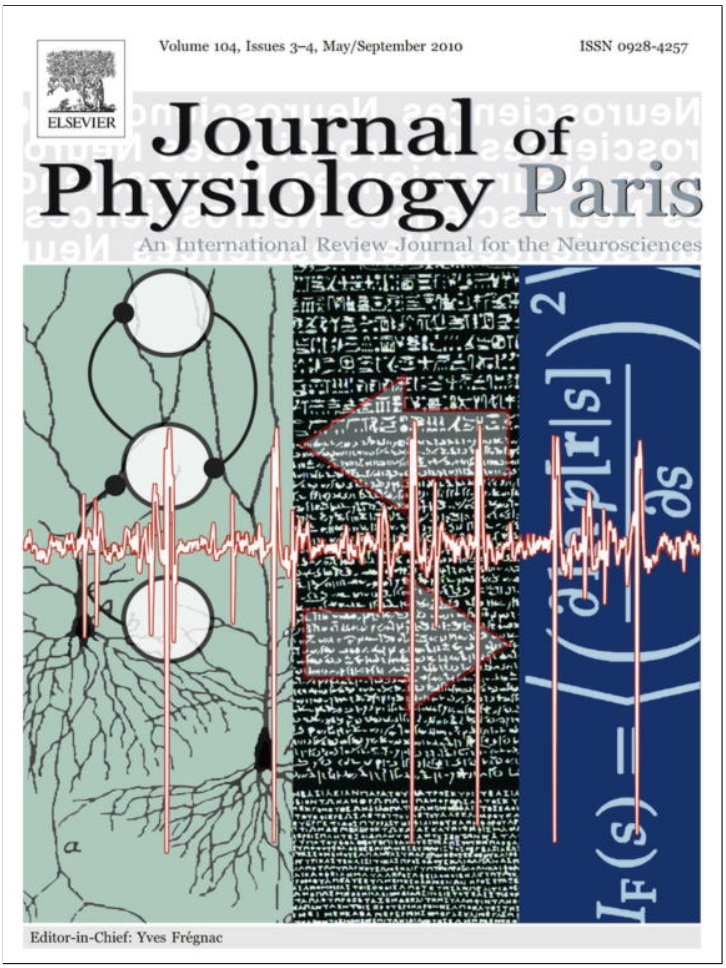

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Contrast induced changes in response latency depend on stimulus specificity

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ABSTRACT

Neurones in visual cortex show increasing response latency with decreasing stimulus contrast. Neurophysiological recordings from neurones in inferior temporal cortex (IT) and the superior temporal sulcus (STS), show that the increment in response latency with decreasing stimulus contrast is considerably greater in higher visual areas than that seen in primary visual cortex. This suggests that the majority of the latency change is not retinal or V1 in origin, instead each cortical processing area adds latency at low contrast. I show that, as in earlier visual areas, response latency is more strongly dependent on stimulus contrast than stimulus identity. There is large variation in the extent to which response latency increases with decreasing stimulus contrast. I show that this between cell variability is, at least in part, related to the stimulus specificity of the neurones: the increase in response latency as stimulus contrast decreases is greater for neurones that respond to few stimuli compared to neurones that respond to many stimuli.

1. Introduction

Neuronal response latency – the time when the stimulus elicited neuronal signal can first be detected – is an example of a neuronal code involving precisely timed spikes (see Oram et al., 2002 for review). Given the dissociation of response magnitude and response latency (Albrecht, 1995; Bair et al., 2002; Carandini and Heeger, 1994; Gawne et al., 1996; Reich et al., 2001a,b) it is not surprising that response latency of individual neurons in primary visual cortex convey information unavailable from the spike count (Gawne et al., 1996; Reich et al., 2001a,b). Indeed, it is has been speculated that changes in response latency are a potential source of the temporal code revealed by principal component and information theoretic analysis of spike waveforms (Optican and Richmond, 1987; Tovee et al., 1993). Thus, understanding factors that influence neuronal response latency may be relevant to studies examining the role of temporal variation in firing rate in visual processing (Eskandar et al., 1992; Heller et al., 1995; McClurkin et al., 1991; Optican and Richmond, 1987; Richmond and Optican, 1990) as well as shed light on the temporal precision of neuronal codes.

The latency of visually responsive neurones in the visual system increases with decreasing stimulus contrast in the retina (Shapley and ictor, 1978), LGN (Lee et al., 1981b), primary visual cortex (Albrecht, 1995; Carandini et al., 1997, 2002; Carandini and Heeger, 1994; Gawne et al., 1996; Movshon et al., 1978; Parker et al., 1982; Reich et al., 2001a,b; Wiener et al., 1998), area MT (Raiguel et al., 1999) and the anterior superior temporal sulcus (Oram et al., 2002; van Rossum et al., 2008). The increment in response latency with decreasing stimulus contrast is considerably greater in higher visual areas such as the anterior superior temporal sulcus (STSa) than that seen in primary visual cortex Fig. 1 and (Oram et al., 2002; van Rossum et al., 2008). Indeed, the average response latency in area STSa increases by 33 ± 3 ms for each halving of stimulus contrast compared to 8 ± 0.8 ms in V1 (van Rossum et al., 2008). The increasing dependency of neuronal response latency on stimulus contrast indicates that latency change is not retinal or V1 in origin, instead suggesting that each cortical processing area adds latency at low contrast (van Rossum et al., 2008).

Stimulus properties other than stimulus contrast influence response latency. For example, changes in spatial frequency influence both response magnitude and response latency of many V1 neurones (Bredfeldt and Ringach, 2002; Mazer et al., 2002). Position of moving gratings relative to the receptive field also influence response latency (Lee et al., 1981a), as does luminance of the stimulus (Maunsell and Gibson, 1992). On the other hand, changes in response magnitude do not necessarily influence latency in V1 (Albrecht et al., 2002; Gawne et al., 1996; Geisler and Albrecht, 1995; Opara and Worgotter, 1996; Reich et al., 2001a,b; Tolhurst and Heeger, 1997; Worgotter et al., 1996). Similarly, response latency of individual neurons in STSa show little dependency on response magnitude (Oram et al., 1993; Oram and Perrett, 1996, 1992) whereas changes in stimulus contrast cause large changes (>200 ms) in response latency (Oram et al., 2002; van Rossum et al., 2008; York et al., 2007).

In this article, I present data indicating that processing complexity may also influences neuronal response latency. Specifically,

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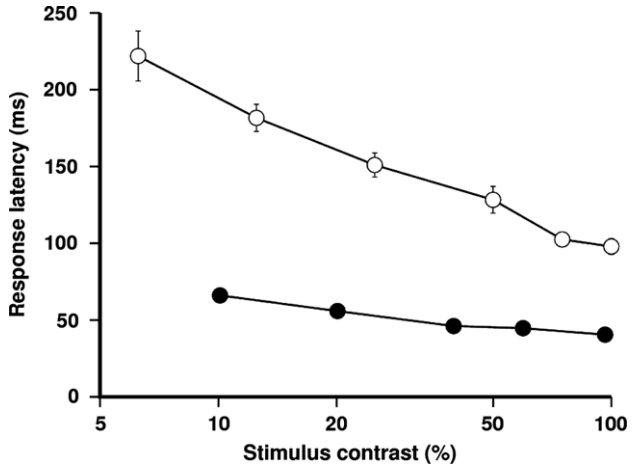

Fig. 1. Stimulus contrast influences response latency more in late visual areas than in early areas. Mean latency (±SEM) is plotted for primary visual cortex (V1, solid symbols, error bars lie under the symbol) and anterior superior temporal sulcus (STS, open symbols), adapted from van Rossum et al. (2008).

I show that the response latency of neurones that respond to a small number of stimuli is more sensitive to changes in stimulus contrast than neurones that respond in a less discriminative or selective fashion. The findings are discussed in terms of current models of visual processing.

2. Methods

The experimental protocols have been described before (Oram et al., 2002; van Rossum et al., 2008). Briefly, extra-cellular single-unit recordings were made using standard techniques from the upper and lower banks of the anterior part of the superior temporal sulcus (STSa) and the inferior temporal cortex of two male monkeys (Macaca mulatta) performing a visual fixation task. The subject received a drop of fruit juice reward every 500 ms of fixation (±3°) while static stimuli (10° by 12.5°) were displayed. During initial screening, images of different perspective views of monkey and human head, animals, fractal patterns, natural scenes, and everyday objects were presented for 110 ms. Visual inspection of on-line rasters and the post-stimulus time histogram (PSTH) to each visual stimulus allowed selection of stimuli that were effective (preferred) and non-effective (non-preferred) in eliciting a response from the recorded neuron.

2.1. Stimuli

Grey-scale images of the cell's preferred and non-preferred stimuli were presented for 333 ms with 333 ms inter-stimulus interval at different contrast levels in random order. Stimulus contrast was determined using foreground regions of the image. The 100% Michelson contrast $(L_{max} - L_{min})/(L_{max} + L_{min})$ was formed by normalising the foreground pixel values such that they occupied the monitor's full luminance range after adjusting the initial grey-scale image to have mid (50%) luminance. Other contrast versions (75%, 50%, 25%, 12.5%, and 6.25%) were achieved by systematically varying the width of the distribution of the foreground pixel values of the 100% contrast version while maintaining the average foreground luminance. Example stimuli are shown in Fig. 2. All manipulations were performed after correcting for the measured gamma function of the display monitor.

2.2. Data analysis

Spike density functions were computed by smoothing a 1 ms bin-width peri-stimulus time histogram with a Gaussian filter (s.d. = 10 ms) for each stimulus at each contrast. *Response magnitude* was taken as the average firing rate in the 333 ms following response latency. The *response latency* was extracted as the point at which the activity exceeded the baseline activity (estimated using the 200 ms before stimulus onset) by three standard deviations for a period of at least 20 ms. The latency was only accepted if the activity of the neuron in the 100 ms following the estimate was significantly ($p < 0.05$) above the baseline activity (paired *t*-test). *Population responses* were generated by normalising the spike density function of each cell to the most effective stimulus by setting the average of the 200 ms prior to stimulus onset to 0 and the peak of the spike density function to 1, average across neurons, and re-normalizing to the range 0–1 (Barraclough et al., 2005; Oram and Perrett, 1996, 1992).

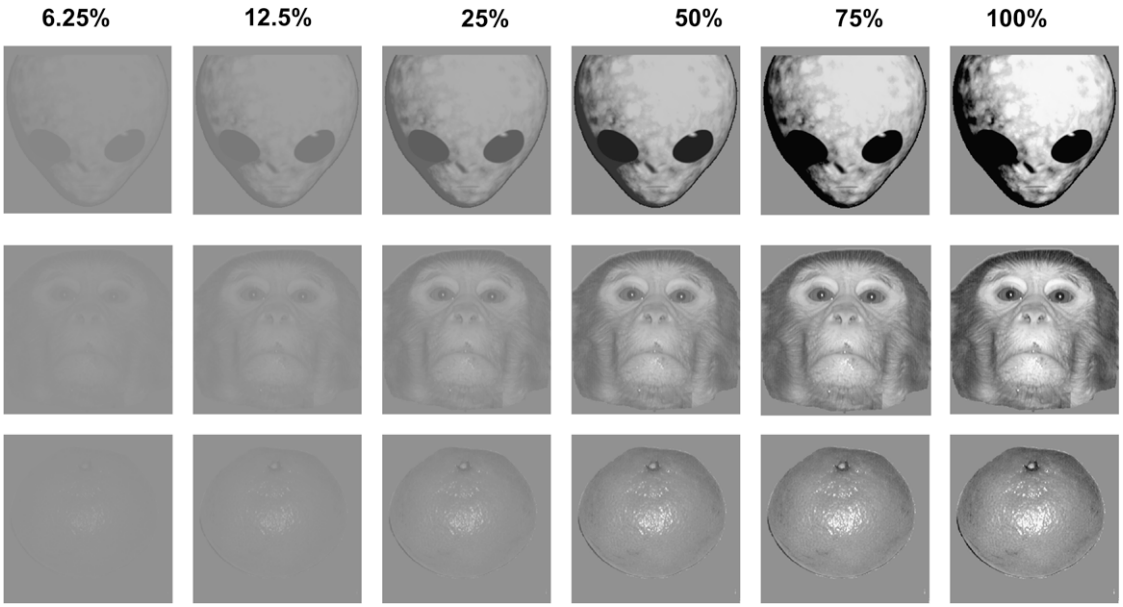

Fig. 2. Example stimuli at different contrasts.

2.3. Histological reconstruction

After each electrode penetration X-ray photographs were taken coronally and para-sagitally. The positions of the tip of each electrode and its trajectory were measured with respect to the intra-aural plane and the skull's midline. Following the final recording session, the subject was sedated with ketamine, and then administered a lethal dose of barbiturate anaesthetic. After transcardial perfusion with phosphate buffered saline and 4% glutaraldehyde/paraformaldehyde fixative, the brain was removed and soaked in successively higher concentrations of sucrose solution or 2% dimethylsulphoxide and 20% glycerol (Rosene et al., 1986). Sections (25 μm) were taken every 500 μm using standard techniques (Harries and Perrett, 1991). Every 0.1 mm during sectioning slide photographs of the remaining tissue were taken. Using the distance of each recorded neuron along the penetration, a three-dimensional map of the position of the recorded cells was calculated. Alignment of sections with the X-ray co-ordinates of the recording sites was achieved using the location of microlesions and injection markers on the sections.

The anterior–posterior extent of the recorded cells was from 7 mm to 9 mm anterior of the inter-aural plane (Fig. 3), consistent with previous studies showing visual responses to static images in this region (Baylis et al., 1987; Bruce et al., 1981; Oram and Perrett, 1992; Perrett et al., 1982). The recorded cells were located in the upper bank (TAa, TPO), lower bank (TEa, TEm) and fundus (PGa, IPa) of STS and in the anterior areas of TE (Tanaka et al., 1991). As defined in previous studies (Barraclough et al., 2006, 2005; Baylis et al., 1987; Bruce et al., 1981, 1986; Desimone and Gross, 1979; Distler et al., 1993; Hikosaka et al., 1988; Saleem et al., 2000; Seltzer and Pandya, 1994; van Rossum et al., 2008) these areas are rostral to FST and we collectively call the anterior STS (Barraclough et al., 2006, 2005; van Rossum et al., 2008).

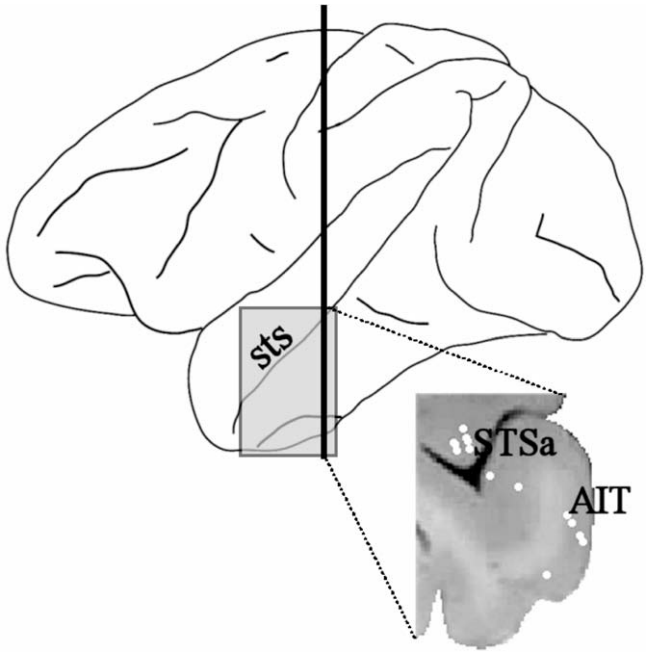

Fig. 3. Histological reconstruction of recording sites. Upper: schematic lateral view of the macaque brain showing the target recording area. Inset: photograph of the macaque brain at 8 mm anterior to the inter-aural plane. White dots mark the location of recorded neurones on this slice from monkey P.

3. Results

3.1. Location of recorded neurones

Recordings were made from 55 neurones in STSa and AIT in two male monkeys. Fig. 3 shows the location of recorded neurones following histological reconstruction. The neurones were located in the upper bank, fundus and lower bank of the STS and in the lateral portion of AIT. No statistical differences in response latency or stimulus specificity were found between the recorded locations. In view of the absence of differences between areas, I report further population statistics after collapsing between areas.

3.2. Stimulus contrast and response latency

The responses of a single neurone in area STSa to stimuli of different contrasts are shown in Fig. 4. For this neurone, the response latency at high (100%, solid line) contrast was 68 ms, increasing to over 416 ms at 6.25% contrast (dotted line). An increase in response latency of ~100 ms when stimulus contrast is reduced from 100% to 6.25% is typical of neurones in STSa (Oram et al., 2002; van Rossum et al., 2008). Note that as stimulus contrast decreases the response magnitude decreases (Oram et al., 2002), as seen in primary visual cortex (Albrecht, 1995; Albrecht and Hamilton, 1982; Carandini et al., 2002; Carandini and Heeger, 1994; Gawne et al., 1996; Movshon et al., 1978; Parker et al., 1982), but the sharp response onset is retained (Oram et al., 2002; van Rossum et al., 2008).

The average responses of the 55 neurons recorded in area STSa to preferred visual stimuli presented at different contrasts are plotted in Fig. 5. The data show large changes in response latency as a function of contrast. Note that the sharp response onset to low contrast stimuli is not seen because of averaging over increasing between cell temporal variation in response latency. The average

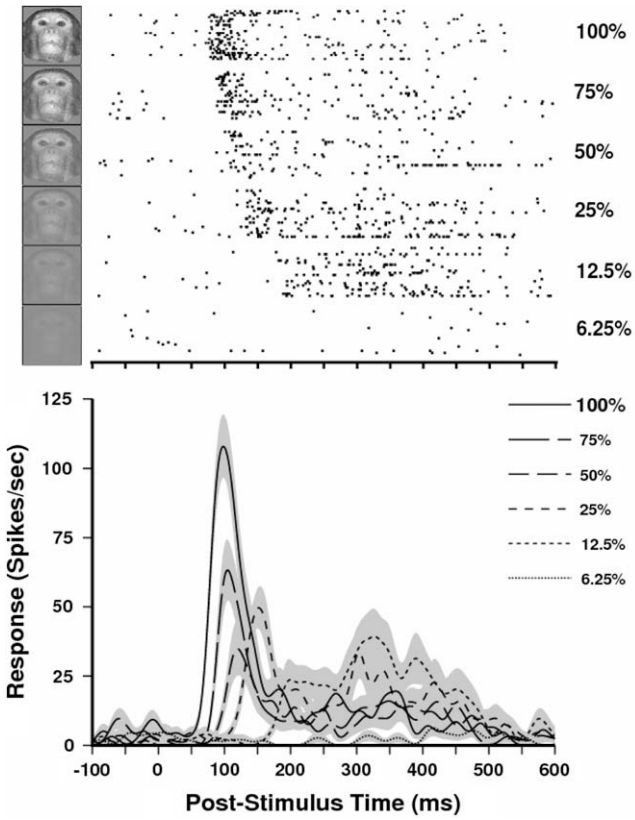

Fig. 4. Responses of an STSa neurone to stimuli of different contrasts. Upper: rastergrams of responses to stimuli shown on the left. Percentage figures give the actual contrast. Lower: spike density functions (kernel σ = 10 ms) of the responses above (shaded region = ±SEM).

latency in the population response ranges from 90 ms for the highest contrast stimuli, to 216 ms for the lowest contrast stimuli.

3.3. Contrast, not stimulus preference determines the latency

Fig. 6 shows the average spike density function of 50 STS neurones to effective stimuli when presented at high (100%) contrast (solid line). When effective stimuli are presented at low (25%) contrast there is a noticeable increase in response latency (dashed). Responses to ineffective stimuli (dotted line) have the same latency as the most effective stimuli despite the very small response (Oram et al., 2002). These findings are indicative that response latency is relatively sensitive to stimulus contrast and relatively independent of response magnitude.

Following (Gawne et al., 1996), the extent to which response amplitude and latency varied with stimulus identity and stimulus contrast was examined. Neuronal response latency varied with stimulus contrast in a systematic manner regardless of response magnitude. An example illustrating this is shown in Fig. 7. The neurone responded well to the face view but poorly to the back view of the head. When the estimated latency is plotted as a function of response strength elicited by the effective stimulus (face), a clear relationship between response strength and latency is observed (Fig. 7a, solid symbols). This relationship is, however, poor for the ineffective stimulus (back view of the head, Fig. 7a, open symbols). However, when response latency is plotted as a function of stimulus contrast a consistent relationship is observed regardless of stimulus identity (Fig. 7b).

For recorded cells tested with different stimuli eliciting significantly different response strengths (spike counts 70–370 ms, ANOVA, $p < 0.05$), stimulus contrast accounted for 67 ± 7% of the variability of response latency and only 33 ± 3% of the variability in spike count. Conversely, stimulus identity accounted for 69 ± 6% of the variability in spike count and only 20 ± 5% of the variability on response latency (Fig. 7c). Thus, in areas STSa and IT stimulus contrast is encoded mostly by response latency whereas stimulus identity is encoded mostly by response magnitude (Oram et al., 2002; van Rossum et al., 2008), the same reversal as observed in V1 (Gawne et al., 1996; Reich et al., 2001b).

3.4. Stimulus specificity determines contrast sensitivity

The neurophysiological recordings from some neurones in higher visual areas can show increases in response latency of up to

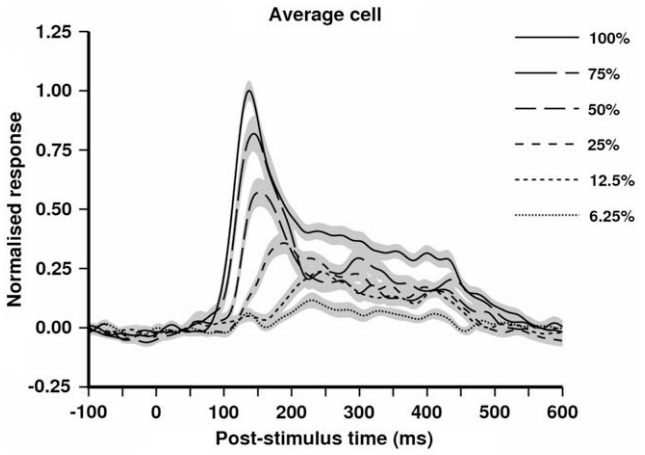

Fig. 5. Average response of STSa neurones to effective stimuli at different contrasts. Response (±SEM) of 55 neurones to stimului of 100–6.25% contrast are shown. The response of each neurone was normalised (peak response at 100% contrast = 1, spontaneous activity = 0) and averaged. For plotting purposes, the average was re-normalised.

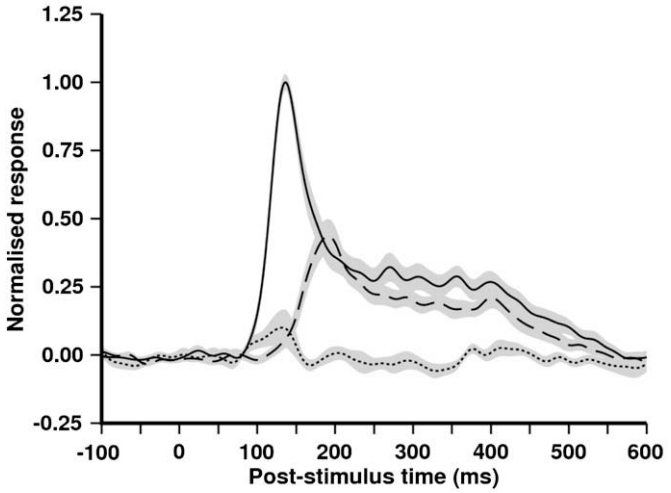

Fig. 6. Stimulus contrast influences response latency more than response magnitude. The average response (±SEM) across 50 neurones to the most effective stimulus (best) at high (100%) and low (25%) contrast (solid and dashed line respectively). The change in contrast induces a latency shift of some 70 ms. In comparison, the almost negligible population response to high (100%) contrast ineffective stimuli (dotted line) has a latency that is indistinguishable from the most effective high contrast stimuli.

300 ms when stimulus contrast is reduced to 6%. Other neurones, however, show barely detectable increases of less than 5 ms over the same range of stimulus contrasts. Example rastergrams and spike density functions from single neurones illustrating this asynchrony are shown in Fig. 8. In Fig. 8a, the response latency of a neurones increases markedly with decreasing stimulus contrast. Another neurone (Fig. 8b) showed almost no change in response latency. Note that the changes in response magnitude are equivalent ($p > 0.1$) and so cannot account for the different latency–contrast relationships of these two neurones. Because the sensitivity of response latency to stimulus contrast varies between neurones, neurones in IT and STS that respond to the same visual stimulus become increasingly asynchronous as stimulus contrast is reduced (van Rossum et al., 2008).

Increasing asynchrony of visual responses at low contrast could simply reflect heterogeneity of neurones. It is also possible that such asynchrony reflects functional aspects of visual processing. One functional aspect of visual processing frequently investigated is the notion of "tuning width": the sensitivity of the neuronal responses to changes in the input stimulus. While tuning width is well defined for simple static stimulus sets such as gratings (spatial frequency, phase, and orientation), it is less well defined for neuronal responses found in higher association cortex.

During initial screening of the neuronal responses (see Section 2) a relatively variable stimulus set was used (all stimuli at 100% contrast). Fig. 9 illustrates the responses to screening set stimuli of two example neurones. At one end of the spectrum are highly selective neurones which respond strongly to only one or two images. Strong responses from the neurone shown in Fig. 9a were elicited by only the image of an alien face (bottom right of the figure), with the neurone responding slightly to the image of a hand (near the centre of the figure) with the other tested stimuli eliciting little or no response. At the other end of the spectrum are neurones which seem non-selective, responding to all the visual stimuli used in the screening set (Fig. 9b).

A median split according to the number of stimuli that elicit a significant ($p < 0.05$) response separated the neurones into two group. One group of neurones responded to (relatively) many stimuli (low specificity), the second group responded to a small number of stimuli (high specificity). The sensitivity of response latency

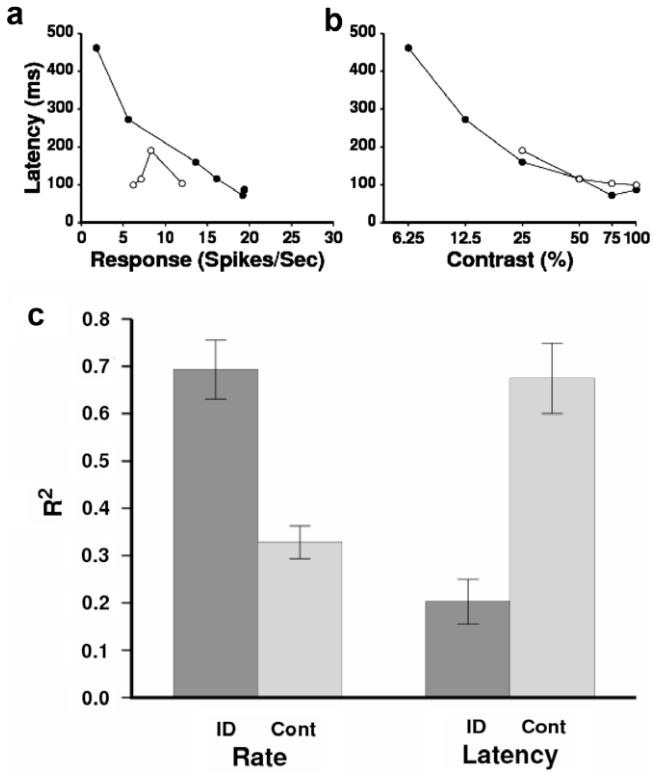

Fig. 7. Stimulus contrast influences response latency more than response magnitude. (a) Relationship between response latency and response strength. (b) Relationship between latency and stimulus contrast. Recordings from this neurone were made while images of the face (solid symbols) and back (open symbols) view of the head were presented. For this neurone, as with most others, response latency is better described as a function of stimulus contrast than of response strength (Response latency – response strength: $R^2 = 0.46$; Response latency – stimulus contrast: $R^2 = 0.91$). (c) Average R^2 of regressions of stimulus identity (ID) or stimulus contrast (Cont) on response rate (left) and response latency (right). The proportion of variability in response rate explained by stimulus contrast (33 ± 3%) was less (paired t-test: $t = 5.8$, $p < 0.001$) than that explained by stimulus identity (69 ± 6%). Conversely, the proportion of variability in response latency explained by stimulus contrast (67 ± 7%) was greater ($t = 4.7$, $p < 0.002$) than that explained by stimulus identity (20 ± 5%).

with stimulus contrast differs between these two groups (Fig. 10). Specifically, the response latency of neurones that respond to many stimuli is less sensitive to change in stimulus contrast than those neurones which respond to only a small number of stimuli. Regression of latency on ln(stimulus contrast) for each neurone confirmed the impression from the population analysis. Response latency increased, on average, by 21.0 ± 3.3 ms for each halving of stimulus contrast for the low specificity neurones compared to an increase of 43.8 ± 5.7 ms for the high specificity group. Finally, the proportion of stimuli in the screening set that elicited a significant response also correlated with the slope of the latency–ln(contrast) relationship (Spearman's correlation coefficient = 0.39, $p < 0.01$), again indicating that the more specific the neuronal selectivity the greater the impact of contrast on response latency.

4. Discussion

The results presented here show that, as in V1, neuronal response latency is heavily influenced by stimulus contrast and is relatively independent of response magnitude. Notably, the changes in response latency in STSa are considerably larger than that seen in earlier visual areas (Oram et al., 2002; van Rossum et al., 2008). However, the average increase in response latency as contrast is reduced hides considerable between cell variability in the relationship between stimulus contrast and response latency. This variability in response latency, as with neuronal response latency in general (Albrecht et al., 2002; Bair et al., 2002; Gawne et al., 1996; Geisler and Albrecht, 1995; Opara and Worgotter, 1996; Oram et al., 2002, 1993; Oram and Perrett, 1996, 1992; Reich et al., 2001a,b; Tolhurst and Heeger, 1997; van Rossum et al., 2008; Worgotter et al., 1996; York et al., 2007), cannot easily be attributed to changes in response magnitude. The specificity of the neuronal responses, here measured simply as the proportion of stimuli that elicit a detectable response, captures at least part of the between cell variability in the contrast–latency relationship.

The analyses presented here do not differentiate between neurones in the upper bank, fundus, lower bank of the STS and lateral AIT. Given that fMRI studies suggest that neurones from these different areas may have different sensitivity to motion (Nelissen et al., 2006) treating the neurones in different sub-regions of STSa as equivalent needs to be treated with caution. However, neurones were included in this study only if they responded to static stimuli, suggesting that differences based on motion selectivity are unlikely to impact on the present results. Furthermore neurones with high and low selectivity were found in the upper bank, fundus lower bank of the STS and lateral AIT. Nevertheless, it remains a possibility that between sub-division difference exist.

4.1. Implications for visual processing

The cell-by-cell differences in the latency–contrast relationship mean that signals will become increasingly asynchronous as the stimulus contrast is reduced, particularly in the higher visual areas such as STSa (van Rossum et al., 2008). While it has been shown that information unavailable from spike count is carried by response latency (Reich et al., 2001a,b), it is not immediately obvious how this information could be used by receiving neurons. The problem for the nervous system in determining response latency (time from stimulus onset to response onset) is undeniable. However, decoding of absolute response latency is not, under normal viewing conditions, necessary. While some sections or areas of a visual scene are likely to contain stimuli – or parts of stimuli – with high contrast features (e.g. object outlines), other areas are likely to contain elements with lower contrast (e.g. internal features of the objects). Thus, relative response latency, rather than absolute latency, can be used. Indeed, a recent study has shown that stimulus related information is encoded by relative rather than absolute response latency in the salamander retina (Gollisch and Meister, 2008).

It has argued that visual processing may, in part, operate using a first-come winner-take-all mechanism (Gautrais and Thorpe, 1998; Perrinet et al., 2004; Thorpe, 1990; Van Rullen et al., 2001; Van Rullen and Thorpe, 2002). The use of relative response or spike latency in decoding may help explain the robustness of human vision against large reductions in stimulus contrast (Mace et al., 2005). However, the increasing asynchrony – up to 200 ms or more – between neurons to low contrast stimuli will disrupt relative spike times between different populations of neurones as much as it disrupts the spike times within populations. Thus, the results reported here suggest that the robustness of relative spike times against changes in stimulus contrast (Mace et al., 2005) is likely to be less than previously argued.

While the current results relate directly to visual processing utilising response latencies, the results also open up the far greater challenge of dealing with asynchronous signals in general. At the subjective phenomenological level, the present results are appealing. When viewing stimuli in low contrast conditions (e.g. fog), the sensation is of an indistinct object gaining form and detail over time. This is commensurate with early signals being indiscriminate (from low specificity neurons) and later signals showing greater

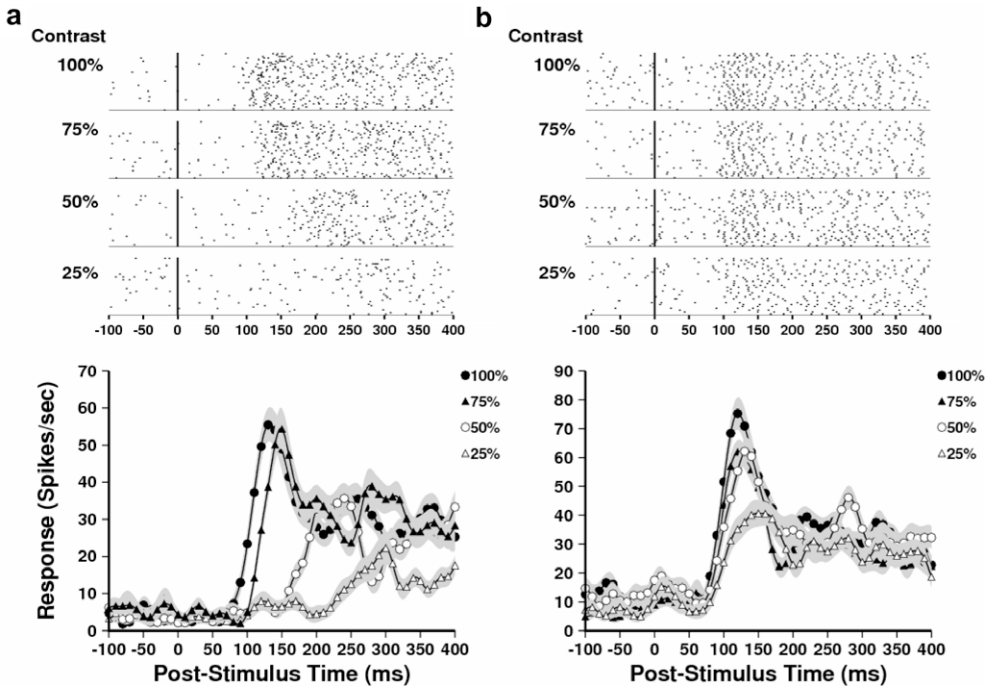

Fig. 8. Different neurones show different changes in response latency as contrast changes. (a) Neurone showing large latency increases as stimulus contrast is reduced. (b) Neurone showing little latency increase as stimulus contrast is reduced. The relative reduction in response magnitude is almost identical for these two neurones, confirming the independence of response magnitude and response latency (see also Figs. 7 and 8).

discrimination (from high specificity neurons). At low stimulus contrast, integration of early signals would provide coarse information, integrators of later signals providing more detailed information. At high stimulus contrast, the information from all integrators would be available simultaneously, allowing subsequent processing to utilise the output associated with detailed information. This is, of course, no more than a restatement of the results and as such assumes that asynchronous signals are not integrated.

Response asynchrony at low contrast will occur in any pool of neurones unless they all share the same contrast–latency relationship. Hence, it is necessary to consider what sorts of mechanism could allow for integration of asynchronous signals without inducing inherent instability (incorrect integration). A potential candidate would be a mechanism that low-pass filters, integrates and then thresholds the inputs (e.g. Bugmann and Taylor, 1993).

Such integrate and threshold models are becoming increasingly dominant in modelling decision making processes. The activity of single neurones can be viewed as evidence supporting the presence of a particular visual stimulus (Barlow, 1972, 1985). Once the evidence or information exceeds threshold, a decision about the presence or absence of the stimulus can be made (Loftus and Ruthruff, 1994). The visual information acquisition and evidence accumulation hypotheses are captured by integrate and threshold processes such as diffusion and accumulator models (Carpenter and Williams, 1995; Gold and Shadlen, 2001; Oram et al., 2002; Palmer and McLean, 1996; Perrett et al., 1998; Ratcliff and Rouder, 2000; Roitman and Shadlen, 2002; Schall, 2002, 2003; Schall and Thompson, 1999; Usher and McClelland, 2001; Verghese, 2001; Ward and McClelland, 1989). Of particular relevance here is the observation that integrate and threshold models can explain performance in dual task experiments when signal asynchrony occurs because of contrast induced latency changes (Oram, 2005). While such integrate and threshold models can cope with asynchronous inputs, they cannot explain the dependency of contrast–latency relationship on stimulus specificity described here.

4.2. Implications for models of contrast induced latency change

Models which low-pass filter and then threshold the signal (see above) have been used to explain V1 latencies (Bair et al., 2002). However, models based on the integrate and threshold act locally and typically predict a relationship between firing rate and latency, a feature which is not observed in the STSa responses described here (see van Rossum et al., 2008 for further discussion). Carandini and Heeger (1994) have suggested inhibitory shunting feedback reduces the membrane time constant at high contrast. However, the effective membrane time constant observed in V1, with a resting membrane time constant typically <20 ms (Anderson et al., 2000; Destexhe et al., 2003; Destexhe and Pare, 1999; Hirsch et al., 1998) is too short to allow for the required ~50 ms decrease in the time constant required to explain contrast induced latency changes observed in V1 (Albrecht, 1995; Gawne et al., 1996; Gawne, 2000). Additionally, physiologically observed levels of inhibition do not match the shunting model (Ahmed et al., 1997; Anderson et al., 2000), although the inclusion of noise and dendritic saturation suggests that the shunting inhibitory model may be applicable (Prescott and De Koninck, 2003). Finally, computational studies indicate that the even shorter synaptic time constant determines the circuit's dynamics (Treves, 1993).

More recent models explaining latency changes with stimulus contrast have examined the role of synaptic depression in either feed-forward connections (Carandini et al., 2002; Chance et al., 1998), or feed-forward and recurrent connections (Kayser et al., 2001; van Rossum et al., 2008). It has recently been shown that in V1 low contrast stimuli elicit greater synaptic activity (as measured from both magnitude and extent of local field potentials) than high contrast stimuli (Nauhaus et al., 2008). This is consistent with reduced synaptic depression (resulting in more prolonged synaptic activity) at low compared to high contrast and inconsistent with shunting inhibitory models where increased synaptic activity (more shunting) is predicted to high contrast stimuli.

Current models that explain latency shifts as a result of changing stimulus contrast will not, at least in their current forms,

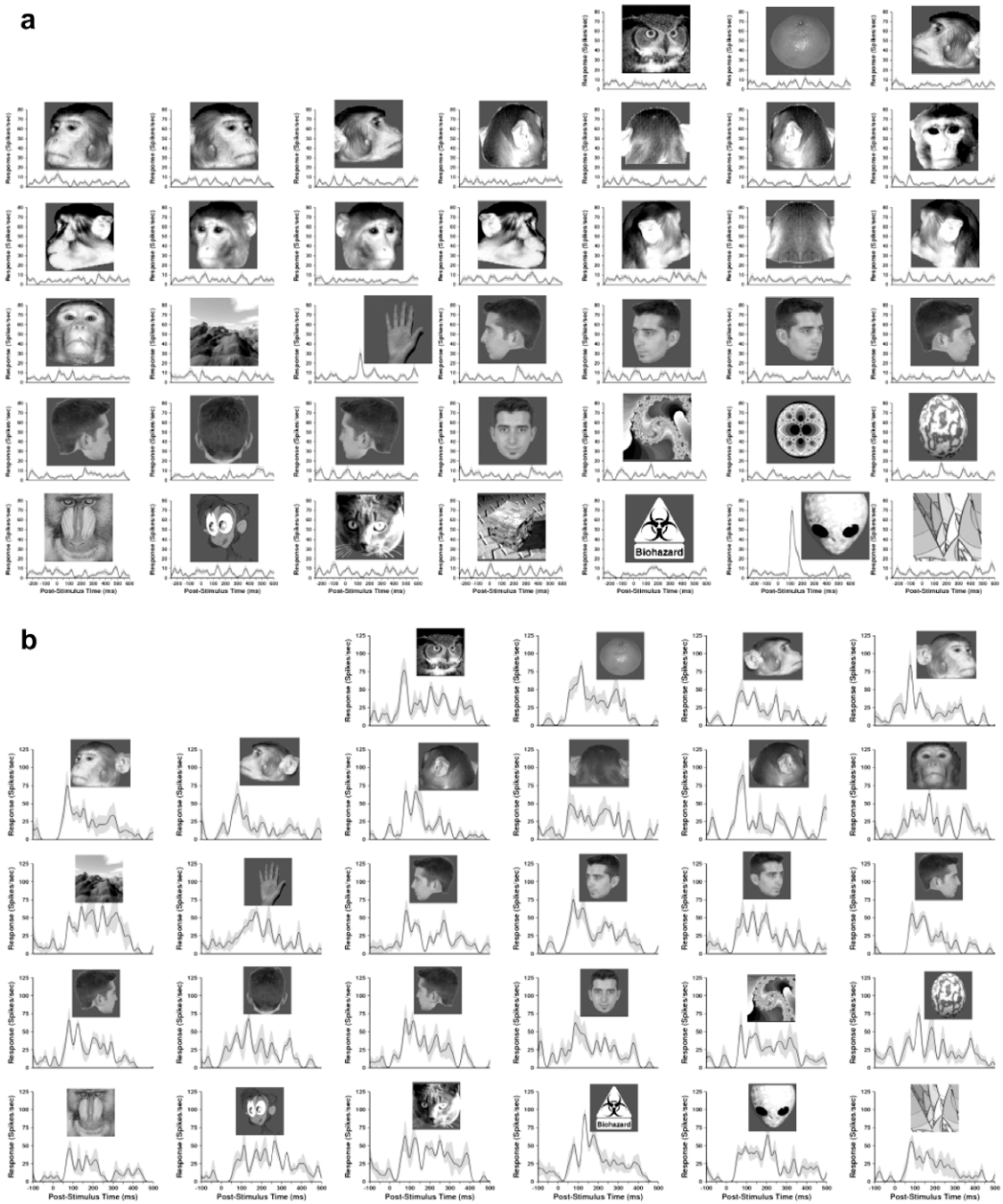

Fig. 9. Stimulus selectivity varies between neurones. (a) An example of a neurone that was highly selective, responding significantly to only 5% (2/38) of the stimuli used in the screening set. (b) Example of a neurone responding significantly to 100% (28/28) of the stimuli used in the screening set.

explain the results reported here. Population based models, such as the shunting inhibition model (Carandini and Heeger, 1994), use pooling of the responses from many neurones with varying stimulus preferences (orientation, spatial frequency, phase, etc.). The use of the pooled activity from many other neurones is assumed to be constant for all neurones and hence would not show the main finding reported here: increases in response latency as stimulus contrast decreases is greater for neurones that respond to few stimuli compared to neurones that respond to many stimuli. Similarly, models with depressing synapses (Carandini et al., 2002; Chance et al., 1998; Kayser et al., 2001; van Rossum et al., 2008) do not differentiate rates of depression on the basis of the specificity of neurons and hence would not automatically show this property. It remains to be seen which of the current models can be adapted to explain the current results.

4.3. Conclusion

Response latency of neurones in STSa is more strongly dependent on stimulus contrast than stimulus identity. There is, however, large variation in the extent to which response latency increases with decreasing stimulus contrast. This between cell variability is, at least in part, related to the stimulus specificity of the neurones: the increase in response latency as stimulus contrast decreases is greater for neurones that respond to few stimuli compared to neurones that respond to many stimuli. These results

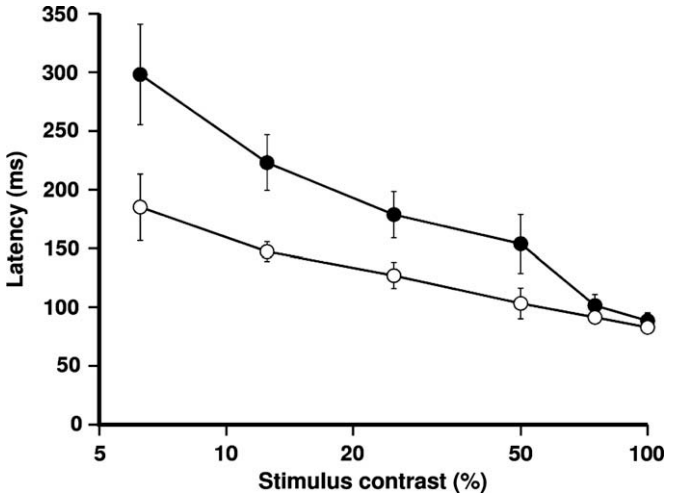

Fig. 10. Sensitivity of response latency to stimulus contrast varies with selectivity. As the stimulus contrast decreased the response latency of neurones which responded to many stimuli (low selectivity, open symbols) increased less rapidly than the response latency of neurones which responded to relatively few stimuli (high selectivity, solid symbols). (Overall ANOVA: effect of contrast, $F_{[5,220]} = 18.9$, $p < 0.00005$; effect of selectivity, $F_{[1,220]} = 16.8$, $p < 0.0005$; interaction $F_{[5,220]} = 4.2$, $p < 0.005$)

challenge current models of contrast gain control and suggest that our understanding of visual processing will require detailed studies of how asynchronous signals can be effectively integrated.

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