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Direction-contingent duration compression is primarily retinotopic

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Abstract
Previous research has shown that prior adaptation to a spatially circumscribed, oscillating grating results in the duration of a subsequent stimulus briefly presented within the adapted region being underestimated. There is an on-going debate about where in the motion processing pathway the adaptation underlying this distortion of sub-second duration perception occurs. One position is that the LGN and, perhaps, early cortical processing areas are likely sites for the adaptation; an alternative suggestion is that visual area MT+ contains the neural mechanisms for sub-second timing; and a third position proposes that the effect is driven by adaptation at multiple levels of the motion processing pathway. A related issue is in what frame of reference – retinotopic or spatiotopic – does adaptation induced duration distortion occur. We addressed these questions by having participants adapt to a unidirectional random dot kinematogram (RDK), and then measuring perceived duration of a 600ms test RDK positioned in either the same retinotopic or the same spatiotopic location as the adaptor. We found that, when it did occur, duration distortion of the test stimulus was direction contingent; that is it occurred when the adaptor and test stimuli drifted in the same direction, but not when they drifted in opposite directions. Furthermore the duration compression was evident primarily under retinotopic viewing conditions, with little evidence of duration distortion under spatiotopic viewing conditions. Our results support previous research implicating cortical mechanisms in the duration encoding of sub-second visual events, and reveal that these mechanisms encode duration within a retinotopic frame of reference.

1. Introduction
The ability to accurately perceive the duration of events is an essential component of meaningful interaction with our environment. Traditionally it has been thought that all event timing is carried out by a centralised internal clock responsible for encoding duration across all modalities (Creelman, 1962; Treisman, 1963; Treisman et al., 1990). However recent studies suggest that this is not the case for sub-second events; rather it is likely there are a number of distributed, modality-specific mechanisms which time events in the sub-second range (Buonomano & Karmarkar, 2002; Grondin, 2010; Karmarkar & Buonomano, 2007; Mauk & Buonomano, 2004). For instance Bueti, Bahrami & Walsh (2008) demonstrated that, when TMS is used to disrupt functioning in Area MT, participants’ ability to accurately time brief visual events suffered; however their ability to accurately time acoustic events was unaffected. Bueti et al. take this as evidence that area MT is critical for the perception of visual (but not auditory) duration.
A series of studies by different research groups has demonstrated the existence of multiple, spatially localized temporal mechanisms that underlie duration encoding in the visual system. The existence of these mechanisms was first demonstrated by Johnston et al. (2006) when they reported that prior adaptation to a 20Hz oscillating sine wave pattern resulted in perceived ‘duration compression’ of a subsequent 600ms 10Hz test pattern presented in the same location. The effect persisted when the grating stimuli were replaced with Gaussian patches that changed in brightness sinusoidally, thus suggesting that the underlying mechanisms driving the effect are temporal frequency tuned. There have been several attempts at identifying where in the motion-processing pathway the mechanisms underlying the duration compression effect reside. Adaptation-induced duration compression has been demonstrated using very narrow (0.75° x 1°) adaptors (Ayhan et al., 2009), and when adaptor and test grating orientations (hence their drift directions) differ by 90°. The former finding implies the involvement of brain regions with small receptive fields, and the orientation-independence of duration compression implicates pre-cortical mechanisms. The pre-cortical interpretation is reinforced by the finding that adapting to temporal frequencies above the flicker fusion threshold, but low enough to stimulate LGN cells, also induces duration compression (Johnston et al., 2008).

While evidence of the involvement of pre-cortical mechanisms in the duration compression effect is compelling, it appears that cortical mechanisms also contribute to the effect. Curran and Benton (2012) had participants adapt to a unidirectional (3°s⁻¹) drifting dot pattern before judging the duration of a 600ms test pattern that drifted in either the same or opposite direction to the adapting stimulus. There was strong duration compression when test and adaptor drifted in the same direction, but no duration distortion was evident when they drifted in opposite directions; in other words the duration compression effect was direction contingent. Given that cortical area V1 marks the earliest point in the primate visual system where direction-sensitive neurons occur (Hubel & Wiesel, 1968), the direction contingent nature of duration compression points to the existence of cortical timing mechanisms. In a follow-up experiment Curran & Benton had participants adapt to a vertically drifting plaid pattern before judging the duration of a random dot test stimulus whose motion direction matched the plaid’s global motion direction. Again there was robust adaptation-induced duration compression; when the plaid adaptor was replaced with a transparent moving dot pattern, whose two motion directions matched the plaid’s component directions (± 70° from vertical), there was no distortion of the test stimulus’s perceived duration. Given that drifting plaids are known to selectively stimulate MT neurons tuned to the plaid’s global motion direction (Movshon et al., 1985), Curran & Benton’s results suggest that cortical timing mechanisms are likely to be found at or beyond area MT.
Marinovic and Arnold (2012) report that prior adaptation to a high speed (410°s⁻¹) circular pattern of rotating dots results in duration compression of a briefly presented dot in the adapted location. They found no evidence that the duration compression was direction contingent, reporting that test dot duration (speed = 205.2°s⁻¹) was underestimated regardless of whether it moved in the same or opposite direction to the adapting pattern. The absence or presence of direction-contingent duration compression reported by Marinovic & Arnold and Curran & Benton, respectively, may be explained by the observation that the former used fast adaptor and test speeds while the latter used slow (3°s⁻¹) adaptor and test speeds. This has been confirmed by Bruno et al. (2013), who found duration compression to be direction contingent when adapting and testing with low temporal frequencies but was direction independent for higher temporal frequencies. They take this as evidence that the mechanism underlying adaptation-induced duration compression comprises both pre-cortical and cortical components, although they argue that the cortical mechanisms are likely to reside in area V1.

There is an on-going debate regarding the frame of reference within which adaptation-induced duration compression occurs, with one line of evidence suggesting that the timing mechanisms underlying the effect operate within a spatiotopic frame of reference (Burr et al., 2007, 2011; Morrone et al., 2010) and another pointing to retinotopic neural timing mechanisms (Bruno et al., 2010). Burr et al. (2007) addressed this question by having participants adapt to an oscillating grating before judging the duration of a test stimulus under three different conditions. In the ‘full adaptation’ condition fixation was maintained at the same location and the test stimulus was presented in the adapted region; in both the spatiotopic and retinotopic conditions participants made a saccade to a new fixation point and the test stimulus was presented at either the same physical location (spatiotopic condition) or the same retinotopic location (retinotopic condition) as the adapting stimulus. When the test stimulus had the same physical speed as the comparison stimulus, duration compression was evident in each of the three conditions. However, when comparison stimulus speed was matched to the perceived speed of the test stimulus, duration compression only occurred in the full and spatiotopic conditions. This was taken as evidence that the neural mechanisms underlying the duration compression effect are localized in real-world, rather than retinal, coordinates. Given the evidence that spatiotopic encoding first occurs in area MT+ (Goosens et al., 2006), and that this cortical region has been previously implicated in subsecond timing (Janssen & Shadlen, 2005; Leon & Shadlen, 2003), Morrone et al. (2010) propose that the duration compression effect is based on the activity of timing mechanisms in MT+. As compelling as
these results are, there remains disagreement on the frame of reference within which the timing mechanisms underlying the duration compression effect operate. Bruno et al. (2010) report a robust duration compression effect for both the ‘full adaptation’ and retinotopic conditions, but found no evidence of duration distortion in their spatiotopic condition, suggesting the existence of a significant retinotopic component for duration encoding; which, in turn, suggests that the mechanisms responsible for the duration compression effect reside at an early stage of the visual system. In Bruno et al.’s experiment the test and comparison stimuli had the same physical speed, a scenario under which Burr et al. found duration compression for both the retinotopic and spatiotopic conditions. Another methodological difference was the presentation order of test and comparison stimuli post-adaptation; whereas Bruno et al. randomized the presentation order, Burr et al. presented the stimuli in the same order – test followed by comparison. Given that the strength of the second of a pair of stimuli tends to be overestimated (Lapid et al., 2008; Nachmias, 2006), Bruno et al. speculate that this temporal order effect may be partly responsible for the duration compression found in Burr et al.’s spatiotopic condition. However, Burr et al. (2011) point out that, because their data were calculated as the difference between adapted and unadapted conditions, any such temporal order effect would have been cancelled out.

So, not only is there disagreement on where the timing mechanisms underlying the duration compression effect are located in the motion-processing pathway, there is also disagreement on the related issue regarding the frame of reference within which the effect occurs. Here we describe an experiment which addresses the retinotopic/spatiotopic debate, in which we use a modified version of the experiments designed by Burr et al. (2007) and Bruno et al. (2010). Rather than using oscillating adaptor and test grating stimuli, we used unidirectional random dot patterns with the adaptor and test stimuli having identical retinotopic and spatiotopic coordinates, identical retinal (but different spatiotopic) coordinates, or identical spatiotopic (but different retinotopic) coordinates. We use a stimulus speed which has been shown to induce direction-contingent duration compression (Curran & Benton, 2012; Bruno et al., 2013). This will ensure that any resulting duration compression is driven by adaptation of cortical mechanisms; if duration compression is underpinned by spatiotopic mechanisms, then this should be revealed through adapting these cortical mechanisms. Our results show a robust duration compression effect in the retinotopic, but not spatiotopic, viewing condition. Furthermore, we show that the direction contingent nature of the duration compression effect (Curran & Benton, 2012) also persists under the retinotopic condition. These results support previous reports that cortical mechanisms are
implicated in the encoding of brief visual motion events; they also demonstrate the encoding of duration within a retinotopic frame of reference.

2. Methods

2.1 Participants
The same 7 participants (3 authors, 4 naïve) were tested in the four adapt-test conditions. All participants had normal or corrected-to-normal vision. The experiment was conducted in accord with the Code of Ethics of the World Medical Association (Declaration of Helsinki) and informed consent was obtained for experimentation with human subjects.

2.2 Apparatus
Stimuli were presented on a Mitsubishi Diamond Pro 2070\textsuperscript{58} monitor (Belfast) and a Sony CPD-500 monitor (Bristol); the former was driven by a Cambridge Research Systems Visage and the latter was driven by a Cambridge Research Systems VSG 2/5 graphics board, both at a frame rate of 120Hz.

2.3 Stimuli
Stimuli were unidirectional, translating random dot kinematograms (100\% coherence, dot diameter = 1.8 arcmin) presented within a circular aperture (6.3\textdegree diameter). The chosen viewing distances (Belfast: 71cm; Bristol: 72cm) ensured stimuli subtended the same visual angle on both experimental set-ups.

2.4 Procedure
Participants adapted to a unidirectional RDK depicting upwards motion while fixating on a black ‘x’ to its lower left. The initial adaptation phase lasted 30 seconds. Following adaptation the fixation marker either remained at the same location (full adaptation condition) or it was re-plotted 7.6\textdegree to the right and participants made a saccade to it. Participants were then presented with the test and comparison stimuli, whose presentation order was randomized. The 600ms test stimulus, which moved in the same direction as the adaptor, was displayed in one of three locations depending on the test condition (Fig 1a). There was one no-saccade condition and three saccade conditions. In the no saccade ‘full adaptation’ condition the test stimulus was presented at the same retinal and spatial location as the adaptor (above and to the right of fixation) and the comparison stimulus was presented below and to the right of fixation; in the saccade ‘retinotopic’ condition the test stimulus was presented at the same retinal location as the adaptor; in the saccade ‘spatiotopic’ condition it
was presented at the same spatial location as the adaptor; and in the saccade ‘control’ condition the test stimulus was positioned to the lower left of fixation. For the three saccade conditions the comparison stimulus, drifting in the opposite direction to the test stimulus, was positioned to the lower right of fixation. Perceived duration of the test pattern was estimated by having participants judge whether the test or comparison stimulus had the longer duration. To maintain adaptation, each subsequent test-comparison pair was preceded with a 5 s presentation of the adapting stimulus. Comparison stimulus duration was chosen by an adaptive method of constants procedure (Watt & Andrews, 1981) thus optimising the estimation of the ‘point of subjective equality’ (PSE), i.e. the duration at which the comparison stimulus was judged to match that of the test stimulus. At the end of each trial the fixation marker reset to its original position. Adaptor and test stimulus speed was fixed at 3°s\(^{-1}\). Comparison stimulus speed was set to match the perceived speed of the test stimulus; thus controlling for adaptation-induced speed distortions (Thompson, 1981), and taking into account previous reports that perceived speed influences apparent duration (Brown, 1995; Kanai et al., 2006; Kaneko & Murakami, 2009). Participants generated four PSEs per viewing condition, with each PSE derived from 64 duration judgments.

Results

Figure 2 (a-d) plots change in perceived duration of the test stimulus for each viewing condition (full, retinotopic, spatiotopic, and control). One-tailed t test analyses reveal significant duration compression in the full (t(6) = 7.788, p < 0.001, d = 2.94) and retinotopic adaptation conditions (t(6) = 6.609, p < 0.001, d = 2.5), but find no evidence of significant duration compression in the spatiotopic condition (t(6) = 1.623, p = 0.156, d = 0.61); two tailed t-test analysis shows no strong evidence of duration distortion in the control condition (t(6) = 0.006, p = 0.995, d = 0.002), but does show a significant duration compression effect in the retinotopic condition relative to the spatiotopic condition (t(6) = 4.142, p = 0.003, d = 2.37). Our results suggest that the duration compression effect takes place primarily within a retinotopic frame of reference; this is clearly depicted in Figure 2b, in which the effect is evident for 6 of the 7 participants. Paired t-test analysis reveals a significant difference between the no-saccade ‘full’ condition and the retinotopic condition (t(6) = 3.585, p = 0.012, d = 1.06); while it is not entirely inconsistent with a weak influence of a spatiotopic effect, this result, in conjunction with an absence of significant compression in the spatiotopic condition, is not a strong endorsement of there being a spatiotopic component. Indeed, the results are just as consistent with there being no such influence.
The one-sample t tests, reported above, measured the difference between perceived duration for each of the conditions and assumed veridical duration perception in the absence of prior adaptation. We repeated our analysis, but this time used Burr et al.’s (2007) approach and assessed duration compression in the full, spatiotopic, and retinotopic conditions relative to perceived duration in the control condition. The control condition had both test and comparison stimuli presented in non-adapted regions, thus serving as a baseline measure for participants’ duration perception. One-tailed t test analysis reveals significant differences in duration perception between the control condition and the full (t(6) = 6.359, p < 0.001, d = 2.66) and retinotopic (t(6) = 4.297, p = 0.003, d = 1.94) conditions, but no significant difference between the control and spatiotopic conditions (t(6) = 0.75, p = 0.24, d = 0.37).

While our results reveal an important role for retinotopic-tuned mechanisms in the duration compression effect it is not possible to infer from these data whether the observed duration compression effect is mediated by pre-cortical or cortical timing mechanisms, simply because retinotopic mapping is prevalent at multiple levels of the motion-processing pathway. We, therefore, tested whether the reported direction contingent nature of the duration compression effect (Curran & Benton, 2012) held in the retinotopic condition; i.e. is the effect null when the adapt and test stimuli move in opposite directions? Given that cortical area V1 marks the earliest point in the primate visual system where direction-sensitive neurons occur (Hubel & Wiesel, 1968), evidence of the duration compression effect being direction contingent would point to it being driven by adaptation of cortical mechanisms. Paired t test analysis found no significant duration compression relative to the control condition (t(6) = 0.443, p = 0.39, d = 0.24); this supports previous assertions that cortical mechanisms are involved in encoding the duration of brief motion stimuli.

**Discussion**

There is an on-going debate regarding the frame of reference – retinotopic or spatiotopic – within which adaptation-induced duration compression of brief visual events occurs, with Burr et al. (2007) finding the effect to be spatiotopic while Bruno et al. (2010) conclude that it is largely retinotopic. It could be argued that the different outcomes are a result of different methodologies being employed by the two research groups. For instance in Bruno et al.’s paradigm the test and comparison were presented in the same hemifield for the three saccade conditions, while Burr et al. had the test and comparison in different hemifields for the spatiotopic and control conditions. However, when adopting the same spatial configuration as used by Burr et al., Bruno et al.’s data were still consistent with retinotopic-tuned timing mechanisms. While there were other minor differences in
the two approaches (Bruno et al.’s stimuli were smaller and their oscillating rate higher than Burr et al.’s) it seems unlikely that these would account for the research groups’ diametrically opposed results. However, there is a difference between the two methodologies which, Burr et al. (2011) argue, may explain the differing results. Whereas Bruno et al. assigned the same physical speed to both the test and comparison stimuli, Burr et al. used both physically speed-matched and perceptually speed-matched test and comparison stimuli. In the physically speed-matched paradigm Bruno et al. (2010) report a strong retinotopic and a smaller spatiotopic duration compression effect, whereas Burr et al. (2007, 2011) find the opposite pattern of results. However, Burr et al. report that, whereas retinotopic duration compression disappears, spatiotopic duration compression persists when the test and comparison stimuli speeds are perceptually matched. Burr et al. explain the presence of retinotopic duration compression in the physically speed-matched condition by noting that perceived test speed following adaptation is slower than that of a comparison stimulus moving at the same physical speed in an unadapted region, and argue that the observed retinotopic duration compression merely reflects previous findings that stimulus speed influences apparent duration.

Despite intensive experimentation by these two research groups, there remains strong disagreement on whether adaptation-induced duration compression is driven by retinotopic- or spatiotopic-tuned temporal mechanisms. In an attempt to resolve this issue we used a different stimulus set to address the retinotopic-spatiotopic question. Given the involvement of pre-cortical and cortical mechanisms in the duration compression effect (Bruno et al., 2013; Curran & Benton, 2012), finding evidence that it may be driven by mechanisms operating within a spatiotopic frame of reference necessitates the use of stimuli known to adapt cortical mechanisms. To this end we used random dot stimuli with a speed of 3°s⁻¹, which are known to induce direction-contingent duration compression (Curran & Benton, 2012). Since direction-tuned neurons are only found in cortex, it follows that direction-specific duration compression will occur only as a consequence of adapting cortical mechanisms. We know that the LGN and area V1 display precise retinotopic mapping; while there is evidence for retinotopic mapping in area MT+, it appears to be rather coarse in comparison to these earlier stages in the motion pathway (Gattass & Gross, 1981; van Essen et al., 1981). However, there is evidence that spatiotopic mapping takes place in area MT+ (d’Avossa et al., 2007; Goosens et al., 2006). Thus evidence of spatiotopic duration compression would point definitively to involvement of spatiotopic-tuned temporal mechanisms in area MT+. If the mechanisms responsible for encoding duration of subsecond motion events are located at the early stages of the motion pathway, we would expect duration compression to occur only within a retinotopic frame of
reference. However, the converse does not apply. That is retinotopic duration compression would not rule out involvement of area MT+; it would demonstrate that, if they contribute to the effect, the relevant temporal mechanisms in MT+ would necessarily operate within retinotopic coordinates.

Our results from the ‘full adaptation’ condition were consistent with previous accounts, showing robust duration compression when test stimulus coordinates matched both the retinal and spatial coordinates of the adapting stimulus. We also obtained strong robust retinotopic duration compression. Whilst we cannot rule out the existence of a spatiotopic component for our stimulus, it is clear that the pattern of results that we obtained is the opposite of that reported by Burr et al. (2007, 2011), despite the fact that in both situations comparison stimulus speed was matched to perceived test speed. They found adaptation-driven duration compression to be primarily spatiotopic, yet we found it to be primarily retinotopic. We have also demonstrated that our retinotopic duration compression effect is direction contingent; i.e. the effect was evident when adapt and test stimuli had the same motion direction, but it was absent when they moved in opposite directions. This shows that the effect was being driven by cortical mechanisms.

It is probable that the contrasting results from our experiments and Burr et al.’s are a consequence of the difference in stimulus parameters between the two setups. While Burr et al. used narrowband sine wave gratings, we used spatially and temporally broadband RDKs. Another difference between the two setups was the motion characteristics of our adaptors; in contrast to Burr et al., who used oscillating adaptor motion, our adapting stimuli were unidirectional. It is likely that these differences between adaptors would lead to different patterns of sustained activity (and hence adaptation) within and/or between neural systems that show a motion-dependent response. If the perceived duration of dynamic stimuli were dependent upon the responses distributed across such systems, then the different patterns of aftereffect could conceivably result from those different patterns of adaptation. However, whilst this might provide a mechanism to explain the difference between our results and those of Burr et al, we are currently unable to explain why, within such a framework, our adaptors should target retinotopic systems whilst those of Burr et al. should target spatiotopic ones.

While test and comparison stimuli for the retinotopic condition were both presented in the right hemifield, for the spatiotopic condition the test was presented in the left hemifield and the comparison in the right hemifield. However, it is unlikely that the absence of spatiotopic duration compression would be explained by this hemifield difference in test stimulus location. Vicario et al.
(2008) report that duration of static stimuli presented in the left hemifield is underestimated while that of stimuli presented in the right hemifield is overestimated. This hemifield difference in duration estimation was not reliant on prior adaptation, a situation which is analogous to our control condition in which both the test and comparison stimuli were presented in non-adapted regions of the left and right hemifields, respectively. The absence of duration distortion in the control condition suggests that there was no inter-hemifield difference in duration estimation, thus making it unlikely that the results of the spatiotopic condition can be explained by appealing to inter-hemispheric differences in duration encoding.

To conclude our results demonstrate that our adaptation-induced duration compression is primarily driven by mechanisms operating within a retinotopic coordinate system. We have also shown duration compression to be direction contingent, thus demonstrating the contribution of cortical mechanisms to the effect. Of course this does not rule out pre-cortical involvement in duration encoding, for which there is compelling evidence (Bruno et al., 2010; Johnston et al., 2008); rather, it supports the view that mechanisms involved in duration encoding occur at multiple levels of the motion processing pathway. While there is evidence that MT+ is implicated in duration encoding of brief visual events (Bueti et al., 2008; Burr et al., 2007; Curran and Benton, 2012), the lack of evidence for spatiotopic duration compression in our experiments suggests that, if MT+ timing mechanisms were adapted with our paradigm, they likely encode duration within a retinotopic frame of reference.
References


Figure Legends

**Figure 1.** (a) The locations of the adaptor and test stimuli for each condition. The location of the fixation is represented by an ‘x’. During adaptation the adaptor was centred 4.2° to the right and 3.5° above fixation. Following adaptation the fixation either remained in the same location (full adaptation condition) or moved 7.6° to the right. In the ‘full adaptation’ condition the test stimulus had the same spatiotopic and retinotopic coordinates as the adapting stimulus. In the spatiotopic condition the test stimulus was centred on the same screen coordinates as the adaptor (3.4° to the left and 3.5° above fixation). In the retinotopic condition the test stimulus was presented at the same retinal location as the adaptor (centred 4.2° to the right and 3.5° above fixation). In the control condition the test stimulus was centred 3.4° to the left and 3.5° below fixation. The comparison stimulus (not shown) was centred 4.2° to the right and 3.5° below fixation in all four conditions. Each stimulus contained equal numbers of black and white dots (12.9 dot/deg²) presented on a mean luminance background (28.5 cd/m²). (b) Experimental timeline showing the retinotopic condition.

**Figure 2.** Percentage change in perceived duration of the test stimulus relative to its actual (600ms) duration in (a) the ‘no saccade’ full adaptation condition, (b) the ‘same direction’ retinotopic condition, (c) the spatiotopic condition, (d) the control condition, and (e) the ‘opposite directions’ retinotopic condition. Negative values indicate duration compression. There was significant duration compression in the ‘no saccade’ full adaptation condition and in the retinotopic (same direction) condition, and there was a small duration compression effect close to significant in the spatiotopic condition. No significant duration was observed in either the control condition, in which test and comparison stimuli were located in unadapted regions, or the retinotopic ‘opposite directions’ condition. 95% confidence intervals were generated by parametric bootstrapping (10000 iterations, percentile method; Wichmann & Hill, 2001). Underlined initials indicate the three authors.
Figure 1