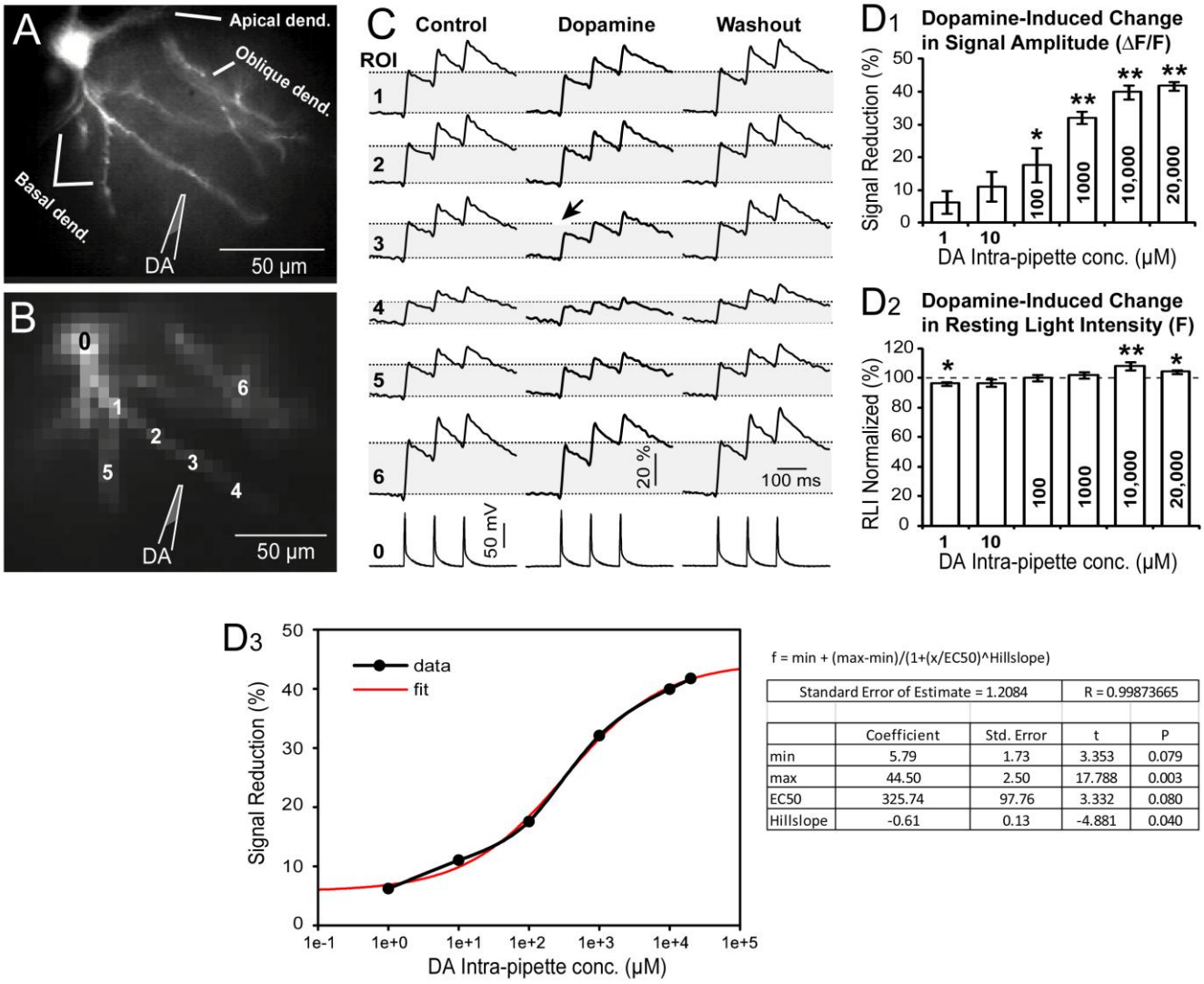
Suppl. Fig. S1.

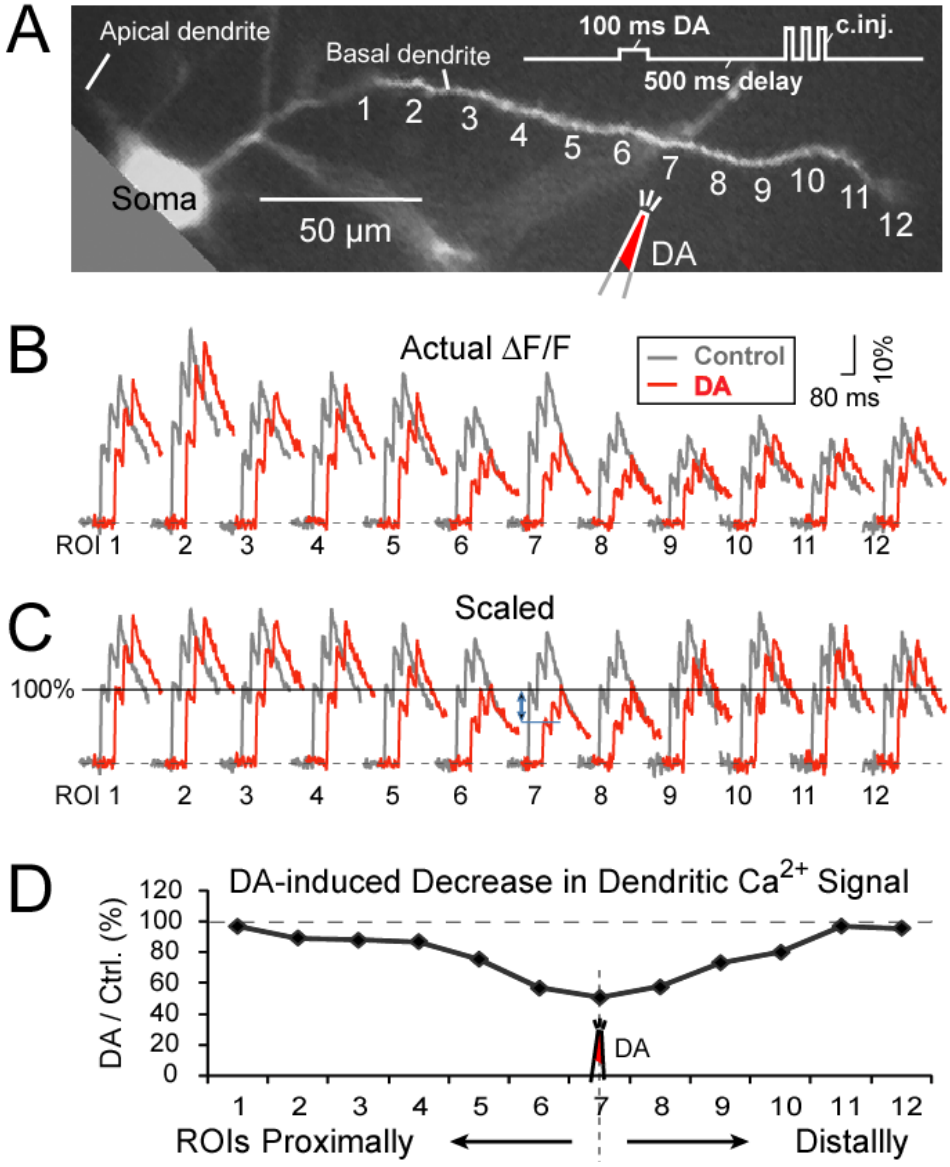


f = min + (max-min)/(1+(x/EC50)^Hillslope)

Standard Error of Estimate = 1.2084			R = 0.99873665
	Coefficient	Std. Error	t	P
min	5.79	1.73	3.353	0.079
max	44.50	2.50	17.788	0.003
EC50	325.74	97.76	3.332	0.080
Hillslope	-0.61	0.13	-4.881	0.040

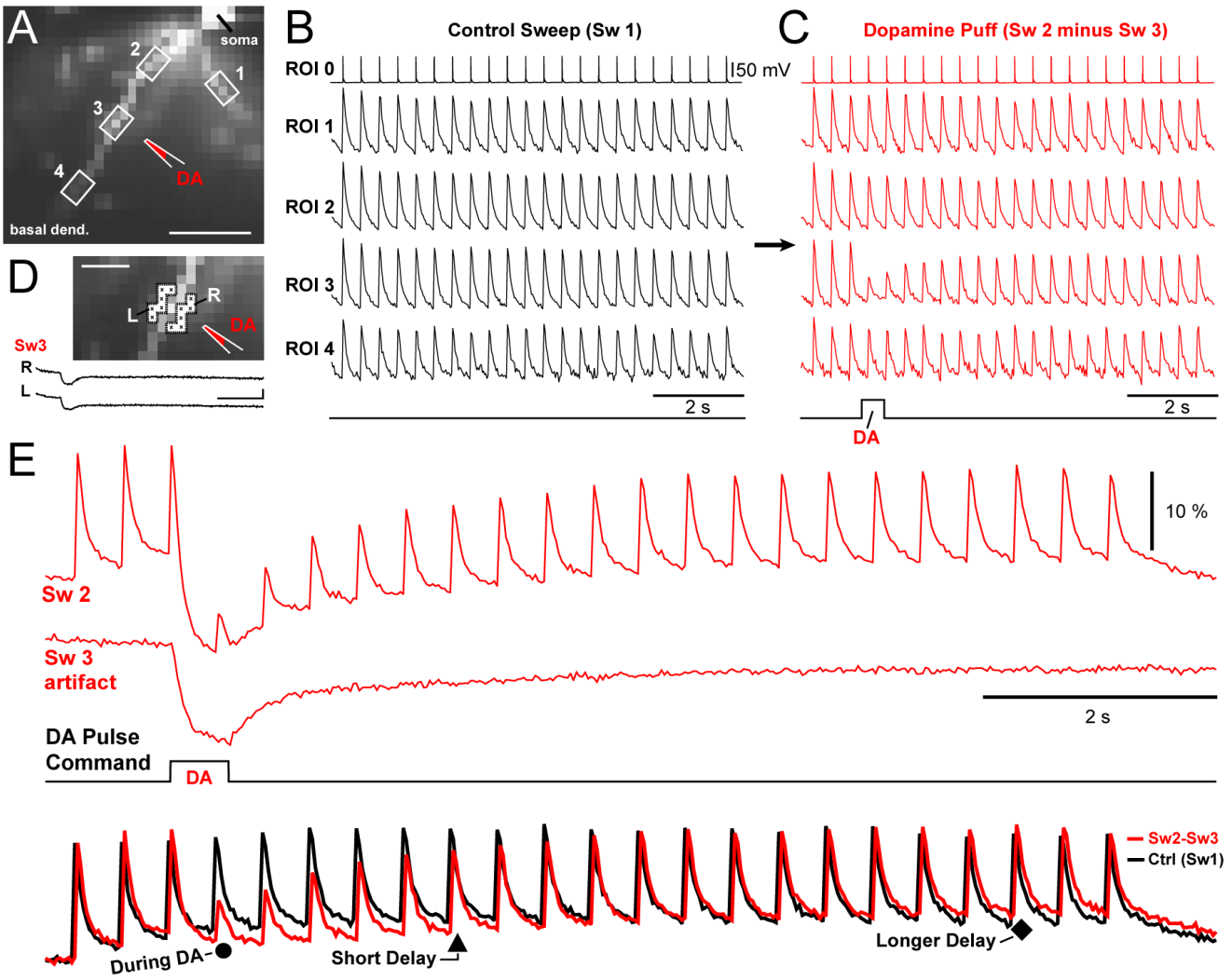
Suppl. Fig. S1. Local DA Application Suppresses AP-mediated Ca^{2+} Transients. This figure is complementary to *Figure 1* of the main manuscript. (**A**) Microphotograph of a pyramidal neuron filled with OGB-1 and Alexa Fluor 594. (**B**) One movie frame captured by fast CCD camera (500 Hz, 80x80 pixel). The drawing marks the position of a DA-filled pipette (1 mM) inserted into the brain slice. (**C**) Somatic current injections were used to produce 3 action potentials. Electrical signal from the soma (region of interest, ROI, 0) is aligned with dendritic recordings from six ROIs (1-6) shown in panel *B*. Upon application of dopamine (DA puff) the amplitude of the first AP-mediated Ca^{2+} transient at ROI 3 (*Dopamine*) is less than that in the measurement obtained just before DA puff (*Control)*, or 90 s after DA puff (*Washout*). Arrow points to a signal which is significantly reduced compared to the control measurements obtained at the same recording location (dashed line). (**D_1**) Ca^{2+} signal decreases with an increase in the concentration of DA inside the application pipette. (**D_2**) Resting light intensity (RLI) changes during DA application. In this and all remaining figures single (*) and double asterisks (**) mark statistical significance paired T-test $P<0.05$ and $P<0.01$, respectively. (**D3**) Data from *Figure 1* of the main manuscript, plotted on logarithmic scale (black line) and fitted (red line) with the equation shown above table on the right. Table contains parameters of the fit, including EC50 = 325.7 ± 98 µM.

Suppl. Fig. S2.



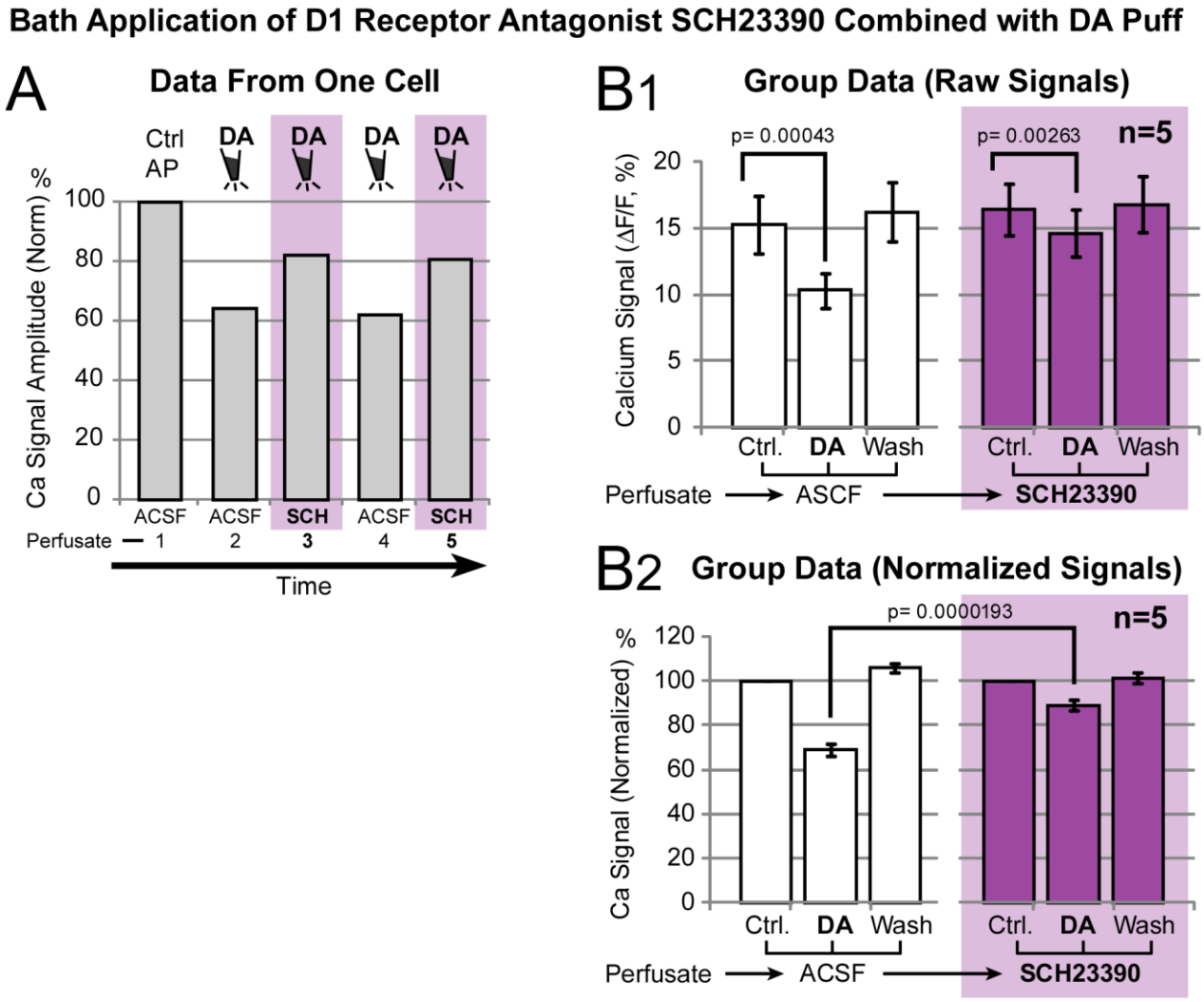
Suppl. Fig. S2. Spatially-restricted effect of Dopamine (DA). This figure is complementary to *Figure 2A* of the main manuscript. (**A**) Pyramidal L5 neuron filled with OGB-1 and Alexa Fluor 594. The drawing marks the position of a DA-filled micropipette [20 mM]. (**B**) AP-induced Ca^{2+} transients were recorded simultaneously at multiple loci (marked in ***A***) before (Control, grey) and 0.5 sec after a brief (100 ms duration) iontophoretic ejection of DA (red). The command pulses for dopamine iontophoresis and somatic current injection are shown in ***A*** (upper right corner). (**C**) Signal amplitudes of the Control recording (grey) were scaled to the same height. This scale is then applied on corresponding red traces (same ROI). Upon DA puff (red) the signal amplitudes were reduced in dendritic segments closest to the tip of the DA application pipette (same scale between corresponding red and grey). (**D**) Quantification of data was performed by calculating the amplitude ratio between DA (red) and Control (grey) for each region of interest (1-12).

Suppl. Fig. S3.



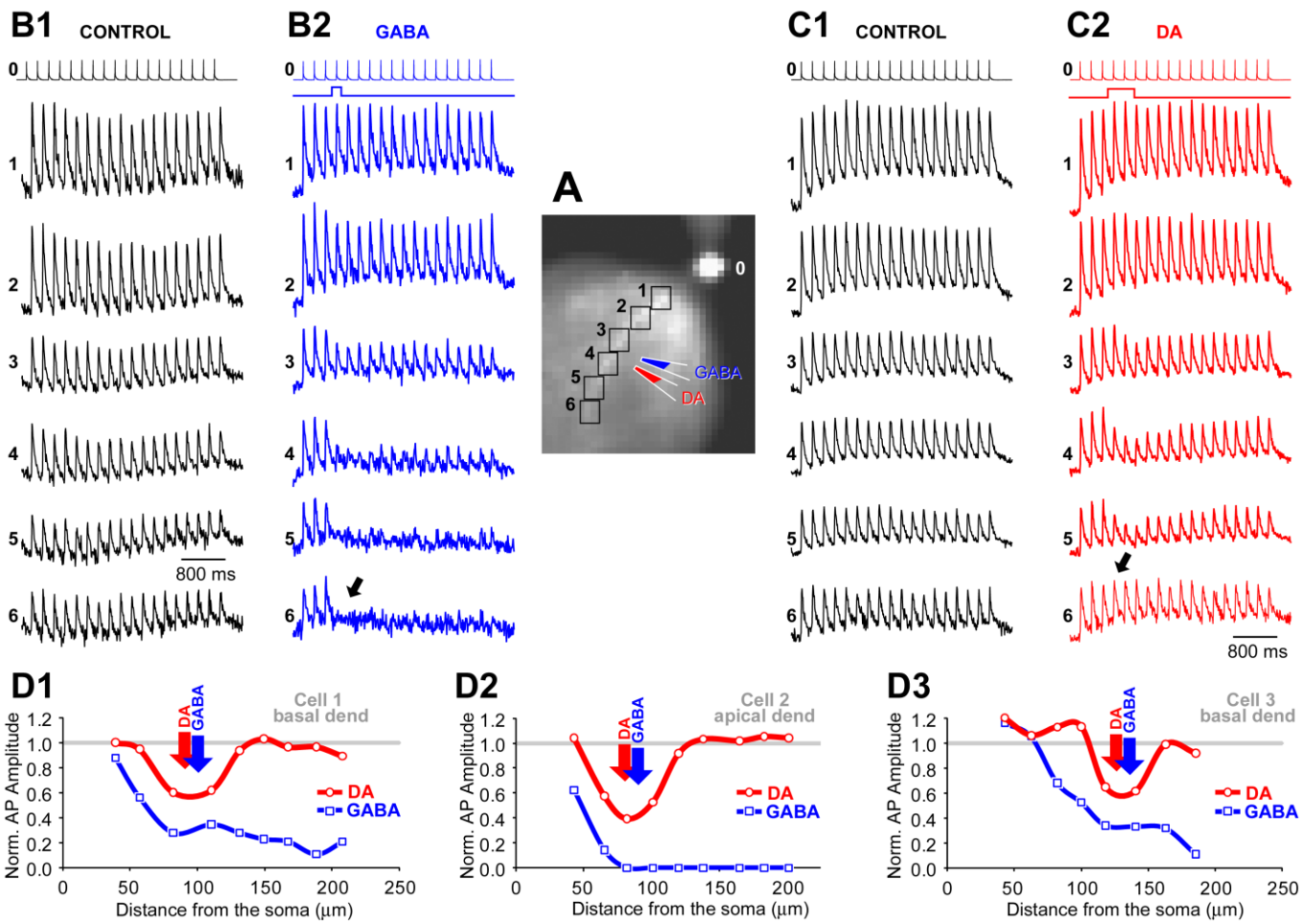
Suppl. Fig. S3. Latency of the DA effect. This figure is complementary to the main ***Fig. 2B***. (**A**) Pyramidal L5 neuron filled with OGB-1 and Alexa Fluor 594. The drawing marks a DA-filled pipette [10 mM IPC] positioned against the basal dendrite. (**B**) In the control sweep (Sw 1 Control) the cell body was injected with short current pulses to produce a train of 23 somatic APs (ROI 0, 2.5 Hz). The corresponding dendritic Ca^{2+} transients were recorded simultaneously from 4 locations marked by boxes in ***A*** (ROI 1 - 4). Signals are high-pass filtered (0.1 Hz) and arbitrarily scaled between ROIs for clarity. (**C**) Same ROI-specific scales as in the previous panel except DA puff was applied locally at time point indicated by the command pulse "DA". (**D**) On sweep 3 (Sw 3), DA was ejected on a resting neuron (no AP firing) to assess the mechanical/optical artifact caused by DA application alone. Both, the left edge (L) and the right edge (R) of the dendritic contour, showed a simultaneous decrease in fluorescent light, thus suggesting that DA-induced change in resting light intensity was not a simple motion artifact. (**E**) Sw2: Pairing of APs and DA pulse. Sw3: Same position and focus as in Sw2, same DA pulse, except APs were not evoked. The command signal for driving the picospritzer valve shows the exact timing of DA pulses in Sw2 and Sw3. Bottom traces: Ca^{2+} signal obtained in the Control condition (black) is superimposed with the one measured during the DA puff (red). Heavy red line is generated by subtracting Sw3 from Sw2. High-pass filtering was not applied on traces in panel ***E***. The same cell shown in ***A-E*** is featuring in ***Fig. 2B*** of the main manuscript.

Suppl. Fig. S4.



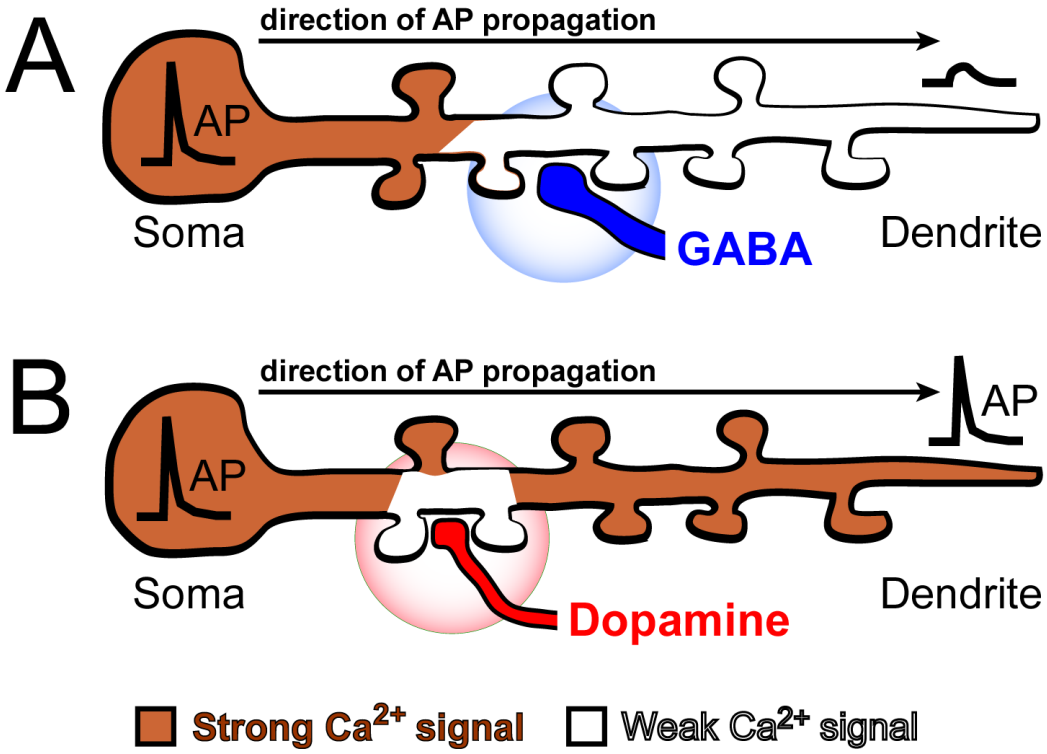
Suppl. Fig. S4. D1 Receptor Antagonist. This figure is complementary to the main ***Fig. 3E***. (**A**) Dendritic Ca^{2+} transients were measured in the same dendrite in 5 trials. The first column on the left (Ctrl AP) represents the baseline amplitude of the dendritic Ca^{2+} transient induced by backpropagating AP in drug-free perfusate (ACSF, Perfusate 1). The second column indicates amplitude of the Ca^{2+} transient in response to DA puff, still in drug-free saline (ACSF, Perfusate 2). The following 3 repetitions of the DA puff were made in the presence of D1 receptor antagonist SCH23390 [150 µM] (SCH, Perfusate 3), upon wash of SCH (ACSF, Perfusate 4), and re-application of D1 antagonist (SCH, Perfusate 5), in that order. Every time the D1 antagonist SCH was present in the bath, the potency of DA puff (to decrease Ca^{2+} signal) was reduced. (**B_1**). Group data on the basal dendrites treated with DA in drug-free ACSF (WHITE columns), and the same dendrites perfused with SCH [100 µM] (PURPLE columns). Regardless of the absence or presence of the D1 antagonist SCH, the dopamine puff caused a statistically significant reduction in the calcium signal amplitude, which indicates that the effect of DA was not eliminated by the D1 receptor antagonist SCH. (**B_2**) Same data as in ***B_1*** but normalized against its corresponding control measurement. The ability of DA puff to reduce dendritic Ca^{2+} transient was significantly less in the presence of the D1 receptor antagonist SCH (paired t-Test, p = 0.0000193, n = 5). The same graph shown here in ***B_2*** is featuring in ***Fig. 3E*** of the main manuscript.

Suppl. Fig. S5.



Suppl. Fig. S5. DA versus GABA on a Same Dendrite. This figure shows the same cell featuring in Fig. 9BC, but in greater detail. (**A**) Pyramidal L5 neuron filled with OGB-1 and Alexa Fluor 594. The schematic drawings mark the positions of DA-filled and GABA-filled pipettes. (**B_1**) The cell body was injected with short current pulses to produce a train of 18 somatic APs (ROI 0). The corresponding dendritic Ca^{2+} transients were recorded simultaneously from 6 locations marked by boxes in ***A***. Each output of the 6 ROIs was arbitrarily scaled for display. (**B_2**) Same detectors, same scales as in previous panel except GABA puff was applied locally at time point indicated by the GABA command pulse. Arrow indicates a dramatic loss of the optical signal, suggestive of a complete failure of AP backpropagation distally from the GABA application site. (**C_1**) Same detectors as in ***B_1***. This recording was made prior to DA application (Control). (**C_2**) Same detectors, same scales as in ***C_1***, except DA was applied at time point indicated by the DA command pulse. Arrow indicates completely intact optical signals, suggestive of a successful AP backpropagation into dendritic segments distal to the DA application site. (**D_1 – D_3**) Drug-induced changes in calcium signal amplitude along a dendritic branch normalized against the corresponding (ROI-specific) control measurements, in three neurons (Cell 1- 3) treated with sequential DA and GABA applications on the same branch. Control measurements were obtained prior to each drug application. **GABA**, IPC = [1 mM], Duration=20-200 ms; **DA**, IPC = [10 mM], Duration=500 ms. Locations of drug-filled pipettes in respect to the distance from the cell body are marked by color-coded arrows.

Suppl. Fig. S6



Suppl. Fig. S6. DA-ergic versus GABA-ergic Modulation of Dendritic AP-induced Ca^{2+} Transient. (**A**) Schematics of a pyramidal neuron. Only one spiny dendrite is shown for clarity. Only one GABA synapse is shown impinging upon the dendrite. A GABA-ergic input (blue sphere) blocks the AP backpropagation process. The AP voltage waveform is lost in the distal dendritic tip. As a consequence of this AP block, the AP-associated dendritic Ca^{2+} signals were drastically diminished, or eliminated, in dendritic segments distal to the GABA synapse (white dendritic region). In order words, while the cell body and dendritic segments proximal to the GABA input site experience strong AP-mediated Ca^{2+} influx (brown), the dendritic segments distal to the GABA input site are left without the AP-mediated Ca^{2+} influx (white). (**B**) Same as in **A** except the GABA-ergic synapse is replaced by a DA-ergic synaptic contact (Dopamine). The activation of the DA-ergic synapse suppresses dendritic Ca^{2+} locally (at the input site, red sphere) but it does not block the propagation of electrical transients (AP backpropagation), so distal dendritic segments receive normal AP-associated Ca^{2+} flux (brown regions). At the DA input receiving locus (red sphere), however, the electrical signal (backpropagating AP) is not accompanied by a strong Ca^{2+} transient (white region).