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People with aphantasia report fewer PTSD intrusions but similar distress after trauma

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Abstract

People with aphantasia, who lack mental imagery, report fewer intrusive memories after trauma. Imagery is central to cognitive models of post-traumatic stress disorder (PTSD), but whether it makes trauma harmful is unknown; previous studies neither measured exposure nor tested questionnaire equivalence without imagery. We surveyed 340 adults with congenital aphantasia, 261 with imagery from the same community and 471 controls. Questionnaires functioned broadly equivalently. At equal exposure, people with aphantasia reported fewer intrusions and less avoidance, more trauma amnesia and less depersonalisation, but similar depression, anxiety, mood and arousal. Trauma was associated with their distress no less strongly than in people with imagery. Importantly, at equal exposure and distress, they had about half the odds of controls of reaching the PTSD screening cut-off. Imagery appears tied to how trauma is re-experienced, not how much distress follows it; low PTSD checklist score without imagery does not mean trauma had little effect.

Introduction

Post-traumatic stress disorder (PTSD) is defined by intrusive re-experiencing, in the form of unwanted memories, nightmares and flashbacks in which the event is relived as if it were happening again (DSM-5; American Psychiatric Association, 2013). These intrusions are predominantly sensory and most often visual (Brewin et al., 2010; Ehlers & Clark, 2000; Ehlers et al., 2004), and mental imagery has a central place in cognitive models of the disorder and in its treatment (Holmes & Mathews, 2010). Clinical theories propose that trauma affects memory in two distinct ways: involuntary perceptual memories of the event are strengthened, giving rise to flashbacks, while voluntary episodic recall becomes fragmented, giving rise to gaps in memory for the event (Brewin, 2014; Brewin, Dalgleish, & Joseph, 1996; Ehlers & Clark, 2000). However, post-traumatic symptoms do not form a unitary construct. Flashbacks are specific to PTSD, whereas most other post-traumatic symptoms are shared with other disorders (Brewin, 2014; Bryant et al., 2011). Factor analyses likewise distinguish intrusions, the component most specific to PTSD and most closely tied to trauma exposure, from a broad dysphoria component comprising numbing, detachment, gaps in memory for the event, and problems with concentration and sleep (Simms et al., 2002). This dysphoria component is closely related to depression and corresponds to the general distress shared across anxiety and depressive disorders (Clark & Watson, 1991; Simms et al., 2002). Cognitive models link imagery to intrusive re-experiencing, but whether imagery is also related to the dysphoric, depression-like symptoms that follow trauma is unknown.

Intrusive memories after trauma may be an extreme form of a common phenomenon. Most people experience involuntary autobiographical memories, memories of past events that come to mind without any attempt to retrieve them, as well as involuntary thoughts about the future, several times a day (Berntsen et al., 2015). Involuntary memories are common in non-clinical samples and in depression, and those in PTSD are distinguished mainly by their sensory detail and sense of occurring in the present (Brewin, Christodoulides, & Hutchinson, 1996; Reynolds & Brewin, 1998). If intrusions are an instance of this broader tendency, individual differences in imagery may influence intrusions partly by modulating the frequency of involuntary retrieval. In a general adult sample, more vivid imagery was associated with more frequent involuntary autobiographical memories, which in turn were associated with a latent factor of depression, stress and neuroticism (Kvamme, Rutiku, et al., 2026). Imagery may therefore be related to distress not only through intrusions after trauma but also through involuntary memory in everyday life.

Aphantasia refers to the absence of voluntary visual imagery; most affected individuals report never having experienced imagery, a form known as congenital aphantasia (Zeman et al., 2015). Their autobiographical memory is characterised by knowledge of past events rather than re-experiencing of them (Dawes et al., 2020; Milton et al., 2021), and involuntary imagery, including imagery in dreams, is also reduced (Krempel & Monzel, 2024). Aphantasia is not associated with a general increase in psychopathology (Monzel, Vetterlein, & Reuter, 2023), although weak imagery may be associated with poorer mental health than its complete absence (Kvamme, Monzel, et al., 2026). Imagery may also be linked to dissociation: in depersonalisation disorder, more severe depersonalisation accompanies an impaired ability to generate images (Lambert et al., 2001), and in PTSD, intrusive re-experiencing and the dissociative subtype have been linked to opposite patterns of anterior insula activity (Kvamme, Nagai, & Silvanto, 2026).

Evidence on aphantasia and trauma comes from three empirical studies. After viewing a distressing film, individuals with aphantasia reported fewer intrusive memories, both immediately and over the following week, and those they reported were predominantly verbal (Keogh et al., 2024). In a survey using the PTSD Checklist for DSM-5 (PCL-5; Weathers, Litz, et al., 2013), people with aphantasia did not differ from controls in total score but reported fewer intrusion and avoidance symptoms, with inconsistent differences in mood and cognition, most of which did not survive correction for multiple comparisons (Dawes et al., 2020). In a subsequent study, people with aphantasia who reported PTSD were much less likely than those with imagery to report intrusive sensory experiences (43% against 93%), although only visual intrusions differed and the prevalence of PTSD itself did not (Mawtus et al., 2024). A narrative review of ten sources likewise reported fewer flashbacks in trauma-exposed people with aphantasia (Northover et al., 2026). The sensory character of intrusive memories thus appears to depend on imagery. However, the laboratory study did not use real trauma, neither survey measured trauma exposure, and the PCL-5 and the Dissociative Experiences Scale (DES; E. M. Bernstein & Putnam, 1986) ask about unwanted memories, dreams and reliving, which people without imagery may be unable to endorse whatever their response to trauma. It therefore remains unknown whether fewer intrusions reflect less distress after trauma or the same distress expressed differently. The answer matters because, if imagery is the route by which trauma causes harm, people with aphantasia should be protected from it. In contrast, if imagery is merely the route by which trauma is re-experienced, people with aphantasia should be as distressed as people with imagery, but their distress may be missed by PTSD measures, which centre on re-experiencing.

Answering this question requires comparing people with and without imagery at equal trauma exposure, on measures shown to function equivalently in both groups. Congenital aphantasia makes such a comparison possible, and two questions follow from the two components of post-traumatic symptoms. The first concerns whether fewer intrusions persist when exposure is held constant. If aphantasia reflects an absence of voluntary imagery only, involuntary re-experiencing should be preserved, whereas if it reflects an absence of imagery altogether, intrusions should be reduced (Cavedon-Taylor, 2022). The second concerns overall distress, which we index by depression, anxiety and post-traumatic stress. People without imagery may show less distress after trauma (a protective account), the same distress expressed through other symptoms (a same-burden account), or more distress if re-experiencing contributes to the processing of trauma, as proposed by dual representation theory, in which attention to flashbacks allows sensory memories to be integrated into episodic memory (a hidden-cost account; Brewin, 2014; Brewin, Dalgleish, & Joseph, 1996). These accounts predict, respectively, a weaker, equal or stronger association between trauma exposure and distress in people without imagery.

The objective of this study was to determine whether the absence of mental imagery is related to the overall level of distress after trauma, or only to its pattern, that is, whether imagery makes trauma harmful or only makes it re-experienced. We compared adults with congenital aphantasia, general-population controls and people with imagery recruited from the same community as the aphantasia group, who provide a reference group free of differences in recruitment source. Measuring childhood and lifetime trauma allowed the groups to be compared at statistically equal exposure, and multi-group factor models tested whether the questionnaires measure the same constructs with and without imagery. We then asked whether the pattern of post-traumatic

symptoms differs at equal exposure, and whether trauma predicts depression, anxiety and post-traumatic stress as strongly without imagery. Secondary analyses examined involuntary autobiographical memory, dissociation, and how these differences affect who meets PTSD screening and diagnostic criteria.

Methods

Participants

Participants were recruited through two routes and completed the same online survey. Members of the Aphantasia Network were invited through a survey link circulated to the Network's membership between 10 January and 4 March 2026; recruitment was open, so the number of respondents could not be fixed in advance, and all responses received were retained for screening after respondents who reported an age under 18 had been removed (n = 1,038). Aphantasia Network members took part voluntarily without compensation. A general-population sample was recruited through Prolific in two waves and paid US$5 each. The first wave (23–28 November 2025; n = 387) was followed, after Network recruitment had closed, by a second wave (5–6 May 2026; n = 108) that brought the sample to a size comparable to the aphantasia group and improved its gender balance (n = 495 in total). This decision was based on sample size and composition only, without reference to outcome data. Gender was self-reported; biological sex was not separately assessed, and data on race, ethnicity and socioeconomic status were not collected. The study was approved by the University of Macau Ethics Committee (Approval ID: HE-0608-2025), and all participants provided informed consent prior to participation.

Groups were defined by the mean score on the Plymouth Sensory Imagery Questionnaire (Psi-Q; see Questionnaire measures). Controls were Prolific participants with a mean score above 4 (n = 471; mean age 48.8 years, SD 14.3; 62.0% women, 37.4% men, 0.6% another gender). Congenital aphantasia was defined as Aphantasia Network members who reported never having had imagery and had a mean score of 4 or below (n = 340; mean age 51.8, SD 16.0; 67.4% women, 30.3% men, 2.4% another gender); of these, 266 rated every item of the visual subscale at zero and formed the core aphantasia subgroup. Aphantasia Network members with a mean score above 4 formed a second reference group with the same recruitment source (n = 261; mean age 48.3, SD 17.0; 77.8% women, 19.2% men, 3.1% another gender). Excluded were 13 Prolific participants with a mean score of 4 or below, 181 participants without Psi-Q data (11 from Prolific, 170 from the Aphantasia Network), and 267 Aphantasia Network members with a mean score of 4 or below who did not report lifelong absence of imagery or did not answer the question.

Questionnaire measures

Imagery was measured with the Psi-Q (Andrade et al., 2014). It consists of 35 items, five for each of seven modalities (vision, sound, smell, taste, touch, bodily sensation and emotional feeling), on which participants rate the vividness of their imagery from 0 (no image at all) to 10 (as vivid as real life). Childhood trauma was measured with the 25 clinical items of the Childhood Trauma Questionnaire short form (CTQ; D. P. Bernstein et al., 2003), summed over its five subscales (emotional, physical and sexual abuse, emotional and physical neglect). Lifetime traumatic events were measured with the Life Events Checklist for DSM-5 (LEC-5; Weathers, Blake, et al., 2013), scored as the number of its 17 events that the participant had experienced or witnessed. Post-traumatic stress was measured with the PCL-5 (Weathers, Litz, et al., 2013), on which participants rate 20 symptoms over the past month from 0 (not at all) to 4 (extremely) in relation to the event they identified as their worst. The PCL-5 was scored as a total and as the four DSM-5 symptom clusters: intrusion (items 1 to 5), avoidance (6 and 7), negative alterations in cognition and mood (8 to 14) and alterations in arousal and reactivity (15 to 20; hereafter arousal). A total of 33 or more was taken as the screening cut-off for probable PTSD (Bovin et al., 2016).

Depression was measured with the Patient Health Questionnaire-9 (PHQ-9; Kroenke et al., 2001) and anxiety with the Generalized Anxiety Disorder-7 (GAD-7; Spitzer et al., 2006), each rated over the past two weeks from 0 (not at all) to 3 (nearly every day); scores of 10 or more were taken to indicate clinical-range symptoms. Dissociation was measured with the 28-item DES (E. M. Bernstein & Putnam, 1986; Carlson & Putnam, 1993), on which participants rate the percentage of time they have each experience, from 0 to 100, and scored as the subscales of amnesia (items 3, 4, 5, 8, 25 and 26), depersonalisation and derealisation (items 7, 11, 12, 13, 27 and 28) and absorption (items 2, 14, 15, 16, 17, 18, 20, 22 and 23). The Multidimensional Assessment of Interoceptive Awareness (MAIA-2; 37 items; Mehling et al., 2018) entered the measurement analyses only. The Involuntary Autobiographical Memory Inventory (IAMI; Berntsen et al., 2015) asks how often memories of past events (ten items) and imagined future events (ten items) come to mind by themselves in everyday situations, rated from 1 (never) to 5 (every hour); each subscale was scored as the item mean. Internal consistency was acceptable to excellent in every group (Cronbach's α 0.73 to 0.99; Supplementary Table S1).

Statistical analysis

Exploratory factor analyses were conducted in Python (version 3.12) with factor_analyzer (version 0.5.1; Biggs, 2024), and the regression, logistic and mediation models with statsmodels (version 0.14.6; Seabold & Perktold, 2010). Confirmatory factor models were fitted in R (version 4.4.0; R Core Team, 2024) with lavaan (version 0.6-21; Rosseel, 2012) and semTools (version 0.5-7; Jorgensen et al., 2025). Each analysis used all participants with complete data on its variables, so sample sizes vary slightly between analyses. Gender entered the models as a binary covariate (women against men and participants of another gender). Unless stated otherwise, scores were standardised on the reference group of each comparison, so that coefficients are in standard deviations of the general-population control group or of Aphantasia Network members with imagery; slopes of distress on exposure and indirect effects were standardised on the controls. The symptom comparisons were repeated in the core aphantasia subgroup. The primary analyses (group differences in symptoms at equal exposure, and group differences in the slopes of distress on exposure) tested planned contrasts and were not corrected for multiple comparisons; the

remaining analyses were secondary or sensitivity analyses and are interpreted as exploratory. The study was not preregistered.

Measurement equivalence. All 181 items of the eight questionnaires entered an exploratory factor analysis (Spearman correlations through a rank transform, minimum residual extraction, oblimin rotation, number of factors by parallel analysis; Horn, 1965). Because items on which the groups differ will correlate in pooled data, we repeated the analysis after standardising the ranks of every item within each group (the three analysed groups and the excluded subgroups), which removes every group difference in item means. The factor structure on the 146 items other than the Psi-Q was compared between controls and congenital aphantasia with Tucker's congruence coefficient (Lorenzo-Seva & ten Berge, 2006), with the number of factors set to the smaller of the two numbers suggested by parallel analysis in each group, and the mean congruence was compared with its distribution across 50 random splits of the controls into a subsample of the size of the aphantasia group and the remainder; in each split the aphantasia group was also compared with the same remaining controls, so that both comparisons had equal group sizes. A coefficient of 0.95 or more was taken to indicate equal factors. For each questionnaire except the Psi-Q, on which the aphantasia group was at the floor, we then fitted multi-group ordinal confirmatory factor models (weighted least squares with mean- and variance-adjusted statistics, WLSMV; delta parameterisation) with the published structure (for the PCL-5, the four DSM-5 clusters as factors), identified as proposed by Wu and Estabrook (2016). Against each reference group we tested, in turn, equal thresholds, equal thresholds and loadings (metric invariance), and in addition equal intercepts (scalar invariance, under which the latent means may differ). Adjacent response categories were merged where a category had fewer than five responses in either group; DES responses were first grouped as 0, 10, 20, 30 to 40, and 50% or more of the time. Each step was accepted when the scaled comparative fit index (CFI) fell by no more than 0.010 and the scaled root mean square error of approximation (RMSEA) rose by no more than 0.015 relative to the preceding step (Chen, 2007). Where scalar invariance failed, the thresholds and intercept of one item at a time were freed in order of their score-test statistics, up to six items, and freeing stopped when it no longer improved the fit, to test partial invariance. Finally, each rank-transformed item was regressed on the mean of the other items of its factor, group, age and gender (in participants who reported being women or men), to identify items answered differently by people with aphantasia at the same level of the factor. Items were classed as biased when the group coefficient reached 0.25 SD, survived Benjamini–Hochberg correction (Benjamini & Hochberg, 1995) and had the same sign against both reference groups.

Exposure and symptom differences. Trauma exposure was compared between groups with Cohen's d. Symptom differences were estimated by linear regression of each standardised score on group, CTQ total, LEC-5 count, age and gender, so that groups were compared at the same level of exposure. Three of the four DES items classed as biased (14, 17 and 18) belong to the absorption subscale and were removed from it, so that absorption comprised items 2, 15, 16, 20, 22 and 23; the fourth, item 9, belongs to none of the three subscales and was analysed separately. The PCL-5 total was also analysed without item 8, and the screening analyses without items 3 and 8. Participants who reported no traumatic event on the LEC-5 completed the PCL-5 for their most stressful experience and were retained in all analyses.

Burden of trauma. To distinguish the protective, same-burden and hidden-cost accounts, depression, anxiety and PCL-5 total were regressed on exposure, group and their interaction, with age and gender as covariates, separately for CTQ total and LEC-5 count. A smaller slope in aphantasia would support the protective account, a slope that did not differ the same-burden account, and a larger slope the hidden-cost account. Because a non-significant difference does not show that two slopes are equal, we also tested each slope difference for equivalence with two one-sided tests (Lakens, 2017), against bounds of ±0.20 SD of the controls per SD of exposure, a small difference in slope. A slope difference was classed as equivalent when its 90% confidence interval lay within these bounds.

Dissociation. To test whether distress is associated with dissociation as strongly in aphantasia as in people with imagery, the same model was fitted with depersonalisation and derealisation as the outcome and each distress measure in turn as the predictor. Within each group with imagery, we computed the Spearman correlation between Psi-Q vividness and depersonalisation and derealisation, without and with partialling out depression and anxiety.

Indirect associations. Indirect associations of childhood trauma with depression were estimated by parallel mediation in each group, with two mediators: the intrusion cluster, and the mean of the items on trauma amnesia (item 8), loss of interest (12), feeling distant (13), trouble feeling positive (14) and difficulty concentrating (19). Because items 12 to 14 and 19 overlap in content with depression, the analysis was repeated with items 8 and 19 only. Models contained no covariates, and all variables were standardised on the controls. Indirect effects and their differences between each pair of groups were tested with 2,000 percentile bootstrap samples.

Trauma amnesia. The association of trauma amnesia with distress was tested by regressing depression and the numbing items (12 to 14) on PCL-5 item 8, with exposure, age and gender as covariates. To test whether trauma amnesia reflects general gaps in autobiographical memory, the group comparison of item 8 and both regressions were repeated with DES item 9 as an additional covariate, and the association of DES item 9 with depression was compared between groups through an interaction term.

Screening and diagnosis. The probability of a PCL-5 total of 33 or more was modelled by logistic regression on group, adjusting in turn for age and gender, for exposure, and for exposure together with depression and anxiety, in participants with complete data on all covariates, and repeated with the total rescored without items 3 and 8 and prorated to 20 items. We also applied the DSM-5 symptom rule to the PCL-5, in which an item rated 2 (“moderately”) or higher counts as a symptom and a provisional diagnosis requires at least one intrusion, one avoidance, two cognition and mood and two arousal symptoms (Weathers, Litz, et al., 2013), and compared the proportion of each group meeting each criterion against each reference group at equal exposure. Because the survey contained no measure of functional impairment, these comparisons were repeated among participants with clinical-range depression or anxiety (PHQ-9 or GAD-7 of 10 or more).

Involuntary memory. To test whether fewer intrusions are part of a general reduction in involuntary memory, the IAMI past-memory score was added to the models of the four PCL-5 clusters, the

PCL-5 total and the cut-off, and the change in the group coefficient was recorded. The slope of intrusions on the IAMI score was compared between groups through an interaction term.

Results

Descriptive statistics for the three groups are shown in Table 1.

Table 1. *Participant Characteristics by Group*

Measure	*Congenital aphantasia (n = 340)*	*General-population controls (n = 471)*	*Aphantasia Network members with imagery (n = 261)*	*n*
Demographics				
Age, years	51.8 (16.0)	48.8 (14.3)	48.3 (17.0)	340 / 471 / 260
Women, n (%)	229 (67.4)	292 (62.0)	203 (77.8)	340 / 471 / 261
Men, n (%)	103 (30.3)	176 (37.4)	50 (19.2)	340 / 471 / 261
Another gender, n (%)	8 (2.4)	3 (0.6)	8 (3.1)	340 / 471 / 261
Imagery (Psi-Q)				
All modalities	0.73 (1.03)	7.79 (1.41)	7.14 (1.83)	340 / 471 / 261
Visual subscale	0.22 (0.56)	8.29 (1.48)	5.35 (3.87)	340 / 471 / 261
Trauma exposure				
CTQ total (25–125)	46.5 (16.9)	44.9 (18.5)	50.2 (19.0)	302 / 469 / 245
Emotional abuse	10.8 (5.8)	10.2 (5.8)	12.6 (6.4)	302 / 469 / 245
Physical abuse	6.9 (3.3)	7.9 (4.4)	7.7 (4.2)	302 / 469 / 245
Sexual abuse	7.8 (4.8)	8.0 (5.2)	8.3 (5.0)	302 / 469 / 245
Emotional neglect	13.4 (5.8)	11.1 (5.4)	13.5 (5.6)	302 / 469 / 245
Physical neglect	7.6 (3.3)	7.7 (3.5)	8.0 (3.6)	302 / 469 / 245
LEC-5 events (0–17)	5.1 (2.9)	5.2 (3.3)	5.8 (3.3)	304 / 463 / 242
At least one event, n (%)	294 (96.7)	437 (94.4)	236 (97.5)	304 / 463 / 242
Depression and anxiety				
PHQ-9 (0–27)	6.8 (5.8)	6.5 (6.0)	8.1 (6.1)	340 / 469 / 259
PHQ-9 ≥ 10, n (%)	101 (29.7)	130 (27.7)	95 (36.7)	340 / 469 / 259
GAD-7 (0–21)	5.1 (5.2)	5.6 (5.4)	6.8 (5.5)	299 / 467 / 235
GAD-7 ≥ 10, n (%)	58 (19.4)	105 (22.5)	68 (28.9)	299 / 467 / 235
Post-traumatic stress (PCL-5)				

Total (0–80)	18.4 (15.8)	23.4 (17.6)	25.8 (16.9)	307 / 467 / 238
Intrusion (0–20)	3.1 (3.8)	6.1 (5.3)	6.1 (5.2)	307 / 467 / 238
Avoidance (0–8)	1.6 (2.1)	2.9 (2.5)	2.6 (2.5)	307 / 467 / 238
Negative alterations in cognition and mood (0–28)	7.8 (7.0)	8.1 (6.9)	9.4 (6.8)	307 / 467 / 238
Arousal and reactivity (0–24)	6.0 (5.1)	6.3 (5.3)	7.6 (5.3)	307 / 467 / 238
Total ≥ 33, n (%)	56 (18.2)	131 (28.1)	75 (31.5)	307 / 467 / 238
Dissociation (DES, 0–100)				
Amnesia	7.8 (12.9)	12.2 (18.6)	10.4 (14.2)	303 / 466 / 244
Depersonalisation and derealisation	6.0 (11.3)	11.3 (18.8)	12.2 (18.5)	303 / 466 / 244
Absorption	19.5 (17.7)	23.6 (20.5)	27.3 (21.1)	303 / 466 / 244
Item 9, no memory of life events	36.4 (34.1)	14.6 (22.8)	23.7 (30.5)	303 / 466 / 244
Involuntary autobiographical memory (IAMI, 1–5)				
Past memories	2.56 (0.88)	3.29 (0.72)	3.64 (0.93)	295 / 466 / 238
Future thoughts	1.94 (0.92)	2.82 (0.88)	3.06 (1.15)	295 / 466 / 238

Note. Values are M (SD) unless stated otherwise. The n column gives the number of participants with complete data on each measure (congenital aphantasia / general-population control group / Aphantasia Network members with imagery). Psi-Q = Plymouth Sensory Imagery Questionnaire; CTQ = Childhood Trauma Questionnaire short form; LEC-5 = Life Events Checklist for DSM-5 (events experienced or witnessed); PHQ-9 = Patient Health Questionnaire-9; GAD-7 = Generalized Anxiety Disorder-7; PCL-5 = PTSD Checklist for DSM-5; DES = Dissociative Experiences Scale; IAMI = Involuntary Autobiographical Memory Inventory. DES absorption excludes items 14, 17 and 18, which were answered differently in aphantasia; item 9, which belongs to no subscale, is shown separately. Ranges of possible scores are given in parentheses. PCL-5 ≥ 33 is given for all participants with PCL-5 data; the screening analyses use the subsample with complete covariate data.

Trauma exposure, depression and anxiety did not differ significantly from controls

We first compared trauma exposure and distress, since fewer post-traumatic symptoms in aphantasia could otherwise reflect less trauma. Participants were not selected for trauma, but nearly all had experienced or witnessed at least one traumatic event (96.7% of people with congenital aphantasia, 94.4% of controls and 97.5% of Aphantasia Network members with imagery; Table 1), so most had an event to which the PCL-5 could refer. People with congenital aphantasia did not differ from the general-population control group in total childhood trauma ($d = 0.08$, 95% CI −0.06 to 0.23, $p = .25$) or in the number of traumatic events they had experienced or witnessed ($d = −0.01$, 95% CI −0.16 to 0.13, $p = .87$; Figure 1), although they reported more emotional neglect ($d = 0.41$, 95% CI 0.26 to 0.56, $p < .001$) and less physical abuse ($d = −0.26$, 95% CI −0.41 to −0.12, $p < .001$). Aphantasia Network members with imagery reported slightly

more childhood trauma (d = 0.21, 95% CI 0.04 to 0.38, p = .016) and more traumatic events (d = 0.22, 95% CI 0.05 to 0.39, p = .011) than the aphantasia group. At equal exposure, people with congenital aphantasia did not differ from controls in depression (0.07 SD, 95% CI −0.06 to 0.20, p = .32) or anxiety (−0.07 SD, 95% CI −0.21 to 0.06, p = .29). Against Aphantasia Network members with imagery, depression did not differ (−0.10 SD, 95% CI −0.26 to 0.05, p = .20), but anxiety was slightly lower (−0.17 SD, 95% CI −0.33 to −0.01, p = .033). Thus people with congenital aphantasia did not differ significantly from general-population controls in trauma exposure, depression or anxiety, although they reported slightly less anxiety than Aphantasia Network members with imagery. Group differences in post-traumatic symptoms are therefore unlikely to reflect large differences in exposure or in overall distress, and all symptom comparisons below were made at equal exposure.

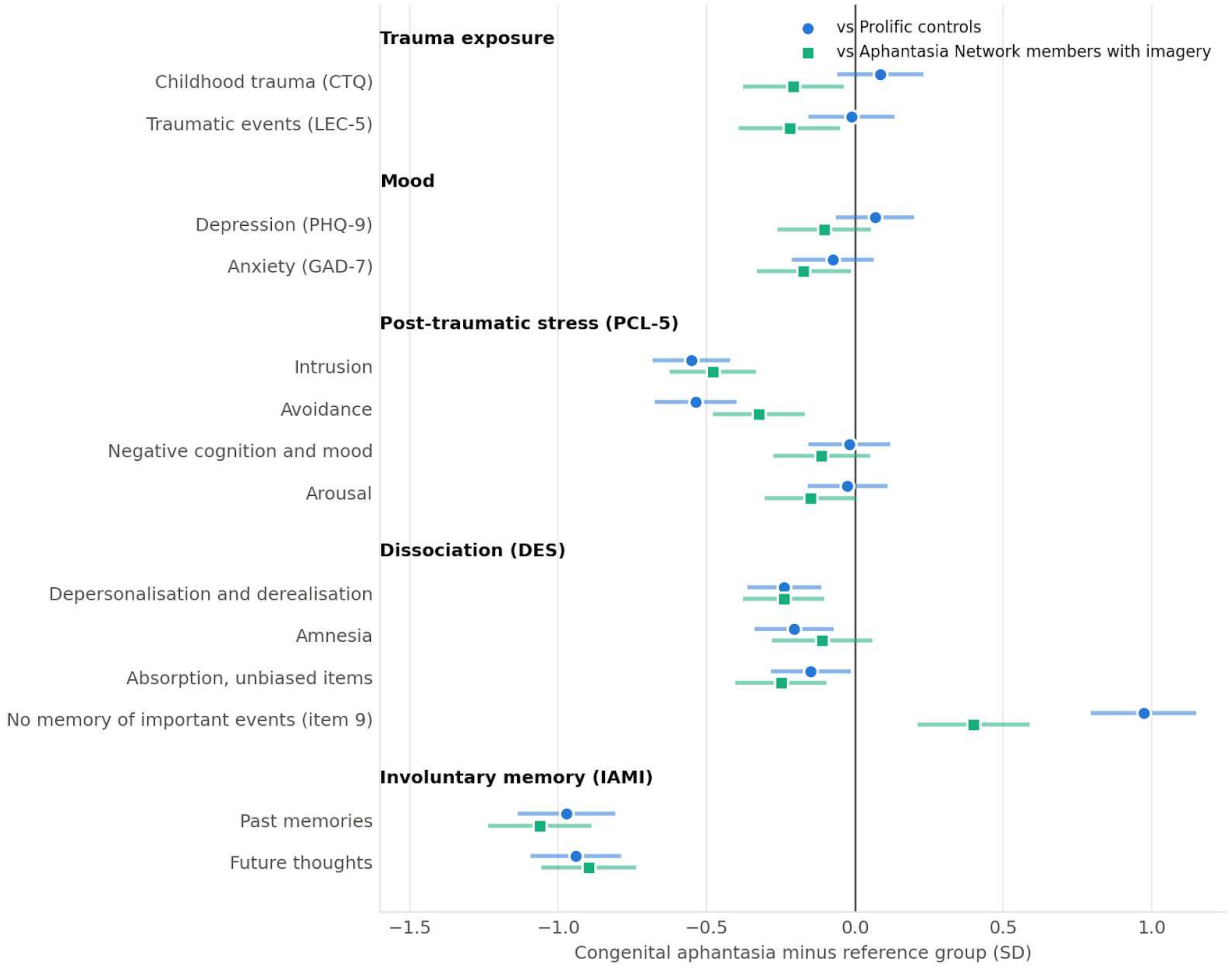


Figure 1. *Trauma exposure, mood, post-traumatic stress, dissociation and involuntary memory in congenital aphantasia (n = 340). Points are differences from the general-population control group (circles, n = 471) and from Aphantasia Network members with imagery (squares, n = 261) in standard deviations of the reference group, with 95% confidence intervals. Group sizes are totals; each comparison used the participants with complete data on its variables (Table 1).*

Exposure rows are Cohen's d without adjustment; all other rows are regression coefficients at equal childhood trauma (CTQ) and traumatic events (LEC-5), adjusted for age and gender. DES absorption excludes items 14, 17 and 18, which were answered differently in aphantasia; DES item 9 is shown separately.

Measurement equivalence of the questionnaires

We next assessed whether the questionnaires measured the same constructs in people with and without imagery, so that group differences in scores could be interpreted as differences in symptoms. In an exploratory factor analysis of all 181 items, most items loaded on factors corresponding to their own questionnaire or subscale, so that the factor structure largely reproduced the published instruments; the exception was that the depression and anxiety items loaded on a single factor together with the mood and arousal items of the PCL-5 (Supplementary Figure S1). Items on which the groups differ will correlate in pooled data for that reason alone, so a group difference in means could produce spurious factors. We therefore repeated the analysis after standardising every item within each group, which removes all differences between the groups while preserving the relations among items within each group. Ten of the 13 factors kept a Tucker congruence of 0.90 or more with the pooled solution (nine of 0.95 or more), indicating that the factor structure did not arise from pooling the groups. To compare the structure directly, we fitted a 10-factor solution, the smaller number suggested by parallel analysis in either group, separately in each group on the 146 items other than the Psi-Q. The solutions for controls and for congenital aphantasia were about as similar as those for two random subsamples of the controls (mean congruence 0.875 against 0.866); compared with the same control subsamples, so that both comparisons had equal group sizes, the aphantasia group's mean congruence was 0.855 (difference from the control-only comparison −0.011, 5th to 95th percentile −0.039 to 0.020). Both values were below the 0.95 usually taken to indicate equal factors (Lorenzo-Seva & ten Berge, 2006).

We then tested each questionnaire separately in multi-group confirmatory models. The PCL-5, CTQ, PHQ-9, GAD-7, MAIA-2 and IAMI met scalar invariance against both reference groups (across the threshold, metric and scalar steps, ΔCFI −0.004 to +0.005 and ΔRMSEA −0.018 to +0.003; criteria of Chen, 2007; Supplementary Figure S2). The DES met threshold and metric invariance but not scalar invariance (ΔCFI −0.027 against controls and −0.042 against Aphantasia Network members with imagery). It reached partial scalar invariance after the thresholds and intercepts of items 14 and 9 were freed against controls, and of items 14, 9 and 18 against Aphantasia Network members with imagery, items that the item-level analysis below also identified. The configural RMSEA of the IAMI, GAD-7 and PHQ-9 was high (0.10 to 0.12), although their configural CFI was 0.949 or higher.

Finally, we tested whether individual items were answered differently by people with aphantasia at the same level of their factor. Seven of the 143 non-imagery items tested (the other three belonged to factors with too few items for the test) were answered differently in both comparisons, with group coefficients from −0.98 to +0.91 SD (Supplementary Table S2), and their content was consistent with the absence of imagery. Two were PCL-5 items: feeling as if the stressful experience were happening again (item 3) was endorsed less, and trouble remembering

important parts of it (item 8) was endorsed more. Four were DES items: having no memory of important life events (item 9), reliving a past event so vividly that it feels as if it is happening again (item 14), becoming so absorbed in a film or television programme as to be unaware of other events (item 17), and becoming so involved in a fantasy or daydream that it feels real (item 18). The seventh was a MAIA-2 item, maintaining awareness of the whole body when a part is in pain or discomfort (item 21). Against the general-population control group alone, 31 items met the criterion, including all seven PCL-5 intrusion and avoidance items. This reflected their loading on a shared factor with mood items in the exploratory solution: when the four DSM-5 clusters were modelled as separate factors, the PCL-5 met scalar invariance against controls (scalar model CFI 0.963, RMSEA 0.080; ΔCFI −0.004).

These analyses show that the questionnaires measured broadly the same constructs in people with and without imagery, and the group comparisons that follow can be read as differences in symptoms, not in how the questions were understood. The exceptions were confined to a few items that ask about reliving events or about remembering them, which people without imagery answered in the direction predicted by their wording. We therefore removed the three biased absorption items from the DES absorption subscale, analysed DES item 9 on its own, analysed the PCL-5 total without item 8, and repeated the screening analyses without items 3 and 8. The IAMI, whose configural fit was poor, was used only in the analyses of involuntary memory, and those results are interpreted with this caution.

Intrusion and avoidance symptoms were lower and trauma amnesia was higher in aphantasia

We next compared post-traumatic symptoms between the groups at equal trauma exposure, adjusting for childhood trauma, traumatic events, age and gender. People with congenital aphantasia differed from people with imagery in the symptoms that involve re-experiencing, but not significantly in mood or arousal. They reported fewer intrusion symptoms than controls (−0.55 SD, 95% CI −0.68 to −0.42, $p < .001$) and less avoidance (−0.54 SD, 95% CI −0.67 to −0.40, $p < .001$), whereas negative alterations in cognition and mood (−0.02 SD, 95% CI −0.16 to 0.12, $p = .79$) and arousal (−0.03 SD, 95% CI −0.16 to 0.11, $p = .71$) did not differ (Figure 1). Every intrusion and avoidance item was lower, including disturbing dreams (−0.53 SD, 95% CI −0.65 to −0.40, $p < .001$; Figure 2). Two items in other clusters were higher: trouble remembering important parts of the event (0.42 SD, 95% CI 0.25 to 0.58, $p < .001$) and difficulty concentrating (0.23 SD, 95% CI 0.09 to 0.38, $p = .002$). Three were slightly lower: loss of interest, irritability and risk-taking (−0.16 to −0.20 SD, $p = .003$ to .031). Against Aphantasia Network members with imagery, the pattern was the same for intrusion (−0.48 SD, 95% CI −0.62 to −0.33, $p < .001$), avoidance (−0.32 SD, 95% CI −0.48 to −0.17, $p < .001$) and trauma amnesia (0.31 SD, 95% CI 0.11 to 0.50, $p = .002$), but not for difficulty concentrating (−0.09 SD, 95% CI −0.26 to 0.08, $p = .29$). In the core aphantasia subgroup ($n = 266$), the estimates against controls were close to those of the whole group (intrusion −0.55 SD, 95% CI −0.70 to −0.41; trauma amnesia 0.41 SD, 95% CI 0.23 to 0.58; both $p < .001$).

Because mood and arousal symptoms, which make up most of the PCL-5, did not differ, the PCL-5 total differed less than the intrusion and avoidance clusters (−0.26 SD, 95% CI −0.39 to −0.13, $p < .001$). Without item 8, which people with aphantasia endorsed more, the difference was slightly larger (−0.29 SD, 95% CI −0.42 to −0.16, $p < .001$). Thus, at the same level of trauma, people without imagery reported a post-traumatic response of a different form: fewer intrusions and less avoidance, more amnesia for the event, and no significant difference in negative mood and arousal.

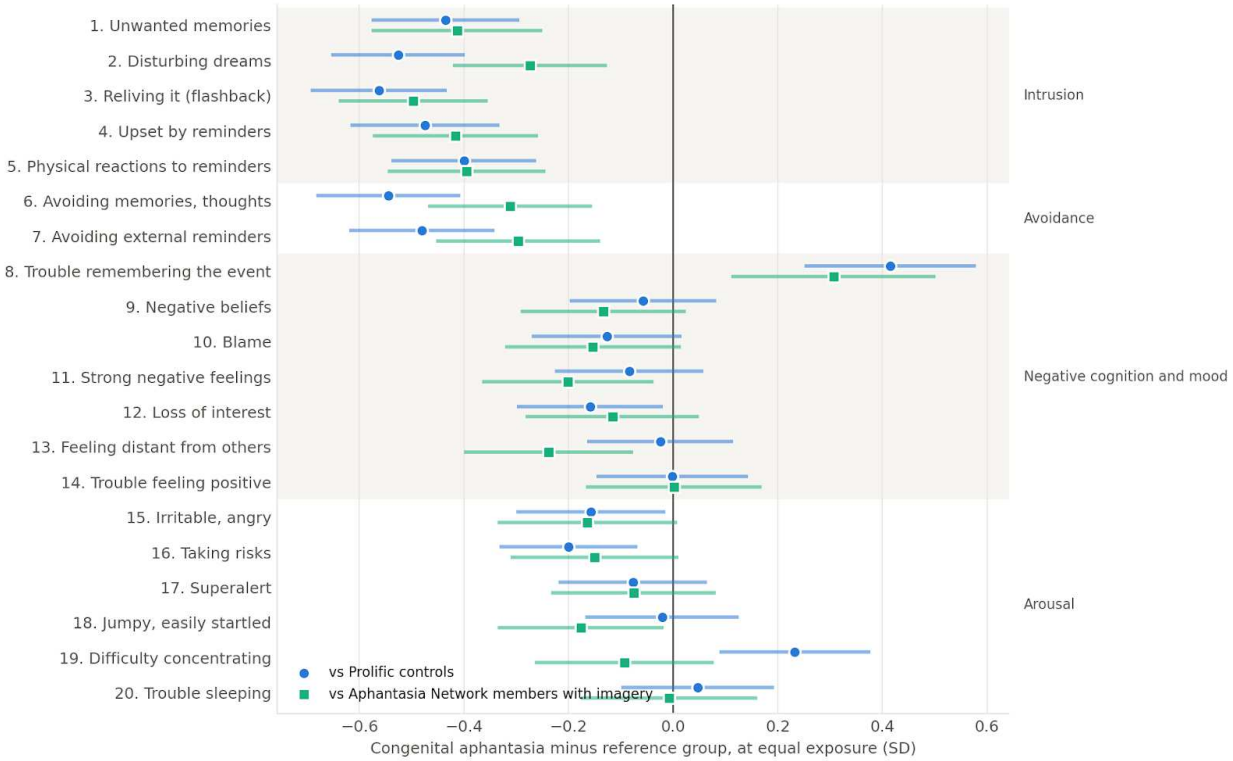


Figure 2. *Each PCL-5 item in congenital aphantasia, relative to the general-population control group (circles) and Aphantasia Network members with imagery (squares), at equal childhood trauma and traumatic events and adjusted for age and gender. Units are standard deviations of the reference group; lines are 95% confidence intervals. Shaded bands mark the four DSM-5 symptom clusters. PCL-5 = PTSD Checklist for DSM-5.*

Dissociation and involuntary memory

We next examined dissociation, with absorption scored without the three items answered differently in aphantasia. People with congenital aphantasia reported less depersonalisation and derealisation than people with imagery. At equal exposure, depersonalisation and derealisation were lower than in controls (−0.24 SD, 95% CI −0.36 to −0.11, p < .001) and than in Aphantasia Network members with imagery (−0.24 SD, 95% CI −0.38 to −0.10, p < .001), as was absorption (−0.15 SD, 95% CI −0.28 to −0.01, p = .030, and −0.25 SD, 95% CI −0.40 to −0.10, p = .001, respectively). Dissociative amnesia was lower than in controls (−0.21 SD, 95% CI −0.34 to −0.07, p = .002) and did not differ significantly from Aphantasia Network members with imagery (−0.11 SD, 95% CI −0.28 to 0.06, p = .20). In contrast, having no memory of important life events (DES item 9) was higher (0.97 SD, 95% CI 0.80 to 1.15, p < .001, against controls; 0.40 SD, 95% CI 0.21 to 0.59, p < .001, against Aphantasia Network members with imagery).

To test whether people with aphantasia respond less to distress or start from a lower level, we compared the slopes of depersonalisation and derealisation on each distress measure. Depersonalisation and derealisation increased with depression at a similar rate in aphantasia and in controls (slope 0.31 in aphantasia against 0.29 in controls; difference 0.02, 95% CI −0.10 to 0.14, p = .71), and the slopes on anxiety, childhood trauma, traumatic events and PCL-5 total did not differ either (all p > .54). At the mean level of depression in controls, scores in aphantasia were 0.27 SD lower (95% CI −0.38 to −0.15, p < .001). Against Aphantasia Network members with imagery, the slopes on depression, anxiety and PCL-5 total did not differ either (all p > .24), but the slope on traumatic events was flatter in aphantasia (0.14 against 0.29, p = .032); the slope on childhood trauma was flatter in the same direction but not significantly (0.22 against 0.35, p = .054).

In depersonalisation disorder, weaker imagery accompanies more severe depersonalisation (Lambert et al., 2001). We therefore asked whether the same holds within the normal range of imagery and found no consistent association. In controls (n = 462), weaker imagery was associated with slightly more depersonalisation and derealisation (rho = −0.10, 95% CI −0.19 to −0.01, p = .031), but not after adjustment for depression and anxiety (partial rho = −0.06, 95% CI −0.15 to 0.03, p = .21), and in Aphantasia Network members with imagery (n = 229) the association had the opposite sign (rho = 0.15, 95% CI 0.02 to 0.27, p = .026; partial rho = 0.11, 95% CI −0.02 to 0.23, p = .11).

Involuntary autobiographical memory was also markedly reduced. People with congenital aphantasia reported fewer involuntary past memories (−0.97 SD, 95% CI −1.14 to −0.81, p < .001) and involuntary future thoughts (−0.94 SD, 95% CI −1.09 to −0.79, p < .001) than controls, with similar differences against Aphantasia Network members with imagery (−1.06 SD, 95% CI −1.24 to −0.89, and −0.90 SD, 95% CI −1.06 to −0.74; both p < .001). This difference, of about one standard deviation, is examined as a possible basis for the lower intrusion scores below.

Overall, congenital aphantasia was associated with less depersonalisation and derealisation, but not with a weaker response to distress: dissociation started from a lower level and rose with depression, anxiety and post-traumatic stress at a rate not significantly different from people with imagery. Within the normal range of imagery, weaker imagery was not reliably associated with more dissociation. Dissociative amnesia in general was not increased (and was lower than in

controls), but gaps in memory for important life events were more common, and involuntary memories of the past and future were about one standard deviation less frequent.

Trauma was associated with distress comparably in people with and without imagery, except for childhood trauma with anxiety and post-traumatic stress

We then asked whether trauma is associated with less, the same or more distress in the absence of imagery. The protective, same-burden and hidden-cost accounts predict, respectively, a flatter, equal or steeper slope of distress on trauma exposure in people with aphantasia, so we compared these slopes between groups. The slopes did not differ significantly. Depression increased with childhood trauma at a similar rate in congenital aphantasia and in controls (slope 0.38 against 0.40; difference −0.02, 95% CI −0.16 to 0.12, p = .78; Figure 3a). The slopes for anxiety (0.40 against 0.29; difference 0.11, 95% CI −0.04 to 0.25, p = .15) and for PCL-5 total (0.45 against 0.34; difference 0.12, 95% CI −0.02 to 0.25, p = .10) did not differ significantly either, although both point estimates were steeper in aphantasia. None of the slopes on traumatic events differed (all p > .37), and the comparison with Aphantasia Network members with imagery gave the same result (all p > .42). Across the range of childhood trauma, PCL-5 totals in aphantasia were lower than in controls (Figure 3b), consistent with the lower intrusion and avoidance scores.

Thus trauma was clearly associated with depression, anxiety and post-traumatic stress in people without imagery (slopes 0.22 to 0.45), and these associations were not significantly weaker than in people with imagery. In the equivalence tests, 10 of the 12 slope differences lay within ±0.20 SD, including all of those for depression and for traumatic events (p for equivalence .005 to .047). The two exceptions were the slopes of anxiety and of PCL-5 total on childhood trauma against controls (90% CI −0.01 to 0.23, p = .11, and 0.00 to 0.23, p = .12), both steeper in aphantasia. In no comparison did the 90% confidence interval extend below −0.20 SD. The data therefore support the same-burden account for depression and for traumatic events, and rule out a protective effect larger than 0.20 SD, but cannot exclude a moderately stronger association of childhood trauma with anxiety and post-traumatic stress in aphantasia.

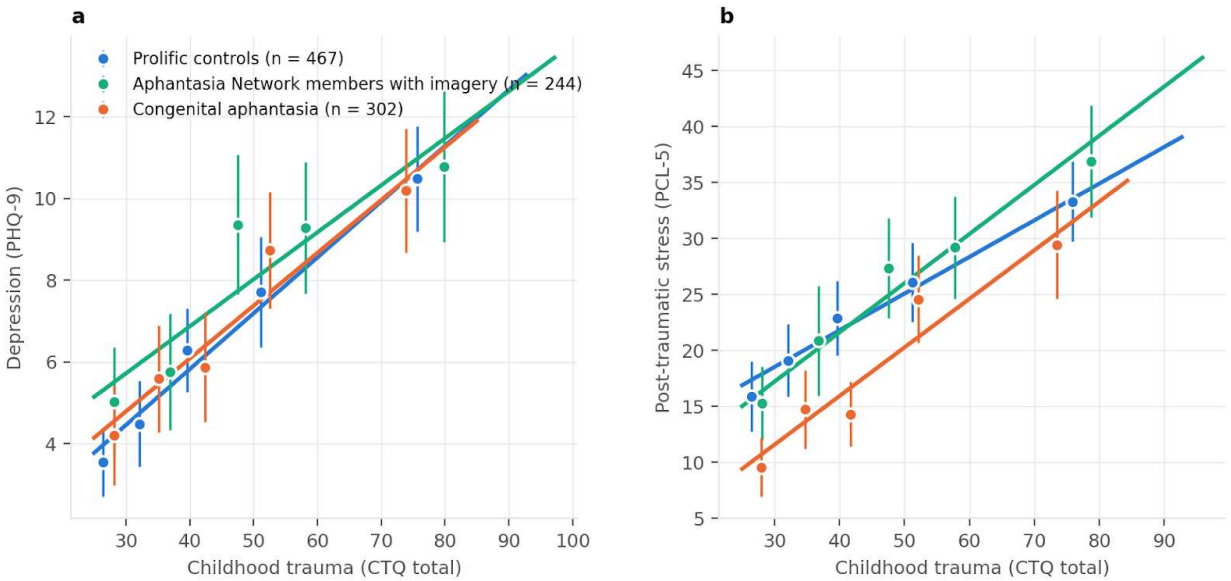


Figure 3. *Depression (a) and post-traumatic stress (b) against childhood trauma in each group. Points are means within quintiles of childhood trauma with 95% confidence intervals; lines are least-squares fits. Tests of the slope differences are reported in the text. CTQ = Childhood Trauma Questionnaire short form; PHQ-9 = Patient Health Questionnaire-9; PCL-5 = PTSD Checklist for DSM-5.*

Indirect associations of childhood trauma with depression

The same-burden account also allows that trauma may be related to depression by different routes: through memory gaps and numbing in people without imagery, and through intrusions in people with imagery. We tested this with a parallel mediation model in each group, in which childhood trauma was associated with depression through two mediators: the intrusion cluster, and a composite of trauma amnesia, numbing and concentration problems (PCL-5 items 8, 12, 13, 14 and 19). In all three groups, the indirect association ran mainly through the composite (indirect effect 0.31 in congenital aphantasia, 0.26 in controls and 0.29 in Aphantasia Network members with imagery) and barely through intrusions (0.01, −0.02 and 0.01), and none of the pairwise group differences in indirect effects was significant.

Because the numbing and concentration items overlap in content with depression (PHQ-9 items on loss of interest and concentration), we repeated the analysis with trauma amnesia and concentration problems (items 8 and 19) as the second mediator. This route still carried an indirect effect in every group (0.16 in congenital aphantasia, 0.14 in controls and 0.10 in Aphantasia Network members with imagery), and intrusions now carried a small one (0.05, 0.02 and 0.10). No difference between congenital aphantasia and either reference group was significant.

Thus, in all groups, childhood trauma was associated with depression mainly through the dysphoric symptoms of post-traumatic stress, which overlap with depression itself. When the second mediator was narrowed to trauma amnesia and concentration problems, both routes were

small. The groups differed more in how often intrusions occurred than in the route by which trauma was related to depression.

Trauma amnesia reflected general gaps in memory but was associated with distress

Higher trauma amnesia in aphantasia could be a benign consequence of remembering events as knowledge rather than as re-experience, or it could be a marker of distress, as it is typically taken to be in PTSD. We therefore tested whether trouble remembering parts of the traumatic event (PCL-5 item 8) was associated with depression and with numbing (PCL-5 items 12 to 14), adjusting for exposure, age and gender. In congenital aphantasia, greater trauma amnesia was associated with more depression (standardised beta 0.24, 95% CI 0.13 to 0.35, $p < .001$) and more numbing (0.37, 95% CI 0.27 to 0.47, $p < .001$), and the associations in controls were of similar size (0.22, 95% CI 0.14 to 0.30, and 0.31, 95% CI 0.23 to 0.40; both $p < .001$). In Aphantasia Network members with imagery, trauma amnesia was not associated with depression (0.01, 95% CI −0.12 to 0.13, $p = .94$) and only weakly and not significantly with numbing (0.09, 95% CI −0.04 to 0.21, $p = .17$).

We next asked whether the higher trauma amnesia in aphantasia was specific to the trauma or reflected a general difficulty in remembering important life events, measured by DES item 9. Most of the difference was general. Among participants with both items, adding DES item 9 as a covariate reduced the group difference in trauma amnesia from 0.40 to 0.14 SD against controls (95% CI −0.03 to 0.31, $p = .11$), and from 0.29 to 0.16 SD against Aphantasia Network members with imagery (95% CI −0.03 to 0.35, $p = .11$). With DES item 9 held constant, trauma amnesia in congenital aphantasia remained associated with depression (0.18, 95% CI 0.07 to 0.29, $p = .002$) and numbing (0.35, 95% CI 0.24 to 0.46, $p < .001$). General gaps in autobiographical memory were also associated with depression, independently of trauma amnesia, in aphantasia (0.22, 95% CI 0.11 to 0.34, $p < .001$) and, not significantly, in controls (0.07, 95% CI −0.02 to 0.15, $p = .12$), although this association did not differ significantly between the groups (interaction $p = .41$).

Thus the higher trauma amnesia in aphantasia largely reflected a general pattern of autobiographical memory rather than a response specific to trauma, but it was still associated with distress: in people with aphantasia, as in controls, not remembering parts of the trauma was associated with depression and numbing, whatever its origin.

Screening cut-off and DSM-5 criteria

Finally, we asked how these differences affect who meets the screening and diagnostic thresholds for PTSD. At equal exposure and equal depression and anxiety, people with congenital aphantasia had about half the odds of controls of reaching the screening cut-off, and they met the intrusion and avoidance criteria less often while meeting the mood and arousal criteria about as

often. A PCL-5 total of 33 or more was reached by 17.6% of people with congenital aphantasia, 28.5% of controls and 32.7% of Aphantasia Network members with imagery (participants with complete covariate data, n = 262, 456 and 217; Figure 4). Against controls, the odds ratio for aphantasia was 0.56 (95% CI 0.38 to 0.82, p = .003) adjusted for age and gender, 0.52 (0.35 to 0.78, p = .002) with exposure added, and 0.47 (0.30 to 0.76, p = .002) with depression and anxiety added. Against Aphantasia Network members with imagery, the corresponding odds ratios were 0.47 (0.31 to 0.73, p = .001), 0.53 (0.33 to 0.85, p = .008) and 0.59 (0.34 to 1.03, p = .064), so that with full adjustment the difference was not significant. Rescoring the PCL-5 without the two biased items (prorated to 20 items) left the odds ratios almost unchanged (0.46, 95% CI 0.28 to 0.74, against controls with full adjustment).

Under the DSM-5 symptom criteria (Table 2), people with congenital aphantasia met the intrusion criterion less often than controls at equal exposure (34.9% against 57.4%; odds ratio 0.38, 95% CI 0.27 to 0.52, p < .001), as well as the avoidance criterion (29.4% against 51.3%; 0.36, 95% CI 0.25 to 0.50, p < .001). Against Aphantasia Network members with imagery, the intrusion and avoidance criteria were also met less often (odds ratios 0.36, 95% CI 0.24 to 0.53, p < .001, and 0.56, 95% CI 0.38 to 0.84, p = .005), whereas the criteria for negative alterations in cognition and mood and for arousal did not differ significantly (Table 2). Against controls, people with congenital aphantasia met the criteria for negative alterations in cognition and mood (49.3% against 49.8%; 0.98, 95% CI 0.71 to 1.35) and for arousal (43.4% against 45.4%; 0.94, 95% CI 0.68 to 1.30) as often, and both of these criteria together almost as often (33.5% against 37.8%; 0.84, 95% CI 0.60 to 1.18). A provisional diagnosis under the full symptom rule was met by 18.8% of people with congenital aphantasia and 27.6% of controls (0.60, 95% CI 0.40 to 0.88, p = .009). Few participants were excluded by the intrusion requirement alone: meeting every criterion except intrusion was rare in both groups (2.9% and 2.2%), so the lower rate in aphantasia reflected fewer intrusion and avoidance symptoms together. At equal exposure, depression and anxiety, the odds ratio for a provisional diagnosis was 0.55 (95% CI 0.36 to 0.86, p = .009) against controls, close to that for the total-score cut-off, and 0.82 (95% CI 0.48 to 1.41, p = .47) against Aphantasia Network members with imagery.

The differences were in the same direction among participants with clinical-range depression or anxiety (PHQ-9 or GAD-7 of 10 or more; participants with complete covariate data), although the differences were smaller relative to their uncertainty in this subsample. The PCL-5 cut-off was reached by 42.0% of participants with congenital aphantasia (n = 88) and 60.1% of controls (n = 148; odds ratio at equal exposure, depression and anxiety 0.61, 95% CI 0.33 to 1.13, p = .12), and a provisional diagnosis by 39.8% and 58.1% (0.56, 95% CI 0.31 to 1.00, p = .049). Against Aphantasia Network members with imagery, the differences were in the same direction (42.0% against 55.6% for the cut-off; odds ratio 0.76, 95% CI 0.38 to 1.55, p = .46).

Thus, at the same level of trauma and the same depression and anxiety, people without imagery reached the screening and diagnostic thresholds for PTSD less often than controls. They reported fewer of the intrusion and avoidance symptoms that the diagnostic rule requires, while their mood and arousal symptoms were about as frequent. Against Aphantasia Network members with imagery, the difference in reaching the thresholds was not significant after adjustment for depression and anxiety.

Table 2. *DSM-5 Symptom Criteria on the PCL-5 in Each Group*

	Meeting criterion (%)			Odds ratio [95% CI]	
Criterion	Congenital aphantasia	General-population controls	Aphantasia Network members with imagery	vs controls	vs Aphantasia Network members with imagery
B, intrusion (≥ 1 of 1–5)	34.9	57.4	63.5	0.38 [0.27, 0.52]	0.36 [0.24, 0.53]
C, avoidance (≥ 1 of 6–7)	29.4	51.3	46.8	0.36 [0.25, 0.50]	0.56 [0.38, 0.84]
D, cognition and mood (≥ 2 of 8–14)	49.3	49.8	61.3	0.98 [0.71, 1.35]	0.72 [0.49, 1.07]
E, arousal (≥ 2 of 15–20)	43.4	45.4	54.5	0.94 [0.68, 1.30]	0.82 [0.55, 1.23]
D and E	33.5	37.8	44.6	0.84 [0.60, 1.18]	0.79 [0.52, 1.20]
All (provisional diagnosis)	18.8	27.6	29.3	0.60 [0.40, 0.88]	0.70 [0.44, 1.11]

Note. Numbers in parentheses are the PCL-5 items for each criterion; an item rated 2 (“moderately”) or higher counts as a symptom. Percentages are of participants with complete childhood trauma, traumatic event and PCL-5 data (congenital aphantasia n = 272, general-population control group n = 460, Aphantasia Network members with imagery n = 222). Odds ratios are for congenital aphantasia against each reference group from logistic regressions on group, childhood trauma (CTQ), traumatic events (LEC-5), age and gender. PCL-5 = PTSD Checklist for DSM-5.

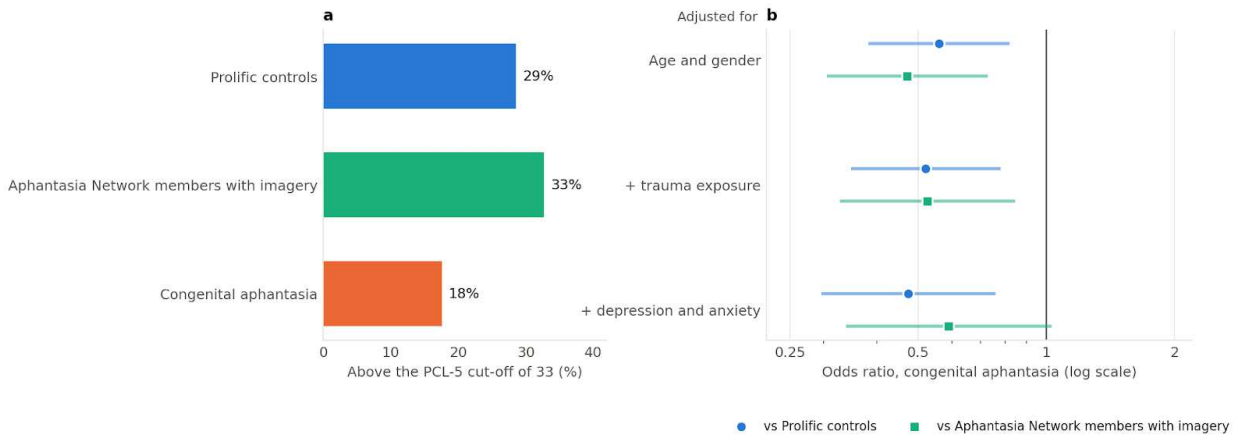


Figure 4. *Screening by the PCL-5. (a) Percentage of each group with a PCL-5 total of 33 or more. (b) Odds ratios for congenital aphantasia from logistic regressions adjusted for age and gender, then also for childhood trauma and traumatic events, then also for depression and anxiety, against the general-population control group (circles) and Aphantasia Network members with imagery (squares), with 95% confidence intervals on a log scale. Percentages and odds ratios are based on participants with complete data on all covariates (n = 262, 456 and 217).*

Involuntary memories and the group difference in intrusions

We then asked whether fewer intrusions in aphantasia were specific to trauma or part of the general reduction in involuntary memory. To test this, we added the IAMI past-memory score to the models of the PCL-5 clusters, total and cut-off. Fewer involuntary memories accounted for part, but not all, of the difference in intrusions. In participants with IAMI data, the group difference in intrusion fell from −0.56 (−0.55 in the full sample) to −0.38 SD against controls (95% CI −0.52 to −0.24, p < .001) and from −0.51 (−0.48 in the full sample) to −0.30 SD against Aphantasia Network members with imagery (95% CI −0.46 to −0.13, p < .001), and the difference in avoidance fell by a third to a half (to −0.36 SD, p < .001, and −0.16 SD, p = .088, respectively). Against controls, the difference in PCL-5 total was no longer significant (−0.04, 95% CI −0.18 to 0.10, p = .57), and the odds ratio for reaching the cut-off at equal exposure, depression and anxiety rose from 0.45 (0.47 in the full sample) to 0.64 (95% CI 0.38 to 1.07, p = .089). Involuntary memories were also less strongly associated with intrusions in aphantasia than in controls (slope 0.12 against 0.26; difference −0.14, 95% CI −0.26 to −0.03, p = .013); against Aphantasia Network members with imagery the slope was flatter in the same direction but not significantly (0.12 against 0.20, p = .15).

The adjustment did not act on intrusions alone because, against controls, adding the IAMI score also moved the estimates for negative alterations in cognition and mood (0.18, 95% CI 0.03 to 0.32) and for arousal (0.18, 95% CI 0.04 to 0.33) above zero, and against Aphantasia Network

members with imagery it moved them from slightly below zero (−0.13 and −0.19 SD) to about zero (0.03 and 0.00 SD), indicating a shift of all clusters, not a link specific to intrusions. Because the IAMI and the intrusion items both ask about memories that come to mind unbidden, adjusting one for the other also removes content they share, and because the configural fit of the IAMI was poor, the adjusted estimates should be interpreted with caution.

In summary, intrusions remained lower in aphantasia after accounting for involuntary memory in daily life, and involuntary memories were less closely tied to intrusions in people without imagery than in controls. The reduction in intrusions overlaps with, but is not fully explained by, a general reduction in involuntary memory, and the present data do not establish this as a specific mechanism.

Discussion

This study used congenital aphantasia as a natural test of the role of mental imagery in psychological trauma. People with congenital aphantasia did not differ significantly from general-population controls in trauma exposure, depression or anxiety. At equal exposure, people with aphantasia reported fewer intrusions and less avoidance, more amnesia for the traumatic event and less depersonalisation and derealisation, with no significant difference in negative mood and arousal. Overall distress, however, did not differ significantly. In people with aphantasia, trauma was associated with depression, anxiety and post-traumatic stress no less strongly than in people with imagery, and comparably except for the associations of childhood trauma with anxiety and post-traumatic stress, which tended to be stronger. In every group, its association with depression ran mainly through dysphoric symptoms, not intrusive ones. In the absence of imagery, the response to trauma thus appeared to be expressed less through re-experiencing and more through negative mood, arousal symptoms, gaps in memory for the event and numbing. As a result, at the same level of distress, individuals with aphantasia reached the screening and diagnostic thresholds for PTSD less often than controls. These findings suggest that mental imagery is related to how trauma is re-experienced, but not detectably to how much distress follows it. This challenges an assumption implicit in imagery-centred models of PTSD, that imagery is what makes trauma harmful; without imagery, the component of the response specific to PTSD was reduced, but the general distress it shares with depression and anxiety was not.

Earlier studies found that people with aphantasia report fewer intrusive memories (Dawes et al., 2020; Keogh et al., 2024; Mawtus et al., 2024), but could not tell whether this reflects less distress after trauma or the same distress expressed differently, because none examined real trauma exposure alongside symptom severity or tested whether PTSD questionnaires function equivalently without imagery. To our knowledge, the present study is the first to show that these questionnaires measure broadly equivalent constructs in people with and without imagery, that the association of trauma with distress is not detectably weaker in the absence of imagery, and that people with aphantasia are less likely than general-population controls to reach PTSD screening and diagnostic thresholds at the same level of distress.

Post-traumatic response without imagery

People with congenital aphantasia showed a selective reduction in the intrusion cluster (intrusive memories, trauma-related nightmares, flashbacks and cue-evoked reactivity) and in avoidance, which is typically secondary to re-experiencing (Ehlers & Clark, 2000). This agrees with models in which intrusions are sensory-perceptual memories triggered by cues (Brewin, 2014; Brewin et al., 2010; Ehlers & Clark, 2000), with experimental evidence that visuospatial but not verbal tasks reduce intrusions (Holmes et al., 2004, 2010), with the laboratory finding of fewer intrusions after a distressing film in aphantasia (Keogh et al., 2024), and with surveys reporting fewer intrusion symptoms on the PCL-5 (Dawes et al., 2020) and fewer intrusive sensory experiences among people with aphantasia reporting PTSD (Mawtus et al., 2024; see Northover et al., 2026 for a review). The present data show that the difference persists at equal trauma exposure, on measures that function equivalently in people with and without imagery. Negative alterations in cognition and mood, for which Dawes et al. (2020) reported inconsistent differences that mostly did not survive correction for multiple comparisons, did not differ here at equal exposure (−0.02 SD, 95% CI −0.16 to 0.12). Lower avoidance is expected if fewer memories return in sensory form, since there is less to avoid. In terms of the structure of post-traumatic symptoms, the absence of imagery was associated with fewer symptoms of the component most specific to PTSD, intrusions, but not with fewer of the mood and arousal symptoms that make up its non-specific dysphoria component (Bryant et al., 2011; Simms et al., 2002).

Two observations could suggest that the lower intrusion scores reflect measurement rather than symptoms. First, two PCL-5 items were answered differently by people with aphantasia at the same level of post-traumatic stress (reliving the experience, item 3, was endorsed less, and trouble remembering it, item 8, more). The intrusion difference did not depend on these items, however. All five intrusion items were lower, removing item 8 did not reduce the difference in PCL-5 total, and removing items 3 and 8 left the screening result essentially unchanged. Second, against the general-population control group, the item-level screen flagged all intrusion and avoidance items. This arose because the screen compares each item with the other items on its factor, and in the exploratory solution the intrusion and avoidance items shared a factor with mood items that did not differ between groups; a genuine difference in intrusions therefore appeared as item bias. When intrusion, avoidance, mood and arousal were modelled as separate factors, following the DSM-5 clusters, the PCL-5 met scalar invariance, indicating that the items measured each cluster equivalently, so that the lower intrusion scores can be read as fewer symptoms rather than as item bias, although the fit of this model was only moderate (RMSEA 0.080).

The one post-traumatic symptom that was more frequent in aphantasia was amnesia for parts of the event. Most of this difference appeared to reflect a general pattern of autobiographical memory: adding gaps for important life events in general reduced it from 0.40 to 0.14 SD against controls, which was no longer statistically significant. This is consistent with the account of autobiographical memory in aphantasia as knowledge of past events rather than re-experience of them (Dawes et al., 2020; Milton et al., 2021), and with earlier observations that the trauma-

amnesia item behaves inconsistently in factor analyses of PTSD symptoms (King et al., 1998; Simms et al., 2002). The pattern also fits the distinction drawn by dual representation theory between involuntary perceptual memory and voluntary episodic memory for trauma, which are proposed to be functionally independent, so that strong involuntary images can coexist with gaps in voluntary recall (Brewin, 2014). In people without imagery, the perceptual component was reduced while gaps in episodic recall were more frequent, suggesting that the two components can be dissociated in a natural population. Trauma amnesia was nevertheless associated with distress in the aphantasia group. As in the general-population control group, but not in Aphantasia Network members with imagery, people with congenital aphantasia who remembered less of the traumatic event reported more depression and numbing. This association remained when general gaps in autobiographical memory were taken into account. Because people with aphantasia scored higher on the trauma-amnesia item, this item raised their PCL-5 totals and partly offset their lower scores on other symptoms. The total therefore understates how different their post-traumatic response was; at equal exposure, the total differed from controls by only about a quarter of a standard deviation (−0.26 SD), and by slightly more when item 8 was removed (−0.29 SD), whereas intrusion and avoidance each differed by more than half a standard deviation (−0.55 and −0.54 SD). A total score thus combines lower re-experiencing with similar mood and arousal symptoms and higher trauma amnesia, and in doing so it conceals the pattern that distinguishes the post-traumatic response in aphantasia. Cluster-level scores, not totals, are therefore needed to characterise post-traumatic stress in people without imagery.

The burden of trauma in aphantasia

We considered three accounts of how the absence of imagery might affect the harm that follows trauma: that trauma is less damaging without imagery (protective), equally damaging but expressed through other symptoms (same burden), or more damaging because re-experiencing contributes to its processing (hidden cost; Brewin, 2014; Brewin, Dalgleish, & Joseph, 1996). These predict, respectively, a weaker, equal or stronger association between trauma exposure and distress in aphantasia. Previous studies could not distinguish these accounts because they did not measure trauma exposure. Here, no slope differed significantly between groups, and equivalence tests supported the same-burden account for depression and for traumatic events, and ruled out a protective effect larger than 0.20 SD in every comparison. For anxiety and PCL-5 total on childhood trauma, however, the slopes were somewhat steeper in aphantasia than in controls and could not be shown to be equivalent, so a moderate hidden cost for these outcomes cannot be excluded.

The mediation analysis bears on the question of why fewer re-experiencing symptoms were not accompanied by less distress. We had expected the same burden to be reached by different routes, through memory gaps and numbing in aphantasia and through intrusions in people with imagery. Instead, the route was similar in every group: the association of childhood trauma with depression ran mainly through trauma amnesia, numbing and concentration problems, and little through intrusions, even in people with imagery. These symptoms correspond to the dysphoria component of post-traumatic stress, which is closely related to depression and reflects the general

distress shared across anxiety and depressive disorders (Clark & Watson, 1991; Simms et al., 2002). Our exploratory factor analysis showed the same overlap, with the depression and anxiety items loading together with the mood and arousal items of the PCL-5. Intrusive memories are also common in depression (Reynolds & Brewin, 1998), yet they carried little of the association of trauma with depression in any group, except in Aphantasia Network members with imagery when the numbing items were left out, where their share was similar to that of trauma amnesia and concentration problems. Without imagery, only the PTSD-specific component of the response was lower, not the general distress shared with depression, which may explain why trauma was associated with depression about as strongly as in people with imagery. The data are cross-sectional, however, so they show how strongly trauma is associated with current distress, not whether trauma that is not re-experienced is processed differently over time. In dual representation theory, attending to flashbacks allows sensory memories of the trauma to be integrated into episodic memory, after which they lose their sense of current threat (Brewin, 2014; Brewin, Dalgleish, & Joseph, 1996). If re-experiencing serves this function, its absence could affect the long-term course of distress, so a hidden cost for long-term outcomes remains possible.

Dissociation: lower depersonalisation without imagery, and a similar increase with distress

People with congenital aphantasia reported less depersonalisation and derealisation than people with imagery. None of the four DES items answered differently in aphantasia (which refer to vivid reliving, absorption in fantasy or film, and gaps in memory) belongs to the depersonalisation and derealisation subscale, so the difference does not depend on items whose content presupposes imagery. Depersonalisation and derealisation in people with aphantasia nevertheless increased with depression, anxiety and post-traumatic stress at a rate that did not differ significantly from people with imagery, although against Aphantasia Network members with imagery the slope on traumatic events was flatter. People with aphantasia thus started from a lower level, with little evidence that they responded less to distress. Invariance tests, however, detect items that function differently between groups, but not a uniform shift across every item of a scale, which appears instead as a lower latent mean. We therefore cannot exclude that part of the difference reflects how people without imagery read the remaining depersonalisation items, for example as describing experiences of imagery rather than of the self.

Lower dissociation in congenital aphantasia is consistent with the interoceptive account of imagery, in which imagery and affect share a substrate in the anterior insula, and in which the dissociative presentation of PTSD involves reduced insular engagement (Kvamme, Nagai, & Silvanto, 2026). It contrasts with depersonalisation disorder, in which poorer imagery accompanies more severe depersonalisation (Lambert et al., 2001). In the present groups with imagery, weaker imagery was associated with slightly more depersonalisation only in controls and only before adjustment for depression and anxiety, consistent with the link between impaired imagery and low mood reported by Lambert et al. (2001). Impaired imagery in depersonalisation disorder may therefore be a feature of a detached and depressed state, whereas congenital aphantasia is a lifelong absence of imagery and was associated with less depersonalisation. The combination of more trauma amnesia with less depersonalisation is also notable, because fragmented memory for trauma is usually associated with greater dissociation (Brewin, 2014). In

aphantasia, gaps in memory for the event appear to reflect a general style of autobiographical memory rather than a dissociative response.

Implications for imagery-based models of PTSD and for accounts of aphantasia

If aphantasia were simply a lack of voluntary imagery, involuntary flashbacks should be spared and intrusions should be as common as in people with imagery. Cavedon-Taylor (2022) favoured this volitional account, partly because people with aphantasia did not differ from controls in PCL-5 total (Dawes et al., 2020). The present data do not support this view. Intrusions, trauma-related nightmares and involuntary memories in daily life were all less frequent, in agreement with the view that aphantasia also affects involuntary imagery (Krempel & Monzel, 2024). Intrusions were reduced, not absent, and about a third of people with aphantasia met the intrusion criterion; in the laboratory their intrusions were mostly verbal (Keogh et al., 2024), consistent with evidence that intrusive images and involuntary verbal thoughts are distinct (Brewin, 2014; Speckens et al., 2007). The alternative, that aphantasia is an absence of imagery of any kind, was taken to predict both fewer intrusions and less psychopathology (Cavedon-Taylor, 2022). Intrusions were fewer, but depression and anxiety were as common as in people with imagery, and no less closely related to trauma. Neither account predicts this combination. The data favour the view that aphantasia involves an absence of involuntary as well as voluntary imagery, but not the assumption, shared by both accounts, that imagery is what makes trauma harmful. Post-traumatic symptoms comprise two components (Brewin, 2014; Bryant et al., 2011; Simms et al., 2002): intrusions, which are specific to PTSD and were lower without imagery, and general distress, which is shared with depression and anxiety and was not detectably related to imagery.

The reduction in intrusions also bears on the question of whether individuals with aphantasia have unconscious imagery. On one account, aphantasia resembles blindsight: the sensory representations of imagery are formed but do not reach awareness (Michel et al., 2025; Nanay, 2021). If trauma memories were reactivated in this form, they could drive distress without being experienced as flashbacks, and this would account for the similar burden of trauma found here. The integration model predicts the opposite (Scholz et al., 2026). It holds that the activity recorded in the visual cortex of people with aphantasia consists of sensory precursors, fragmentary codes that are not integrated into perceptual representations and therefore cannot act as imagery, consciously or unconsciously. Two findings favour the second account. People with aphantasia show no rise in skin conductance when they read and imagine frightening stories, although they respond normally when they view frightening pictures (Wicken et al., 2021). In the present data, involuntary memories of the past and involuntary thoughts about the future were both less frequent, not only intrusions. The distress associated with trauma may therefore have been carried by routes other than imagery, such as negative appraisal, rumination and loss of interest, which the PCL-5 assesses in its cognition and mood cluster.

Empathy shows a similar pattern. Self-reported empathy is lower in aphantasia when it is measured with verbal questionnaires and vignettes, but not when it is measured with pictures (Monzel, Keidel, & Reuter, 2023). In both cases the emotional response to what is seen is intact, and what is missing is the response to what must be imagined. A traumatic event is first perceived

and only later re-experienced, which may explain why people with aphantasia were not detectably less distressed by trauma but had fewer intrusions. The questionnaires used here are also verbal, and items that ask about reliving or remembering were answered differently in aphantasia. The trauma film paradigm instead presents the event visually and records intrusions as they occur, yet it also found fewer intrusions in aphantasia (Keogh et al., 2024). The present data cannot exclude unconscious reactivation. A direct test would combine the trauma film paradigm with a physiological measure, such as skin conductance, during later cued recall of the film.

A similar dissociation has been reported after traumatic brain injury, where the memory of the event, rather than imagery, is lost. In a prospective study of mild brain injury, PTSD developed in 23% of patients who remembered the event but in 6% of those who did not, and the difference lay in the re-experiencing cluster, whereas avoidance and hyperarousal did not differ (Gil et al., 2005). After road traffic accidents, PTSD, anxiety and depression a year later were as common in people who had been unconscious as in those who had not, and the intrusive memories of those who had been unconscious usually concerned events just before or after the accident (Mayou et al., 2000). After moderate to severe brain injury, intrusive memories were common among patients with PTSD despite amnesia for the event, possibly through memories reconstructed afterwards or through implicit memory (Alway et al., 2016). Losing access to the trauma memory, like lacking imagery, therefore appears to reduce re-experiencing more than the other symptoms. Rubin et al. (2008) argued from such findings that PTSD depends on the memory of the trauma rather than on the event itself, and showed that the symptom of being unable to recall an important aspect of the trauma fits poorly with the other symptoms in factor analyses. In people with aphantasia, by contrast, trauma amnesia was associated with depression and numbing.

Clinical implications

The present results have clinical implications in relation to diagnosis. PTSD screening and diagnosis require intrusive re-experiencing, and at the same level of trauma, depression and anxiety, people with aphantasia had about half the odds of general-population controls of reaching the PCL-5 cut-off or meeting the DSM-5 symptom rule. This did not reflect lower distress; they reported fewer of the intrusion and avoidance symptoms, while meeting the mood and arousal criteria as often as controls. This is consistent with reports that PTSD in people with aphantasia is often missed or misdiagnosed (Mawtus et al., 2024). A low PTSD checklist score in a person without imagery should therefore not be taken as evidence that trauma has had little effect, and clinicians may need to ask about imagery ability and give more weight to mood, arousal and memory symptoms when assessing such patients.

Several treatments for PTSD are thought to work by changing how the trauma memory is re-experienced. Psychotherapy assisted by 3,4-methylenedioxymethamphetamine (MDMA), for example, has been proposed to act by reducing the accessibility and emotional intensity of the traumatic memory when it is reactivated in therapy (Sarmanlu et al., 2024), and people with aphantasia rate therapies that rely on visual imagery as less effective than people with imagery do (Mawtus et al., 2024). The present results suggest that treatments targeting re-experiencing would mainly reduce intrusions, and that general distress may need to be treated separately.

Imagery ability could therefore be measured in clinical trials, alongside the measures of memory vividness and accessibility proposed for trials of MDMA-assisted psychotherapy (Sarmanlu et al., 2024).

Limitations

The main limitation of the present study is that all measures were self-reports collected at a single time point. The PCL-5 was answered for a self-chosen worst event, which may differ between groups, and trauma exposure was reported retrospectively, so people with aphantasia, who report more gaps in autobiographical memory, may have under-reported events, although their similar exposure scores make a large under-report unlikely. Furthermore, the study contained no measure of functional impairment or clinical interview, so we cannot determine whether people with aphantasia who fall below the PTSD thresholds need clinical help as often as those with imagery who reach them. In addition, equivalence was tested for the slopes of distress on exposure but not for the group differences in the level of depression and anxiety, so the conclusion that the level of distress was similar rests on non-significant differences. Finally, autistic and attention-deficit traits, which are more common in aphantasia (Kvamme, Nagai, & Silvanto, 2026), were not measured and may affect how symptoms are reported.

Conclusion

Using congenital aphantasia as a natural test, we investigated the link between mental imagery and psychological trauma. People without imagery did not differ significantly from controls in trauma exposure, anxiety or depression, and trauma was associated with their distress no less strongly than in people with imagery. Their post-traumatic response, however, took a different form, with fewer intrusions and less avoidance, more amnesia for the event, and less depersonalisation. Imagery therefore appears to be related to how trauma is re-experienced, but not detectably to how much distress follows it. People with aphantasia also reached the screening and diagnostic thresholds for PTSD less often than controls at the same level of distress, and a low score on a PTSD checklist should not be taken as evidence that trauma has had little effect in a person without imagery.

Declarations

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Ethics

The study was approved by the University of Macau Ethics Committee (HE-0608-2025). All participants provided informed consent prior to participation.

Competing interests

The authors declare no competing interests**.**

Data and code availability

The analysis code that reproduces every result reported here is available at https://github.com/timokvamme/aphantasia-trauma-ptsd and archived on Zenodo (https://doi.org/10.5281/zenodo.23375587). Because participants were informed that their responses would be reported only in the aggregate, the participant-level data are not publicly available; the anonymised data are available from the authors on reasonable request.

Author contributions

C.J.S.C.: Conceptualization, Methodology, Investigation, Data curation, Formal analysis, Visualization, Project administration, Writing – original draft, Writing – review & editing. T.L.K.: Methodology, Software, Formal analysis, Visualization, Writing – original draft, Writing – review & editing. J.S.: Conceptualization, Methodology, Supervision, Funding acquisition, Writing – original draft, Writing – review & editing.

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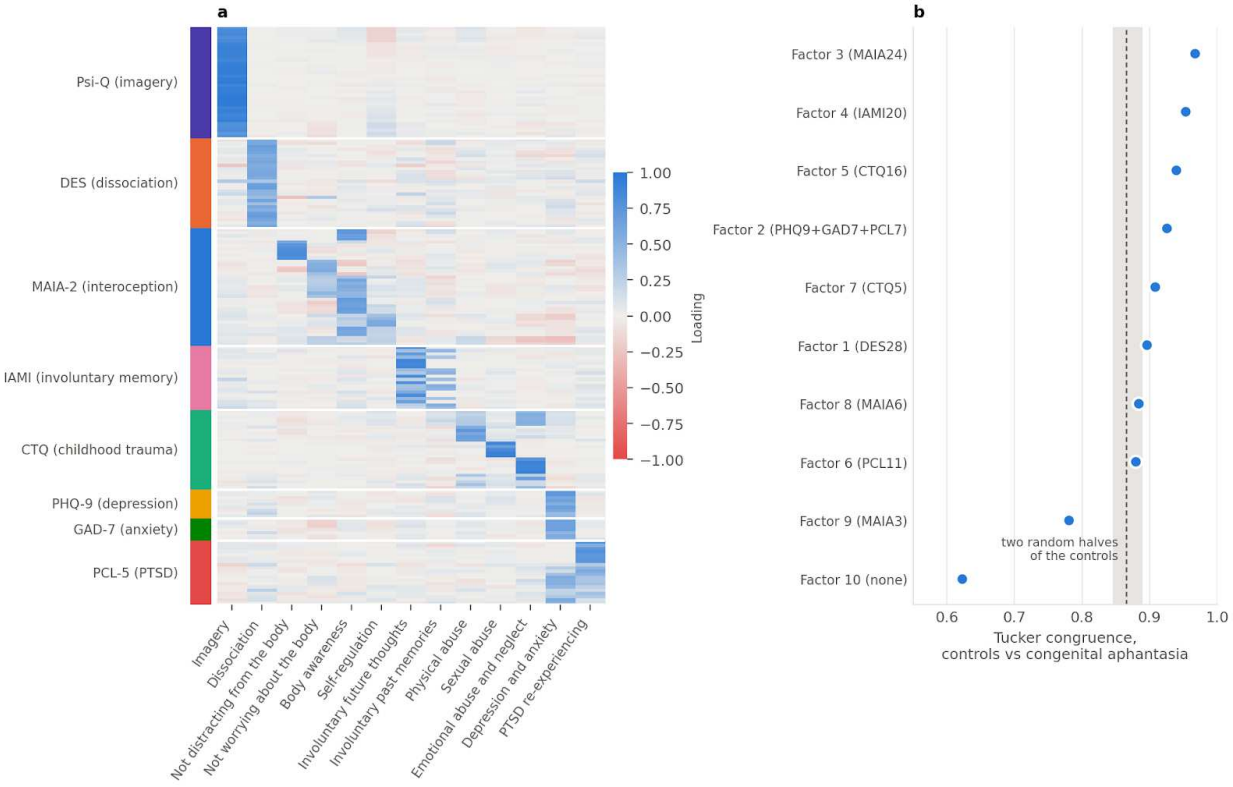
Supplementary material

Supplementary Table S1. *Internal Consistency (Cronbach's α) of Each Scale in Each Group*

Scale (items)	Congenital aphantasia	General-population controls	Aphantasia Network members with imagery
Psi-Q, all items (35)	0.96	0.96	0.96
Psi-Q, visual subscale (5)	0.89	0.83	0.99
CTQ total (25)	0.93	0.94	0.94
Emotional abuse (5)	0.91	0.94	0.91
Physical abuse (5)	0.81	0.89	0.87
Sexual abuse (5)	0.94	0.95	0.93
Emotional neglect (5)	0.94	0.95	0.93
Physical neglect (5)	0.73	0.77	0.74
PHQ-9 (9)	0.88	0.90	0.87
GAD-7 (7)	0.91	0.93	0.90
PCL-5 total (20)	0.94	0.95	0.93
Intrusion (5)	0.84	0.92	0.89
Avoidance (2)	0.86	0.90	0.86
Negative alterations in cognition and mood (7)	0.89	0.89	0.86
Arousal and reactivity (6)	0.81	0.83	0.79
DES amnesia (6)	0.80	0.93	0.81
DES depersonalisation and derealisation (6)	0.77	0.94	0.87
DES absorption (6)	0.81	0.89	0.84
IAMI past memories (10)	0.93	0.90	0.94
IAMI future thoughts (10)	0.94	0.94	0.96

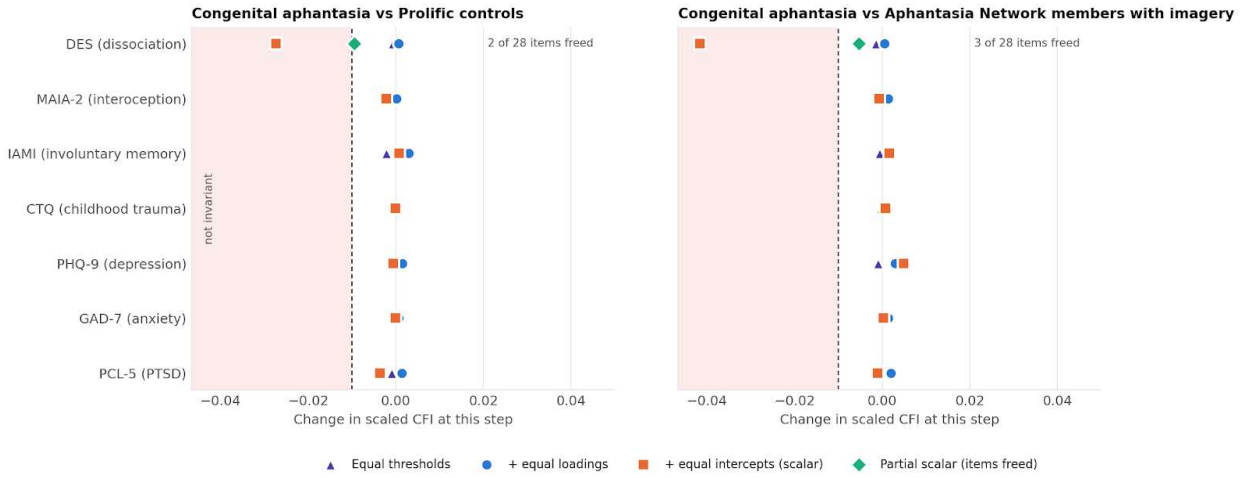
Note. Values are Cronbach's α among participants with complete data on each scale. The DES absorption subscale excludes items 14, 17 and 18, which were answered differently in aphantasia, as in the analyses. Psi-Q values in the congenital aphantasia group are computed on a restricted range near the floor of the scale. Psi-Q = Plymouth Sensory Imagery Questionnaire; CTQ = Childhood Trauma Questionnaire short form; PHQ-9 = Patient Health

Questionnaire-9; GAD-7 = Generalized Anxiety Disorder-7; PCL-5 = PTSD Checklist for DSM-5; DES = Dissociative Experiences Scale; IAMI = Involuntary Autobiographical Memory Inventory.



Supplementary Figure S1. *Factor structure of the questionnaire items. (a) Pattern loadings of all 181 items on the 13 factors of the pooled exploratory factor analysis (Spearman correlations, minimum residual extraction, oblimin rotation, number of factors by parallel analysis), with rows grouped by questionnaire and columns labelled by the content of the items loading on each factor. Most items loaded on factors corresponding to their own questionnaire or subscale; the depression and anxiety items loaded on a single factor together with the mood and arousal items of the PCL-5. (b) Tucker congruence of each factor between the general-population control group and congenital aphantasia, from the 10-factor solution (the smaller number suggested by parallel analysis in either group) fitted separately in each group on the 146 items other than the Psi-Q. Labels give the questionnaires and number of items loading on each factor (|loading| ≥ 0.40); Factor 10 had no items above this threshold. The dashed line and grey band show the mean and 5th to 95th percentile of the mean congruence across 50 random splits of the controls into a subsample of the size of the aphantasia group and the remainder, so the band should be compared with the mean congruence between groups (0.875), not with individual factors. Panel*

a uses the 1,148 participants with complete data on all items, and panel b the 454 controls and 246 people with congenital aphantasia among them.



Supplementary Figure S2. *Measurement invariance of each questionnaire in congenital aphantasia, compared with the general-population control group (left) and Aphantasia Network members with imagery (right). Each point is the change in scaled CFI at one step of a multi-group ordinal confirmatory factor model (WLSMV, identified as in Wu & Estabrook, 2016) with the published factor structure: from the configural model to equal thresholds (triangles), then to equal loadings (metric; circles), then to equal intercepts (scalar; squares), and, where scalar invariance failed, from the metric model to a partial scalar model after the thresholds and intercepts of items were freed in order of their score-test statistics (diamonds). The PCL-5 was modelled with the four DSM-5 symptom clusters as factors. The dashed line and shaded region mark a fall in CFI larger than 0.010, the criterion for rejecting invariance (Chen, 2007); invariance also required a rise in RMSEA of no more than 0.015 (not shown). The DES reached partial scalar invariance after two items were freed against controls (items 14 and 9) and three against Aphantasia Network members with imagery (items 14, 9 and 18). Each model used participants with complete data on that questionnaire.*

Supplementary Table S2. *Items Answered Differently by People With Congenital Aphantasia at the Same Level of Their Factor*

Item	Wording (abbreviated)	Against general-population controls	Against Aphantasia Network members with imagery
DES 14	Remembering a past event so vividly that one feels as if reliving it	−0.98	−0.77
DES 18	So involved in a fantasy or daydream that it feels as though it were really happening	−0.66	−0.75
MAIA-2 21	Maintaining awareness of the whole body when a part is in pain or discomfort	−0.50	−0.34
PCL-5 3	Feeling or acting as if the stressful experience were actually happening again	−0.44	−0.36
DES 17	So absorbed in a film or television story as to be unaware of other events	−0.37	−0.31
PCL-5 8	Trouble remembering important parts of the stressful experience	+0.43	+0.38
DES 9	No memory for some important events in	+0.91	+0.54

	one’s life		

Note. Cells are the group coefficient from a regression of the rank-transformed item on the mean of the other items of its factor, group, age and gender, in SD units; negative values mean lower endorsement in aphantasia. Items were classed as biased when the group coefficient reached 0.25 SD, survived Benjamini–Hochberg correction and had the same sign against both reference groups. DES = Dissociative Experiences Scale; MAIA-2 = Multidimensional Assessment of Interoceptive Awareness, Version 2; PCL-5 = PTSD Checklist for DSM-5.