Accepted for publication in Annual Review of Neuroscience

Cite as: Janacsek, K., Evans, T. M., Kiss, M., Shah, L., Blumenfeld, H., & Ullman, M. T. (2022). Subcortical Cognition: The Fruit Below the Rind. *Annual Review of Neuroscience*, *45*, 361-86, https://doi.org/10.1146/annurev-neuro-110920-013544

Subcortical Cognition: The Fruit Below the Rind

Karolina Janacsek,[1,2] Tanya M. Evans,[3] Mariann Kiss,[2,4] Leela Shah,[3] Hal Blumenfeld,[5] and Michael T. Ullman[6]

[1]Centre for Thinking and Learning, Institute for Lifecourse Development, School of Human Sciences, Faculty of Education, Health and Human Sciences, University of Greenwich, London, United Kingdom

[2]Institute of Psychology, ELTE Eötvös Loránd University, Budapest, Hungary

[3]School of Education and Human Development, University of Virginia, Charlottesville, Virginia, USA

[4]Department of Cognitive Science, Faculty of Natural Sciences, Budapest University of Technology and Economics, Budapest, Hungary

[5]Departments of Neurology, Neuroscience and Neurosurgery, Yale School of Medicine, New Haven, Connecticut, USA

[6]Brain and Language Lab, Department of Neuroscience, Georgetown University, Washington, DC, USA; email: michael@georgetown.edu

Abstract

Cognitive neuroscience has highlighted the cerebral cortex while often overlooking subcortical structures. This cortical proclivity is found in basic and translational research on many aspects of cognition, especially higher cognitive domains such as language, reading, music, and math. We suggest that, for both anatomical and evolutionary reasons, multiple subcortical structures play substantial roles across higher and lower cognition. We present a comprehensive review of existing evidence, which indeed reveals extensive subcortical contributions in multiple cognitive domains. We argue that the findings are overall both real and important. Next, we advance a theoretical framework to capture the nature of (sub)cortical contributions to cognition. Finally, we propose how new subcortical cognitive roles can be identified by leveraging anatomical and evolutionary principles, and we describe specific methods that can be used to reveal subcortical cognition. Altogether, this review aims to advance cognitive neuroscience by highlighting subcortical cognition and facilitating its future investigation.

Keywords

subcortical, cognition, connectivity, co-optation, network, dynamic

1. THE PROMISE OF SUBCORTEX

Research investigating the neural substrates of human cognition has focused on cerebral cortex while often neglecting subcortical structures. This predilection for cortex—"rind" or "bark" in Latin, referring to the gray matter surrounding the cerebrum—is widespread. It is found in both theoretical and empirical work across basic and translational research in multiple domains of cognition. A cortical inclination can be found especially in the study of higher-level aspects of cognition, such as language, reading, music, and math, though it occurs even to some extent for lower-level functions, such as attention, executive function, and memory (see **Supplemental Appendix 1**). Conversely, research on subcortical (noncortical) structures—that is, gray matter structures below cortex—has often focused on noncognitive rather than cognitive functions (Koziol & Budding 2009, Noback et al. 2005). This propensity for noncognitive functions, which has deep historical roots (e.g., "the reptilian brain"; MacLean 1988), is also prevalent in the clinical community; for example, clinicians tend to highlight motor rather than cognitive impairments in basal ganglia and cerebellar disorders (Jankovic 2008, Whaley et al. 2011). Thus, overall, there has been a much stronger emphasis on the role of cortex than subcortex in cognition.

Nevertheless, recent years have seen an emerging interest in subcortical contributions to cognition. Much of this attention has focused on the basal ganglia (a key component of the posited reptilian brain) and the cerebellum, not only for aspects of lower cognition but also for some higher aspects such as language (Kotz & Schwartze 2010, Mariën et al. 2014, Murphy et al. 2021, Schmahmann et al. 2019, Ullman et al. 2020, Vargha-Khadem et al. 2005). (We use the term higher cognition to refer to cognitive functions that appear to be found only in humans, in particular in their more complex forms; other functions are classified within lower cognition.) Indeed, the basal ganglia and/or the cerebellum has been implicated in a variety of disorders involving atypicalities in higher as well as lower aspects of cognition, including attention-deficit/hyperactivity disorder (Dickstein et al. 2006), autism (Amaral et al. 2008), developmental language disorder (Ullman et al. 2020), dyslexia (Nicolson & Fawcett 2007, Ullman et al. 2020), dyscalculia (Evans & Ullman 2016), aphasia (Crosson et al. 2007, De Smet et al. 2013), alexia (Mariën et al. 2009), amusia (Sihvonen et al. 2016), and acalculia (Delazer et al. 2004, Roşca 2009). Thus, some evidence implicates at least certain subcortical structures in some aspects of

cognition, including higher-level functions.

1.1. Beyond the Tip of the Iceberg

We posit that the subcortical contributions summarized above represent only the tip of the subcortical cognition iceberg. That is, we suggest that subcortical structures play much more extensive roles in human cognition than has generally been acknowledged. Specifically, we hypothesize that multiple subcortical structures throughout the brain—well beyond the basal ganglia and cerebellum—make significant contributions to multiple aspects of both higher and lower cognition. Indeed, even structures such as the basal ganglia that have been reasonably well studied regarding their cognitive contributions likely play much more substantial cognitive roles than has generally been recognized. For a discussion of the biases and methodological limitations that may explain why known or potential contributions of subcortical structures to cognition have not been emphasized more to date, see **Supplemental Appendices 2** and **3**, as well as Parvizi (2009).

Extensive subcortical involvement is expected for both anatomical and evolutionary reasons. First of all, there are clear anatomical reasons to expect, a priori, that a wide range of subcortical structures should play prominent roles in human cognition. It has become clear that cognitive functions are carried out not by isolated structures but by brain networks (Bassett & Sporns 2017, Bertolero et al. 2015). Until now, such networks have generally emphasized (neo)cortical structures, especially for higher-level cognitive functions such as language, reading, music, and math (Dehaene et al. 2004, Feng et al. 2020, Friederici & Gierhan 2013, Janata 2005, Yeo et al. 2011). However, evidence from structural and functional connectivity studies suggests that these cortical regions are linked to subcortical structures throughout the brain, via direct connections as well as indirect connections through other (sub)cortical structures. For example, the basal ganglia are connected directly (e.g., through the striatum) and indirectly (e.g., via the thalamus) to neocortical regions across the cerebrum, including many that underlie higher cognition (Draganski et al. 2008, Postuma & Dagher 2006, Saunders et al. 2015). Even structures in the brainstem whose cognitive roles are much less well studied, such as the red nucleus and the pedunculopontine nucleus, show structural and/or functional direct and indirect connections with cortical regions subserving higher and lower cognition (Martinez-Gonzalez et al. 2011, Nioche et

al. 2009). Indeed, it is likely that all subcortical structures are directly or indirectly connected to multiple neocortical regions (Ji et al. 2019). Moreover, certain subcortical structures are the major loci of specific neurotransmitters (e.g., norepinephrine from the locus coeruleus and dopamine from midbrain structures) that play important cognitive roles thanks to their projections to (sub)cortical regions that underlie cognition (Sara 2009, Ullman et al. 2020). Thus, overall, the brain networks underlying higher as well as lower cognition should be expected to include subcortical as well as cortical structures.

The expectation of subcortical contributions to cognitive networks is further strengthened by the sheer extent of subcortex in humans: The fruit below the rind is substantial. From a developmental structural perspective, cortex is but part of a part of a part of the entire brain (Haines 2004). That is, cerebral cortex is only part of the telencephalon, which additionally includes multiple subcortical structures. The telencephalon, in turn, is only part of the prosencephalon (forebrain), which also includes the diencephalon—all of which is subcortical. And the prosencephalon is only one part of the entire brain, which also includes the mesencephalon (midbrain) and rhombencephalon (hindbrain), which itself is made up of the metencephalon (pons and cerebellum) and the myelencephalon (medulla)—none of which are part of cerebral cortex. Beyond this developmental partitioning indicating the prevalence of subcortex, the actual number of subcortical structures underscores this point: The brain includes at least 80 subcortical (noncortical) structures and substructures (see the figures in this review and **Supplemental Tables 1–4**), not even including the cerebellum. Indeed, subcortical structures constitute approximately 18% (with the cerebellum representing 10% and the remaining subcortex 8%) of the total gray and white matter mass of the human brain (Azevedo et al. 2009).

There are also strong evolutionary reasons to predict substantial subcortical contributions to cognition. Various lower-level cognitive functions that are not unique to humans, such as attention, executive function, working memory, and declarative memory, depend importantly on subcortical structures in nonhuman animals (Givens & Olton 1990, Vann & Aggleton 2004, Wright et al. 2015). The frequent preservation of structure–function mappings in evolution suggests a similar dependence in humans as well (Lee et al. 2013, Leszczyński & Staudigl 2016, Tsivilis et al. 2008). This logic may also extend to higher cognition, since in nonhuman animals possible analogs of or precursors to higher cognitive functions such as language or math also rely

on noncortical structures (Collins et al. 2017, Fitch 2000, Hage & Nieder 2016, Hunt et al. 2008).

Subcortical cognitive roles may also be expected according to the basic evolutionary and biological principle of co-optation (exaptation), that is, the reuse of existing structures or mechanisms for new functions (Gould & Vrba 1982). It follows from this principle that cognitive functions in humans (as well as nonhuman animals) are likely to have piggybacked on cortical and subcortical structures that predate these functions—whether or not these structures have been modified further, either evolutionarily or developmentally, for these new functions (Dehaene & Cohen 2007; Ullman 2004, 2016). Thus, even cognitive functions that may be unique to humans are likely to depend on structures and mechanisms that predate the emergence of these functions, as well as *Homo sapiens* itself. Brain structures that subserve noncognitive functions sharing characteristics with cognitive functions, which may be co-opted to support these functions, are promising candidates, as are structures that underlie lower aspects of cognition that could be appropriated for higher-level cognitive functions. For example, evidence suggests that both noncognitive and lower cognitive functions involving the basal ganglia have been co-opted for language, such as motor functions for naming motor-related words like "hammer" (Johari et al. 2019) and procedural learning for grammar (Ullman 2016). Even (sub)cortical substrates for higher cognition may be co-opted for other higher functions (e.g., as has been proposed by the "neuronal recycling hypothesis" for reading; Feng et al. 2020). Moreover, since structures can be co-opted for completely new functions (e.g., feathers seem to have evolved for thermal regulation, but then were co-opted for flight; Gould & Vrba 1982), subcortical structures might have been hijacked for aspects of cognition even if these structures previously subserved very different noncognitive (or cognitive) functions. Finally, given the functional flexibility conferred by neural plasticity, the reuse of older structures for new functions is particularly likely in the brain. Indeed, subcortical structures such as the basal ganglia and the cerebellum appear to show substantial plasticity (Hansel et al. 2001, Kreitzer & Malenka 2008), underscoring the likelihood of the reuse of these structures for new functions.

2. A GUIDE TO TERRA COGNITA: A REVIEW OF THE KNOWN STRUCTURE–FUNCTION MAP OF SUBCORTICAL COGNITION

In this section, we comprehensively review the evidence to date on subcortical cognition. The review is comprehensive in that our search encompassed subcortical structures and substructures

throughout the brain and, moreover, targeted a wide range of both lower and higher cognitive functions, with a focus on the latter. However, we emphasize that our review is not systematic, in that not all relevant papers were included. For the methods employed in the review, see **Supplemental Appendix 4**.

The review is designed to achieve two broad goals. First, it tests whether subcortical structures indeed play substantial roles across higher and lower cognitive domains. Second, the structure–function map generated by our review is designed to help researchers incorporate subcortical structures into existing or new theoretical frameworks, as well as to guide them in hypothesis-driven empirical research and in the interpretation of subcortical findings in data-driven neurocognitive studies.

Figures 1–**4** present a graphical summary of the review. The figures show which higher and lower cognitive domains have been empirically linked to which subcortical (sub)structures throughout the brain: in the lower brainstem (medulla and pons; **Figure 1*a***), upper brainstem (i.e., the midbrain; **Figure 1*b***), diencephalon (**Figure 2**), and telencephalon (**Figure 3**); see also **Figure 4** for lower-level substructures of the diencephalon and telencephalon. Our review identifies structure–function mappings on the basis of a wide range of evidence, including from neuroimaging, brain stimulation, electrophysiological, and lesion studies. The figures do not include any cortical regions, in either neocortex or allocortex. Thus, we exclude olfactory cortex, the hippocampus, and other medial temporal lobe allocortical regions. White matter structures are also excluded. In the interest of brevity, we do not include the cerebellum, which has perhaps been the best-studied subcortical structure regarding cognition (Caligiore et al. 2017, Mariën et al. 2014, Schmahmann et al. 2019).

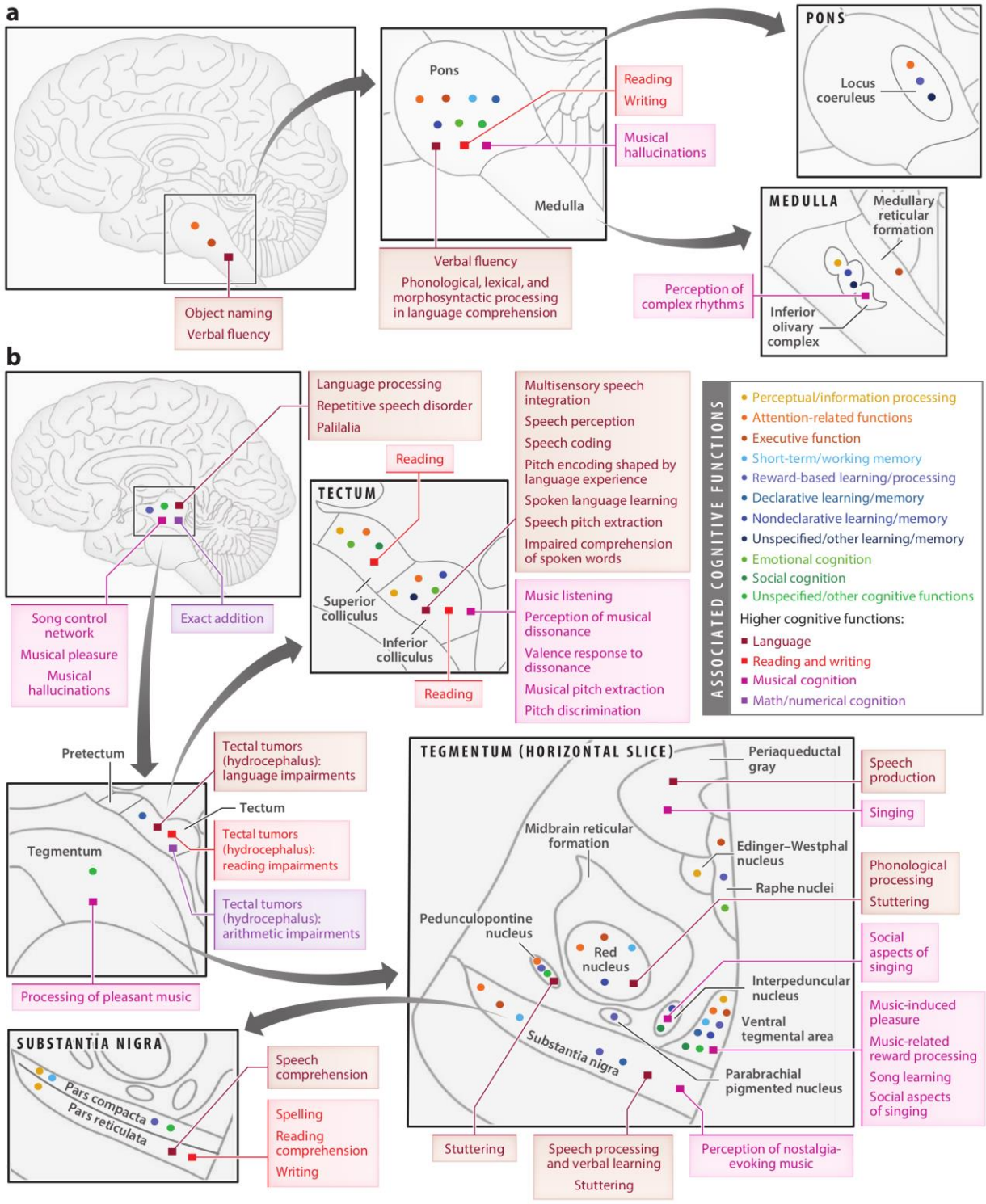

Figure 1 Structure–function map of subcortical cognition for (*a*) the lower brainstem (medulla and pons) and (*b*) the upper brainstem (midbrain), with a focus on higher cognition. Evidence of cognitive involvement for each (sub)structure is indicated with different-colored circles (lower cognitive domains) and squares (higher cognitive domains). Within each subcortical

(sub)structure, the circles and squares are generally organized (*top*/*down*, *left*/*right*) according to their order in the legend. Specific functions and dysfunctions linked to each (sub)structure are listed in colored boxes for higher cognitive domains only. The figure is designed for illustrative purposes only, and thus the neuroanatomy of the subcortical structures (e.g., their sizes and shapes) is not exact. Because the auditory brainstem is composed of the inferior colliculus together with other structures, it is not shown in panel *b*; the parabigeminal nucleus is also not shown in this panel (for both of these, see **Supplemental Table 2**). For further details, including for specific lower cognitive and noncognitive (dys)functions, see **Supplemental Table 1** (lower brainstem) and **Supplemental Table 2** (upper brainstem).

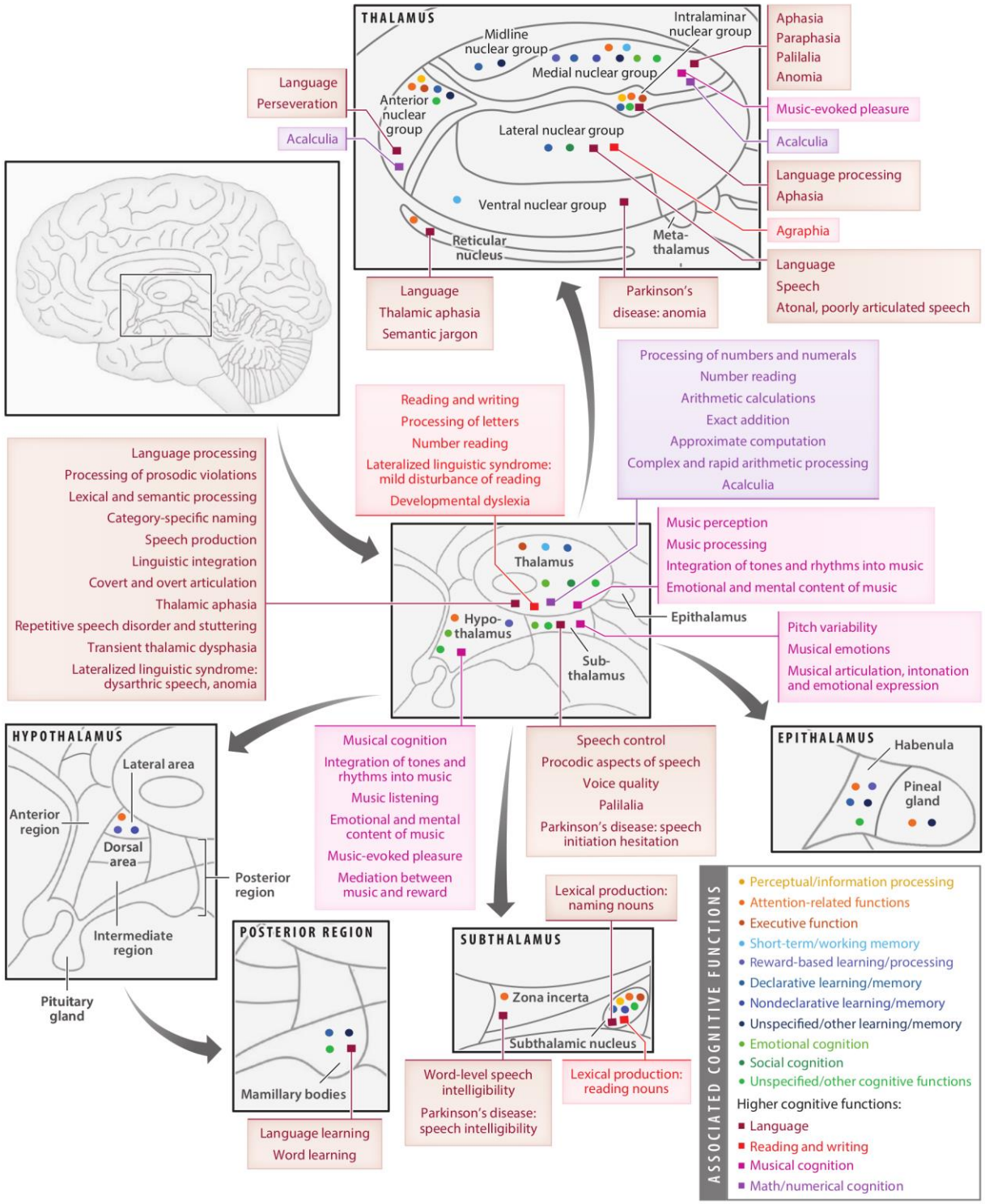

Figure 2 Structure–function map of subcortical cognition for the diencephalon, with a focus on higher cognition. Evidence of cognitive involvement for each (sub)structure is indicated with different-colored circles (lower cognitive domains) and squares (higher cognitive domains). Within each subcortical (sub)structure, the circles and squares are generally organized

(*top*/*down*, *left*/*right*) according to their order in the legend. Specific functions and dysfunctions linked to each (sub)structure are listed in colored boxes for higher cognitive domains only. The neuroanatomy of the subcortical structures (e.g., their sizes and shapes) is not exact. For further details, including for specific lower cognitive and noncognitive (dys)functions, see **Supplemental Table 3** (diencephalon).

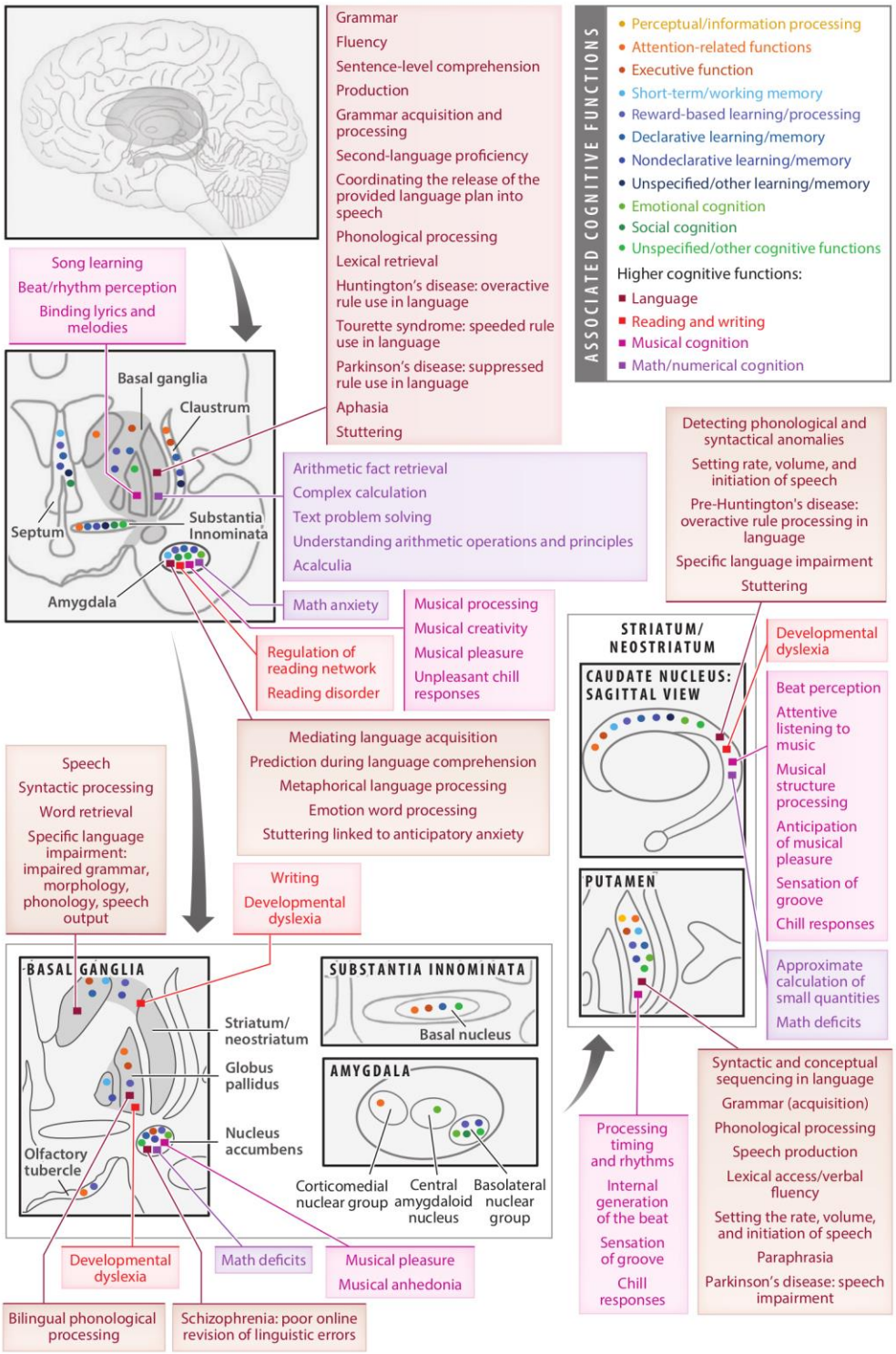

Figure 3 Structure–function map of subcortical cognition for the telencephalon, with a focus on higher cognition. Evidence of cognitive involvement for each (sub)structure is indicated with different-colored circles (lower cognitive domains) and squares (higher cognitive domains).

Within each subcortical (sub)structure, the circles and squares are generally organized (*top*/*down*, *left*/*right*) according to their order in the legend. Specific functions and dysfunctions linked to each (sub)structure are listed in colored boxes for higher cognitive domains only. The neuroanatomy of the subcortical structures (e.g., their sizes and shapes) is not exact. Because the lentiform nucleus, dorsal striatum, and ventral striatum are all composed of other structures (e.g., the putamen and globus pallidus for the lentiform nucleus), they are not shown here (for these structures, see **Supplemental Table 4**). For further details, including for specific lower cognitive and noncognitive (dys)functions, see **Supplemental Table 4** (telencephalon).

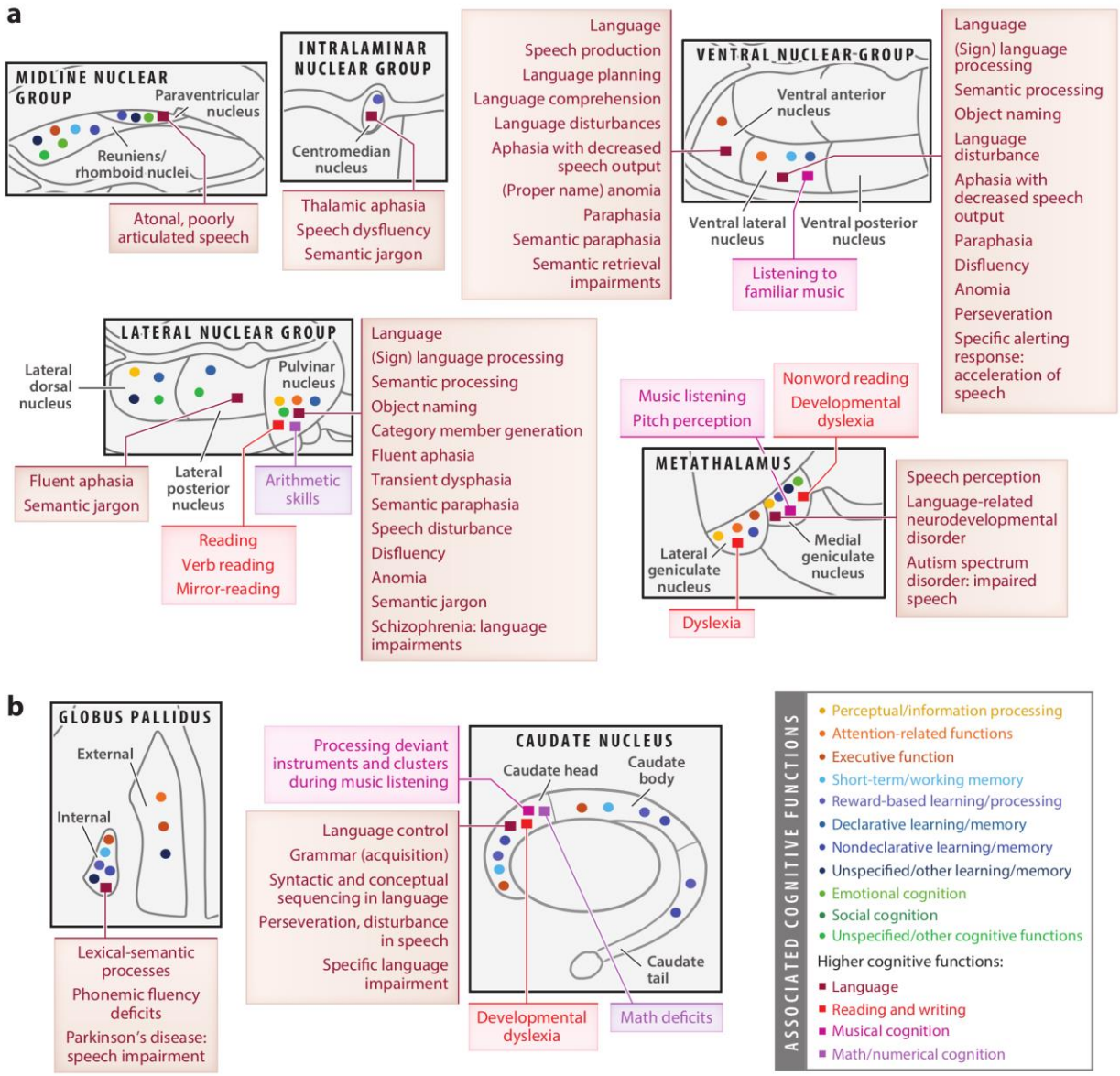

Figure 4 Structure–function map of subcortical cognition for lower-level substructures of (*a*) the thalamus in the diencephalon and (*b*) the basal ganglia in the telencephalon, with a focus on

higher cognition. Evidence of cognitive involvement for each (sub)structure is indicated with different-colored circles (lower cognitive domains) and squares (higher cognitive domains). Within each subcortical (sub)structure, the circles and squares are generally organized (*top*/*down*, *left*/*right*) according to their order in the legend. Specific functions and dysfunctions linked to each (sub)structure are listed in colored boxes for higher cognitive domains only. The neuroanatomy of the subcortical structures (e.g., their sizes and shapes) is not exact. For further details, including for specific lower cognitive and noncognitive (dys)functions, see **Supplemental Tables 3** (diencephalon) and **4** (telencephalon).

Supplemental Tables 1–4 provide additional information over and above what is shown in the four figures. Specifically, these tables present lists of the specific functions and dysfunctions of lower cognitive domains that have been linked to each (sub)structure (only those for higher domains are shown in the figures), additional details on the (dys)functions listed in the figures for higher domains, noncognitive functions linked to each (sub)structure (see Section 3.1), and references to empirical studies and review articles for all entries.

2.1. Evidence Suggests that Multiple Subcortical Structures Play Multiple Cognitive Roles

As can be seen in **Figures 1–4** and **Supplemental Tables 1–4**, a wide range of subcortical structures throughout the brain have been found to play roles in multiple aspects of cognition, including higher cognitive domains. The review thus supports the idea that subcortical cognition is extensive. The following observations on the review further elucidate subcortical contributions to cognition.

First of all, the findings suggest cognitive roles for subcortical (sub)structures that have been essentially ignored until now in cognitive neuroscience, especially in the study of higher cognition. These include the inferior olivary complex, pons, periaqueductal gray, pedunculopontine nucleus, red nucleus, raphe nuclei, habenula, pineal gland, zona incerta, septal nuclei, and substantia innominata. Indeed, many of these structures, which are found from the lower brainstem up to the telencephalon, are probably not even on the cognitive radar of most cognitive neuroscientists. Yet the evidence from studies using a variety of methods, including electrophysiological, neuroimaging, stimulation, and lesion approaches, suggests that all of these structures play roles in cognition, including in aspects of higher cognition, across language (pons, periaqueductal gray, pedunculopontine nucleus, red nucleus, zona incerta), reading (pons), and music (inferior olivary complex, pons, periaqueductal gray).

Our review further indicates that subcortical regions already well known by cognitive

neuroscientists to play noncognitive functions in fact also appear to underlie multiple aspects of cognition, including in higher cognitive domains. For example, the findings reveal that (portions of) the hypothalamus have thus far been implicated in attention, reward learning (reinforcement learning), declarative memory (episodic memory), nondeclarative memory (associative learning), other learning and memory (object learning and memory), and both language (language/word learning) and musical cognition (integration of tones and rhythms into music, mediation between reward and music listening, and other functions) (**Figure 2**; **Supplemental Table 3**). Along the same lines, the amygdala seems to underlie aspects of attention, working memory, reward learning and processing (motivating behavior through reward), declarative memory (episodic memory), nondeclarative memory (associative learning), social cognition (theory of mind), other cognitive functions such decision-making, and even language (aspects of both language acquisition and comprehension), reading, music (musical processing, musical creativity), and math (math anxiety) (**Figure 3**; **Supplemental Table 4**). These findings suggest that greater integration and communication between affective neuroscience, which has focused to a fair extent on these structures, and cognitive neuroscience, which has not, may be warranted.

The figures and tables also reveal that subcortical structures that are often discussed regarding specific lower cognitive functions in fact play wider cognitive roles, in both lower and higher domains. For example, the basal ganglia, whose links to cognition have been particularly well studied, have often been discussed with respect to aspects of learning or executive function, mainly regarding portions of the striatum. Yet the evidence suggests that the basal ganglia (defined as a telencephalic set of structures; **Figures 3** and **4*b***; **Supplemental Table 4**) are involved in a wide range of cognitive functions, including attention; executive function (cognitive control, planning); working memory; declarative memory; reward learning and nondeclarative memory, in particular procedural memory; other learning and memory (route recognition); decision-making; and various higher cognitive domains, including language (grammar acquisition, language control), reading, writing, music (processing of timing and rhythm, beat perception), and math (arithmetic fact retrieval, complex calculation, text problem-solving). Moreover, the basal ganglia substrates of at least some of these cognitive functions extend beyond the striatum to the globus pallidus (**Figures 3** and **4*b***; **Supplemental Table 4**), as well as to midbrain and diencephalic structures that are also generally considered to be part of the basal ganglia complex, namely the substantia nigra and subthalamic nucleus (**Figures 1*b*** and

2; **Supplemental Tables 2** and **3**).

Finally, the findings presented in the figures and tables underscore the cognitive contributions of subcortical structures that are already reasonably well known to underlie cognition. In particular, our review shows that the thalamus, which is often discussed as a relay station for sensory, motor, and other functions but has also been implicated in cognition, appears to underlie multiple lower and higher cognitive domains. These include aspects of perceptual processing (higher-order visual object processing), attention, executive function (inhibitory control), working memory, reward processing, declarative memory, nondeclarative memory, other learning and memory (spatial memory), emotional cognition, social cognition, and other cognitive functions such as spatial navigation and decision-making, as well as higher domains, in particular language (lexical, semantic, and prosodic processing; category-specific naming; speech production; linguistic integration), reading (letter processing), writing, music (music perception and processing), and math (number processing, arithmetic calculations, exact addition) (**Figures 2** and **4*a***; **Supplemental Table 3**).

Whereas **Supplemental Tables 1–4** and **Figures 1**–**4** present structure–function mappings organized by subcortical structure, a clear exposition of this information organized by cognitive domain may also be useful. Thus, **Supplemental Table 5** lists, for each domain (e.g., declarative memory, language), all the subcortical structures that have been associated with that domain, together with the particular functions and dysfunctions in that domain that these structures have been linked to. For example, the entry for language in this table lists the subcortical structures in the lower brainstem that have been associated with language, together with the particular language (dys)functions these structures have been tied to, and similarly for the upper brainstem, diencephalon, and telencephalon. The table contains the exact same information as **Supplemental Tables 1-4** but presented by cognitive domain rather than by subcortical structure. An indication of this structure-function information organized by domain can also be gleaned from **Figures 1**–**4**, namely in the set of structures in which each colored circle (lower cognitive domain) or square (higher cognitive domain) is found. Sticking with the example of language, examining the burgundy squares across the four figures indicates the wide range of subcortical structures that underlie this domain. The evidence presented in **Supplemental Table 5** and the figures suggests that multiple aspects of cognition—including higher cognitive domains—depend on multiple subcortical structures, which contribute a variety of specific

functions. For example, they suggest that language involves numerous subcortical structures throughout the brain, including the pons (lower brainstem); the inferior colliculus, periaqueductal gray, pedunculopontine nucleus, red nucleus, and substantia nigra (upper brainstem); the thalamus, the mammillary bodies in the hypothalamus, and both the subthalamic nucleus and zona incerta in the subthalamus (diencephalon); and the amygdala and multiple substructures in the basal ganglia (telencephalon). Although our review focused on individual subcortical structures rather than networks (largely because most empirical studies also focus on individual structures), the aggregation of structures by function as shown in the figures and the table reveals the extent of subcortical involvement in the networks underlying cognition. Overall, the pattern underscores the conclusion that cortical networks for both lower and higher cognitive domains extend to subcortical structures.

2.2. An Example of Subcortical Contributions to Higher Cognition: Lexical Functioning

In the previous section, we discussed the breadth of subcortical roles in cognition that were revealed by our review. In this section, we examine the role of subcortex in one higher cognitive function in some depth to demonstrate how the review may be useful for furthering the understanding of subcortical contributions to particular aspects of cognition. We focus on lexical functioning, defined broadly as the learning and processing of lexical knowledge, including aspects of conceptual/semantics and phonology (Ullman 2007). As with other higher cognitive functions, research on this topic has concentrated on cortex, especially neocortex, and more recently on allocortex (e.g., the hippocampus) as well (Ullman 2007).

As can be seen in **Supplemental Table 5** and the figures, evidence in fact suggests that a wide range of subcortical (sub)structures, across the lower brainstem, upper brainstem, diencephalon, and telencephalon, subserve lexical functioning. Moreover, the findings reveal which particular subcortical structures have been implicated in which particular lexical functions, including aspects of word learning (the hypothalamus, specifically the mammillary bodies, and the lentiform nucleus in the basal ganglia), lexical retrieval (the lower brainstem not further specified; the pons; the thalamus; within the thalamus the centromedian nucleus, medial nuclear group/medial dorsal nucleus, lateral posterior nucleus, pulvinar nucleus, ventral nuclear group, ventral anterior nucleus, ventral lateral nucleus, and reticular nucleus; the subthalamic nucleus in the subthalamus; the striatum/neostriatum and lentiform nucleus and, more specifically, the

putamen and internal globus pallidus within the basal ganglia), conceptual/semantic processing (the pulvinar nucleus, ventral anterior nucleus, and ventral lateral nucleus within the thalamus; the caudate nucleus head, putamen, and internal globus pallidus within the basal ganglia), (lexical) phonological processing (the red nucleus, the basal ganglia, the caudate nucleus and globus pallidus within the basal ganglia), and other aspects of lexical processing (the pons, the inferior colliculus, the thalamus, the zona incerta in the subthalamus, the amygdala, and the nucleus accumbens). Moreover, subcortical contributions to these rather broad aspects of lexical functioning can be further specified. For example, **Supplemental Table 5** and the figures reveal the particular subcortical structures that have thus far been implicated in specific tasks probing aspects of lexical retrieval such as verbal fluency tasks and (object) naming tasks.

Although the exact contributions of the subcortical structures implicated in these aspects of lexical functioning, and how they work together and with cortex as a functional network, are not yet understood, we suggest that both their connectivity and the co-optation of existing functions can elucidate this issue. Take word learning, for which two subcortical structures have thus far been implicated: the mammillary bodies and the lentiform nucleus. The mammillary bodies underlie declarative memory (e.g., **Supplemental Table 3**) due to their close anatomical links with the hippocampi, each of which project to this structure via the fornix. Given that word learning has been linked to declarative memory, it is likely and indeed expected that the declarative memory functions of these hypothalamic structures have been co-opted to play a role in word learning (Ullman 2004, 2016). The lentiform nucleus (composed of the putamen and globus pallidus) likely makes distinct contributions to word learning. This structure has thus far been implicated in word learning during word memorization in a word list–learning task (**Supplemental Table 5**), which heavily relies on working memory. Given that (verbal) working memory been linked to the lentiform nucleus and its anatomical components (**Supplemental Table 4**), it seems plausible that the working memory function of this structure has been hijacked for word learning as well.

We emphasize that this evidence for subcortical roles in word learning likely represents only a small portion of the true subcortical contributions—not only because subcortical roles in cognition have been neglected in general (see Section 2.3 and **Supplemental Appendices 2** and **3**) but also because the neural substrates of word learning have probably been less well studied than those of word processing, which indeed has been linked to more subcortical structures.

Thus, further studies are clearly needed to elucidate the exact nature of subcortical contributions to word learning, as well as lexical functioning more generally. Nevertheless, we hope that our dive into the role of subcortex in lexical functioning, in particular word learning, has demonstrated the utility of our review in advancing the understanding of subcortical contributions to (higher) cognitive functions.

2.3. Are the Findings Both Real and Important?

One might argue that many of the findings discussed above and presented in the figures and tables do not reflect real effects but rather are false positives due to confounds, small sample sizes, insufficient correction for multiple comparisons, and so on. As with any set of findings, we agree that this is possible for some of the results. Nevertheless, we suggest that the overall pattern of subcortical contributions reported in the review, as well as a large number of the individual structure–function pairs, is real.

Our comprehensive review revealed a myriad of structure–function mappings, across multiple subcortical structures and multiple lower and higher cognitive domains, from a large number of studies using a range of methods. This suggests that the overall contribution of subcortical structures to both lower and higher cognition is indeed substantial. That is, the amount and diversity of evidence argue against the possibility that the broad pattern of findings is spurious. This logic also applies to many of the individual structures whose cognitive roles are well studied, since these roles are often supported by converging evidence from an array of studies, with a variety of methodological approaches (**Figures 1**–**4**; **Supplemental Tables 1–5**).

In fact, we suggest that the evidence reported in our review likely represents an undercount of subcortical contributions to cognition, given the biases and methodological limitations that have thus far hindered research on this topic (see **Supplemental Appendices 2 and 3**). Additionally, quite a few papers were likely omitted from our review, both because the review was not systematic and because we largely ignored certain lines of evidence, such as genetic or other molecular/cellular findings. Thus, even the findings reported in our comprehensive review likely capture a relatively small portion of the subcortical cognition iceberg.

One might also argue that subcortical contributions to cognition are not important, even if they are real. In particular, it might be suggested that subcortical structures often play only secondary or supporting roles in cognition, especially for higher cognitive domains. This

argument could take different forms, two of which are laid out here. First, if a structure is known to subserve noncognitive roles such as motor or sensory functions, or lower cognitive roles such as attention or memory, then perhaps its roles in (higher) cognition should be interpreted as simply being due to those lower functions. For example, because evidence suggests that the neostriatum underlies motor functions, inhibitory control, working memory, and procedural memory, then perhaps its role in language should be attributed solely to one or more of these lower-level functions, which in turn are involved in language. On this interpretation, the neostriatum would not actually subserve language itself but rather these lower functions that then play roles in language. Second, whether or not a structure subserves a (higher) cognitive domain directly, if it plays roles only in relatively specific circumstances, then it could be argued that it should not be interpreted as making an important contribution in that domain and should not be considered to be part of the underlying brain network that supports it. For example, if a structure's role in language appears to be restricted only or primarily to specific aspects of that domain (e.g., articulation or particular syntactic relations) or to certain contexts (e.g., during learning, or while processing certain types of stimuli such as emotion-related words), perhaps one should not interpret this structure as being important for language, or part of the "language network." Note that the possible interpretations laid out in both scenarios in this paragraph would apply to cortical as well as subcortical structures.

We take a different position and suggest that many if not most subcortical contributions to cognition are important—including contributions that one might argue are only secondary or supporting, such as in the scenarios described above.

First of all, the figures and tables reveal that lesions or abnormalities of subcortical structures often lead to cognitive impairments, for higher as well as lower cognitive functions. Such dysfunction following damage has often been taken to suggest that the affected structures are necessary for cognition rather than simply being involved in supporting but nonessential roles (Mah et al. 2014, Rorden & Karnath 2004). Lesion evidence implicates a wide range of subcortical structures in (higher) cognitive functions (**Figures 1–4**; **Supplemental Tables 1–5**). For example, structures for which the listed symptoms or disorders affect various aspects of language include—but are not limited to—the inferior colliculus, red nucleus, pedunculopontine nucleus, substantia nigra, lateral posterior thalamic nucleus, pulvinar nucleus, ventral anterior thalamic nucleus, caudate nucleus, putamen, nucleus accumbens, and internal globus pallidus.

This lesion evidence suggests that these structures are indeed critical for language, despite the fact that many of the structures may fit the description in the first scenario above, namely they have often been thought of as primarily subserving lower (noncognitive or cognitive) functions. Moreover, the language dysfunctions associated with these structures range widely and include impaired comprehension of spoken words, stuttering, fluent aphasia, semantic jargon, paraphasia, semantic retrieval impairment, specific language impairment, overactive rule processing, and speech impairment. Underscoring the impact of subcortical lesions on (higher) cognition, cognitive impairments are included in the figures and tables not only as symptoms or disorders but also in the lists of cognitive functions associated with the various (sub)structures, since these lists also often include lesion studies.

Thus, even lesion evidence alone counters the view that many subcortical structures simply play secondary or supporting cognitive roles. Rather, this evidence suggests that multiple subcortical structures are critical for human cognition, including in higher cognitive domains. We also point out that although subcortical lesions could lead to cognitive impairments due to the dysfunction of proximal or connected cortical regions (diaschisis) (Carrera & Tononi 2014, Mah et al. 2014), which could be taken to suggest that subcortical structures are not necessary for such cognitive functions, the converse is also true, in that cortical lesions leading to cognitive deficits could be due to the dysfunction of nearby or connected subcortical structures.

What about those subcortical structures for which there does not yet appear to be lesion evidence implicating (higher) cognition, or for which the lesion evidence seems unclear? We suggest that many such structures may also contribute importantly to (higher) cognition. Just because lesion evidence has not yet been reported, or was not identified in our review or was unclear, does not mean that convincing lesion evidence will not be found. Moreover, if damage to a structure does not yield an impairment of a particular function, this would not imply that the structure is not important for the function. Not only is it a null result (which could have many explanations), but also, crucially, there may be other structures playing redundant roles for the function, which could take over the function and thus obscure the importance of the damaged structure (see Section 2.4 for further discussion).

We also suggest that the possible interpretations presented above for the two scenarios are problematic in their own right. Regarding the first scenario, it would be difficult to empirically demonstrate that a particular (subcortical or cortical) structure only subserves a (higher)

cognitive function through the action of a lower one. Even if this were shown, it may be just as appropriate, or more so, to interpret the scenario as indicating that the structure simply underlies both the lower and higher functions (see Section 2.4 for further discussion). This interpretation seems even more appropriate if the structure appears to underlie the higher function via multiple lower functions (e.g., language via inhibitory control, working memory, and procedural memory), since its role in the higher function would be even more important, or if the structure's role in the lower function(s) shows subspecialization (as a result of evolution or development) for a higher domain (e.g., verbal working memory for language), since its role would then in fact be for that domain.

Similar arguments apply to the second scenario. It would be difficult to show empirically that a given structure underlies only one specific function of a domain or subserves the domain in only one specific context. Even if this were shown, it is not at all obvious that one should interpret the structure as not being part of the domain's underlying brain network (also see Section 2.4). First, the structure is in fact playing a role, even if it might be argued in some cases that the role is not paramount for the domain (e.g., processing emotion-related words in language). Second, many specific roles are essential for a domain (e.g., articulation or certain syntactic relations for language). Third, many structures appear to play several specific roles in (higher) cognitive domains, indicating that the structures' contributions to these domains are quite extensive. For example, even the amygdala, which historically has been tied closely to affect, does not appear to underlie only emotion-related functions in (higher) cognition (**Figure 3**; **Supplemental Table 4**).

2.4. What Is the Nature of (Sub)Cortical Contributions to Cognition?

We have argued above that subcortical contributions to higher as well as lower cognition are not only widespread but also, broadly, both real and important. However, we have not yet discussed just how subcortical (and cortical) structures contribute to cognition, that is, what the overall nature of their contributions may be. Building on our review as well as prior work, in this section we present a theoretical framework of (sub)cortical cognition that may help interpret existing (sub)cortical contributions and predict new ones. The framework is premised on three principles.

According to the first principle, subcortical and other brain structures (or substructures) often subserve core computations that can underlie a wide range of lower and higher functions

(consistent with co-optation), yielding mappings of one (structure) to many (functions) (Genon et al. 2018, Pessoa 2014). For example, something akin to selection may constitute the core computation of the basal ganglia (Ullman et al. 2020), with this computation emerging from the interplay between the direct and indirect pathways within the structure, leading to the disinhibition of selected cortically based representations and the inhibition of other representations (Friend & Kravitz 2014). This basic computation may underlie a wide range of functions (Stephenson-Jones et al. 2011), as suggested by the variety of lower and higher cognitive abilities supported by the basal ganglia such as inhibitory control, working memory, planning and switching, lexical retrieval, procedural memory, and grammar acquisition. In some cases, the selection computation may play a reasonably transparent role in the function (e.g., inhibitory control, working memory, lexical retrieval), while in others the role of selection may be more subtle (Ullman 2016, Ullman et al. 2020). For example, in procedural learning (including of sequences, categories, and grammar), the basal ganglia may be selecting a cortical representation of the predicted outcome (e.g., the next item in a sequence), with learning occurring (modifying cortical-basal ganglia-cortical connections to the selected representation) on the basis of the correctness of the predicted outcome (Ullman et al. 2020). We suggest that the existence of specific and possibly fixed core computations such as selection is widespread among subcortical (sub)structures, though such computations may also be found in portions of cortex, including allocortex and sensory and motor neocortex. In contrast, association neocortex may generally have greater computational and representational flexibility. Based on the evidence from our review as well as the vast literature on cortical function, the interplay between these contributions—as seen, for example, in the relation between the basal ganglia and cortex—appears to be critical for cognition.

The second principle posits that each lower or higher cognitive function relies on a network of supporting structures, constituting mappings of many (structures) to one (function) (Pessoa 2014, Uddin et al. 2019). That is, each function (e.g., working memory, lexical retrieval, grammar, or language) depends on many structures or even many sets of structures, or circuits (or streams or pathways). These (sets of) structures work together in at least two ways. First, consistent with a traditional view of networks, they can play complementary roles. That is, the different structures or circuits make different contributions to a given function or domain. For example, the dorsal and ventral streams are thought to play largely distinct functional roles in

vision or language (Friederici & Gierhan 2013). Second, we suggest that different structures or circuits can also play analogous functional roles and thus may be considered at least partly redundant (Pessoa 2014, Ullman 2016, Ullman et al. 2020). For example, evidence indicates that learning sequences, categories, grammar, and other skills can depend not only on basal ganglia–based procedural memory but also on hippocampal-based declarative memory (both of whose circuits involve cortical and subcortical structures) (Ullman 2016, Ullman et al. 2020).

And according to the third principle, the contributions of (sub)cortical structures and circuits in a functional network are dynamic in that these contributions can vary across conditions, including at different points in time (e.g., during learning versus processing or at different points during learning or processing), in different contexts (e.g., with different kinds of input, with different stimuli, and so on), and even for different individuals or populations (Uddin et al. 2019, Ullman 2016, Ullman et al. 2020). Structures can vary dynamically in their contributions in different ways. First, any single structure or circuit (e.g., the basal ganglia) may play different roles (e.g., inhibitory control, working memory, lexical retrieval, procedural memory) in different conditions (e.g., at different points during learning or processing) in its support of a given function or domain (e.g., language). Second, two or more structures/circuits in a functional network can make both (complementary) and similar (redundant) contributions with respect to each other, with the nature of these contributions varying across conditions. For example, in language, the declarative memory circuit appears to be necessary for learning lexical information, whereas the procedural memory circuit is often relied on for learning grammar, and thus in this case the two circuits play complementary roles, which may indeed occur at different points during language learning (Ullman et al. 2020). However, as we have seen above, learning grammar (and sequences, categories, and other skills) can also rely on declarative memory, and thus the two learning circuits can also play redundant roles in language. Crucially, the extent to which grammar (and these other skills) depends on one versus the other circuit varies dynamically across conditions, including as a function of time (e.g., these skills can rely more on declarative memory early on during learning and more on procedural memory later on), learning context (e.g., more on declarative memory with explicit input, or with slow or no feedback, and more on procedural memory without explicit input or with rapid feedback), and population (e.g., more on declarative than procedural memory for females than males, adults than children, second than first language, and patients with procedural memory deficits such as those with

Parkinson's disease or developmental language disorder as compared to healthy controls, and so on) (Ullman 2016, Ullman et al. 2020).

Thus, our explanatory framework posits that (sub)cortex underlies cognition via a system of many-to-many mappings between structures and functions (many structures supporting each function, as described in the second principle, and each structure supporting many functions, as described in the first principle), in which the contributions of structures to a given function can vary dynamically across conditions. We refer to this framework as the MaMa (many-to-many) dynamic network model of brain and cognition.

According to MaMa, the network underlying a given cognitive function is not composed of a small set of structures [e.g., a few cortical regions in what is often called the language network (Fedorenko & Thompson-Schill 2014, Friederici & Gierhan 2013)] but rather involves numerous subcortical and cortical structures that can play a variety of (complementary and redundant) roles, which can vary across different points in time, different contexts, and different individuals and populations. In this view, all structures contributing to a function are part of the network for that function, even if some may be more critical than others and most if not all also underlie other functions. Certain structures may be more critical at least partly because their core computations underlie more than one role for a given function or domain. For example, both the basal ganglia and Broca's region appear to play multiple roles in language at different levels [e.g., inhibitory control, working memory, procedural memory, grammar, lexical retrieval (Ullman 2006)] and thus are especially important for this domain. We also emphasize that anatomical subspecialization within a structure is consistent with our framework, since different portions of a given structure could become specialized for different functions (e.g., the parallel loops or channels within the basal ganglia that have different cortical connectivity and subserve different motor or cognitive functions) as a result of evolution and/or development, even if the same basic computation underlies these functions (Stephenson-Jones et al. 2011, Ullman 2020, Ullman et al. 2020).

MaMa makes a range of testable predictions. Networks underlying both higher and lower cognitive functions should encompass subcortical as well as cortical structures. Subcortical and other structures in the network will often have underlying core computations. These can support multiple roles for a given function or domain (e.g., inhibitory control and procedural memory for language), as well as different functions/domains altogether, perhaps in part via the same roles

(e.g., inhibitory control and procedural memory for math). A given network includes multiple structures or circuits, which can play complementary and/or redundant roles with respect to each other. The dependence on particular structures or circuits in a given network is dynamic in that it varies across a range of conditions, examples of which are laid out above.

Overall, the MaMa model suggests that our understanding of brain function in cognition could benefit from focusing on particular key issues. First, future research should probe the existence and nature of core computations of subcortical and other structures, how these computations underlie the functions that are subserved by these structures, and how the computations emerge from the underlying neurobiology of the structures, including their molecular/cellular bases and their connectivity (Friend & Kravitz 2014, Stephenson-Jones et al. 2011). Thus, our framework may facilitate the further integration of cognitive neuroscience, computational neuroscience, and molecular/cellular and systems neuroscience. Second, targeted investigations of each functional network should examine which structures play which complementary and/or redundant roles, how the structures interact [e.g., do they compete with each other (Ullman et al. 2020)], under what conditions their roles change dynamically, and how this takes place. All in all, we hope that the MaMa dynamic network model can advance our understanding not only of subcortical contributions to cognition but of brain function in cognition more generally.

3. EXPLORING TERRA INCOGNITA: HOW WE CAN EXPAND THE STRUCTURE–FUNCTION MAP OF SUBCORTICAL COGNITION

Section 2 presents the known structure–function map of subcortical cognition and how it may be interpreted. However, evidently not all aspects of subcortical cognition have already been discovered—particularly given the biases and methodological limitations that have thus far stymied progress in understanding subcortical cognition (**Supplemental Appendices 2** and **3**). In this section, we propose a road map for expanding the map of subcortical cognition. First, we suggest where to look for new structure–function mappings, on the basis of anatomical and evolutionary principles. Second, we lay out just how such mappings can be revealed, by describing specific methodological approaches that are appropriate for examining subcortical

roles in cognition.

3.1. Where to Look: Leveraging Anatomical and Evolutionary Principles

As discussed in Section 1.1, a major reason why subcortical structures are expected to play prominent roles in (higher) cognition is that they are widely connected—via both direct connections and indirect connections through other (sub)cortical structures—to cortical regions that have already been shown to underlie (higher) cognition. It follows straightforwardly that subcortical structures that show strong structural or functional connectivity with cortical regions implicated in particular cognitive functions are also likely to subserve these functions. Such subcortical structures thus constitute excellent candidates for new structure–function mappings. Along the same lines, subcortical (and, indeed, cortical) structures connected to other subcortical structures that underlie particular cognitive functions are also promising candidates for subserving those functions. Thus, overall, previously undiscovered cognitive functions of subcortical structures may be identified by leveraging structural and functional connectivity between these structures and other (sub)cortical structures that have already been shown to underlie cognitive functions.

Evolutionary principles can also be leveraged to discover new structure–function mappings (also see Section 1.1). First, if a subcortical (or cortical) structure in nonhuman animals underlies a particular cognitive function (e.g., working memory) or an apparent precursor to a human (higher) cognitive function (e.g., language), this structure or its homolog might be expected to subserve such functions in humans as well. Second, following the principle of co-optation, (sub)cortical structures may be hijacked for cognitive functions. At least three kinds of subcortical (and cortical) co-optation for cognitive functions may be expected.

First, subcortical structures underlying noncognitive functions in humans or other animals may have been co-opted for cognitive functions, particularly those with analogous computations or characteristics. For example, if a structure is involved in temporal processing, it may have been co-opted to play a role in music processing, whereas if a structure is involved in arousal, it may also play a role in attention. In order to facilitate the identification of noncognitive functions that may have been co-opted for cognition, for each (sub)structure in **Supplemental Tables 1–4** we have included the noncognitive functions (in addition to the cognitive functions) associated with that structure—for example, autonomic, sleep, arousal, sensorimotor, and (noncognitive)

emotion- and motivation-related functions. Note that these noncognitive functions are not shown either in **Supplemental Table 5** or in the figures.

As shown in **Supplemental Tables 1–4**, there are indeed hints that subcortical structures that have not yet been clearly implicated in (higher) cognitive functions might subserve such functions as a result of co-optation from noncognitive functions. For example, the timing functions of the inferior olivary complex in the medulla oblongata signal that this substructure could play similar roles in cognitive domains; in fact, it has been implicated in the perception of complex rhythms, and thus might also contribute to musical cognition (**Supplemental Table 1**). Similarly, the role of the pineal gland in sleep suggests a possible role for this structure in alertness or attention, as indeed may be the case (**Supplemental Table 3**). Other such indicators of possible co-optation are left to the reader.

Second, subcortical structures subserving lower cognitive functions—again, whether in animals or humans—may have been co-opted for higher functions in humans. For example, working memory may be expected to play key roles in language, math, and other higher domains, since these often require information to be temporarily maintained and manipulated (Gathercole & Baddeley 2014, Logie et al. 1994). Thus, subcortical (and cortical) structures supporting working memory may have been co-opted for these higher-level functions, whether or not the structures have become subspecialized for these functions as a result of evolution and/or development. Similarly, higher cognitive domains require substantial learning and thus may be expected to depend on preexisting learning and memory circuits such as declarative and procedural memory, which could have been co-opted to support them (Evans & Ullman 2016, Ullman et al. 2020). For example, the procedural learning roles of the basal ganglia may be expected to underlie learning higher-level cognitive domains. Indeed, evidence already supports this hypothesis for language (Ullman 2016, Ullman et al. 2020), and recent findings suggest that it may hold for aspects of reading and math as well (also see **Supplemental Table 5**) (Earle et al. 2020, Evans & Ullman 2016, Lum et al. 2013).

More generally, subcortical structures that subserve lower (noncognitive or cognitive) functions that have already been shown to underlie particular (higher) cognitive functions constitute especially promising candidates for subserving these functions as well. For example, exactly because working memory and procedural memory appear to play roles in language and other higher domains, any (sub)cortical structure that underlies these lower functions may also

subserve these domains. Along the same lines, if one lower cognitive function (e.g., inhibitory control) has been linked to another (e.g., working memory), then a (sub)cortical structure that has thus far been shown to underlie only one of these may also support the other.

Third, subcortical structures supporting certain higher-level cognitive functions may have been co-opted for other higher functions. For example, given the similarities between language and music, it has been suggested that the neurocognitive underpinnings of one may have been co-opted for the other (Fitch 2005, Patel 2003). Indeed, as our review shows, language and music seem to share some subcortical substrates (e.g., the pons, inferior colliculus, and basal ganglia, including both the caudate nucleus and putamen). Importantly, those subcortical structures that have been implicated thus far in only one of these domains (e.g., the hypothalamus and interpeduncular nucleus in music) serve as potential candidates for the other. Similar comparisons and identification of lacunae in the figures and tables, which are left to the reader, could be examined for any pair of (potentially) related cognitive functions, such as language and reading, or language and math.

Overall, we encourage readers to carefully examine the noncognitive as well as lower and higher cognitive roles of the (sub)structures in the figure and tables with the goal of identifying possible analogous roles in cognitive processing that might have emerged through co-optation. Moreover, because structures can be co-opted for quite different functions (Section 1.1), co-optation may be expected even for aspects of cognition that might not, at first blush, resemble the original functions. Thus, the functions in the figures and tables should be examined with a truly open mind.

3.2. How to Look: A Guide to Appropriate Methodologies for Revealing Subcortical Cognition

Identifying where subcortical contributions to cognition are likely to be found is only a first step in furthering our understanding of subcortical cognition. In addition, just how subcortical cognition is investigated is critical. Appropriate investigations require not only appropriate experimental designs but also appropriate methodologies. Not all techniques or data processing and analysis approaches are equally well suited for assessing whether subcortical structures underlie cognitive functions. As discussed in **Supplemental Appendix 3**, some approaches are inherently less apt for probing subcortical cognition, while others are often misinterpreted for

this purpose or have failed to employ more recent technological, data processing, or analysis advances that can facilitate the investigation of subcortical cognition. Here we provide a brief guide that is designed to help researchers select the most appropriate methods for revealing subcortical cognition.

Various approaches can be used to probe subcortical cognition in functional and structural magnetic resonance imaging (MRI) studies. To begin with, researchers should simply avoid cortical biases, including of analysis and interpretation, of the sort described in **Supplemental Appendices 1**–**3**. Acknowledging the known subcortical–cognitive mappings presented in our review should help in this respect. The use of appropriate techniques is also critical and can overcome some of the methodological limitations described in **Supplemental Appendix 3**. Hypothesis-driven focal scanning of subcortical structures should overcome some of the problems associated with the smaller volumes of many of these structures, such as lower signal-to-noise ratios. The use of higher-field scanners (e.g., 7 T, 10.5 T) would be appropriate, as these yield increased signal-to-noise and contrast-to-noise ratios as well as smaller voxels and thus increased spatial resolution. Functional MRI (fMRI) sequences that substantially increase the signal-to-noise ratio should be used, such as the three-shot spiral dual-echo out-out sequence, which has recently been successfully employed in the investigation of small subcortical structures (Savjani et al. 2018). Appropriate MRI data processing and analytic approaches can also help to more accurately characterize subcortical cognition. For example, anatomical alignment across subjects can be improved by using specialized atlases for subcortical structures (Diedrichsen 2006, Feng et al. 2017). The signal-to-noise ratio can be ameliorated with analytic techniques that reduce motion and other noise, such as measuring nonneuronal physiological fluctuations (e.g., pulse, breathing) simultaneously with fMRI acquisition and controlling for such measures using modeling approaches (Caballero-Gaudes & Reynolds 2017). Finally, even with whole-brain imaging, the use of hypothesis-driven anatomically defined regions of interest and small-volume correction can mitigate the problem of small cluster sizes in corrections for multiple comparisons (Poldrack 2007).

Studies employing transcranial electrical stimulation (tES) methods, such as transcranial direct current stimulation (tDCS), can also use various approaches to help reveal subcortical cognition. Although, as mentioned in **Supplemental Appendix 3**, early current-flow models suggested that the current may be strongest under the anode (Miranda et al. 2006), more recent

computational modeling studies indicate that some conventional tDCS montages induce increased current flow in subcortical structures as well (DaSilva et al. 2015, Parazzini et al. 2011). Moreover, neuroimaging studies have revealed tES-driven alterations of subcortical areas that may be functionally connected to the stimulated cortical area (Chib et al. 2013, Keeser et al. 2011). These modeling and neuroimaging studies suggest that tES can indeed be used to stimulate subcortical structures, with either direct targeting (through specific montages resulting in the current reaching its global maxima in specific structures below the cortical mantle) or indirect targeting (by leveraging cortico-subcortical connectivity) (Santarnecchi et al. 2015). Direct targeting of subcortical structures should use state-of-the-art neuroanatomical target analysis (with high-resolution realistic head models) (DaSilva et al. 2015) to determine optimal electrode montages for subcortical targets, in hypothesis-driven research. The precision of subcortical stimulation can be increased both with structural MRI prior to stimulation, to improve the model, and with confirmatory fMRI during or soon after stimulation. Similarly, indirect targeting of subcortical structures may be improved by performing fMRI prior to tES, in order to determine subject-specific functional connectivity maps and to select cortical stimulation sites on the basis of their functional connections with the subcortical structure(s) of interest. Additionally, as in direct targeting, fMRI may be used during and/or after stimulation to monitor how cortico-subcortical networks are affected by the stimulation (Chib et al. 2013). Finally, recent advances in stimulation devices and the modeling of tES-induced electric fields enable multisite stimulation, which can further support a network-oriented approach for transcranial stimulation (Santarnecchi et al. 2015). Such multisite stimulation may be used to stimulate multiple cortico-subcortical networks simultaneously, for example, by downregulating one network and simultaneously upregulating another.

Intracranial brain recording and stimulation may also be fruitful for the investigation of subcortical cognition. Indeed, the use of both cortical and depth intracranial electrodes, including in subcortical structures, is becoming increasingly common, both presurgically (e.g., for intractable epilepsy and tumors) and therapeutically (e.g., for Parkinson's disease, essential tremor, dystonia, alcohol dependence, Tourette syndrome, depression) (Engel et al. 2005, Hariz & Robertson 2010, Lachaux et al. 2012). For example, the subthalamic nucleus, globus pallidus, and thalamus are all frequent therapeutic targets for movement disorders, including in Parkinson's disease, idiopathic dystonia, and essential tremor (Brandt et al. 2015, Eltahawy et al.

2004, Papavassiliou et al. 2004), while the nucleus accumbens is targeted in obsessive-compulsive disorder, major depression, and alcohol dependence (Bewernick et al. 2012, Heinze et al. 2009, Münte et al. 2008), and the lateral geniculate nucleus in photosensitive epilepsy (Krolak-Salmon et al. 2003). Intracranial recording has a spatial precision comparable to that of fMRI and a temporal precision similar to that of scalp electroencephalography (EEG) and magnetoencephalography (MEG), combined with a higher signal-to-noise ratio than these methods. Therefore, it offers a unique opportunity for direct investigations of the neural mechanisms in focal neuronal populations such as in subcortical structures.

Although the spatial precision of scalp MEG and EEG (M/EEG) is lower than that of fMRI, the temporal precision of these techniques provides a valuable view into brain activity. Importantly, scalp M/EEG signals reflect subcortical as well as cortical activity (**Supplemental Appendix 3**). Indeed, newer M/EEG source estimation models include certain deep brain structures, taking into account their neural characteristics (e.g., cell types, cell density, volume, and distance from the cortical surface), thus enabling the reconstruction of activity in a number of subcortical structures, including the basal ganglia, thalamus, and amygdala (Attal et al. 2007, Krishnaswamy et al. 2017). Moreover, in the absence of source modeling, the mere knowledge of the role of subcortical structures in cognition, and their contributions to the M/EEG signal, should lead to greater caution in attributing M/EEG patterns (e.g., in event-related potential studies) to cortical sources (**Supplemental Appendix 3**).

Transcranial magnetic stimulation (TMS) can also be used to probe for subcortical cognition. First of all, emerging evidence indicates that subcortical structures are affected by cortical TMS via their functional connections (Li et al. 2004, Strafella et al. 2001). Even common commercial TMS devices can be used for indirect targeting when combined with prior fMRI to determine subject-specific cortical stimulation sites on the basis of their functional connections with the subcortical structure(s) of interest (Ulrich et al. 2018). Additionally, as with tES, targeted stimulation efficacy can be monitored by fMRI soon after stimulation. Such prior and confirmatory fMRI has been used successfully for indirect targeting with theta-burst TMS to leverage the functional connectivity between the ventral tegmental area and ventrolateral prefrontal cortex (Ulrich et al. 2018). In addition, recently developed Halo coils are capable of deep TMS, with improved stimulation penetration depths of up to 6 cm, enabling the direct stimulation of subcortical structures (Roth et al. 2007). It must be kept in mind, however, that

even though these new coils are capable of deep TMS, they do not spare the superficial (cortical) regions from being stimulated. Nevertheless, this limitation may be overcome by well-planned experimental designs (e.g., comparisons of cortical versus deep TMS) and by monitoring the effect of TMS on the stimulated cortical and subcortical structures with fMRI.

Note that the use of functional near-infrared spectroscopy (fNIRS) for revealing subcortical cognition is less clear than the techniques discussed above, since in most fNIRS systems the typical depth sensitivity is approximately 1.5 cm (Quaresima et al. 2012). Nevertheless, future advances (e.g., the development of depth-compensated diffuse optical tomography) may overcome this limitation (Tian & Liu 2014), potentially enabling the use of fNIRS in the study of subcortical cognition.

Thus, a variety of current and incipient methodologies may be effectively used to reveal subcortical cognition. This can be done both directly [e.g., with (f)MRI, tES with specific montages, intracranial brain recording and stimulation, EEG and MEG with newer source estimation models, and deep TMS] and indirectly, taking advantage of cortico-subcortical networks (e.g., using tES or TMS to stimulate cortical sites that are functionally connected to subcortical structures of interest).

Finally, we emphasize the importance of hypothesis-driven research in the investigation of subcortical cognition. That is, subcortical structures and their functions should be tested in hypothesis-driven studies, using appropriate methods. Nevertheless, since not all cognitive neuroscience is strongly hypothesis driven, and serendipitous results can be obtained even in hypothesis-driven research, at the very least techniques should be used that do not bias results in favor of cortical (or subcortical) structures, or, if they do, this should be clearly acknowledged.

4. IN CLOSING

Overall, this review aims to stimulate interest in subcortical cognition, in particular for higher cognitive domains and for the multitude of subcortical structures across the brain, even beyond those that have already been reasonably well studied. Enhancing research in subcortical cognition is likely to significantly advance our understanding of both cognitive function and dysfunction, as well as the nature of subcortical structures themselves, and could lead to important translational benefits. The time may be ripe to shine light on the fruit below the rind.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

AUTHOR CONTRIBUTIONS

M.U. conceived the idea for this article. K.J., together with T.E., supervised the searches, which were performed primarily by M.K. and L.S. K.J. and M.U. designed the tables and figures. K.J. and M.K. constructed the tables, with help from T.E. and L.S. and input from M.U. as well as H.B. K.J. and M.K. created the figures, with help from L.S. and input from M.U. M.U. and K.J. wrote the article, with input from T.E. and H.B.

DATA AVAILABILITY

The results from the literature searches are presented in **Supplemental Tables 1–5** and are thus available to all readers.

ACKNOWLEDGMENTS

We thank Alexandra Constantinescu, Rich Ivry, Will Saban, Kyle Shattuck, and John VanMeter for helpful comments. This research was supported by the National Brain Research Program (project 2017–1.2.1-NKP-2017–00002), the Hungarian Scientific Research Fund (NKFIH-OTKA PD 124148), and a Janos Bolyai Research Fellowship from the Hungarian Academy of Sciences to K.J.; by National Science Foundation (NSF) grant DRL 1252453 to T.M.E.; and by National Institutes of Health grant R21 HD 087088 and NSF grants BCS 1439290 and BCS 1940980 to M.T.U.

LITERATURE CITED

Amaral DG, Schumann CM, Nordahl CW. 2008. Neuroanatomy of autism. *Trends Neurosci.* 31:137–45

Attal Y, Bhattacharjee M, Yelnik J, Cottereau B, Lefèvre J, et al. 2007. Modeling and detecting deep brain activity with MEG & EEG. In *2007 29th Annual International Conference of the*

IEEE Engineering in Medicine and Biology Society, pp. 4937–40. Piscataway, NJ: IEEE

Azevedo FA, Carvalho LR, Grinberg LT, Farfel JM, Ferretti RE, et al. 2009. Equal numbers of neuronal and nonneuronal cells make the human brain an isometrically scaled-up primate brain. *J. Comp. Neurol.* 513:532–41

Bassett DS, Sporns O. 2017. Network neuroscience. *Nat. Neurosci.* 20:353–64

Bertolero MA, Yeo BT, D'Esposito M. 2015. The modular and integrative functional architecture of the human brain. *PNAS* 112:E6798–807

Bewernick BH, Kayser S, Sturm V, Schlaepfer TE. 2012. Long-term effects of nucleus accumbens deep brain stimulation in treatment-resistant depression: evidence for sustained efficacy. *Neuropsychopharmacology* 37:1975–85

Brandt J, Rogerson M, Al-Joudi H, Reckess G, Shpritz B, et al. 2015. Betting on DBS: effects of subthalamic nucleus deep brain stimulation on risk taking and decision making in patients with Parkinson's disease. *Neuropsychology* 29:622–31

Caballero-Gaudes C, Reynolds RC. 2017. Methods for cleaning the BOLD fMRI signal. *NeuroImage* 154:128–49

Caligiore D, Pezzulo G, Baldassarre G, Bostan AC, Strick PL, et al. 2017. Towards a systems-level view of cerebellar function: the interplay between cerebellum, basal ganglia, and cortex. *Cerebellum* 16:203–29

Carrera E, Tononi G. 2014. Diaschisis: past, present, future. *Brain* 137:2408–22

Chib VS, Yun K, Takahashi H, Shimojo S. 2013. Noninvasive remote activation of the ventral midbrain by transcranial direct current stimulation of prefrontal cortex. *Transl. Psychiatry* 3:e268

Collins E, Park J, Behrmann M. 2017. Numerosity representation is encoded in human subcortex. *PNAS* 114:E2806–15

Crosson B, McGregor K, Gopinath KS, Conway TW, Benjamin M, et al. 2007. Functional MRI of language in aphasia: a review of the literature and the methodological challenges. *Neuropsychol. Rev.* 17:157–77

DaSilva AF, Truong DQ, DosSantos MF, Toback RL, Datta A, Bikson M. 2015. State-of-art neuroanatomical target analysis of high-definition and conventional tDCS montages used for migraine and pain control. *Front. Neuroanat.* 9:89

De Smet HJ, Paquier P, Verhoeven J, Mariën P. 2013. The cerebellum: its role in language and

related cognitive and affective functions. *Brain Lang.* 127:334–42

Dehaene S, Cohen L. 2007. Cultural recycling of cortical maps. *Neuron* 56:384–98

Dehaene S, Molko N, Cohen L, Wilson AJ. 2004. Arithmetic and the brain. *Curr. Opin. Neurobiol.* 14:218–24

Delazer M, Domahs F, Lochy A, Karner E, Benke T, Poewe W. 2004. Number processing and basal ganglia dysfunction: a single case study. *Neuropsychologia* 42:1050–62

Dickstein SG, Bannon K, Castellanos FX, Milham MP. 2006. The neural correlates of attention deficit hyperactivity disorder: an ALE meta-analysis. *J. Child Psychol. Psychiatry* 47:1051–62

Diedrichsen J. 2006. A spatially unbiased atlas template of the human cerebellum. *NeuroImage* 33:127–38

Draganski B, Kherif F, Klöppel S, Cook PA, Alexander DC, et al. 2008. Evidence for segregated and integrative connectivity patterns in the human basal ganglia. *J. Neurosci.* 28:7143–52

Earle FS, Del Tufo SN, Evans TM, Lum JA, Cutting LE, Ullman MT. 2020. Domain-general learning and memory substrates of reading acquisition. *Mind Brain Educ.* 14:176–86

Eltahawy HA, Saint-Cyr J, Giladi N, Lang AE, Lozano AM. 2004. Primary dystonia is more responsive than secondary dystonia to pallidal interventions: outcome after pallidotomy or pallidal deep brain stimulation. *Neurosurgery* 54:613–21

Engel AK, Moll CK, Fried I, Ojemann GA. 2005. Invasive recordings from the human brain: clinical insights and beyond. *Nat. Rev. Neurosci.* 6:35–47

Evans TM, Ullman MT. 2016. An extension of the procedural deficit hypothesis from developmental language disorders to mathematical disability. *Front. Psychol.* 7:1318

Fedorenko E, Thompson-Schill SL. 2014. Reworking the language network. *Trends Cogn. Sci.* 18:120–26

Feng X, Altarelli I, Monzalvo K, Ding G, Ramus F, et al. 2020. A universal reading network and its modulation by writing system and reading ability in French and Chinese children. *eLife* 9:e54591

Feng X, Deistung A, Dwyer MG, Hagemeier J, Polak P, et al. 2017. An improved FSL-FIRST pipeline for subcortical gray matter segmentation to study abnormal brain anatomy using quantitative susceptibility mapping (QSM). *J. Magn. Reson. Imaging* 39:110–22

Fitch WT. 2000. The evolution of speech: a comparative review. *Trends Cogn. Sci.* 4:258–67

Fitch WT. 2005. The evolution of language: a comparative review. *Biol. Philos.* 20:193–203

Friederici AD, Gierhan SM. 2013. The language network. *Curr. Opin. Neurobiol.* 23:250–54

Friend DM, Kravitz AV. 2014. Working together: basal ganglia pathways in action selection. *Trends Neurosci.* 37(6):301–3

Gathercole SE, Baddeley AD. 2014. *Working Memory and Language*. New York: Psychology

Genon S, Reid A, Langner R, Amunts K, Eickhoff SB. 2018. How to characterize the function of a brain region. *Trends Cogn. Sci.* 22:350–64

Givens BS, Olton DS. 1990. Cholinergic and GABAergic modulation of medial septal area: effect on working memory. *Behav. Neurosci.* 104:849–55

Gould SJ, Vrba ES. 1982. Exaptation—a missing term in the science of form. *Paleobiology* 8:4–15

Hage SR, Nieder A. 2016. Dual neural network model for the evolution of speech and language. *Trends Neurosci.* 39:813–29

Haines DE. 2004. *Neuroanatomy: An Atlas of Structures, Sections, and Systems*. New York: Lippincott, Williams & Wilkins

Hansel C, Linden DJ, D'Angelo E. 2001. Beyond parallel fiber LTD: the diversity of synaptic and non-synaptic plasticity in the cerebellum. *Nat. Neurosci.* 4:467–75

Hariz MI, Robertson MM. 2010. Gilles de la Tourette syndrome and deep brain stimulation. *Eur. J. Neurosci.* 32:1128–34

Heinze H-J, Heldmann M, Voges J, Hinrichs H, Marco-Pallares J, et al. 2009. Counteracting incentive sensitization in severe alcohol dependence using deep brain stimulation of the nucleus accumbens: clinical and basic science aspects. *Front. Hum. Neurosci.* 3:22

Hunt S, Low J, Burns K. 2008. Adaptive numerical competency in a food-hoarding songbird. *Proc. R. Soc. B* 275:2373–79

Janata P. 2005. Brain networks that track musical structure. *Ann. N. Y. Acad. Sci.* 1060:111–24

Jankovic J. 2008. Parkinson's disease: clinical features and diagnosis. *J. Neurol. Neurosurg. Psychiatry* 79:368–76

Ji JL, Spronk M, Kulkarni K, Repovš G, Anticevic A, Cole MW. 2019. Mapping the human brain's cortical–subcortical functional network organization. *NeuroImage* 185:35–57

Johari K, Walenski M, Reifegersete J, Ashrafi F, Behroozmand R, et al. 2019. A dissociation between syntactic and lexical processing in Parkinson's disease. *J. Neurolinguistics* 51:221–

35

Keeser D, Meindl T, Bor J, Palm U, Pogarell O, et al. 2011. Prefrontal transcranial direct current stimulation changes connectivity of resting-state networks during fMRI. *J. Neurosci.* 31:15284–93

Kotz SA, Schwartze M. 2010. Cortical speech processing unplugged: a timely subcortico-cortical framework. *Trends Cogn. Sci.* 14:392–99

Koziol LF, Budding DE. 2009. *Subcortical Structures and Cognition: Implications for Neuropsychological Assessment*. Berlin: Springer

Kreitzer AC, Malenka RC. 2008. Striatal plasticity and basal ganglia circuit function. *Neuron* 60:543–54

Krishnaswamy P, Obregon-Henao G, Ahveninen J, Khan S, Babadi B, et al. 2017. Sparsity enables estimation of both subcortical and cortical activity from MEG and EEG. *PNAS* 114:E10465–74

Krolak-Salmon P, Hénaff MA, Tallon-Baudry C, Yvert B, Guénot M, et al. 2003. Human lateral geniculate nucleus and visual cortex respond to screen flicker. *Ann. Neurol.* 53:73–80

Lachaux J-P, Axmacher N, Mormann F, Halgren E, Crone NE. 2012. High-frequency neural activity and human cognition: past, present and possible future of intracranial EEG research. *Prog. Neurobiol.* 98:279–301

Lee DJ, Gurkoff GG, Izadi A, Berman RF, Ekstrom AD, et al. 2013. Medial septal nucleus theta frequency deep brain stimulation improves spatial working memory after traumatic brain injury. *J. Neurotrauma* 30:131–39

Leszczyński M, Staudigl T. 2016. Memory-guided attention in the anterior thalamus. *Neurosci. Biobehav. Rev.* 66:163–65

Li X, Nahas Z, Kozel FA, Anderson B, Bohning DE, George MS. 2004. Acute left prefrontal transcranial magnetic stimulation in depressed patients is associated with immediately increased activity in prefrontal cortical as well as subcortical regions. *Biol. Psychiatry* 55:882–90

Logie RH, Gilhooly KJ, Wynn V. 1994. Counting on working memory in arithmetic problem solving. *Mem. Cogn.* 22:395–410

Lum JA, Ullman MT, Conti-Ramsden G. 2013. Procedural learning is impaired in dyslexia: evidence from a meta-analysis of serial reaction time studies. *Res. Dev. Dis.* 34:3460–76

MacLean PD. 1988. Triune brain. In *Comparative Neuroscience and Neurobiology*, ed. LN Irwin, pp. 126–28. Berlin: Springer

Mah Y-H, Husain M, Rees G, Nachev P. 2014. Human brain lesion–deficit inference remapped. *Brain* 137:2522–31

Mariën P, Ackermann H, Adamaszek M, Barwood CH, Beaton A, et al. 2014. Consensus paper: language and the cerebellum: an ongoing enigma. *Cerebellum* 13:386–410

Mariën P, Baillieux H, De Smet HJ, Engelborghs S, Wilssens I, et al. 2009. Cognitive, linguistic and affective disturbances following a right superior cerebellar artery infarction: a case study. *Cortex* 45:527–36

Martinez-Gonzalez C, Bolam JP, Mena-Segovia J. 2011. Topographical organization of the pedunculopontine nucleus. *Front. Neuroanat.* 5:22

Miranda PC, Lomarev M, Hallett M. 2006. Modeling the current distribution during transcranial direct current stimulation. *Clin. Neurophysiol.* 117:1623–29

Münte TF, Heldmann M, Hinrichs H, Marco-Pallares J, Krämer UM, et al. 2008. Nucleus accumbens is involved in human action monitoring: evidence from invasive electrophysiological recordings. *Front. Hum. Neurosci.* 2:11

Murphy E, Hoshi K, Benítez-Burraco A. 2021. Subcortical syntax: reconsidering the neural dynamics of language. PsyArXiv. https://psyarxiv.com/29cjw/

Nicolson RI, Fawcett AJ. 2007. Procedural learning difficulties: reuniting the developmental disorders? *Trends Neurosci.* 30:135–41

Nioche C, Cabanis E, Habas C. 2009. Functional connectivity of the human red nucleus in the brain resting state at 3T. *Am. J. Neuroradiol.* 30:396–403

Noback CR, Strominger NL, Demarest RJ, Ruggiero DA. 2005. *The Human Nervous System: Structure and Function*. Berlin: Springer

Papavassiliou E, Rau G, Heath S, Abosch A, Barbaro NM, et al. 2004. Thalamic deep brain stimulation for essential tremor: relation of lead location to outcome. *Neurosurgery* 54:1120–30

Parazzini M, Fiocchi S, Rossi E, Paglialonga A, Ravazzani P. 2011. Transcranial direct current stimulation: estimation of the electric field and of the current density in an anatomical human head model. *IEEE Trans. Biomed. Eng.* 58:1773–80

Parvizi J. 2009. Corticocentric myopia: old bias in new cognitive sciences. *Trends Cogn. Sci*.

13:354–59

Patel AD. 2003. Language, music, syntax and the brain. *Nat. Neurosci.* 6:674–81

Pessoa L. 2014. Understanding brain networks and brain organization. *Phys. Rev.* 11:400–35

Poldrack RA. 2007. Region of interest analysis for fMRI. *Soc. Cogn. Affect. Neurosci.* 2:67–70

Postuma RB, Dagher A. 2006. Basal ganglia functional connectivity based on a meta-analysis of 126 positron emission tomography and functional magnetic resonance imaging publications. *Cereb. Cortex* 16:1508–21

Quaresima V, Bisconti S, Ferrari M. 2012. A brief review on the use of functional near-infrared spectroscopy (fNIRS) for language imaging studies in human newborns and adults. *Brain Lang.* 121:79–89

Rorden C, Karnath H-O. 2004. Using human brain lesions to infer function: a relic from a past era in the fMRI age? *Nat. Rev. Neurosci.* 5:812–19

Roşca EC. 2009. Arithmetic procedural knowledge: a cortico-subcortical circuit. *Brain Res.* 1302:148–56

Roth Y, Amir A, Levkovitz Y, Zangen A. 2007. Three-dimensional distribution of the electric field induced in the brain by transcranial magnetic stimulation using figure-8 and deep H-coils. *J. Clin. Neurophysiol.* 24:31–38

Santarnecchi E, Brem A-K, Levenbaum E, Thompson T, Kadosh RC, Pascual-Leone A. 2015. Enhancing cognition using transcranial electrical stimulation. *Curr. Opin. Behav. Sci.* 4:171–78

Sara SJ. 2009. The locus coeruleus and noradrenergic modulation of cognition. *Nat. Rev. Neurosci.* 10(3):211–23

Saunders A, Oldenburg IA, Berezovskii VK, Johnson CA, Kingery ND, et al. 2015. A direct GABAergic output from the basal ganglia to frontal cortex. *Nature* 521:85–89

Savjani RR, Katyal S, Halfen E, Kim JH, Ress D. 2018. Polar-angle representation of saccadic eye movements in human superior colliculus. *NeuroImage* 171:199–208

Schmahmann JD, Guell X, Stoodley CJ, Halko MA. 2019. The theory and neuroscience of cerebellar cognition. *Annu. Rev. Neurosci.* 42:337–64

Sihvonen AJ, Ripollés P, Leo V, Rodríguez-Fornells A, Soinila S, Särkämö T. 2016. Neural basis of acquired amusia and its recovery after stroke. *J. Neurosci.* 36:8872–81

Stephenson-Jones M, Samuelsson E, Ericsson J, Robertson B, Grillner S. 2011. Evolutionary

conservation of the basal ganglia as a common vertebrate mechanism for action selection. *Curr. Biol.* 21(13):1081–91

Strafella AP, Paus T, Barrett J, Dagher A. 2001. Repetitive transcranial magnetic stimulation of the human prefrontal cortex induces dopamine release in the caudate nucleus. *J. Neurosci.* 21:RC157

Tian F, Liu H. 2014. Depth-compensated diffuse optical tomography enhanced by general linear model analysis and an anatomical atlas of human head. *NeuroImage* 85:166–80

Tsivilis D, Vann SD, Denby C, Roberts N, Mayes AR, et al. 2008. A disproportionate role for the fornix and mammillary bodies in recall versus recognition memory. *Nat. Neurosci.* 11:834–42

Uddin LQ, Yeo BT, Spreng RN. 2019. Towards a universal taxonomy of macro-scale functional human brain networks. *Brain Topogr.* 32:926–42

Ullman MT. 2004. Contributions of memory circuits to language: the declarative/procedural model. *Cognition* 92:231–70

Ullman MT. 2006. Is Broca's area part of a basal ganglia thalamocortical circuit? *Cortex* 42:480–85

Ullman MT. 2007. The biocognition of the mental lexicon. In *The Oxford Handbook of Psycholinguistics*, ed. MG Gaskell, pp. 267–86. Oxford, UK: Oxford Univ. Press

Ullman MT. 2016. The declarative/procedural model: a neurobiological model of language learning, knowledge, and use. In *Neurobiology of Language*, ed. G Hickok, SL Small, pp. 953–68. Amsterdam: Elsevier

Ullman MT. 2020. The declarative/procedural model: a neurobiologically motivated theory of first and second language. In *Theories in Second Language Acquisition*, ed. B VanPatten, GD Keating, S Wulff, pp. 128–61. New York: Routledge

Ullman MT, Earle FS, Walenski M, Janacsek K. 2020. The neurocognition of developmental disorders of language. *Annu. Rev. Psychol.* 71:389–417

Ulrich M, Lorenz S, Spitzer MW, Steigleder L, Kammer T, Grön G. 2018. Theta-burst modulation of mid-ventrolateral prefrontal cortex affects salience coding in the human ventral tegmental area. *Appetite* 123:91–100

Vann SD, Aggleton JP. 2004. The mammillary bodies: two memory systems in one? *Nat. Rev. Neurosci.* 5:35–44

Vargha-Khadem F, Gadian DG, Copp A, Mishkin M. 2005. *FOXP2* and the neuroanatomy of speech and language. *Nat. Rev. Neurosci.* 6:131–38

Whaley NR, Fujioka S, Wszolek ZK. 2011. Autosomal dominant cerebellar ataxia type I: a review of the phenotypic and genotypic characteristics. *Orphanet J. Rare Dis.* 6:33

Wright NF, Vann SD, Aggleton JP, Nelson AJ. 2015. A critical role for the anterior thalamus in directing attention to task-relevant stimuli. *J. Neurosci.* 35:5480–88

Yeo BT, Krienen FM, Sepulcre J, Sabuncu MR, Lashkari D, et al. 2011. The organization of the human cerebral cortex estimated by intrinsic functional connectivity. *J. Neurophysiol.* 106:1125–65