Supplementary Fig. 1) Specificity of GABA-immunoreactivity in the zebra finch brain.

A) Low power photomicrograph depicting GABA-like immunoreactivity in the nidopallium of a male zebra finch. This section was incubated with the anti-GABA antibody that was pre-absorbed with 50μM of unbound BSA. B) Photomicrograph of a section incubated with the anti-GABA antibody pre-absorbed with 50μM of the GABA-BSA conjugate (see Methods). Note that GABA-like immunoreactivity was completely abolished after the pre-absorption with the GABA-BSA conjugate. Scale bar = 100μm.

Supplementary Fig. 2) GABA Immunoreactivity in Known GABAergic Neurons in Zebra Finches.

A) Low-power view of part of a cerebellar folium, depicting labeling of Purkinje cells; arrowheads indicate examples of immunopositive cells with clear immunonegative nucleus. Arrows depict small labeled cells in the molecular layer. B) High power view of cerebellar Purkinje cells and respective dendritic arborizations. Arrow depicts putative basket cell in the molecular layer. C) Low power photomicrograph of the transition between the GP and CSt. Note that GABAergic cells are strongly labeled and highly concentrated in the dorsal aspect of the GP, but are found at very low densities in the CSt. D) High power view of GP depicting somata of labeled cells and respective dendrites extending into the CSt (arrows). E) Detail view of GABAergic cells in RA and adjacent arcopallium. Dashed line indicates the boundary of RA based on Nissl counterstaining. F) High power photomicrograph of GABA-labeled cells within RA, consistent with previous observations. Arrowheads indicate immunolabeled processes that extend from

labeled cells, branch, and form an intricate fiber network. Scale bars (in μm): 50 (A); 25 (B); 100 (C); 25 (D); 50 (E); 25 (F). ml=molecular layer; pcl=Purkinje cell layer; gcl=granular cell layer; CSt=caudal striatum; GP=globus pallidus; A=arcopallium; RA=robust nucleus of the arcopallium.

Supplementary Fig. 3) The Large Majority of sPSCs in NCM Are Mediated By $GABA_A$ Receptors.

A) Spontaneous GABAergic events recorded in a representative NCM neuron. B) Application of 20 μM BIC abolishes the large majority of the spontaneous events. C) The remaining spontaneous events, which exhibit both lower frequency and smaller amplitude, are blocked by addition of 20 μM DNQX to the recording bath. Scale bar applies to all traces.

Supplementary Fig. 4) Song-Evoked Spiking Pattern in NCM in a Representative Recording Session (Prior to Drug Treatment).

This figure illustrates the typical extracellular spiking pattern observed in NCM in response to the presentation of a conspecific song. This auditory area typically exhibits low baseline activity, but responds robustly and in phase with the stimulus. Importantly, NCM responses display sustained (tonic) firing throughout the duration of the stimulus, which typically includes the large majority of the silent inter-syllable intervals. The top four traces illustrate representative extracellular recordings obtained from four electrodes placed in the right (experimental) NCM; the next three traces illustrate recordings obtained with the three electrodes placed in the left (control) NCM, and the bottom trace

illustrates the amplitude envelope of the corresponding conspecific song. Note that this auditory stimulus triggers vigorous responses in both hemispheres.

Supplementary Fig. 5) The Effects of BIC and PTX Treatment on NCM Responses to Song Are Not Significantly Different.

The effects of drug application (BIC-red; PTX-green) on amplitude (A) and standard deviation (B) of NCM responses to song were compared for both control and experimental hemispheres by using a post/pre ratio (detailed in Fig. 6). Vertical bars denote 95% confidence intervals. Multi-factorial ANOVA revealed no significant side differences for the response amplitude and no interaction with drug treatment was detected for this parameter. For STD comparisons, multi-factorial ANOVA revealed no interaction between hemispheres and drug treatment, although the ratio was found to be higher on the experimental than on the control hemisphere for both drugs. These data illustrate that the effects of BIC and PTX on the physiology of NCM neurons are quantitatively and qualitatively similar.

Supplementary Fig. 6) Action Potential Properties of NCM Neurons Under BIC.

Representative traces of action potentials recorded from NCM neurons in slices after BIC (20 μM) application to the recording bath. Action potentials were evoked by a depolarizing square current from resting membrane potential and recorded in a potassium gluconate solution. A) Example of tonic firing mode seen in 1 out of 13 neurons. B) Example of a tonic-accomodating firing mode seen in 3 out of 13 recorded neurons. C)

Example of phasic firing observed in 9 out of 13 neurons. Firing frequency of NCM neurons under these conditions ranged from 10 to 50 Hz.

Supplementary Fig. 7) The Response of NCM to Auditory Stimuli Under BIC is Unaffected by Spontaneous Bursts Immediately Preceding Stimulus Onset. Representative traces obtained from NCM sites under BIC where a spontaneous burst of activity occurred prior to the onset of auditory stimuli - a pure tone in (A) and a conspecific song in (B). In both situations, we observed highly similar firing activity in response to the stimulus, even though the spontaneous burst occurred within 100 ms of stimulus onset (~50 ms in [A] and ~80 ms in [B]). Each graph shows the responses recorded at the same site with (upper trace) and without (bottom trace) the spontaneous burst.