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Monocular channels have a functional role in phasic alertness and temporal expectancy

William Saban[1] · Noam Weinbach[2] · Shai Gabay[1]

Abstract

The literature has long emphasized the neocortex's role in the tangled phasic-alertness and temporal-expectancy processes. In this work, we examined whether subcortical, monocular mechanisms have a functional role in these processes. This was done by assessing phasic alertness and temporal expectancy independently using a cue–target eye-of-origin manipulation. Participants performed target detection tasks in which a central cue and its ensuing peripheral target were each presented either to the same eye or to a different eye. In Experiment 1, phasic alertness, independent of temporal expectancy, was manipulated by presenting an alerting cue prior to the target presentation. The alerting effect elicited by the cue lasted for a longer duration when the cue and target were presented to the same eye than when they were presented to different eyes, indicating the involvement of subcortical regions in phasic alertness. In Experiment 2, the cue's temporal predictability regarding the target's onset time was manipulated by changing the cue–target interval's foreperiod distribution. A modulation in temporal expectancy was found when both the cue and the target were presented to the same eye, demonstrating the importance of subcortical mechanisms in temporal expectancy. Together, the results demonstrate that monocular channels are functionally involved in both phasic alertness and temporal expectancy. This study suggests that both phasic alertness and temporal expectancy are functionally dependent on monocular channels of the visual stream, and highlights the importance of direct examination of primitive, subcortical regions in higher cognitive functioning (e.g., temporal expectancy).

Keywords Attention · Cortex · Temporal expectancy · Phasic alertness · Subcortical regions

Performance improves after a preceding signal. This simple effect was already well scrutinized in the 19th century, as Wundt (1887) demonstrated that the human response time (RT) to a target is facilitated by prior presentation of a cue. Although the neural basis of the cognitive processes that facilitate behavior following a cue is enjoying a research boom (for a review, see Nobre, Correa, & Coull, 2007), a clear, well-defined taxonomy is still needed. Such processes may reflect benefits caused by phasic alertness (i.e., an increase in arousal level), but also may involve increased temporal expectancy (i.e., orienting attention

to a specific moment in time; Weinbach & Henik, 2012). The literature has long emphasized the role of cortical mechanisms in these cognitive abilities (Bueti, Bahrami, & Walsh, 2008; Coull & Nobre, 1998; Coull, Frith, Büchel, & Nobre, 2000; Coull, Vidal, Nazarian, & Macar, 2004; Genovesio, Tsujimoto, & Wise, 2006; Ghose & Maunsell, 2002; Janssen & Shadlen, 2005; Leon & Shadlen, 2003; Onoe et al., 2001; Périn, Godefroy, Fall, & De Marco, 2010; Posner, 1988; Sturm & Willmes, 2001; Stuss and Alexander, 2005; Vallesi, McIntosh, Shallice, & Stuss, 2009; Vallesi & Shallice, 2007; Vallesi, Shallice, & Walsh, 2007; Yanaka, Saito, Uchiyama, & Sadato, 2010); however, is it possible that subcortical regions may also play a functional role in phasic alertness and in temporal expectancy, when they are examined separately?

Phasic alertness refers to a state of increased arousal that is elicited by an external event and has been suggested to regulate the intensity of attention to stimuli (Posner & Petersen, 1990). A typically used manipulation to induce this state of phasic alertness is to present an alerting cue (e.g., an auditory sound or a visual event) prior to the presentation of a target and to compare the averaged RT in this condition to that in a

✉ William Saban
williamsaban@gmail.com

Shai Gabay
shaigaba@gmail.com

[1] Department of Psychology and the Institute of Information Processing and Decision Making (IIPDM), University of Haifa, Haifa, Israel

[2] Department of Psychology, University of Haifa, Haifa, Israel

no-cue condition. Typically, alertness reaches the optimal level when the cue–target interval is 500 ms (Posner & Boies, 1971), but this effect may last up to 900 ms or longer after the cue (e.g., Weinbach & Henik, 2013; Yoshida et al., 2013).

One difficulty in using alerting cues is that besides alertness, they may also elicit temporal expectancy, and therefore help participants prepare for the temporal onset of the target, irrespective of the increase in arousal level. To eliminate temporal expectancy following an alerting cue, it is possible to use multiple cue–target intervals (the stimulus onset asynchrony; henceforth, SOA) and to employ a nonaging foreperiod distribution of SOAs (e.g., Niemi & Näätänen, 1981; Weinbach & Henik, 2013; the exact method will be further discussed below). A nonaging foreperiod distribution ensures an equal probability of the target's appearance at any time point after the cue. In this way, participants cannot use the cue to predict the temporal onset of the target. Previous studies have shown that the alerting effect (i.e., faster RTs following an alerting cue than following a no-cue condition) is evident even when a nonaging foreperiod distribution is used (Weinbach & Henik, 2013; Whitehead, 1991), encapsulating a "pure" influence of phasic alertness level, voided of temporal expectancy.

A hallmark of temporal expectancy is the presence of a foreperiod effect (Niemi & Näätänen, 1981). The foreperiod effect is characterized by a decrease in RTs as the cue–target interval increases. The foreperiod effect demonstrates the participant's growing anticipation of the target's appearance over time. This phenomenon is due to the fact that the probability of target appearance increases as time passes following the appearance of the cue. Temporal expectancy can be manipulated by employing three foreperiod distributions: (1) an *aging* distribution, in which there is an equal number of trials for each SOA, and as time passes from the appearance of the cue, the probability of target presentation increases—hence, the cue provides temporal information regarding the target onset time; (2) a *nonaging* foreperiod distribution, as we previously described, in which there is a constant probability of target appearance in each SOA, and the cue does not provide any temporal information regarding target appearance (e.g., Niemi & Näätänen, 1981); and (3) an *accelerated-aging* distribution, in which the distribution of SOAs is the opposite of that found in the nonaging distribution (Baumeister & Joubert, 1969; Gabay & Henik, 2008, 2010), and in which the cue provides the most temporal information regarding target appearance (see Table 3 below for the number of trials and predictability of target appearance as a function of each foreperiod distribution in our Exp. 2). Therefore, despite the similar levels of phasic alertness that the cue elicits in all three foreperiod distributions, only in the accelerated-aging and aging distributions do the cues also provide temporal information about the target's time of appearance. Hence, a comparison between the nonaging distribution (which does not provide any temporal information) and the aging and accelerated-

aging distributions (which do provide temporal information) can be used as an index for measuring the influence of temporal expectancy without the effect of phasic alertness.

The neural substrates of phasic alertness and temporal expectancy

Since most studies do not apply methods to dissociate between phasic alertness and temporal expectancy, finding a clear, well-defined dissociation between them is somewhat challenging (Weinbach & Henik, 2012). Studies that have examined phasic alertness have suggested the involvement of mostly cortical regions (e.g., Coull et al., 2000; Périn et al., 2010; Sturm & Willmes, 2001; Yanaka, Saito, Uchiyama, & Sadato, 2010). In addition, several studies have also demonstrated the involvement of subcortical regions in phasic alertness, such as the locus coeruleus (LC; Coull, Frith, Frackowiak, & Grasby, 1996; Fan, McCandliss, Fossella, Flombaum, & Posner, 2005; for a review of superior colliculus [SC] involvement, see Sara, 2009). To summarize, the literature reflects that phasic alertness is encoded not by a single area, but by a wide subcortical–cortical network.

Temporal expectancy studies have emphasized the involvement of cortical regions such as fronto-parietal areas, in both humans and monkeys (Genovesio et al., 2006; Ghose & Maunsell, 2002; Janssen & Shadlen, 2005; Leon & Shadlen, 2003; Onoe et al., 2001; Stuss & Alexander, 2005; Stuss et al., 2005; Vallesi et al., 2009; Vallesi & Shallice, 2007; Vallesi et al., 2007). Patient studies have also demonstrated that lesions to the prefrontal cortex reduce the ability to show an RT benefit at long SOAs (Stuss et al., 2005; Triviño, Correa, Arnedo, & Lupiáñez, 2010; Vallesi et al., 2007), which has been suggested to reflect temporal expectancy. Correspondingly, a transcranial magnetic stimulation (TMS) study also confirmed the role of the prefrontal cortex in temporal expectancy among healthy individuals (Vallesi et al., 2007). In recent functional magnetic resonance imaging (fMRI) and TMS studies, researchers have also found evidence for the involvement of early cortical regions of the visual stream (in contrast to the prefrontal cortex). In particular, they found involvement of striate and extrastriate areas such as V1, V2, and V3 in temporal coding processes (Bueti, Bahrami, Walsh, & Rees, 2010; Salvioni, Murray, Kalmbach, & Bueti, 2013). Finally, the brain's norepinephrine (NE) system arises in the LC of the midbrain, and several studies have demonstrated that the effect of an alerting cue is reduced, or even eliminated, by norepinephrine antagonists (Coull et al., 1996; Marrocco & Davidson, 1998). Importantly, the LC–NE system includes major nodes in the fronto-parietal regions (e.g., Morrison & Foote, 1986; Sara, 2009). Hence, it is possible that the LC (subcortical region) distribution of NE to cortical regions may generate the activity

of a subcortical–cortical network not only in phasic alertness, but also in temporal expectancy.

As we mentioned, studies examining the neural substrates of temporal expectancy have focused mainly on cortical networks—somewhat neglecting lower structures of the visual stream. Because subcortical regions are involved in phasic alertness, an outstanding question is whether subcortical mechanisms do more than just channel information. In particular, do they also have a functional role in temporal expectancy? On theoretical grounds, it is reasonable to assume that subcortical regions might also be involved in temporal expectancy. For example, evolutionarily older species that lack a cortex (e.g., fish) often face the need to prepare for and temporally predict events in their surroundings in order to optimize foraging and avoid predators. Hence, temporal expectancy is crucial for their survival, and in those species temporal expectancy most likely involves subcortical regions. In a recent study, it was demonstrated that the archer fish, which does not have cortical structures, has the ability to orient attention endogenously (Saban, Sekely, Klein, & Gabay, 2017) and can predict a target's location. In addition, by using a sensitive behavioral manipulation, researchers have accumulated evidence suggesting that subcortical structures have a functional role in diverse cognitive processes, such as orienting attention, face recognition, and even executive functions (Batson, Beer, Seitz, & Watanabe, 2011; Gabay & Behrmann, 2014; Gabay, Burlingham, & Behrmann, 2014a; Gabay, Nestor, Dundas, & Behrmann, 2014b; Karni & Sagi, 1991; Saban, Gabay, & Kalanthroff, 2018a, b, c; Self & Roelfsema, 2010). In this study, using monocular/dichoptic presentation, we examined whether phasic alertness and temporal expectancy might be functionally dependent on subcortical regions.

How to behaviorally probe the functional role of subcortical structures

Visual input is propagated monocularly until it reaches striate (V1) and extrastriate regions (Horton, Dagi, McCrane, & de Monasterio, 1990; Menon, Ogawa, Strupp, & Uğurbil, 1997). Hence, cortical regions are mostly binocular and are insensitive to the visual information's eye of origin, whereas subcortical regions are mostly eye-dependent. By using a stereoscope, one can present different visual information to each eye separately, thereby dissociating the contributions of monocular (mostly subcortical) versus binocular (mostly cortical) visual channels in the cognitive process (Batson et al., 2011; Gabay & Behrmann, 2014; Gabay, Burlingham, & Behrmann, 2014a; Gabay, Nestor, et al., 2014b; Karni & Sagi, 1991; Saban, Gabay, & Kalanthroff, 2018a; Saban, Klein, & Gabay, 2018b; Saban, Sekely, Klein, & Gabay, 2018c; Self & Roelfsema, 2010). If subcortical regions or V1 are not involved in a cognitive process (e.g., if they only channel information to higher brain regions in the visual stream), then segregating the visual information to different eyes should not affect performance. For example, a recent study has demonstrated that endogenous orienting of attention to spatial locations occurs earlier when the same monocular channel is presented with both the cue and the target (Saban, Sekely, et al., 2018c). Importantly, this novel finding has highlighted the significance of studying the involvement of monocular channels in cognitive processes such as spatial attention. However, the literature has long demonstrated that spatial attention and temporal attention are two distinct cognitive processes, both behaviorally and neuroanatomically (e.g., Callejas, Lupiáñez, & Tudela, 2004; Fan et al., 2005; Petersen & Posner, 2012). Hence, the remaining outstanding question is whether monocular channels have a functional role in temporal attention, and specifically in phasic alertness and temporal expectancy.

To examine the involvement of subcortical structures in phasic alertness and temporal expectancy, we employed a target detection task in which a central cue was presented prior to the appearance of a peripheral target. Using a stereoscope, we manipulated the eye to which the cue and target were presented: In the different-eye condition, the cue and target were presented to different eyes, and in the same-eye condition, both were presented to the same eye. In the first experiment, a nonaging foreperiod distribution was implemented, in which a preceding cue manipulated phasic alertness without any involvement of temporal expectancy. In line with the above-mentioned literature, subcortical regions are involved in phasic alertness, and hence we would expect to find a larger or longer alerting effect in the same-eye condition (in which both cue and target are presented to lower monocular regions) than in the different-eye condition (in which the cue and target are presented to different monocular channels). In the second experiment, we implemented all three foreperiod distributions, such that a comparison of the foreperiod distributions would provide an index of temporal expectancy. This manipulation enabled us to examine the influence of temporal expectancy by comparing a temporally nonpredictive cue (nonaging condition) with a temporally predictive cue (aging or accelerated-aging conditions). According to the above-mentioned literature and the evolutionary theoretical perspective, the effect of temporal expectancy should be stronger or should be observed only in the same-eye condition (vs. the different-eye condition). This would indicate a functional role of subcortical visual channels in temporal expectancy.

Experiment 1

In this experiment we aimed to examine whether monocular channels contribute to phasic alertness. By using a nonaging foreperiod distribution (see Table 1) and manipulating the

alerting cue and target's eye of origin, the independent effect of phasic alertness on task performance was measured.

Method

Participants A total of 20 participants (mean age 24.51 years; 13 females, seven males) volunteered to participate in exchange for payment or course credit. All had normal or corrected-to-normal vision. A power analysis using G*Power 3.1 (Faul, Erdfelder, Lang, & Buchner, 2007) was conducted. The phasic alerting effect is known to be robust, producing very large effect sizes (e.g., Fan et al., 2005, $\eta_p^2 = .786$; Spagna et al., 2014, $\eta_p^2 = .80$; Yanaka et al., 2010, $\eta_p^2 = .639$). Hence, a large effect size was also expected in the present experiment $\left(\eta_p^2 = .14\right)$. The analysis showed that 18 participants would be required for a power = 80% with a Type I error rate of $\alpha = .05$. Therefore, the sample for the present experiment was sufficiently powered. The study was approved by the University of Haifa ethics committee.

Stimulus and apparatus Stimulus presentation was performed using a HP Z200 computer, operating with a Windows 7 system. The stimuli were displayed on a Samsung LCD monitor (model S24C650PL) with a resolution of 1,680 × 1,050. Responses were made using a DELL Hebrew–English Extended Keyboard (model RT7D50 SK-8115). The computer monitor was positioned 57 cm in front of a stereoscope (model ScreenScope LCD SA200LCD), blocking the participant's direct view of the monitor (see Fig. 1). The monitor presentation was divided into two halves (each half was presented to a different eye) and consisted of two rectangles (4.8° in width and 14.2° in height), placed 8.5° from the center of the screen and 16.7° from each other. Each rectangle contained three boxes (2.3° width and height) in a vertical alignment. The upper and lower boxes were placed 5.9° from the center of the rectangle, and the central box was placed in the rectangle's center. A central fixation cross composed of two lines (0.7° width and height) was placed in the central box. The target was preceded by a brightening of the central box, which was accomplished by widening the box's outline from 1 to 5 mm, and was presented to one eye. An asterisk
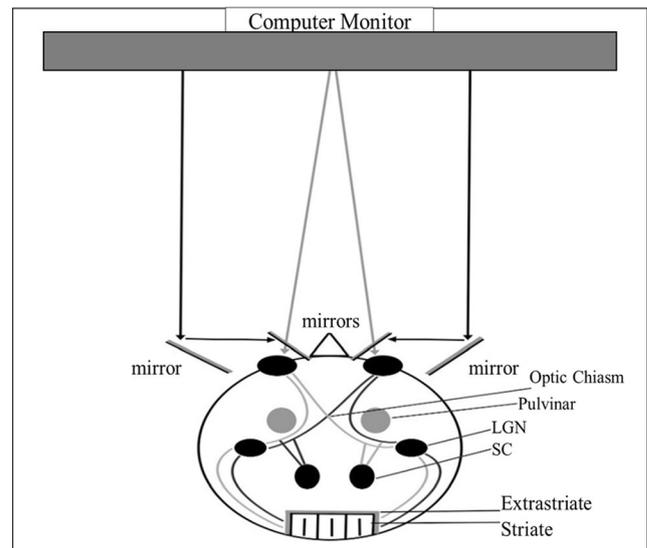
Fig. 1 Schematic illustration of the experimental apparatus and visual pathways from the eyes to the brain. Each side of the computer monitor provided visual information to a different eye. From the eye, the visual information passes first through monocularly segregated subcortical regions (gray curves, left eye; black curves, right eye). This information is then projected to the lateral geniculate nucleus (LGN) and subsequently reaches striate and binocular extrastriate regions

target (0.5° width and height), placed in one of the peripheral boxes, was then presented to one of the eyes. All stimuli were white figures against a black background.

Procedure A typical experimental trial is depicted in Fig. 2. Three vertically aligned boxes were presented within a larger rectangle throughout the experiment. Each trial began with a fixation cross appearing for 500 ms in the center of the central box. To manipulate phasic alertness, 200 ms after fixation disappeared the cue—a brightening of the central box outline—was presented for 100 ms. This alerting cue was present in 50% of the trials, and cue and no-cue trials were randomly intermixed (half of the no-cue trials were defined as same-eye, and the other half as different-eye). After a variable SOA of 500 or 1,000 ms, a white target asterisk appeared for 3,000 ms or until a response was detected. To eliminate temporal expectancy, we used the nonaging foreperiod distribution. As a result, at any time point during a trial, there was a constant probability that the target would appear in the upcoming SOA (Table 1 shows the number of trials and temporal predictability of target appearance in the nonaging foreperiod distribution). This was done by assigning more trials to the short SOA condition than to the longer SOA condition. The target could appear in the upper or lower boxes randomly and with equal probabilities. The cue and target were presented to the left or right eye randomly and with equal probabilities. Participants were requested to respond to the target's appearance by pressing the space bar

Table 1. Distribution of the number of trials and the probability of target appearance for each experimental condition (same-eye, different-eye) in the nonaging foreperiod distribution

SOA (ms)	Number of Trials [Probability of Target Appearance]
500	96 [.5]
1,000	48 [.5]
Catch trials	48 [0]
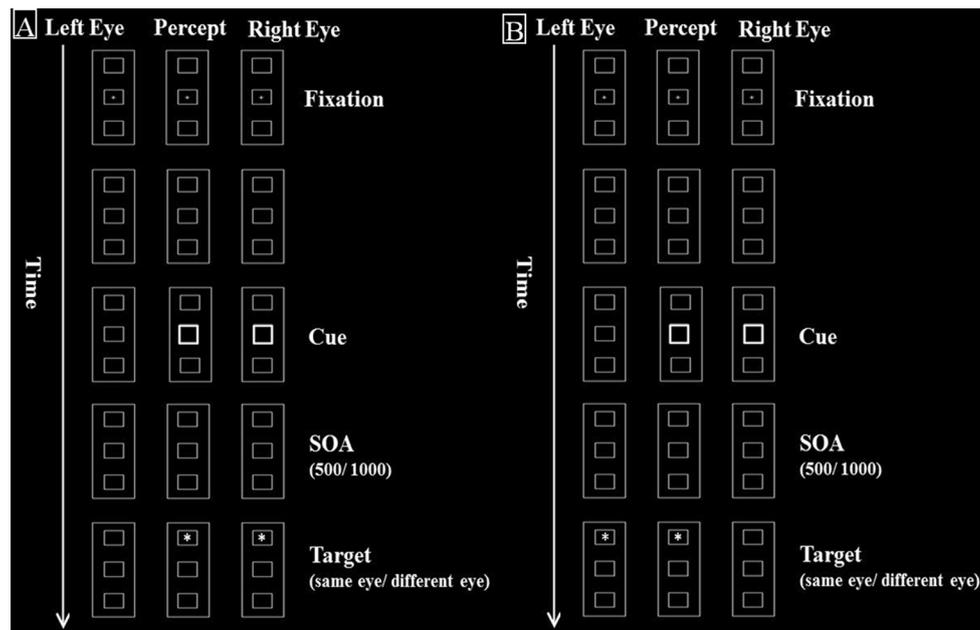
Fig. 2 (A) A typical *same-eye* condition trial, in which the cue (brightening square) is presented to the right eye (right column) and the target is also presented to the right eye (right column) in the upper square. The middle column represents the participant's fused perception. (B) A

typical *different-eye* condition trial, in which the cue (brightening square) is presented to the right eye (right column) and the target is presented to the left eye (left column) in the upper square. The middle column again represents the participant's fused perception

of the keyboard with their dominant hand as fast as possible. After each manual response, an intertrial interval of 500 ms was introduced. Each participant completed 16 practice trials before the experiment began. To preclude any confounding effect of perceptual differences between the two eye-of-origin conditions, and to determine whether participants experienced a well-fused percept in all conditions, we conducted two tests before the practice block. First, we asked participants whether they saw a single rectangle or two overlapping rectangles when looking through the stereoscope (note that two rectangles were presented throughout the task, one to each eye, and all stimuli were presented inside those rectangles). If participants reported seeing two overlapping rectangles, the stereoscope was calibrated in order to achieve a fused percept of a single rectangle. Second, participants were also instructed to close one eye (this was done for each eye separately) and asked whether they saw a full rectangle (to make sure that the visual display was full for each eye separately). If participants reported seeing only a part of the rectangle, the stereoscope was re-calibrated. These tests assured us that the percept was well fused during the task, in both eye-of-origin conditions. It is important to mention that the results also supported this notion, by demonstrating no significant difference between the same-eye and different-eye conditions with respect to general RTs (for details, see the Results section). In 96 trials (25%), no target appeared (i.e., catch trials), and the participants were instructed not to respond. The catch trials were dispersed randomly

across the trials. Each participant completed a total of 384 experimental trials, divided into four blocks. The different experimental conditions were presented randomly so that participants could not adopt a specific strategy.

Results

The STATISTICA software (version 7) was used to perform all analyses. Trials in which the RT was longer than 2,500 ms or shorter than 100 ms were excluded from the analysis (less than 1%). Participants responded on less than 1% of catch trials. We carried out a three-way analysis of variance (ANOVA) with eye of origin (same vs. different), SOA (500 vs. 1,000 ms) and cue (cue, no cue) as within-subjects factors, and RT as the dependent measure. The main effects of eye of origin and SOA were not significant [$F(1, 19) = 0.005, p > .25$; $F(1, 19) = 0.839, p > .25$, respectively]. As expected, the main effect of cue was significant [$F(1, 19) = 60.066, p < .001, \eta_p^2 = .76$], indicating lower RTs when the cue appeared (vs. the no-cue condition). All of the two-way interactions were not significant [Eye of Origin × SOA, $F(1, 19) = 1.328, p > .25$; Eye of Origin × Cue, $F(1, 19) = 2.033, p = .170$; SOA × Cue, $F(1, 19) = 0.20, p > .25$]. Most importantly, the three-way interaction between SOA, eye of origin, and cue was significant [$F(1, 19) = 5.656, p = .028, \eta_p^2 = .23$].

Planned-comparison analyses revealed that with the short SOA, the interaction between eye of origin and cue was not significant [$F(1, 19) = 0.223, p > .25$]. However, with the long

Non-Aging Alerting effect as a function of eye-of-origin and SOA
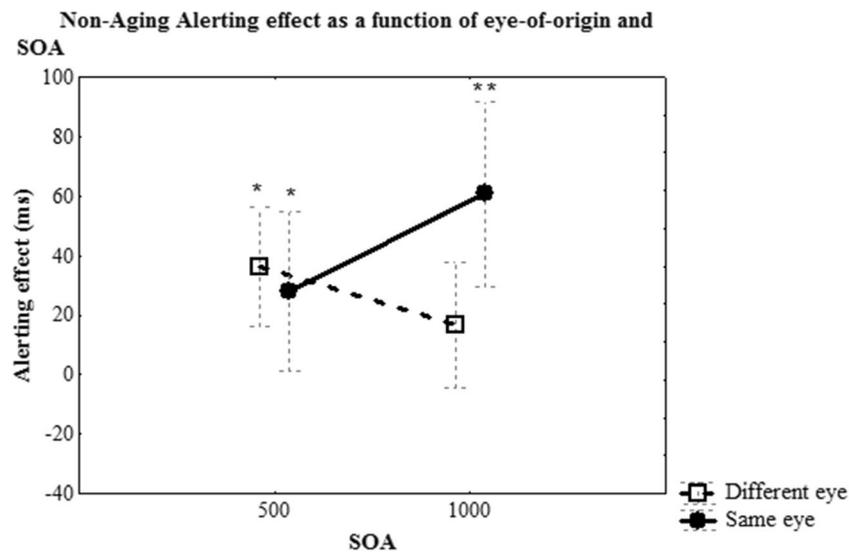
Fig. 3 Alerting effect as function of eye of origin and SOA; 95% confidence intervals are shown in the error bars. The two functions have been slightly offset horizontally to allow visualization of the error bars. $^{*}p < .05$, $^{**}p < .001$

SOA, the interaction between eye of origin and cue was significant [$F(1, 19) = 7.085$, $p = .015$], indicating a larger alerting effect (cue vs. no-cue) in the same-eye than in the different-eyes condition. Follow-up analyses revealed that the alerting effect was only significant at the same-eye condition [$F(1, 19) = 16.698$, $p < .001$; different-eye condition: $F(1, 19) = 2.752$, $p = .113$]. Figure 3 presents the alerting effect as a function of SOA and eye of origin, and Table 2 presents RTs and the standard errors for each condition.[1]

Discussion

The results of the experiment demonstrated that at the 500-ms SOA, the alerting effect did not change as a function of the eye-of-origin presentation. By contrast, at the 1,000-ms SOA, the alerting effect was significant only in the same-eye condition. One possible explanation is that in binocular channels (mostly cortical regions), phasic alertness activation dissipates over time. By contrast, in monocular channels (mostly subcortical regions), alertness dissipates more slowly over time. It is possible that the influence of an alerting cue is more robust in the monocular channel that perceived it, since the neural activation generated by presentation of the alerting cue is more restricted to that monocular channel in lower portions of the visual system. Hence, the influence of alerting cues is maintained for a longer duration in monocular regions. To conclude, thus far the results demonstrate the functional involvement of monocularly segregated (i.e., subcortical) regions in phasic alertness.

Experiment 2

The following experiment was designed to explore whether monocular regions are functionally involved in temporal expectancy. This experiment was similar to the first experiment, but in order to measure the influence of temporal expectancy without the confounding effect of phasic alertness, we implemented a foreperiod distribution manipulation (e.g., Gabay & Henik, 2008, 2010). In contrast to the first experiment, in all experimental trials a cue appeared before target appearance. This was done in order to provide optimal conditions for extracting the predictive value of the cue regarding the target's time of appearance. As a result, in the present experiment the cue was more frequent (appearing in all trials) than in the first experiment, and hence the arousal effect elicited by the cue might differ from that in the first experiment.

We manipulated three foreperiod distributions as a between-subjects variable: (1) a nonaging foreperiod distribution, in which the cue did not provide any temporal information regarding the time of the target's appearance; (2) an aging foreperiod distribution, which is the distribution typically used in experiments, in which the cue did provide temporal information; and (3) an accelerated-aging foreperiod distribution,

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[1] When comparing all the experimental and no-cue conditions, no significant effect was found [$F(3, 57) = 0.59$, $p = .624$].

Table 2. Response times (with standard errors) for the different experimental conditions

SOA (ms)	Same Eye		Different Eye	
	Cue	No Cue	Cue	No Cue
500	462 (32)	490 (29)	446 (24)	482 (26)
1,000	440 (22)	500 (26)	473 (30)	489 (27)

in which the cue was most predictive of the temporal onset of the target.

Method

With the following exceptions, the methods were similar to those used in Experiment 1. A total of 61 participants (mean age 21.68 years; 48 females, 13 males) volunteered to participate in exchange for payment or course credits. Participants were randomly assigned to one of three experimental groups: aging (21 participants), nonaging (20 participants), or accelerated aging (20 participants). A power analysis was conducted to assess the sample size required for testing a within-/between-subjects factor interaction (i.e., foreperiod distribution group, SOA, and eye of origin). The studies that have examined foreperiod distributions and SOA interaction effects have revealed very large effect sizes (e.g., Gabay & Henik, 2008, $\eta_p^2 = .457$; Gabay & Henik, 2010, $\eta_p^2 = .45$). Hence, a medium to large effect size was expected in the present experiment ($\eta_p^2 = .09$). The analysis revealed that for a power = 90% with $\alpha = .05$, we would need 52 participants. Therefore, the sample size of the present study was sufficiently powered. The study was approved by the University of Haifa ethics committee.

In all trials, and in contrast to the first experiment, 200 ms after the fixation cross disappeared, the cue—a brightening of the central square—was presented for 100 ms. In 64 trials (25%), no target appeared (i.e., catch trials). In all foreperiod distribution groups, each participant completed a total of 256 experimental trials. See Table 3 for the distributions of the numbers of trials and the probabilities of target appearance for each experimental condition.

Results

The STATISTICA software (version 7) was used to perform all analyses. As in Experiment 1, trials in which the RT was longer than 2,500 ms or shorter than 100 ms were excluded from the analysis (less than 1%). Participants responded in 1.51% of the catch trials. We carried out a three-way ANOVA with eye of origin (same vs. different) and SOA (500 vs. 1,000 ms) as within-subjects factors, and foreperiod distribution group

Table 3. Distributions of the numbers of trials and the probabilities of target appearance for each experimental condition

SOA (ms)	Nonaging [Probability]	Aging [Probability]	Accelerated Aging [Probability]
500	128 [.5]	96 [.375]	64 [.25]
1,000	64 [.5]	96 [.6]	128 [.75]
Catch trials	64 [0]	64 [0]	64 [0]

(nonaging, aging, accelerated aging) as a between-subjects factor, and RT as the dependent measure. The main effects of eye of origin, SOA, and foreperiod distribution group were not significant [$F(1, 58) = 2.46$, $p = .122$, $\eta_p^2 = .04$; $F < 1$; $F(2, 58) = 2.24$, $p = .114$, $\eta_p^2 = .071$, respectively]. The eye of origin and foreperiod distribution group two-way interaction was not significant ($F < 1$), but the Eye of Origin × SOA and SOA × Foreperiod Distribution Group two-way interactions were significant [$F(1, 58) = 4.07$, $p = .048$, $\eta_p^2 = .065$; $F(2, 58) = 5.85$, $p = .004$, $\eta_p^2 = .167$, respectively].

Most importantly, the three-way interaction between SOA, eye of origin, and foreperiod distribution group was significant [$F(2, 58) = 3.31$, $p = .043$, $\eta_p^2 = .102$]. Figure 4 presents RTs as a function of SOA and foreperiod distribution group, depicted for each eye-of-origin condition separately. To further investigate the three-way interaction, we conducted two separate two-way ANOVAs for each eye-of-origin condition, with SOA as a within-subjects factor and foreperiod distribution group as a between-subjects factor. A significant interaction was found for the same-eye condition [$F(2, 58) = 8.24$, $p < .001$, $\eta_p^2 = .221$]. We further investigated this interaction by conducting two independent planned comparisons. Figure 5 depicts the SOA effect (RT for the short SOA minus RT for the long SOA) as a function of eye of origin and foreperiod distribution group. First, we compared the SOA effect in the nonaging distribution group with the averaged SOA effects of the aging and accelerated-aging foreperiod distribution groups, and this difference was significant [$F(1, 58) = 7.15$, $p = .009$, $\eta_p^2 = .109$]. This was a result of a more negative slope (faster RT for the long SOA than for the short SOA) for the averaged aging and accelerated-aging groups than for the nonaging foreperiod distribution group. Second, we compared the SOA effect for the aging group with the SOA effect for the accelerated-aging group. A significant effect was also found for this comparison [$F(1, 58) = 9.55$, $p = .003$, $\eta_p^2 = .141$]. These analyses revealed that in same-eye condition, the SOA effects were significantly modulated by the different foreperiod distributions. By contrast, in the different-eye condition the interaction between SOA and foreperiod distribution group was not significant [$F(2, 58) = 2.39$, $p = .100$, $\eta_p^2 = .076$]. Even though the two-way interaction was not significant, we continued to analyze it. The comparison of the SOA effects between the nonaging and the averaged aging and accelerated-aging groups, and the comparison of the SOA effects between the aging and accelerated-aging groups, both were not significant [$F(1, 58) = 1.70$, $p = .196$, $\eta_p^2 = .028$; $F(1, 58) = 3.13$, $p = .081$, $\eta_p^2 = .051$, respectively].[2]

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[2] To preclude sequential-effects explanations, we conducted a similar analysis only for trials in which the trial $n - 1$ had a short SOA. In this analysis, a similar pattern of results was observed.
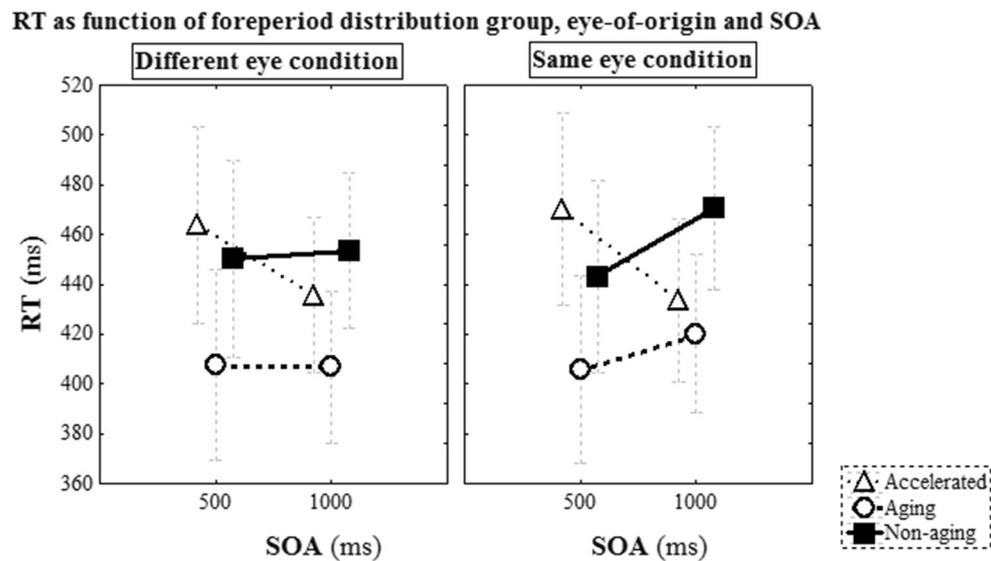
Fig. 4 RT as a function of foreperiod distribution group and SOA for each eye-of-origin condition; 95% confidence intervals are shown in the error bars. The two functions have been slightly offset horizontally to allow for visualization of the error bars

We also conducted additional follow-up analyses for the three-way interaction, by examining the interaction between eye of origin and SOA in each foreperiod distribution group separately. The only significant interaction between eye of origin and SOA was in the nonaging group [$F(1, 58) = 7.085$, $p = .01$; accelerated aging: $F(1, 58) = 0.75$, $p = .389$; aging: $F(1, 58) = 2.94$, $p = .091$].

Discussion

The present study has demonstrated for the first time that the effect of temporal expectancy is modulated by an eye-of-origin manipulation. The results demonstrate that when both the cue and target are presented to the same eye, the cue's temporal information modulates performance to a greater extent. This pattern of results suggests that subcortical structures play a functional role not only in phasic alertness, but also in temporal expectancy. Since the brain's LC–NE system arises in subcortical regions and studies have demonstrated its involvement in alerting cue effects (Coull et al., 2000; Coull et al., 1996; Marrocco & Davidson, 1998; for a review, see Sara, 2009), it is possible that this subcortical structure may also be involved in the modulation of performance as a result of changes in temporal expectancy.

The pattern of results is also in line with previous studies suggesting that a foreperiod distribution manipulation can influence temporal expectancy. Replicating previous studies (e.g., Gabay & Henik, 2008, 2010; Niemi & Näätänen, 1981), our results showed that the effect of SOA can be influenced by foreperiod distribution group (e.g., the nonaging vs. the accelerated-aging groups). In addition, the present study is

also in line with a previous study that demonstrated that in an aging distribution group, RTs do not decrease as SOAs increase (Gabay & Henik, 2008, 2010; Tipper & Kingstone, 2005). Tipper and Kingstone manipulated the percentage of catch trials (the number of trials in which a target did not appear) and found that a high percentage of catch trials (25%, as in the present study) eliminates the decline in RT as the SOA increases. In addition, another study (Gabay & Henik, 2008) suggested that the high percentage of catch trials might have decreased general alertness in Tipper and Kingstone's study. These studies are in line with the present finding of no foreperiod effect in the aging distribution group as a result of a high percentage of catch trials. However, the present foreperiod distribution manipulation modulated performance as expected, which indicates that the foreperiod distribution manipulation did influence temporal expectancy.

Note that additional follow-up analyses of the interaction between eye of origin and SOA in each foreperiod group indicated that monocular channels influenced performance only in the nonaging group. In the field of temporal expectations, studies have long differentiated between different types of expectations (for a review, see Nobre et al., 2007), which are related to different brain regions (e.g., Coull & Nobre, 2008). It is possible that the differences between the analyses mentioned above emerged from an examinations of different expectancy processes that were involved in the presently used foreperiod manipulation. In accordance with a previous study (Correa, Lupiáñez, Milliken, & Tudela, 2004), we suggest an additional viewpoint, in which the nonaging foreperiod group can also be considered an early-expectancy manipulation, whereas the accelerated-aging foreperiod group can be
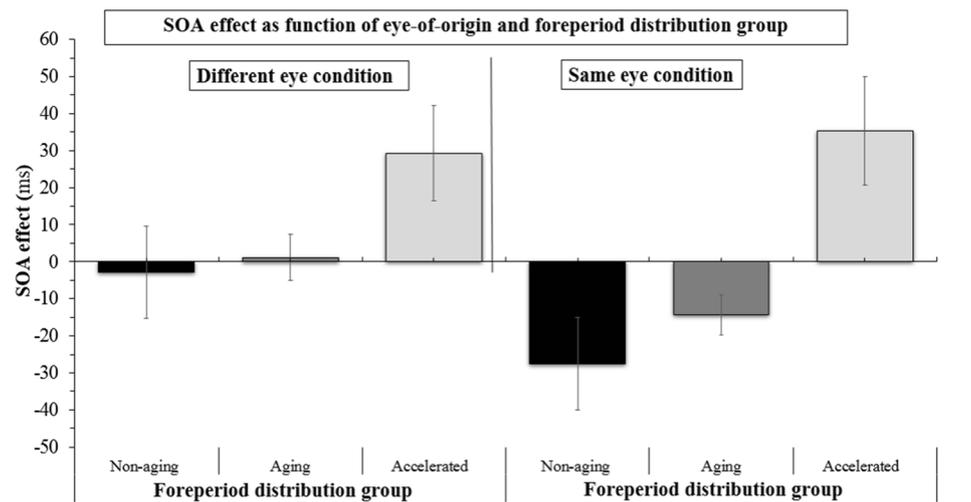
Fig. 5 SOA effect (RT for the short SOA minus RT for the long SOA) as a function of eye of origin and foreperiod distribution group. Error bars indicate standard errors

considered a late-expectancy manipulation (for the differences between the two types of expectancies, see Correa et al., 2004). We suggest that in the nonaging foreperiod group, in which there are more short-SOA trials (vs. long), participants may learn to expect short-SOA trials (since they have the highest frequency). However, in the accelerated-aging group, a late-expectancy process is involved, and participants expect that the target will probably appear at a long SOA. The present finding suggest that subcortical regions are not involved in late-expectancy processes, since we did not find an eye-of-origin modulation in the accelerated-aging group.

Both our first analysis and the insights from the second analysis indicate that primitive–subcortical regions are involved in temporal expectancy process. The added value of the second analyses is that those regions might be involved only with an early expectancy, but not with a late expectancy.

General discussion

The aim of the present study was to test whether lower portions of the visual stream are functionally involved in phasic alertness and temporal expectancy, by manipulating the cue's and the target's eye of origin. Simple target detection tasks were administrated in this study with a variable foreperiod distribution. Using a stereoscope, we manipulated whether the cue and target were presented to the same eye or to different eyes. This technique allowed us to examine the involvement of monocularly segregated (mostly subcortical) regions of the visual processing stream.

In the first experiment, participants' temporal expectancy was eliminated by using a nonaging foreperiod distribution in order to assess the pure influence of phasic alertness, irrespective of temporal expectancy. It was found that the alerting effect was modulated as a function of eye of origin.

Specifically, at the second SOA, the alerting effect was larger when the cue and target were presented to the same eye than when they were presented to different eyes. In line with previous studies, the result of the first experiment demonstrated the involvement of monocularly segregated regions in phasic alertness.

In the second experiment, the contribution of monocular channels in temporal expectancy was examined. This was done by employing a stereoscope cue–target eye-of-origin manipulation, while implementing three (between-subjects) foreperiod distributions: nonaging, aging, and accelerated aging. The level of phasic alertness was controlled by presenting a preceding cue in all trials. Most importantly, in the same-eye condition the temporal expectancy manipulation influenced performance to a greater extent. These results allow us to conclude that monocular portions of the visual stream are also involved in temporal expectancy. Temporal expectancy, as indexed by changes in the foreperiod effect, was modulated by the cue's temporal information to a greater extent when both the cue and target were presented to the same monocular channel.

When examining the influence of monocular channels at each foreperiod group separately, we observed that monocular channels affected performance only for the nonaging group. This analyses is important because, in the field of temporal expectations, studies have long differentiated between different types of expectations (for a review, see Nobre et al., 2007), which are related to different brain regions (e.g., Coull & Nobre, 2008). It is possible that in the currently used foreperiod manipulation, different expectancy processes were examined. In accordance with a previous study (Correa et al., 2004), the nonaging group might have assessed an early-expectancy process, whereas the accelerated-aging group might have assessed a late-expectancy process. We suggest that subcortical regions might be involved only in the early-expectancy, but not in the late-expectancy, process.

In studies employing alerting cues, a clear, well-defined dissociation between phasic alertness and temporal expectancy might be challenging (e.g., Weinbach & Henik, 2012). Since studies have long demonstrated the involvement of the LC–NE system in the effects of alerting cues (Coull et al., 2000; Coull et al., 1996; Marrocco & Davidson, 1998; for a review, see Sara, 2009), it is possible that this subcortical structure may be involved in both phasic alertness and temporal expectancy.

By contrast, as we discussed in the introduction, the previous literature has long emphasized the involvement of mainly cortical regions in phasic alertness and temporal expectancy (e.g., parietal, premotor, and prefrontal cortical regions). This cortico-centric emphasis is not unexpected when methodological limitations are taken into consideration. For instance, the power of fMRI to establish a direct causal relation between brain regions (especially subcortical regions) and cognitive processes is limited and prone to the confounding effects of epiphenomenal brain activations (e.g., LaBar, Gitelman, Mesulam, & Parrish, 2001).

In addition, although the present study provides converging evidence for the involvement of monocular channels in phasic alertness and temporal expectancy, it is also possible that these low-level structures may be necessary but not sufficient to elicit both processes. Since the visual system has many feedback connections (e.g., Bullier, 2001; Lamme, Supèr, & Spekreijse, 1998), dynamic interactions between cortical and subcortical regions might be involved in these processes. It should be noted that in order for feedback connections from cortical regions to explain the differences between the two eye-of-origin conditions, the connections should target monocularly segregated neurons specifically.

For example, similar to most subcortical visual regions, V1 also has monocularly segregated neurons that might be responsible for the differences between the two eye-of-origin conditions. V1, which projects monosynaptically to the SC, and thus provides a source of cortical inputs, could play a role in attention. Indeed, recent experimental evidence supports the idea that V1 creates a bottom-up saliency map that plays a role in guiding attention (Zhaoping, 2008). As Zhaoping suggested, if V1, through its SC connections, is involved in attentional orienting, it is possible that the same low-level neuronal mechanisms are also involved in phasic alertness and temporal expectancy.

The presence of an alerting effect at the short SOA when the cue and target were presented to different eyes suggests the involvement of higher cortical regions in this process and implies that phasic alertness may not depend solely on subcortical involvement. Although the explanation is speculative, the finding that the alerting effect was maintained for a longer duration at monocular channels (vs. binocular channels) might be suggested to result from a reactivation of the same group of neurons that processed the cue in the same-eye condition, which are also activated by the target. In contrast, in the different-eye condition, different groups of neurons were activated by the cue and the target, and hence, the neurons that responded to the target had not previously been activated. This might explain why the alerting effect is maintained for a shorter duration in the different-eye condition.

It is possible that distinct subcortical brain areas are involved in phasic alertness and temporal expectancy. Several studies have demonstrated the involvement of the LC in phasic alertness (Coull et al., 1996; Fan et al., 2005; for a review of SC involvement, see Sara, 2009), whereas other studies have demonstrated the involvement of the thalamus in temporal expectancy (Coull et al., 2000). In the present study, we observed a dissociation in the foreperiod over which subcortical involvement was observed. It is possible that the involvement of early subcortical brain structures has its effect at different foreperiods. This pattern of results indicates that the involvement of primitive–subcortical regions in more basic and reflexive processes (i.e., phasic alertness) appears over a longer temporal duration, whereas the involvement of subcortical regions in volitional processes (i.e., temporal expectancy) appears over shorter temporal durations (early expectancy). We suggest that future research should examine whether or not the two functions have different subcortical neural substrates.

To conclude, in the present study we measured the involvement of monocular channels in phasic alertness and temporal expectancy independently. It was found that monocular channels of the visual processing stream do more than just channel information: They also have a functional role in phasic alertness and temporal expectancy. In addition, subcortical involvement in different temporal processes might be differential and may depend on the nature of the cognitive function in the reflexive-to-volitional axis.

References

Batson, M. A., Beer, A. L., Seitz, A. R., & Watanabe, T. (2011). Spatial shifts of audio-visual interactions by perceptual learning are specific to the trained orientation and eye. *Seeing and Perceiving*, *24*, 579–594. https://doi.org/10.1163/187847611X603738

Baumeister, A. A., & Joubert, C. E. (1969). Interactive effects on reaction time of preparatory interval length and preparatory interval frequency. *Journal of Experimental Psychology, 82*, 393.

Bueti, D., Bahrami, B., & Walsh, V. (2008). Sensory and association cortex in time perception. *Journal of Cognitive Neuroscience, 20*, 1054–1062. https://doi.org/10.1162/jocn.2008.20060

Bueti, D., Bahrami, B., Walsh, V., & Rees, G. (2010). Encoding of temporal probabilities in the human brain. *Journal of Neuroscience, 30*, 4343–4352. https://doi.org/10.1523/jneurosci.2254-09.2010

Bullier, J. (2001). Integrated model of visual processing. *Brain Research Reviews, 36*, 96–107.

Callejas, A., Lupiáñez, J., & Tudela, P. (2004). The three attentional networks: On their independence and interactions. *Brain and Cognition, 54*, 225–227. https://doi.org/10.1016/j.bandc.2004.02.012

Correa Á., Lupiáñez, J., Milliken, B., & Tudela, P. (2004). Endogenous temporal orienting of attention in detection and discrimination tasks. *Perception & Psychophysics, 66*, 264–278. https://doi.org/10.3758/BF03194878

Coull, J., & Nobre, A. C. (2008). Dissociating explicit timing from temporal expectation with fMRI. *Current Opinion in Neurobiology, 18*, 137–144. https://doi.org/10.1016/j.conb.2008.07.011

Coull, J. T., Frith, C. D., Büchel, C., & Nobre, A. C. (2000). Orienting attention in time: Behavioural and neuroanatomical distinction between exogenous and endogenous shifts. *Neuropsychologia, 38*, 808–819. https://doi.org/10.1016/S0028-3932(99)00132-3

Coull, J. T., Frith, C. D., Frackowiak, R. S. J., & Grasby, P. M. (1996). A fronto-parietal network for rapid visual information processing: A PET study of sustained attention and working memory. *Neuropsychologia, 34*, 1085–1095.

Coull, J. T., & Nobre, A. C. (1998). Where and when to pay attention: The neural systems for directing attention to spatial locations and to time intervals as revealed by both PET and fMRI. *Journal of Neuroscience, 18*, 7426–7435.

Coull, J. T., Vidal, F., Nazarian, B., & Macar, F. (2004). Functional anatomy of the attentional modulation of time estimation. *Science, 303*, 1506–1508.

Fan, J., McCandliss, B. D., Fossella, J., Flombaum, J. I., & Posner, M. I. (2005). The activation of attentional networks. *NeuroImage, 26*, 471–479. https://doi.org/10.1016/j.neuroimage.2005.02.004

Faul, F., Erdfelder, E., Lang, A., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods, 39*, 175–191. https://doi.org/10.3758/BF03193146

Gabay, S., & Behrmann, M. (2014). Attentional dynamics mediated by subcortical mechanisms. *Attention, Perception, & Psychophysics, 76*, 2375–2388. https://doi.org/10.3758/s13414-014-0725-0

Gabay, S., Burlingham, C., & Behrmann, M. (2014a). The nature of face representations in subcortical regions. *Neuropsychologia, 59*, 35–46.

Gabay, S., & Henik, A. (2008). The effects of expectancy on inhibition of return. *Cognition, 106*, 1478–1486. https://doi.org/10.1016/j.cognition.2007.05.007

Gabay, S., & Henik, A. (2010). Temporal expectancy modulates inhibition of return in a discrimination task. *Psychonomic Bulletin & Review, 17*, 47–51. https://doi.org/10.3758/PBR.17.1.47

Gabay, S., Nestor, A., Dundas, E., & Behrmann, M. (2014b). Monocular advantage for face perception implicates subcortical mechanisms in adult humans. *Journal of Cognitive Neuroscience, 26*, 927–937.

Genovesio, A., Tsujimoto, S., & Wise, S. P. (2006). Neuronal activity related to elapsed time in prefrontal cortex. *Journal of Neurophysiology, 95*, 3281–3285. https://doi.org/10.1152/jn.01011.2005

Ghose, G. M., & Maunsell, J. H. (2002). Attentional modulation in visual cortex depends on task timing. *Nature, 419*, 616–620.

Horton, J. C., Dagi, L. R., McCrane, E. P., & de Monasterio, F. M. (1990). Arrangement of ocular dominance columns in human visual cortex. *Archives of Ophthalmology, 108*, 1025–1031.

Janssen, P., & Shadlen, M. N. (2005). A representation of the hazard rate of elapsed time in macaque area LIP. *Nature Neuroscience, 8*, 234–241.

Karni, A., & Sagi, D. (1991). Where practice makes perfect in texture discrimination: Evidence for primary visual cortex plasticity. *Proceedings of the National Academy of Sciences, 88*, 4966–4970.

LaBar, K. S., Gitelman, D. R., Mesulam, M. M., & Parrish, T. B. (2001). Impact of signal-to-noise on functional MRI of the human amygdala. *NeuroReport, 12*, 3461–3464.

Lamme, V. A. F., Supèr, H., & Spekreijse, H. (1998). Feedforward, horizontal, and feedback processing in the visual cortex. *Current Opinion in Neurobiology, 8*, 529–535. https://doi.org/10.1016/S0959-4388(98)80042-1

Leon, M. I., & Shadlen, M. N. (2003). Representation of time by neurons in the posterior parietal cortex of the macaque. *Neuron, 38*, 317–327.

Marrocco, R. T., & Davidson, M. C. (1998). Neurochemistry of attention. In R. Parasuraman (Ed.), The attentive brain (pp. 35–50). Cambridge: MIT Press.

Menon, R. S., Ogawa, S., Strupp, J. P., & Uğurbil, K. (1997). Ocular dominance in human V1 demonstrated by functional magnetic resonance imaging. *Journal of Neurophysiology, 77*, 2780–2787.

Morrison, J. H., & Foote, S. L. (1986). Noradrenergic and serotoninergic innervation of cortical, thalamic, and tectal visual structures in Old and New World monkeys. *Journal of Comparative Neurology, 243*, 117–138.

Niemi, P., & Näätänen, R. (1981). Foreperiod and simple reaction time. *Psychological Bulletin, 89*, 133–162. https://doi.org/10.1037/0033-2909.89.1.133

Nobre, A. C., Correa, A., & Coull, J. T. (2007). The hazards of time. *Current Opinion in Neurobiology, 17*, 465–470.

Onoe, H., Komori, M., Onoe, K., Takechi, H., Tsukada, H., & Watanabe, Y. (2001). Cortical networks recruited for time perception: A monkey positron emission tomography (PET) study. *NeuroImage, 13*, 37–45.

Périn, B., Godefroy, O., Fall, S., & De Marco, G. (2010). Alertness in young healthy subjects: An fMRI study of brain region interactivity enhanced by a warning signal. *Brain and Cognition, 72*, 271–281.

Petersen, S. E., & Posner, M. I. (2012). The attention system of the human brain: 20 years after. *Annual Review of Neuroscience, 35*, 73–89. https://doi.org/10.1146/annurev-neuro-062111-150525

Posner, M. I. (1988). Structures and functions of selective attention. In T. Boll & B. Bryant (Eds.), Master lectures in clinical neuropsychology and brain function: Research, measurement, and practice (pp. 171–202). Washington, DC: American Psychological Association.

Posner, M. I., & Boies, S. J. (1971). Components of attention. *Psychological Review, 78*, 391–408. https://doi.org/10.1037/h0031333

Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience, 13*, 25–42. https://doi.org/10.1146/annurev.ne.13.030190.000325

Saban, W., Gabay, S., & Kalanthroff, E. (2018a). More than just channeling: The role of subcortical mechanisms in executive functions—Evidence from the Stroop task. *Acta Psychologica, 189*, 36–42. https://doi.org/10.1016/j.actpsy.2017.03.001

Saban, W., Klein, R. M., & Gabay, S. (2018b). Probabilistic versus "pure" volitional orienting: A monocular difference. *Attention, Perception, & Psychophysics, 80*, 669–676. https://doi.org/10.3758/s13414-017-1473-8

Saban, W., Sekely, L., Klein, R. M., & Gabay, S. (2017). Endogenous orienting in the archer fish. *Proceedings of the National Academy of Sciences, 114*, 7577–7581. https://doi.org/10.1073/pnas.1700574114

Saban, W., Sekely, L., Klein, R. M., & Gabay, S. (2018c). Monocular channels have a functional role in endogenous orienting. *Neuropsychologia*, *111*, 1–7. https://doi.org/10.1016/j.neuropsychologia.2018.01.002

Salvioni, P., Murray, M. M., Kalmbach, L., & Bueti, D. (2013). How the visual brain encodes and keeps track of time. *Journal of Neuroscience*, *33*, 12423–12429.

Sara, S. J. (2009). The locus coeruleus and noradrenergic modulation of cognition. *Nature Reviews Neuroscience*, *10*, 211–223. https://doi.org/10.1038/nrn2573

Self, M. W., & Roelfsema, P. R. (2010). A monocular, unconscious form of visual attention. *Journal of Vision*, *10*(4), 17. https://doi.org/10.1167/10.4.17

Spagna, A., Martella, D., Sebastiani, M., Maccari, L., Marotta, A., & Casagrande, M. (2014). Efficiency and interactions of alerting, orienting and executive networks: The impact of imperative stimulus type. *Acta Psychologica*, *148*, 209–215. https://doi.org/10.1016/j.actpsy.2014.02.007

Sturm, W., & Willmes, K. (2001). On the functional neuroanatomy of intrinsic and phasic alertness. *NeuroImage*, *14*, S76–S84.

Stuss, D. T., & Alexander, M. P. (2005). Does damage to the frontal lobes produce impairment in memory? *Current Directions in Psychological Science*, *14*, 84–88. https://doi.org/10.1111/j.0963-7214.2005.00340.x

Tipper, C., & Kingstone, A. (2005). Is inhibition of return a reflexive effect? *Cognition*, *97*, B55–B62. https://doi.org/10.1016/j.cognition.2005.02.003

Triviño, M., Correa, A., Arnedo, M., & Lupiáñez, J. (2010). Temporal orienting deficit after prefrontal damage. *Brain*, *133*(Part 4), 1173–1785. https://doi.org/10.1093/brain/awp346

Vallesi, A., McIntosh, A. R., Shallice, T., & Stuss, D. T. (2009). When time shapes behavior: fMRI evidence of brain correlates of temporal monitoring. *Journal of Cognitive Neuroscience*, *21*, 1116–1126.

Vallesi, A., & Shallice, T. (2007). Developmental dissociations of preparation over time: Deconstructing the variable foreperiod phenomena. *Journal of Experimental Psychology: Human Perception and Performance*, *33*, 1377–1388. https://doi.org/10.1037/0096-1523.33.6.1377

Vallesi, A., Shallice, T., & Walsh, V. (2007). Role of the prefrontal cortex in the foreperiod effect: TMS evidence for dual mechanisms in temporal preparation. *Cerebral Cortex*, *17*, 466–474.

Weinbach, N., & Henik, A. (2012). Temporal orienting and alerting—The same or different? *Frontiers in Psychology*, *3*, 236. https://doi.org/10.3389/fpsyg.2012.00236

Weinbach, N., & Henik, A. (2013). The interaction between alerting and executive control: Dissociating phasic arousal and temporal expectancy. *Attention, Perception, & Psychophysics*, *75*, 1374–1381. https://doi.org/10.3758/s13414-013-0501-6

Whitehead, R. (1991). Right hemisphere processing superiority during sustained visual attention. *Journal of Cognitive Neuroscience*, *3*, 329–334.

Wundt, W. (1887). Über Ziele und Wege der Völkerpsychologie. *Philosophische Studien*, *4*, 1–27.

Yanaka, H. T., Saito, D. N., Uchiyama, Y., & Sadato, N. (2010). Neural substrates of phasic alertness: A functional magnetic resonance imaging study. *Neuroscience Research*, *68*, 51–58. https://doi.org/10.1016/j.neures.2010.05.005

Yoshida, Y., Tanabe, H. C., Hayashi, M. J., Kawamichi, H., Kochiyama, T., & Sadato, N. (2013). The neural substrates of the warning effect: A functional magnetic resonance imaging study. *Neuroscience Research*, *76*, 230–239. https://doi.org/10.1016/j.neures.2013.05.008

Zhaoping, L. (2008). Attention capture by eye of origin singletons even without awareness—A hallmark of a bottom-up saliency map in the primary visual cortex. *Journal of Vision*, *8*(5), 1.1–18. https://doi.org/10.1167/8.5.1