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Accessibility
A Direct Projection from Mouse Primary Visual Cortex to Dorsomedial Striatum

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Abstract

The mammalian striatum receives inputs from many cortical areas, but the existence of a direct axonal projection from the primary visual cortex (V1) is controversial. In this study we use anterograde and retrograde tracing techniques to demonstrate that V1 directly innervates a topographically defined longitudinal strip of dorsomedial striatum in mice. We find that this projection forms functional excitatory synapses with direct and indirect pathway striatal projection neurons (SPNs) and engages feed-forward inhibition onto these cells. Importantly, stimulation of V1 afferents is sufficient to evoke phasic firing in SPNs. These findings therefore identify a striatal region that is functionally innervated by V1 and suggest that early visual processing may play an important role in striatal-based behaviors.

Methods

Mice

All experimental manipulations were performed on mice in accordance with the protocols approved by the Harvard Standing Committee on Animal Care and with the guidelines described in the US National Institutes of Health Guide for the Care and Use of Laboratory Animals. All animals used in this study carried the Rbp4-Cre transgene (GENSAT #RP24-283K21). For imaging studies, some Rbp4-Cre mice were bred to mice bearing an allele encoding EGFP expressed in a Cre-dependent manner under a CAG promoter (referred to as ZSGreen1 reporter transgene; Ai6; the Jackson Laboratory, stock number 007906) to reveal the distribution of Cre+ cells. For electrophysiology experiments Rbp4-Cre animals were bred to Drd2-EGFP transgenic mice (GENSAT #RP23-161H15), which express EGFP under control of a bacterial artificial chromosome containing the type 2 dopamine receptor genomic locus to permit distinction between the direct- and indirect-pathways SPNs. All animals were maintained on a C57Bl/6 background. Both male and female animals were used.

Viruses and stereotaxic intracranial injections

Conditional expression of EGFP or of the H134R variant of ChR2 in Cre-containing neurons was achieved using recombinant adeno-associated viruses (AAVs) encoding a double-floxed inverted open reading frame (DIO) of ChR2-mCherry or EGFP, as

Introduction

Understanding the functional circuitry of the basal ganglia requires a thorough characterization of the sources of afferents to the striatum – the principal input structure of the basal ganglia – and their organization within it. Cortex provides one of the major excitatory inputs to the striatum, which was first described by Webster in the 1960s in rat and cat [1,2]. Similar studies subsequently emerged from other groups describing corticostriatal afferents in rabbit and monkey [3,4]. These studies indicated that all major cortical areas project to the striatum, noting that inputs from visual cortices targeted rather restricted striatal areas compared to other cortical regions. However, these early studies were performed using focial cortical lesions followed by a silver impregnation of degenerating axons, making it difficult to isolate specific areas of visual cortex and their striatal target regions. Several subsequent investigations have provided conflicting findings; whereas some studies revealed the existence of small corticostriatal inputs from V1 in rodents [5-8], others have failed to observe projections originating in V1 in the striatum of monkeys, cats and rodents [9-14]. Functional studies have reported visually-driven units in the dorsal striatum of rodents, cats and monkeys [15-18], but the relative contributions of primary and extrastriate visual cortices, and of other visual structures such as the superior colliculus are difficult to distinguish in vivo. It therefore remains unclear whether V1 functionally innervates the dorsolateral striatum.

In this study, we examined whether V1 establishes a direct, functional connection with the striatum using a combination of anatomical tracing methods, electrophysiology and optogenetics. We demonstrate that corticostriatal afferents originating in V1 form strong excitatory connections with both direct- and indirect-pathway striatal projection neurons (referred to as dSPNs and iSPNs, respectively), indicating that V1 may directly participate in visually-evoked basal ganglia behaviors.

Competing Interests: The authors have declared that no competing interests exist.

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Data Availability: The authors confirm that all data underlying the findings are fully available without restriction. All relevant data are within the article.

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described previously [19,20]. Briefly, for intracranial injections, mice (postnatal day 30–40) were anesthetized with isoflurane and placed in a small animal stereotaxic frame (David Kopf Instruments). After exposing the skull under aseptic conditions, a small burr hole was drilled for microinjections. All microinjections were performed through a pulled glass pipette using a UMP3 microsyringe pump (World Precision Instruments). Virus (AAV-DIO-EGFP or AAV-DIO-ChR2-mCherry) was injected into V1 (0.3 mm lateral from Lambda, and 0.6 mm below pia) at the rate of 50 nl per minute (500–750 nl total volume). Fluorescent latex microspheres (Red Retrobeads, Lumaduall) were injected into dorsomedial striatum (0.9 mm anterior and 1.5 mm lateral from Bregma, 2.0 mm below pia) at the rate of 100 nl per minute (150 nl total volume). Following the injection, the skin was sutured using Vicryl 7.0 silk sutures (Ethicon) and mice were returned to their home cage for at least 21 days for AAV injections and 7 days for experiments using fluorescent retrobeads.

**Tissue processing for imaging**

Mice were deeply anesthetized with isoflurane and perfused transcardially with 4% paraformaldehyde in 0.1 M sodium phosphate buffer. Brains were post-fixed for 24 hours at room temperature, washed in phosphate buffer saline (PBS) and sectioned (50 μm) coronally using a vibratome (Leica VT1000). Brain sections were mounted on superfrost slides, dried and coverslipped using ProLong antifade reagent containing DAPI (Molecular Probes). Whole brain sections were imaged with an Olympus VS110 slide-scanning microscope. High-resolution images of regions of interest were subsequently acquired using an Olympus FV1000 confocal microscope (Harvard Neurobiology Imaging Facility). All confocal images were acquired in a single plane and processed using ImageJ.

**Acute slice preparation and electrophysiology**

Acute brain slices and whole-cell voltage-clamp recordings from identified SPNs were obtained using standard methods, as described previously [19,20]. Briefly, mice (50–80-days old) were anesthetized by isoflurane inhalation and perfused transcardially with ice-cold artificial cerebrospinal fluid (ACSF) containing (in mM): 125 NaCl, 2.5 KCl, 25 NaHCO3, 2 CaCl2, 1 MgCl2, 1.25 NaH2PO4 and 11 glucose (290 mOsm per kg). Cerebral hemispheres were removed, placed in cold choline-based cutting solution consisting of (in mM): 110 chloride choline; 25 NaHCO3, 2.5 KCl, 7 MgCl2, 0.5 CaCl2, 1.25 NaH2PO4, 25 glucose, 11.6 ascorbic acid, and 3.1 pyruvic acid), blocked and transferred into a 2.5 KCl, 7 MgCl2, 0.5 CaCl2, 1.25 NaH2PO4, 25 glucose, 11.6 ascorbic acid, and 3.1 pyruvic acid), blocked and transferred into a

**Data acquisition and analysis**

Membrane currents and potentials were amplified and low-pass filtered at 3 kHz using a Multiclamp 700B amplifier (Molecular Devices), digitized at 10 kHz and acquired using National Instruments acquisition boards and a custom version of ScanImage written in MATLAB (Mathworks). Electrophysiology data was analyzed offline using Igor Pro (Wavemetrics) and imaging data was analyzed using ImageJ (National Institutes of Health). Averaged waveforms of 3–5 consecutive sweeps were used to obtain current onset and peak current amplitude. Detection threshold for IPSCs and EPSCs was set at 10 pA. Current onset was measured using a threshold set at three standard deviations of baseline noise. Peak amplitudes were calculated by averaging over a 2 ms window around the peak. For experiments using drug perfusions, peak amplitudes of three consecutive light-evoked responses 3–4 min after drug perfusion onset were averaged, normalized to baseline averages and compared statistically with values obtained at corresponding times in control preparations bathed in ACSF. Data (reported in text and figures as mean ± s.e.m.) were compared statistically using a Mann-Whitney U test. P values less than 0.05 were considered statistically significant.

**Reagents**

Drugs (all from Tocris) were applied by bath perfusion: gabazine (SR95531; 10 μM), 2,3-dihydroxy-6-nitro-7-sulfamoylbenzo[f]quinoxaline (NBQX; 10 μM), R-3-(2-carboxypiperazin-4-yl)propyl-1-phosphonic acid (CPP; 10 μM).

**Results**

Anterograde labeling reveals a direct projection from V1 to dorsomedial striatum (DMS)

To address whether layer 5 pyramidal cells in primary visual cortex (V1) project to the striatum of mice, we injected an adeno-associated virus (AAV) encoding Cre-dependent EGFP into V1 of Rbp4-Cre transgenic mice, which express Cre in a subset of layer 5 pyramidal neurons throughout cortex [21]. Projecting axon bundles emerging from V1 were readily traced across sections throughout the brain of 8 mice, and followed stereotyped trajectories and innervation patterns in all brains studied (Figure 1A–F). As axons exited V1, EGFP-labeled axons formed a thick bundle that traveled ventrally in the ipsilateral external...
capsule (Figure 1A–B). These axons innervated well-defined projection areas, including the superior colliculus (Figure 1B) and the dorsal part of the lateral geniculate nucleus (Figure 1C–D), confirming the site of injection as V1 [22–24]. Interestingly, V1 axons also left the external capsule (Figure 1D) to enter the most posterior part of the ipsilateral striatum and then coursed anteriorly within the striatum in tight axonal bundles lining dorsal and medial regions. Terminal fields, which were identified by the splaying of filiforms throughout the neuropil, were observed most prominently in the anterior half of the DMS (Figure 1E–F). Corticostriatal axons from V1 targeted a restricted longitudinal strip of the ipsilateral DMS lining the lateral ventricle, forming two dense innervation regions: one at the more dorsal aspect of the strip and the other on its ventral side. At higher magnification, sparse axons were occasionally detected in more medial parts of dorsal striatum (data not shown). Interestingly, V1 neurons also extended axons medially and anteriorly towards the corpus callosum to innervate the contralateral V1 as well as the contralateral DMS (not shown). These axonal projections were comparatively much more sparse and weakly labeled, and were consequently not studied further. Our results therefore reveal that layer 5 pyramidal neurons in the mouse primary visual cortex extend bilateral axonal projections into the striatum.

Retrograde labeling of V1 projections to DMS

To determine whether axons originating in V1 establish synaptic contacts within the DMS, we employed a retrograde labeling method using fluorescently labeled microspheres (referred to as red retrobeads or RRBs) that are absorbed by the terminal zones of axons and transported back to the cell body [25]. Retrobeads are not transported trans-synaptically and therefore only label the soma of neurons that directly project into the injection region. Following stereotaxic injection of RRBs in the DMS (Figure 2A), we observed retrogradely labeled cells in several brain regions known to innervate dorsal striatum, including the substantia nigra pars compacta (SNc; Figure 2B) as well as the intralaminar thalamus, a region providing the densest thalamic input to the striatum [23] (Figure 2H). Importantly, we observed dense retrograde labeling within V1, confirming that V1 innervates DMS. Interestingly, microsphere-labeled cells were not found in either auditory (Figure 2B) or somatosensory cortex (Figure 2H), indicating that these cortical areas do not extend sizeable axonal arbors into the medial-most region of dorsal striatum. These observations are consistent with the notion that cortical inputs into the dorsal striatum are largely topographic, with primary auditory cortex innervating primarily the posterior portion of striatum and primary somatosensory cortex projections occupying mainly the dorsolateral aspect of anterior striatum ([6,14], Allen Institute for Brain Science Mouse Brain Connectivity Atlas [http://connectivity.brain-map.org] [26]).

The majority of pyramidal neurons in somatosensory and motor cortices that project to the striatum lie within the upper half of layer 5, although corticostriatal neurons in layer 2/3 have also been described [6,27,28]. Consistent with the distribution of corticostriatal neurons in these other brain regions, most of the retrogradely labeled cell bodies in V1 rested within layer 5 (Figure 2C): out of 1139 retrogradely-labeled cortical cells in V1 (n = 4 mice), 943 (81%) distributed to layer 5, while only 216 (19%) were in layers 2/3 (Figure 2D). On rare occasions, cells in layer 6 were observed, but they constituted a negligible percentage of the total number of corticostriatal neurons.

Because the transgenic line used in our anterograde study (Rbp4-Cre; Figure 1) restricts Cre expression to layer 5 neocortical neurons in V1, it represents an attractive tool for studying the function of visual cortex neurons projecting to striatum. To determine the fraction of corticostriatal neurons expressing Cre in these mice, we injected red fluorescent retrobeads in the DMS of Rbp4-Cre mice expressing EGFP in the soma of Cre-containing neurons (ZsGreen1, Figure 2E). Out of 331 layer 5 cells in V1 that innervate striatum (RRB-positive), the majority (345, 63%) also expressed Cre (EGFP’) and the remaining 186 cells (35%) were EGFP+ (Figure 2G). This indicates that the Rbp4-Cre line expresses Cre in two-thirds of layer 5 corticostriatal neurons, confirming the usefulness of this line in labeling striatally-projecting cortical neurons. Interestingly, the majority of neurons double-labeled for EGFP and RBB distributed to upper layer 5, suggesting that most of the DMS-projecting corticostriatal neurons labeled in Rbp4-Cre mice are infralaminacenic (IT) cortical cells [29–32].

V1 inputs into DMS drive action potentials in SPNs

To determine whether V1 corticostriatal neurons form functional synaptic connections in the striatum, we expressed channelrhodopsin (ChR2-mCherry) in V1 of Rbp4-Cre;Drd2-EGFP mice and obtained recordings from direct- (dSPN) and indirect-pathway striatal projection neurons (iSPNs) in coronal sections of DMS (Figure 3A). Stimulation of V1 axons using 1 ms light flashes reliably evoked action potentials in SPNs in both cell-attached (n = 4; Figure 3B) and whole-cell current-clamp recording configurations (n = 3; Figure 3C), indicating that corticostriatal axons originating in V1 are functional and strong enough to reliably depolarize SPNs above spike threshold. We used whole-cell voltage-clamp recordings from SPNs to characterize the synaptic conductances evoked by optogenetic activation of V1 axons. In SPNs clamped at −70 mV (the Cl− reversal potential), ChR2 stimulation reliably evoked large excitatory postsynaptic currents (EPSCs) that were blocked by gabazine (mean amplitude: 746 ± 182 pA, n = 9; P < 0.01 vs. baseline, Mann-Whitney U Test; Figure 3D, G), indicating that these corticostriatal synaptic events result from monosynaptic activation of ionotropic glutamate receptors. As anticipated, the GABA A receptor antagonist gabazine did not affect EPSCs (mean amplitude: −283 ± 92 pA, n = 3; P = 0.5 vs. baseline; Mann-Whitney U Test; Figure 3G).

Stimulation of corticostriatal afferents in slice potently engages feed-forward inhibition, which is mediated by striatal fast spiking interneurons and SPN collaterals [33]. To determine if corticostriatal afferents arising in V1 similarly recruit local inhibitory circuits in striatum, we recorded light-evoked responses in SPNs clamped at 0 mV (the reversal potential of ionotropic glutamate receptors) to reveal inhibitory postsynaptic currents (IPSCs). Under these conditions, we consistently observed large IPSCs (mean amplitude: 746 ± 182 pA, n = 9) that were blocked by gabazine (mean amplitude: 4 ± 2 pA, n = 3; P < 0.01 vs. baseline, Mann-Whitney U Test), indicating that corticostriatal stimulation triggers synaptic release of GABA (Figures 3D,G). Consistent with a disynaptic origin, IPSCs displayed synaptic latencies twice as long as those of EPSCs (0.3 ± 0.4 ms, n = 9; P < 0.001, Mann Whitney U Test; Figure 3F) and were eliminated in the presence
Discussion

The dorsal striatum plays a crucial role in motor planning, motivation, as well as procedural and reward learning behaviors [34–37]. As one of the major sources of excitatory inputs into the dorsal striatum, cortex provides an important signal that shapes striatal function and subsequent behavioral outcomes [38,39]. Our results reveal that, in addition to well-established somatosensory, auditory and motor corticostriatal afferents, the striatum of mice also receives strong synaptic inputs from primary visual cortex in its dorsal-medial quadrant. This finding therefore suggests that early visual processing may play an important role in visually guided striatal-based behaviors.

Figure 1. Layer 5 neurons in primary visual cortex project to dorsomedial striatum. A–F. Representative serial coronal sections from the brain of a Rbp4-Cre mouse virally injected in the primary visual cortex (V1) with an ARV encoding Cre-dependent EGFP to label the cell bodies and axonal processes of neurons in layer 5 (A), EGFP-labeled axons leave V1 in through the external capsule (B) and innervate various structures including several layers of the superior colliculus (SC; B) and dorsal lateral geniculate nucleus (DLG; C). V1 axons enter the posterior tail of the striatum (caudate/putamen, CPU; D) and course throughout the length of the striatum (E) before innervating the anterior dorsomedial quadrant (F). Left, entire hemisphere stained with DAPI to highlight different brain structures overlaid with the EGFP fluorescence. Middle, detailed view of regions outlined in white in the corresponding left panel. Right, corresponding images from Paxinos Mouse Brain Atlas highlighting the putative structures where EGFP fluorescence is detected. Similar results were observed eight different mouse brains. M1; primary motor cortex. S1BF; primary somatosensory cortex barrel field.

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Figure 2. Retrograde labeling of V1 neurons innervating the DMS. A. Coronal striatal section showing the site of red retrobead (RRB) injection into the dorsomedial striatum (DMS). The section is counterstained with DAPI. B. Coronal brain section of V1 containing RBBS retrogradely transported from the dorsomedial striatum. Secondary visual cortex (V2ML and V2L) and secondary auditory cortex (AuD), but not Au1 (primary auditory cortex) were also labeled. As expected, retrobeads were also detected in putative dopaminergic neurons within the substantia nigra pars compacta (SNc). C. Detailed view of V1 showing the relative distribution of retrobead-labeled cells bodies across cortical layers. D. Distribution of retrobead-labeled neurons in V1 across cortical layers. The majority (81%) of labeled cells are situated within layer 5, while only 19% distribute to layers 2/3. E. Representative coronal section from a Rbp4-Cre;ZsGreen1 brain showing EGFP fluorescence concentrated in the cell bodies of Cre-containing cortical neurons in layer 5. The section is counterstained with DAPI. F. Detailed view of V1 in a Rbp4-Cre;ZsGreen1 brain injected with RBBS in the DMS illustrating the extensive overlap between retrobead- and Cre-containing (EGFP-positive) cells in upper layer 5. G. Percentage of all retrobead-labeled layer 5 neurons that also contain Cre (RRB/EGFP+; 65%, shown in yellow) vs. cells that are only RBB+ (35%, red). H. Coronal brain section showing red retrobead labeled cells in thalamus. ILT: Intralaminar thalamic nuclei. Scale: 1 mm for panels A, B, E and H; 100 μm for panels C and F.

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performed across a wide variety of species, including monkeys, rats, rabbits, and hamsters. Second, some labeling methods might have lacked the sensitivity required to reveal this projection. Third, the use of focal cortical lesions for mapping corticostriatal inputs might have presented difficulties in isolating V1 from neighboring regions. In this study, we employed sensitive labeling, imaging and optogenetic methods to conclusively demonstrate that V1 establishes a direct and functionally strong axonal projection into the DMS in mice. Using viral expression of EGFP in V1 and injection of retrogradely transported beads in the DMS, we confirmed that V1 neurons innervate a longitudinal strip of the DMS that lines the ventricle. In agreement with the distribution of corticostriatal afferents in other cortical regions, we found that V1 neurons projecting to the DMS lie in both layer 5 and layers 2/3, with the overwhelming majority of them in upper layer 5. Moreover, we used optogenetics and electrophysiological recordings from SPNs to show that the axons of neocortical L5 neurons in V1 functionally innervate striatum. Our anatomical findings are supported by studies in rats [5,6,14], as well as in mice [Allen Institute for Brain Science Mouse Brain Connectivity Atlas, (26)], which reported a similar projection from V1 that mainly terminates ipsilaterally in a longitudinal strip of the DMS.

Two broad groups of corticostriatal afferents can be distinguished anatomically: intra-telecephalic (IT) neurons mainly distribute to upper layer 5 and innervate the striatum bilaterally, while pyramidal tract (PT) neurons rest in lower layer 5 and extend collaterals ipsilaterally into the striatum, thalamus and midbrain on their way to the brainstem and spinal cord [29–32]. Although we did not observe V1 axons in the ponds or pyramidal tract, EGFP

Studies dating back to the 1960s have attempted to map corticostriatal inputs [1–4,6,12]. Many of these and subsequent studies investigated whether V1 provides a direct synaptic input into the striatum, but the existence of this connection has remained controversial. Several experimental factors might have contributed to conflicting reports. First, these studies were
and pharmacogenetic manipulations of these neurons on behavior. Nevertheless, the \( Rbp4-Cre \) transgenic line should prove useful in studying general principles of corticostriatal transmission and circuitry.

Together, our findings indicate that corticostriatal inputs originating in primary visual cortex can efficiently recruit neuronal circuits in defined regions of the striatum, suggesting that early visual processing might contribute to corticostriatal synaptic plasticity as well as visually guided motor behaviors.

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Author Contributions

Conceived and designed the experiments: LK NT BS. Performed the experiments: LK NT. Analyzed the data: LK NT. Contributed reagents/materials/analysis tools: LK NT. Contributed to the writing of the manuscript: LK NT BS.

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