional salience, which makes it hard to conclude that the differences in activity are reflecting only the cognitive processes under investigation.*

We agree with the reviewer that our control task differs from autobiographical memory in many different ways. In fact, for this first investigation of the neural correlates of autobiographical memory in aphantasia, this is precisely the reason why we chose this mental arithmetic (MA) task. We know from previous studies, that MA is, as much as possible,

not dependent on hippocampal memory processes (Addis, et al. 2007, McCormick et al. 2015, 2017, Leelaarporn et al., 2024). The main goal of the current study was to establish whether there are any differences between individuals with aphantasia and controls. In the next investigation, we can now build on these findings to disentangle in more detail what this difference reflects.

> *Overall, I believe that this is a timely and important contribution to the field and will inspire novel avenues for further investigation.*

This highly positive conclusion is much appreciated.

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McCormick, C., Moscovitch, M., Valiante, T. A., Cohn, M., & McAndrews, M. P. (2018). Different neural routes to autobiographical memory recall in healthy people and individuals with left medial temporal lobe epilepsy. *Neuropsychologia*, *110*, 26-36.

> ***Recommendations for the authors:***
>
> ***Reviewer #1 (Recommendations For The Authors):***
>
> *This is a very interesting article that makes a substantial contribution to the field of the study of aphantasia as well as the neural mechanisms of autobiographical memory. I would strongly recommend this manuscript to be accepted (with these minor revisions), as it makes a substantial and well-evidenced contribution to the research, and it opens up many interesting avenues for researchers to explore. I was especially excited to see that the Autobiographical Interview had been paired with an fMRI paradigm, something which this field of research highly benefits from, as there are yet so few fMRI studies into aphantasia. I understand that it is the authors' decision whether to accept or reject any of the revisions I recommend here, but I would like to stress that I encourage accepting the recommended revisions, especially as there are some minor inaccuracies in the manuscript as it currently stands. Finally, I would like to stress that though I am based in the area of cognitive science, am not trained in fMRI imaging techniques, and therefore do not stand in a position where I can comment on the methodology pertaining to this part of the study - I encourage the Editors to seek a second reviewer's opinion on this.*

Thank you for the positive evaluation of our manuscript as well as your comments. We have revised our manuscript according to your important suggestions as further explained below.

> *Line 33: "aphantasia prohibits people from experiencing visual imagery". This characterisation of aphantasia is too strong, especially as the authors use 32 as a cut-off point on the VVIQ, which represents weak and dim imagery. I would recommend using language like 'people with aphantasia have reduced visual imagery abilities', as this more accurately captures the group of people studied. Please revise throughout the manuscript. Please consult Blomkvist and Marks (2023) on this point who have discussed this problem in the aphantasia literature.*

We agree that aphantasics may experience reduced visual imagery abilities. We have revised our wording throughout the manuscript.

> *Line 49: The authors conclude that their results 'indicate that visual mental imagery is essential for detail-rich, vivid AM', but this seems to be a bit too strong, for example since AM can be detail-rich with external (rather than internal) detail, and a person could potentially use mnemonic tricks such as keeping a detail-rich diary in order to boost their memory. That visual imagery is 'essential' implies that it is the only way to achieve detail-rich vivid AM, and this does not seem to be supported by the findings. I would recommend rephrasing it as 'visual mental imagery plays an important role in detail-rich, vivid AM' or 'visual mental imagery mediated detail-rich vivid AM'.*

We altered the sentence in Line 49 using one of the recommended phrases:

‘Our results indicate that visual mental imagery plays an important role in detail-rich, vivid AM, and that this type of cognitive function is supported by the functional connection between the hippocampus and the visual-perceptual cortex.’

> *Line 69: Blomkvist and Marks (2023) have warned against calling aphantasia a 'condition' and this moreover seems to fit with the authors' previous research (Monzel, 2022). Please consider instead calling aphantasia an 'individual difference' in mental imagery abilities.*

Thank you for the suggestion. We have revised our wording throughout the manuscript, avoiding the term ‘condition’.

> *Line 72: Add reference for emotional strength which has also been researched (Wicken et al. 2021, https://doi.org/10.1016/j.cortex.2020.11.014).*

We have added the suggested reference in Line 75:

‘Indeed, a handful of previous studies report convergent evidence that aphantasics report less sensory AM details than controls (Bainbridge et al., 2021; Dawes et al., 2020, 2022; Milton et al., 2020; Zeman et al., 2020), which may also be less emotional (Monzel et al., 2023; Wicken et al., 2021).’

> *72-73: 'absence of voluntary imagery' - too strong as many people with aphantasia report having weak/dim mental imagery on the VVIQ.*

We agree that aphantasics may experience reduced visual imagery. We have revised this notion throughout the manuscript.

> *74: Add reference to Bainbridge study which found a difference between recall of object vs spatial memory. This would be relevant here.*

We have added the suggested reference in Line 76:

'Spatial accuracy, on the other hand, was not found to be impaired (Bainbridge et al., 2021).'

> *Lines 94-97: The authors mention 'a prominent theory' but it is unclear which theory is referred to here. The article cited by Pearson (2019) does not suggest the possibility that aphantasia is due to altered connectivity between the hippocampus and visual-perceptual cortices. It suggests that aphantasia is due to impairment in the ventral stream, and in fact says that the hippocampus is unlikely to be affected due to spared spatial abilities in people with aphantasia. Specifically, Pearson claims: "Accordingly, memory areas of the brain that process spatial properties, including the hippocampus, may not be the underlying cause of aphantasia." (page 631). The authors further come back to this point in the discussion section (see comment below), saying that the hypothesis attributed to Pearson is supported by their study. I do not disagree with the point that the hypothesis is supported by the data, but it is unclear to me why the hypothesis is attributed to Pearson.*

Thank you for pointing out this inaccuracy. We have edited the text to spell out our entire train of thought (see Lines 96-102):

'A prominent theory posits that because of this hyperactivity, small signals elicited during the construction of mental imagery may not be detected (Pearson, 2019, Keogh et al., 2020). Pearson further speculates that since spatial abilities seem to be spared, the hippocampus may not be the underlying cause of aphantasia. In agreement, Bergmann and Ortiz-Tudela (2023) speculate that individuals with aphantasia might lack the ability to reinstate visually precise episodic elements from memory due to altered feedback from the visual cortex.'

> *Line 97: Blomkvist reference should be 2022 (when first published online).*

The article 'Aphantasia: In search of a theory' by Blomkvist was first published on 1st July 2022. However, a correction was added on 13th March 2023. Therefore, we had cited the corrected version in this manuscript. However, we agree that the first publication date should be used and edited the reference accordingly.

> *Line 116: 'one aphantasic' could be seen as offensive. I would suggest 'one aphantasic participant'.*

We have altered the paragraph according to your suggestion.

> *Line 138: In line with the recommendations put forward by Blomkvist and Marks (2023), I would suggest removing the word 'diagnosed', as this medicalises aphantasia in a way that is not consistent with its not being a kind of mental disorder (Monzel et al., 2022). I would say that aphantasia is instead operationalised as a score between 16-32. However, note that Blomkvist (2022) and Blomkvist and Marks (2023, https://doi.org/10.1016/j.cortex.2023.09.004) point out that there is also a lot of inconsistency in this score and how it is used in different studies. In your manuscript, I would recommend removing all wording that indicates that people with aphantasia have no experience of mental imagery, as you have operationalised for a score up to 32 which indicates vague and dim imagery. Describing vague and dim imagery as no imagery/absence of imagery is inconsistent (but common practice in the literature).*

Thank you for your suggestion. We have revised the entire manuscript to eliminate any ambiguous meanings regarding the definition of aphantasia. Moreover, we replaced the word ‘diagnosed’ with ‘identified’ in Line 146.

> *Line 153: maybe 'correlated with imagery strength' rather than 'measures imagery strength'?*

We have altered the sentence according to your suggestion in Line 160:

‘Previous studies have shown that the binocular rivalry task validly correlated with mental imagery strength.’

> *Line 162: "For participants who were younger than 34 years, the middle-age memory was replaced by another early adulthood memory". Is there precedence for this? Please add one sentence to explain/justify for the reader why a memory from this time period was chosen.*

To maintain the homogeneous data set of acquiring five episodic autobiographical memories from five different periods of life per one individual, we asked the participants who were at the time of the interview, younger than 34 years old, to provide another early adulthood memory instead of middle age memory, as they had not reached the age range of middle age. According to Levine et al. (2002), younger adults (age < 34 years old) selected 2 events from the early adulthood period. Hence, all participants provided the last time period with memories from their previous year. We have added an additional explanation in this section in Line 170:

‘In order to acquire five AMs in every participant, the middle age memory was replaced by another early adulthood memory for participants who were younger than 34 years old (see Levine et al., 2002). Hence, all participants provided the last time period with memories from their previous year.’

> *Line 169: "During the general probe, the interviewer asked the participant encouragingly to promote any additional details." Consider a different word choice, 'promote' sounds odd.*

We have altered the sentence according to your suggestion in Line 180:

‘During the general probe, the interviewer asked the participant encouragingly to provide any additional details.’

> *Line 196-198: the phrasing of these questions could have biased participants toward reporting it being more difficult. Did the authors control for this possibility in any way? The phrasing ‘How easy is it for you to [x]?’ might also be considered in a future study.*

Thank you for pointing this out. These debriefing questions were thought of as open questions to get people to talk about their experiences. They were not meant as rigorous scientific experiments. Framing it in a positive way is a good idea for future research.

We have edited the manuscript on Line 394-396:

‘The debriefing questions were employed as a way for participants to reflect on their own cognitive abilities. Of note, these were not meant to represent or replace necessary future experiments.’

> *Line 197: This question is ungrammatical. Is this a typo, or was this how the question was actually posed? What language was the study conducted in?*

All interviews within this study were conducted in German. Hence, the questions listed in this current manuscript were all translated from German into English. We have added this information in the Materials and Methods section in Line 169 as well as restructured the referred questions from Line 208-210:

‘All interviews were conducted in German.’

(1) Typically, how difficult is it for you to recall autobiographical memories?

(2) Typically, how difficult is it for you to orient yourself spatially?

(3) Typically, how difficult is it for you to use your imagination?’

> *Line 211: The authors write that participants were asked to "re-experience the chosen AM and elaborate as many details as possible in their mind's eye" was this the instruction used? I think stating the explicit instruction here would be relevant for the reader. If this is the word choice, it is also interesting as the autobiographical interview does not normally specify to re-experience details 'in one's mind's eye'.*

The instructions gi‘en to ’he par’Icipa’ts were to choose an AM and re-experience/elaborate it in their mind with as many details as possible without explaining them out loud. We have clarified this in Lines 221-223.

‘For the rest of the trial duration, participants were asked to re-experience the chosen AM and try to recall as many details as possible without speaking out loud.’

> *Line 213: Were ‘vivid’ and ‘faint’ the only two options? Why was a 5-point scale (like the VVIQ scale) not used to better be able to compare?*

During the scanning session, the participants were given a button box which contained two buttons with 'vivid' by pressing the index finger and 'faint' by pressing the middle finger. The 5-point scale was not used to avoid confusion with the buttons during the scanning session. We have clarified this in Line 224:

‘We chose a simple two-button response in order to keep the task as easy as possible.’

> *Line 347: Do the authors mean the same thing by 'imagery strength' and 'imagery vividness'? This would be good to clarify as it is not clear that these words mean the same thing.*

Imagery strength is often used to describe the results of the Binocular Rivalry Task, whereas vividness of mental imagery is often used to describe the results of the VVIQ. Although both tasks are correlated, the VVIQ measures vividness, whereas the dimension of the Binocular Rivalry Task is not clearly defined. We added this information in a footnote on page 10.

> *Lines 353 - 356: When the authors first say that aphantasics described fewer memory details than controls, does this refer to external + internal details? Please clarify.*
>
> *Lines 353-360: The authors first say that aphantasics report "internal details (M = 43.59, SD = 17.91) were reported more often than external details (M = 20.64, SD = 8.94)" (line 355). But then they say: "a 2-way interaction was found between the type of memory details and group, $F(1, 27)= 54.09$, $p < .001$, $\eta p2 = .67$, indicating that aphantasics*

> *reported significantly less internal memory details, t(27) = 5.07, p < .001, d = 1.83, but not significantly less external memory details, t(27) = 0.13, p = .898, compared to controls (see Figure 1b)" (line 358). This seems to first say that aphantasics didn't report fewer details than controls, but then that they did report fewer internal details than controls. Please clarify if this is correct.*
>
> *Line 383: Results from controls are not reported in this section.*

We have first reported the main effects of the different factors; thus, aphantasics reported less details than controls (no matter of group and type of memory details), the internal details were reported more often than external details (no matter of group and memory period), and more details were reported for recent than remote memories (no matter of group and type of memory details). Subsequently, we report the simple effects for aphantasics and controls separately. To further clarify, we added the following segment in line 360:

'Regarding the AI, we found significant main effects of memory period, F(1, 27) = 11.88, p = .002, ηp2 = .31, type of memory details, F(1, 27) = 189.03, p < .001, ηp2 = .88, and group, F(1, 27) = 9.98, p = .004, ηp2 = .27. When the other conditions were collapsed, aphantasics (M = 26.29, SD = 9.58) described less memory details than controls (M = 38.36, SD = 10.99). For aphantasics and controls combined, more details were reported for recent (M = 35.17, SD = 14.19) than remote memories (M = 29.06, SD = 11.12), and internal details (M = 43.59, SD = 17.91) were reported more often than external details (M = 20.64, SD = 8.94). More importantly, a 2-way interaction was found between type of memory details and group, F(1, 27) = 54.09, p < .001, ηp2 = .67, indicating that aphantasics reported significantly less internal memory details, t(27) = 5.07, p < .001, d = 1.83, but not significantly less external memory details, t(27) = 0.13, p = .898, compared to controls (see Figure 1b).'

Overall, the results were reported for aphantasics and controls separately in Lines 368-372.

> *Line 386: The question does not specify that it's asking about using imagination in daily life, even though this is what results report. I'm not sure that the question implies the use of imagination in daily life, so I would recommend removing this reference here.*

We have removed the "in daily life" since this was not part of the original debriefing question.

> *Line 394: Could this slowness in response reflect uncertainty about the vividness?*

Since the reason for this slowness is not known, we have refrained from adding this to the discussion. However, we added this as a short insertion in line 406:

'Moreover, aphantasics responded slower (M = 1.34 s, SD = 0.38 s) than controls (M = 1.00 s, SD = 0.29 s) when they were asked whether their retrieved memories were vivid or faint, t(28) = 2.78, p = .009, possibly reflecting uncertainty in their response.'

> *Line 443: Graph E, significance not indicated on the graph.*

After preprocessing, the fMRI data were statistically analyzed using the GLM contrast AM versus MA. The resulting images were then thresholded at p < 0.001, so that the illuminated voxels in Fig. 3 A, B, C, and D show only voxel in which we know already that there is a statistical difference between our conditions. Graph E illustrates only the descriptive means and variance of the significant differences in Fig. 3 C and D. This display is useful since the reader can more easily assess the difference between two conditions and two groups at a glance. For a general discussion on this topic, please also see circular analysis in fMRI (Kriegeskorte et al. 2009)

> *Line 521-522: The authors claim that Pearson (2019) forwards the hypothesis that heightened activity of visual-perceptual cortices hinders aphantasics from detecting small imagery-related signals. However, I find no statement of this hypothesis in Pearson (2019). It is unclear to me why this hypothesis is attributed to Pearson (2019). Please remove this reference or provide a correct citation for where the hypothesis is stated. Further, it is not clear from what is written how the results support this hypothesis as this is rather brief - please elaborate on this.*

We attributed this hypothesis to Pearson (2019) according to his Fig. 4, which states: 'A strong top-down signal and low noise (bottom left) gives the strongest mental image (square), whereas a high level of neural noise and a weak top-down imagery signal would produce the weakest imagery experience (top right).'

We have edited our manuscript to reflect Pearson better in Lines 543-550:

'In a prominent review, Pearson synthesizes evidence about the neural mechanism of imagery strength (Pearson, 2019). Indeed, activity metrics in the visual cortex predict imagery strength (Cui et al., 2007; Dijkstra et al., 2017). Interestingly, lower resting activity and excitability result in stronger imagery, and reducing cortical activity in the visual cortex via transcranial direct current stimulation (tDCS) increases visual imagery strength (Keogh et al., 2020). Thus, one potential mechanism of aphantasia-related AM deficits is that the heightened activity of the visual-perceptual cortices observed in our and previous work hinders aphantasics to detect weaker imagery-related signals.'

> *Line 575: Consider citing Blomkvist (2022) who has argued that aphantasia is an episodic memory condition*

We added the suggested reference in Line 601.

> *Line 585: Consider citing Bainbridge et al (2021) https://doi.org/10.1016/j.cortex.2020.11.014*

We have added the suggested reference in Line 612.

> *Line 581: It might be relevant here to also discuss non-visual details, which have indeed been investigated in your present study. E.g. the lower emotional details, temporal details, place details, etc.*

We have edited our discussion to reflect the non-visual details better in Line 605:

'In fact, previous and the current study show that aphantasics and individuals with hippocampal damage report less internal details across several memory detail subcategories, such as emotional details and temporal details (Rosenbaum et al., 2008; St-Laurent et al., 2009; Steinvorth et al., 2005), and these deficits can be observed regardless of the recency of the memory (Miller et al., 2020). These similarities suggest that aphantasics are not merely missing the visual-perceptual details to specific AM, but they have a profound deficit associated with the retrieval of AM.'

Place details are discussed on page 37 onwards.

> *Line 605: I agree with this interesting suggestion for future research. It would also be relevant to reference Bainbridge (2021) here who tested spatial cognition in a drawing task and found that aphantasic participants correctly recalled spatial layouts of rooms but reported fewer objects than controls. It might also be worth pointing out that the*

> *present study does not actually test for accuracy in spatial cognition, so it could be the case that people with aphantasia feel confident that they can navigate well, but they might in fact not. Future studies relying on objective measures should test this possibility.*

We have added the suggested reference in Line 625.

> *Lines 609-614: Is there any evidence that complex decision-making and complex empathy tasks depend on constructed scenes with visual-perceptual details? This hypothesis seems a bit far-fetched without any supporting evidence. In fact, it seems unlikely to be supported as we also know that people with aphantasia generally live normal lives, and often have careers that we can assume involve complex decision-making (see Zeman 2020 who report aphantasics who work as computer scientists, managers, etc). I would recommend that the authors provide evidence of the role of mental imagery in complex decision-making and complex empathy tasks, mediated by scene construction, to support this hypothesis as viable to test for future research. It is also unclear how this point connects to the argument made by Bergmann and Ortiz-Tudela (2023). In fact, Bergmann and Ortiz-Tudela seem to make the same argument as Pearson (2019) does - that aphantasia results from impairments in the ventral stream, but that the dorsal stream is unaffected. However, Blomkvist (2022) argues that this view is too simplistic to be able to account for the variety of deficits that we see in aphantasia. I would recommend either engaging more fully with this debate or cutting it, as it currently is too vague for a reader to follow.*

We have decided to leave the discussion about scene construction and its connection to complex decision making and empathy out of the current manuscript. We have included the argument of Bergmann & Ortiz-Tudela (2023) in the Introduction (Line 101):

'In agreement, Bergmann and Ortiz-Tudela (2023) speculate that individuals with aphantasia might lack the ability to reinstate visually precise episodic elements from memory due to altered feedback from the visual cortex.'

> ***Reviewer #2 (Recommendations For The Authors):***
>
> *In general, I really enjoyed reading this paper.*

Thank you very much for the positive evaluation of our manuscript as well as your comments.

> *There were only a few things that I had some concerns about. For example, it was unclear to me whether the whole-brain analysis (Figures 3 and 4) was corrected for multiple comparisons or why only a small volume correction was applied for the functional connectivity analysis. If these results are borderline significant, this should be made more explicit in the manuscript. I don't think this is a major issue as the investigation of both the hippocampus and visual cortex was strongly hypothesis-driven, but it would still be good to be explicit about the strength of the findings.*

For the whole-brain analysis, we applied a threshold of $p < .001$, voxel cluster of 10, but no other multiple comparisons correction applied. The peak in the right hippocampus did survive the whole-brain threshold but we decided to lower this threshold just for display purposes in Figure 3, so that the readers can easily see the cluster.

We have made the statistical thresholds more easily assessable for the reader on the following pages:

Figure 3 (Page 27): ‘Images are thresholded at p < .001, cluster size 10, uncorrected, except (D) which is thresholded at p < .01, cluster size 10, for display purposes only (i.e., the peak voxel and adjacent 10 voxels also survived p < .001, uncorrected).’

Figure 4 (Page 30): ‘Image is displayed at p < .05, small volume corrected, and a voxel cluster threshold of 10 adjacent voxels.’

I was wondering whether it would be possible to use DCM to investigate the directionality of the connectivity. Given that there are only two ROIs and two alternative hypotheses (top-down versus bottom-up) this seems like an ideal DCM problem.

We thank the reviewer for this suggestion and will consider testing the effective connectivity between both regions of interest in a future investigation.

> *Line 385: typo: 'great' should be 'greater'.*

We have altered the typo from ‘great’ to ‘greater’ in Line 397.

> *Line 400: absence of evidence of an effect is not evidence of absence of an effect.*

We agree with the reviewer that this was unclear. We changed the wording in Line 412:

‘In addition, aphantasics and controls did not differ significantly in their time searching for a memory in AM trials, t(19) = 1.03, p = .315.’

> *Typo line 623: 'overseas'.*

We have altered the mistyped word from ‘overseas’ to ‘oversees’ in Line 647.

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