Repetition Suppression in Monkey Inferotemporal Cortex: Relation to Behavioral Priming

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McMahon DBT, Olson CR. Repetition suppression in monkey inferotemporal cortex: relation to behavioral priming. J Neurophysiol 97: 3532–3543, 2007. First published March 7, 2007; doi:10.1152/jn.01042.2006. In tasks requiring judgments about visual stimuli, humans exhibit repetition priming, responding with increased speed when a stimulus is repeated. Repetition priming might depend on repetition suppression, a phenomenon first observed in monkey inferotemporal cortex (IT) whereby, when a stimulus is repeated, the strength of the neuronal visual response is reduced. If the reduction resulted in sharpening of the cortical representation of the stimulus, and did not just scale it down, then speeded processing might result. To explore the relation between repetition priming and repetition suppression, we monitored neuronal activity in IT while monkeys performed a symmetry decision task. We found 1) that monkeys exhibit repetition priming, 2) that IT neurons simultaneously exhibit repetition suppression, 3) that repetition priming and repetition suppression do not vary in a significantly correlated fashion across trials, and 4) that repetition suppression scales down the representation of the stimulus without sharpening it. We conclude that repetition suppression accompanies repetition priming but is unlikely to be its cause.

INTRODUCTION

Repetition priming is a behavioral phenomenon, widely studied in humans, whereby prior experience with a stimulus leads to faster discrimination on subsequent encounters. Priming is both rapid and long-lasting: it can be induced by a single presentation of a visual stimulus, and has been shown to persist for as long as a year (Cave 1997). The fact that priming effects are not dependent on explicit memory has led to the suggestion that they are mediated by a distinct implicit memory system that operates on representations in sensory cortex (Schacter and Buckner 1998; Tulving and Schacter 1990).

Repetition priming has been of particular interest in recent years as a behavioral probe for characterizing the structure of neural representations in humans (Henson 2003). In experiments based on this approach, subjects are required to respond to a series of visual stimuli some of which reappear in a transformed version, for instance at a different scale or orientation. Insofar as behavioral responses are speeded by prior exposure, the populations of neurons representing the original and the transformed version are inferred to overlap. Conversely, if priming does not occur, the populations of neurons representing the two images are inferred to be distinct (Bar and Biederman 1999; Biederman and Cooper 1991; Schacter et al. 1990; Srinivas 1995).

Repetition suppression is a physiological phenomenon, first demonstrated in recording studies of monkey inferotemporal cortex (IT), whereby the strength of the neuronal response to a visual stimulus declines over successive presentations. Repetition suppression occurs irrespective of whether monkeys are required to process visual stimuli or must simply engage in passive fixation (Baylis and Rolls 1987; Li et al. 1993; Riches et al. 1991; Sobotka and Ringo 1993). An analogous phenomenon—magnetic resonance (MR) adaptation—has been observed in studies of humans: when a visual stimulus is presented repeatedly the induced blood oxygenation level–dependent (BOLD) response in ventral stream visual cortex declines (Buckner et al. 1998; Grill-Spector et al. 1999).

Repetition suppression, like repetition priming, has been widely used as a probe for characterizing the structure of neural representations in humans. In a manner analogous to the use of behavioral measures in priming experiments, the MR adaptation technique seeks to draw inferences about the response properties of single neurons from the degree of stimulus invariance of BOLD signal adaptation (Grill-Spector et al. 1999; Kourtzi and Kanwisher 2001; Vuilleumier et al. 2002). The general logic of this approach is as follows (Grill-Spector and Malach 2001). First, subjects view an adapting stimulus. Then BOLD responses are evoked by a test stimulus, which may differ in some respect from the adapting stimulus. The degree to which MR adaptation occurs despite the stimulus transformation is taken as indicating the degree to which individual neurons are driven by both the adapting stimulus and the test stimulus. A somewhat different procedure has been employed in earlier visual cortex, in which a rebound in BOLD signal following a prolonged period of adaptation is interpreted as indicating selectivity for the feature dimension that was changed between the adapting stimulus and the test stimulus. In this approach, the observation of interest is the recovery from adaptation, rather than the magnitude of adaptation itself (Tolias et al. 2001).

It has been proposed that repetition priming at the behavioral level depends on repetition suppression at the physiological level. The notion that the two phenomena are related is suggested by several similarities between them (reviewed by Wiggs and Martin 1998). First, under some circumstances repetition suppression induced by a single stimulus presentation can be extremely long lasting (Fahy et al. 1993). Second, both priming and repetition suppression are cumulative over multiple presentations (Li et al. 1993). Third, the magnitude of

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Suppression decreases as the interval between first and second stimulus presentations increases (Fahy et al. 1993; Li et al. 1993). Finally, decreases in visual response strength accompany behavioral priming in human imaging studies (Buckner et al. 1998; Grill-Spector et al. 2006; Henson 2003).

Whether repetition suppression indeed contributes to repetition priming must depend in part on how it affects the stimulus selectivity of neurons. Two models of its effect have been put forward. In the sharpening model, repetition suppression occurs among neurons weakly responsive to the stimulus and not among neurons strongly responsive to it (Desimone 1996; Wiggs and Martin 1998). When the stimulus is repeated, neurons selective for it (those that previously fired strongly) respond at full strength, whereas neurons selective for other stimuli (those that previously fired weakly) respond at reduced strength. Thus the cortical representation of the stimulus is sharpened. This could give rise to an improvement in visual discrimination performance. In the scaling model, when a stimulus is repeated, the strength of the response is reduced from its previous level by a fixed proportion. Thus the cortical representation of the stimulus is unaltered except insofar as it is weakened. The scaling model is assumed in studies that use MRI adaptation to characterize neuronal selectivity (Avidan et al. 2002). It is difficult to see how a scaling reduction could give rise to repetition priming. The only idea put forward to date is that a reduction in the firing rate might be accompanied by an increase in the degree to which neurons fire spikes synchronously (Gotts 2003).

The first aim of the present study was to determine whether repetition suppression, as measured at the neuronal level in IT, occurs during repetition priming, as measured at the behavioral level. The second aim was to determine, in the event of its occurring, whether it has properties indicative of its causing behavioral priming. Repetition priming has not previously been demonstrated in monkeys. Most priming tasks commonly used with humans, such as naming pictures and making lexical decisions, require a semantic judgment about the stimulus and consequently are not appropriate for use with nonhuman species (Buckner et al. 1998; Dobbins et al. 2004; Maccotta and Buckner 2004; Sayres and Grill-Spector 2006; reviewed by Henson 2003). Tasks requiring symmetry judgments are an exception. Symmetry judgments are both sensitive to priming in humans (Kersteen-Tucker 1991) and possessed of the critical feature that they depend on the global structure of an image rather than on its semantic attributes. Accordingly, we trained two rhesus macaques to discriminate between symmetric and asymmetric images and recorded from neurons in IT while they made symmetry judgments in response to the first and second presentations of numerous images. Using this approach, we addressed the following specific questions. 1) Do monkeys exhibit repetition priming? 2) Does repetition suppression accompany priming? 3) Is the degree of priming correlated across trials with the degree of suppression? 4) Does repetition suppression sharpen or simply scale down the cortical representation of the stimulus?

**METHODS**

**Surgical procedures**

Two adult male rhesus macaque monkeys [laboratory designations EG and PH; hereafter referred to as m1 (10.2 kg) and m2 (11.1 kg)] were used. All experimental procedures were approved by the Carnegie Mellon University Animal Care and Use Committee and were in compliance with the guidelines set forth in the United States Public Health Service Guide for the Care and Use of Laboratory Animals. At the outset of the training period, each monkey underwent sterile surgery under general anesthesia maintained with isofluorane inhalation. The top of the skull was exposed, bone screws were inserted around the perimeter of the exposed area, a continuous cap of rapidly hardening acrylic was laid down so as to cover the skull and embed the heads of the screws, a head-restraint bar was embedded in the cap, and scleral search coils were implanted on the eyes, with the leads directed subcutaneously to plugs on the acrylic cap. After initial training, a 2-cm-diameter disk of acrylic and skull overlying the right hemisphere was removed to allow for the positioning of a vertically oriented cylindrical recording chamber. The chamber was centered at approximately anterior 18 mm and lateral 18 mm with respect to the Horsley–Clarke reference frame (Fig. 1).

**Monitoring single-neuron activity and eye position**

At the beginning of each day’s session, a varnish-coated tungsten microelectrode with an initial impedance of several megohms at 1 kHz (FHC, Bowdoinham, ME) was advanced through the dura into the underlying cortex. The electrode was introduced through a translational guide tube advanced to a depth such that its tip was about 10 mm above IT. The electrode could be advanced reproducibly along tracks forming a square grid with 1-mm spacing. The action potentials of a single neuron were isolated from the multi-neuronal trace by means of an on-line spike-sorting system using a template matching algorithm (Signal Processing Systems, Prospect, Australia). The spike-sorting system, on detection of an action potential, generated a pulse the time of which was stored with 1-ms resolution. Eye position was monitored by means of a scleral search coil system (Riverbend Instruments, Birmingham, AL) and the x and y coordinates of eye position were stored with 4-ms resolution. To measure saccadic reaction times, we first identified the saccade by locating the peak of the eye velocity trace. We then located the onset of the saccade, which was defined as the time before the velocity peak at which the eye velocity crossed a threshold of 15°/s.

**Task and stimuli**

All aspects of the behavioral experiment, including stimulus presentation, eye position monitoring, and reward delivery, were under control of a Pentium PC running Cortex software (provided by R. Desimone). Both monkeys were trained to perform the task dia-
The monkey initiated each trial of the task by fixating within approximately 2° of a central white dot presented against a black background. After a 200-ms-fixation interval a white stimulus appeared at the center of the screen. The stimulus was either symmetric or asymmetric across the vertical midline. Simultaneously with stimulus onset, two white target dots appeared 4.8° above and below the horizontal midline. Monkey m1 was rewarded for making a saccade to the upper target if the stimulus was symmetric or to the lower target if the stimulus was asymmetric. The association between symmetry and saccade direction was reversed for monkey m2. Incorrect trials were followed by a pause of 1,200 ms. The stimulus onset asynchrony averaged over all trials was about 1,900 ms.

Sixty symmetric and 60 asymmetric stimuli were presented two times each over the course of a single session, for a total of 240 trials. The order of stimulus presentation was constrained so that the lag between the first and second presentations of every stimulus was either 0, 1, 2, 4, 8, or 16 intervening different stimuli (Fig. 2B). The number of consecutive runs of the same type of stimulus (either symmetric or asymmetric) was constrained to be consistent with the runs generated by a binomial process, so that the correct response on any given trial was independent of the correct response on any other trial. To generate sequences that satisfied these criteria in an efficient manner, we developed a recursive algorithm that randomly generated segments of 24 trials, containing 12 symmetric and 12 asymmetric trials that included repeats at all six lags. Ten different such segments were then concatenated together to produce the sequence for a single experimental session.

Each white stimulus consisted of a central bar 0.5° wide and 2.3° high, a right-side flanking shape, and a left-side flanking shape. The entire stimulus, consisting of the central body and the flanking shapes, fit within a 2.3 × 2.3° square. For each session, a new set of 120 stimuli was constructed by random selection without replacement from a library of 180 available right-side flanking shapes. From 60 of these shapes were constructed 60 symmetric stimuli, each consisting of the central bar, the right-side shape (on the right), and its mirror image (on the left). From the remaining 120 shapes were constructed 60 asymmetric stimuli, each consisting of the central bar, one right-side shape (on the right), and the mirror image of another right-side shape (on the left). This procedure ensured that the monkeys were exposed to all flanking shapes with equal frequency and that all flanking shapes had the same probability of being contained in a symmetric versus an asymmetric stimulus. However, it led to the consequence that, across multiple sessions, symmetric stimuli tended to recur, whereas asymmetric ones did not. This followed from the fact that there were only 180 possible symmetric stimuli, whereas there were 32,220 possible asymmetric ones.

Assessing response bias effects

The effect of stimulus type (symmetric vs. asymmetric) was quantified according to the formula

$$ B = p(S | S) - p(a | A) $$

where $p(S | S)$ is the probability of a correct response to a symmetric stimulus and $p(a | A)$ is the probability of a correct response to an asymmetric stimulus. Thus response bias could range from +1 (if the monkey always responded “symmetric”) to −1 (if the monkey always responded “asymmetric”) and would be 0 if there were no bias.

Assessing priming and suppression effects

Effects of stimulus repetition on both behavioral reaction time (RT) and neuronal firing rate were assessed by pooling responses across sessions. Behavioral priming was assessed as $RT_1 - RT_2$, and neuronal suppression was assessed as $FR_1 - FR_2$, where $RT_1$ and $RT_2$ are the reaction times and $FR_1$ and $FR_2$ are the firing rates associated with the first and second presentations of a stimulus. All analysis was restricted to stimuli to which the monkeys responded correctly on both first and second presentations. Trials on which the RT was faster than 200 ms or slower than 600 ms (which amounted to <1% of the total trials) were excluded from analysis. To compute the firing rate response to each stimulus, we counted the number of spikes fired within an adjustable time window that started 80 ms after stimulus onset (on account of the latency of visual response) and ended with the faster of the two saccadic response times $RT_1$ and $RT_2$. Computing firing rate in this manner, as opposed to using a fixed integration window, ensured that spikes fired after the saccade did not contribute to our FR measurements. To restrict our analysis to responses to visual stimuli that were effective at driving the neurons, we computed a threshold using a procedure modeled after signal-detection theoretical analysis. First, we obtained a distribution of “noise” responses by measuring the spike counts obtained during the 200-ms-fixation period before stimulus onset. Second, we obtained a distribution of putative “signal” responses by measuring the spike counts obtained during 80–280 ms after stimulus onset. We then chose a threshold that resulted in the best separation between the signal and noise distributions. This procedure was carried out separately for each neuron, so that cells with high baseline activity would be assigned a higher threshold than neurons with low baseline activity. Because there was a tendency toward a transient, nonstimulus-specific decrease in both RT and firing rate very early in a session, we excluded the first 12 trials of each session from subsequent analysis to restrict our focus to data collected under stationary conditions.
Memory kernel analysis of response repetition effects

To determine whether repetition of the motor response (as distinct from repetition of the stimulus) contributed to priming, we performed a memory kernel analysis characterizing the impact of motor response repetition on RT (Maljkovic and Nakayama 1994). This procedure involved computing (RT_{different} − RT_{same}), where RT_{different} is the mean RT on trials requiring that the monkey make a saccade in the direction opposite to the direction of the preceding trial’s saccade and RT_{same} is the mean RT on trials involving a different stimulus but requiring the monkey to make a saccade in the same direction. Only pairs of trials on which the monkey responded correctly to both presentations were included in the analysis. This procedure was carried out independently for each lag in the range 0 to 16. The lag 16 analysis, for example, characterized the impact on RT of the motor response executed on the trial immediately prior to 16 intervening trials. The time course of response priming was fit to an exponential decay function \( y = A \cdot e^{-t/\tau} + C \). Significance of kernels was evaluated for each monkey and each lag by rank-sum tests, with Bonferroni correction.

Assessing the latency of suppression

To determine the latency of the suppression effect at the population level, we analyzed data from all pairs of trials in which a neuron fired at least one spike during the interval from 80 to 280 ms after stimulus onset during either the first or second presentation of the stimulus and in which the monkey had responded correctly on both trials. Spikes were counted within a 40-ms sliding boxcar window that was moved incrementally along the time axis in 1-ms steps. At each step, we used a two-sided, paired t-test to compare the firing rates evoked on the first and second presentations of each stimulus. The boxcar continued to slide until the result of the t-test indicated a statistically significant difference \( P < 0.05 \) between the first and second presentation for ten consecutive steps of the boxcar. Having thus found a period of sustained differences between the two responses, we then identified the latency of suppression as the earliest time point inside the first window in the string of ten.

To assess whether any observed differences in latency across lags were statistically significant, we performed a bootstrap analysis. A single simulation was carried out by pooling all trials from two conditions together (for instance, all trials from lags 0 and 1) and then obtaining two simulated populations of trials by resampling the pooled set of trials randomly with replacement. The latency of suppression was then computed for these two simulated populations using the same procedure applied to the experimentally observed data sets. We conducted 1,000 such simulations to obtain a distribution of latency differences that could be expected to occur by chance. Experimentally observed latency differences were compared with this distribution and were considered statistically significant if they were greater than the 95th percentile of simulated latency differences.

Assessing the correlation between priming and suppression

To assess whether neuronal suppression and behavioral priming were correlated, we performed a Pearson correlation analysis across all pairs of trials between measures of both priming and repetition suppression. The analysis was confined to cases in which the monkey responded correctly to both the first and second presentations of the stimulus. We estimated the power of this correlation analysis by conducting simulations in the following manner. Distributions of neuronal and behavioral responses were obtained from the experimental data set and were designated as X and Y, respectively. These distributions were then resampled in the following manner to generate four simulated observations. The physiological responses to each artificial stimulus were computed as

\[
\text{fr}_1 = \text{Pois}(X_i) \\
\text{fr}_2 = \text{Pois}(X_i + s)
\]

(2)

where fr_1 and fr_2 represent the simulated firing rate on first and second presentations. X_i is the expected firing rate for artificial stimulus n, determined by a random sample drawn with replacement from X. The function Pois generates a Poisson random number with a mean of X_i. The constant s is a suppression factor, derived from the data and set to 0.95. The behavioral responses to each artificial stimulus were computed as

\[
\text{rt}_1 = \text{Resam}(Y) \\
\text{rt}_2 = \text{Resam}(Y - b(\text{fr}_1 - \text{fr}_2))
\]

(3)

where rt_1 and rt_2 represent the simulated reaction time on first and second presentations. The function Resam draws a random sample with replacement from the RT distribution Y. The constant b, which introduces a dependency between the simulated priming and suppression, was set to 1. Priming and suppression were computed as

\[
\text{priming} = \text{rt}_1 - \text{rt}_2 \\
\text{suppression} = \text{fr}_1 - \text{fr}_2
\]

(4)

Thus the parameter b is the expected slope of the priming–suppression correlation. We conducted 1,000 simulations in total. Each consisted of paired behavioral and neuronal responses to 9,407 artificial stimuli, a number equal to that of the paired responses obtained experimentally. For each simulated data set, we computed a Pearson correlation analysis in a manner identical to the analysis performed on the experimentally obtained data set.

Assessing the test–retest reliability of stimulus selectivity

To determine whether the selectivity profiles of IT neurons were adequately characterized by a single presentation per stimulus, we conducted an analysis independently for each neuron of the correlation across all stimuli between the firing rate evoked on the first presentation and that evoked on the second presentation. The analysis was confined to cases in which the monkey responded correctly to both presentations.

Assessing the impact of suppression on stimulus selectivity

Analysis of the relation between suppression magnitude and visual response strength was based on pairs of stimuli repeated at lags 0 through 4 because these shortest lags produced significant suppression effects. The analysis was confined to cases in which the monkey responded correctly to both presentations of a stimulus and in which the stimulus was effective at driving the cell. For each trial, we first computed the firing rate in a window extending from 150 ms after stimulus onset to the faster RT of the two presentations. The selection of this window was based on the observation that repetition suppression did not develop until 150 ms after stimulus onset. A tuning curve reflecting stimulus selectivity was then constructed for each neuron on the basis of mean firing rate averaged over the first and second presentations of the same stimuli. These mean firing rates were then adjusted for baseline firing rate by subtracting the mean firing rate during the fixation epoch 200 ms before stimulus onset. Each tuning curve was constructed by 1) ranking the n stimuli from 1 to n according to the strength of the elicited response, with ties decided arbitrarily; 2) assigning to each stimulus a rank index r corresponding to its location in the sequence; 3) computing for each stimulus a normalized rank index \( r = r/b \), where b is the rank of the top-ranked stimulus; and 4) computing, for each of 30 bins of width 1/30 spanning the range 0 to 1, the average firing rate elicited by stimuli in that bin. Adopting a uniform binning procedure made it possible to combine data across neurons despite the fact that the number of stimuli included in the analysis differed from neuron to neuron. The
RESULTS

Behavioral signs of repetition priming

Each monkey performed a symmetry judgment task (Fig. 2). On each trial of this task, a white shape appeared at fixation. The monkey was required to make a saccade in one direction in response to a symmetric shape and a saccade in the opposite direction in response to an asymmetric shape. Across a session consisting of 240 trials, each of 120 stimuli was presented twice. Half of the stimuli were symmetric and half asymmetric. With equal frequency, the lag between the first and second presentations (the number of intervening trials) was 0, 1, 2, 4, 8, and 16. Further details are given in METHODS, Task and stimuli.

Behavioral and electrophysiological data were collected simultaneously during 118 sessions (67 in m1; 51 in m2). Both monkeys performed the symmetry decision task with a high degree of accuracy and speed: the mean percent of correct responses was 84.3% in m1 and 80.2% in m2, whereas the mean reaction time (RT) of correct responses was 311 ms in m1 and 321 ms in m2. Ninety percent of all saccades fell between 235 and 453 ms. Both monkeys, and particularly m2, displayed a response bias favoring symmetric stimuli. This bias was reflected both by a shorter RT for symmetric stimuli (shorter by 15 ms in m1; 127 ms in m2) and by greater accuracy (symmetry bias: +0.04 in m1; +0.28 in m2). Both measures were significantly biased in each monkey, and the bias was significantly greater in m2 than in m1 (P < 0.05, chi-squared tests). In light of this effect of stimulus type on behavior, we present the results for each monkey and stimulus type separately, as well as describing the pooled data.

Because particular symmetric stimuli appeared more often across sessions than particular asymmetric stimuli, there is a possibility that the monkeys responded faster to symmetric stimuli because they were more familiar. Familiarity results in faster reaction times in lexical decision tasks in human subjects (McKone 1995). To assess this possibility, we conducted a trend analysis of symmetry bias as a function of experimental session. Because the stimulus set used for data collection was entirely different from the set used to train the monkeys, both symmetric and asymmetric stimuli were initially novel. Because particular symmetric stimuli recurred from one session to the next (on average, 6.7 of 60 symmetric stimuli were the same between any given pair of sessions), they presumably became familiar. Thus a trend toward increasing symmetry bias over session number would reflect a familiarity effect that could be distinguished from response bias attributable to symmetry in itself. In m1, there was no significant trend in bias over sessions (R = 0.086, P = 0.49), indicating that familiarity did not contribute to response bias. In m2, there was a significant trend (R = 0.47, P = 4.5 × 10−3), however, a strong symmetry bias was clearly present even at the outset of data collection. The γ-intercept of the regression line was significantly greater than zero (+0.21, P = 1.4 × 10−11), in harmony with the observation that the mean symmetry bias over the first five sessions was +0.22. We conclude that both monkeys exhibited a response bias in favor of symmetry that was independent of stimulus familiarity.

To determine whether the monkeys exhibited repetition priming, we pooled behavioral data across all 118 experimental sessions. The number of paired stimulus presentations that met the criteria for analysis (see METHODS, Assessing priming and suppression effects) ranged, across the six lags, from 1,434 to 1,500 (mean: 1,461). Stimulus repetition indeed resulted in robust priming evidenced by a reduction of reaction time from the first to the second presentation of a stimulus (Fig. 3). Priming was significant at all lags (P < 5 × 10−7, one-sided Wilcoxon signed-rank test) and was especially pronounced at lag 0 (P = 10−28). When the data were subdivided by monkey (Fig. 3A) or by stimulus type (Fig. 3B), priming persisted, attaining significance in 20 of the 24 cases obtained by crossing four categories (m1, m2, symmetric, asymmetric) with six lags (0, 1, 2, 4, 8, 16).

The experiment was not designed to test explicitly the impact of time interval, as distinct from the number of intervening trials, between repetitions. Moreover, because the monkeys were motivated to respond as quickly as possible in order to collect the reward efficiently, the time interval between stimulus presentations tended to be tightly linked to the lag. Although the fact that inaccurate responses were followed by a 1,200 ms pause resulted in some variance in the time interval between 235 and 453 ms. Both monkeys performed the symmetry decision task with a high degree of accuracy and speed: the mean percent of correct responses was 84.3% in m1 and 80.2% in m2, whereas the mean reaction time (RT) of correct responses was 311 ms in m1 and 321 ms in m2. Ninety percent of all saccades fell between 235 and 453 ms. Both monkeys, and particularly m2, displayed a response bias favoring symmetric stimuli. This bias was reflected both by a shorter RT for symmetric stimuli (shorter by 15 ms in m1; 127 ms in m2) and by greater accuracy (symmetry bias: +0.04 in m1; +0.28 in m2). Both measures were significantly biased in each monkey, and the bias was significantly greater in m2 than in m1 (P < 0.05, chi-squared tests). In light of this effect of stimulus type on behavior, we present the results for each monkey and stimulus type separately, as well as describing the pooled data.

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between the first and second stimulus presentations for long lag repeats, there was no significant correlation between absolute time interval and the magnitude of RT priming ($P > 0.1$) at any of the lags. Nonetheless, in light of human priming studies specifically investigating this issue (McKone 1998), it is likely that both the elapsed time between repeats and interference from intervening stimulus presentations contribute to the reduction in priming magnitude observed at longer lags.

To assess the contribution of specific experimental parameters to the priming effect, we conducted a three-way ANOVA on the magnitude of priming ($\text{RT}_1 - \text{RT}_2$) with lag, stimulus type, and monkey as factors. There were highly significant main effects of all three factors (Table 1). The main effect of lag arose from the particularly strong effect at lag 0, which was significantly higher than effects at all other lags. Lags 1 through 16 did not differ among themselves, as determined by a Tukey post hoc test. The main effects of monkey and type reflected the fact that priming tended to be stronger in m1 (Fig. 3A) and for symmetric stimuli (Fig. 3B). These effects were themselves lag dependent, as indicated by a significant interaction effect lag $\times$ type, reflecting a greater lag 0 priming effect for symmetric than for asymmetric stimuli (Tukey test).

Because two presentations of the identical stimulus necessarily called for eye movements in the same direction, the behavioral priming effects of stimulus repetition could in principle have been related either to processing the same stimulus or to producing the same response. Response priming has been observed in both human and monkey studies (Dorris et al. 1999; Maljkovic and Nakayama 1994). To determine whether the priming effects we observed could be partially attributable to response repetition, we adapted from Maljkovic and Nakayama (1994) the method of memory kernel analysis (see METHODS, Memory kernel analysis of response repetition effects). At the two shortest lags, we observed a tendency for the behavioral reaction to be slower when the required response was in the same direction as on a preceding trial as compared to when it was in the opposite direction (Fig. 4). This effect is opposite to the one observed in response priming. It is consonant instead with inhibition of return (Bichot and Schall 2002). We conclude that the repetition priming as observed in our study is genuinely due to stimulus repetition and that there is no contribution from response priming.

### TABLE 1. Three way ANOVA on behavioral priming effects

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>Sum Sq.</th>
<th>Mean Sq.</th>
<th>F-Statistic</th>
<th>P-Value</th>
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<td>4.47 $\times 10^4$</td>
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Results from a multi-factor ANOVA assessing the effects of the factors lag (0, 1, 2, 4, 8, or 16), stimulus type (symmetric or asymmetric), and monkey (m1 or m2) on behavioral reaction time differences between prime and probe stimuli ($\text{RT}_1 - \text{RT}_2$).

**FIG. 4.** Responses (as distinct from stimuli) were not primed at any lag. Data are shown for the 2 monkeys together and for m1 and m2 separately. Response priming was computed as mean $\text{RT}_{\text{diff}}$ - mean $\text{RT}_{\text{same}}$, where $\text{RT}_{\text{diff}}$ is the reaction time on a trial requiring a saccade opposite to that executed on a trial preceding at the indicated lag and $\text{RT}_{\text{same}}$ is the reaction time on a trial requiring a saccade in the same direction as that executed on a trial preceding at the indicated lag with consideration restricted to cases in which the stimuli were different. A positive value would indicate speeding of the saccadic reaction time on trials requiring that a given saccade be repeated (response priming). The negative values observed at lags 0 and 1 indicate that there was actually a slowing (inhibition of return). Solid line indicates the best fit to an exponential decay function.

**Electrophysiological signs of repetition suppression**

Having established that monkeys exhibit repetition priming, we proceeded to evaluate whether it is accompanied by suppression of visual responses at the single-neuron level. We monitored the activity of 152 neurons in anterior IT (85 in m1; 67 in m2) during the same 118 sessions on which the behavioral analysis was based. The number of paired stimulus presentations that met the criteria for analysis (see METHODS, Assessing priming and suppression effects) ranged, across the six lags, from 1,064 to 1,173 (mean: 1,136). Stimulus repetition indeed resulted in a robust reduction in the strength of the neuronal response from the first to the second presentation of a stimulus (Fig. 5). The suppression effect was highly significant at lags of 0 and 1 ($P = 3 \times 10^{-5}$ and $P = 6 \times 10^{-4}$, respectively, one-sided signed-rank test) and decreased as a function of lag, with suppression becoming insignificant at lags 8 and 16. When the data were subdivided by monkey (Fig. 5A) or by stimulus type (Fig. 5B), suppression persisted, attaining significance in 10 of the 24 cases obtained by crossing four categories (m1, m2, symmetric, asymmetric) with six lags (0, 1, 2, 4, 8, 16).

To assess the contribution of specific experimental parameters to the suppression effect, we conducted a three-way ANOVA on the suppression measure ($\text{RT}_1 - \text{RT}_2$) with lag, stimulus type, and monkey as factors (Table 2). There was a significant main effect of lag (stronger suppression at short lags), but no other significant effects. We conclude that repetition suppression in single IT neurons accompanies repetition priming at the level of behavior.

The preceding analysis was based on the spike count within an adjustable time window after stimulus onset. The results
indicate clearly that, when a stimulus is repeated, the net strength of the neuronal visual response is reduced. This reduction might or might not be accompanied by a change in the dynamics of the response. There is a model, the accumulator model, according to which, when a stimulus is presented for a second time, there is a change in the dynamic pattern of the neuronal response: firing builds up more rapidly than on the first occasion, attains threshold sooner than on the first occasion, and then is truncated, with the result that fewer spikes are fired than in response to the first presentation (James and Gauthier 2006). To determine whether the neuronal response dynamics were altered on repetition of a stimulus, we constructed histograms representing the average firing rate, across all neurons and stimuli, as a function of time after the first and second presentations (see METHODS, Assessing the latency of suppression). In agreement with previous reports obtained outside the context of the priming paradigm (Li et al. 1993; Ringo 1996), we found that the initial phase of the visual response was identical between the first and second presentations and that suppression took hold only late in the response (Fig. 6). The population visual response began 80 ms after stimulus onset on both the first and second presentations, but repetition suppression did not emerge until 147 ms at lag 0 (see METHODS, Assessing the latency of suppression). The latency of suppression was progressively later at longer lags, although a bootstrap analysis indicated that this trend was not significant and may simply be due to the decrease in magnitude of the effect. These findings place several constraints on the nature of the mechanism that underlies repetition suppression. The observation that the firing rate did not rise more sharply during the second presentation is not consistent with the accumulator model. The observation that suppression takes hold only late in the response argues against its being inherited from an earlier visual area where effects such as contrast adaptation are generally evident at the onset of the visual response (Muller et al. 1999). It suggests instead that suppression arises from recurrent circuitry either intrinsic to IT or reciprocally linking IT to other areas.

Comparison between effects of stimulus repetition on behavioral and neuronal responses

Repetition priming and repetition suppression were demonstrated above as trends across all pairs of stimulus presenta-
tions. Across pairs of presentations there was considerable variability. We now ask whether variations in the strength of repetition priming were correlated with variations in the strength of repetition suppression. This question is of interest because the answer could cast light on whether the two phenomena are causally related. To assess whether there was a correlation, we performed a Pearson correlation analysis on measures of repetition priming and repetition suppression on observations pooled across all 152 neurons (Fig. 7A). There was no evidence of a correlation between behavioral priming and neuronal suppression ($R = 0.0067$, $P = 0.51$). This analysis was based on all stimuli to which the monkey responded correctly on both the first and second presentations ($n = 9,407$). It could be argued that, by using this permissive selection criterion, we included a high degree of noise that might obscure a true underlying correlation. To address this possibility, we applied the following filters to the data set. First, neurons were excluded if they failed to display repetition suppression when responses to all stimuli were averaged. Second, neurons that failed to display significant test–retest reliability (i.e., those neurons that fell to the right of $P = 0.05$ in Fig. 8B) between first and second presentations were excluded from analysis. Third, we excluded responses to stimuli that were identified as subthreshold on the basis of the signal-detection thresholding filter (described in METHODS, Assessing priming and suppression effects). There was still no significant correlation when the correlation analysis was repeated on this more restricted data set ($n = 2869$, $R = 0.0071$, $P = 0.70$). Finally, we asked whether the motor response on the immediately preceding trial might have introduced noise that masked a true correlation between priming and suppression. Although this possibility seemed plausible in light of the finding that consecutive responses in the same direction led to inhibition of return (Fig. 4), the correlation between priming and suppression remained insignificant when the data were divided into subsets according to whether the monkey responded in the same or the different direction on the immediately preceding trial ($P > 0.05$ for both same and different response directions). Thus there was no detectable correlation between the strength of repetition suppression and the strength of repetition priming.

Because the correlation analysis was of limited power, we cannot rule out the possibility that there was a very weak cross-trial correlation between suppression and priming. To investigate how strong a correlation would have escaped being identified as significant by our analysis, we studied simulated data sets generated by resampling the observed RT and firing rate distributions. In each simulation, a dependency was artificially introduced between priming and suppression. The parameter $b$ (see METHODS) was set to 1, simulating a situation in which, on average, the reaction-time measure of priming ($RT_1 - RT_2$) increased by 1 ms for every increase of 1 spike/s in the firing-rate measure of suppression ($FR_1 - FR_2$). The distribution of $R$ values obtained from 1,000 simulated data sets is shown in Fig. 7B. The $R$ value obtained from the experimental data set ($R = 0.0067$) is less than the mean of the simulated values ($R = 0.026$) and falls below the fifth percentile of the simulated distribution (percentile = 3.5). Thus we can reject (one-tailed test, $P < 0.05$) the hypothesis that the experimental value was drawn from the simulated distribution. Conversely, we can reject the hypothesis that there was a correlation in the experimental data set as strong as the simulated correlation. From the fact that, with $b = 1$, the experimental value is close to the fifth percentile of the simulated distribution, it follows that, with $b \ll 1$, it would be above the fifth percentile. Accordingly, we cannot reject the presence in the experimental data set of a correlation considerably less than that obtained with $b = 1$. How severe a limit is this? To answer this question, we consider that the fraction of cross-trial variance in reaction time explained by firing rate is equal to $R^2$. Given the mean value of $R$ across the simulated cases (0.026), we conclude that our analysis would have identified as significant a correlation such that firing rate explained only a tiny fraction (0.026$^2 = 0.00068$) of cross-trial variance in reaction time. For all practical purposes, then, we have ruled out the existence of a correlation.

Effect of repetition suppression on stimulus selectivity

In eliciting behavioral priming, we necessarily used a large set of stimuli chosen without respect to the individual neuron’s preferences (pretesting the stimuli would have compromised their efficacy as primes and probes) and presented each stimulus only twice (as required for demonstrating priming). It might be questioned whether the neurons were commonly selective for the stimuli and whether, if they were selective, we could demonstrate their selectivity using so few presentations. To answer these questions, we conducted a linear regression analysis on the visual responses evoked by first and second presentations across all cases in which the monkey responded correctly to both presentations (see METHODS, Assessing the test–retest reliability of stimulus selectivity). An example is shown in Fig. 8A. For this cell, the relation between the firing rates on the first and second presentations was highly signifi-
Neurons exhibited significant selectivity for the visual stimuli. A: data from a single neuron from m1. For each of 78 stimuli (all of those to which the monkey responded correctly on both presentations), the number of spikes fired in response to the first presentation is plotted against the number of spikes fired in response to the second presentation. The response to the second presentation was suppressed as indicated by the fact that the majority of the points fall below the identity line (hatched). However, there was a clear trend for stimuli eliciting a strong response on the first presentation also to do so on the second presentation as indicated by the fact that the best-fit line (solid) has a positive slope. The correlation between the number of spikes fired in response to the first presentation and the number fired in response to the second presentation was highly significant \( R = 0.67, P = 3 \times 10^{-11} \). B: results obtained for all 152 neurons by the correlation analysis depicted in A. Each point represents one neuron. On the y-axis is plotted the corresponding \( P \) value. There was a positive correlation in 142 of 152 neurons (points above the hatched horizontal line). This reached statistical significance \( (P < 0.05) \) in 91 neurons (points to the left of the solid vertical line). Star represents the neuron in A.

Having established that IT neurons responded selectively to the stimuli, we proceeded to analyze the impact of repetition suppression on the pattern of selectivity. The aim of the analysis was to determine which of two models provided a better fit to the data. According to the sharpening model (Desimone 1996; Wiggs and Martin 1998), repetition suppression is proportionally greater for stimuli that elicit a weak response from the neuron than for stimuli that elicit a strong response, with the consequence that selectivity is sharpened at the time of the second presentation (Fig. 9A). According to the scaling model (Avidan et al. 2002), repetition suppression reduces visual responses by a constant proportion regardless of stimulus efficacy, with the consequence that selectivity is unaffected (Fig. 9B).

To visualize directly the impact of repetition suppression on stimulus selectivity, we constructed, for each neuron, selectivity profiles representing response strength as a function of stimulus rank from the least effective to the most effective stimulus (see METHODS, Assessing the impact of suppression on stimulus selectivity). We did this for all first presentations and likewise for all second presentations. Then, averaging across neurons, we constructed population selectivity profiles for the first and second presentations. These are shown as black and red curves in Fig. 9C. They clearly conform more closely to the predictions of the scaling model (Fig. 9B) than to those of the sharpening model (Fig. 9A). In fact, when the black and red selectivity profiles were normalized to eliminate the overall difference in firing rate, they were not significantly different, as predicted by the scaling model \( (P = 1, \text{Kolmogorov–Smirnov test}) \).

Changes in neural selectivity have been most commonly assessed by fitting Gaussian functions to a set of physiological responses, and then comparing the best fit parameters across different conditions (McAdams and Maunsell 1999a,b). This approach is not feasible for IT neurons because IT tuning profiles are not generally well fit by Gaussian functions, nor are the dimensions of feature selectivity necessarily manipulable parametrically. For this reason, we compared the predictions of

![Fig. 8](image_url)

![Fig. 9](image_url)
the scaling versus the sharpening models by examining the effect of repetition suppression on absolute neural responses, rather than on tuning profiles based on relative stimulus rankings. The simplest prediction of the scaling model is that the magnitude of suppression will be proportional to the magnitude of the mean visual response. In contrast, the sharpening model makes the opposite prediction, that the relation between suppression and mean visual response will have a negative slope, or at most a zero slope. To determine which model best accounted for our data, we plotted repetition suppression (FR₁ − FR₃) against the mean strength of visual response (FR₁ + FR₃)/2 across all observed pairs of stimulus presentations (Fig. 10). The correlation between the two measures was indeed positive and highly significant (n = 5,765 stimulus pairs, P < 3 × 10⁻¹¹, R = 0.09). The magnitude of suppression, indicated by the slope of the regression line, was roughly 9% (upper and lower confidence intervals 6–11%) of the strength of the original response. The positive correlation between mean firing rate and the magnitude of suppression remained significant when stimuli repeated at lag 0, 1, or 2 were considered separately and was reduced to a nonsignificant trend for lag 4 (P = 0.14). Thus repetition suppression resulted in a uniform scaling down of visual responses, rather than a sharpening of selectivity.

Differences between responses to symmetric and asymmetric stimuli

Our use of symmetric and asymmetric stimuli was dictated by the need to elicit behavioral priming in monkeys rather than by a desire to compare responses evoked by the two stimulus types. Nevertheless, we note here an incidental observation of some interest. Symmetric stimuli tended to evoke stronger responses from IT neurons than did asymmetric stimuli (P = 0.001, rank-sum test). On average, responses to symmetric stimuli were 3.1% greater than responses to asymmetric stimuli (3.6 and 2.6% for m₁ and m₂, respectively). This effect was observed irrespective of whether the monkey responded correctly or incorrectly to the stimulus.

**DISCUSSION**

Is there a relation between repetition priming and repetition suppression?

We developed a monkey model of repetition priming in order to investigate what changes in neural activity accompany priming. Using this model, we demonstrated that repetition suppression at the level of single neurons in IT occurs under the same conditions that induce priming. However, the measures of behavioral priming and neuronal suppression were not significantly correlated across trials, as would have been expected if repetition suppression caused repetition priming. We cannot absolutely rule out the possibility that the firing rates and reaction times were subject to large independent sources of noise that masked an underlying weak correlation between suppression and priming. We would not have detected an underlying correlation so weak as to explain <0.07% of the variance in the measures. However, we question whether a correlation so weak would, if present, possess functional significance. This result is consistent with the findings of several recent studies that failed to detect any correlation across human subjects between priming of behavior and BOLD signal reduction in regions of ventral stream visual cortex (Maccotta and Buckner 2004; Sayres and Grill-Spector 2006; Wig et al. 2005). In contrast, a significant correlation has been observed in prefrontal cortex (Dobbins et al. 2004; Maccotta and Buckner 2004; Wig et al. 2005). This might reflect either the participation of prefrontal cortex in priming or its participation in semantic judgments affected by priming.

What is the behavioral significance of scaling?

In previous studies involving weeks and months of intensive discrimination training, neuronal stimulus selectivity in IT became sharper as a result of training (Baker et al. 2002; Freedman et al. 2005; Rainer and Miller 2000). How are we to explain the difference between the effects of prolonged training and the effects of a single exposure to a stimulus? Perhaps sharpening of selectivity requires slowly developing synaptic changes, whereas scaling of neural responses, either up or down, can be achieved by a rapidly acting process. The rapid process might mediate not only the effect of priming but also the effect of attention (McAdams and Maunsell 1999a). A rapid scaling-up of neural response strength would be expected to enhance the signal-to-noise ratio of the stimulus representation (McAdams and Maunsell 1999b). What benefit would attach to scaling-down the response strength is more difficult to understand. Rendering responses weaker can, under certain circumstances, enhance the synchronization of spikes across neurons (Gotts 2003). Repetition-induced increases in synchrony were reported in the insect olfactory system (Stopfer and Laurent 1999). Increased synchrony at the level of IT could result in enhanced post-synaptic efficacy and stronger firing in areas of frontal cortex receiving projections from IT. Consistent with this hypothesis, one repetition priming study demonstrated that BOLD signal decreases in extrastriate visual cortex are accompanied by BOLD signal increases in frontal cortex (van Turenout et al. 2000).
What is the relation of repetition suppression to MR adaptation?

The prospect of using BOLD signal adaptation to circumvent the limited spatial resolution of MRI has attracted a great deal of interest (Grill-Spector and Malach 2001; Henson et al. 2003). This approach assumes that repetition-induced reductions in BOLD signal are the result of firing rate reductions in visual cortex, as recently demonstrated in a study combining the approaches of physiology and functional imaging (Sawamura et al. 2006). Interpreting reductions in BOLD signal in terms of the response properties of single units depends on the assumption that the cells with highest firing rates are also the ones that undergo maximal suppression (Avidan et al. 2002). Our finding that visual responses are reduced proportionally supports the validity of this assumption.

Impact of symmetry on behavioral and neuronal responses

Although the main focus of this study was not on the effect of image symmetry in itself, we did observe consistent effects of symmetry that are worth noting. Both monkeys were faster to respond to symmetric stimuli and exhibited a bias to classify stimuli as symmetric. Moreover, priming was both greater in magnitude and more strongly dependent on lag for repeated symmetric stimuli than for repeated asymmetric stimuli. Stronger priming effects for symmetric stimuli have also been previously observed in human subjects (Kersten-Tucker 1991). Because symmetry is often associated with living things (such as faces or body parts), enhanced priming for symmetrical objects may reflect a mechanism whereby perceptual learning is facilitated for stimuli with a high degree of behavioral relevance. In addition to the impact of symmetry on behavior, the average firing rate elicited by symmetric stimuli was greater than that elicited by asymmetric stimuli even on the first presentation. This finding, which parallels functional imaging results obtained in humans (Sasaki et al. 2005; Tyler et al. 2005), may be related to the fact that IT neurons respond similarly to the two members of any given lateral mirror image pair (Rollenhagen and Olson 2000). Enhanced processing of symmetric stimuli might serve a useful function in a natural environment because symmetry detection has the potential to facilitate the structural interpretation of three-dimensional objects (Vetter et al. 1994) and exerts a strong influence on figure-ground organization (Peterson and Gibson 1994).


