

FIG. S1. Effect of gabazine on unsorted, multiunit sites in control and D1-spared rats. Multiunit sites were classified as control (n = 12), shifted (n = 8); in which >80% of component single units had shifted receptive fields), or unshifted (n = 4); in which >80% of component single units had unshifted receptive fields). Four sites were excluded because they contained a more equal mix of shifted and unshifted single units. *Left:* control sites. Gabazine preferentially enhanced D1 (SW) responses. Black, all sites with non-0 predrug responses to D1–D3. Gray, all sites with nonzero predrug responses to D1 and D2. Bars are SE. *Middle:* shifted sites in D1-spared rats. The trend toward preferential disinhibition of deprived PW responses was similar to that found for shifted single units (Fig. 4) but was weaker and nonsignificant (P = 0.07; Wilcoxon test), consistent with the inclusion of $\leq 20\%$ of nonshifted single units within "shifted" multiunit sites. *Right:* unshifted sites in D1-spared rats.

which are readily suppressed below spike threshold by even modest amounts of inhibition. Thus we distinguish below between the net functional effect of inhibition on whisker tuning, which can be inferred from these data, and the magnitude of inhibitory potentials or conductances, which cannot.

Inhibitory sharpening of receptive fields in control animals

In control animals, gabazine preferentially disinhibited surround whisker responses, thereby broadening whisker receptive fields. This effect was reflected in the D1-di, which increased significantly with gabazine, indicating that when inhibition was blocked, tuning broadened to include more surround D1 whisker responses. We interpret these results to indicate that GABAergic conductances on L4 and L2/3 neurons normally act to preferentially suppress surround whisker responses and sharpen whisker receptive fields. This finding is consistent with previous studies in S1 (Kelly et al. 1999; Kyriazi et al. 1996b, 1998; Simons and Carvell 1989) and other cortical areas (Foeller et al. 2001; Miller et al. 2001; Sompolinsky and Shapley 1997; Wang et al. 2002; Wehr and Zador 2003).

How inhibitory conductances sharpen whisker tuning is not clear. L4 and L2/3 neurons receive tonic and whisker-evoked inhibition from local interneurons (Brumberg et al. 1996; Bruno and Simons 2002; Douglas and Martin 2004; Porter et al. 2001; Simons and Carvell 1989; Swadlow and Gusev 2002; Welker et al. 1993). In a classical lateral inhibition model, nonpreferred (SW) inputs are hypothesized to evoke a larger inhibitory conductance than preferred (PW) inputs, leading to preferential suppression of SW responses. Alternatively, inhibitory conductance may be untuned, broadly tuned, or co-tuned with excitatory inputs. The existence of any of these patterns of inhibitory conductance would preferentially suppress spiking responses to weak (SW) excitatory inputs, relative to strong (PW) excitatory inputs, because weak inputs are more readily reduced below spike threshold by either subtractive or divisive inhibition (Anderson et al. 2000; Heeger 1992; Miller et al. 2001; Wehr and Zador 2003). Thus in these models, inhibition and the spike threshold act together to sharpen the tuning of the cell's spiking output around the whisker that elicits the strongest excitatory synaptic input. The current data do not distinguish between these models, although whole cell recording experiments in vivo argue against the lateral inhibition model (Brecht et al. 2003; Moore and Nelson 1998).

Receptive field plasticity and the effect of inhibition in univibrissa rats

Following univibrissa experience, many L2/3 neurons showed decreased responses to the deprived PW, consistent with previous descriptions of whisker map plasticity (Glazewski and Fox 1996). However, we did not observe a second, previously reported effect of univibrissa experience, an increase in responses to the spared SW whisker (Glazewski and Fox 1996). This discrepancy may reflect the fact that potentiation of spared whisker responses required ≥ 20 days of univibrissa experience in prior studies, whereas most rats (6/7) in our study were plucked <20 days (Glazewski and Fox 1996). Alternatively, this effect may be less robust in 2-wk-old rats, compared with the 1- and 4-wk-old rats studied previously (Fox 1992; Glazewski and Fox 1996).

For L2/3 units the receptive fields of which were shifted substantially away from the PW by univibrissa experience, gabazine preferentially disinhibited responses to the deprived PW, opposite to its effect in controls (Fig. 4). As a result, gabazine application tended to restore the D1-dominance index of shifted units toward values observed in control animals (Fig. 5). We interpret these results to indicate that in shifted units, GABA_A conductances preferentially suppressed PW responses, rather than SW responses as in controls, and therefore that inhibition helped to sharpen whisker tuning around the spared SW, thereby promoting the receptive field shift away from the deprived PW. This finding is consistent with early studies of monocular deprivation and strabismus, in which deprived eye responses could be restored by application of the GABA_A-receptor antagonist bicuculline (Mower et al. 1984; Sillito et al. 1981). In contrast, the effect of gabazine on receptive fields was unaltered for L2/3 units with unshifted receptive fields and in L4, where receptive field plasticity did