Visual stimulation decorrelates neuronal activity

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The accuracy of neuronal encoding depends on the response statistics of individual neurones and the correlation of the activity between different neurones. Here, the dynamics of the neuronal response statistics in the anterior superior temporal sulcus of the macaque monkey are described. A transient reduction in the normalised trial-by-trial variability and de-correlation of the responses with both the activity of other neurones and previous activity of the same neurone is found at response onset. The variability of neuronal activity and its correlation structure return to the levels observed in the resting state 50-100ms after response onset, except for marked increases in the signal correlation between neurones. The transient changes in the response statistics are seen even if there is little or no stimulus elicited activity, indicating the effect is due to network properties rather than activity changes per se. Modelling also indicates that the observed variations in response variability and correlation structure of the neuronal activity over time cannot be attributed to changes in firing rate. However, a reset of the underlying spike generating process, possibly due to the driving input changing from recurrent to feed-forward inputs, captures most of the observed changes. The non-stationarity indicated by the changes in correlation structure around response onset increases coding efficiency: compared to the mutual information calculated without regard to the transitory changes, the de-correlation increases the information conveyed by the initial response of modelled neuronal pairs by up to 24% and suggests that an integration time of as little as 50ms is sufficient to extract 95% the available information during the initial response period.
Introduction

The ability of the brain to encode information is determined by the response characteristics of the individual neurones and the correlation structure between the responses of different neurones. Reports of the neuronal coding of visual stimuli have typically analysed data using sample windows that are fixed both in time and size (e.g. a window size of 250ms starting 50ms after stimulus onset). More recently, studies have started to examine changes in the response characteristics at different time points within the stimulus elicited response (e.g. Muller et al., 2001; Churchland et al., 2006; Amarasingham et al., 2006; Smith and Kohn, 2008). The study of the dynamics of response statistics can help elucidate the properties of underlying neural circuits and constrain computational models in addition to quantifying how dynamic changes in response characteristics influence the stimulus related information carried by the responses.

To determine the impact of changes in response statistics over time in terms of information, it is necessary to measure response variability. For individual neurones, the more variable the responses to a given stimulus, the less information those neurones can encode. The trial-by-trial variability increases from retina to LGN to V1 (Kara et al., 2000) and differences have been noted between cortical areas (e.g. visually responsive neurones in MT/MST show greater trial-by-trial variability than motor related neuronal activity in area5, Maimon and Assad, 2009). Studies frequently report the fano-factor (variance/mean spike count in a given sample window). While some studies report fano-factors less than 1 (Gur et al., 1997; Bair, 1999; Kara et al., 2000; Gur and Snodderly, 2006; Amarasingham et al., 2006) or larger than 2 (Vogels and Orban, 1991), fano-factors are typically greater than 1 and below 1.5 in LGN (Levine and Troy, 1986; Oram et al., 1999; Reich et al., 1997), primary visual cortex, V1 (Dean, 1981; Tolhurst et al., 1981; Tolhurst et al., 1983; Bradley et al.,...
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1987; Victor and Purpura, 1996; Geisler and Albrecht, 1997; Carandini, 2004), V4 (McAdams and Maunsell, 1999), infero-temporal cortex and anterior superior temporal sulcus, IT/STSa (Vogels et al., 1989; Baddeley et al., 1997; Wiener et al., 2001; Oram et al., 2002; Amarasingham et al., 2006), middle temporal area MT, (Snowden et al., 1992; Britten et al., 1993; Buracas et al., 1998; Uka and DeAngelis, 2003), parietal and motor cortices (Lee et al., 1998; Maynard et al., 1999; Oram et al., 2001) and supplementary motor cortex (Averbeck and Lee, 2003). While the variability of neuronal responses has typically been examined using a “fixed” analysis window, the variability of the initial response is less than in the sustained or latter part of stimulus or motor induced activity in pre-motor cortex, V1, IT and STSa (Vogels and Orban, 1991; Oram and Perrett, 1992; Muller et al., 2001; Churchland et al., 2006; Amarasingham et al., 2006) suggesting that the initial transient response will be more informative than later parts of the response (Gershon et al., 1998).

If the responses of a neurone to repeated presentations of a stimulus are not Possion distributed, as indicated when the fano-factor differs from unity, then the responses will have correlation over time (Oram et al., 1999; Oram et al., 2001; Kass and Ventura, 2006). The correlation between successive time windows within the responses of a single neurone (sequential auto-correlation) has been found in parietal, MT, IT and motor cortices (Lee et al., 1998; Bair et al., 2001; Uka and DeAngelis, 2003; Oram et al., 2007; Lee et al., 1998; Osborne et al., 2004). Knowledge of both the time course of and how sequential auto-correlation varies with temporal window size provides insight into the temporal resolution of integration of signals available within the brain.

Evaluation of stimulus related information coded by a population of neurones requires quantification of the correlations between the responses of different neurones when a stimulus is presented. Responses from simultaneously recorded neurones allow evaluation of both the correlation of the signal between neurones (estimated from the mean responses to
different stimuli) and the between neurone correlation of the trial-by-trial variability to different stimuli (the so called “noise” correlation). The noise correlation between neurones may be small, particularly when receptive field similarity or overlap is small (DeAngelis et al., 1999; Maynard et al., 1999; Lee et al., 1998; Bair et al., 2001; Averbeck and Lee, 2006; Smith and Kohn, 2008). The influence of the between neurone signal and “noise” correlation on encoded information is complex. While the presence of correlation will reduce the entropy of the population response, the levels of correlation observed between cortical neuronal responses, $r=0.1–0.3$ (Gawne et al., 1996; Reich et al., 2001b; Kohn and Smith, 2005; Zohary et al., 1994; Bair et al., 2001; Gawne and Richmond, 1993; Lee et al., 1998; Maynard et al., 1999) can increase or decrease the mutual information between population responses and stimuli compared to that expected when the neurones are independent (Oram et al., 1998; Abbott and Dayan, 1999; Panzeri et al., 1999; Wilke and Eurich, 2001; Wilke and Eurich, 2002; Schneidman et al., 2006; Nirenberg and Latham, 2003; Averbeck and Lee, 2004; Michel and Jacobs, 2006; Franco et al., 2004; Anderson et al., 2007).

In this article, I describe the dynamics of the statistics of neuronal responses induced by visual stimuli in the anterior superior temporal sulcus (STSa) of the macaque monkey and quantify their impact on the information carried by the responses. Stimulus elicited activity starts with a brief period in which the trial-by-trial variability of neuronal activity is reduced and independent of previous activity. The between cell correlation of the trial-by-trial variability also shows a decrease at response onset, coincident with the rise in the signal correlation. Extension of previous modelling work (Oram et al., 1999; Oram et al., 2001) suggests that these phenomena are related and can be explained by a dual-process model in which the activity generating process undergoes a “reset” at response onset. The results are discussed in the context of neuronal output activity being governed by recurrent activity in the “pre-stimulus” period, then changing at the onset of the stimulus elicited response to
being largely dominated by feed-forward connections. Thus, the study of the dynamics of
response statistics informs us about the neural code, helps discriminate models of response
statistics and helps elucidate the underlying circuits (Oram et al., 2007; Smith and Kohn,
2008).
Methods

Responses elicited by a wide range of visual stimuli, including faces, bodies, hands, animals and abstract images from single and pairs of single neurones in the superior temporal sulcus of 2 macaque monkeys were collected. The experimental protocols have been described before (Foldiak et al., 2003; Oram et al., 2002; van Rossum et al., 2008). Extracellular single-unit recordings were made using standard techniques for single electrode recording from the upper and lower banks of the anterior part of the superior temporal sulcus (STSa) of two monkeys (Macaca mulatta) performing a visual fixation task. Briefly, recording chambers were implanted under pentobarbitol (Sagatal) anaesthesia with full sterile precautions over both hemispheres to enable electrode penetrations to reach the STSa. Cells were recorded using a single tungsten-in-glass microelectrode inserted through the dura mater. The electrical signal was amplified (Neurolog NL104) and then filtered using a 50 Hz notch filter together with low- (20KHz) and high- (300Hz) pass filters (Neurolog NL125). The subject’s eye position (±1°) was monitored (IView, SMI, Germany). A Pentium IV PC with a Cambridge electronics CED 1401 interface running Spike 2 recorded eye position, spike arrival and stimulus on/offset times. The animal procedures were conducted under UK Home Office project licence (PPL 60/3186) and approved by the Animal Welfare and Ethical Committee of the University of St Andrews.

The subject received a drop of fruit juice reward every 500ms of fixation (±3°) while static stimuli (19° by 19°) were displayed. During initial screening, 24 bit colour images of different perspective views of monkey and human head, animals, fractal patterns, natural scenes, and everyday objects were presented (22-41 stimuli, Edwards et al., 2003). Visual stimuli were presented centrally on a black monitor screen (Sony GDM-20D11, resolution
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25.7 pixels/degree, refresh rate 72Hz, 57cm from the subject in a random sequence for 333ms with a 333ms inter-stimulus interval.

Spikes were recorded during the period of fixation. If the subject looked away for longer than 100ms, both spike recording and presentation of stimuli stopped until the subject resumed fixation for ≥500msecs. The results from initial screening were used to select stimuli that elicited large responses from the neurone (effective stimuli) and to select stimuli that elicited small or no response (ineffective stimuli). For different neurones effective and ineffective stimuli included different views of the head (Perrett et al., 1991), abstract patterns and familiar objects (Foldiak et al., 2003). Details of the stimulus selectivity of these neurones has been reported elsewhere (Oram et al., 2002; Foldiak et al., 2003; Edwards et al., 2003; Barraclough et al., 2005; Barraclough et al., 2006; van Rossum et al., 2008; Barraclough et al., 2009). The anterior-posterior extent of the recorded cells was from 7mm to 10mm anterior of the interaural plane, consistent with previous studies showing visual responses to static images in the anterior superior temporal sulcus, STSa (Bruce et al., 1981; Perrett et al., 1982; Baylis and Rolls, 1987; Perrett et al., 1991).

Data analysis

Offline isolation of single neurones was performed using a template matching procedure and principal components analysis on the continuous recording (Spike2, Cambridge Electronic Design, Cambridge, UK). Each neurone’s response to a stimulus was calculated by aligning spike times on each occurrence of that particular stimulus (trials). For each neurone a PSTH was generated and a spike density function (SDF) calculated for each stimulus by summing across trials (bin size = 1ms) and smoothing (Gaussian kernel, = 10ms). Background or spontaneous activity was measured in the 200ms period prior to stimulus onset. Data from a neurone was accepted if at least 5 trials were obtained per stimulus (median=19, inter-quartile range = 11..29) and the peak response (measured in the
100ms after response latency) to at least one stimulus was greater than 5 spikes/sec above
background activity.

**Single cell analysis**

The analysis of the data from a single cell is shown schematically in Figure 1. The variance of the responses was related to the mean (e.g. in the period 70-270ms post-stimulus onset, \( \ln(\sigma^2) = 0.17 + 1.05*\ln(\mu), r^2=0.79, p <0.00001 \)). The variability of the responses was therefore assessed using the fano-factor (variance/mean of spike count), \( FF_{stim} \):

\[
FF_{stim} = \frac{\sigma_{stim}^2}{\mu_{stim}}
\]

where \( \mu_{stim} \) and \( \sigma_{stim} \) are the mean and standard deviation of the spike counts to stimulus \( stim \).

The \( FF_{stim} \) were aligned to the centre point of each bin (Figure 1a, dashed lines). The fano-factor averaged across stimuli gives the neurone fano-factor, \( FF_{neurone} \), the average of the \( FF_{neurone} \) giving the population estimate, \( FF \).

The sequential or auto-correlation was assessed for each cell/stimulus combination using Pearson’s correlation coefficient between two successive, non-overlapping sample windows. The sequential correlation is broken down into the correlation of the signal (\( r_{auto\_signal} \)) and correlation of the noise (\( r_{auto\_noise} \), Figure 1b), corresponding to the correlation between successive sample windows of the mean response to each stimulus and the correlation between the successive sample windows of the trial-by-trial variability about those means.

The signal correlation of each neuron (\( r_{neuron\_auto\_signal} \)) is the correlation between the mean spike counts of successive sample windows across stimuli:

\[
r_{neuron\_auto\_signal} = \frac{\sum_{stim}(\mu_{w1} - \overline{\mu_{w1}})(\mu_{w2} - \overline{\mu_{w2}})}{\sqrt{\sum_{stim}(\mu_{w1} - \overline{\mu_{w1}})^2}\sum_{stim}(\mu_{w2} - \overline{\mu_{w2}})^2}
\]
where $\mu_{w1}$ and $\mu_{w2}$ are the mean spike counts in window 1 and window 2 to each stimulus stim, $\bar{\mu}_{w1}$ and $\bar{\mu}_{w2}$ are the mean spike counts across stimuli of window 1 and window 2 respectively.

The simple average of $r_{\text{neuron\_auto\_signal}}$ will result in an underestimate of the true value of the population estimate $r_{\text{auto\_signal}}$ because of the skewed distribution of correlation coefficients (Snedecor and Cochran, 1980). Instead, the average of Fisher’s transformation of $r_{\text{neuron\_auto\_signal}}$

$$z_{\text{neuron\_auto\_signal}} = \frac{1}{2} \ln \left( \frac{1 + r_{\text{neuron\_auto\_signal}}}{1 - r_{\text{neuron\_auto\_signal}}} \right)$$

gives a population estimate $z_{\text{auto\_signal}}$, which can be inverse transformed to obtain a better estimate of the population sequential correlation of the signal, $r_{\text{auto\_signal}}$. The Fisher transformation can, however, result in a small over-estimate of the true value of correlation (Snedecor and Cochran, 1980): the results reported here are also seen using the (under) estimate from the average of $r_{\text{neuron\_auto\_signal}}$.

The correlation of the trial-by-trial variability between two non-overlapping windows can be calculated to each stimulus (Figure 1b). These values can then be averaged (using Fisher’s transform) to obtain an estimate of the sequential correlation of the noise for the neurone. However, such a procedure does not work well with small windows if either window has no measurable variability (i.e. no spikes) in the limited number of trials. To allow calculation of the sequential correlation of the trial-by-trial variability of each neuron ($r_{\text{neuron\_auto\_noise}}$) for small windows, the spike counts were collapsed across all stimuli by transforming each response to each stimulus $s$ ($r_s$) into its z-score, $r_z = \frac{r_s - \bar{r}}{\sigma_s}$, where $\bar{r}$ is the mean and $\sigma_s$ is the standard deviation of the responses to stimulus $s$, setting $r_z=0$ when $\sigma_s = 0$. Without this normalisation, the measure of noise correlation assessed over all trials
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will be artifactually high because of the inclusion of the impact of the stimulus (signal). The correlation of the \( r_z \) between windows w1 and w2 for each neuron

\[
\begin{align*}
    r_{\text{neuron sequ noise}} &= \frac{\sum_{\text{trials}}(r_{zw1} - \bar{r}_{zw1})(r_{zw2} - \bar{r}_{zw2})}{\sqrt{\sum_{\text{trials}}(r_{zw1} - \bar{r}_{zw1})^2 \sum_{\text{trials}}(r_{zw2} - \bar{r}_{zw2})^2}}
\end{align*}
\]

is therefore the sequential correlation of the normalised trial-by-trial response variability about the mean and does not reflect correlation of the mean response (signal), nor is it biased towards those stimuli with greater absolute response variability (Lee et al., 1998; Oram et al., 2001). Averaging \( r_{\text{neuron auto noise}} \) across neurones, using Fisher’s transformation,

\[
    z_{\text{neuron sequ noise}} = \frac{1}{2} \ln \left( \frac{1 + r_{\text{neuron sequ noise}}}{1 - r_{\text{neuron sequ noise}}} \right)
\]

results in mean, \( z_{\text{auto noise}} \), which can be inverse transformed to obtain an estimate of the population sequential correlation, \( r_{\text{auto noise}} \).

**Between cell analysis**

The recordings allowed separation of pairs, three and in two cases 4 isolated neurones from the same electrode (Spike 2, Cambridge Electronic Design, PCA based algorithm; minimum Mahalanobis distance = 1.5). Recordings in which the waveforms drifted over time were rejected. All separated spike clusters were then examined visually, and only if the separation was clear (i.e. no “unclassified” spike waveforms lay between the clusters) were the isolated neurones accepted.

The correlation between the responses of pairs of neurones can also be described using the signal correlation, \( r_{\text{cross signal}} \), and the “noise” correlation, \( r_{\text{cross noise}} \) (Gawne and Richmond, 1993). The signal correlation for each neurone (\( r_{\text{neuron cross signal}} \)) is the correlation between the mean spike counts to each stimulus:
where $\mu_1$ and $\mu_2$ are the mean spike counts of neurone 1 and neurone 2 to each stimulus, $\overline{\mu_1}$ and $\overline{\mu_2}$ are the mean across stimuli of neurone 1 and neurone 2 respectively. The Fisher’s transformed $r_{\text{neuron\_cross\_signal}}$ were averaged and inverse transformed to obtain a population based estimate, $r_{\text{cross\_signal}}$. The trial-by-trial or noise correlation between neurones was calculated in an analogous manner to the calculation of the $r_{\text{auto\_noise}}$. The cross-correlation, $r_{\text{cross\_noise}}$, used the z-scored firing rates and calculating the correlation of the trial z-scores from all stimuli. Combining of data across stimuli is supported by the observation that the trial-by-trial or noise cross-correlation does not vary with stimulus (Kohn and Smith, 2005).

The values of $\text{FF}$, $r_{\text{auto\_signal}}$, $r_{\text{auto\_noise}}$, $r_{\text{cross\_signal}}$ and $r_{\text{cross\_noise}}$ were calculated every 5ms using sample windows of 5, 10, 25, 50, 100 and 200ms duration. Estimates obtained of $\text{FF}$, $r_{\text{auto\_signal}}$, $r_{\text{auto\_noise}}$, $r_{\text{cross\_signal}}$ and $r_{\text{cross\_noise}}$ every 5ms are not independent when the window size is greater than 5ms. Statistical analyses used non-overlapping, independent samples from each neurone, but data is displayed every 5ms to aid visual interpretation of these summary statistics.

**Modelling**

The three models used are shown schematically in Figure 2. All three models use the spike density function (SDF, Figure 2 top) as the starting point. The non-homogenous Poisson process (NHPP) model used independent binomial processes with one millisecond time bins, the value of the spike density function (SDF) giving the probability, $p$, of a spike occurring in that time bin (Figure 2, middle row, left). The effects of non-Poissonian spike count distribution, particularly with respect to correlations, are critically dependent on the precise spike counts (Oram et al., 1999). The spike count matched (SCM) model (Oram et al.,
Stimulus induced decorrelation (Oram, 1999; Oram et al., 2001; Oram, 2005; Oram et al., 2002) was used to match the observed coarse temporal statistics as closely as possible. Briefly, for each cell, the spike density function (SDF) is transformed into a cumulative spike density function (CSDF, Figure 2, middle) for each stimulus at each time point $t$ ($\sum_{i=1}^{t} SDF(i)$). Normalization by the value at the end of the sample period ($t=T$) gives the cumulative spike probability function $CSPF(t) = \frac{CSDF(t)}{CSDF(T)}$. The spike times of each artificial trial are found by applying the inverse of the cumulative probability distribution to random numbers in the interval $(0,1)$, $R[0..1]$, black arrows Figure 2, middle. The time bin (width $\delta t$, here 1ms) in which a spike occurs, $t_{\text{spike}}$, is such that $t_{\text{spike}}$ satisfies $CSPF(k) \leq R[0..1] < CSPF(k+1)$, the time of the $k$th bin being $k\delta t - (k+1)\delta t$.

When generating surrogate data for pairs of neurones using the SCM model (Oram et al., 2001), each pair of spike count distributions and their correlation are preserved by stepping through the experimental data trial by trial and forcing each simulated trial of each cell to have the same number of spikes as the corresponding experimental trial. The SCM model therefore incorporates the slow variation in firing rate and the distribution of spike counts generated by individual neurons as well as the coarse temporal correlation of the spike counts and the correlation between the spike density functions (i.e. correlation between the slow variation in firing rate of the individual neurons over time, see Oram et al., 2001 for details).

The SCM model simulates spike trains assuming a single process (here running from 200ms before to 600ms post-stimulus onset). A two process extension, SCM2, is modelled by applying a “reset”. This is achieved by running the SCM, including the normalizations to generate a CSDF for the period -200 to 69ms. The SCM model is then re-run, with re-calculation of the CSDF, using the data from 70ms to 600ms post-stimulus onset (Figure 2,
Therefore, in the SCM2 model the spike counts are matched on a trial-by-trial basis separately for the two different intervals (Figure 2, middle row, right: solid arrows for period -200..69ms, open arrows for period 70..600ms). The reset or change of spike generation process at 70ms was chosen to approximate the response latency of STS/IT neural populations (Oram and Perrett, 1992; Oram and Perrett, 1996; Eifuku et al., 2004; Kiani et al., 2005; Sary et al., 2006). Thus, the SCM2 model can be interpreted as assuming a “non-stimulus driven” and a separate (independent) “stimulus driven” spike generation process. An absolute refractory period of 1ms was applied to the SCM and SCM2 models. To control for potential biases due to limited sampling, the number of trials from each neurone was also matched. The spike trains from the NHPP, SCM and SCM2 models were analysed using the same methods as used for the experimental data.
Results

Visually elicited responses from 74 neurones from two Rhesus macaque monkeys were analysed following presentation of stimuli for 333ms. 51 neurones were tested with effective (>70% of maximum) and ineffective (<30% of maximum response) stimuli. The 74 neurones allowed for 67 pairs of simultaneously recorded neurones to be examined.

Response statistics of single neurones

An example of the calculation of the fano factor for a single neurone/stimulus combination (\(FF_{\text{stim}}\)) using 100ms windows is shown in Figure 1a. Across the population, the average fano-factor (see methods) was significantly greater than 1 at all time points for sample windows of 10ms or longer (Figure 3, upper row). As expected, the fano-factor increases with window size (de Ruyter van Steveninck R et al., 1997; Baddeley et al., 1997; Teich et al., 1996; Carandini, 2004; Buracas et al., 1998; Osborne et al., 2004).

Figure 3b allows comparison of the time course of the fano-factor averaged across the 74 neurones using 100ms windows with the time course of the neuronal activity (average spike density function, grey). There is a transient dip in fano-factor at the onset of neuronal responses (Uka and DeAngelis, 2003; Osborne et al., 2004; Churchland et al., 2006; Amarasingham et al., 2006). The time of the minima estimated from different window sizes ranged from 80 to 140 ms post-stimulus onset, approximately the time of peak response. The dip in the fano-factor at ~70ms is significant (ANOVA, \(p<0.005\)) for window sizes down to 10ms and a trend (\(p=0.08\)) is present for 5 ms windows. Detection of the dip in normalised variability down to such short sample windows suggests a rapid underlying mechanism.

An example of the sequential auto-correlation of the trial-by-trial variability assessed between adjacent bins, \(r_{\text{auto_noise_stim}}\), is shown in Figure 1b for the same cell/stimulus as in Figure 1a. Note the marked decrease (dip) in the correlation around response onset, in this
case showing negative correlation. The average sequential correlation of the trial-by-trial variability ($r_{\text{auto\_noise}}$) is shown in Figure 3d-f. Like the fano-factor, $r_{\text{auto\_noise}}$ shows a dip at ~70ms and increases systematically with window size, reaching a stable value at window sizes of ~100ms (Figure 3d,f). The average sequential correlation for windows ≥100ms in both the pre-stimulus period and sustained period (0.13±0.02) are similar to values reported previously (0.14-0.16) in parietal cortex, MT, IT and motor cortices (Lee et al., 1998; Bair et al., 2001; Uka and DeAngelis, 2003; Oram et al., 2007) although not as high as found in some studies (e.g. 0.4 in MT Osborne et al., 2004). These previous findings are extended here by showing that the dip in $r_{\text{auto\_noise}}$ around response onset (Figure 3f) is significant for window lengths of 25ms or more (p<0.005 each comparison), with a trend (p=0.09) for windows of length 10ms. As with the dip in the Fano factor, detection of the dip in $r_{\text{auto\_noise}}$ in short sample windows suggests a rapid underlying mechanism.

The sequential (auto-) correlation of the “signal”, assessed using the mean response to each stimulus in two adjacent bins, $r_{\text{auto\_signal}}$ is shown in Figure 3g-i). Combined with the presence of sequential correlation of the noise ($r_{\text{auto\_noise}} > 0$), the small numerical differences the mean pre-stimulus response estimates due to limited sampling are maintained in time, giving rise to non-zero estimates of $r_{\text{auto\_signal}}$. While $r_{\text{auto\_signal}}$ does not vary with the size of the sample window in the pre-stimulus (p=0.22) and onset periods (p=0.54, see Figure 3i), $r_{\text{auto\_signal}}$ does vary with sample window during the sustained period (ANOVA: p<0.001).

The reduction in the average sequential correlation $r_{\text{auto\_noise}}$ and fano-factor around response onset is evident cell by cell. Figure 4 plots the frequency distribution of the time for the minimum (solid line) and maximum (dashed line) of the $r_{\text{auto\_noise}}$ (Figure 4, upper) and fano-factor (Figure 4, lower) of each neurone assessed using 100ms windows. The clustering of the minima of both $r_{\text{auto\_noise}}$ and the fano-factor in the period 75-175 was significant for all window sizes ($\chi^2$, p<0.05 each comparison). If the clustering were due to increased variability
of the estimates of $r_{auto\_noise}$ and the fano-factor, it would be expected that the maxima would also cluster in this period. However, the maxima tend to occur outside this period ($r_{auto\_noise}$: window sizes 50, 100ms $p<0.01$; windows 10, 25ms $p<0.1$; fano-factor: window sizes 5,25-200ms $p<0.05$).

It is also of interest to know whether the response statistics vary with response amplitude or stimulus effectiveness. The $r_{auto\_noise}$ and fano-factor observed in responses to stimuli that elicited large (>70% of maximum) and small (<30% of maximum) responses were examined separately (Figure 5). [Splitting the data into groups by stimuli that elicit either large or small mean responses removes any meaningful assessment of $r_{auto\_signal}$]. The average spike density function (Figure 5, inset) shows that there are negligible responses to ineffective stimuli and strong responses to effective stimuli. The consistency of $r_{auto\_noise}$ in the pre-stimulus period (effective 0.15±0.03; ineffective 0.13±0.02), during onset (effective 0.02±0.04; ineffective 0.05±0.03) and sustained period (effective 0.18±0.04; ineffective 0.12±0.03) indicates that the dip in sequential correlation is not attributable to changes in response amplitude. The dip in mean $r_{auto\_noise}$ was present in the responses elicited by ineffective (and effective) stimuli for windows as small as 10ms, although statistically significant ($p<0.05$) only for windows of 25ms or more. The fano-factor in the pre-stimulus period (effective 1.5±0.1; ineffective 1.5±0.1), during onset (effective 1.3±0.2; ineffective 1.3±0.1) and sustained period (effective 1.5±0.1; ineffective 1.4±0.1) indicates that the dip in fano-factor at response onset (Figure 5, upper) is not attributable to changes response amplitude. The transient “dip” in fano factor was statistically significant ($p<0.05$) for window sizes down to 10ms in the responses elicited by both effective and ineffective stimuli.

While the presence of a dip in sequential correlation and fano-factor around response onset cannot be attributable to differences in response magnitude, the time course of the response statistics varies with response magnitude, being somewhat faster to “recover” to
pre-stimulus levels for effective than ineffective stimuli (Figure 5). For example, although the
time of the minima of the sequential correlation are almost identical (90ms for ineffective
stimuli, 95ms for effective stimuli), the recovery to pre-stimulus levels takes only 60ms with
effective stimuli compared to 185ms with ineffective stimuli. The slower recovery to pre-
stimulus levels in the responses to ineffective stimuli is also seen with the fano-factor. Thus,
both response variability and sequential correlation of neuronal activity are reduced at around
the time when neurones in STSa become active, even in neurones which are not strongly
activated by the input stimulus. Although the magnitude and time of the “dip” is similar to
both effective and ineffective stimuli, the effect is shorter lasting in responses to effective
than in response to ineffective stimuli.

Next, I extend previous studies by examining the impact of lag between sample
windows. With a constant spike generating process the correlation between successive short
sample windows will be independent of the lag between two sample windows. Now consider
a spike generating process that operates at short time scales. Between time 1 and 2, we can
explain $R^2$ of the variance at time 2 based on the data at time 1. Similarly, between time 2 and
3 we can explain $R^2$. Assuming no higher order correlation, we can therefore explain $R^2 \times R^2$
of the variance of the response at time point 3 based on the values at time point 1. Given the
$R^2$ is necessarily $\leq 1$, this gives an exponential decay in the $R^2$ (and the correlation
coefficient, $r$) as the lag is increased. Thus, if the spike generating process operates at
(multiple) short time scales, the correlation between successive sample windows will
decrease exponentially as the lag between the two sample windows is increased.

With 100ms sample window (Figure 6), the magnitude of the sequential correlation of
the trial-by-trial variability ($r_{auto\_noise}$) decreases with increasing lag (Figure 6a-b). The “dip”
in $r_{auto\_noise}$ is numerically present for lags up to 225ms (Figure 5a), being statistically
significant ($p<0.05$) for lags up to 75ms and showing a trend ($p<0.1$) for lags up to 150ms.
The exponential decay of $r_{\text{auto\_noise}}$ with lag is evident for all sample window sizes (see Table 1).

The time constant for the decay of $r_{\text{auto\_noise}}$ with increasing lag between the sample windows obtained from the pre-stimulus data ($107\pm6\text{ms}$) was less than in both the onset ($188\pm50\text{ms}$) and the sustained periods of the response ($143\pm17\text{ms}$). Including a constant offset in the regression (i.e. fitting $r_{\text{seq}} = \text{Offset} + \beta_1 e^{\log(\tau)/\text{Lag}}$) showed significant ($p<0.05$) offset for the pre-stimulus data ($0.015\pm0.002$ using $100\text{ms}$ windows, Figure 6b), significant offsets being observed in the pre-stimulus period for window sizes down to $5\text{ms}$ (Table 1). The absence of evidence for a long-term offset of $r_{\text{auto\_noise}}$ in the sustained period is attributable to the de-correlation (the dip in $r_{\text{auto\_noise}}$) at onset. Specifically, the sustained period of the responses are taken from $300-400$ post-stimulus onset. At a lag of $200\text{ms}$, the two $100\text{ms}$ windows are ($0..100$) and ($300..400$). The first of these windows falls before the typical response latency ($70-100\text{ms}$) of STSa neurones (Oram and Perrett, 1992; Oram and Perrett, 1996; Kiani et al., 2005), precisely the period where decorrelation occurs (see Figures 1,3-4). The exponential of the decay in $r_{\text{auto\_noise}}$ with lag between the samples across multiple window sizes indicates the presence of multiple short time scale correlations present in the spike generating process. As with MT (Bair et al., 2001) and V1 (Kohn and Smith, 2005), the correlation of the trial-by-trial variability within the responses of single STSa neurones can last over prolonged periods (indicated by significant offset values). However, the present data suggest the prolonged correlation is disrupted by the presentation of a stimulus even though such correlation can last across many stimulus presentations (Bair et al., 2001).

The sequential correlation of the signal ($r_{\text{auto\_signal}}$) is also sensitive to the delay between the samples (Figure 6c-b, Table 1). While the “dip” in $r_{\text{auto\_noise}}$ is locked to the onset of the response (Figure 6a), the rise in $r_{\text{auto\_signal}}$ is found when both windows are within the stimulus elicited period of the response (Figure 6c). The requirement for both sample
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windows to be in the stimulus elicited response explains (1) the decreasing time constant of
the decline in $r_{\text{auto signal}}$ with increasing window size (Table 1) and (2) the accelerating slope
of $r_{\text{auto signal}}$ with increasing lag. For example, the sustained period of the responses are taken
from 300-400 post-stimulus onset. At a lag of 150ms, the two 50ms windows (100-150) and
(300-350) both fall within the period of stimulus elicited activity. At a lag of 150ms, the first
of the 100ms windows (50..150) falls partially outside the stimulus elicited response, the
effect being even greater for the 200ms window (-50..150). Longer sample windows at longer
lags therefore include data from periods which are not directly driven by the stimulus, leading
to a decline of $r_{\text{auto signal}}$ with increasing slope.

In summary, the low variability (fano-factor) (Uka and DeAngelis, 2003; Osborne et
al., 2004; Churchland et al., 2006; Amarasingham et al., 2006; Oram et al., 2007) and auto-
correlation of the trial-by-trial variability during the initial response (Oram et al., 2007)
reflects a transient dip from pre-stimulus levels. This dip is seen even when the stimulus
elicits no response, suggesting a network property rather than mechanisms intrinsic to the
neuronal activity per se. The magnitude of the auto-correlation decays exponentially with
delay between sample windows, indicating that the correlation is due to processes operating
at multiple time scales. As with cross-correlation in areas MT and V1 (Bair et al., 2001; Kohn
and Smith, 2005; Smith and Kohn, 2008), the auto-correlation seems to last for extended
periods. However, the present study indicates that the long-lasting sequential correlation in
STSa is transiently disrupted by the presentation of a visual stimulus.

A two process model captures the basic statistics of single neuronal responses

A drop in the fano-factor is expected when activity, assessed using short duration bins
with no more than 1 spike in, increases. Short duration bins can be considered as binomially
distributed with probability $p$ of a spike in a given ms bin. Such bins have mean $p$, variance
$p(1-p)$ and hence a fano-factor of $(1-p)$. It follows that as the firing rate ($p$) increases, the
fano-factor \((1-p)\) decreases. A series of such binomial bins, as can be used to model a spike train, will have mean and variance given by the sum of the mean and variances of the individual bins if and only if the bins are independent. Thus, spike count distributions assessed over an extended time will have a fano-factor of \(~1\) only if the bins are independent. As the fano-factor is greater than 1, the bins (activity) must be correlated in time (Oram et al., 1999; Oram et al., 2001; Kass and Ventura, 2006). Second, spike count distributions are truncated at 0, implying that estimates of correlation between two spike counts will change as a function of the mean (de la Rocha et al., 2007). It is natural to therefore ask (1) are the high levels of \(r_{\text{auto\_noise}}\) in the pre-stimulus and late response periods due to the fano-factor being greater than 1 and (2) is the decrease in \(r_{\text{auto\_noise}}\) due to the rapid change in mean firing rates around response onset?

Artificial spike trains were generated using the binomial approximation of a time varying (non-homogenous) Poisson (NHPP) process. As the time bins in the NHPP model are independent, the sequential correlation should be 0. The spike count matched model forces the fano-factor of extended windows (here -300 to 600 post-stimulus onset) to match that seen in the data (Oram et al., 1999; Oram et al., 2001; Oram et al., 2002; Oram, 2005). Thus, the SCM model was used to investigate the impact of a fano-factor > 1 on the autocorrelation measures \(r_{\text{auto\_noise}}\). A drop in auto-correlation of the trial-by-trial variability is also indicative of a change in the (spike) generating process (Kohn and Smith, 2005). The spike count matched model was therefore run as two independent processes with a change in process at 70 ms (SCM2 model, see methods for details).

The sequential correlation \((r_{\text{auto\_noise}}\) and \(r_{\text{auto\_signal}}\)) and the fano-factor using sample window sizes ranging from 5 to 200ms of the three spike train models are shown in Figure 7. As expected, the binomial approximation of the non-homogenous Poisson process (NHPP, left) gives a fano-factor slightly below one (Figure 7a) and zero sequential correlation
Stimulus induced decorrelation (Figure 7d) irrespective of sample window size. The SCM and SCM2 models (Figure 7, middle and right columns respectively) have a fano-factor greater than 1 (Figure 7b,c) which, as described above, induces non-zero $r_{\text{auto\_noise}}$ (Oram et al., 1999; Oram et al., 2001; Kass and Ventura, 2006 and see Figure 7e,f). Consistent with the neurophysiological data, the SCM and SCM2 models show an increase of both fano-factor and $r_{\text{auto\_noise}}$ with increasing window size.

The dip in fano-factor and $r_{\text{auto\_noise}}$ around response onset is absent in the NHPP and SCM simulated spike trains. The dips seen in the neurophysiological data are, however, captured by the SCM2 model (Figure 7c,f). The transient changes shown by the SCM2 model also occur without change in firing rate (data not shown). Furthermore, the fano-factor and $r_{\text{auto\_noise}}$ of the SCM2 model data are, like the neurophysiological data, comparable between the “pre-stimulus” and “sustained” periods (Figure 7c,f), despite the difference in firing rate. The similarity of $r_{\text{auto\_signal}}$ across the three models (Figure 7g-i) is expected as the same spike density functions (mean activities) are used for all three models.

The variation of the fano-factor and $r_{\text{auto\_noise}}$ with window size and lag in the different spike train models is shown in Figure 8. The ability of the different models to explain the neurophysiological results was assessed using the correlation co-efficient between model and neuronal data (Table 2). The absence of modulation over time in the fano-factor or the $r_{\text{auto\_noise}}$ with either window size or lag of the NHPP model data (Figure 8a,d,g) is reflected in the poor fits to the neuronal data both overall and for the individual window size, with a clear tendency towards negative correlation (Table 2). While the SCM model captures some of the dependency of fano-factor and $r_{\text{auto\_noise}}$ on window size and delay between samples (overall $R^2 = 0.81$ and 0.29 respectively), the values are consistent across pre-stimulus, onset and sustained response periods. The SCM2 model captures the dip in the fano-factor and $r_{\text{auto\_noise}}$ around response onset and therefore provides a better description overall (fano-factor: $R^2 = 0.81$ and 0.29 respectively).
0.96; $r_{\text{auto\_noise}}: R^2 = 0.64$) and for window sizes greater than 10ms (Table 2) than the other models.

In summary, comparison of the results from the NHPP model with the results from the SCM and SCM2 models indicate that observed high levels of $r_{\text{auto\_noise}}$ in the pre-stimulus and late response periods are largely attributable to the fano-factor being greater than 1. The dip around response onset of fano-factor and sequential correlation is not seen in the NHPP and SCM models and hence cannot be attributed to the change in firing rate at response onset nor bias due to limited sample size (see methods). While the SCM2 model captures much of the “shape” of the response statistics, it is not a complete description. The SCM2 models predicts lower than observed values of the sequential correlation found using small windows (e.g. 40% of the observed values using 5ms windows) and overestimates the values for large windows (e.g. 136% of the observed values using 200ms windows). This mismatch suggests the SCM2 model is too simple in that it does not include multiple times scales. However, it is noteworthy that the mismatch in sequential correlation between observed and model values is smaller for the SCM2 model than either of the other models.

**Response statistics between neurones**

The average signal and noise correlation of 67 pairs of neurones simultaneously recorded from STSa are shown for different window sizes as a function of time in Figure 9. As expected (Reich et al., 2001a; Averbeck and Lee, 2003; Kass and Ventura, 2006), the correlation of the trial-by-trial variability (“Noise” correlation) between neurones and the correlation of the mean (signal) between neurones increased with increasing window size (Figure 9b: $r_{\text{cross\_noise}}$; 8d: $r_{\text{cross\_signal}}$). Despite the variation in the magnitude of the noise correlation $r_{\text{cross\_noise}}$, the dip is detectable even with a 5ms sample window. The reduction in the cross-correlation of the noise is transitory, returning to levels comparable to the pre-stimulus period at ~150-200ms post-stimulus onset. The signal correlation shows a slight dip
after response onset but remains high until after the response itself has stopped (~450ms, see
figure 3).

Taking the 100ms sample window as an example, the correlation of the trial-by-trial
variability across neurones (Figure 9, upper) is relatively high in the pre-stimulus period
($r_{cross\_noise}=0.28\pm0.02$) and equivalent ($p=0.1$) during the sustained period of the response
(0.24±0.02). The noise correlation at response onset (0.18±0.02) is significantly less than
either pre-stimulus or sustained periods of the response ($p<0.005$ each comparison). The
values of $r_{cross\_noise}$ in the sustained period using sample windows of 100ms or more are
typical of cortical visual areas V1 (Gawne et al., 1996; Reich et al., 2001b; Kohn and Smith,
2005), MT (Zohary et al., 1994; Bair et al., 2001) and IT (Gawne and Richmond, 1993).
However, the correlation of the trial-by-trial variability between neurones falls significantly
below the pre-stimulus level 40ms after stimulus onset, reaching a minimum of 0.16±0.02
some 75ms post-stimulus onset. The $r_{cross\_noise}$ then rises, being statistically indistinguishable
(p>0.05) from pre-stimulus levels 175ms after stimulus onset.

Trivially, the signal correlation between neurones rises around response onset (Figure
9c). While $r_{cross\_signal}$ increases with increasing window size, it plateaus at window sizes of
50ms or more (Figure 9d). The $r_{cross\_signal}$ at response onset (0.80±0.10 with a 100ms window)
and in the period 300-400 ms (0.81±0.08) is comparable with, albeit slightly less than,
previous studies examining signal correlation of visually responsive neurones recorded from
single electrodes (e.g. 0.86 in MT Bair et al., 2001).

The signal correlation is non-zero in the pre-stimulus period (e.g. with 100ms
window, $r_{cross\_signal} = 0.31\pm0.04$ in the 200ms before stimulus onset), indicating the mean
activity level in the pre-stimulus period varied with the subsequent stimulus. The neuronal
response to one stimulus influences responses to a following stimulus in visual (Macknik and
Livingstone, 1998; Kourtzi and Kanwisher, 2001; Grill-Spector et al., 2006; Sawamura et al.,
Stimulus induced decorrelation (Oram 2006; Felsen et al., 2002; Dragoi et al., 2002), auditory (Wehr and Zador, 2005) and somatosensory systems (Khatri et al., 2004) even with intervening stimuli (Felsen et al., 2002; Perrett et al., 2009). Although stimuli were presented in random order, the presentations were blocked such that each stimulus was presented once before any individual stimulus was repeated. As the stimuli were specifically selected to be either effective or ineffective, the presentation of an effective stimulus is likely to have been preceded by an ineffective stimulus and vice versa. Hence, interaction between stimulus presentations (Perrett et al., 2009) would produce this initially unexpected observation.

The impact of the lag between sample windows on $r_{\text{cross\_noise}}$ and $r_{\text{cross\_signal}}$ is shown in Figure 10. The cross-correlation of the trial-by-trial variability ($r_{\text{cross\_noise}}$, Figure 10a,b) decays smoothly as a function of the lag between the sample windows. The cross-correlation of the signal ($r_{\text{cross\_signal}}$, Figure 10c,d) with increasing lag also decays smoothly in the pre-stimulus and sustained periods of the response (Figure 10, d dotted and solid lines respectively). However, there is a marked “step” in $r_{\text{cross\_signal}}$ with lags around response onset (Figure 10d, dashed line), with a rapid decline from lags of 50 to 75 ms. Indeed, at lags of 25 ms (not shown) and 50 ms (Figure 10c, dashed line) there is a drop in $r_{\text{cross\_signal}}$ around 50-70 ms that is not captured in the 70-120 ms onset period. Thus, the signal at response onset of a neurone is relatively uncorrelated with previous activity of other neurones.

The SCM2 model captures the basic statistics of responses between neuronal pairs

The SCM2 model also captures the transient reduction (dip) in the cross-correlation of the between neuron trial-by-trial variability. A dip in the cross-correlation of the SCM2 modelled data is evident for all window sizes (Figure 11, upper) at the time when a change of process (70 ms) occurs. The dip in $r_{\text{cross\_noise}}$ is not seen using either the NHPP or ~SCM models (not shown). The presence of the dip shows that when sample windows span the time
at which a change in process occurs, a change of process in the individual neurones induces
detection of a drop in the cross-correlation of the trial-by-trial variability between neurones.

As with the responses of single neurones, the SCM2 model does not provide a full
description of the neurophysiological data despite capturing the main features. The absence
of decay with lag in the SCM2 modelled data (Figure 11, lower) is to be expected simply
because a single, constant process is used in the interval [-300..70ms] and a second process in
the interval [71..600ms]. The SCM2 models predicts substantially lower than observed values
of $r_{\text{cross}_\text{noise}}$ for all windows sizes examined, the mismatch being proportionally smaller for
large windows ($r_{\text{cross}_\text{noise}}$ : 200ms sample window, SCM2≈0.22, STSa≈0.30; 10ms sample
window SCM2≈0.02, STSa≈0.15).

**Implications for neuronal coding**

The drop in response variability and the drop in cross-correlation of the trial-by-trial
variability associated with response onset have implications for the information that the
neuronal responses carry. Specifically, I ask what is the impact of the transient dip in the
fano-factor and $r_{\text{cross}_\text{noise}}$ on the mutual information between the responses and the input
stimuli? To calculate the mutual information between a stimulus set and the responses of a
pair of model neurones, the mean response of each neuron to each stimulus needs to be
known, as does the trial-by-trial variability of the responses about that mean (fano-factor). It
is also necessary to know the co-variance structures between the neurons, both of the signal
($r_{\text{cross}_\text{signal}}$) and the trial-by-trial variability ($r_{\text{cross}_\text{noise}}$). Thus, the information needs to be
calculated twice: once with the low fano-factor and $r_{\text{cross}_\text{noise}}$ observed at response onset and
once with the higher values observed in both the pre-stimulus and sustained sections of the
activity. As the dip in fano-factor and $r_{\text{cross}_\text{noise}}$ occurs at the time of response onset when
there is a large transient peak in firing rate, comparison of the mutual information during the
onset and sustained periods would confound changes in mean response with changes in the
variability and correlation structure. Therefore, it is necessary to model the impact of the transient dip in the fano-factor and $r_{\text{cross,noise}}$ on the mutual information.

The distribution of the signal (mean response to different stimuli) was modelled by setting each neuron to have the average tuning curve found in area STS to changes in perspective view of the head (Perrett et al., 1991; Oram, 2005). The relative “position” of the tuning curves between the pair of model neurons was adjusted to give the observed signal correlation ($r_{\text{cross,signal}}$). The mutual information was calculated using 128 stimuli, the mean firing rate (signal) to each stimulus given by the tuning curve. The stimuli were uniformly distributed across the modelled stimulus space (perspective view), the tuning curve ensuring the distribution within “response space” is typical of STSa neurons. The peak response of the tuning curve was taken as the response to the effective stimuli at 100 ms post-stimulus onset (Figure 5, inset), scaled by the sample window (10, 25, 50, 100 or 200ms). The mutual information between the pair of modelled responses and 128 stimuli was assessed using the average fano-factor, signal correlation ($r_{\text{cross,signal}}$) and cross-correlation of the noise ($r_{\text{cross,noise}}$) observed at 100ms post-stimulus onset (Figure 12, solid line). As can be seen, the information increases with the integration time (window size), reaching 95% of the maximum with a window size of 50ms (see also Gershon et al., 1998). The mutual information in the absence of the transient dip of the fano-factor and cross-correlation of the noise was assessed using the average fano-factor, and cross correlation of the noise from the pre-stimulus period whilst maintaining the same distribution of mean responses (signal) and signal correlation (Figure 12, dashed line).

The increase in the mutual information between the stimuli and pairs of neuronal responses due to the transient dips in the fano-factor and $r_{\text{cross,noise}}$ is evidenced as the difference between the solid and dashed lines of Figure 12. The increase was minimal for small windows (10ms sample window the increase in the mutual information was only 1%,
25ms increase 7%). The dips in fano-factor and $r_{\text{cross\_noise}}$ around response onset found with windows of 50, 100 and 200ms resulted in 13, 19 and 24% increases in mutual information respectively compared to the mutual information when the fano-factor and $r_{\text{cross\_noise}}$ were set to the values observed in the pre-stimulus periods. Given the similarity between the pre-stimulus and sustained periods of the response with respect to the fano-factor (Figure 3), $r_{\text{cross\_noise}}$ and $r_{\text{cross\_signal}}$ (Figure 9) it is not surprising than almost identical results were obtained using the values from the sustained period of the response (300-400ms). Thus, the transient dip in fano-factor and cross-correlation of the noise described here can result in appreciable changes in the efficiency of the neural coding, particularly when the integration time is 100ms or more.
Discussion

The response statistics of single and pairs of visually responsive neurones in the anterior sections of the superior temporal cortex were examined. As the trial-by-trial variability of neuronal responses is proportional to the mean firing-rate, the normalised variability (variance/mean) was used to examine changes in response variability. The data indicate that the normalised variability is high in the pre-stimulus period (i.e. in the absence of a stimulus) and roughly equivalent to that in the sustained period of the response. There is a dip, however, in normalised variability of the initial visually evoked response in STSa, as seen in V1 (Muller et al., 2001) and in previous studies of inferior temporal cortex and STSa (Vogels and Orban, 1991; Oram and Perrett, 1992; Amarasingham et al., 2006). Variability is reduced for high response magnitudes (Hartveit and Heggelund, 1994). However, the drop in fano-factor is seen with stimuli that elicit negligible responses (Figure 5) and is absent in both the NHPP and SCM models (Figure 7) which include the relevant changes in activity over time. Thus, changes in activity at response onset cannot explain the observed changes in response variability. The fixation window used here was quite large (6 degrees across). Previous studies have noted that a considerable proportion of trial-by-trial response variability in V1 and LGN can be attributed to small variations in eye position (Gur et al., 1997). As already noted, the values for the normalised variability (fano factor) and auto-correlation ($r_{auto\_noise}$) are almost identical to those obtained in previous studies with much tighter eye movement control (e.g. Bair, 1999), as is the presence of the “dip” around response onset (e.g. Amarasingham et al., 2006; Churchland et al., 2006). Furthermore, the response variability of IT neurones is relatively independent of the amount of eye movement so long as the stimulus remains well within the receptive field (Wiener et al., 2001). Thus, while the lack of precise eye-movement control may have introduced small errors in the
absolute values of response variability, the impact of this on the normalised response
statistics used here is likely to be negligible.

As with response variability, the response auto-correlation has a marked “dip” in the
first 100ms of the stimulus elicited response. The transient reduction in response variability
and auto-correlation are also seen in the responses to ineffective stimuli (Figure 5), indicative
that the effects are the results of a network property rather than intracellular activity
dependent mechanisms. The auto-correlation decayed in an exponential fashion with
increasing lag between the two sample windows (Figure 6), the effect being seen at multiple
time scales (sample windows of 10 to 200ms, Table 1). It is also noteworthy that the
sequential correlation does not decay to 0, instead falling to some low value (~0.02). Thus,
like others (Bair et al., 2001), we find evidence for multiple time scales, including long term
correlation of neuronal activity.

Comparison of the NHPP and SCM models (Figure 7) with the neuronal data shows
that the correlation induced by the non-Poisson distribution of spike count (Oram et al., 1999;
Kass and Ventura, 2006) is insufficient to explain the levels of correlation in the data.
Previous studies have shown that a dip in cross-correlation between neurones can result from
a change in response statistics (Poisson to gamma processes) without a change in firing rate
(Tetzlaff et al., 2008). The present simulations suggest that the “dip” in response variability
and auto-correlation of single neurones around response onset can be induced simply by a
change of process without any change in the underlying output statistics and that such
changes are reflected in the between neurone correlation.

Perhaps the simplest mechanism that could underlie a change in spike generation
process at response onset involves a change in the relative importance of feed-forward inputs.
The number of feed-forward synapses to layer 4 V1 neurones of the cat is estimated to be
only ~2% of the total (Binzegger et al., 2004; Douglas and Martin, 2007), while ~12% of
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synapses to LGN come from retinal inputs (Van Horn et al., 2000). While small in number, these feed-forward connections can clearly drive the target neurones. However, the relative contribution of feed-forward connections during un-driven (pre-stimulus) activity is likely to be substantially less than in the driven state. In the absence of driving input, the neuronal activity will be determined largely by feedback and lateral connections. When receiving sensory inputs the balance might change, such that the activity driving the spike generation process is now governed by the small proportion of active feed-forward inputs. Over time the relative importance of recurrent activity will rise as the stimulus elicited activity spreads through the network and activates the recurrent connections. The transient change in setting the neurone’s activity from recurrent to feed-forward inputs and then back to recurrent inputs would provide the change of generating process suggested by the SCM2 model.

Recent studies indicate that mechanisms other than a change in process may also play a role in the dip of response variability and correlation at response onset. Variability of responses in LGN, as measured by the fano-factor, is reduced by cortical feedback from V1 (Andolina et al., 2007). The transient dip in fano-factor and auto-correlation reported here and elsewhere could therefore be due to increased feedback. However, feedback to LGN is dominated from extra-area (V1) input whereas the majority of recurrent connections in cortex are within cortical area (Binzegger et al., 2004; Douglas and Martin, 2007). It remains to be seen whether the dips in fano-factor, auto-correlation and cross-correlation in STSa neural responses are still seen in the absence of feedback from other cortical areas.

The trial-by-trial variability between neurones becomes less correlated for ~100ms starting at response onset (Smith and Kohn, 2008), at the same time as the signal correlation increases (Figure 9c-d). The dip in noise correlation is concurrent with the increase in signal correlation, suggesting that the “noise” from one input neurone is independent of other neurones, even when those other neurones convey related information. The overall impact of
the transient dips in response variability of individual neurones and the covariance of the
trial-by-trial variability between neurones is to increase the information conveyed by pairs of
neurones by up to 25%.

Whenever neurones are recorded on the same electrode, there is always a concern that
incomplete or inaccurate clustering will lead to artifactually high measures of cross-
correlation. Considerable effort was made to ameliorate this possibility in the spike sorting
stage by using a high threshold for cluster separation (see methods) and through visual
examination of the spike wave-forms. It is also noteworthy that the cross-correlation
coefficients reported here are towards the lower end of those reported in other studies using
spikes sorted from single neurones (e.g. Bair et al., 2001) and where multiple electrodes were
used (e.g. Kohn and Smith, 2005; Smith and Kohn, 2008). The data presented here are
consistent with previous reports and, if anything, suggest lower rather than higher correlation
than previous studies, suggesting that the potential confounds are small. Furthermore, the
focus here has been in the “dip” in the correlation rather than the absolute magnitude and it is
highly unlikely that this is a result of cross-contamination between clusters.

The mean firing rates of neurones were found to be correlated in the pre-stimulus
period (Figure 9c-d), an effect probably reflecting limited number of stimuli (typically 8-20)
and interactions between stimuli. The ongoing activity related to previous stimuli is,
however, independent of subsequent stimulus elicited activity (Figure 10c-d) and is therefore
probably different from those mechanisms underlying similar effects associated with
attention (Reynolds and Chelazzi, 2004). The SCM2 model shows that this effect may, like
the dips of the within neurone response statistics, be an artefact of sampling an instantaneous
change of process in the individual neurones rather than a change in the between neurone
statistics (Figure 11). Thus, while, the correlation between pairs LGN neurones is reduced by
cortical feedback from V1 (Andolina et al., 2007), the present study shows that a change of
generating process within the activity of single neurones explains much of the transient changes in the between cell correlation.

While the simple SCM2 model shows the transient “dip” in fano-factor, auto-correlation and cross-correlation of the responses, the model fails to capture the absolute magnitudes and the dependency of the correlation measures on lag. The decay of the correlation measures with lag between the sample windows indicates that the neuronal activity is governed at multiple time scales (Bair et al., 2001). The high level of interconnectivity within (Stratford et al., 1996; Binzegger et al., 2004; Douglas and Martin, 2007) and between (Felleman and Van Essen, 1991; Young, 1993; Young et al., 1994) cortical areas implies that the feedback/lateral inputs will operate through multiple loops of differing lengths and thus show auto-correlation and cross-correlation of their activity across multiple time scales. It remains to be seen whether incorporation of recurrent activity through multiple loops of different length into models of neural activity induce more accurate absolute measures of correlation as well as show the decrease of correlation with lag between sample windows.

In summary, the main neurophysiological findings reported here are of a transient drop in response variability and correlation of the trial-by-trial variability both within and between neurones at response onset. While these decreases are detectable using sample windows as small as 5ms duration, the effects are more noticeable with sample windows up to 100ms. Furthermore, the effects are seen in the absence of significant stimulus induced changes in activity levels. While the transient changes in response statistics appear relatively complex, modelling indicates that a simple change or resting of underlying process generating the neuronal activity within individual neurones can give rise to the complex observed neurophysiological phenomena. Critically, a change of process within single modelled neuronal spike trains induces changes in the response statistics of those spike trains.
between neurones. This simple mechanistic account of the transient changes in the response
statistics of both single and pairs of neurones may underlie up to a 25% increase in the
information conveyed by a neuronal population in the initial stimulus induced response.
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Stimulus induced decorrelation


Tolhurst DJ, Movshon JA and Dean AF. The statistical reliability of signals in single neurons in cat and monkey visual cortex. *Vis Res* 23: 775-785, 1983.


Figure Legends

**Figure 1. Measurement of single cell response statistics.** Rastergrams of the responses of a single cell to a single stimulus are shown in the top panels. (a) The fano factor from a window of 100 ms (upper, shaded area) was calculated every 5ms. The estimate was assigned to the mid-point of the window (dashed line). (b) The sequential correlation of the trial-by-trial variability (noise) was assessed using spike counts in two non-overlapping windows (upper, light and dark grey areas). The resultant estimate was assigned to the time of the start of the second window (dashed line).

**Figure 2. Modelling spike trains.** The three models all use spike density function (SDF, upper) of a neuron’s responses. To obtain modelled spike trains using the non-homogenous Poisson process (NHPP), the SDF can be normalised to give the probability of a spike, \( p(\text{spike}) \), in each millisecond (middle row, left). If a random number \([0..1]\) is less than \( p(\text{spike}) \), then a spike is allocated into the spike train (dashed lines, solid arrow to bottom left). In the spike count matched model (Oram et al., 1999, 2001; SCM, middle), the SDF is converted to the cumulative spike density function (CSDF). For each spike observed in the spike train (here 8 spikes), a uniform random number \([0..1]\) is transformed into a spike time by the inverse of the CSDF (solid arrows, dashed lines, bottom middle). In the two process SCM model (SCM2, right), the response period is split into two sections at 70ms, the typical latency of IT/STS neurones. The SCM model is then run for the initial period \([-200..69ms]\) (dashed line, solid arrows) and, separately, for the period \([70..600ms]\) (dashed line, open arrows).
Figure 3. Response statistics of single neurones assessed using varying window size.

Sequential response statistics averaged across 74 single neurones (see methods) are shown.

Upper row (a-c): The variability of the responses (fano factor±sem) assessed in a single sample window. A: Fano-factor plotted at the mid point of the sample window every 5 ms for sample windows of 5, 10, 25 50, 100 and 200ms duration. B: Comparison of the time course of the changes in the fano-factor (100ms window) and the average response (mean±sem, light gray). Dark shaded bars indicate periods used to estimate sequential correlation in the pre-stimulus (-200 to 0 ms), onset (70-120ms post-stimulus onset) and sustained periods (300-400ms post-stimulus onset) of the response. C: The mean correlation assessed in the pre-stimulus, onset and sustained periods of the responses as a function of window size. Middle row (d-f): The sequential correlation of the trial-by-trial variability (r_{auto.noise}±sem) between two successive windows. d: Correlation coefficient r_{auto.noise} plotted at the time of the start of the second window (end of the first window) every 5 ms for sample windows of 25 and 200ms. e: Comparison of the time course of the changes in r_{auto.noise} (100ms windows) and the average response (mean±sem, light gray). f: The mean r_{auto.noise} assessed in the pre-stimulus (dotted), onset (dashed) and sustained (solid) periods of the responses as a function of window size. Lower row (g-i): The sequential correlation (r±sem) of the mean responses to each stimulus (r_{auto.signal}±sem) between two successive windows. g: Correlation coefficient r_{auto.signal} plotted at the time of the start of the second window (end of the first window) every 5 ms for sample windows of 25 and 200ms. h: Comparison of the time course of the changes in r_{auto.signal} (100ms windows) and the average response (mean±sem, light gray). i: The mean r_{auto.signal} assessed in the pre-stimulus, onset and sustained periods of the responses as a function of window size.
Figure 4. Timing of minimum and maximum fano-factor and sequential-correlation. The time of the maximum (dashed lines) and minimum (solid lines) fano-factor (upper) and sequential correlation of the trial-by-trial variability, $r_{\text{auto\_noise}}$ (lower) of each neurone is plotted as a frequency distribution. Minimum values cluster in the period 50-150ms for both measures. Maxima occur in the time periods outside this.

Figure 5. Transient decrease in fano-factor and sequential-correlation to effective and ineffective stimuli. The average fano-factor (upper) and $r_{\text{auto\_noise}}$ (lower) assessed using 100ms window for 51 neurones where responses to both effective (solid line, dark gray=SEM) and ineffective (dashed line, light grey=SEM) stimuli were obtained. Inset shows the average spike density functions to the effective and ineffective stimuli.

Figure 6. Decay of sequential correlation. (a) The mean sequential correlation of the trial-by-trial variability ($r_{\text{auto\_noise}} \pm \text{sem}$) assessed using 100ms windows is plotted as a function of post-stimulus onset time for delays or lags between the sample windows of 0, 50, 100, 150 and 200 ms (solid, long dashed, dashed, short-dashed and dotted lines respectively). Values are plotted using the start of the later window. Thus, the correlation of the data from window (100..200) at lag 200ms corresponds to the window (-150..-100), lag 100ms corresponds to window (-100..0), lag 50ms (-50..50) and lag 0 (0..100). All these lags are all aligned at time 100. (b) $r_{\text{auto\_noise}} (\pm \text{sem})$ as a function of the lag between 100ms sample windows for the pre-stimulus (-200..0ms, dotted line), onset (70..120ms post-stimulus, dashed line) and sustained (300..400ms post-stimulus, solid line). (c) The sequential correlation of the mean responses ($r_{\text{auto\_signal}} \pm \text{sem}$) assessed using 100ms windows for lags of 0 to 200ms between the sample windows (line coding as in (a)). (d) $r_{\text{auto\_signal}} (\pm \text{sem})$ as a function of the lag between 100ms sample windows (line coding as in (b)).
Figure 7. Modelling statistics of single neurones. The fano-factor (a-c), the sequential correlation of the noise ($r_{\text{auto\_noise}}$, d-f) and sequential correlation of the signal ($r_{\text{auto\_signal}}$, g-i) of the non-homogenous Poisson process (NHPP, left column), the spike count matched model (SCM, middle column) and two process spike count matched model (SCM2, right column) are shown. Values were calculated using windows of 10, 50, 100 and 200ms (solid, short-dash, dashed, long-dash, solid lines respectively). Shaded areas represent ±sem.

Figure 8. Dependency of modelled statistics on window size and lag. The mean fano-factor (a-c), the sequential correlation of the noise ($r_{\text{auto\_noise}}$, d-f) as a function of window size, and $r_{\text{auto\_noise}}$ as a function of lag between samples (g-i) of the non-homogenous Poisson process (NHPP, left column), the spike count matched model (SCM, middle column) and two process spike count matched model (SCM2, right column). Shaded areas represent ±sem. Values were calculated using independent samples in the periods -200..0ms (Prestim, dotted lines), 70..120ms (Onset, dashed lines) and 300..400ms (Sustained, solid lines) post-stimulus onset.

Figure 9. Correlation between pairs of neurones. The mean cross-correlation (±sem) of the noise ($r_{\text{cross\_noise}}$, a,b) and signal (c,d) from pairs of simultaneously recorded neurones. Panels a and c show the cross-correlation for windows of 5,10,25,50,100 and 200ms (dotted, short-dash, dashed, long-dash, v long dash, solid lines respectively). Right panels show the mean cross-correlation of the noise (b) and signal (d) in the pre-stimulus (-200 to 0ms post-stimulus onset, dotted line) period, during response onset (70-120ms post-stimulus onset, dashed line) and in the sustained period of the response (300-400ms post-stimulus onset, solid line) as a function of the sample window size.
**Figure 10. Correlation between pairs of neurones as a function of lag between samples.**
The mean cross-correlation (±sem) of the noise ($r_{\text{cross\_noise\ a,b}}$) and signal ($r_{\text{cross\_signal\ c,d}}$) from pairs of simultaneously recorded neurones. Panels a and c show the cross-correlation for 100ms windows with a lag or delay of 0, 50, 100, 150 and 200ms (solid, long-dash, dash, short-dash, dotted lines respectively). Right panels show the mean cross-correlation of the noise (b) and signal (d) in the pre-stimulus (-200 to 0ms post-stimulus onset, dotted) period, during response onset (70-120ms post-stimulus onset, dashed) and in the sustained period of the response (300-400ms post-stimulus onset, solid) as a function of the lag between the sample windows.

**Figure 11. SCM2 model predicts dip in cross correlation.** The mean cross-correlation (±sem) of the noise ($r_{\text{cross\_noise}}$) from the SCM2 model. The cross-correlation was calculated using 100 surrogate trials for every trial in the neurophysiological data. **Upper:** The cross-correlation of the noise assessed using windows of 10, 50, 100 and 200ms are shown (short-dash, dash, long-dash and solid lines respectively). A dip in the cross-correlation is evident for all window sizes at the time when a change of process (70ms) occurs. **Lower:** The cross-correlation of the noise assessed using a 100ms window with lags of 0, 50, 100 and 200ms are shown (solid, long-dash, dash, short-dash and dotted lines respectively). While the change of process in the SCM2 model causes de-correlation (the dip), the correlation, unlike the neural data does not decay with time.

**Figure 12. Mutual information carried by pairs of neurones.** The mutual information between pairs of neurones for the response magnitudes at 100ms post-stimulus onset is plotted as a function of sample window size. The information using the observed values of
the average of the fano-factor of the individual neurones and $r_{\text{cross\_noise}}$, the trial-by-trial cross correlation, (solid line) is higher than using the average values from the pre-stimulus period (-200..0, dashed line). The signal correlation between the pairs of neurones was fixed at the value observed at 100ms post-stimulus onset. The information when the fano-factor and $r_{\text{cross\_noise}}$ were taken from the sustained period was almost identical to that taken from the pre-stimulus period.
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Table 1. Decrease in sequential correlation. Measures of the decrease in the sequential correlation of the trial-by-trial variability ($r_{\text{auto\_noise}}$) and the mean responses ($r_{\text{auto\_signal}}$) are shown for the window sizes of 5, 10, 25, 50, 100 and 200ms. **Sequential correlation of the noise.** Dip/Trend: the delays between the sample windows up to which $r_{\text{auto\_noise}}$ in the onset period is significantly below (p<0.05) or shows a trend (p<0.1) to be below both the pre-stimulus and sustained period estimates. **Tau:** The time constant for the exponential decay of $r_{\text{auto\_noise}}$. **Tau/Offset:** The time constant and offset from the regression $r_{\text{sequ}} = \text{Offset} + \beta e^{\text{log}/\tau}$. **Sequential correlation of the signal.** **Tau:** The time constant for the exponential decay of $r_{\text{auto\_signal}}$. **Tau/Offset:** The time constant and offset from the regression $r_{\text{ sequ}} = \text{Offset} + \beta e^{\text{log}/\tau}$. *=significant at p<0.05.
Table 2. Correlation of model and neuronal data. The correlation coefficient between the neural data and the different models (NHPP = non-homogenous Poisson process, SCM = spike count matched model, SCM2 = dual process spike count matched model) of the fano-factor, the sequential correlation of the trial-by-trial variability ($r_{auto\_noise}$) and the sequential correlation of the mean response ($r_{auto\_signal}$).
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Table 2