

# Spatial probability cuing and right hemisphere damage

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## ARTICLE INFO

### Article history:

Accepted 31 August 2012

Available online 17 October 2012

### Keywords:

Attention

Spatial neglect

Priming

Statistical learning

Reaction time

## ABSTRACT

In this experiment we studied statistical learning, inter-trial priming, and visual attention. We assessed healthy controls and right brain damaged (RBD) patients with and without neglect, on a simple visual discrimination task designed to measure priming effects and probability learning. All participants showed a preserved priming effect for item color. Contrary to healthy controls and RBD participants without neglect, RBD participants with neglect did not show positional priming and both RBD groups learned the underlying spatial probability distribution of target locations to a lesser degree. To see if the latter deficiency could be improved, we tested a patient with long standing chronic spatial neglect on three separate days and observed improved identification times for left sided, high probability, targets. In summary, we found preservation of priming per se in people with spatial neglect. However, this was only clearly demonstrable for color priming and not for positional priming. Associated with this impairment was a difficulty in learning the overall statistical structure of target locations. In a patient with severe persistent neglect we were able to demonstrate that the deficit in statistical learning was not absolute, as this subject improved his identification times for targets appearing in high probability regions of the test display.

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## 1. Introduction

The visual world is formed by a complex combination of features and contains more information than the visual system can process. Therefore, we need mechanisms that help us select what is behaviorally relevant. This selection of incoming stimuli, more specifically called visual attention, is guided by mechanisms such as the salience map – where the most salient object attracts attention thanks to its physical distinctiveness (Fecteau & Munoz, 2006; Itti & Koch, 2001; Itti, Koch, & Niebur, 1998) – or by the use of spatial, temporal and statistical information in order to facilitate the direction of attention (Maljkovic & Nakayama, 1994, 1996; Perruchet & Pacton, 2006; Roser, Fiser, Aslin, & Gazzaniga, 2011). The combination of those two elements forms what Fecteau and Munoz (2006) called the priority map, which unites the salience and relevance of the target. In the present paper, we explore how diverse forms of statistical regularities, such as priming and distributional learning, can be used as cues to direct attention and if lesions in the right hemisphere (RH) impair this ability.

### 1.1. Statistical learning and priming in visual search

Our sensitivity to the statistical structure of our environment has long been recognized (Estes, 1950), and has recently been extended to attentional phenomena. For example, Chun and Jiang (1998); Chun (2002) found that learning context information drives spatial attention and makes the identification of the objects that occur in repeated, common contexts, faster. Others researchers (Geng & Behrmann, 2002, 2006; Jiang, Swallow, & Rosenbaum, in press-a, in press-b; Walthew & Gilchrist, 2006) have found that detecting or identifying items is quicker when they are biased to repeat the same position. In those studies, the authors have concluded that our classification of items in visual search tasks is sensitive to the statistical structure of targets but also to the structure of distractors (Vincent, 2011; Vincent, Baddeley, Troscianko, & Gilchrist, 2009). Even a classical attentional phenomenon like inhibition of return is modulated by the probability that target locations will be repeated (Farrell, Ludwig, Ellis, & Gilchrist, 2010).

Typically, spatial probability learning is inferred when there is quicker identification of targets in high probability regions. However, if the number of high probability regions of a display is limited then those locations will have many sequential repeats and therefore faster identification might be more related to location priming (Maljkovic & Nakayama, 1994, 1996), unless specific efforts are made to avoid this (Jones & Kaschak, 2012; Walthew & Gilchrist, 2006).

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Being the first to report position priming, Maljkovic and Nakayama (1996) had a target repeat or switch its location on subsequent trials. The authors found that when the stimulus was repeated at the same position, participants were faster to detect it than when it was switched, and this facilitation lasted until five to eight successive trials. The finding that positional repetition speeds detection has been replicated in different conditions and studies (Hillstrom, 2000; Kristjánsson & Campana, 2010), and has led some researchers (Walthew & Gilchrist, 2006) to suggest that the improved target detection in regions of high probability might be explained by positional priming.

However, positional priming does not appear to be a sufficient explanation, since reducing the frequency of sequential repeats (by either increasing the number or possible positions where targets can appear or by not repeating the same position for successive trials) still results in faster reaction times (RT) in high probability regions (Druker & Anderson, 2010; Jiang et al., in press-a, in press-b; Jones & Kaschak, 2012). The extent of position priming's contribution to spatial statistical learning remains unclear.

We hypothesize that statistical learning relies on detecting sequential regularities, though it is not simply an expression of priming effects. This hypothesis predicts that an impairment of priming will lead to impaired statistical learning. The evidence for this interpretation follows from the demonstration that the learning of spatial statistical distributions (Geng & Behrmann, 2002) is lessened when spatial repeats are limited (Walthew & Gilchrist, 2006), and that the demonstration of statistical learning depends principally on adjacent elements. For example, the robust learning of word borders depends on the relation of adjacent syllables and does not occur for non-adjacent syllables (Newport & Aslin, 2004). In addition, this relationship is bound up with attentional systems, since the ability to learn adjacency relations, especially if irrelevant distractors appear, is absent for unattended sequences (Turk-Browne, Jung, & Scholl, 2005).

## 1.2. Neural network for priming and statistical learning

A possible confirmation of the correlation between priming and statistical learning can be provided by fMRI studies focusing on the neural correlates of those two mechanisms, as well as by studies of patients suffering a lesion in those particular regions. In a study where they reproduced the paradigm of Maljkovic and Nakayama (1994, 1996), Kristjánsson, Vuilleumier, Schwartz, Macaluso, and Driver (2007) found a repetition-suppression BOLD activity in regions responsible of attentional control, including the intraparietal sulci. The authors also discovered a difference in the activation of the brain regions between the color and the location priming: location priming was associated with a reduction in the contralateral inferior parietal and frontal areas, whereas color priming was associated with inferior temporal region. The authors also revealed that the right inferior parietal cortex was activated for both the left visual field and right visual field, which was not the case for the left hemisphere, highlighting a greater implication of the RH in spatial attention.

The role of the RH in statistical learning was also confirmed in a study conducted by Roser et al. (2011) on a split-brain patient during a visual task. Also, Miller, Valsangkar-Smith, Newman, Dumont, and Wolford (2005) showed that the estimation of future events from past experience was associated with greater RH activation.

In order to investigate if a lesion in the RH affects position priming and statistical learning to the same extent, and to probe the relation between spatial priming, priming in general, spatial probability learning and brain attentional systems, we undertook a study of target classification contrasting two different forms of priming (spatial and color) with overall spatial probability learning

in participants who suffered a RH damage with and without attentional impairment.

## 1.3. Spatial neglect

Spatial neglect is a multifaceted deficit characterized by a variety of symptoms (Danckert & Ferber, 2007) making a global understanding of the disorder difficult. However, certain trends can be discerned. Neglect patients with a lesion in their RH generally have some attentional impairment, e.g. they often fail to explore or direct their attention to contralesional visual space (Halligan, Fink, Marshall, & Vallar, 2003).

In addition, people with neglect have difficulty in detecting environmental regularities underlying spatial probability learning. They show spatial working memory impairments, prolonged attentional blinks, and impaired covert spatial priming (Danckert & Ferber, 2007; Ferber & Danckert, 2006; Husain & Rorden, 2003; Husain et al., 2001; Samuelsson, Hjelmquist, Jensen, Ekholm, & Blomstrand, 1998). In a recent study where subjects played the children's game Rock, Paper, Scissors against a computer opponent, right brain damaged (RBD) patients failed to exploit the computer's biased strategy of selecting the same option 80% of the time. Left brain damaged participants and controls did not (Danckert, Stöttinger, Quehl, & Anderson, 2011).

## 1.4. Our study

The present study aims to compare priming and statistical learning in a set of RBD patients with and without neglect, as well as a comparison group of older controls. Our procedure used a simple visual search task with no distractors to prevent neglect participants from becoming fixated on distractors on the right side of the screen. To improve our confidence that participants had actually detected the target, we had them report the color of the stimuli. We adapted the method of Druker and Anderson (2010) to include a large number of target locations, allowing us to assess both positional priming and statistical learning. Furthermore, the location of the lesion of our participants in the RH allows us to investigate the implication of this region for these two mechanisms for directing spatial attention, and to determine if a distinction exists between neglect and non-neglect patients. As a difference exists between color and location priming (Kristjánsson, Vuilleumier, Malhotra, Husain, & Driver, 2005; Kristjánsson et al., 2007), our hypothesis was that patients with and without neglect will show color priming but will be impaired for location priming, an impairment that should extend to statistical learning. Lastly, as we found impairment in our mild neglect participants, we extended our study and undertook a single case study to see if this deficit in statistical learning was absolute or remediable. We tested a single participant with chronic, stable, severe neglect on three separate days with the same paradigm.

## 2. Methods

The study received approval from the University of Waterloo Office of Research Ethics and all participants gave written informed consent to participate in the experiment.

### 2.1. Participants

Sixteen subjects participated in the main study. All participants had normal or corrected-to-normal vision. There were five healthy control participants (four females and one male, average age of 67.6, and standard deviation (sd) of 2.2), recruited through the Waterloo Research in Aging Participant Pool (WRAP). The control

group was chosen to generally match the age range of two brain damaged group. We did not control for gender. There were 11 (see Table 1) brain-damaged participants (five females and six males of average age of 60.8 and sd of 13.9 years): five without neglect (RBD – N), and six with signs of mild clinical neglect (RBD + N). The presence or absence of neglect was established by falling outside the normal range for any two of three neglect tasks from the Behavioral:behavioural Inattention Test (BIT) (see Table 1; further details below). Additionally, one chronic neglect participant (RR, 69 years old male) was recruited from the Neurological Patient Database (NPD) and was studied as a single case. All our patients had their stroke within the last five years, but we qualified RR as a chronic neglect patient because, contrary to the other patients who have shown fluctuations or improvement in their neglect, RR has been regularly tested since his stroke in 2008 and has shown consistent impairment on diverse tests of neglect.

## 2.2. Brain lesions

The majority of participants from the two brain damaged groups had lesions of the right cerebral hemisphere (see Table 1). Four of the participants had a lesion of the right basal ganglia, three had a lesion of the right parietal lobe, one had a lesion of the right temporal lobe. There was also one participant with a right parietal–occipital lesion, two participants with a right fronto-parietal lesion and finally one participant had a lesion of the left thalamic region. The chronic neglect participant suffered an embolic stroke in the right fronto-parietal subcortical deep white matter, as well as right parietal cortex and right basal ganglia.

## 2.3. Neglect assessment

To determine if participants manifested signs of clinical neglect or a possible neurological deficit, they all performed three subtests of the BIT (Wilson, Cockburn, & Halligan, 1987): line bisection, figure copying and letter cancellation. Based on the convention for the BIT test, performance was classified as abnormal when participants had a mean deviation of more than 5% from the center of the lines for the lines bisection test, omitted more than 10% of the “E” and “R” letters on the left side or failed to copy the left side of the star, cube and flower for the figure copying test. Following Halligan, Marshall, and Wade (1989), spatial neglect was determined to be present when participants fulfilled at least two of these diagnostic criteria.

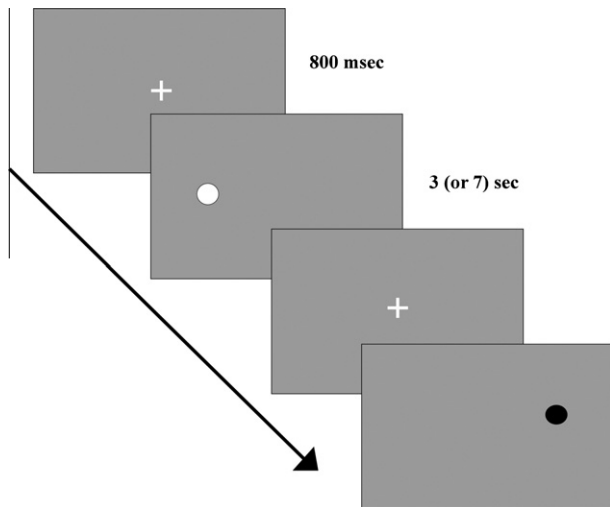
## 2.4. Experimental task

The experiment was created with the Python programming language, using PsychoPy, a psychophysics module for Python (Peirce, 2007). We used a 17 in. screen with a resolution of 1366 × 768. The computer background was grey (see Fig. 1). Each trial began with a white fixation cross at the center of the screen for 800 ms followed by a 300 ms blank (grey) screen before the stimulus was presented. The stimulus was a circle of 0.24° of visual angle that had 50% of chances to be either of a white or black color. The choice of the black or white color for the target was distributed randomly. Dots were presented on the screen for 3 s (or 7 s for the chronic neglect participant). Participants were asked to report the color of the target and they used a key press to indicate if the circle was white or black. They did so by pressing the left or right arrow on the computer keyboard, and because the color of the target was chosen randomly, the left or right key presses were equally likely for left and right-sided targets.

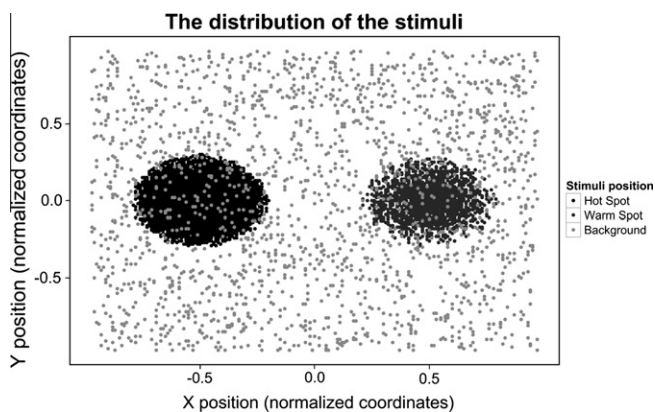
The primary manipulation was a non-uniform, cone shaped distribution (as previously described by Druker & Anderson,

**Table 1**  
12 Brain damaged participants: five without signs of spatial neglect, six with signs of mild clinical neglect and one participant with chronic neglect.

Participant	Sex	Group	Age	Handedness	Lesion	Line bisection (–mm for L and mm for R)	Mean deviation in % for line bisection (%)	Letter cancellation (L,R)	Shape copying
1. DM	M	BD	64	R	L thalamus	–17, –5, 1	7.6	20/20, 20/20	OK
2. DO	F	BD	67	R	R parietal	8, 3, –2	4.2	20/20, 20/20	OK
3. RS	F	BD	69	R	R basal ganglia	9, 7, 0	5.17	20/20, 19/20	OK
4. JH	M	BD	30	L	R parietal	4, 4, –5	4.2	20/20, 19/20	Cube different shape on the left side
5. GP	M	BD	54	R	R basal ganglia	–7, 0, –11	5.83	20/20, 18/20	OK
6. RB	M	BD – Neglect	82	R	R parietal occipital	1, 12, 9	7.12	20/20, 19/20	Cube shorter on the left
7. CP	M	BD_Neglect	49	R	R basal ganglia	–14, –20, 9	13.9	18/20, 17/20	Cube bottom part missing
8. BR	F	BD – Neglect	65	R	R temporal	28, –10, –18	18.19	9/20, 9/20	Cube left part missing
9. CB	F	BD – Neglect	70	L	R parietal	5, –3, –10	5.85	17/20, 18/20	Cube left side missing
10. DB	F	BD – Neglect	68	L	R fronto parietal	45, 14, –19	25.36	12/20, 14/20	Cube and flower left side missing
11. TC	M	BD – Neglect	51	R	R basal ganglia	4, 44, 55	33.3	14/20, 17/20	OK
12. RR	M	BD – Chronic Neglect	68	R	R fronto parietal	1st Session: 27, –4, –30 2nd Session: 32, 5, –22	19.80	0/20, 17/20	Cube and flower missing the left part
							19.18	7/20, 20/20	Star, cube and flower missing the left part



**Fig. 1.** Experimental design. Participants saw a white fixation cross for 800 ms, then the fixation cross disappeared and a black or white dot appeared for 3 or 7 s. Participants had to push the right arrow on a computer keyboard if the dot on the screen was black, and the left arrow if the dot was white.



**Fig. 2.** The distribution of the stimuli. The dots were biased to appear 75% in the hot spot (black), 12.5% of the time in the warm spot (dark grey) and 12.5% for the background, all over the screen (grey).

2010) of target locations. 75% of the time targets came from a distribution centered 6.9° visual angle to the left, which we called the “hot spot” (see Fig. 2). 12.5% of the time target location was selected from the same distribution but mirrored, so that it was located on the participant’s right side, centered 6.9° visual angle to the right, which we called the “warm spot”. Finally, 12.5% of the time, the target location was selected from the uniform distribution, called “background”.

Participants sat approximately 70 cm away from the computer screen. All trials were completed in a single session with the exception of the chronic neglect participant. There were four blocks of 200 trials. Participants were instructed to complete trials as fast and accurately as possible.

The chronic neglect participant, RR, participated in three different sessions on three different days. For the first session, RR failed to complete most trials because the three-second response window was too brief, and trials frequently timed out. Although he was presented with the full number of trials, response data were generally lacking and could not be evaluated. For the second testing session, RR completed three blocks of 200 trials and had 7 s to respond and for the third session, he completed two blocks of 200

trials and one block of 81 trials, as he was too tired to complete the 200 trials forming the full block and he needed a break.

## 2.5. Statistics

The primary analytic method was linear mixed effect modeling (Baayen, 2008). In spirit, the method is similar to conventional analysis of variance (ANOVA); one examines for linear relations between independent variables and a dependent variable. However, for the linear mixed model there are both fixed effects, similar to the factors of a conventional ANOVA, and random effects, which in our case includes the participants, as they represent a random sample from the pool of all potential participants. With the mixed effect models, the standard deviation and coefficients are estimated for the random and the fixed effects, respectively (Baayen, 2008). The use of mixed models increases the power of the analysis and reduces the inter-subject variability unrelated to group assignments. This method is increasingly being used in psychological studies. For example, Marangolo and colleagues (2010) used this analysis approach in their study of brain damaged participants to overcome small sample size limitations. Baayen (2008) provides an excellent tutorial review, and software packages are available. We used R, a statistical program (version 2.13.2, R Development Core Team, 2011) with the lme4 and the languageR packages (version 0.999375-42 and version 1.2). The last package contains the Markov chain Monte Carlo (MCMC) analysis, which reports the *p*-values and confidence intervals of the *t*-statistic (Baayen, 2008).

## 3. Results

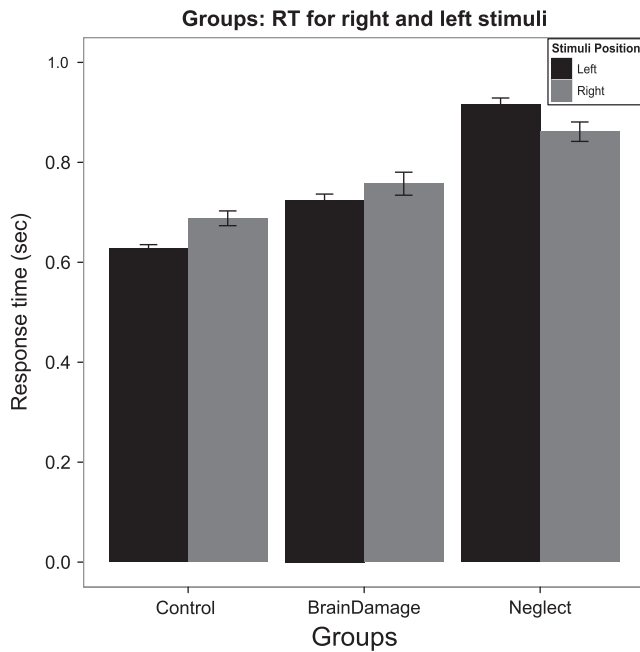
Healthy controls performed the computer-based task with a small number of errors; 1.4% of responses were incorrect and 0.2% were no-answers (NAs). The group of brain damaged participants without neglect had 2.7% errors and 0.52% of NAs. The group of neglect participants had 2.52% errors and 4.6% of NAs. Finally, RR, the chronic neglect participant made 8.1% errors and missed 67% of the targets when the display timed out after 3 s. For the second session, RR made 3.1% errors and 4.7% NAs, and 0.83% errors and 5.4% NAs for the third session. For RT analyses, we used only correct trials. We eliminated NAs and anticipations (trials faster than 0.1 s). Given the high number of NAs for the first session for the chronic neglect participant, only the data from the second and the third session were analyzed.

### 3.1. How different are the RTs of the three groups on the left and right sides?

We analyzed the RTs of the three groups by isolating stimuli that were presented on the left side of the screen or on the right side. For each group, participants were considered as a random effect and the side of the screen was analyzed as a fixed effect. Controls and RBD – N participants were faster for left-sided stimuli, contrary to RBD + N participants that showed the opposite effect, consistent with their clinical condition.

Demonstrating a sensitivity for the main manipulation of the experiment, the group of healthy controls was 60.3 ms faster for targets presented on the high probability left side of the screen compared to the targets on the right side ( $p = 0.0001$ , 95% confidence intervals (CI) 44–76 ms). The RBD – N participants were also 35 ms faster for stimuli on the left ( $p = 0.0006$ , 95% CI 14–54). RBD + N participants demonstrated a different effect, as they were 66.3 ms slower for stimuli on the left side (see Fig. 3), which represents their contra-lesional side, compared to the trials on the right side ( $p = 0.0001$ , 95% CI 42–90).





**Fig. 3.** RT based on the position of the stimuli for each group. Healthy control participants and brain damaged without neglect are faster for targets presented on the left side of the screen, where stimuli were biased to appear, contrary to neglect participants, who are faster for targets presented on their ipsilesional side. Error bars represent 95% confidence intervals.

### 3.2. Priming effects

We isolated consecutive trials that were either of the same color or that had been located near each other (defined as within 5° of visual angle). All three groups showed significant priming effects for color repeats. The results reveal (Fig. 4a) that the healthy

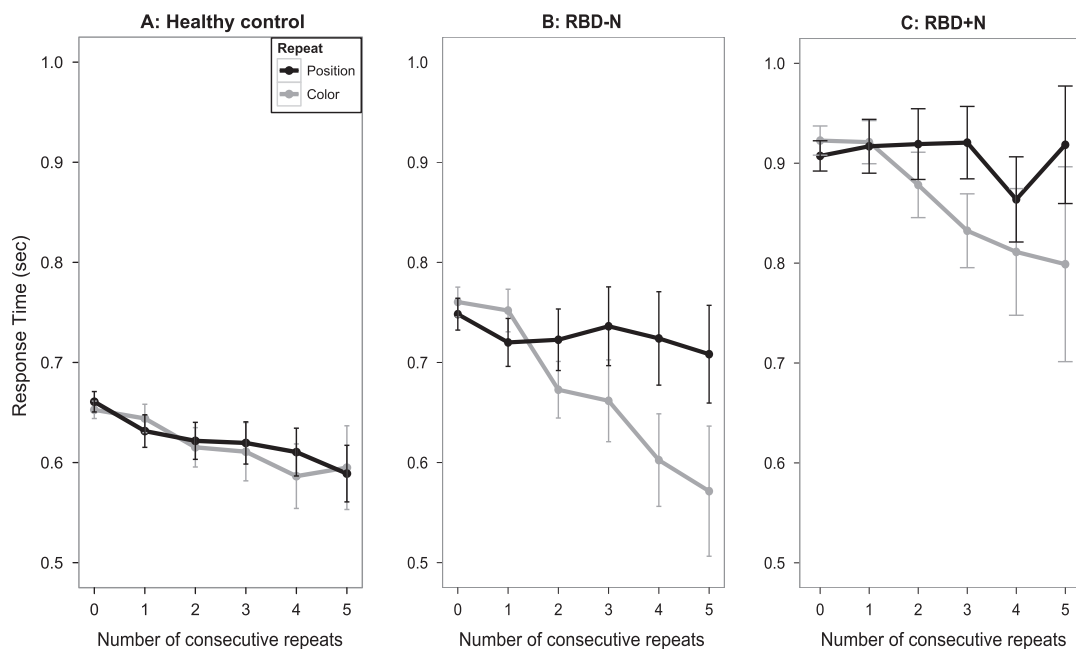
control group was 28.8 ms faster to detect consecutive targets of the same color ( $p = 0.0001$ , 95% CI 16–41 ms). The two groups of brain damaged participants also demonstrated preserved color priming. The group RBD – N was 47 ms faster ( $p = 0.0001$ , 95% CI 31–63 ms) and RBD + N participants were 29 ms quicker ( $p = 0.002$ , 95% CI 10–49 ms) when the color of the stimulus was repeated.

Controls and RBD – N showed significant decreases in RT when trials were repeated in the same location. The control group was 36.5 ms faster ( $p = 0.0001$ , 95% CI 24–49 ms) and RBD – N was 26.8 ms quicker ( $p = 0.0006$ , 95% CI 10–42 ms) for targets repeated at the same region. However, this was not true for RBD + N, which failed to show positional priming (they were 5 ms faster when the target repeated location but  $p = 0.56$ ).

To assess if this depended on the side of the screen, we repeated the analysis separately for hot-spot trials (left side of the screen) and warm-spot trials (right side of the screen). Within neither area was there a significant positional priming effect for the RBD + N, but on the left, there was a trend towards significance (24 ms faster,  $p = 0.075$ ). These negative results are in contrast to those for color priming, which is present in regions of the display that do not show position priming. For example, when restricted to targets in the high probability region on the their left side, RBD + N were 36 ms faster when a hot spot trial was preceded by a similarly colored trial than when it was not ( $p = 0.001$ , 95% CI 13–58 ms).

As can be seen in Fig. 4a–c, the effect of increasing the number of times a stimulus is repeated strengthens the priming effect, but there are differences between the groups. Here “repeat number” refers to the number of consecutive repetitions. For example, if the target color sequence were White–Black–Black–Black this trial would have a repeat count of 3 for color.

All three groups show an increased priming effect as the number of consecutive color repeats increases, at least out to five trials. For positional priming, the effect for RBD – N subjects is attenuated and the effect is carried by trials with multiple repeats and not just a single, isolated repetition, as appears the case for



**Fig. 4.** (A–C) Priming effect for the position and the color. (A). Healthy control participants improved their RT for more than 50 ms when the same color or the same position was repeated 1–5 times. (B). Brain damaged participants without neglect demonstrated a higher improvement of their RT when the color of the target was repeated 1–5 times compared to when the position was repeated. (C). Neglect participants, who are slower overall compared to the two other groups, also demonstrate a better improvement of their RT when the color of the target is repeated 1–5 times, but they do not have a significant improvement for the repetition of the same location. Error bars represent 95% confidence intervals.

controls. This is more pronounced for RBD + N subjects who do not show any statistically significant position priming with an increased number of consecutive positional repeats (for one repeat compared to no repeats ( $p = 0.35$ ); for two repeats compared to one repeat ( $p = 0.8$ )).

### 3.3. Distribution learning

To investigate if participants learned the probability distribution of the targets separately from the effect of positional priming, we evaluated how the RT for particular screen locations evolved across blocks. We also repeated analyses for the subset of trials that were not repeats (i.e. did not match the prior trial for color or location).

The linear mixed model analysis for the healthy control group showed a significant effect on RT for stimuli presented on the hot spot area; participants were 42.3 ms faster overall to detect targets located on the high probability region ( $p = 0.002$ , 95% CI 14–72 ms) compared to targets falling in the mirrored image warm spot.

To further evaluate if participants were learning the probability distribution, we examined if RT decreased across blocks for trials falling in the high probability hot spot as compared to other screen regions.

The results showed that healthy control participants very rapidly learned to favor the high probability region within the first block. By the second block, trials located in the hot spot were detected 70 ms faster than in the first block ( $p = 0.005$ , 95% CI 23–120 ms) while improvement for the less probable warm spot had not significantly changed (18 ms faster;  $p = 0.5$ ). Control participants were 91 ms faster during the third block ( $p = 0.0001$ , 95% CI 43–142 ms) and 93 ms in the fourth block ( $p = 0.0001$ , 95% CI 46–140 ms) when comparing trials falling in the high probability region to similar trials in the first block.

Contrary to the hot spot area, control participants did not show a significant improvement of their RT for the warm spot during the third block (73 ms slower but  $p = 0.8$ ) or the fourth block (57 ms faster but  $p = 0.07$ ) compared to the first block.

We conducted the same analysis for RBD – N. This group revealed a faster RT for trials presented at the high probability region (51.4 ms faster for the hot spot area compared to the warm spot;  $p = 0.02$ , 95% CI 6–90 ms). We also compared their RT across the blocks and found that RBD–N participants were 136 ms faster ( $p = 0.01$ , 95% CI 38–243 ms) to detect targets during the fourth block compared to the first block for the warm spot region. The trend was similar, but not statistically significant for the hot spot area (53 ms faster;  $p = 0.12$ , 95% CI 13–122 ms). In summary, for RBD–N participants the improvement in response times across blocks was greater for the high probability regions of the screen than the lower probability background region.

RBD + N participants demonstrated a different pattern. There was a suggestion that some degree of statistical learning may be intact, as the RBD + N group was 93 ms faster for non-repeated trials presented on the warm spot (which is their ipsilesional side;  $p = 0.0001$ , 95% CI 50–136 ms). There was no change for trials in the hot spot region. While their neglect makes RBD + N participants slower to report left sided targets, it might still be the case that they are sensitive to the non-uniform spatial distribution of targets within their impaired hemi-space. To assess for local effects, we analyzed trials falling within the hot spot (or warm spot) and compared them with trials from background region on the same side only. We did this for all three groups.

The controls were 51.7 ms faster for left sided hot spot trials compared to left-sided background trials ( $p = 0.0002$ , 95% CI 26–77 ms). And although only 12.5% of the targets were presented on the warm spot, the group of older healthy control was also

31.5 ms faster for those targets compared to right-sided background trials ( $p = 0.02$ , 95% 5–65 ms).

The group of RBD – N participants were also sensitive to the probability distribution, but not to the same extent as healthy participants. The RT of RBD – N participants for trials falling in the hot spot was numerically slightly faster than the other trials presented on the background on the left, but this difference was not statistically significant ( $p = 0.69$ ). As RBD–N participants were substantially faster for left sided targets in general (Fig. 3), the two results interpreted together suggest a possible decrease in spatial precision.

Interestingly, RBD + N participants did show that hot spot trials were faster than other left sided trials (65.5 ms;  $p = 0.0008$ ; 95% CI 25–106 ms), even though these participants were slower on the left side overall (which is after all their contralesional side). This leaves open the possibility that neglect subjects may still be partially sensitive to probability structure. This result motivated us to examine these effects in a patient with more severe neglect where we could acquire a larger number of trials.

### 3.4. Chronic neglect

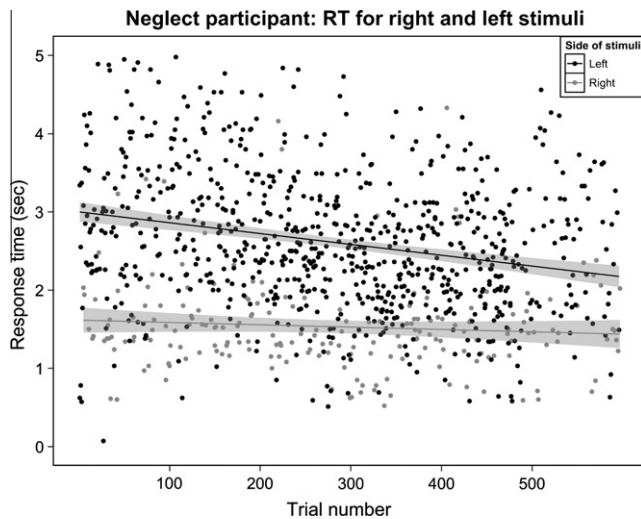
As this was a single participant, analyses were conventional ANOVAs. Similar, to our group of RBD + N participants the chronic neglect participant did not show any detectable location priming. During the second session (first session trials were not evaluable due to the high number of NAs), RR was 313 ms slower when the position of the target was repeated within 5 degrees visual angle of the prior trial; this was significantly different from trials that were not repeated at the same location ( $F(1,534) = 11.41$ ,  $p = 0.0007$ ). But again, like the RBD + N group, RR did benefit from color priming. He was 297 ms faster when the color of the stimulus was repeated ( $F(1,534) = 10.29$ ,  $p = 0.001$ ).

For the third session, RR again failed to demonstrate any benefit from spatial repetitions: he was 436 ms slower for targets that were repeated at the same position ( $F(1,441) = 24.52$ ,  $p < 0.0001$ ), but showed that he was faster when color primed, (150 ms,  $F(1,441) = 2.8$ ,  $p = 0.09$ ).

To assess for RR's ability to learn the spatial probability distribution we conducted a conventional ANOVA with location and block factors. For the second session, RR had a significant RT effect for the target distribution factor ( $F(2,527) = 39.90$ ,  $p < 0.001$ ) and for the blocks ( $F(2,527) = 20.8$ ,  $p < 0.001$ ). RR also showed a target distribution by block interaction ( $F(4,527) = 4.34$ ,  $p = 0.001$ ).

For the third session, although he did only 81 trials during the second block because of fatigue effects, RR again demonstrated an effect for target distribution factor ( $F(2,434) = 95.95$ ,  $p < 0.001$ ), and for block ( $F(2,434) = 23.87$ ,  $p < 0.001$ ), as well as a significant interaction between the two ( $F(4,434) = 2.42$ ,  $p = 0.04$ ). This result is not affected by the fewer trials of the second block, as the final results of this session are not affected even if we disregard this block (indeed, without the second block, RR would still show an effect of block  $F(1,360) = 33.83$ ,  $p < 0.0001$ , an effect of target distribution  $F(2,360) = 80.14$ ,  $p < 0.0001$  and an interaction between the two  $F(2,360) = 4.7$ ,  $p = 0.009$ ).

Since there was a consistent pattern of results across the two sessions, we collapsed across sessions and assessed how RR was learning the spatial distribution of targets for left and right sides by conducting an ANCOVA with hemispace as a factor and trial number as the co-variate. If RR is learning something about the probability of targets per se, as opposed to just learning the task in general, we would predict a greater effect of experience on his RT for targets falling on the left side, despite left sided trials being slower overall (consistent with his neglect;  $F(1,977) = 246.56$ ,  $p < 0.0001$ ).



**Fig. 5.** A scatterplot of RT by trial. The RT is in seconds and the x-axis is trial number. General linear hypotheses test for the RT based on the trial number and side shows a significant effect of side, which revealed that there was a difference in the RT for the left and the right sides over the sessions. RT improved for responses on the left side over the sessions, while the RT remained the same for responses on the right side.

To illustrate this relationship in more detail, we performed a general linear hypotheses test for the RT based on the trial number and the side (Fig. 5). We found a significant effect of the side ( $t = -3.28$ ,  $p = 0.001$ ), demonstrating a difference in the improvement in RT for the left side compared to the right side over trials. In summary, the greatest gain from experience for RR was in his neglected hemispace: the hemispace toward which we had systematically biased the target location probability.

#### 4. Discussion

In this experiment we studied color priming, spatial priming, and the learning of spatial probability distribution in Controls, RBD-N, and RBD+N. Our goal was to investigate how priming and statistical distributions can be used as cues to direct attention. The design of our paradigm allowed us to consider these two functions separately. Sequential trials that were of the same color or in the same position allowed us to assess priming effects, and by eliminating such trials and comparing regions where the target was biased to appear, we could assess for the ability to learn the probability distribution of target locations. To some extent this approach meant we were able to test what Fecteau and Munoz (2006) called the priority map, which is a combination of relevance – formed by top-down spatial expectations driven by the process of statistical learning – and of salience, which in our experiment could be represented by a bottom-up basis for the priming effects. Finally, because two of our groups suffered damage in the RH, an impairment in attentional functions would highlight the involvement of the RH in priming and statistical learning.

A simple scenario for the development of spatial top-down attentional effects is that statistical inhomogeneity lead to spatial repeats. This short time repetition leads to priming, and from an accumulation of priming effects a higher order map of target probabilities is constructed and biases spatial expectancy. To explore the implications of our data for this simple model, we consider in turn our priming and statistical results from the different participant groups.

##### 4.1. Priming effects

Our definition of priming is based on that of Maljkovic and Nakayama (1994, 1996). We tested if our participants were faster when the target color or position repeated. All our participant groups; the healthy controls and the RBD participants with and without neglect, showed a sensitivity to repeating target characteristics on a short time scale. All groups showed a robust color priming effect, demonstrating that priming per se is not affected by either brain injury or neglect.

Only a few researchers have investigated priming in brain damaged participants before (for example Kristjánsson, Vuilleumier, Husain, Macaluso, & Driver, 2004; Kristjánsson et al., 2005), and those studies have also shown a preserved color priming effect. Furthermore, Saevarsson, Joelsdottir, Hjaltason, and Kristjánsson (2008) found that priming was preserved in the contralesional side in neglect patients when distractor sets were repeated; the authors highlight that repetition improved search ability in the neglected side.

While we did not find an impairment for priming per se, we did find impairments for spatial priming. This was partial in our RBD – N group, which seemed to require more than one repeat to benefit from spatial repetition, and was complete in our RBD + N participants, who showed no spatial priming. These data are at some variance with the results of Kristjánsson et al. (2005) who did find intact spatial priming. The differences in experimental procedures may well account for the different findings. In their study, there were only three relevant locations and each location was always occupied by a relevant token. In our task, the location of targets was unpredictable and varied widely from trial to trial. Of note, Kristjánsson et al. (2005) failed to find position priming when shortening the display time and preventing overt recognition of the target. Thus, of the two forms of priming, color and position, Kristjánsson et al. (2005) also found position priming to be the weaker. While the RBD – N and RBD + N differed on measures of position priming, they did not show obvious differences in the brain regions injured. Thus, the absence of a position priming effect correlates better with the presence of persistent attentional impairment in the form of spatial neglect than it does with any one brain region.

##### 4.2. Statistical learning

Our paradigm was also designed to test for probability learning separate from position priming effects. We did this by having a large number of potential target positions and a large number of trials. This meant that even after we excluded trials that were spatial repeats, we still had sufficient numbers of trials for statistical analysis. Because all screen locations were not equally probable, these remaining trials differed in their spatial probability. We chose 5° of visual angle as the cut off for spatial repetition because of evidence that position priming effects are gradual (Maljkovic & Nakayama, 1996).

Considering first the older controls, they replicate our earlier work in young healthy controls (Druker & Anderson, 2010). Our older healthy participants prime for color and position, and when eliminating such trials, show performance benefits across blocks that are greater in the high probability regions of the screen. In addition, these participants are faster for screen regions of high probability than are their responses to ipsilateral, lower probability, locations.

Is this benefit secondary to shifts in gaze? Although we instructed participants to maintain central fixation, we did not train them nor measure their eye position. So, we cannot exclude this as a *mechanism* whereby learning statistical information contributes to improved attentional processing, since attention and gaze

generally correlate (Corbetta, 1998). However, we emphasize that while such a shift, if present, would be a mechanism for improved response speed, it would not explain away that fact that statistical learning does take place, spontaneously, without specific instruction, and when participants are generally not overtly aware of the target distribution (Druker & Anderson, 2010). Lastly, eye position biases cannot be a complete mechanism, since our older healthy participants were simultaneously faster for both the right and left sided high probability locations than for ipsilateral lower probability screen locations.

We began our discussion by suggesting a simple sequence for the development of spatial attentional expectancies: spatial repeats are associated with positional priming and this leads to the development of an attentional bias. Our data however, are more ambiguous. For example, while our RBD – N participants were faster for trials presented on the left higher probability side of the screen, they were not faster in the left-side hotspot compared to other left sided locations, and they showed an impoverished position priming effect that appeared to require multiple sequential repeats. On the other hand, our RBD + N participants showed no position priming, but still demonstrated some retained ability to learn to a partial degree how likely targets were to appear in different regions of the computer screen.

The RBD + N patients were faster for targets presented in the hot spot compared to other left sided target locations. And our single patient showed a retained ability to improve his response more in the high probability, left side of the screen, despite being slower overall on this side, and also having no position priming effect.

Our finding of a retained ability for probability learning in people with spatial neglect is in accordance with earlier work by Geng and Behrmann (2002, 2006). The authors found that neglect patients were able to learn the statistical distribution of the position of the target on their contralesional side and use it as a cue to direct their attention and improve their performances. Because there were relatively few target locations in those studies, the risk that the effect was being mediated by position priming was suggested (Walshaw & Gilchrist, 2006). But in light of our retained evidence for probability learning and the absence of evidence for position priming, the most parsimonious explanation for all the data is that patients with spatial neglect retain an ability to learn target probabilities and that this retained ability is not simply a consequence of position priming, contrary to our initial hypothesis. Further, while our data demonstrate a relationship among RBD, attention, and statistical learning, the nature of that relationship requires further investigation. A central question is whether RH systems for spatial attention and statistical learning overlap or are distinct.

Given our demonstration of some preserved statistical learning in the contralesional hemisphere for RBD + N participants, it is possible that their attentional impairments simply act as a mask on their retained statistical learning abilities. Determining whether the statistical learning impairments are primary or secondary could have practical implications for rehabilitation.

One-way forward on this question will be to look at performance with non-lateralized stimuli, to see how priming and statistical learning are affected for items near fixation. A second approach would be to combine the present methods with eye tracking. Many RBD + N patients are known to have difficulty making contralesional exploratory saccades (Girotti, Casazza, Musicco, & Avanzini, 1983). If the way control participants benefit from knowing the statistics of spatial distributions is through facilitating exploratory eye movements, then this could explain the impairment of RBD + N subjects without requiring a primary impairment in statistical learning. Answers to these questions will require further experiments.

In summary, the analyses we conducted for statistical learning show varying results for the ability of RBD, with or without neglect,

to learn spatial statistical distribution and to use that as a cue to direct their attention. While the RBD – N participants were faster on the high probability side, they did not show an improvement confined to the highest probability area on that side. The RBD + N despite being slower on the left, did show an advantage for the hotspot region. These results are ambivalent and there is a risk they reflect chance variation or are a consequence of the size of our brain damaged subject pools.

Despite our small group size, our results contribute to the literature on priming effects after brain injury. We find a category impairment for spatial but not color priming. More importantly, we do find evidence of a retained ability to learn statistical features of the environment after RH injury. This is interesting in light of recent results of the RH's importance for statistical learning. This has been demonstrated in previous studies by Danckert et al. (2011), Roser et al. (2011), and Turk-Browne, Scholl, Chun, and Johnson (2009). If, as our data and these other studies suggest, similar RH systems contribute to the learning of spatial probability and positional salience, then efforts to rehabilitate neglect could make spatial statistical learning a therapeutic target.

## Acknowledgments

We thank our patients for participating in this study. We also would like to thank Deltcho Valtchanov and Brilé Anderson for their comments on an earlier version of this manuscript.

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