Functional Independence of Layer IV Barrels in Rodent Somatosensory Cortex

Daniel Goldreich, Harold T. Kyriazi, and Daniel J. Simons
Department of Neurobiology, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania 15261

Goldreich, Daniel, Harold T. Kyriazi, and Daniel J. Simons. Functional independence of layer IV barrels in rodent somatosensory cortex. J. Neurophysiol. 82: 1311-1316, 1999. Layer IV of rodent primary somatosensory cortex is characterized by an array of whisker-related groups of neurons, known as “barrels.” Neurons within each barrel respond best to a particular whisker on the contralateral face, and, on deflection of adjacent whiskers, display relatively weak excitation followed by strong inhibition. A prominent hypothesis for the processing of vibrissal information within layer IV is that the multi-whisker receptive fields of barrel neurons reflect interconnections among neighboring barrels. An alternative view is that the receptive field properties of barrel neurons are derived from operations performed on multiwhisker, thalamic inputs by local circuitry within each barrel, independently of neighboring barrels. Here we report that adjacent whisker-evoked excitation and inhibition within a barrel are unaffected by ablation of the corresponding adjacent barrel. In supragranular neurons, on the other hand, excitatory responses to the ablated barrel’s associated whisker are substantially reduced. We conclude that the layer IV barrels function as an array of independent parallel processors, each of which individually transforms thalamic afferent input for subsequent processing by horizontally interconnected circuits in other layers.

I N T R O D U C T I O N

Sensory areas of the cerebral cortex are characterized by collections of interconnected neurons having similar receptive fields. The extent to which these local circuits interact remains poorly defined, even at the earliest stages of cortical processing. For example, some models of simple-cell orientation selectivity in cat visual cortex assume antagonistic interactions between separate minicolumns serving the same retinal location but activated by stimuli having orthogonal orientations (Crok et al. 1997; Sillito et al. 1980). Other models base orientation selectivity on convergent thalamic input solely (Ferster 1987; Hubel and Wiesel 1962; Reid and Alonso 1995) or in conjunction with locally mediated, iso-orientation excitation and/or inhibition (Ferster 1988; Troyer et al. 1998). Layer IV of rodent somatosensory cortex contains anatomically identifiable collections of neurons, called barrels, that represent distinctly different peripheral locations, i.e., facial whiskers (Woolsey and Van der Loos 1970). Although differing from orientation minicolumns in this and other respects, the degree to which barrels interact with each other remains similarly controversial. Some investigators have suggested a prominent role for horizontal connections in creating receptive fields encompassing multiple neighboring vibrissae (Armstrong-James et al. 1991; Fox 1994), whereas others have proposed that interactions among neighboring whiskers reflect local, intrabarrel processing of multiwhisker thalamic inputs (Simons and Carvell 1989). In both visual and somatosensory cortices, horizontal connections are thought to contribute substantially to receptive field properties in nongranular layers.

To what extent do neighboring local circuits function independently of one another? Because of its anatomic organization, the somatosensory cortex of rodents is well suited for addressing this issue. A barrel consists of several thousand synaptically interconnected neurons, each of which receives the bulk of its afferent input from neurons in an homologous “barreloid” within the ventral postero medial (VPM) thalamic nucleus (Land and Simons 1985). Neurons within the barrel and throughout its associated column are maximally excited by a principal whisker (PW) but, depending on laminar location, they respond also to neighboring whiskers to varying degrees (Armstrong-James and Fox 1987; Simons 1978). Deflection of two or more whiskers in rapid sequence reveals the presence of surround inhibitory effects that are considerably stronger in cortical than thalamic neurons (Brumberg et al. 1996; Simons and Carvell 1989).

Previously, we proposed that inhibitory interactions among neighboring whiskers in the layer IV barrel reflect direct engagement of local circuitry by thalamic inputs (Simons and Carvell 1989). We hypothesized that inputs to a barrel from nonprincipal whiskers arise directly from thalamic afferents, because neurons within thalamic barreloids, although driven most strongly by the PW, also respond robustly to neighboring whiskers (Nicolelis et al. 1993; Simons and Carvell 1989). The absence of a direct barrel-to-barrel pathway (Akhtar and Land 1989; Bernardo et al. 1990b; Hoeflinger et al. 1995) further supports the idea that barrels function independently of each other. Accordingly, destruction of a cortical barrel should have little, if any, effect on the influence of its corresponding whisker in neighboring barrels (see Fig. 1). Here we demonstrate that adjacent whisker-evoked excitation and inhibition within a barrel are virtually unaffected by ablation of the adjacent barrel.

M E T H O D S

This study was conducted using adult female Sprague-Dawley rats (202–315 g; Hilltop). Surgery and anesthetic procedures were similar to those previously described (Brumberg et al. 1996). Briefly, animals were anesthetized with halothane (~1.5% in a 50–50 mixture of Nz and O2) and tracheotomized. Venous and arterial catheters were inserted for later drug delivery and blood pressure monitoring. The
animal was placed in a stereotaxic frame, and a craniectomy was made in the skull overlying part of the whisker representation area of the right primary somatosensory cortex. A small steel post was attached to the skull with dental acrylic to hold the animal’s head during the experiment. An acrylic dam was placed around the craniectomy and was kept filled with saline.

The underlying cortex was roughly mapped by multiunit recordings made through the dura using tungsten microelectrodes (Frederick Haer, Brunswick, ME; medium point, 3–5 MΩ at 1 kHz), combined with manual stimulation of the whiskers on the contralateral face. The underlying cortex was roughly mapped by multiunit recordings made through the dura using tungsten microelectrodes (Frederick Haer, medium tip, 0.010-in. shank diameter, 10–12 MΩ at 1 kHz) was inserted normal to the pial surface overlying the estimated barrel center. To minimize dimpling of the brain surface and to achieve reproducible penetration depths, the electrode was advanced initially to a depth of 1,500 μm and then withdrawn to 1,050 μm. Because preliminary experiments indicated that electrolytic lesions made with these electrodes produced a conical abscess that spread superficially, DC (30 μA for 30 s, electrode negative) was passed initially at a depth of 1,050 μm followed by a second application at 700 μm, which we routinely find to correspond to the layer III/IV boundary. Immediately thereafter spontaneous unit activity could be recorded deep to the lesion but not at middle or superficial cortical depths.

Subsequently, we examined the receptive field properties of units in a barrel/column (test barrel) immediately adjacent to the ablated barrel (see Fig. 1). We intentionally selected units that gave vigorous excitatory responses to the PW, because we assumed at the outset that such units were unlikely to be in close proximity to damaged tissue (but see RESULTS). Also, we assumed that suppression of such responses by adjacent whisker (AW) stimulation would be a robust indicator of intact inhibitory mechanisms within the test barrel. Unit recordings were obtained using double-barreled glass micropipettes, one barrel of which contained 3 M NaCl and the other 10% wt/vol horseradish peroxidase (HRP) for marking selected recording sites (Simons and Land 1987).

Electromechanical stimulators were used to deflect the test barrel’s PW and two AWS (see Simons and Carvell 1989), one corresponding to the lesion-ablated barrel (AWL) and the other to an intact (normal) barrel (AWN) on another side of the test barrel (see Figs. 2 and 4). The excitatory influence of each AW was quantified as the average number of spikes/stimulus taken over eight angles of deflection. Each deflection angle was repeated 10–20 times. Spike counts were measured during the 5–25 ms following stimulus onset. To quantify inhibitory AW effects, the AW was deflected in each of eight directions followed 20 ms later by PW deflection at its maximally effective angle. A video camera attached via a beam-splitter to a surgical microscope was used to photograph the brain surface, using green light illumination to enhance blood vessel contrast. A detailed map of the targeted region of the barrelfield was then made using fine-tipped, glass/carbon fiber microelectrodes (see Kyriazi et al. 1996), with special attention being paid to the barrel chosen for ablation. To ablate a single barrel, an unused, high-impedance tungsten microelectrode (Frederick Haer: medium tip, 0.010-in. shank diameter, 10–12 MΩ at 1 kHz) was inserted normal to the pial surface overlying the estimated barrel center. To minimize dimpling of the brain surface and to achieve reproducible penetration depths, the electrode was advanced initially to a depth of 1,500 μm and then withdrawn to 1,050 μm. Because preliminary experiments indicated that electrolytic lesions made with these electrodes produced a conical abscess that spread superficially, DC (30 μA for 30 s, electrode negative) was passed initially at a depth of 1,050 μm followed by a second application at 700 μm, which we routinely find to correspond to the layer III/IV boundary. Immediately thereafter spontaneous unit activity could be recorded deep to the lesion but not at middle or superficial cortical depths.

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condition-test ratio (CTR) was calculated as the ratio of the average response to the PW when deflected after the AW to the response to PW deflection alone. Data from the responses evoked by the two AWs were compared using two-tailed paired-sample t-tests. For all trials in which at least one spike occurred during the stimulus onset window, the time of the first spike was measured at 0.1-ms resolution, and the mean and modal latencies across all trials and deflection angles were determined. For modal latencies, spikes were placed in 0.5 ms bins, and the bin with the greatest number of spikes was taken as the mode. No modal latencies were returned for units in which no bin contained more than one spike. All data are expressed as means ± SD unless indicated otherwise.

In two control experiments, the nerves innervating a whisker follicle were reversibly inactivated by infusion of 5 μl of 4% lidocaine (Xylocaine, Astra USA). Under halothane anesthesia, a 30-gauge needle was inserted 2–3 mm into the hair follicle on the caudal side of the whisker (where the afferent fibers enter the capsule). PE-10 tubing connected the needle to a Hamilton syringe, and the entire assembly was positioned such that the needle was suspended in the approximate plane of the whisker. This minimized mechanical effects of the needle's presence on the mystacial pad and permitted attachment of a stimulator to the whisker.

At the conclusion of each experiment, animals were administered a lethal dose of pentobarbital sodium (Nembutal, Abbott Laboratories) and perfused transcardially with phosphate-buffered saline followed by a solution of 2% paraformaldehyde and 1.5% glutaraldehyde. Brains were sectioned on a freezing microtome in the tangential plane, by a solution of 2% paraformaldehyde and 1.5% glutaraldehyde. Brains were sectioned on a freezing microtome in the tangential plane, and alternate sections were stained for HRP or cytochrome oxidase.

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FUNCTIONAL INDEPENDENCE OF WHISKER BARRELS

RESULTS

Data are reported from 12 experiments in which we estimate from serial tangential sections that, on average, 90% of a barrel was destroyed. Figure 2 shows photomicrographs of barrel fields from four experiments that illustrate the range of destruction as well as the locations of the test barrels. The distribution of lesion sizes, which ranged from 70–100% destruction, for all experiments is plotted in Fig. 3. In the vertical dimension, damage extended downward, having a blunt conical shape whose apex extended well into layer V. From upper layer V through layers IV and lower layer III, the lesion was shaped cylindrically. More superficially, the lesion also tapered conically; the largest lesions extended almost to the pial surface. Lesions rarely extended into neighboring barrels but typically involved the septum between the lesion and the test barrel.

Dendrites of barrel neurons in rats have an average radius of 100 μm, and neurons near the barrel side can have dendrites that extend into the septum and even into the neighboring barrel (Simons and Woolsey 1984). We therefore classified the recorded units into two groups: those in the cytochrome oxidase-rich barrel center at least 100 μm distant from the side of the barrel nearest the lesion (hereafter denoted as “Barrel”) and those within this 100 μm zone (“Near-lesion”). The latter included some cells located in the barrel side and possibly in the immediately adjacent septum. The relative proportions of units studied in experiments involving different lesion sizes were approximately equal (see Fig. 3). Because all lesions were extensive and because the number of units studied in each experiment was relatively small, we pooled data across experiments. All but 4 of 30 supragranular units were located above the barrel center; those 4 Near-lesion units were not included in the analyses.

Peristimulus-time histograms of recordings taken from the E2 barrel of an E1 barrel-ablated animal are shown in Fig. 4. The onset and offset of PW (E2) deflection elicit prominent excitatory responses. AW N (E3) evokes a clear, but weaker, excitatory response and a pronounced suppression of the response evoked by subsequent PW deflection. Most notably, the AW L (E1) also evokes virtually identical excitatory and inhibitory effects, despite the near-total ablation of the E1 barrel. On average, a Barrel unit’s AW-evoked excitatory response was ~20% that of its PW. Pooled Barrel results demonstrate that neither AW L-evoked excitation nor AW L-evoked inhibition were diminished by destruction of the AW’s associated barrel (Fig. 5). Furthermore, there were no significant differences between the AW L and AW N latencies, either mean (15.4 ± 1.9 ms, 15.9 ± 1.4 ms, mean ± SD, n = 27), or modal (14.2 ± 3.6 ms, 15.4 ± 3.3 ms, n = 21). The PW latencies (mean:12.9 ± 1.7 ms, n = 43, modal: 11.6 ± 2.2 ms, n = 35) were significantly shorter than those of either AW (all P values <0.001).

Near-lesion units displayed statistically significantly less AW L-evoked excitation and inhibition compared with those evoked by AW N. In addition, excitatory AW L responses were 42% smaller than those at locations more distant from the lesion, and these effects were greater with larger lesions (R2 = 0.47, P < 0.001). PW-evoked excitatory responses in Near-lesion units also were slightly (~17%) but not significantly smaller than those of Barrel units. Interestingly, AW N-evoked inhibition was stronger in Near-lesion than in Barrel units, and this, too, was correlated with lesion size (R2 = 47, P = 0.007).

We also recorded from neurons in layers II/III superficial to the center of the test barrel (Fig. 5). Condition-test ratios evoked by AW L and AW N were not significantly different from each other. There was, however, a 54% reduction in the size of the AW L-evoked excitatory response (P = 0.03, paired t-test); no correlation with lesion size was observed.

One possible explanation for the normal levels of AW L-evoked excitation and inhibition observed in the Barrel recordings is that AW L effects were mediated by adjacent barrel
remnants that may have survived the lesion and continued to communicate with the test barrel by means of direct, straight-line connections across the interbarrel septum (see Fox 1994). We therefore performed one experiment in which 14 additional, smaller lesions (10 μa, 10 s) were made in 7 penetrations, at 1,050 and 700 μm depths, in a line along the septal region between the ablated adjacent barrel and the test barrel. This procedure resulted in extensive damage to both the ablated barrel and the intervening septum and eliminated any possibility of direct, straight-line barrel-to-barrel communication. Nevertheless, AWL-evoked excitation and inhibition remained at normal levels (Barrel, spikes/stimulus: AWL = 0.65 ± 0.28, AWN = 0.69 ± 0.30; condition-test ratio: AWL = 0.50 ± 0.15, AWN = 0.56 ± 0.15; n = 5).

Another possible explanation for the ineffectiveness of the lesion is that slight, unintended movements of the test barrel’s PW that occur on AW deflection directly activate the test barrel. This may be of particular concern when the PW remains held by a stationary stimulator during AW deflection (Simons 1985). To address this issue, we performed two experiments in which the peripheral nerves innervating the PW were reversibly inactivated by injection of lidocaine into the follicle. Immediately after injection, units in the test barrel were completely unresponsive to the PW and partially responsive to the AW. Within 45 min after injection, AW-evoked excitation had recovered to near-normal levels, whereas responses to the (anesthetized) PW were absent entirely or reduced to below AW levels for an additional 15–60 min. We therefore consider it unlikely that mechanical transmission across the mystacial pad accounts for the bulk of the AWL or AWN response in the test barrel.

**DISCUSSION**

The major finding of this study is the remarkable preservation in the center of the test barrel of AW-evoked excitatory and inhibitory effects despite near-total ablation of the AW’s barrel. We consider layer VI neurons deep to the lesion an unlikely source of the normal levels of AWL-evoked effects, because at the minimum their apical dendrites were severely damaged by the lesion, which extended well into layer V. Results support our hypothesis that thalamic afferents normally provide direct excitatory AW input to a barrel (Fig. 1B). These thalamic inputs may originate from multiwhisker neurons in the homologous thalamic barreloid (Brumberg et al. 1996; Simons and Carvell 1989) and/or from neurons in adjacent, nonhomologous barreloids (Land et al. 1995; Land and Simons 1985). Further, AW-evoked inhibition is, on average, weaker in barreloid than barrel neurons. The observation of normal levels of AWL-evoked inhibition in the test barrel is consistent with the idea that thalamic activation of barrel circuitry on AW stimulation evokes surround inhibition by a feed-forward, intrabarrel mechanism, enhancing the response tuning of barrel neurons (Brumberg et al. 1996; Kyriazi and Simons 1993; Simons and Carvell 1989).

We attribute the abnormalities observed in Near-lesion cells to altered synaptic circuitry resulting from direct damage to thalamic afferents and/or dendritic processes of test barrel
neurons. Lesion by-products, e.g., elevated levels of extracellular potassium or glutamate, may also have contributed to Near-lesion abnormalities. If excitatory by-products disproportionately affect inhibitory barrel neurons, which are normally highly excitable, this could account for the somewhat paradoxical finding that AW$_{se}$-evoked inhibition was greater in Near-lesion than in Barrel units. In either case, the fact that receptive field abnormalities are observed in some neurons (Near-lesion) but not in other, more distant ones (Barrel) residing in the same barrel suggests that a barrel may contain several relatively independent subnetworks (see Chmielowska et al. 1989; McCasland et al. 1992). The nature and sizes of such possible circuits, and the degree to which they may or may not interact, remain to be determined.

In contrast to results in layer IV, adjacent barrel lesion led to a clear reduction in AW$_L$ responses recorded in layers superficial to the test barrel. We suggest that AW input normally reaches supragranular layers via several pathways (see Bernardo et al. 1990a,b; Gottlieb and Keller 1997): 1) an intracolumnar, vertical pathway originating within the test barrel itself, 2) a pathway originating in the adjacent barrel, which includes an additional horizontal, intercolumnar component within the supragranular layers, and 3) recurrent collaterals from infragranular neurons deep to the adjacent barrel. The lesions eliminated the second and possibly the third of these routes. The present findings in layer IV differ markedly from those of two previous studies, which used a similar experimental paradigm but in urethan-anesthetized animals (Armstrong-James et al. 1991; Fox 1994). In those studies, barrel lesion reduced excitatory AW$_L$ responses in proportion to the extent of the lesion, and modal latencies increased from 15.2 to 24.3 ms. Inhibitory interactions were not assessed. The authors concluded that direct barrel-to-barrel connections normally mediate AW responses (see Fig. 1A). In the study of Armstrong-James et al., the mean barrel destruction was 58% compared with 90% in the present study. Moreover, it appears that the present lesions extended deeper and more superficially (judging from Fig. 4 in Armstrong-James et al. 1991). Although Armstrong-James and colleagues did not categorize the barrel units with respect to their proximity to the lesion, as done here, it is clear from their METHODS section that most of their data were obtained >100 μm from the side of the barrel closest to the lesion. Thus differences in results cannot be explained by differences in the location of the recorded units or by differences in lesion size.

The most likely explanation for the discrepant findings is that AW-evoked excitatory responses are qualitatively different in the two experimental preparations. In terms of AW response latency and magnitude, relative to PW responses, the present data are comparable to values obtained previously in awake, undrugged rats (Simons et al. 1992). As discussed in that study, urethan anesthesia increases the magnitude and duration of AW-evoked responses, possibly through involvement of N-methyl-D-aspartate (NMDA) receptors (see Armstrong-James et al. 1993). After exposure of tangential barrel field slices to bicuculline methiodide, NMDA-dependent paroxysmal discharges can propagate across the barrel field (Fleidervish et al. 1998). Thus it appears that long latency, long-duration, barrel-dependent AW responses are expressed under conditions where NMDA-dependent synaptic transmission may be more prominent.

Taken together with results of previous modeling studies (Kyriazi and Simons 1993; Pinto et al. 1996), the present findings demonstrate that local, intrabarbrel circuitry is sufficient to account for the integration, both excitatory and inhibitory, of multithreshold information within individual layer IV barrels. Although there are almost certainly some means for barrels to influence each other, directly or indirectly, interactions are likely to be modulatory, perhaps contributing to the overall excitability of the barrel field during different behavioral states (see McCasland et al. 1997 for a discussion). Whatever role such interactions might play, available anatomic and physiological evidence indicates that barrels function as an array of independent, parallel processors of afferent information. Accordingly, barrel circuitry transforms multiple-whisker inputs into predominantly single-whisker outputs, which are then distributed to other layers of the cortical column, where larger and more complexly organized receptive fields are synthesized via intercolumnar connections.

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Address reprint requests to D. J. Simons.

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