ing 10 mM bic was placed within the forelimb area, 0.5 to 2.0 mm lateral to the stimulating electrode at a depth of 1.8 mm (layer V). In some experiments a glass multibarrelled micropipette was used for the separate application of bic, ACh, or glutamate. The bic was ejected continuously (50 to 150 nA) at this forelimb site for 7 to 80 min, while movements and EMG evoked by stimulating at the vib test site were recorded at 5- to 15-min intervals.
10. In 12 of the 15 rats, multiple bic applications were made. Second and third applications were made only after the shift was no longer observed. Each application produced the same reorganization.
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12. We applied 0.3 μM (−)[^{3}H]bicuculline methyl chloride through our usual glass pipettes with +5 nA for 2 hours.
13. Sections of unfixed tissue were exposed to autoradiographic film and then developed. Radioactive labeling was measured by a computer-based densitometer. The decrease in labeling density from the center of the application site fit a Gaussian distribution reaching two standard deviations below the peak value 600 μm from the ejection center.
14. Threshold ± SEM for vibrissa movement were 28.1 ± 2.6 μA, n = 43, before and 33.3 ± 2.2 μA, n = 65 (not significant, $P > 0.05$, t test), after bic application.
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D1 Dopamine Receptors in Prefrontal Cortex: Involvement in Working Memory

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The prefrontal cortex is involved in the cognitive process of working memory. Local injections of SCH23390 and SCH39166, selective antagonists of the D1 dopamine receptor, into the prefrontal cortex of rhesus monkeys induced errors and increased latency in performance on an oculomotor task that required memory-guided saccades. The deficit was dose-dependent and sensitive to the duration of the delay period. These D1 antagonists had no effect on performance in a control task requiring visually guided saccades, indicating that sensory and motor functions were unaltered. Thus, D1 dopamine receptors play a selective role in the mnemonic, predictive functions of the primate prefrontal cortex.

IN NONHUMAN PRIMATES, THE PREfrontal cortex (PFC), in particular its dorsolateral region, is critical for the cognitive process of working memory (*1*). Several lines of research suggest that dopamine (DA) may influence this process. The concentration of DA in the PFC is among the highest in all cortical areas in monkeys (*2*), and both DA-containing fibers and DA receptors are prominent in the primate PFC (*3, 4*). Local depletion of DA in the PFC of monkeys induces impairment in tasks that require delayed response (*5*), and neuronal activity related to delayed response performance is augmented by iontophoretically applied DA (*6*). These findings suggest that DA receptors may be involved in the mnemonic processes of the PFC.

There are two types of DA receptors in the central nervous system, D1 and D2 receptors (*7*); the PFC of primates, including humans, contains a high level of D_1 receptors (*4, 8, 9*) and a relatively low or negligible level of D2 receptors (*4, 9, 10*). These findings imply that the D1 receptors are likely to be involved in the mnemonic process mediated by the PFC, but direct evidence is lacking. This hypothesis can now be directly tested by the use of potent D1 antagonists, SCH23390 (*11*) and SCH39166 (*12*), in combination with sensitive behavioral paradigms for assessing working memory.

We used an oculomotor delayed-response (ODR) task in which animals were trained to fixate a central spot on a cathode-ray tube while a visual cue was presented briefly in one of several (6 to 22) locations in the visual field. The cue then disappeared, and after a 1.5- to 6-s delay the animal was required to make a memory-guided saccade to where the target had been presented seconds before. Therefore, to achieve criterion performance on the ODR task the animal had to remember visuospatial data in order to make the correct response at the end of the delay. To distinguish a deficit in mnemonic function from deficits in eye movements or sensory perception, we used a control procedure in which the target remained on during the delay period and the subject made a sensory-guided saccade to the target. Neuronal activity in the dorsolateral PFC of monkeys is involved in the ODR task (*13*), and lesions of the dorsolateral PFC induce deficits in the memory-guided saccades required by the task (*14*). However, to examine the role of neurotransmitters or receptors on specified cortical functions, a method of targeting specific brain regions was required. Therefore, we combined the ODR paradigm with the intracerebral injection of SCH23390 and SCH39166 and now report that the activation of D1 receptors plays a critical role in the mnemonic process mediated by the primate PFC.

Three rhesus monkeys (T, J, and N) were trained in the ODR and control tasks (*15*).

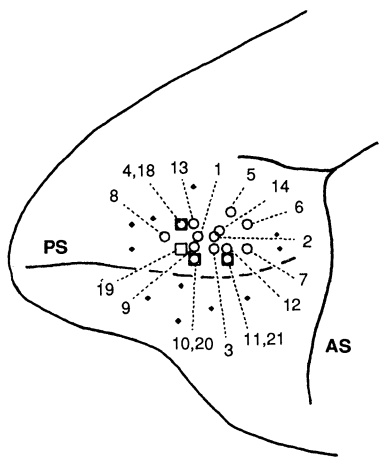

Fig. 1. Injection sites of SCH23390 and SCH39166 in two of the monkeys illustrated on a lateral view of the left PFC. The sites were reconstructed in reference to cortical sulci. Data on injection sites in one monkey, N, that has not yet been killed are not included. However, the effective sites in this monkey are distributed in the same region on the basis of the position of the cylinder and the coordinates of the micromanipulator used for injections; ○, effective sites of SCH23390 on ODR performance; □, effective sites of SCH39166 on ODR performance; ■, ineffective sites with either drug. The number of the injection site in Fig. 1 corresponds to the injection site shown in Table 1. PS, principal sulcus; AS, arcuate sulcus. Injections were placed 2 to 4 mm deep to the dural surface; no sites were located within the banks of the principal sulcus.

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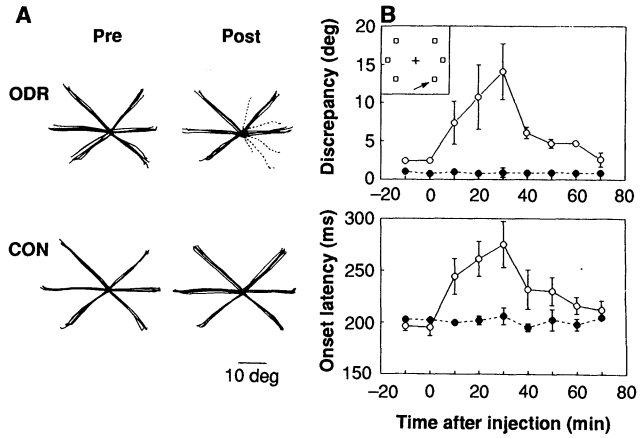

Fig. 2. The effect of the D1 antagonist SCH23390 on the oculomotor delayed-response (ODR) and control (CON) tasks. SCH23390 (50 μg) was injected into the left PFC (site 1 in Table 1). The cue was presented at a total of six locations that were separated by 45° or 90° in direction, and their eccentricity was 20°. (**A**) Superimposition of two-dimensional trajectories of the saccade to each target in the ODR and CON tasks. Pre, before injection; Post, 10 to 20 min after injection. After the injection, the variability and discrepancy between target and memory-guided saccades made to it increased for a specific target located at the lower right (dashed trajectories). (**B**) Time course of changes in the accuracy (top) and the onset latency (bottom) of the saccade for the ODR task and CON tasks. The data are for trials at the location indicated by the arrow (inset). Mean ± SEM; n = 4 to 7 trials per time point.

Table 1. Onset latency of the response for each injection site that was associated with a deficit on the ODR task after injection of SCH23390 or SCH39166. The data given for each injection are for the target affected most strongly and for the postdrug block or blocks for which the score was significantly different from the predrug score. Data for 5- to 6-s delays are shown. Scores before and after injection were compared by analysis of variance, and multiple comparisons were made with the Newman-Keuls procedure (*17*). The number of trials is shown in parentheses. Injections labeled a, b, c, and d were placed with the same coordinates as those used for injections 4, 10, 11, and 16, respectively. *, $P < 0.05$; **, $P < 0.01$.

Injection site (Fig. 1)	Dose (μg)	Onset latency (ms) (mean ± SD)	
		Predrug	Postdrug
		SCH23390	
Monkey T			
1	50	195.9 ± 11.4(8)	269.9 ± 47.4**(11)
2	30	197.6 ± 28.2(10)	282.0 ± 48.1**(12)
3	60	208.8 ± 15.0(10)	256.2 ± 21.2**(10)
3	30	208.5 ± 10.6(8)	240.6 ± 13.7**(11)
3	15	203.5 ± 4.8(8)	224.6 ± 11.8**(9)
4	30	180.8 ± 8.0(4)	262.2 ± 24.7**(5)
5	20	202.6 ± 16.2(14)	248.9 ± 15.7**(15)
6	10	212.4 ± 14.1(10)	234.4 ± 10.0**(10)
7	30	198.4 ± 14.1(9)	242.4 ± 12.9**(14)
8	10	211.6 ± 21.8(10)	235.9 ± 23.2*(8)
9	60	198.6 ± 19.2(11)	259.4 ± 18.3**(8)
9	30	193.8 ± 11.6(10)	240.4 ± 19.3**(10)
9	15	206.1 ± 7.9(9)	230.9 ± 28.0*(8)
10	80	203.6 ± 15.6(10)	260.7 ± 25.0**(17)
11	30	196.3 ± 15.2(7)	249.0 ± 25.2**(7)
	15	202.2 ± 16.9(10)	227.5 ± 20.0**(13)
Monkey J			
12	60	209.7 ± 17.7(11)	302.0 ± 52.5**(13)
12	30	206.6 ± 19.8(7)	257.2 ± 37.0**(10)
13	30	195.0 ± 8.5(7)	244.5 ± 31.6**(10)
14	30	194.1 ± 22.8(10)	258.3 ± 19.5**(11)
Monkey N			
15	30	238.6 ± 10.6(12)	292.7 ± 32.2**(15)
16	30	239.4 ± 24.4(12)	313.6 ± 36.8**(15)
17	30	228.0 ± 10.8(9)	249.5 ± 24.2**(12)
		SCH39166	
Monkey T			
18[a]	5	199.1 ± 8.2(14)	221.3 ± 8.0**(9)
19	5	207.8 ± 17.6(10)	272.0 ± 43.0**(10)
20[b]	5	199.7 ± 10.3(10)	227.0 ± 10.7**(11)
21[c]	1	205.2 ± 13.4(10)	231.0 ± 18.6**(11)
Monkey N			
22[d]	10	231.8 ± 10.3(14)	277.7 ± 19.7**(10)
23	10	233.7 ± 23.3(9)	304.5 ± 37.5**(10)
23	5	247.3 ± 13.7(14)	274.0 ± 28.9**(12)
24	5	243.0 ± 7.2(8)	280.6 ± 17.2**(11)

While the monkey performed these tasks, 3 μl of SCH23390, SCH23388, ketanserin, or SCH39166 was injected locally into the dorsolateral PFC with a 10-μl Hamilton syringe (*16*). The injection of SCH23390 (10 to 80 μg) induced reversible deficits in ODR performance in all three monkeys at a total of 17 sites in the principal sulcal region of the dorsolateral PFC (Table 1 and Fig. 1) (*17*). The impairment was quantified as an increase in error, measured by the discrepancy of the position of the to-be-remembered target and the saccade performed by the monkey at the end of each trial (Fig. 2) and by an increase in latency to respond (*18*). The trajectories of the saccades were abnormal (Fig. 2A), although their velocity did not change (Fig. 3). These deficits usually occurred within 10 min after the injection (in most cases, within 1 to 3 min after the injection), reached a peak at 20 to 40 min, and recovered at 60 to 90 min after the injection (Fig. 2B). The change in performance was restricted to a few specific target locations, which varied with the injection site and were usually contralateral to it. By contrast to the performance of the ODR task, we observed no significant changes in the performance of the control task after the injection of SCH23390 during the same experimental session in which performance on the ODR task was impaired (Fig. 2). The accuracy and onset latency of sensory-guid-

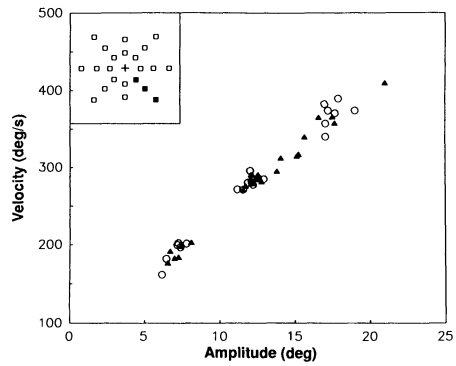

Fig. 3. Relations between amplitude and velocity of the memory-guided saccades in the ODR task before and after injection of SCH23390. SCH23390 (30 μg) was injected into the left PFC while the monkey performed the ODR task; discrepancy and onset latency of memory-guided saccades to a target location were significantly increased after the injection (site 13 in Table 1). The cue was presented at a total of 22 locations that were separated by 45° or 90° in radial direction, with eccentricities of 7°, 13°, and 20°. The velocity was plotted as a function of the amplitude of the saccade for each trial. The data are for trials at the three locations with the same direction indicated by closed squares (inset). The memory-guided saccades in this direction were those most strongly affected by the injection; ○, before injection; ▲, 10 to 40 min after injection. The relation between amplitude and velocity was similar before and after the SCH23390 injection.

ed saccades in the control task were the same before and after the injection for all target locations. Furthermore, the deficit in the ODR task induced by SCH23390 was sensitive to the duration of the delay period, and longer delays were associated with larger errors (Fig. 4A). Moreover, the degree of the deficit on the ODR task depended on the dose of injected drug; higher doses of SCH23390 produced larger increases in errors of the memory-guided saccades (Fig. 4B).

Although SCH23390 is a selective antagonist of D1 receptors, it also appears to bind to serotonin (5-HT2) receptors (*19*) and it might be argued that the effect of SCH23390 was due to effects at 5-HT2 receptors. We therefore examined the effects of a selective antagonist of the 5-HT2 receptor, ketanserin, and an inactive analog of SCH23390, SCH23388, on ODR performance in two monkeys. Local injection of ketanserin (100 μg, five sites) or SCH23388 (100 μg, four sites) near the same sites at which the injection of SCH23390 induced deficits in the ODR task did not produce any clear changes in performance on either the ODR or the control task. In addition, the injection of 0.9% saline (5 to 10 μl) into sites that were associated with deficits in ODR performance after injection of SCH23390 failed to induce any significant changes in performance on either the ODR or the control task. Thus, the effect of SCH23390 does not appear to be a consequence of its nonspecific effects or any effect on 5-HT2 receptors.

We further tested the pharmacological specificity of the effect by injecting SCH39166 into two of the three monkeys. Unlike SCH23390, this D1 antagonist has a negligible affinity for 5HT-2 receptors (*12*). When we injected SCH39166 (1 to 10 μg) into seven sites in the principal sulcal region (Table 1 and Fig. 1), the injection induced deficits in performance in the ODR task but not in the control task. The impairment was similar to that induced by SCH23390: after the injection of SCH39166, the error and onset latency of memory-guided saccades were increased for a few specific target locations, and the deficits were sensitive to the duration of delay (Fig. 4C). Furthermore, we injected SCH39166 into four sites that were associated with deficits after SCH23390 injection and obtained the same results as those independently obtained with SCH23390. In contrast, injection of 0.9% saline (5 to 7 μl) into these same sites and others in which SCH39166 caused deficits did not affect either the ODR or the control task performance. Moreover, we also examined the effects of injecting the selective D2 receptor antagonist raclopride (*20*) (100 μg) into seven sites associated with deficits produced by SCH39166 or SCH23390; raclopride did not affect the ODR or the control task performance (Fig. 4D).

Our findings indicate that the activation of D1 receptors is critical for the memory processes mediated by the primate PFC. They do not, however, rule out possible roles for receptors other than D1 or interactions between D1 receptors and other receptors in the mnemonic function of the PFC. Also, our results do not indicate the specific type of neuron that is affected by D1 receptor antagonism (*21*). However, cells or processes with D1 receptors are heavily concentrated in the deep layers of the PFC, where neurons that project to the thalamus, caudate nucleus, and superior colliculus are located (*4, 22*). Disruption of neuronal activity of one or more of these cell classes may be responsible for the mnemonic deficit.

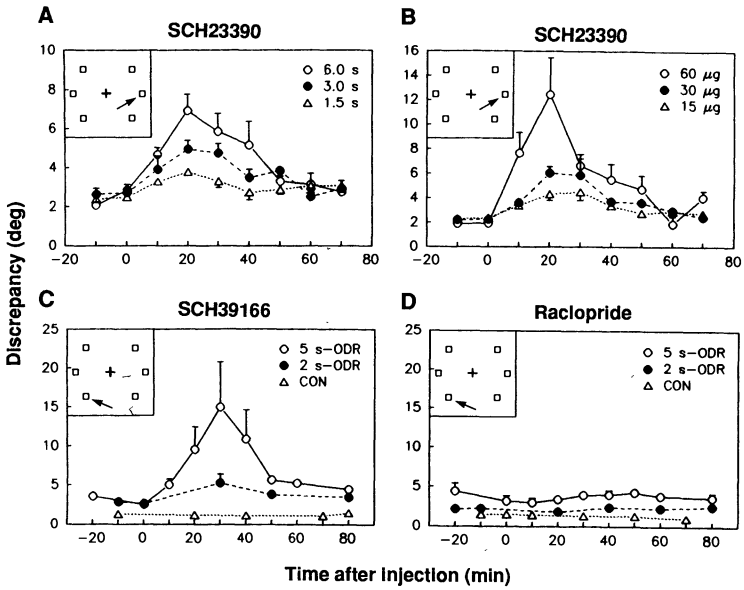

Fig. 4. (**A**) The effect of SCH23390 on the ODR at different delay lengths. SCH23390 (30 μg) was injected into the left PFC while the monkey performed the ODR task with different durations of delay (site 2 in Table 1). The data are for trials at the location indicated by the arrow (inset), since the memory-guided saccade to this target location was the most strongly affected by the injection. Mean ± SEM; n = 4 to 8 trials per time point. (**B**) The effect of SCH23390 on the ODR at different doses. SCH23390 was injected into the same sites in the left PFC in different daily sessions (site 3 in Table 1). The injection sites were near the site of the case shown in (A). The data are for trials at the location indicated by the arrow (inset); the memory-guided saccade to this target location was the most strongly affected by the injection. Mean ± SEM; n = 4 to 8 trials per time point. (**C**) The effect of the D1 antagonist SCH39166 on the ODR and CON tasks. SCH39166 (5 μg) was injected into the right PFC while the monkey performed the ODR task with 2- or 5-s delays and the CON task with a 5-s delay (site 24 in Table 1). The data are for trials at the lower-left target location (inset); the memory-guided saccade to this target location was the most severely impaired. Mean ± SEM; n = 4 to 9 trials per time point. (**D**) The effect of the D2 antagonist raclopride on the ODR and CON task. Raclopride (100 μg) was injected into the same site as that of the case shown in (C). The data are for trials at the lower-left target location (inset). Mean ± SEM; n = 4 to 6 trials per time point. No significant changes in discrepancies or onset latencies were observed after the injection for any task. However, comparisons of discrepancies and latencies revealed a significant difference between the ODR and CON tasks for almost all time periods. This difference existed before drug injection as well as after and reflects the greater difficulty of the ODR compared to the CON task.

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15. In the ODR task, the monkeys fixated a central spot on a cathode-ray tube, and a visual cue came on for 0.3 s, followed by a delay period. The cue was presented randomly at one of several peripheral locations (n = 6 to 22, usually six locations), which were separated by 45° or 90° and whose eccentricities were 7° to 20° (usually 20°). After the delay period (1.5 to 6 s, but usually 5 s), the fixation spot was then extinguished, which instructed the monkeys to make a memory-guided saccade to the location that had been cued before the delay period. The correct response was rewarded by a drop of juice 0.2 s after the response. Trials were separated by an intertrial interval of 3.5 s. The control task was exactly the same as the ODR task except that the target remained on during the "delay" period, thus providing sensory guidance for saccade in the response period.
16. A micromanipulator on a cylinder mounting on the skull was used to insert the syringe into the cortex and to control it precise localization and relocalization in subsequent sessions. The spread of 3 μl of injected solution into the cerebral tissue is about 3 mm in diameter [R. D. Myers, *Psychol. Behav.* **1**, 171 (1966)].
17. The experimental sessions consisted of blocks of trials with a time length of 5 or 10 min. In each block, the monkey performed the ODR or control (CON) task, and the blocks associated with the CON task were intermixed with the blocks associated with the ODR task in most cases. The monkeys performed two to four CON and two to four ODR blocks before and at least four blocks of each task after the injection. The following behavioral parameters were examined: discrepancy between the target location and the end point of the saccade during the response period; the onset latency of the response after the onset of the go signal; trajectories of saccades; and amplitude and velocity of saccades. Velocity was measured from the amplitude and duration of the saccades. The predrug blocks were combined into one score for each task, and this predrug block was compared with every postdrug block by a one-way analysis of variance followed by the Newman-Keuls procedure for multiple comparisons. The data in Table 1 are for injections in which at least one ODR postdrug block differed significantly from the predrug block.
18. Bradykinesia (slowing of responses) is characteristic of Parkinson's disease, a disease in which DA loss in the neostriatum is the cardinal pathognomonic finding. Our result of an increased latency to respond in the ODR task indicates that slowing of at least some memory-guided responses might be attributable to cortical DA dysfunction.
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Technical Comment

Form, Motion, and Binocular Rivalry

If one looks at two grossly dissimilar images—such as orthogonal gratings—through a stereo viewer, only one eye's field of vision is seen at a time. This phenomenon is called binocular rivalry (*1*). N. K. Logothetis and J. D. Schall (*2*) performed an ingenious experiment to explore the neural basis of binocular rivalry. A monkey looked at a downward-moving "conveyor belt" of horizontal stripes through one eye and at upward-moving horizontal stripes through the other eye. While the monkey "reported" rivalry by pressing the appropriate key, the electrical activity of direction-selective neurons in the middle temporal (MT) area in the superior temporal sulcus was monitored. One might suppose that neural responses corresponding to the suppressed image would be silenced while neurons corresponding to the other image would be active. Although 10% of the cells showed the expected suppression, in most neurons no simple suppression was observed—certainly nothing similar to the complete occlusion that occurs perceptually. Indeed, sometimes there was an enhancement of neural responses to the suppressed image.

One possible explanation for this neuronal response would be that rivalry is a "network" property that cannot be studied in single cells, but this statement is not useful, even if it were true. A second explanation would be that rivalry is not a complete occlusion of one eye's input at an early stage, rather, it occurs at multiple sites and can selectively involve some neural channels while sparing others (*3, 4*). For example, stereopsis can be experienced in the presence of "form rivalry" (*4, 5*) even though only one image is perceived at a time. In the case of downward-moving stripes for the left eye and upward for the right eye, it is true that only one image is seen at a time, but is this really "motion rivalry" caused by inhibition between motion channels within the MT area itself? Even though the stripes are horizontal for both eyes, at any given instant the stripes are likely to be vertically misaligned. This would tend to generate form rivalry by stimulating noncorresponding retinal points (*5*). Perhaps it is this form rivalry that gates neural motion signals (*6*)—there may be no motion rivalry per se occurring within the MT.

I did an experiment recently (*6*) to study these effects. After I viewed the "conveyor belt" display for several minutes, two motion after-effects were generated. On looking at the world with the right eye I perceived downward movement, and on looking with the left eye I perceived upward motion. What happened when I opened both eyes depended on what I looked at. A stationary grating (or any pattern) usually looked stationary—the brain simpy averaged the motion after-effects from both eyes. But on presenting diagonal, orthogonal, stationary gratings to both eyes—so that I experienced form rivalry—I experienced motion rivalry as well! The left eye perceived upward motion and the right eye perceived downward motion.

I conclude that the motion signals from the two eyes were averaged only when I looked at the same form with both eyes. If there was form rivalry, on the other hand, the motion signals inhibit each other. Apparently, what happened in the form channels influenced what happened in the motion channels. Since the MT area is concerned with motion rather than form, these results may explain why Logothetis *et al.* did not observe a simple suppression of one eye's motion signals. Indeed, our results suggest that the best place to look for rivalry would be in the "form area" DL, V4, or IT rather than in MT. The presence of rivalry in these areas might modulate the activity of cells in the MT area in complex ways or interact with cells in higher motion areas such as the medial superior temporal area rather than in the MT itself.

A third explanation would be in terms of the theory of F. H. C. Crick and C. Koch (*7*), according to which the basis of conscious visual awareness is the synchronization of 40HTZ oscillations (*8*). If one is aware of an object, the firing of all neurons that are simultaneously activated by that object alone becomes synchronized. This synchronization does not include other neurons that are activated by objects that one is not attending to. A reanalysis of the data of Logothetis and Schall to look for synchronized oscillations (rather than suppression) may therefore be worthwhile. Such data could provide a test for the hypothesis that synchronized oscillations are actually involved in consciousness and not merely in binding features together for object segmentation.

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