

1 Home assessment of visual working memory in pre-
2 schoolers reveals associations between behaviour, brain
3 activation and parent reports of life stress.

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16

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33 **Research highlights**

- 34 • Home-assessments of brain activation and behavioural performance on a
35 visual working memory (VWM) task.
- 36 • Low-performing children showed increasing activation with increasing load
37 across the left frontal and bilateral parietal cortices.
- 38 • High-performing children showed no modulation of activation with increasing
39 load across the left frontal and bilateral parietal cortices.
- 40 • The difference in activation between the highest and the lowest load in the left
41 parietal cortex partially mediated the association between parental life stress
42 and VWM performance.

43

44 **Abstract**

45 Visual working memory (VWM) is reliably predictive of fluid intelligence and academic
46 achievements. The objective of the current study was to investigate individual
47 differences in pre-schoolers' VWM processing by examining the association between
48 behaviour, brain function and parent-reported measures related to the child's
49 environment. We used a portable functional near-infrared spectroscopy system to
50 record from the frontal and parietal cortices of 4.5-year-old children ($N=74$) as they
51 completed a colour change-detection VWM task in their homes. Parents were asked
52 to fill in questionnaires on temperament, academic aspirations, home environment,
53 and life stress. Children were median-split into a low-performing (LP) and a high-
54 performing (HP) group based on the number of items they could successfully
55 remember during the task. LPs increasingly activated channels in the left frontal and
56 bilateral parietal cortices with increasing load, whereas HPs showed no difference in
57 activation across the loads. Our findings suggest that LPs recruited more neural
58 resources than HPs when their VWM capacity was challenged. We employed
59 mediation analyses to examine the association between the difference in activation
60 between the highest and lowest loads and variables from the questionnaires. The
61 difference in activation across loads in the left parietal cortex partially mediated the
62 association between parent-reported stressful life events and VWM performance.
63 Critically, our findings show that the association between VWM capacity, left parietal
64 activation, and indicators of life stress is important to understand the nature of
65 individual differences in VWM in pre-school children.

66

67

68 **Keywords:**

69 Visual Working Memory; Home testing; fNIRS; Individual differences; Life stress; Pre-
70 school children.

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72

73 **Introduction**

74 Visual working memory (VWM) is a short-term storage system responsible for
75 detecting changes in the world as they occur. VWM capacity is reliably predictive of
76 cognitive functions (Fukuda et al., 2010) and a host of academic skills (Alloway &
77 Alloway, 2010; R. Bull & Scerif, 2001; Gathercole et al., 2004; Swanson & Beebe-
78 Frankenberger, 2004). Critically, it accounts for around 40% of individual differences
79 in global fluid intelligence (Fukuda et al., 2010), and for up to 46% of individual
80 differences in performance on a cognitive battery of tasks (Johnson et al., 2013).

81 A common task for assessing VWM is the change-detection task (Luck & Vogel,
82 1997), where subjects are consecutively presented two arrays of items and asked to
83 identify if the items were identical across both arrays. This task is particularly well-
84 suited for examining human development, as age-related changes in capacity can be
85 captured by varying the number of presented items. Using this task, Simmering (2012)
86 found that three-year-olds had a capacity of 1.5 to 2 items. Capacity increased to 2 to
87 3 items by five years of age. This finding was confirmed by Buss and colleagues
88 (2014), who found that three-year-old children had a capacity of 1.2 items while four-
89 year-old children had a capacity of 1.8 items when attending to a shape change
90 detection task.

91 Consistent behavioural findings from manipulating VWM load have been
92 accompanied by the involvement of a distributed brain network in adults. VWM
93 capacity is associated with activity in the posterior parietal and superior occipital
94 cortices, with activation increasing as the number of items in the array increases, and
95 reaching a plateau at maximum capacity (Todd & Marois, 2004). Activity in the
96 posterior parietal cortex has also been shown to predict individual differences in
97 capacity in adults (Todd & Marois, 2005). Further, load-dependent responses have
98 been found in the right intra-parietal sulcus and the right superior intraparietal sulcus
99 (Ambrose et al., 2016). Koenigs and colleagues (2009) assessed a wide range of
100 memory functions in patients with lesions in either hemisphere of the superior parietal
101 cortex and found that this area was directly involved in the manipulation and re-
102 arrangement of information for both auditory-verbal and visuospatial stimuli.
103 Separately, another body of research has shown that the prefrontal cortex is
104 responsible for maintaining and encoding representations of task-relevant information

105 into working memory (Baddeley, 2003; Miller & Cohen, 2001). More recently,
106 Christophel and colleagues (2017) proposed that working memory relies on the
107 interplay between sensory regions in the posterior cortex that retain low-level features
108 and frontal regions that retain more abstract stimuli that are gradually transformed to
109 an appropriate behavioural response. Taken together, these findings suggest that
110 working memory emerges from a coordinated system involving a distributed network
111 of brain regions across the posterior and anterior cortices.

112 While fMRI studies have provided valuable insights into the neural processes
113 underlying VWM in adults, there are limitations in its application in early development.
114 fMRI scanners are noisy, and participants are required to lie still, an obstacle for young
115 children. More recently, early developmental work has employed an alternative
116 technique - functional near-infrared spectroscopy (fNIRS). fNIRS systems shine near-
117 infrared light (ranging from 650nm-1000nm) through the head to detect changes in
118 oxygenated haemoglobin [HbO] and deo-oxygenated haemoglobin [HbR] (Boas et al.,
119 2014). fNIRS has been reliably used across the lifespan to study changes in activation
120 in the frontal and parietal networks underlying VWM (Buss et al., 2014; Sato et al.,
121 2013; Tsujimoto et al., 2004; Wijekumar et al., 2019; Wijekumar, Huppert, et al.,
122 2017; Wijekumar, Magnotta, et al., 2017). Using fNIRS, Tsujimoto and colleagues
123 (2004) examined VWM performance on a change-detection task in a sample of adults
124 and five-year-old children. They found that activity in the bilateral areas of the
125 prefrontal cortex was similar in both groups, characterised by an increase in HbO with
126 the onset of the memory array. These results demonstrated for the first time that the
127 left prefrontal cortex is also involved in VWM processing in pre-schoolers. An fNIRS
128 study examining VWM using a shape change-detection task in three- and four-year-
129 old children found more robust activation in the parietal cortex in four-year-olds relative
130 to three-year-olds (Buss et al., 2014). However, in contrast to what has been
131 consistently found in the adult literature, the hemodynamic response did not plateau
132 at maximum capacity – suggesting this neural effect may develop only after four years
133 of age. Wijekumar and colleagues used a portable fNIRS system to investigate the
134 neural networks underlying VWM processing in infants and children in rural India. They
135 found that VWM performance was inversely correlated with activation in the bilateral
136 frontal cortices, such that increased activation in this area was associated with poorer
137 VWM performance (Wijekumar et al., 2019). This study highlights another crucial

138 advantage of fNIRS over other neuroimaging modalities - its portability, allowing
139 researchers to freely explore cognitive processes outside of a traditional lab setting.

140 Individual differences in the development of VWM are predictive of subsequent
141 academic achievements such as math abilities (Bull, Espy, & Wiebe, 2008) and
142 reading comprehension (Swanson & Berninger, 1996). These individual differences
143 might stem from exposure to specific parental and home environmental factors.
144 Fishbein and colleagues showed that cognitive function in children was associated
145 with certain home child-rearing conditions (Fishbein et al., 2019). Specifically, positive
146 aspects of the home environment predicted better performance on two tasks
147 assessing visual information processing and working memory. Parental/home
148 measures might also be associated with brain structure and brain function. Higher
149 family income has been linked to greater white matter integrity in cortical and
150 subcortical brain areas (Noble et al., 2015; Ursache et al., 2016). Early childhood
151 deprivation is also associated with prefrontal cortical volume and surface area
152 (Mackes et al., 2020). In rural settings in India, weaker activation in parts of the frontal
153 cortex in response to a preferential looking VWM task in children was linked to poorer
154 maternal education and income (Wijeakumar et al., 2019). It is possible that such
155 associations between a specific brain structure or function and parent/home measures
156 could inform effects on behavioural performance and/or other cognitive functions
157 subserved by the same areas in later development. However, the nature of trivariate
158 associations between behavioural performance, brain function and parental
159 and home environment measures are not well-understood. In the current study, we
160 probe this association by investigating how individual differences in VWM performance
161 and underlying brain function are related to parental and home environment factors.
162 We recruited a specific group of pre-schoolers with little variation in age to investigate
163 individual differences during the critical period before children begin formal education.
164 The present study capitalised on the portability of the fNIRS system and, to the best
165 of our knowledge, is the first to examine individual differences in VWM processing
166 using home-based testing. Laboratory testing allows for experiments to be conducted
167 under strictly controlled conditions. However, in doing so, children are tested in an
168 artificial and stressful environment. By conducting all testing in a home environment,
169 we hope to provide children with the opportunity to perform the task in a relaxed
170 environment and increase the ecological validity of the paradigm.

171 Based on findings by Simmering (2012), we hypothesized that overall, VWM
172 performance would decrease as load increases. Our predictions of brain activation
173 patterns in low and high-performing children are informed by contrasting accounts
174 from previous findings. First, in general, challenging task demands through increasing
175 VWM load elicits increasing brain activation in adults until capacity limit is severely
176 strained (Linden et al., 2003; Rypma & D’Esposito, 1999; Todd & Marois, 2004, 2005).
177 Second, along the same vein, Jaeggi et al. (2007) showed that low-performing adults
178 elicited greater load-dependent activation than high-performing adults in parts of the
179 frontal cortex. Here, the authors argued that low-performers might recruit additional
180 attentional and strategy-related mechanisms during difficult conditions that might even
181 be detrimental to their performance, whereas, high-performers were able to
182 demonstrate efficient processing, stabilize their resources and improve performance.
183 Third, in contrast to these two previous set of findings, Buss et al. (2014) showed that
184 four-year-old children showed better performance and greater activation in the parietal
185 cortex compared to three-year-old children suggesting that behavioural gains were
186 associated with greater, and not lesser activation. However, Buss and colleagues
187 investigated *developmental* changes and not *performance-related* changes, affording
188 the question whether with a larger sample of four-year-old children and testing
189 performance differences, they might have observed similar findings to that of Jaeggi
190 et al. (2007). Further, they did not formally test the association between behavioural
191 performance and brain activation. Lastly, Wijekumar and colleagues found that
192 greater activation in parts of the frontal cortex was associated with poorer VWM
193 performance in a preferential looking task where a changing and non-changing
194 flashing display of items were presented side by side (Wijekumar et al., 2019). They
195 suggested that the inability to suppress distraction from the non-changing side elicited
196 greater activation in children with poor VWM performance. Taking these accounts into
197 consideration, we predicted that low-performing children from our sample would
198 require greater cognitive effort to meet the challenging VWM loads of the task, and as
199 a result, would elicit more activation than high-performing children.

200

201 **Methods**

202 *Participants:*

203 Ninety-five 4.5-year-olds (45 females, *Age* = 53.5 months, *SD* = 1.2) participated in
204 the study. Additionally, one of the parents of each child also took part in the study by
205 filling out a series of questionnaires. We recruited participants by contacting gateway
206 organizations such as nurseries and leisure centres and providing them with our study
207 information. Parents of eligible children got in touch with us via our website, email, or
208 over the phone to schedule a testing session. Data were collected on participants in
209 their homes across Scotland. Children received a small honorarium and gift for
210 participation. All participants had normal or corrected to normal vision, no history of
211 colour-blindness, no neurological conditions, and all mothers experienced a full-term
212 pregnancy (37 to 42 weeks) with an uncomplicated birth. Parents gave written
213 informed consent and children gave verbal assent prior to testing. The research was
214 approved by the General University Ethics Panel (GUEP 375) at the University of
215 Stirling.

216 Data from twenty-one children had to be excluded from analyses; five children
217 refused to participate in the task, twelve children interfered with the neuroimaging set-
218 up (pulled the cap off) before the completion of the task, two children had thick hair
219 that prevented contact between the optodes and the scalp and caused poor signal
220 quality, and data from two children was lost due to experimenter error. A total of 74
221 children (37 females, *Age* = 53.5 months, *SD* = 1.3) contributed to the final analyses.

222 *Experimental task:*

223 The colour change-detection task (Simmering, 2012) was used to measure VWM
224 performance in children. The task was explained using 3x3 inch flashcards with
225 coloured squares, to ensure children understood the rules. The experimenter placed
226 the first card (with one coloured square) on the table for approximately 2 seconds and
227 asked the child to remember the card. Then, the experimenter turned over the first
228 card and placed a second card (with one square of the same or different colour) on
229 top. The child was asked if the two cards were the same or different. Once the child
230 responded, the experimenter turned over both cards and praised the child if they had
231 correctly answered the question and corrected them if they had given the wrong
232 answer. This practice session was repeated with flashcards containing two and then,
233 three coloured squares. The flashcards were displayed again if the child made a
234 mistake. Once the child had correctly answered all the practice trials, the experimental

235 task was run in E-prime V.3 software on an HP laptop with a 14-inch screen. The
236 computer task began with three practice trials, the first trial had one square, the second
237 trial had two squares, and the third trial had three squares. Children were corrected if
238 they made a mistake before commencing the experimental trials. Each trial of the
239 experimental task began with a memory array of coloured squares presented for 2
240 seconds, followed by a delay of 1 second, and finally, by the test array of coloured
241 squares (see Fig. 1). The test array remained on the screen until a response was
242 made. During 'same' trials, the colours in both arrays were identical. During 'different'
243 trials, the colour of one square in the test array was different from the otherwise
244 identical memory array. At the end of each trial, the experimenter asked the child if the
245 two cards were the same or different. Children gave a verbal response, which the
246 experimenter recorded on the laptop. An inter-trial interval of 1 second (50% of the
247 trials), 3s (25% of the trials), or 5s (25% of the trials) was used at the end of each trial.
248 During every trial, the memory and test arrays were presented one after another,
249 occupying the same position on the screen. Across trials, arrays were presented on
250 alternating sides of the screen to avoid confusion in children who tried to compare the
251 test array in trial 1 with the sample array in trial 2. VWM load was manipulated from 1
252 to 3 square items (load 1, load 2, and load 3). Each load was presented in a block
253 consisting of randomised presentations of 8 same and 8 different trials.

254

255 *fNIRS data acquisition:*

256 fNIRS data were collected at 7.81 Hz using a NIRSport system 8x8 (8 sources 8
257 detectors) / release 2.01 with wavelengths of 850 and 760 nm. Fiber optic cables
258 carried light from the machine to a NIRS cap. Probe geometry was designed by
259 collating regions of interest (ROI) from previous fMRI VWM literature (Wijeakumar et
260 al., 2015) . Probe geometry consisted of four channels each on the left and right frontal
261 cortices, and three channels each on the left and right parietal cortices (see Fig. 2).
262 Note that short-source-detector channels were not used to regress scalp
263 hemodynamics as all the channels were directed toward maximising coverage of the
264 frontal and parietal cortices. Four cap sizes (50cm, 52cm, 54cm, and 56cm) were used
265 to accommodate different head sizes. Source-detector separation was scaled
266 according to cap size (50cm cap: 2.5cm; 52cm cap: 2.6cm; 54cm cap: 2.7cm and

267 56cm cap: 2.8cm). To synchronise behavioural and fNIRS data, a McDAQ data
268 acquisition device (www.mccdaq.com) was used to send information from the task
269 presentation laptop to the fNIRS system. The trigger was sent at the start of the
270 memory array in each trial.

271

272 *Procedure:*

273 We collected data from children in their homes which allowed us to move away from
274 a traditional lab setting. Two researchers were present during each session. One
275 researcher was responsible for checking the quality of the fNIRS signals, while the
276 other researcher attended to the child and parent. We requested all children to sit on
277 a chair at a table to make sure that all the equipment could be safely set up. The head
278 circumference of the child was measured so that the researchers could select the
279 appropriately sized fNIRS cap. Once the cap was fitted to the child's head,
280 measurements were taken from the inion to the nasion and from the two peri-auricular
281 points to make sure that the cap was centred. Children were given an iPad to watch
282 cartoons during the set-up process. Once the set-up was complete (approx. 15
283 minutes, see Fig. 3), the experimenter began the session by introducing the task as
284 "the colour game" and explained the rules using the flashcards. Children were then
285 told they were going to play the same game on the computer. Children were rewarded
286 with one sticker after they completed all the trials in each load regardless of their
287 performance (correct or incorrect) to maintain their motivation.

288 *Parental Questionnaires:*

289 Parents were given a booklet of questionnaires to complete. Variables from these
290 questionnaires have previously been shown to be associated with cognitive function
291 in children. The Strengths and Difficulties Questionnaire (Goodman, 1997) assesses
292 children's behavioural and emotional characteristics. This questionnaire has been
293 used to investigate individual differences in pre-schoolers executive function (Dias et
294 al., 2017; Hughes et al., 1998, 2000; Sulik et al., 2015). The Parenting Daily Hassles
295 scale (Crnic & Booth, 1991; Crnic & Greenberg, 1990) assesses the frequency and
296 impact of events that routinely occur in families with young children. Previous work
297 has shown a negative association between frequent daily hassles and children's

298 cognitive ability (Stuart, 2005). The Confusion, Order and Hubbub Scale (Matheny et
299 al., 1995) measures the amount of noise, confusion and disorganisation present in the
300 home. Twin studies have found that chaos in the home is an independent predictor of
301 cognitive outcomes (Hart et al., 2007; Petrill et al., 2004). The Parenting Stress Index
302 (Abidin et al., 2013) assesses the degree of stress in the parent-child relationship. Two
303 previous studies found that parenting stress predicted lower cognitive scores in
304 children (de Cock et al., 2017; Harewood et al., 2017). Lastly, a socio-economic scale
305 that assessed income, education and parental aspirations was also included. Several
306 studies have shown a strong association between socio-economic status and
307 children's cognitive ability and achievement (Ardila et al., 2005; Duncan et al., 2011;
308 Hackman et al., 2014, 2015; Hackman & Farah, 2009; Noble et al., 2012; Wijekumar
309 et al., 2019). For a detailed list of subscores, see Supplementary Table 1.

310 Data analyses:

311 *Behavioural analyses:*

312 Accuracy (A') and capacity (K) was calculated from hits (H) and false alarms (FA)
313 based on the behavioural responses. A' was calculated using Grier's (1971) formula,
314 updated by Aaronson and Watts (1987), where $A' = 1$ indicates perfect performances,
315 and $A' = 0.5$ indicates chance performance. The following formula is calculated to
316 account for a "yes" bias.

317 If $H \geq FA$: $A' = \frac{1}{2} + \{ [(H - FA) * (1 + H - FA)] / [4 * H * (1 - FA)] \}$

318 If $H < FA$: $A' = \frac{1}{2} - \{ [(FA - H) * (1 + FA - H)] / [4 * FA * (1 - H)] \}$

319 As demonstrated in Simmering (2016), for the cases where H and FA were equal to
320 each other, accuracy was set to 0.5. A' represents how accurately individuals perform
321 at each load in the task.

322 K was calculated for each load using Pashler's (1988) formula:

323 $K = Load * (H - FA) / (1 - FA)$

324 K represents the number of items that are successfully stored in working memory.
325 Note that, at most, K can equal the maximum number of items as the presented load.
326 Maximum K was estimated as the highest K value across all loads. Thus, maximum K
327 can, at most, equal the highest load. We ran a repeated-measures ANOVA with a

328 within-subjects factor of load to investigate how A' changed as a function of load. We
329 also applied median-splitting on maximum K estimates to divide our sample into low-
330 performers (LPs) and high-performers (HPs). We relied on maximum K for this
331 categorization as the number of items stored is the most important measure of visual
332 working memory performance.

333 *Pre-processing fNIRS signals:*

334 fNIRS data were pre-processed using the Homer2 package
335 (<https://www.nitrc.org/projects/homer2/>). Raw data were pruned using the
336 *enPrunechannels* function (dRange= 0.01 - 300, SNRthresh=2, SDrange=0 – 45).
337 Signals were converted from intensity values to optical density (OD) units using the
338 *Intensity2OD* function. Data was corrected for motion using the
339 *hmrMotionCorrectPCArecurse* function, (tMotion=1, tMask=1, STDEVthresh=50,
340 AMPthresh=0.5, nSV=0.97, maxIter=5, turnon=1). Data was scanned for motion
341 artifacts using *hmrMotionArtifactByChannel* function (tMotion=1, tMask=1,
342 STDEVthresh=50, AMPthresh=0.5). Then, the function *enStimRejection* (tRange=-1
343 to 10) was used to turn off stimulus triggers during any segments that contained motion
344 artifacts. The data were band-pass filtered using *hmrBandpassFilt* to include
345 frequencies between 0.016Hz and 0.5Hz. Using the function *hmrOD2Conc*, the OD
346 units were converted to concentration units (partial pathlength factor = 6 for each
347 wavelength). Lastly, the function *hmrBlockAvg* was used to calculate the block
348 average for a time window of -1 to 12 seconds. The mean activation from -1 to 0 s was
349 used as the baseline to subtract from activation in the rest of the window (as specified
350 in function *hmrBlockAvg*).

351 *fNIRS group analyses:*

352 Only correct trials were included in the fNIRS group analyses. We chose a
353 hemodynamic response window from the 3rd to 6th second for further analyses. This
354 time window was selected based on findings from Buss and colleagues (2014) and on
355 our observations of the peak of the hemodynamic response of the task. Note that we
356 do not consider a 'later' window (after the 6th second) as trials with shorter inter-trial
357 intervals would also include succeeding trials.

358 We computed the mean hemodynamic activation for the chosen window for each load
359 (1, 2, 3), trial type (same, different), chromophore (HbO, HbR), channel (1 to 14), and
360 participant ($N=74$). Note that the mean number of correct trials included in the
361 neuroimaging analyses for HPs were 15 ± 0.17 trials for load 1, 14 ± 0.34 trials for load
362 2, and 12 ± 0.32 trials for load 3. The mean number of correct trials included in the
363 neuroimaging for LPs were 15 ± 0.24 trials for load 1, 12 ± 0.43 trials for load 2, and
364 10 ± 0.27 trials for load 3.

365 A repeated measures ANOVA was run for each of the 14 channels. Each ANOVA
366 included within-subject factors of load (1, 2 and 3 items), trial type (same and different)
367 and chromophore (HbO, HbR) and a between-subjects factor of group (HPs and LPs).
368 We only focussed on effects that showed a significant interaction with chromophore to
369 maximize the possibility of observing differences between HbO and HbR activation.
370 We examined channels with a significant interaction between load and chromophore
371 to assess if activation increased with increasing load as shown in previous studies.
372 Next, we examined channels that showed a significant interaction between group, load
373 and chromophore to examine differences between low-performers and high-
374 performers. The Benjamini-Hochberg test was run with a false discovery rate of 0.05
375 to control for the number of channels that were included in the analyses. Here,
376 individual p-values from interaction between group, load, and chromophore for each
377 channel were ranked in ascending order, with 1 being the smallest p-value, 2 being
378 the second smallest value, and so forth. We then calculated each individual p-value's
379 critical Benjamini Hochberg value using the following formula: $(i/m)Q$, where, i =
380 individual p-value rank, m = total number of tests, and Q = false discovery rate of 0.05.
381 Finally, we compared our original p-values to the critical Benjamini-Hochberg value to
382 find p-values that were smaller than the critical value. All channels with p-values for
383 the interactions between group, load and chromophore below this highest p-value
384 were considered significant. Lastly, Bonferroni correction was applied to the post-hoc
385 tests conducted in following up these significant interactions.

386

387 *Correlations between behaviour, brain function and parental questionnaire data:*

388 The questionnaire data were log-transformed to account for skewed distributions. All
389 data (questionnaires, behaviour and brain function) was screened for outliers that were

390 3 standard deviations above or below the mean. We identified nine outliers - one in
391 the right parietal cortex, one in the left parietal cortex, three in the Strengths and
392 Difficulties Questionnaire, one in the Parenting Daily Hassles Scale, two in the parental
393 aspirations measure, and one in the parental education measure. Next, we winsorized
394 these outliers by replacing their values with the observation closest to them but are
395 just below the threshold for defining outliers. Then we correlated our questionnaire
396 variables with our behavioural and brain measures. The Benjamini-Hochberg test run
397 with a false discovery rate of 0.1 was able to control for the number of significance
398 tests on correlations that we performed. All correlations with p-values below the critical
399 p-value were considered significant.

400

401 **Results**

402 *Behavioural results:*

403 The repeated measures ANOVA on A' revealed that the main effect of load was
404 significant ($F[2, 146] = 90.37, p < .001$, partial eta square (η_p^2) = 0.301). Follow-up
405 pairwise comparisons revealed that accuracy at load 1 ($M=0.96, SD=0.04$) was greater
406 than at load 2 ($M=0.86, SD=0.17, t[73] = 5.29, p < .001$), and load 3 ($M=0.82, SD=0.13$,
407 $t[73] = 9.5, p < .001$). Further, accuracy at load 2 was greater than accuracy at load 3,
408 ($t[73] = 2.27, p = .026$) – see Fig. 4(a). These results were in agreement with previous
409 findings (Simmering, 2012).

410 Median-splitting was applied to the maximum K estimates. The median
411 maximum K was 2 items, with 13 children performing at the median. We decided to
412 include these 13 children into the HP group because their individual accuracy scores
413 at load 3 fell above the median accuracy. This resulted in a total of 39 HPs and 35
414 LPs. Figure 4(b) shows the mean maximum K estimates for HPs (2.4 ± 0.06 items)
415 and LPs (1.5 ± 0.06 items).

416 *fNIRS Results:*

417 Channels showing significant interactions between load and chromophore and group,
418 load and chromophore are shown in Table 1. Note that only effects in channels that
419 survived the Benjamini-Hochberg correction are reported. The interaction between
420 load and chromophore was significant in channels overlying the left middle frontal

421 gyrus ($F[2,144] = 8.599, p=.000$; $F[2,144] = 4.857, p=.009$) and left inferior frontal
422 gyrus ($F[2,144] = 7.542, p=.001$). Posthoc tests revealed that activation at load 3 was
423 greater than activation at load 1 and load 2. Figure 5 shows the change in HbO
424 activation with load for channel 6 overlying the left middle frontal gyrus.

425 A significant interaction between group, load and chromophore was observed
426 in channels overlying the left middle frontal gyrus ($F[2,144] = 4.773, p=.01$ – Figure
427 6a, 6c and 6e), left inferior frontal gyrus ($F[2,144] = 4.616, p=.011$ – Figure 6b, 6d and
428 6f), right angular gyrus ($F[2,144] = 6.604, p=.002$ – Figure 7a, 7c and 7e), right
429 supramarginal gyrus ($F[2,144] = 6.005, p=.003$ – Figure 7b, 7d and 7f), left inferior
430 parietal lobule ($F[2,144] = 4.843, p=.009$ – Figure 8a, 8c and 8e), and left
431 supramarginal gyrus ($F[2,144] = 4.646, p=.011$ – Figure 8b, 8d and 8f). Across all
432 these areas, LPs showed greater activation at load 3 than at load 1. Importantly, HPs
433 did not demonstrate this modulation of activation with increasing load. HPs showed
434 greater activation than LPs at load 1 in channels overlying the left middle gyrus, left
435 inferior frontal gyrus, right angular gyrus, right supramarginal gyrus, and left
436 supramarginal gyrus. Finally, LPs showed greater activation than HPs at load 3 in a
437 channel overlying the left middle frontal gyrus.

438 *Correlations between behavioural performance, brain activation and parental*
439 *questionnaires:*

440 We averaged activation across channels that showed a significant interaction between
441 group, load and chromophore to create three brain clusters as they showed the same
442 trend. The three brain clusters were left frontal cortex (averaging channel 6 and
443 channel 8), right parietal cortex (averaging channel 9 and channel 11) and left parietal
444 cortex (averaging channel 12 and channel 14). Next, we calculated the difference in
445 activation between load 3 and load 1 in these brain areas (for each participant) as this
446 key measure differentiated LPs from HPs. As expected, the difference in activation
447 between loads in the three brain clusters was negatively correlated with maximum K
448 suggesting that this association was still strong even when the variables were
449 considered as continuous measures (see Supplementary Fig. 1). The difference in
450 activation between loads in each of the three brain areas was correlated with data
451 from the parental questionnaires. We pooled all 31 correlations and applied the
452 Benjamini-Hochberg correction separately for each brain area (left frontal cortex, left

453 parietal cortex, and right parietal cortex) with a false discovery rate of 0.1. Only those
454 correlations that survived the Benjamini-Hochberg correction are reported. We found
455 that the difference in activation between loads in the left parietal cortex was positively
456 correlated with the life stress subscore from the Parenting Stress Index ($r=0.293$,
457 $p=.011$, see Fig. 9). Life stress in this scale refers to any major life event that
458 significantly increases life stress. We also found that the difference in activation
459 between loads in the left parietal cortex was negatively associated with parental
460 aspirations ($r=-0.323$, $p=.006$). Parental aspirations were the highest qualification
461 parents hoped their children would achieve.

462 *Mediation Analyses:*

463 We conducted mediation analyses to further understand the association between
464 behaviour, brain activation, and parental factors. Using mediation, we aimed to extend
465 upon the two significant findings from the questionnaire correlation analyses and
466 determine whether a trivariate relationship exists between behavioural performance,
467 brain function and parental factors. Concretely, we investigated if the difference in
468 brain activation was a potential mediator between the distal predictor (parental factor)
469 and the outcome measure (behavioural performance). According to Shrout and Bolger
470 (2002), when a predictor is not experimental-based and distal in nature (e.g., home
471 environment), an association between the predictor and the mediator is sufficient to
472 warrant a test for mediation. Therefore, we focused on the significant bivariate
473 relationships we identified from the correlation analyses described in the previous
474 section: (1) the association between the life stress score and the difference in
475 activation between loads in the left parietal cortex and (2) the association between
476 parental aspirations and the difference in activation between loads in the left parietal
477 cortex. For our analyses, we used the mediation function with bootstrapping in R. We
478 found that the difference in activation between loads in the left parietal cortex partially
479 mediated the relationship between life stress and maximum K (indirect path $=-0.1429$,
480 $p=.016$). To determine if socioeconomic factors had an indirect effect on this
481 association, we controlled for both parental education and income. This association
482 remained significant even after accounting for parental education and income
483 ($p=.0014$). Specifically, we found that higher life stress was related to a larger
484 difference in activation between loads in the left parietal cortex, which in turn was
485 related to lower maximum K. Next, we found that the difference in activation between

486 loads in the same area partially mediated the relationship between parental aspirations
487 and maximum K (indirect path =0.4643, $p=.026$). However, this mediation effect did
488 not hold after controlling for parental education and income suggesting an influence of
489 socioeconomic factors on the association between behavioural performance, brain
490 activation and parental aspirations for their children.

491

492 **Discussion**

493 VWM is an essential cognitive system with a highly limited capacity that is reliably
494 predictive of future academic achievements, making it important to understand the
495 nature of individual differences in children (Bull et al., 2008; Swanson & Berninger,
496 1996). Critically, it is important to try to fulfil this objective in experimental designs
497 without the effect of varying age within the cohort. In the current study, we investigated
498 how individual differences in VWM performance and brain function are linked to
499 parental and home environment factors. We recorded behavioural and brain activation
500 data from 4.5-year-old children with little variation in age as they completed a colour-
501 change-detection task in their homes. Our research also demonstrated the feasibility
502 of collecting neural data on children in their homes. Home-based testing present with
503 unique challenges that require experimenters to be creative while maintaining a
504 standardized procedure. As examples, experimenters are required to manage space
505 constraints while setting up equipment and to deal with distractions for themselves
506 and the children being tested in a respectful manner in different households. Despite
507 these difficulties, we attribute the low drop-rate to an efficient experimental design,
508 extensive training in collecting behavioural and brain imaging data with children and
509 the resourcefulness of the experimenters to maintain a good rapport with children and
510 their parents to create a fun and engaging atmosphere where children did not feel they
511 were being “tested”.

512 In the current study, we observed load-dependent increases in accuracy and brain
513 activation. In general, this finding is in agreement with previous VWM work in children
514 and adults (Ambrose et al., 2016; Buss et al., 2014; Jha & McCarthy, 2000; Linden et
515 al., 2003; Todd & Marois, 2004; Wijekumar, Magnotta, et al., 2017). Our first critical
516 question was to investigate whether differences in activation in the fronto-parietal
517 network would underlie differences in behavioural performance. We found that LPs

518 showed greater activation at load 3 than at load 1 in the left frontal, left parietal and
519 right parietal cortices. By contrast, the HPs showed no modulation of activation
520 between loads. HPs showed greater activation than LPs at load 1 across all three
521 cortices and LPs showed greater activation than HPs at load 3 in the left frontal cortex.
522 We posit that HPs were more 'prepared' through achieving a *heightened state of*
523 *attention* eliciting greater activation at the lowest load (at the start of the experimental
524 task) compared to the LPs. We further suggest that HPs were able to efficiently
525 manage the more challenging demands of increasing VWM loads by utilizing similar
526 levels of neural resources as they did not show any significant increases in activation.
527 On the other hand, LPs showed increasing activation and poor performance with
528 increasing VWM load. We argue that LPs would have needed to *effortfully* attend to
529 the demands of increasing VWM load by increasing activation and thus, recruited more
530 neural resources. Critically, this increase in activation was not accompanied by an
531 increase in performance in LPs, moving the interpretation away from a compensatory
532 account of neurocognitive function often reported in older adults wherein greater neural
533 resources are recruited to maintain the same performance as younger adults (Cabeza
534 et al., 2018). In agreement with our finding, Honey and colleagues (2000) found that
535 adults who performed poorly on a verbal working memory task showed increased
536 activation in the bilateral posterior parietal cortex. They surmised that this increase in
537 parietal activation could be reflective of an increase in attentional demands and the
538 use of visuospatial strategies. Along the same vein, Jaeggi et al. (2007) found that
539 low-performing adults engaging in a challenging dual task displayed large load-
540 dependent increases in activation when their capacity limitations were challenged.
541 They reported that the increase in activation observed in low-performers was due to
542 the additional recruitment of attentional and strategy-related resources. Further, they
543 found that high-performers did not show an increase in activation with increasing task
544 difficulty, which they suggested reflected more efficient processing. However, these
545 findings are not in line with results from an adult study conducted by Nagel and
546 colleagues (2009) who reported that young high-performing adults showed increasing
547 activation with increasing load in a spatial working memory task. In the current study,
548 it is also possible that LPs were unable to suppress distraction or irrelevant information
549 in the event of increasing task demands, thus increasing activation with a decline in
550 performance. The inability to suppress distraction to irrelevant information and as a

551 result, poorer VWM performance during a preferential looking task has been associated
552 with greater activation in the frontal cortex in children in rural settings in India
553 (Wijeakumar et al., 2019).

554 Our second critical question was to examine whether home environment and
555 parental factors could shed light on the nature of individual differences in behaviour
556 and associated brain activation. We found that the relationship between the frequency
557 of stressful life events and poor behavioural performance in the children in the VWM
558 task was mediated by activation in the left parietal cortex. Higher life stress was
559 measured as a greater number of stressful life events including but not limited to,
560 divorce, change in job/school, death of a family member, and substance abuse.
561 Further, this finding remained significant after we controlled for parent income and
562 education, suggesting it is unrelated to socio-economic factors. Our finding is in line
563 with an EEG study by Troller-Renfree and colleagues (2020) who found that infants of
564 mothers who experienced higher chronic physiological stress showed altered brain
565 activation patterns during the first year of life. They posit that maturational lags in
566 development can persist into later life and have an effect on cognitive processing.
567 Further, Hanson and colleagues (2012) assessed cumulative life stress in children and
568 found a negative association with working memory performance. It is also possible
569 that stressful life events can significantly affect the quantity and/or quality of time that
570 parents spend with their children. Crnic and colleagues (2005) found that parent-
571 reported life stress assessed over two years had a negative association with maternal
572 parenting behaviour and the quality of parent-child interactions, which in turn
573 contributed to poor behavioural functioning in children at age 5. Taken together, in the
574 current study, we suggest that a greater number of stressful life events might cause
575 instability in the child's life, resulting in changes to their cognitive processing. In a
576 household with shifting/shifted stability, children might be constantly distracted and
577 unable to consistently sustain attention and maintain information to efficiently
578 accomplish goals or tasks. In addition, it is also possible that parents distracted by
579 stressful life events might struggle to fulfil daily goals to manage care-taking and might
580 transfer poor skills of goal maintenance and traits such as easy distractability to their
581 children. It is important to acknowledge that such children who are negatively impacted
582 by a stressful home environment might have yielded a more pronounced atypical
583 brain-behaviour response since they were afterall subjected to home-based testing

584 and assessments. We recommend that future studies should assess if brain-behaviour
585 responses observed in such children during testing in their home environment can be
586 similarly observed under controlled lab conditions.

587 In summary, our findings revealed that LP children showed a greater difference
588 in activation between the low and high loads in a fronto-parietal VWM network. On the
589 other hand, HP children did not show any modulation in activation with increasing
590 VWM load. We also found that this difference in activation between loads in a left
591 parietal cortex partially mediated the relationship between parent-reported life stress
592 and VWM performance.

593

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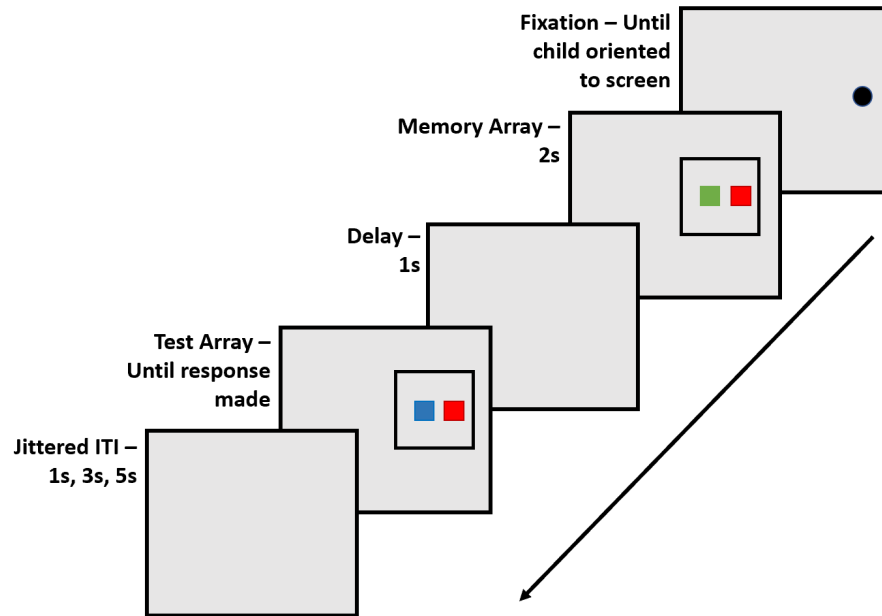
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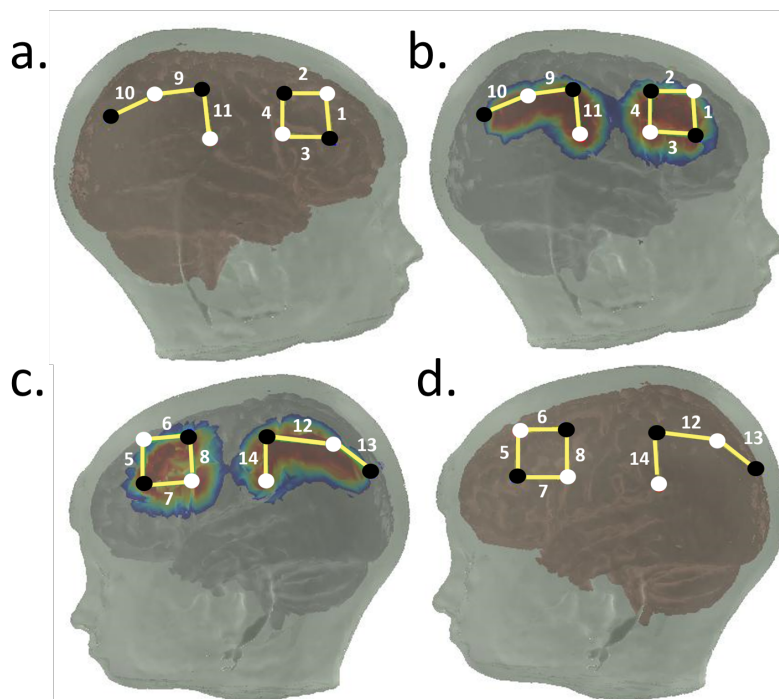
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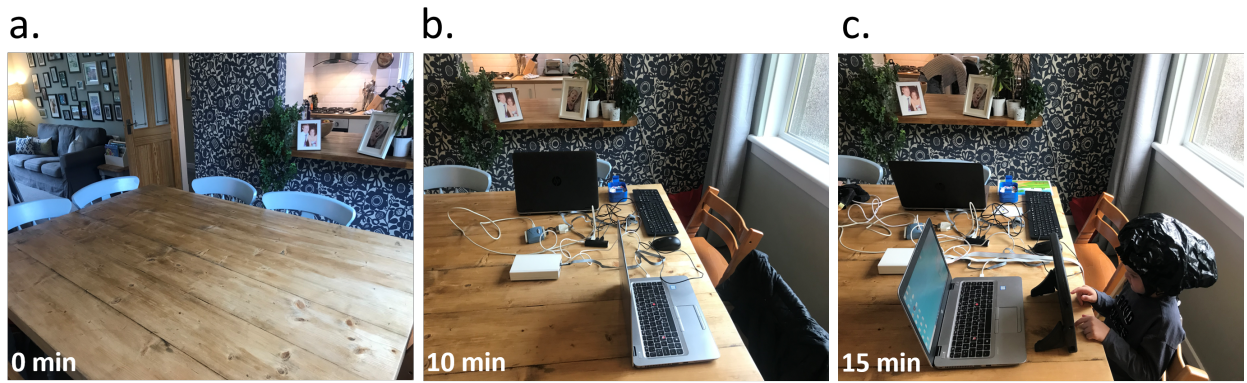
825 **Fig. 1** Colour CD task – Load 2 different trial.



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827 **Fig. 2(a & d)** Probe geometry over the right and left hemispheres. The white circles represent
828 the sources and the black circles represent the detectors. **(b & c)** Sensitivity profiles after
829 running Monte Carlo Simulations with 100 million photons.

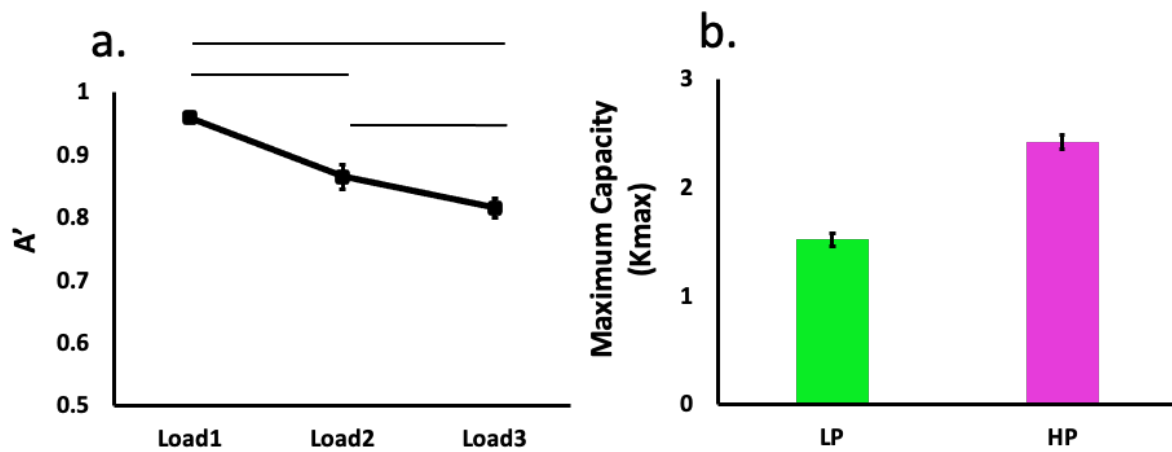
HOME ASSESSMENT OF VISUAL WORKING MEMORY



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831 **Fig. 3.** Experimental set-up inside a participant's home. (a) Beginning of set-up (b) 10 minutes

832 into set-up (c) Complete set-up after 15 minutes.

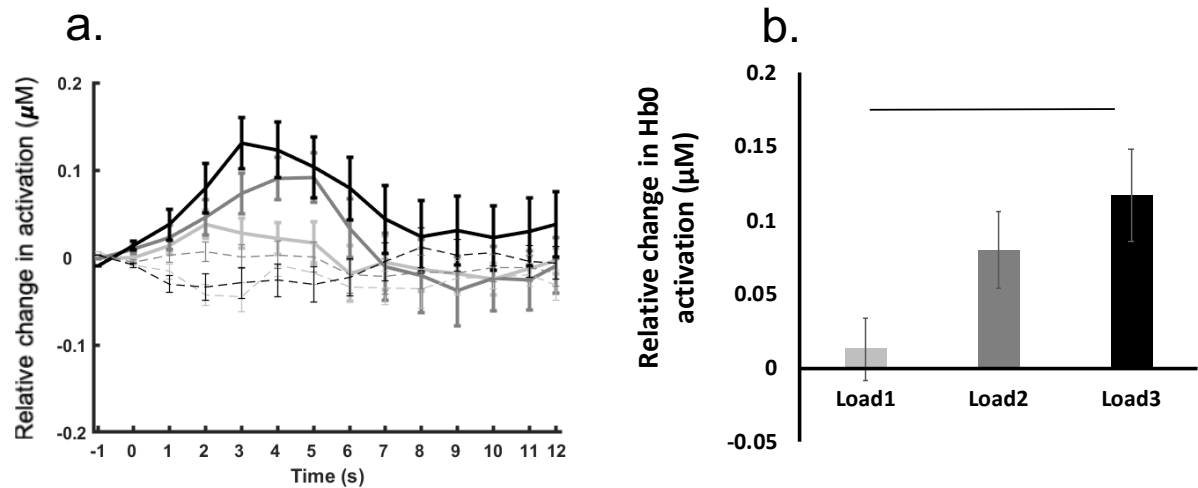


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834 **Fig. 4(a)** A' decreased as load increased from 1 to 3 items. (b) Maximum K estimates for high835 and low performers. Error bars show SEM. '-' indicates significance at $p < .05$.

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839 **Fig. 5(a)** HbO (solid lines) and HbR (dashed lines) activation in channel 6 overlying the left
840 middle frontal gyrus. Load 1 is shown in light grey, load 2 in dark grey, and load 3 in black.
841 Error bars show 1 SE averaged over 1sec intervals. **(b)** Bar plot showing greater HbO
842 activation at load 3 compared to load 1. Error bars show SEM. '-' indicates significance at
843 $p < .05$.

844 **Table. 1.** Channels showing significant interactions between load and chromophore and
 845 group, load and chromophore. Posthoc results are shown for HbO activation.

Channel No.	Brain area (MNI coordinates)	Load x Chromophore (HbO)	Group x Load x Chromophore (HbO)
Channel 1	Right middle frontal gyrus		
Channel 2	Right middle frontal gyrus		
Channel 3	Right inferior frontal gyrus		
Channel 4	Right inferior frontal gyrus		
Channel 5	Left middle frontal gyrus	Load 3 > Load 1 (p=.001) Load 3 > Load 2 (p=.002)	
Channel 6	Left middle frontal gyrus	Load 3 > Load 1 (p=.011)	Load 3: LPs > HPs (p=.037) LPs: Load 3 > Load 1 (p<.001) Load 1: HPs > LPs (p=.024)
Channel 7	Left inferior frontal gyrus	Load 3 > Load 1 (p=.01)	
Channel 8	Left inferior frontal gyrus		LPs: Load 3 > Load 1 (p=.004) LPs: Load 2 > Load 1 (p=.045) Load 1: HPs > LPs (p=.016)
Channel 9	Right angular gyrus		LPs: Load 3 > Load 1 (p=.004) Load 1: HPs > LPs (p=.001)
Channel 10	Right superior occipital gyrus		
Channel 11	Right supramarginal gyrus		LPs: Load 3 > Load 1 (p=.011) Load 1: HPs > LPs (p=.025)
Channel 12	Left inferior parietal lobule		LPs: Load 3 > Load 1 (p=.009)
Channel 13	Left angular gyrus		
Channel 14	Left supramarginal gyrus		LPs: Load 3 > Load 1 (p=.033) Load 1: HPs > LPs (p=.01) Load 2: HPs > LPs (p=.036)

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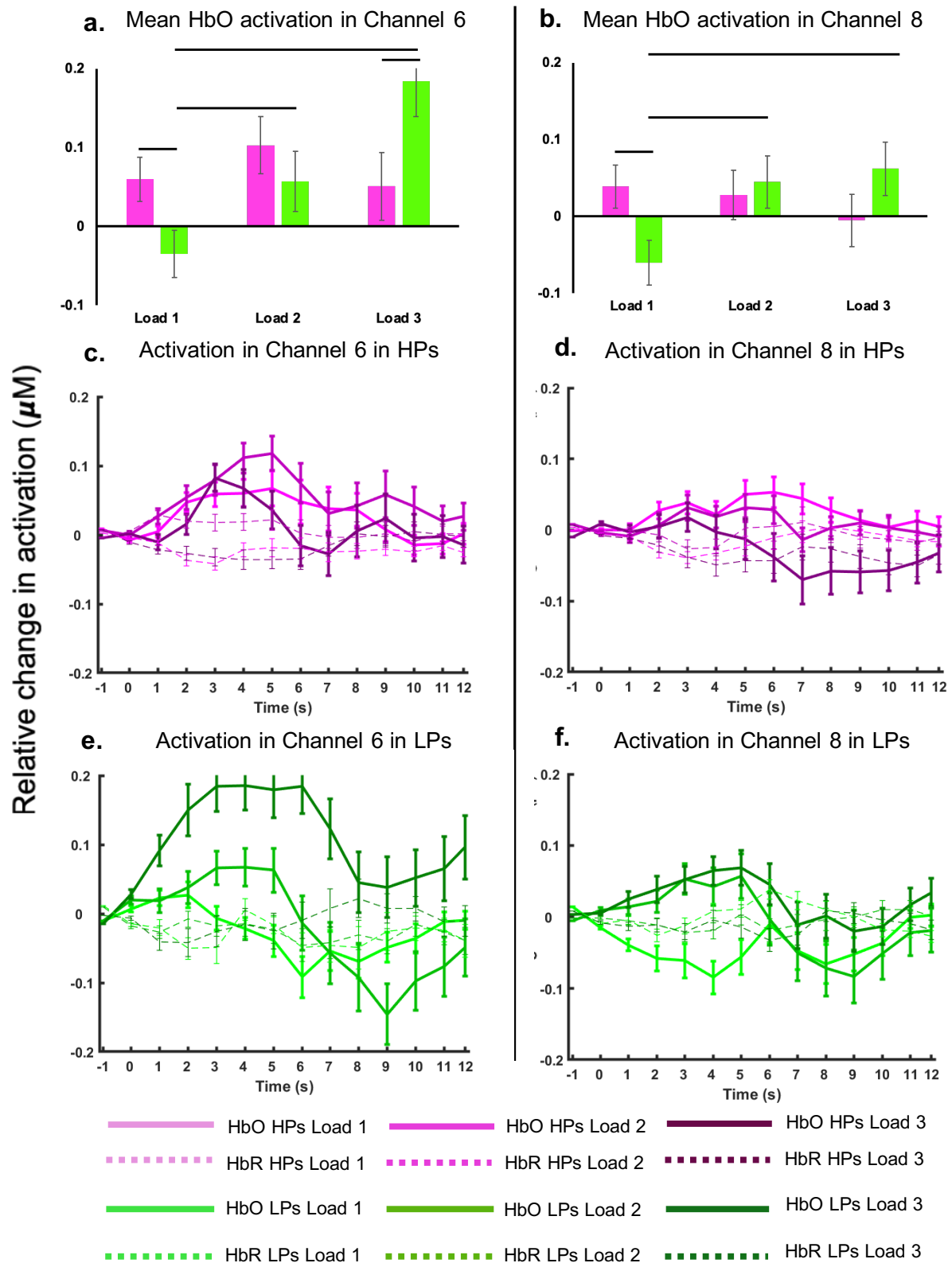
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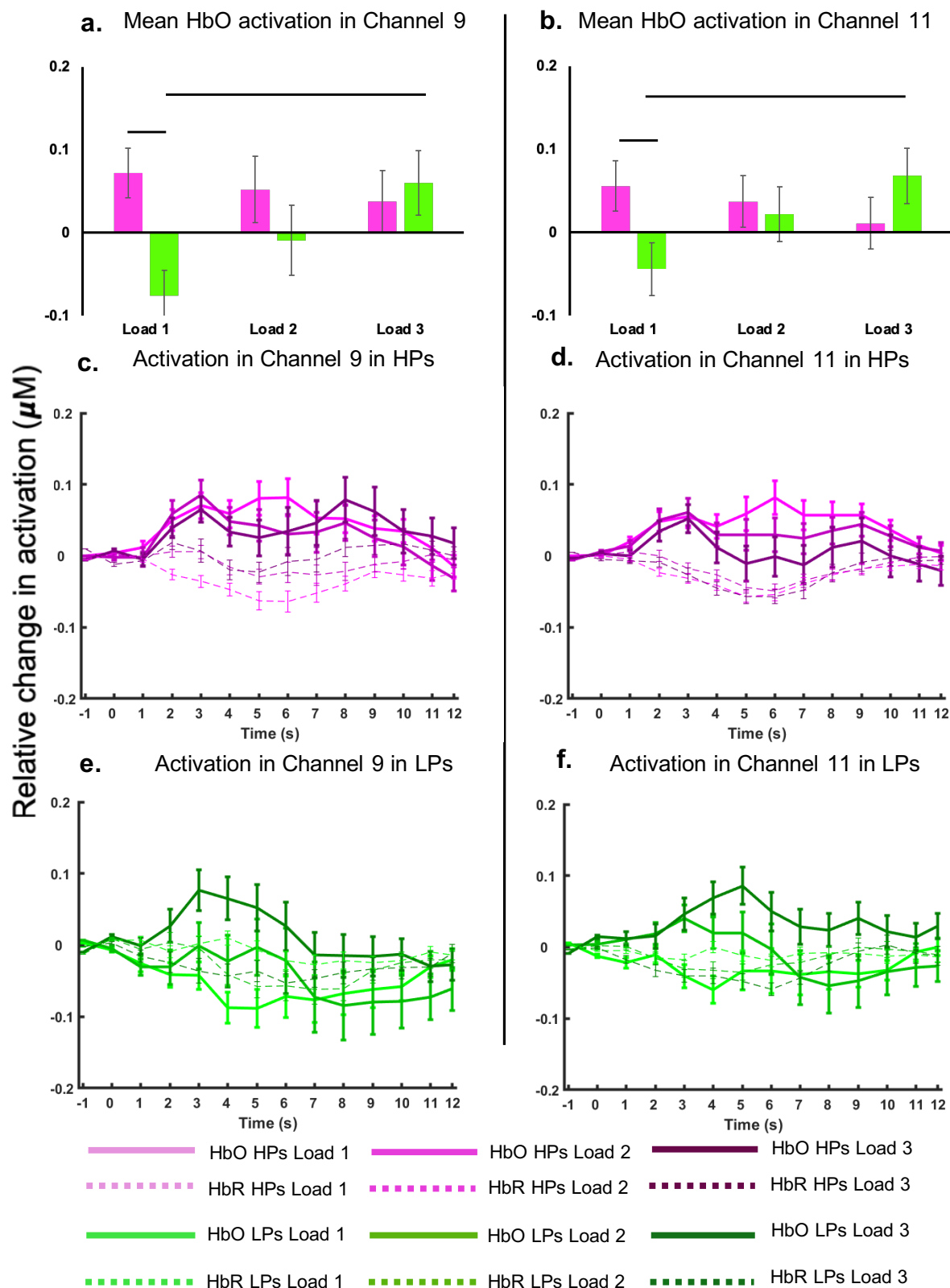


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855 **Fig. 6 (a) and (b).** Bar plots showing mean HbO activation for the time window between 3 s
 856 and 6 s for LPs (green) and HPs (magenta) in channel 6 and channel 8 (overlying the left
 857 frontal cortex) respectively. **(c) and (d)** Hemodynamic activation for HPs in channel 6 and
 858 channel 8 respectively. **(e) and (f)** Hemodynamic activation for LPs in channel 6 and channel

859 8 respectively. Error bars show 1 SE averaged over 1sec intervals. '-' indicates significance at
860 $p < .05$.

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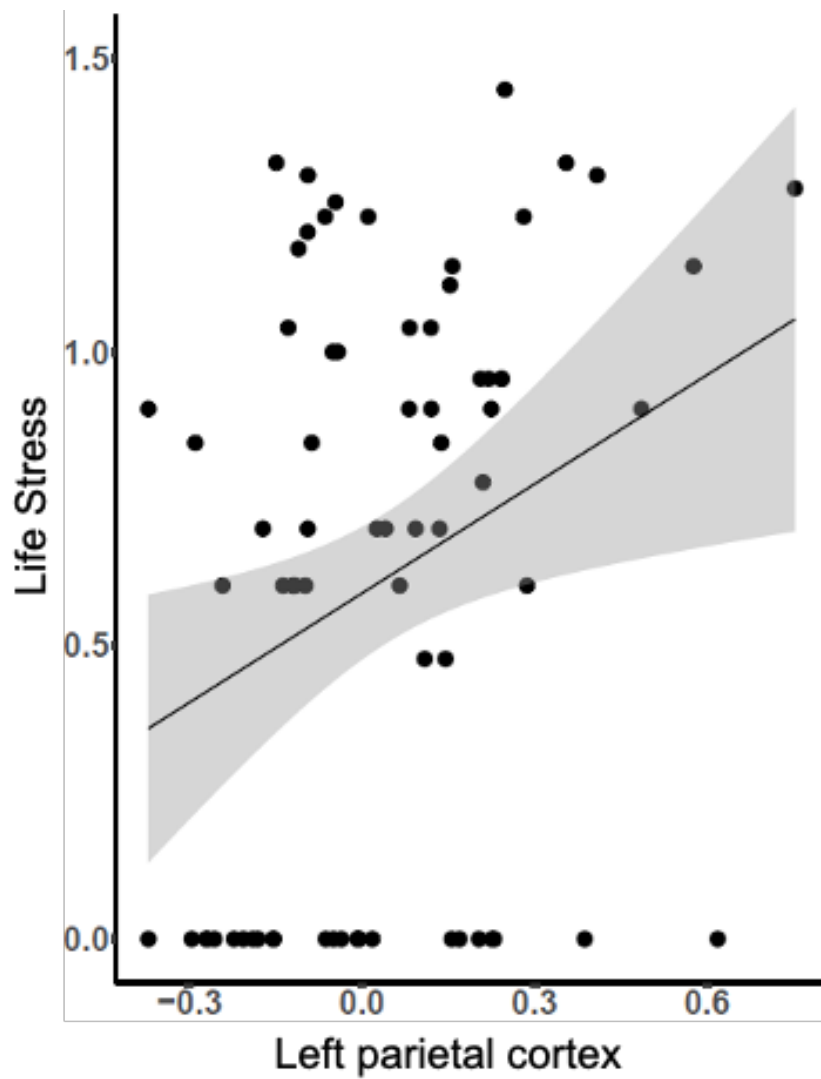


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863 **Fig. 7 (a) and (b).** Bar plots showing mean HbO activation for the time window between 3 s
 864 and 6 s for LPs (green) and HPs (magenta) in channel 9 and channel 11 (overlying the right
 865 parietal cortex) respectively. **(c) and (d)** Hemodynamic activation for HPs in channel 9 and
 866 channel 11 respectively. **(e) and (f)** Hemodynamic activation for LPs in channel 9 and channel

873 channel 14 respectively. **(e) and (f)** Hemodynamic activation for LPs in channel 12 and
874 channel 14 respectively. Error bars show 1 SE averaged over 1sec intervals. ‘-’ indicates
875 significance at $p < .05$.

876



877

878 **Fig. 9.** Plot showing a positive correlation between the difference in HbO activation in the left
879 parietal cortex and the life stress subscore of the Parental Stress Index.