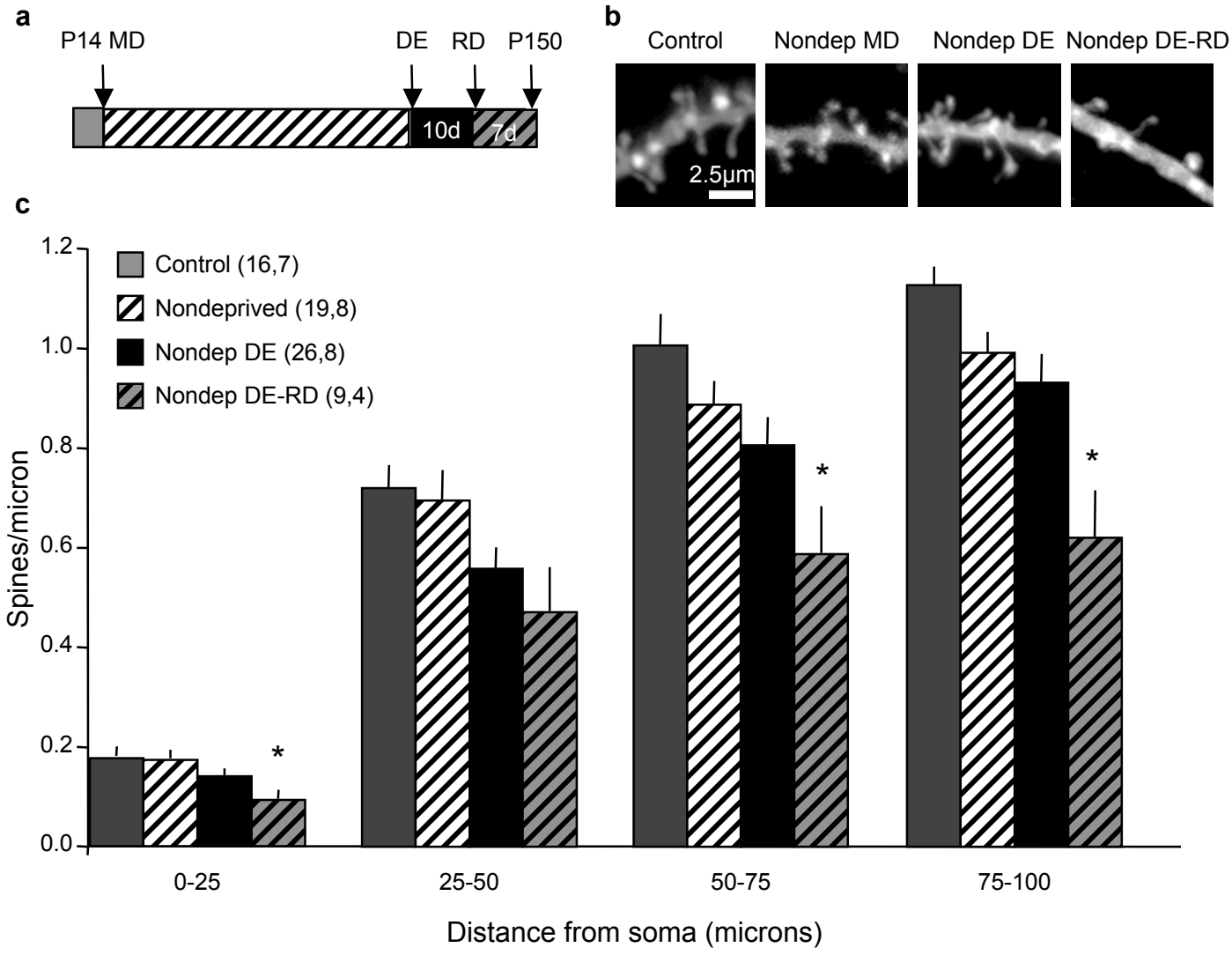
monocular deprivation is reversed by dark exposure and reverse deprivation, *p<0.020, KS test.

Figure 5: Enhancement of the thalamocortical VEP in the deprived visual cortex during the recovery from chronic monocular deprivation. A. Top: Post stimulus time histograms plot action potentials (in 50 ms bins) versus time using a visual stimulus reversing every 500 ms (arrows). Intracortical infusion of muscimol+SCH50911 induces a reversible silencing of cortical spiking and reduction in VEP amplitude. Cortical spiking and VEP amplitude recover following washout (~ 10 hours), scale bars = 50 ms, 50 μvolts. B and C. Pharmacological isolation of the thalamocortical VEP in subjects that received chronic monocular deprivation and chronic monocular deprivation followed by dark exposure and reverse deprivation (*p<0.005 paired t-test). D. Normalization of the thalamocortical VEP to the total cortical VEP reveals the recovery from chronic monocular deprivation is accompanied by an enhancement of thalamocortical VEP amplitude (*p=0.027 unpaired t-test). E. Inhibition of cortical spiking does not modify the VEP contralateral bias in subjects that received chronic monocular deprivation, or after chronic monocular deprivation is reversed by dark exposure and reverse deprivation.

Supplemental figure 1: Dendritic spines in the visual cortex ipsilateral to the occluded eye (non-deprived cortex) are stable across experimental conditions, until anatomical plasticity is reactivated by dark exposure in adulthood. A. Experimental timeline. B. Representative DiI labeled basolateral dendrites from pyramidal neurons in the binocular region of the non-deprived visual cortex (ipsilateral to occlusion) in control, chronically monocularly-deprived (Nondep cMD), chronically monocularly-deprived plus dark exposure (Nondep cMD DE) and chronically monocularly-deprived plus dark exposure and reverse deprivation (Nondep cMD DE-RD). In each case the dendritic segment shown is 75–100 μm from cell body. C. No change in spine density (0-100 μm from the soma) in the non-deprived cortex following chronic monocular deprivation or dark exposure. However a reduction in dendritic spine density was observed along segments of non-deprived visual cortex when chronic monocular deprivation as followed by dark exposure and reverse deprivation (average ± SEM 0-25 μm control=0.16±0.02,

Nondep=0.16±0.02, Nondep DE=0.13±0.01, Nondep DE-RD=0.08±0.02; one-way ANOVA, $F_{(3,69)}$=3.4272, p=0.0220, *p<0.05 versus control, Tukey-Kramer HSD post-hoc; 25-50 μm control=0.67±0.04, Nondep=0.64±0.06, Nondep DE=0.52±0.04, Nondep DE-RD=0.44±0.08; one-way ANOVA, $F_{(3,69)}$=3.5132, p=0.0199, *p<0.05 versus control, Tukey-Kramer HSD post-hoc; 50-75 μm control=0.93±0.06, Nondep=0.82±0.04, Nondep DE=0.74±0.05, Nondep DE-RD=0.54±0.09 spines/μm; one-way ANOVA $F_{(3,68)}$=5.6344, p=0.0017, *p<0.05 versus control, Tukey-Kramer HSD post-hoc; 75-100 μm: control=1.04±0.03, Nondep=0.92±0.04, Nondep DE=0.86±0.05, Nondep DE-RD=0.57±0.09 spines/μm; one-way ANOVA, $F_{(3,61)}$=9.7476, p<0.0001, *p<0.05 versus control, Tukey-Kramer HSD post-hoc; n=neurons, subjects).


a
P14 MD
DE
RD
P150
10d
7d
b
Control
Nondep MD
Nondep DE
Nondep DE-RD
2.5µm
c
Control (16,7)
Nondeprived (19,8)
Nondep DE (26,8)
Nondep DE-RD (9,4)
Spines/micron
1.2
1.0
0.8
0.6
0.4
0.2
0.0
*
*
*
0-25
25-50
50-75
75-100
Distance from soma (microns)