Complementary and Integrative Health Intervention Use in Binge Eating Disorder: A Narrative Review

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**SUPPLEMENTARY MATERIAL**

## Supplemental Material S1

Feeding and eating behaviors are thought to involve complex interactions between physiological and neural systems associated with hunger/satiety mechanisms, ingestive (homeostatic/appetitive and consummatory) mechanisms, and hedonic/reward/reinforcement-based mechanisms (96, 97) (96-99) (**Figure S1**). Implicated neuralcircuitry are illustrated in [Figures 1](#_Figure_1) and [2](#_Figure__2) as well as in the **Supplementary Figures S1–S16**, and include (96, 98-114):

* The **mesostriatal dopamine circuit/pathway** (AKA the **ventral pathway**) is primarily associated with reward salience and motivational drive (**Supplementary Figure S2**). In this circuit, dopaminergic neurons in the medial **ventral tegmental area (VTA)** innervate the **ventral striatum** (mainly the medial **nucleus accumbens shell (NAcS),** which sends GABAergic and dopaminergic projections to the **ventral pallidum (VP)** in turn, conveying the reward salience of pleasurable experiences and substances (e.g., food, drugs, etc.))(115, 116). The VP integrates accumbal inputs with dopaminergic inputs directly from the VTA to influence consummatory behaviors, including consumption, cue-induced feeding, taste reactivity, and food preference (116). Impaired inhibition of the mesolimbic portion of the mesostriatal dopamine pathway is also associated with impulsivity ([Figure 2](#_Figure__2); **Supplementary Figure S3**)(119, 120).
* The **nigrostriatal dopamine pathway** (AKA the **dorsal pathway**) is associated primarily with reward sensitization and compulsivity (**Supplementary Figure S4**). Dopaminergic neurons in the **VTA** innervate the **dorsomedial and dorsolateral striatum**, including the **caudate nucleus** and **putamen**, which are essential for orchestrating goal-directed and habitual decision-making (121-123). The dorsal pathway largely involves a circuit by which dorsal striatal projections innervate the **paraventricular nucleus of the thalamus (PVN/PVT)**, which integrates homeostatic and hedonic feeding signals with physiological and environmental stress signals, anticipatory feeding needs, and cognitive inputs to regulate food-seeking and consumption (124). The thalamus (innervates the **orbitofrontal cortex (OFC)** in turn (also thought to be involved in reward and punishment processing and reward-based decision-making (125-129)), which feeds back onto the striatum (130). Additionally, preclinical findings demonstrate that dopamine sensitization in the dorsal striatum accelerates the development of habit formation from previously goal-directed behaviors (123, 131-133), possibly contributing to compulsivity ([Figure 2](#_Figure__2); **Supplementary Figure S5)**(357-359).
* The **corticolimbic system** also contributes to reward and motivational processes, and includes the amygdala, hippocampus, and prefrontal cortex (PFC) (**Supplementary Figures S6A–C**)(134). The VTA sends dopaminergic neurons to the **amygdala** and **hippocampus**, which are thought to be involved in learning and remembering reward cues and innervate the NAc as part of the mesolimbic pathway. The **orbitofrontal cortex (OCF)** in the **prefrontal cortex (PFC)** is also thought to be involved in reward and punishment processing and reward-based decision-making (125-129). It receives and integrates various sensory inputs (e.g., taste, smell, touch, vision, sound) and learns (and reverses) associations between stimuli and their outcomes (e.g., foods and their salient/rewarding properties/pleasantness), adapting to valuation changes as needed (e.g., changes in food value), thus contributing to reward-based decision-making processes that guide behavior (125-129). The **insula** and **thalamus** (described above), **hypothalamus**, and **brainstem/pons** are also thought to be part of the corticolimbic circuitry that drives eating and binge eating behaviors (98).
* The **frontoparietal control system** (also called the **central executive network (CEN)** and the **executive control network** **(ECN)**) are generally associated with executive functioning, decision-making, coordinating goal-driven behaviors, self-regulation, regulating appetitive responding, and regulating the hedonic/reward/reinforcement-based corticolimbic and mesostriatal dopamine circuits **(Supplementary Figures S7)** (96, 98-107, 135-139). This network/system primarily includes the **dorsolateral PFC** (especially the **OFC** and **middle frontal gyrus**), **anterior cingulate cortex (ACC)**, and **posterior parietal cortex (PCC)** (around the intraparietal sulcus), as well as the **middle cingulate gyrus (mCG)**, **dorsomedial thalamus**, **caudate nucleus head**, **dorsal precuneus**, and **posterior inferior temporal lobe** (138, 139). Specifically, the OFC sends projections and reward information to the ACC, which integrates spatial and action-related information from the parietal cortical areas, thus connecting rewards to actions and reward-cues, while the **PCC** projects to the hippocampal system, thus storing reward memories (140).
* The **basal ganglia network (BGN)** (also known as the **reward network**) is a complex network implicated in drive for reward-based eating and sensitivity to rewards and rewarding foods (**Supplemental Figure 8, Supplemental Figures 9–17)** (Donnelly et al., 2018; Frank, 2013; McFadden et al., 2014; Monteleone et al., 2018 as cited in Chen et al., 2021 (139, 141-144)). This complex network involves components of the mesostriatal and nigrostriatal dopamine pathways (e.g., the VTA, ventral striatum/NAc, ventral and globus pallidum (VP and GP), the dorsomedial and dorsolateral striatum (caudate nucleus and putamen), OFC, PFC), corticolimbic structures (e.g., the amygdala, hippocampus, insula, and thalamus), and frontoparietal systems (e.g., ACC, PCC), and cortical structures/areas (e.g., PFC, OFC, premotor-, motor-, and somatosensory cortices). The primary components of the basal ganglia are:
  + The striatum, including the dorsal striatum (caudate and putamen, involved in goal-directed and habitual decision-making) and the ventral striatum (NAc, involved in reward salience and motivational drive).
  + The pallidum, including:
    - The **globus pallidus interna and externa (GPi, GPe)**, which receives dopaminergic and inhibitory GABAergic projections from the NAC and contain primarily inhibitory GABAergic neurons, causing it to have an inhibitory or disinhibitory impact on its targets. The GPe has an inhibitory effect on the Subthalamic Nucleus (STN/SNc), which excites the GPi and substantia nigra in turn. The GPi has a disinhibitory impact on its tartest, which include the thalamus and substantia Nigra (SN) (which sends dopaminergic and inhibitory GABAergic projections to the thalamus also.
    - The **ventral pallidum (VP)**, which receives inhibitory GABAergic and dopaminergic projections from the NAcS, conveying the reward salience of pleasurable experiences and substances (e.g., food, drugs, etc.))(115, 116). The VP also integrates accumbal inputs with dopaminergic inputs directly from the VTA to influence consummatory behaviors, including consumption, cue-induced feeding, taste reactivity, and food preference (116).
  + The **Substantia Nigra (SN)**, which consists of the **pars compacta (SNc)** and the **pars reticula (SNr)**, and: 1) produces dopaminergic projections that maintain balance in the striatal pathway; 2) works with the GPi to inhibit the thalamus.
  + The **Subthalamic Nucleus (SN/StN)**, \*\*\*

The BGN involves two pathways that inhibit and disinhibit the thalamus respectively (and regulate reward-related consummatory behaviors in turn).

* + In the **indirect inhibitory pathway**, the VTA sends dopaminergic projections to the NAc in the ventral striatum, which sends GABAergic inhibitory projects to the globus pallidus externa (GPe), inhibiting the thalamus in turn (**Supplemental Figure 9**).
  + In the **direct excitatory/disinhibitory pathway**, the VTA sends dopaminergic projections to the NAc in the ventral striatum, which inhibit the globus pallidus interna (GPi)/SNr and disinhibits the thalamus in turn. However the speed of the direct pathway would not be concordant with the indirect pathway in this model leading to dysregulation of the system. Overcoming this problem, a **hyperdirect excitatory/disinhibitory pathway** has been proposed, in which the cortex sends glutamatergic projections through the subthalamic nucleus exciting the inhibitory GPe under the *center surround model*, as well as a shorter indirect pathway.
* The BG is generally divided into five main pathways (See supplementary material S2 for figures):
  + The **Motor Loop** involves a loop of projections from the supplementary motor area to the arcuate premotor area, the motor cortex, somatosensory cortex, putamen (in the dorsal striatum), GP, SNr, and back to the cortex in turn.
  + The **Oculomotor Loop** involves a loop from the frontal visual field(s) to the **dorsal PFC (dPFC)**, **posterior parietal cortex (PPC)**, caudate (in the dorsal striatum), caudal dorsomedial GPi, ventral SNr, **lateral ventralis anterior pars magnocellularis (VAmc)**, and back to the cortex to the close the loop.
  + In the **First Cognitive/Associative Pathway**, the dorsolaterateral dPFC projects to the dorsal caudate (in the dorsal striatum) and on through the GPi to the SNr, which projects to the medial pars magnocellularis and the lateral VAmc.
  + The **Second Cognitive/Associative Pathway** involves a loop of projections from the lateral OFC to the temporal gyrus to the ACC, to the ventromedial caudate in the dorsal striatum, then projecting to the lateromedial GPi, rostrolateral SNr, medial VAmc, and back onto the cortex to close the loop.
  + In the **Limbic Circuit**, the ACC, hippocampus, entorhinal cortex, and insula project onto the ventral striatum (NAc), which projects onto rostrodorsal GPi, which projects onto the VP, rostrodorsal SNr, posteromedial dorsal nucleus, and back onto the cortex in turn.

Supplemental Figure 1: The complex neurobiological underpinnings of binge eating disorder.

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## Supplemental Figure 1: The complex neurobiological underpinnings of binge eating disorder.

Supplemental Figure 2: Mesostriatal Dopamine Circuit (Ventral Pathway)

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**Supplemental Figure 2: The mesostriatal dopamine circuit/pathway** (AKA the ventral pathway) is primarily associated with reward salience and motivational drive (Figure 2). In this circuit, dopaminergic neurons in the medial ventral tegmental area (VTA) innervate the ventral striatum (mainly the medial nucleus accumbens shell (NAcS), which sends GABAergic and dopaminergic projections to the ventral pallidum (VP) in turn, conveying the reward salience of pleasurable experiences and substances (e.g., food, drugs, etc.)){Poisson, 2021 #8181;Root, 2015 #8182}. The VP integrates accumbal inputs with dopaminergic inputs directly from the VTA to influence consummatory behaviors, including consumption, cue-induced feeding, taste reactivity, and food preference {Root, 2015 #8182}. Impaired inhibition of the mesolimbic portion of the mesostriatal dopamine pathway is also associated with impulsivity (Figure 2){Barbosa, 2022 #8193;Hiser, 2018 #8196}. **Abbreviations:** DA, dopamine; NAcS, nucleus accumbens shell; VP, ventral pallidum; VS, ventral striatum; VTA, ventral tegmental area.

Supplemental Figure 3: Impulsivity in binge eating disorder

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**Supplemental Figure 3:** Impulsivity is associated with deficient inhibition of the **ventral/ mesolimbic dopamine pathway** (**Figure 1**), by which dopaminergic neurons in the medial VTA innervate the ventral striatum (mainly the medial NAc), which projects onto the thalamus and then onto the ACC (see **Figure 1** for description) which projects back onto the ventral striatum both directly and through the ventromedial prefrontal cortex (vmPFC). The vmPFC is largely associated with exerting inhibitory control over the NAc and reward-seeking behaviors in turn {Richard, 2013 #8194;Ghazizadeh, 2012 #8195}, with decreased vmPFC thickness in binge eating and obesity thought to render this inhibitory system unable to prevent binge eating {Barbosa, 2022 #8193}. The resulting disinhibition is thought to contribute to impulsivity, both in impulsive, disinhibited binge eating and in general {Barbosa, 2022 #8193;Hiser, 2018 #8196}. **Abbreviations:** ACC, anterior cingulate cortex; NAcS, nucleus accembens shell; vmPFC ventromedial prefrontal cortex; VTA, ventral tegmental area.

Supplemental Figure 4: Nigrostriatal Dopamine Pathway (Dorsal Pathway)

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**Supplemental Figure 4. The nigrostriatal dopamine pathway** (AKA the dorsal pathway) is associated primarily with reward sensitization and compulsivity (Figure 2). Dopaminergic neurons in the VTA innervate the dorsomedial and dorsolateral striatum, including the caudate nucleus and putamen, which are essential for orchestrating goal-directed and habitual decision-making {Yin, 2005 #8197;Yin, 2004 #8198;Yu, 2022 #8202}. The dorsal pathway largely involves a circuit by which dorsal striatal projections innervate the paraventricular nucleus of the thalamus (PVN/PVT), which integrates homeostatic and hedonic feeding signals with physiological and environmental stress signals, anticipatory feeding needs, and cognitive inputs to regulate food-seeking and consumption {Petrovich, 2021 #8207}. The thalamus (innervates the orbitofrontal cortex (OFC) in turn (also thought to be involved in reward and punishment processing and reward-based decision-making {Seabrook, 2020 #8184;Rolls, 2023 #8185;Londerée, 2021 #8186;Suzuki, 2017 #8190;Kringelbach, 2005 #8191}), which feeds back onto the striatum {Lipton, 2019 #8188}. Additionally, preclinical findings demonstrate that dopamine sensitization in the dorsal striatum accelerates the development of habit formation from previously goal-directed behaviors {Belin-Rauscent, 2012 #8199;Wickens, 2007 #8200;Guo, 2014 #8201;Yu, 2022 #8202}, possibly contributing to compulsivity {Yin, 2005 #8197;Yin, 2004 #8198;Yu, 2022 #8202}. **Abbreviations:** DA, dopamine; OFC, orbitofrontal cortex (in the prefrontal cortex); PVN, paraventricular nucleus (in the thalamus); VTA, ventral tegmental area.

Supplemental Figure 5: Compulsivity in binge eating disorder

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**Supplemental Figure 5: Compulsivity** is associated with the dorsal/nigrostriatal dopamine pathway (Figure 1), by which dopaminergic neurons in the VTA innervate the dorsomedial and dorsolateral striatum, including the caudate nucleus and putamen, which are essential for orchestrating goal-directed and habitual decision-making {Yin, 2005 #8197;Yin, 2004 #8198;Yu, 2022 #8202}. Dorsal striatal projections innervate the paraventricular nucleus of the thalamus (PVN/PVT), which integrates homeostatic and hedonic feeding signals with physiological and environmental stress signals, anticipatory feeding needs, and cognitive inputs to regulate food-seeking and consumption {Petrovich, 2021 #8207}. The thalamus innervates the the insular cortex (the primary taste cortex involved in appetite, motivated behavior, emotional processing, and emotional, stress-driven eating {G. Anversa, 2023 #8203}) and orbitofrontal cortex (OFC), which are situated in junction to one another and both project back onto the striatum {G. Anversa, 2023 #8203;Lipton, 2019 #8188;Chikama, 1997 #8205;Wang, 2022 #8206}. The insular cortex also projects to the OFC as well as to the mPFC, amygdala, cingulate, and autonomic centers {Mathiasen, 2023 #8204;Wang, 2022 #8206}. The OFC then feeds back onto the striatum in turn {Lipton, 2019 #8188}. Preclinical findings demonstrate that dopamine sensitization in the dorsal striatum accelerates the development of habit formation from previously goal-directed behaviors {Belin-Rauscent, 2012 #8199;Wickens, 2007 #8200;Guo, 2014 #8201;Yu, 2022 #8202}, possibly contributing to compulsivity.

The shift from impulsivity to compulsivity involves neuroplastic changes in the mesolimbic dopamine system (esp. in the NAc) and in prefrontal systems, including the extended amygdala {Koob, 2010 #8189}, as well as dopamine desensitization in the dorsal striatum (of the nigrostriatal dopamine system) that accelerates the development of habit formation from previously goal-directed behaviors {Belin-Rauscent, 2012 #8199;Wickens, 2007 #8200;Guo, 2014 #8201;Yu, 2022 #8202}. The impulsivity and compulsivity are often associated with abbarent “top-down” cognitive control and dysregulated interactions between executive top-down self-regulatory prefrontal circuits and more reptilian mesolimbic circuits associated with behavioral engagement {Robbins, 2012 #8171;Dalley, 2011 #8172;Bari, 2013 #8173;Boswell, 2021 #8169;Morales, 2020 #6362}. The glucocorticoid stress system is also thought to be implicit in these dysregulated changes {Lutter, 2009 #6498;Kuckuck, 2023 #8170;Bray, 2020 #3575;Barr, 2017 #3065}. **Abbreviations:** DA, dopamine; OFC, orbitofrontal cortex (in the prefrontal cortex); PVN, paraventricular nucleus (in the thalamus); VTA, ventral tegmental area.

Supplemental Figure 6A–C: Corticolimbic System

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**Supplemental Figure 6A–C.** The corticolimbic system contributes to reward and motivational processes, and includes the amygdala, hippocampus, and prefrontal cortex (PFC) {Rusbridge, 2020 #8183}. **A)** The VTA sends dopaminergic neurons to the amygdala and hippocampus, which are thought to be involved in learning and remembering reward cues and innervate the NAc as part of the mesolimbic pathway. **B)** The orbitofrontal cortex (OCF) in the prefrontal cortex (PFC) is also thought to be involved in reward and punishment processing and reward-based decision-making {Seabrook, 2020 #8184;Rolls, 2023 #8185;Londerée, 2021 #8186;Suzuki, 2017 #8190;Kringelbach, 2005 #8191}. It receives and integrates various sensory inputs (e.g., taste, smell, touch, vision, sound) and learns (and reverses) associations between stimuli and their outcomes (e.g., foods and their salient/rewarding properties/pleasantness), adapting to valuation changes as needed (e.g., changes in food value), thus contributing to reward-based decision-making processes that guide behavior {Seabrook, 2020 #8184;Rolls, 2023 #8185;Londerée, 2021 #8186;Suzuki, 2017 #8190;Kringelbach, 2005 #8191}. **C)** The insula and thalamus (described above), hypothalamus, and brainstem/pons are also thought to be part of the corticolimbic circuitry that drives eating and binge eating behaviors {Boswell, 2021 #8169}. **Abbreviations:** DA, dopamine; NAc, nucleus accumbens; OFC, orbitofrontal cortex (in the prefrontal cortex); PFC (prefrontal cortex); VTA, ventral tegmental area.

Supplemental Figure 7: Frontoparietal Control System

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**Supplemental Figure 7:** The frontoparietal control system (also called the central executive network (CEN) and the executive control network (ECN)) are generally associated with executive functioning, decision-making, coordinating goal-driven behaviors, self-regulation, regulating appetitive responding, and regulating the hedonic/reward/reinforcement-based corticolimbic and mesostriatal dopamine circuits {Marek, 2018 #8179;Rosenbloom, 2012 #8180;Polk, 2017 #2023;Berridge, 2010 #6363;Olszewski, 2007 #6356;Berridge, 2009 #1543;Morales, 2020 #6362;Lindgren, 2018 #2214;Gordon, 2018 #6218;Novelle, 2018 #6359;Valbrun, 2020 #6258;Boswell, 2021 #8169;Lutter, 2009 #6498;Menon, 2011 #8439;Menon, 2011 #8445;Chen, 2021 #8442}. This network/system primarily includes the dorsolateral PFC (especially the OFC and middle frontal gyrus), anterior cingulate cortex (ACC), and posterior parietal cortex (PCC) (around the intraparietal sulcus), as well as the middle cingulate gyrus (mCG), dorsomedial thalamus, caudate nucleus head, dorsal precuneus, and posterior inferior temporal lobe{Menon, 2011 #8445;Chen, 2021 #8442}. Specifically, the OFC sends projections and reward information to the ACC, which integrates spatial and action-related information from the parietal cortical areas, thus connecting rewards to actions and reward-cues, while the PCC projects to the hippocampal system, thus storing reward memories {Rolls, 2019 #8192}. **Abbreviations:** ACC, anterior cingulate cortex; OFC, orbitofrontal cortex (in the prefrontal cortex); PFC (prefrontal cortex), PCC, posterior parietal cortex.

Supplemental Figure 8: Basal Ganglia Network (BGN)

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**Supplemental Figure 8: Basal Ganglia Network (BGN)**. The indirect inhibitory pathway in the basal ganglia results in inhibition of the globus pallidus externus (GPe), allowing for disinhibition of the globus pallidus internus (GPi) through the subthalamic nucleus (STN) allowing it to inhibit the thalamus. **Abbreviations:** GP, globus pallidus; SNr, substantia nigra.

Supplemental Figure 9 A–C: The Indirect Inhibitory-, Direct Excitatory/Disinhibitory-, and Hyperdirect Excitatory Pathways of the Basal Ganglia

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**Supplemental Figure 9A–C: Disinhibition in the direct and indirect pathways through the basal ganglia.** A) In the direct pathway, transiently inhibitory neurons in the caudate and putamen project to tonically active inhibitory neurons in the *internal* segment of the globus pallidus, which project in turn to the VA/VL complex of the thalamus. Transiently excitatory inputs to the caudate and putamen from the cortex and substantia nigra are also shown, as is the transiently excitatory input from the thalamus back to the cortex. B) In the indirect pathway (shaded), transiently active inhibitory neurons from the caudate and putamen project to tonically active inhibitory neurons of the *external* segment of the globus pallidus. Note that the influence of nigral dopaminergic input to neurons in the indirect pathway is inhibitory. The globus pallidus (external segment) neurons project to the subthalamic nucleus, which also receives a strong excitatory input from the cortex. The subthalamic nucleus in turn projects to the globus pallidus (internal segment), where its transiently excitatory drive acts to oppose the disinhibitory action of the direct pathway. In this way, the indirect pathway modulates the effects of the direct pathway. (Purves Neuroscience). Fig 18-7.

The brake–accelerator model for basal ganglia motor disorders. (a) The direct pathway (leading to release of movement) consists of two successive GABAergic connections, from the striatum to the internal pallidum and from the internal pallidum to the thalamus. This flow diagram suggests that excitatory (glutamate; Glu) inputs from the neocortex to the striatum would disinhibit thalamic neurons. Dopamine modulates the system mainly in the striatum, where it activates D1-class and D2-class dopamine receptors. (b) In the indirect pathway (leading to inhibition of movement), there is an extra step after the external pallidum, so that the subthalamic nucleus excites the internal pallidum. (c) Balance is achieved when these antagonistic systems are combined under normal circumstances. Graybiel. “The basal ganglia.” (200).

**Abbreviations:** DA, dopamine; GP, globus pallidus; GPe, globus pallidus externa; GPi, globus pallidus interna; SNc, substantia nigra pars compacta; STN, subthalamic nucleus (in the subthalamus).

Supplemental Figure 10: Direct/Disinhibitory Pathway of the Basal Ganglia

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**Supplemental Figure 10: Direct/Disinhibitory Pathway** **of the Basal Ganglia**. Text here. **Abbreviations:** GP, globus pallidus; SNr, substantia nigra.

Supplemental Figure 11: Motor Loop of the Basal Ganglia

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**Supplemental Figure 11: Motor Loop of the Basal Ganglia**. Text here. **Abbreviations:** GP, globus pallidus; SNr, substantia nigra.

Supplemental Figure 12: Oculomotor Loop of the Basal Ganglia

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**Supplemental Figure 12: Oculomotor Loop of the Basal Ganglia**. Text here. **Abbreviations:** dlPFC, dorsolateral prefrontal cortex; GPi, globus pallidus interna; PPC, posterior parietal cortex; SNr, substantia nigra; VAmc, lateral ventralis anterior pars magnocellularis.

Supplemental Figure 13: First Cognitive/Associative Pathway of the Basal Ganglia

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**Supplemental Figure 13: First Cognitive/Associative Pathway of the Basal Ganglia**. Text here. **Abbreviations:** D. Striatum, dorsal striatum; dlPFC, dorsolateral prefrontal cortex; ldmGPi, lateral dorsomedial globus pallidus interna; lVAmc, lateral ventralis anterior pars magnocellularis; mPmc, medial pars magnocellularis SNr, substantia nigra.

Supplemental Figure 14: Second Cognitive/Associative Pathway of the Basal Ganglia

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**Supplemental Figure 14: Second Cognitive/Associative Pathway of the Basal Ganglia**. Text here. **Abbreviations:** ACC, anterior cingulate cortex; D. Striatum, dorsal striatum; GPi, globus pallidus interna (specifically, laterodorsal GPi); lOFC, lateral orbitofrontal cortex (in the prefrontal cortex); SNr, substantia nigra (rostrolateral); mVAmc, meidal lateral ventralis anterior pars magnocellularis.

Supplemental Figure 15: Limbic Circuit of the Basal Ganglia

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**Supplemental Figure 15: Limbic Circuit of the Basal Ganglia**. Text here. **Abbreviations:** ACC, anterior cingulate cortex; GPi, globus pallidus interna; MDN, medial dorsal nucleus (in the thalamus); SNr, substantia nigra (rostrodorsal); VP, ventral pallidum.