An Overview of the Striatum – from motor skills to cue-reward association

1. Introduction

To adapt behavior to a changing environment, the brain must maintain representations of where, when, and how to acquire the components necessary for survival. These representations are built from experience with sensory predictors in the environment and are constantly updated to facilitate survival. In the psychological literature, a reinforcer is an event that increases the future likelihood of behavior that preceded it. In the neuroscience literature, the term reward has come to represent discrete tangible reinforcers, including food, drugs, and other forms of pleasure. While critical to action selection and the attainment of biological needs, the same trajectory from sensory predictor to reward achievement also underlies maladaptive behaviors and pathologies, including addiction, schizophrenia, and PTSD. While important advances have been made in the identification of neural substrates of reward-relevant sensory information, little is known about the integration of these signals to form complex actions and how they are transformed into habits and/or skills.

Abuse of a number of psychoactive substances can eventually control an individual behavior by producing dependence and/or addiction. After a 170% rise in drug abuse and suicide (2009-2018), 2020 saw record-high overdoses, giving 2020 the title of “The deadliest year in drug history” (Source: addictioncenter.com, 2020). In the United States in 2024, we saw the steepest decline in drug overdose seen in decades. With over 100,000 drug overdose deaths still occurring every year, experts warn that the problem is still far from over and that this decline, while encouraging, should not downplay the significant

need for continued research on the brain circuits involved. Scientists continue to encourage more focus and funding on addiction treatment and harm reduction strategies at both the national and state levels, specifically in underserved communities where overdose rates are still particularly high (addictioncenter.com 2024).

There is increasing evidence suggesting that drug addiction represents a conditioning phenomenon that is largely dependent on associations between drug effect and environmental cues. An appropriate balance between neurotransmitters is necessary for the proper evaluation of stimuli associated with rewarding of aversive events (Sulzer, 2011; Lüscher, 2013; Lipton et al., 2019). In this pre-paper and presentation, I will discuss three independent projects—each with distinct objectives—that collectively reveal how striatal functional heterogeneity shapes motivated behavior in health and disease.

2. The striatum

The striatum, a key component of the basal ganglia, is central to learning, motivation, and action selection. It is traditionally divided into three functionally distinct regions: the dorsal striatum (DS), ventral striatum (VS), and tail of the striatum (TS). Each of these regions contributes to different aspects of behavior, yet their functions are deeply interconnected, forming a dynamic system that adapts to environmental demands.

- The DS is critical for motor skill acquisition and habit formation. It is generally thought that it is mostly involved in movement, particularly automatized fine skills and micromovements embedded in an action (Yin et al., 2009; Thorn et al., 2010). For example, learning to ride a bicycle or play a musical instrument relies on the DS's ability to chunk actions into seamless sequences (Graybiel, 1998). Nevertheless, the DS

integrates sensory and motor information to refine movements, transitioning goal-directed actions into automatic skills (Yin et al., 2009; Cataldi et al., 2021). Dysfunction here is implicated in Parkinson's disease, where motor initiation and skill learning are impaired (DeLong et al., 1984; Marinelli et al., 2017).

- The VS is the hub of reward-based learning and motivation. It receives projection from limbic cortices and amygdala and is more broadly involved in goal-related movements, a process by which the animal encodes values to the movement performance (O'Doherty, 2004; Liljeholm & O'Doherty, 2012). It associates cues with outcomes, driving behaviors like seeking food or addictive substances (Liljeholm & O'Doherty, 2012; Sulzer, 2011).
- The TS, a less-studied region, filters sensory stimuli and is implicated in safety and fear learning (Menegas et al., 2017; Valjent & Gangarossa, 2021). It helps distinguish relevant from irrelevant cues, a process disrupted in PTSD, where neutral stimuli trigger trauma responses (van Rooij & Jovanovic, 2019).

While these regions are often studied in isolation, their collaboration is essential for adaptive behavior. For instance, the VS may initiate reward-seeking actions, the DS refines them into habits, and the TS suppresses distractions. My work explores these subregions separately—through treadmill running (dorsal), operant conditioning (ventral), and fear/safety paradigms (TS)—but the findings collectively reveal how the striatum balances learning, action, and environmental interaction. This integrated perspective is vital for understanding disorders like addiction, Parkinson's, and PTSD, where striatal circuits are disrupted.

The vast majority of the striatum is composed of spiny projections neurons (SPNs), accounting for up to 95% of all striatal neurons in rodents (Kemp & Powell, 1971; DiFiglia et al., 1976; Matamales et al, 2009) and approximately 75% in primates (Fox et al., 1971; Graveland & Difiglia, 1985). Striatal SPNs form predominantly two distinct neural pathways. The direct pathway consists of GABAergic neurons D1 dopamine receptors (D1-SPNs, Gerfen et al., 1990; Kawaguchi et al., 1990; Gangarossa et al., 2013). The indirect pathway neurons are also GABAergic but express D2 dopamine receptors (D2-SPNs, Gangarossa et al., 2013; Gerfen et al., 1990; Kawaguchi et al., 1990).

The striatum receives dopaminergic innervations from the midbrain, as well as excitatory innervation from cortex, hippocampus, and amygdala, regions shown to be important substrates for reward-related memory and sensory perception. Together, this collection of inputs as well as the striatum's role in action selection and motor control make the striatal SPN a key integrative locus for reinforcement (Cataldi et al., 2021).

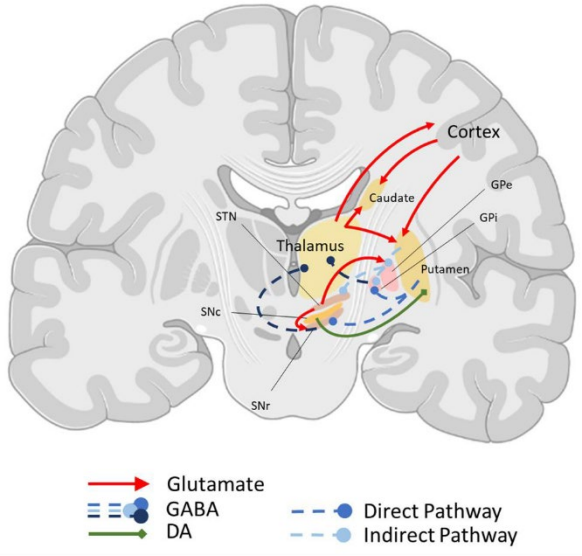

Fig. 1. Simplified schematic of the basal ganglia circuit involved in motor learning in the human brain. Connections to and from the dorsal portion of the striatum are indicated. The caudate and putamen receive glutamatergic projections (red solid arrows) from cortex and thalamus, while sending GABAergic projections (blue dashed lines) to downstream structures. The direct pathway is composed of GABAergic D1R-expressing SPNs from the striatum to the GPi and SNr. The inhibition of these structures by the direct pathway, and therefore disinhibition of the thalamus, promotes movement and is classically referred to as the 'go' pathway. In contrast, the indirect pathway via D2R-expressing GABAergic projections inhibits the GPe, which in turn causes disinhibition of the GPi and STN, leading to reduced activity of the thalamus and is known as the 'no-go' pathway. dopaminergic projections from the SNc (green solid line) modulates activity of both caudate and putamen. Location and size of each region are altered for presentation purposes. Figure created with BioRender.com. GPe, globus pallidus external; GPi, globus pallidus internal; SNr, substantia nigra pars reticulata; SNc, substantia nigra pars compacta; STN, subthalamic nucleus; DA, dopamine; SPNs, spiny projection neurons.

2.1 Dopamine and the striatum in health and disease

The striatum receives dense innervation by dopamine neurons from the midbrain, which have long been shown to encode reward prediction error and salience. As animals engage with external stimuli, they associate cues with specific rewarding or aversive events. Dopamine activity eventually shifts from responding to the rewarding or aversive event to the presence of the associated cue. Most if not all drugs of abuse are modulators of dopamine signaling. In laboratory experiments, animals have been shown to self-administer drugs when they are paired with specific cues, a method widely used to model addiction in animals (Olds & Milner, 1954). These studies demonstrate how strongly the brain ties actions to rewards, particularly in addiction, where the anticipation of a drug-induced reward becomes encoded in the brain's reward pathways (Koob & Le Moal, 2008). Addiction fundamentally alters how the brain responds to cues. The repeated use of addictive substances leads to changes in the striatum (Konova et al., 2013). Over time, the brain begins to associate cues related to drug use (such as drug tools or places where drugs were taken) with the pleasure derived from the drug itself, leading to what is called cue-induced craving (Konova et al., 2013). This association can trigger compulsive behaviors where the mere sight or sound of a related cue provokes drug-seeking, even in the absence of the drug itself.

Similarly, in schizophrenia, dopamine dysregulation plays a crucial role in abnormal behavior and cognition, particularly in how individuals process environmental cues. In schizophrenia, hyperactivity of the dopamine system, especially in the associative striatum, leads to impaired response inhibition and difficulties in distinguishing between

relevant and irrelevant stimuli (Frankle et al., 2022). This can result in auditory hallucinations where the brain misinterprets neutral sounds or thoughts as real, external voices, a symptom driven by overactive dopamine signaling (Howes & Falkenberg, 2011). These hallucinations demonstrate how excessive dopamine activity can amplify the brain's response to environmental cues, leading to maladaptive behavior similar to that seen in addiction. In PTSD, dopamine is also involved in the heightened sensitivity to environmental cues associated with trauma. Patients with PTSD may experience intense reactions to seemingly innocuous stimuli, like sounds or smells, that remind them of the traumatic event (van Rooij & Jovanovic, 2019). These conditioned responses are difficult to extinguish because the dopamine system reinforces the association between the cue and the fear response. As in addiction, the brain becomes wired to react strongly to specific stimuli, even when the actual threat is no longer present.

Overall, dopamine's involvement in cue-related learning is fundamental in both normal and pathological behaviors. Whether it's the cravings triggered by drug-related cues in addiction, the hallucinations provoked by hyperactive dopamine signaling in schizophrenia, or the fear responses in PTSD, understanding these mechanisms helps to illuminate the neural basis of these complex disorders.

Dopamine modulates the activity of SPNs (Gerfen & Surmeier, 2011), but much less is known about the specific function of SPNs, particularly in relation to how they contribute to complex behaviors such as reward-seeking and habit formation. Recent studies suggest that SPNs are not merely passive recipients of dopamine signals but actively shape the brain's response to rewards and actions. Understanding the exact role of these neurons in

processes such as reinforcement learning, addiction, and other mental health disorders could provide new insights into how the brain encodes habits and maladaptive behaviors.

3. Our Experimental Approach

A central goal of systems neuroscience is to predict behavior based on neural dynamics. The recent development of neuron type-specific genetics and *in vivo* imaging techniques now allows us to probe the activity dynamics of these intermixed populations during naturalistic behaviors. Through fiber photometry and genetic calcium sensors we can record D1-SPN bulk dynamics during behavior that I use to determine the predictivity of neural signatures on behavioral performance.

3.1 Animal model

Mice are a crucial model in neuroscience research due to their genetic and behavioral similarity to humans and the ease with which their genetics can be manipulated. Moreover, mouse models allow for longitudinal studies, enabling researchers to track changes in behavior and brain function over time, which is critical in understanding how these circuits adapt or become dysregulated in conditions like addiction or schizophrenia (Crawley, 2007). D1-Cre mice are genetically modified to express Cre-recombinase under the control of the D1 dopamine receptor gene, allowing for targeted expression of proteins, like calcium sensors, specifically in D1-SPNs. In our experiments, we use the calcium sensor GCaMP6f. This enables us to measure the activity of these neurons with high precision during complex behaviors.

Since mice are nocturnal animals, they are kept on a reversed light cycle, where lights are off during the day to align their awake phase with daytime experimental

conditions. Experiments are conducted in the dark to minimize stress and ensure natural, relaxed behavior.

3.2 Fiber Photometry: Measuring Brain Activity

To measure the activity of D1-SPNs that express the GCaMP6f protein, we use a technique called fiber photometry. This method allows us to track changes in calcium levels in the neurons, which is an indicator of their activity. The mouse has a tiny fiber optic cable implanted in its brain in the region of interest. This cable shines a specific light into the brain, and when the neurons are active, calcium flows into them and the GCaMP protein glows. A detector captures this glow, and the amount of light emitted tells us how active the neurons are. This gives us a way to directly measure neural activity in real time, allowing us to see when certain neurons are involved in actions like pressing a lever or receiving a reward.

3.3 DeepLabCut: Tracking Movements with Precision

One of the challenges in studying behavior is tracking the animal's movements with high precision. For this, we use a tool called DeepLabCut (Mathis et al., 2018), which uses advanced machine learning to analyze videos and detect specific body parts, such as the mouse's paws or head, as they move around. DeepLabCut allows us to track these movements in fine detail. In our experiments, this is particularly useful for analyzing how mice body movement changes over time as they learn the task. It also helps us align their movements with the neural data we collect through fiber photometry, giving us a complete picture of how behavior and brain activity are connected.

3.4 The Role of Python Coding

A significant part of our research involves analyzing large amounts of data, which comes from both the behavior of the mice and their brain activity. To make sense of all this data, we write custom Python code that processes it automatically. This code allows us to: synchronize the behavioral data with the neural activity data, identify patterns in the data, such as how quickly the mouse learns the task or how its brain responds to different phases of the task, and create visual representations of the data so that we can easily interpret the results. Python is a flexible and powerful programming language, which is why it's widely used in both research and industry for tasks like data analysis and machine learning.

3.5 Our Experimental Setup: Custom-Made Equipment

To observe learning in real time, we designed and built all of the equipment used for behavioral experiments.

- To study how the dorsal striatum refines motor skills, we developed a custom motorized treadmill that precisely controls locomotion while monitoring neural activity. Mice were placed on a 1-meter-long clear belt moved by acrylic wheels operated via an Arduino, which is a small programmable microcontroller. The setup included a mirrored floor for simultaneous video tracking from the side and from the bottom of the treadmill. DeepLabCut and Python analysis was used to quantify gait and limb coordination. During 12 days of training, mice improved their coordination while running, transitioning from hesitant steps to fluid running—a hallmark of dorsal striatum-dependent skill consolidation (Yin et al., 2009; Cataldi et al., 2022).

- For analysis of ventral striatum activity, we built a custom operant box that allows mice to press levers and receive rewards while we monitor their brain activity. The box is made of a type of acrylic plexiglass that allows us to track the mice's movements even in the dark using infrared light and an infrared camera. We control various elements of the experiment—like the lights, sound cues, and levers—using an Arduino. In this case the Arduino is like the brain of the box. It allows us to precisely control when things happen, such as turning on the sound or dispensing the reward. It is programmed to execute these tasks automatically using custom codes, in the Arduino coding language. The Arduino code also provides inputs at each time the mouse is licking from the spout to acquire the reward or pressing the lever to access the spout. The code records each individual lick and the actual physical force applied to the lever. I developed Python codes to analyze the information provided and establish the engagement of the mouse with the different components of the test and the level of learning over multiple trials (Cataldi et al., 2024). I will expand more on operant conditioning in the next paragraph.

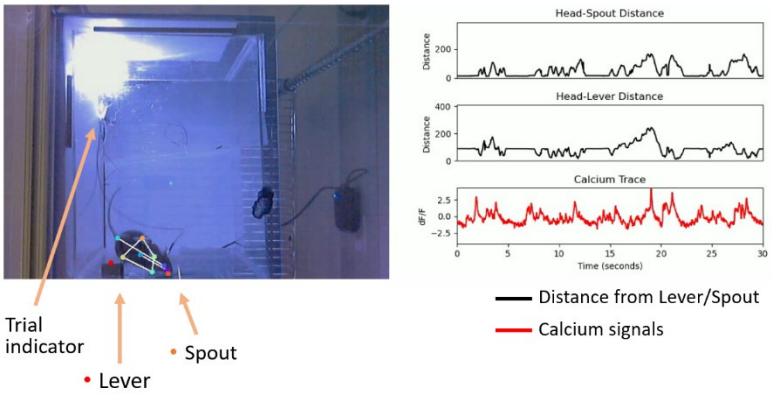

Figure 2. Example of analyzed behavior video. Videos are recorded in the dark, using an infrared camera placed below the operant box. Left. In this picture the mouse is seen licking from the spout at the center-bottom of the image. Using DeepLabCut, the lever is labeled with a red dot, while the spout is labeled in orange. The infrared LED light at the top right corner of the picture indicates that the mouse has pressed the lever and the trial has started. Right. At the top is a sample trace (black) indicating the distance from the mouse head to the spout. The middle plot (also black) shows the distance from the head of the mouse to the lever. The bottom trace (red) corresponds to the calcium signals as recorded using fiber photometry while the mouse is performing the experiment.

- To probe the tail of the striatum's (TS) role in threat processing, we used a three-day fear/safety conditioning protocol. Mice were exposed to tones (conditioned stimuli, CS) paired with mild foot shocks (unconditioned stimuli, US) in a controlled chamber. In the

fear conditioning group, shocks happened simultaneously with tones, creating fear associations. In the safety conditioning group, tones and shocks were explicitly unpaired, teaching mice that tones predict safety. A third control group heard tones alone. During recall, threat-conditioned mice froze to tones in a novel context (measuring generalized fear), while safety-conditioned mice suppressed freezing in the original context—a TS-dependent process (manuscript submitted).

3.4 Classical and Operant Conditioning: How We Learn

Because the main goal of my projects is to understand the striatum in the context of addiction, I will expand here on the concept of classical and operant conditioning. Classical conditioning is a simple form of learning. It happens when we associate two things: one that naturally triggers a response (like food) and a neutral signal (like a sound). Classical conditioning is used to train people or animals to respond automatically to certain triggers. The most famous example -- Pavlov's dogs. Ivan Pavlov was a Russian psychologist. He observed that dogs salivated when food was put in front of them. That's natural, or what's called an unconditioned response. But then Pavlov noticed that the dogs began to salivate shortly before their food arrived, possibly because the sound of the food cart triggered their anticipation of mealtime. In his experiment, at mealtimes, he sounded a bell shortly before the food arrived. Eventually, the dogs began to salivate when they heard the bell. That was a trained, or **conditioned**, response to the sound of the bell.

Operant conditioning is different because it involves learning from the consequences of our actions. If you press a button and receive a reward, you'll learn to

press the button again when you want that reward. Operant conditioning involves active participation from the subject—in our case, mice pressing a lever to receive a reward. Through operant conditioning, behavior that is rewarded is likely to be repeated and behavior that is punished is prone to happen less.

4. Conclusion

The striatum's functional diversity—spanning motor skill acquisition, reward learning, and threat detection—highlights its role as a neural orchestrator of adaptive behavior. By studying its subregions in distinct behavioral contexts (dorsal: treadmill running; ventral: operant conditioning; TS: fear/safety learning), we uncover how specialized circuits collaborate to guide actions. While each project addressed unique hypotheses, together they emphasize that striatal function depends on **experimental design**, **cell-type specificity**, and **environmental cues**. For example, dopamine's role shifts from driving reward-seeking in the VS to optimizing movement in the DS, while the TS modulates responses to threats. It is unclear if similar patterns are seen in striatal neurons.

In summary, my research uses a variety of advanced tools and techniques to study how the brain learns complex behaviors. By combining behavior with fiber photometry, DeepLabCut, and Python programming, I can precisely measure how neurons activity changes during learning. The ultimate goal is to gain a better understanding of how reward-based learning works, which could provide insights into conditions like addiction, as well as Parkinson's Disease, Schizophrenia, PTSD, and other neurological disorders where these processes go awry.

References:

- Cataldi, S., Stanley, A. T., Miniaci, M. C., & Sulzer, D. (2021). Interpreting the role of the striatum during multiple phases of motor learning. The FEBS Journal, febs.15908. https://doi.org/10.1111/febs.15908
- Crawley, J. N. (2007). What's Wrong With My Mouse? What's Wrong With My Mouse? https://doi.org/10.1002/0470119055
- DeLong MR, Alexander GE, Georgopoulos AP, Crutcher MD, Mitchell SJ & Richardson RT (1984) Role of basal ganglia in limb movements. Hum Neurobiol 2, 235–244.
- DiFiglia, M., Pasik, P., & Pasik, T. (1976). A Golgi study of neuronal types in the neostriatum of monkeys. Brain Research, 114(2), 245–256. https://doi.org/10.1016/0006-8993(76)90669-7
- Fox, C. A., Andrade, A. N., Hillman, D. E., & Schwyn, R. C. (1971). The spiny neurons in the primate striatum: a Golgi and electron microscopic study. Journal Fur Hirnforschung, 13(3), 181–201. https://europepmc.org/article/med/5005223
- Frankle, W. G., Himes, M., Mason, N. S., Mathis, C. A., & Narendran, R. (2022). Prefrontal and Striatal Dopamine Release Are Inversely Correlated in Schizophrenia. Biological Psychiatry, 92(10), 791–799. https://doi.org/10.1016/J.BIOPSYCH.2022.05.009
- Gangarossa, G., Espallergues, J., Mailly, P., De Bundel, D., de Kerchove d'Exaerde, A., Hervé, D., Girault, J. A., Valjent, E., & Krieger, P. (2013). Spatial distribution of D1R- and D2R-expressing medium-sized spiny neurons differs along the rostro-caudal axis of the mouse dorsal striatum. Frontiers in Neural Circuits, 7(JUL). https://doi.org/10.3389/fncir.2013.00124
- Gerfen, C. R., Engber, T. M., Mahan, L. C., Susel, Z., Chase, T. N., Monsma, F. J., & Sibley, D. R. (1990). D1 and D2 dopamine receptor-regulated gene expression of striatonigral and striatopallidal neurons. Science, 250(4986), 1429–1432. https://doi.org/10.1126/science.2147780
- Gerfen CR, Surmeier DJ. Modulation of striatal projection systems by dopamine. Annu Rev Neurosci. 2011;34:441-66. doi: 10.1146/annurev-neuro-061010-113641. PMID: 21469956; PMCID: PMC3487690.
- Graveland, G. A., & Difiglia, M. (1985). The frequency and distribution of medium-sized neurons with indented nuclei in the primate and rodent neostriatum. Brain Research, 327(1–2), 307–311. https://doi.org/10.1016/0006-8993(85)91524-0
- Graybiel AM. The basal ganglia and chunking of action repertoires. Neurobiol Learn Mem. 1998 Jul-Sep;70(1-2):119-36. doi: 10.1006/nlme.1998.3843. PMID: 9753592.
- Howes, O. D., & Falkenberg, I. (2011). Early detection and intervention in bipolar affective disorder: targeting the development of the disorder. Current Psychiatry Reports, 13(6), 493–499. https://doi.org/10.1007/S11920-011-0229-8
- Kawaguchi, Y., Wilson, C. J., & Emson, P. C. (1990). Projection subtypes of rat neostriatal matrix cells revealed by intracellular injection of biocytin. Journal of Neuroscience, 10(10), 3421–3438. https://doi.org/10.1523/jneurosci.10-10-03421.1990
- Kemp, J. M., & Powell, T. P. (1971). The structure of the caudate nucleus of the cat: light and electron microscopy. Philosophical Transactions of the Royal Society of London. B, Biological Sciences, 262(845), 383–401. https://doi.org/10.1098/rstb.1971.0102
- Konova, A. B., Moeller, S. J., & Goldstein, R. Z. (2013). Common and distinct neural targets of treatment: changing brain function in substance addiction. Neuroscience and Biobehavioral Reviews, 37(10 Pt 2), 2806–2817. https://doi.org/10.1016/J.NEUBIOREV.2013.10.002
- Koob, G. F., & Le Moal, M. (2008). Addiction and the brain antireward system. Annual Review of Psychology, 59, 29–53. https://doi.org/10.1146/ANNUREV.PSYCH.59.103006.093548
- Liljeholm, M., & O'Doherty, J. P. (2012). contributions of the striatum to learning, motivation, and performance: An associative account. In Trends in Cognitive Sciences (Vol. 16, Issue 9, pp. 467–475). Elsevier Ltd. https://doi.org/10.1016/j.tics.2012.07.007

- Lipton, D. M., Gonzales, B. J., & Citri, A. (2019). Dorsal striatal circuits for habits, compulsions and addictions. In Frontiers in Systems Neuroscience (Vol. 13, p. 28). Frontiers Media S.A. https://doi.org/10.3389/fnsys.2019.00028
- Lüscher, C. (2013). Cocaine-evoked synaptic plasticity of excitatory transmission in the ventral tegmental area. Cold Spring Harbor Perspectives in Medicine, 3(5). https://doi.org/10.1101/cshperspect.a012013
- Marinelli L, Quartarone A, Hallett M, Frazzitta G & Ghilardi MF (2017) The many facets of motor learning and their relevance for Parkinson's disease. Clin Neurophysiol 128, 1127–1141.
- Matamales M, Bertran-Gonzalez J, Salomon L, Degos B, Deniau JM, Valjent E, Hervé D & Girault JA (2009) Striatal medium-sized spiny neurons: identification by nuclear staining and study of neuronal subpopulations in BAC transgenic mice. PLoS One 4, e4770.
- Mathis, A., Mamidanna, P., Cury, K. M., Abe, T., Murthy, V. N., Mathis, M. W., & Bethge, M. (2018). DeepLabCut: markerless pose estimation of user-defined body parts with deep learning. Nature Neuroscience, 21(9), 1281–1289. https://doi.org/10.1038/s41593-018-0209-y
- Menegas W, Babayan BM, Uchida N & Watabe-Uchida M (2017) Opposite initialization to novel cues in dopamine signaling in ventral and posterior striatum in mice. Elife 6, e21886.
- O'Doherty, J. P. (2004). Reward representations and reward-related learning in the human brain: Insights from neuroimaging. In Current Opinion in Neurobiology (Vol. 14, Issue 6, pp. 769–776). Curr Opin Neurobiol. https://doi.org/10.1016/j.conb.2004.10.016
- Olds, J., & Milner, P. (1954). Positive reinforcement produced by electrical stimulation of septal area and other regions of rat brain. Journal of Comparative and Physiological Psychology, 47(6), 419–427.
- Sulzer, D. (2011). How Addictive Drugs Disrupt Presynaptic Dopamine Neurotransmission. In Neuron (Vol. 69, Issue 4, pp. 628–649). Neuron. https://doi.org/10.1016/j.neuron.2011.02.010
- Thorn, C. A., Atallah, H., Howe, M., & Graybiel, A. M. (2010). Differential Dynamics of Activity Changes in Dorsolateral and Dorsomedial Striatal Loops during Learning. Neuron, 66(5), 781–795. https://doi.org/10.1016/j.neuron.2010.04.036
- Valjent E & Gangarossa G (2021) Trends in the tail of the striatum: from anatomy to connectivity and function. Trends Neurosci 44, 203–214.
- van Rooij, S. J. H., & Jovanovic, T. (2019). Impaired inhibition as an intermediate phenotype for PTSD risk and treatment response. Progress in Neuro-Psychopharmacology and Biological Psychiatry, 89, 435–445. https://doi.org/10.1016/J.PNPBP.2018.10.014
- Wall, N. R., DeLaParra, M., Callaway, E. M., & Kreitzer, A. C. (2013). Differential innervation of direct- and indirect-pathway striatal projection neurons. Neuron, 79(2), 347–360. https://doi.org/10.1016/j.neuron.2013.05.014
- Yin, H. H., Mulcare, S. P., Hilário, M. R. F., Clouse, E., Holloway, T., Davis, M. I., Hansson, A. C., Lovinger, D. M., & Costa, R. M. (2009). Dynamic reorganization of striatal circuits during the acquisition and consolidation of a skill. Nature Neuroscience, 12(3), 333–341. https://doi.org/10.1038/nn.2261