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EMERGENTISM AND LANGUAGE DISORDERS

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Introduction

The human brain is the most complicated structure in the known universe, and language is the most complex mental function, relying on large parts of the cerebral cortex and midbrain. Additional complexity arises from the social and developmental forces that produce continual variation in the shapes of words, sounds, and communications. It is difficult to imagine a full account of language disorders that does not come to grips with this great complexity, both in terms of neural processing and the shape of language itself.

The biggest challenge facing a theory of language disorder is that there are so many alternative forms of language across individuals and so many possible expressions of language disorder. Given the complexity of language, there are good reasons to expect that patterns in language disorders should be at least as complex as disorders of other biological systems, such as the immune system or the skeletal system. Within this complex system, there may well be pivotal mechanisms that trigger a disorder. However, the behavioral and neurological effects of that pivotal mechanism are surely going to be modified by other components of the system.

Traditionally, there have been two competing approaches to explaining language disorders. The nativist approach emphasizes the ways in which variations in genetic structures can lead to language disorder. For example, studies of mutations in the FOXP2 gene in the KE family (Fisher & Scharff, 2009) have been shown to impact aspects of language production, although they also impact motor behaviors and control more generally (Vargha-Khadem, Gadian, Copp, & Mishkin, 2005). Nativist accounts typically view language as controlled by distinct brain modules that function automatically with non-interactive informational encapsulation (Fodor, 1983). They also emphasize the structuring of language through recursive rule systems (Hauser, Chomsky, & Fitch, 2002) which are viewed as the core event in language evolution.

In contrast, empiricist accounts of language disorders emphasize the extent to which language learning and processing rely on general cognitive resources (Christiansen & Chater, 2008; Elman et al., 1996) and environmental inputs. These accounts see neural processing as involving dynamic associations between highly interactive areas (McClelland, Mirman, & Holt, 2006). They view language learning as the acquisition of constructions, rather than rules (Goldberg, 2006), and as being driven by usage (Bybee, 2010) and statistics (Conway, 2020).

These two approaches have made important contributions to our understanding of language, brain, and disorders. However, they also suffer from core weaknesses. The nativist emphasis on modularity fails to account for the dynamic interplay of neural functioning (Hagoort, 2013) during

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actual language processing. Nativist emphasis on syntactic rules and recursion as the core features of human language fails to account for the equally important roles of articulation, audition, lexicon, and interaction in language functioning and language evolution (MacWhinney, 2005b).

Empiricist accounts have difficulty assigning a role to genetic and epigenetic causes of language disorders, such as familial inheritance of stuttering (Frigerio-Domingues & Drayna, 2017). Although genetic determination may account for not more that 16% of cases of stuttering, it is still useful to understand these relations and the relations between this type of direct causation and other etiologies. Another weakness of some empiricist accounts is their failure to fully consider developmental or epigenetic changes in language functioning across the lifespan.

Often the opposition between nativism and empiricism is characterized by questions such as "how much of a given behavior is due to nature and how much is due to nurture". This poses the problem as a forced choice between the two approaches. We can avoid this forced choice by saying that we need to take the best insights from each, but then the question is how exactly to do this. The most promising way to achieve this integration involves consideration of both language and the brain as complex dynamic systems (Beckner et al., 2009). To best explore this option, we need consider the theory of emergentism with particular attention to the theory of neuroemergentism (Hernandez et al., 2019). This framework has the great advantage of allowing us to piece together a view that incorporates insights from a diversity of component models.

Emergentism

Scientific approaches to large complex systems rely on the formulation of multiple interlocking component theories. Geological accounts of the past and present of our planet rely on theories about radioactive decay, crystal formation, element separation in magma, state transitions, fluid dynamics, crustal movements, vulcanism, and plate tectonics. Similarly, to understand language processing and language disorders we need to invoke many well-developed theories, ranging from gene expression (Wong, Morgan-Short, Ettlinger, & Zheng, 2012) to the impact of stress on stuttering (Bloodstein & Bernstein Ratner, 2008) or the role of corrective feedback in children's language learning (MacWhinney, 2004). Emergentism provides a way of linking these already developed component theories into a coherent whole. It does this by articulating the role of four core analytic frameworks: competition, mechanisms as constraints on structures, emergent levels, and time/process frames (MacWhinney, 2015).

Competition

The theory of competition builds on Darwin's (1859) vision of evolution and adaptation as arising from the operation of three processes: proliferation, competition, and selection. For individual speakers, proliferation of language forms and functions is driven by the rich language variety to which they are exposed. Learners pick up thousands of sound forms, referents, words, multiword expressions, constructions, syntactic patterns, and conversational practices. These forms and functions vary markedly based on dialect, genre, and speaker variation (Hymes, 1962). The learner's task is to deal with the competition and cooperation created by this great proliferation. This is a task not just for language, but for all cognitive processing (Rosenbaum, 2015).

During both comprehension and production, the learner must rely on a system of cue validity to select winning forms and functions. The component model that articulates this process is the Unified Competition Model (MacWhinney, 2021). This model provides a functionalist account of how languages are learned across the lifespan and how they are processed in real time. Research based on this model has shed light on aspects of first language learning, second language learning, bilingual processing, developmental language disorders, and language loss in aphasia. The model's fundamental claim is that, during comprehension, alternative interpreta-

tions compete online in terms of their relative cue validity. Similarly, during production, alternative expressions compete in terms of the validity of their match to intentions. To probe these various competitions, researchers have used multifactorial experimental designs to measure the process of cue competition. As summarized in MacWhinney (2021) and elsewhere, the predictions of the model have been uniformly supported across four decades of research involving 15 different languages (MacWhinney & Bates, 1989).

We can supplement behavioral evidence for competition with evidence from Cognitive Neuroscience. Computational models of brain functioning rely on facts about lateral inhibition (Kohonen, 2001), cell assembly structure (Hebb, 1949; Pulvermüller, 2003), and connectivity (Hickok, 2009; Valiant, 2014) to explain how competition is processed both locally and between cortical areas. These models have been articulated for many levels of language processing, including lexical selection, code-switching, sentence processing, and speech recognition.

Models of neural competition can help us understand both speech errors and stuttering. The fact that speakers can detect and subsequently correct their speech errors (Maclay & Osgood, 1959) has led researchers to postulate a system that monitors or compares candidate output forms with the auditory shape of the intended target (Roelofs, 2011). However, evidence from the ERN (error-related negativity) component of the EEG suggests that the competition between candidate forms arises immediately during the process of initial form activation (Nozari & Novick, 2017). Nozari and Hepner (2019) show how a signal detection model of the competition between lexical forms can account for observed details in the pattern of speech errors. This model views hesitation pauses and retraces as arising from a lower level of confidence regarding the outcome of a competition on the lexical level. The drift-diffusion model (Ratcliff & McKoon, 2008) provides a similar account.

Competition must also be involved in stuttering, although in a way that is more complex than what we see in speech errors from fluent speakers. One possibility is that problems in the cortico-basal ganglia-thalamocortical (cortico-BG) loop for motor activation could further exacerbate problems arising from competitions between lexical items (Chang & Guenther, 2020). The cortico-BG loop account can then be integrated with other component models, including the model for speech errors proposed by Nozari and Hepner (2019), Guenther's DIVA feedback model (Guenther & Vladusich, 2012), the theory of dopamine action on the basal ganglia (Civier, Bullock, Max, & Guenther, 2013), and the model of segregated basal ganglia circuitry (Alexander, DeLong, & Strick, 1986). The need to piece together component models in this way is driven by the complexity of language and the brain.

Mechanisms constraining structures

Human language is structured into a series of interactive levels, including audition, articulation, lexicon, morphology, syntax, discourse, narrative, and conversation. On each of these levels, we find that structures emerge from the impact of mechanisms that impose constraints on possible forms and structural levels.

Nature abounds with examples of emergent structures. Whether we are talking about the shape and properties of water, soap bubbles, ocean tides, honeycomb cells, protein molecules, optical dominance columns, mental representations, neurolinguistic modules, linguistic forms, or social groups, we can view all structures in the natural world as emerging from the force of mechanisms that impose constraints on how these structures can be configured.

As Mill (1859) noted, water provides a perfect example. To produce water from hydrogen and oxygen, one must apply a spark of energy to break the covalent bonds. After that, the process of molecular formation produces its own energy, and the reaction will go to completion. The first emergent property of this new molecule is its polarity, which then responds to constraints on the molecular level to produce hydrogen-bonding of each water molecule with up to four additional

water molecules. These new links then produce water's high surface tension as a further emergent property, as well as its high thermal conductivity, specific heat capacity, heat of vaporization, and heat of fusion. As water accumulates in larger bodies like lakes and oceans, these local properties shape new emergent patterns such as snowflakes, rain drops, ocean currents, glaciers, thunderstorms, and many other features of our planet and its climate.

Language is shaped by a wide range of constraints, and researchers have formulated accounts and models for each of these constraints. These models constitute important components of the overall emergentist framework. Here we can list the mechanisms or constraints that have been most thoroughly studied.

1. Functional mapping. Language forms are designed to express communicative functions. As Bates and MacWhinney (1981) noted, "the forms of natural languages are created, governed, constrained, acquired and used in the service of communicative functions." This is a core constraint in accounts such as the Unified Competition Model or Construction Grammar (Goldberg, 2006).
2. Conversational determination. Possible lexical forms are constrained by the possibilities of achieving systematic coreference with other speakers (Goldstone, 2002). Syntactic structures adapt to frequent conversational patterns (Ochs, Schegloff, & Thompson, 1996). Possible conversational patterns are shaped by social practices (Goodwin, 2013) and preference management (Korniol, 1995).
3. Embodied determination. The functions expressed by language forms are grounded on our embodied experiences as human actors (MacWhinney, 2005a).
4. Generalization. Language forms organize into groups or gangs, based on similarity and this organization then produces patterns that can generalize to new forms (McClelland, 2015). Generalization plays a major role in systems as diverse as morphological categories, metaphor, constructions, narrative, and phonotactics.
5. Self-organization and error correction. The formation of groups and new patterns can arise either through self-organization in which forms that behave similarly are grouped together (Kohonen, 2001) or through error correction in which we compare what we say or understand with what we should have said or should have understood (Berwick, 1987).
6. Simplicity and expressiveness. The mapping of forms to functions is governed by the operation of two major competing constraints: simplicity and expressiveness. Language seeks to be simple by creating a minimal number of forms for a function. At the same time, it seeks to be expressive by creating forms for fine-grained differences in meaning. Much of language complexity arises from the competition between these two constraints (MacWhinney, Malchukov, & Moravcsik, 2014).
7. Physical constraints. The formation of articulatory gestures and their linkage into syllables and words are constrained by the mechanics of the vocal system and neural control (Browman & Goldstein, 1992).
8. Item-based patterns. The linking of words into sentences is constrained by the operation of argument slots on lexical forms (MacWhinney, 2014).
9. Incrementality. Possible syntactic patterns are constrained by the incremental "now or never" functioning of sentence processing (Christiansen & Chater, 2016; O'Grady, 2005).
10. Perceptual recording. Both infants and adults can apply general-purpose mechanisms to record and learn sequential patterns (Conway, 2020).
11. Chunking. On each structural level, forms and functions that occur together frequently are treated as a single chunk for memory storage and processing (Hebb, 1949; Newell, 1990).
12. Resonance. Apart from chunking within levels, the integration of new information with old information and across levels can lead to strengthening of associations (Schlichting & Preston, 2015), greater fluency (Dominey & Boussaoud, 1997), and improved recall (Pavlik & Anderson, 2008).

13. Connectivity and localization. The need to communicate information across neural regions constrains the localization of processing levels to areas that are well connected with other areas required for their computations (Dronkers, 2011). Moreover, the detailed shape of cortical areas maintains a topological connection to the body in terms of retinotopic, tonotopic, and somatotopic maps. This principle of topological mapping extends even further to control areas such as the thalamus and hippocampus.
14. Imitation. We can learn both forms and functions by recording speech and then imitating it (Whitehurst & Vasta, 1975). Imitation or copying is a fundamental mechanism for usage-based linguistics (Diessel, 2017).
15. Plasticity. Processes of neural reuse and plasticity permit reorganization of neural functioning (Zerilli, 2022).

As we will see in the next section, these mechanisms and others not listed in this overview operate in different ways across the emergent levels of language structure. A given language disorder could impact relative reliance on any given mechanism. For example, the motor disorder in the KE family caused by SNP (single nucleotide polymorphism) mutations of the Fox2P gene impact phonology, morphology, and syntax, but in different ways for each level. Moreover, these mechanisms interact in different ways for different disorders. For example, problems with syntactic control lead to argument omissions in non-fluent aphasia (Thompson et al., 2013), whereas they lead to disfluencies in stuttering. The fact that some linguistic structures are particularly vulnerable echoes findings from linguistic analysis regarding the emergence of complexity in syntax (Chomsky, 2007; Culicover, 2013).

Emergent levels

Once new forms emerge from the actions of constraints, they become subject to new constraints. This interplay of forms, constraints, and levels can be illustrated by examining the process of protein folding which goes through four structural levels to determine a protein's final folded form. On the primary level, a simple chain of amino acids emerges from the ribosome. This structure is constrained or shaped by the operation of messenger RNA and transfer RNA. On the secondary level, constraints from hydrogen bonding across the amino acid chain serve to create either helices or pleated sheets, based on the nature of the sequences of amino acids derived from the primary level. On the tertiary level, the helices and pleated sheets twist into other forms based on the new constraints of hydrophobic and hydrophilic attractions. On the quaternary level, multiple polypeptide chains formed on the tertiary level combine further to produce still more complex 3-D patterns appropriate for functioning, such as the ability of hemoglobin in transport oxygen, or the ability of antibodies to engulf viruses. On each of these four levels, folding is further guided or constrained by catalysts and molecular chaperones. Once proteins are available for neuronal functioning, they can further determine the structure of neurons, transmitters, and hormones. As neurons group on higher levels, they are subject to constraints from neuronal packing, local and distal connectivity patterns, activation thresholds, gang effects, and other properties of neural assemblies and areas.

This analysis of the emergence of structural levels through the creation of new forms subject to new constraints also applies to language and language learning. In children's language learning, the shape of the basic levels of linguistic structure emerges from the operation of constraints on those levels. One component of the theory of neuroemergentism (MacWhinney, 2019) is that data tends to self-organize into a particular brain area based on the connections of that area with other areas that optimize functioning. For example, the organization of sound patterns into auditory cortex is facilitated by its linkage to medial geniculate body of the thalamus, the planum temporale, and lexical processing in the ventral pathway of the temporal lobe. Auditory cortex is also well con-

nected to the dorsal pathway for support of motor aspects of speech perception (Hickok, 2009). In children with early deafness, this pathway receives only weak input, thereby allowing it to reorganize for visual motion detection (Shiell, Champoux, & Zatorre, 2015).

The emergence of distinct areas for lexical processing is also driven by the connectivity constraints on these areas (Gow, 2012). This leads to a concentration of phonetic information in the inferior parietal, a concentration of core lexical information in the superior temporal sulcus and medial temporal gyrus, and a concentration of item-based syntactic frame information in the anterior temporal lobe. Similar patterns of connectivity determine structuring for the levels of articulation, clausal syntax, mental model processing, and control of conversation. Note that self-organization may occur during neural organization before birth, as the fetus is able to process the mother's voice while in the womb (Webb, Heller, Benson, & Lahav, 2015).

This view of the emergent and adaptable nature of processing areas contrasts with the nativist view of genetically fixed, encapsulated neural modules (Fodor, 1983; Galton, 1883; Pinker, 1994). It also provides us with a fuller understanding of the complex nature of language disorders. During online production, these multiple structural levels interact dynamically. As we listen to messages from our conversational partners, we are also formulating our own contributions. These ideas are shaped into clauses as we activate words bit by bit into phrases and begin to articulate these ideas, even before all the components of the utterance are fully formed. The interactions of these levels involves just-in-time processing (Christiansen & Chater, 2016) along with gating between areas to make sure that articulations are not begun until the underlying message is at least approximately correct. These demands underscore the key role of fluency in language production and comprehension. Preschoolers are still piecing together basic elements for fluency and, even as adults, we can become disfluent when the components of our messages are not well practiced.

Timescale/process constraints

In a process like protein folding, the movement across structural levels occurs within a span varying from an hour to microseconds with larger proteins taking longer to fold (Naganathan & Muñoz, 2005). For language, new structures emerge across three very divergent timescales. One is the timescale of learning in the individual which extends from seconds to decades. The second is the timescale of language change in the community which extends from decades to centuries. The third is the timescale of language evolution which includes thousands and even millions of years.

For understanding language disorders, the most important timescales are those that impact the individual speaker. Here, we can consider ideas from the theory of neuroemergentism (Hernandez et al., 2019) which focuses on three processes leading to cortical reorganization. The first is what Dehaene calls "cultural recycling" (Dehaene & Cohen, 2007). This is the process involved in the reshaping of the left face form area (FFA) to become the visual word form area (VWFA) used in reading. This type of repurposing of an area depends both on the cytoarchitectonic structure of an area and its pattern of connectivity with other areas involved in a type of processing. In the case of learning to read, the ability of the left FFA to encode precise visual patterns, along with its connectivity to the ventral stream of language processing (Hickok & Poeppel, 2004) allow it to take over the function of visual word processing.

An equally remarkable example of recycling involves the use of IFG (inferior frontal gyrus, Broca's area) by signers to support syntactic processing in sign language and the use of STG (superior temporal gyrus, Wernicke's area) to support lexical processing in sign (Hickok, Bellugi, & Klima, 2001). Similar effects arise through increases in the part of the hippocampus dedicated to route finding for London taxicab drivers (Woollett & Maguire, 2011), increases in a variety of auditory areas as a result of musical training (Olszewska, Gaca, Herman, Jednoróg, & Marchewka, 2021), and greater functional connectivity as a result of learning new words in Chinese as a second language (Li, Legault, & Litcofsky, 2014). Recycling may have important consequences for lan-

guage disorders. On the one hand, inadequate language input could lead to temporary deficits in language functioning (Hart & Risley, 1995). On the other hand, recycling suggest that deficits can be mitigated or reversed through training (Recanzone & Merzenich, 1993) and exposure (Roberts, Rosenfeld, & Zeisel, 2004).

A second neuroemergentist process involves neural reuse. Looking at fMRI activation data for 968 brain regions, Anderson (2010) found that brain regions are often involved in 20 different tasks or more and that, on average, a brain region is active in 4.32 clearly different domains. This pattern of reuse for multiple functions is a fundamental aspect of emergence in biology, as noted by Darwin (1862, p. 348) for organs or West-Eberhard (2003) for processes in epigenetic and phenotypic control of developmental plasticity. The theory of neuronal recycling is highly compatible with the theory of neural reuse. If an area can serve multiple functions in alternative configurations of functional neural circuits, then recycling can be directly supported during ontogenesis and later.

The third neuroemergentist process involves what Johnson (2011) calls "interactive specialization." This framework elaborates on the motto from Bates (1999) that, "modules are made, not born." For example, Edelman (1987) shows how processes of Darwinian neural competition shape emerging cortical areas during embryogenesis and early infancy. Carrying this further into infancy, Johnson (2011) shows how the formation of visual areas in precocial birds involves an interaction between the genetic guidance of vision through CONSPEC and its shaping by the process of CONLEARN. This same process operates in human infants. Johnson shows how the ability of infants to focus on their mother's eyes sets the stage for further development of an interactional and communication bond with the mother which then leads to specialization of visual areas for face perception.

Language disorders

Reacting against his failure to locate the engrams of memory, Lashley (1951) proposed that all cognitive functioning is global. However, given what we now know about the details of neural connectivity (Schmahmann et al., 2007; Van Essen, Felleman, DeYoe, Olavarria, & Knierim, 1990), it is difficult to deny that different neuronal areas have different functions. However, functional differentiation does not fully invalidate Lashley's insight. To understand how specific impairments can lead to general disorders, we can think of language processing as an acrobat who is simultaneously juggling across seven separate dimensions. At any given moment, there is a contribution from attentional areas, lexical processing, links from lexicon to syntax, and often elaboration of a mental model. If processing in any one of these coordinated areas suddenly "crashes" or breaks down, then the larger process is disrupted. In the case of normal speakers, the juggler is so skillful that this seldom happens, and when it does, there is a quick recovery. In a speaker with impairments, problems in any area can impact the whole system. Because of this, the Unified Competition Model (MacWhinney, 2021) places an emphasis on overall patterns of cognitive *cost* or cognitive *load*. If stress to the system causes failure primarily in a highly "vulnerable" or costly area of language, then within a language, there should be a common tendency across disorders for similar structures and processes to be harmed. That is, aphasics and SLI patients may display similar deficits in terms of which elements of language are impaired, either in comprehension or production.

Evidence for the systemic properties of language disorders comes from non-disordered individuals under cognitive load. First, we know that marked increases in cognitive load can impair normal comprehension (Just & Carpenter, 1992). Moreover, varying the type and quality of cognitive load creates a performance profile in normal college students that closely resembles the one found in aphasics (Dick et al., 2001). Because we know there is no systematic physiological or genetic damage to the language system in these control participants, results like these support a model of language as a broad, complex, resource-intensive system that depends on smooth coordination between diverse local resources.

Aphasics and children with SLI have similar deficits in terms of the elements of language that are impaired, both in comprehension and production. This similarity shifts the emphasis in language disorders from specific competency deficits (e.g., inflectional morphology in Broca's aphasics) and moves it to consideration of the relative vulnerability of a linguistic form or process to damage. The resemblance between the areas of language affected under cognitive load in normal speakers and those affected by SLI is a good example of this vulnerability effect.

The Competition Model does not suggest there are no differences among different disorders. Differences and dissociations are very informative in understanding neural specialization and other properties of language. However, the deciding role of the weakest link in a chain leads us to expect many commonalities across disorders. To illustrate this, let us consider two major disorder groups in further detail: SLI (Specific Language Impairment) and aphasia.

Specific Language Impairment

SLI is characterized by normal cognitive function combined with poor performance on language tasks. As such, the disorder is a logical testing ground for hypotheses about the domain-generality of language as well as genetic vs. learning bases of grammatical abilities.

Some researchers have argued that SLI is a genetic disorder resulting in a phenotypically unified competence deficit. For example, the Extended Optional Infinitive Hypothesis (Rice & Wexler, 1996) proposes that SLI involves a failure to develop tense and agreement marking, thereby delaying grammatically correct production. Similarly, the G-SLI model (van der Lely, 2005) proposes that there is a subgroup of SLI patients whose essential deficit involves grammatical processing of canonical linking chains with no problems in word learning, phonology, or working memory.

These analyses advance three main claims: (1) the cause of SLI is genetic in origin, (2) the deficits seen in SLI are fundamentally domain-specific, and (3) there are diagnostic characteristics that mark the fundamental difference between SLI and normally developing individuals. Let us examine each of these claims.

The cause of SLI is genetic in origin

To characterize SLI as a disorder with a genetic cause, several pieces of evidence are needed. First, the argument requires an identifiable genetic source of the disorder. For example, language disorder in the KE family is associated with a mutation in FOXP2 which determines dominant inheritance (Pinker, 1994).

Although we can relate language deficits in this family to a mutation in FOXP2, this does not provide evidence for a general role of FOXP2 in SLI. A large-scale study of 270 four-year-old language-impaired children from a general population sample of 18,000 children (Meaburn, 2002) did not find a FOXP2 mutation in any participants. Therefore, there must be some alternative account for SLI in general. Moreover, mutations of FOXP2 in the KE family are also associated with small-scale orofacial motor control. Thus, behavioral deficits in these individuals extend beyond functional language processing to motor control (including motor control that is necessary for speech). Vargha-Khadem and colleagues (1995) note that the disorder in affected members "indicates that the inherited disorder does not affect morphosyntax exclusively, or even primarily; rather, it affects intellectual, linguistic, and orofacial praxic functions generally" (p. 930). Given the complex range of deficits, it is unclear how a mutation in this area could yield a phenotypically unified disorder such as that proposed by van der Lely (2005).

There are cases in which a disorder can be closely linked to a specific genetic pattern. In the cases of sickle cell anemia or phenylketonuria we know the exact pathways of gene expression that lead to the disorders. No such simple relation has yet been found for any language disorder. We understand the complex genetic determination of chromosomal abnormalities in Downs Syndrome and

Williams Syndrome. But we do not know how the expression of these genetic factors impacts language. For these and other disorders, a complex model, involving interactions between multiple genetic factors with possible epigenetic expression, seems most probable. Recently, Vernes et al. (2008) traced the down-regulation of FOXP2 on CNTNAP2, a gene that encodes a neurexin that influences cortical development. Looking at a British database of 847 individuals from families with at least one child with SLI, this group then focused on nine CNTNAP2 polymorphisms. Each of these had a significant association with non-word repetition scores. The most powerful association was for a haplotype labeled *ht1* linked to a lowering of non-word repetition scores by half a standard deviation. However, this same pattern is also heavily associated with autism. Interactions of this type argue for the emergentist view of language processing as an integrated system with points of failure that are revealed differentially across syndromes and comorbidities.

Van der Lely sought to identify a highly specified subgroup of SLI language users. However, attempts to replicate this selection specificity (Bishop, Bright, James, Bishop, & van der Lely, 2000) have not succeeded. Moreover, even if such a distinct subtype were identified, and if there were an association between that disorder and some genetic mutation or set of mutations, we would still need to construct a cognitive or neural model by which the mutations could be linked mechanistically to the disorder in question.

SLI deficits are domain-specific

Claims of specific competence deficits in children with SLI have been used to support nativist views regarding the "faculty of language" (Hauser et al., 2002). The idea is that the specificity of this disorder implies that language learning and processing depend on a separate linguistic module, rather than on domain-general processes, and that damage to the module causes highly specified symptoms as hypothesized in SLI. However, the comorbidity of non-linguistic task difficulties for children with SLI (Barry, Yasin, & Bishop, 2006) calls this interpretation into question.

Studies have found various deficits in non-linguistic tasks in SLI patients, seemingly disputing the definition of SLI as an exclusively linguistic (or exclusively grammatical) disorder. SLI patients have impaired phonological short-term memory (Evans & MacWhinney, 1999); the KE family and others have comorbid motor problems (Vargha-Khadem et al., 1995); and SLI children take longer to respond in a word gating task (Mainela-Arnold, 2008).

SLI is a deficit in linguistic competence

The strongest form of nativist analysis views SLI as a deficit in linguistic competence. Specific hypothesized failures include non-termination of the Optional Infinitive stage (Rice & Wexler, 1996) or misapplication of canonical linking rules (van der Lely, 1994). Van der Lely and Christian (2000) describe the choice between processing models and competence deficit models as hinging on whether or not "impaired input processes and processing capacity cause SLI" (p. 35). However, for each of the putative competence deficits, there exist plausible processing deficit accounts. For example, crosslinguistic patterns that have been used to support the Optional Infinitive Hypothesis can also be explained through learning models such as MOSAIC (Freudenthal, Pine, Aguado-Orea, & Gobet, 2007). Van der Lely was able to pick out a group to match the criterion that focused on grammatical problems, but this process of careful selective exclusion then leaves us with no explanation for all the remaining SLI sub-types. Nor does it help us understand the status of children that show grammatical deficits along with additional linguistic, cognitive, and motoric impairments.

There is substantial evidence that the SLI diagnosis can be further sub-divided based on whether the impairment in language competence extends to receptive as well as expressive language use (Evans & MacWhinney, 1999). It is difficult to see how a competence account alone can explain this further dissociation. The Competition Model can account for this asymmetry in terms of

differences in processing. Expressive SLI functions much like Broca's aphasia. In typical speakers, Broca's area serves to gate the firing of lexical items during production. In expressive SLI, as in Broca's aphasia, disruption in the connectivity between Broca's and Wernicke's areas interrupts the smooth gating of lexical items for production. This gating is only important during production and is much less involved in comprehension. In the case of receptive-expressive SLI, then, we would expect to see a different, more general problem of information exchange between brain areas, affecting connections between IFG, DLPFC, MTG, and attentional areas generally.

Aphasia

Neuroemergentism can also help us understand varying patterns in aphasia. The cause of aphasia is well understood, because it arises when a brain lesion from trauma or stroke produces a linguistic impairment. Traditionally, aphasia has been divided into three main categories: Broca's or nonfluent aphasia; Wernicke's or fluent aphasia; and anomia for problems with word finding. Additional types include global, conduction, transcortical sensory, and transcortical motor. Because the etiology of aphasia is much clearer than that of SLI, and because the injuries are easier to map, aphasia provides a useful counterpoint to SLI. In SLI, the functional deficits are well defined, but etiology remains unclear. In aphasia, the opposite is true.

Although aphasia has a clear etiology, lesion site is not a strong predictor of symptom pattern. Two patients with lesions in very different areas will often have similar linguistic profiles. Similarly, patients with lesions in the same area often end up with very different profiles in language performance (Dronkers, Wilkins, Van Valin, Redfern, & Jaeger, 2004). Moreover, if a person with Wernicke's aphasia is impaired in grammaticality judgment in a way that resembles a person with Broca's aphasia, this does not necessarily mean that Broca's and Wernicke's areas perform the same processing tasks, or that they are neuronally identical. Rather, it means that grammar is a complex computational task with vulnerable components that can be impaired in similar ways through damage to various parts of the language network. In this way, aphasia sometimes teaches us more about language than about the brain (McDonald & MacWhinney, 1989).

Crosslinguistic studies of aphasia (Bates, Wulfeck, & MacWhinney, 1991) have illustrated and validated this approach. There is a rich literature demonstrating differences between Broca's aphasics who are native speakers of different languages. For example, the use of agreement in aphasic patients whose native language is Italian is relatively less impaired than in comparison patients whose native language is English. This result is predictable in a Unified Competition Model framework, given the strength of agreement cues in Italian compared to English. In both fluent and non-fluent aphasics, obligatory structures such as SVO word order in German and Italian patients are preserved (Bates et al., 1988). These structures are also the most valid, least costly (as defaults in the language), and most highly frequent. Similarly, when Turkish speakers become aphasic, they still maintain the use of SOV word order, which is the standard in Turkish. As Bates has said, "You can take the Turks out of Turkey, but you can't take the Turkish out of the Turks." In other words, the major determinant of cue survival in aphasia is the relative strength of the cue in the person's language.

The status of competence accounts in aphasia parallels their status in SLI. In SLI, competence accounts look for a simple causal association between a damaged component (such as a specific mutation) and a language deficit. In aphasia, competence accounts also require that a specific lesioned local area or module be the root cause of the aphasic disability. In both cases, the competence approach fails to consider the broader context of the language system, wherein levels of processing (semantics, syntax, lexicon, audition, comprehension) interact within a distributed functional neural network of brain areas (Dronkers, 2011; Kemmerer, 2015).

In the neuroemergentist analysis, the effects of lesions are understood in terms of the damage inflicted on both grey matter and white matter. Damages to grey matter impact the content of the

representational maps that organize structural levels. Damages to white matter tracts impact coordination and gating between areas. Thus, observed patterns of aphasia relate not just to the processing in local maps, but also to disorders in connectivity and processing that occur as two or more maps attempt to work in synchrony.

Gupta et al. (2003) showed that, in children who had had early focal lesions, learning was quantitatively delayed in word learning, non-word repetition, and serial recall tasks. Although the level of performance was impaired overall, the relation between measures of verbal working memory and word learning was maintained, and those relations were like the control group. These data are consistent with the finding that children with focal lesions are able to achieve functional language use, although their overall reaction times are often slower than those of controls (MacWhinney, Feldman, Sacco, & Valdes-Perez, 2000).

A similar, and perhaps even more striking, finding comes from Wilson and Saygun (2004). They report evidence in direct contradiction to models that hypothesize that Broca's area is the unique site for comprehension of maximal trace projections (Grodzinsky, 2000). In Wilson and Saygun's study, all patient groups, including anomics, shared a general impairment pattern, although the quantitative performance of the patients varied, as expected. These results show that different injuries to the language network can create similar performance profiles. These data fit well with the analysis of the emergentist Competition Model.

Summary and conclusion

This chapter has examined ways in which neuroemergentism as expressed in the Unified Competition Model can be used to understand language disorders. Apart from providing a comprehensive theoretical approach, emergentism provides clear methodological guidance through its emphasis on component models. The complexities of language, structural emergence, mechanisms, and the brain make it imperative to rely on multiple component models to understand language disorders. Earlier, we considered the disorder of stuttering as an example. For this, we need to model the time course of language production as it moves through an interconnected functional neural circuit from formulation to articulation. We need models of neural activation and competition to consider whether incomplete gating signals are being transmitted between areas. We need to elaborate models of basal ganglia control of fluency or proceduralization to evaluate the contributions of the cortico-BG loop (Chang & Guenther, 2020) and within that model the relative contributions of the loop to learning vs. processing. We also need to understand the role of dopamine (Civier et al., 2013) in control of the loop. We need to consider neuroemergentist accounts of the ways in which stutterers develop compensatory strategies (Bloodstein & Bernstein Ratner, 2008) across the lifespan, and the extent to which specific linguistic structures can trigger stuttering. By linking component models in this way, we can derive at a fuller understanding of stuttering and other language disorders.

For each language disorder, we need to consider the involvement of mechanisms such as the 16 listed earlier, as well as specific genetic variations that impact these mechanisms as they unfold either in embryogenesis or through epigenesis and interactive specialization. During these explorations, we may discover genetic or processing mechanisms that play a pivotal role in shaping the disorder. However, we can be sure that the effects of these pivotal mechanisms will be further modified by interactions with other constraints and processes across diverse timescales.

Further reading

Bishop, D. (2013). Developmental cognitive genetics: How psychology can inform genetics and vice versa. *Quarterly Journal of Experimental Psychology*, *59*(7), 1153–1168.

MacWhinney, B. (2021). The competition model: Past and future. In J. Gervain (Ed.), *A life in cognition* (pp. 3–16). New York: Springer Nature.

References

Alexander, G. E., DeLong, M. R., & Strick, P. L. (1986). Parallel organization of functionally segregated circuits linking basal ganglia and cortex. *Annual Review of Neuroscience*, *9*(1), 357–381.

Anderson, M. L. (2010). Neural reuse: A fundamental organizational principle of the brain. *Behavioral and Brain Sciences*, *33*(4), 245–266.

Barry, J., Yasin, I., & Bishop, D. (2006). Heritable risk factors associated with language impairments. *Genes, Brain and Behavior*, *6*(1), 66–76.

Bates, E. (1999). Plasticity, localization, and language development. In S. Broman & J. M. Fletcher (Eds.), *The changing nervous system: Neurobehavioral consequences of eary brain disorders* (pp. 213–253). New York: Oxford University Press.

Bates, E., & MacWhinney, B. (1981). Second language acquisition from a functionalist perspective: Pragmatic, semantic and perceptual strategies. In H. Winitz (Ed.), *Annals of the New York academy of sciences conference on native and foreign language acquisition* (pp. 190–214). New York: New York Academy of Sciences.

Bates, E., Wulfeck, B., & MacWhinney, B. (1991). Crosslinguistic research in aphasia: An overview. *Brain and Language*, *41*(2), 123–148. Retrieved from https://psyling.talkbank.org/years/1991/overview.pdf.

Beckner, C., Blythe, R., Bybee, J., Christiansen, M., Croft, W., Ellis, N., … Schoenemann, T. (2009). Language is a complex adaptive system: Position paper. *Language Learning*, *59*(s1), 1–26.

Berwick, R. (1987). Parsability and learnability. In B. MacWhinney (Ed.), *Mechanisms of language acquisition*. Hillsdale, NJ: Lawrence Erlbaum Associates.

Bishop, D., Bright, P., James, C., Bishop, S. J., & van der Lely, H. (2000). Grammatical SLI: A distinct subtype of developmental language impairment. *Applied Psycholinguistics*, *21*(2), 159–181.

Bloodstein, O., & Bernstein Ratner, N. (2008). *A handbook on stuttering* (6th ed.). Delmar, NY: Cengage.

Browman, C. P., & Goldstein, L. (1992). Articulatory phonology: An overview. *Phonetica*, *49*(3–4), 155–180.

Bybee, J. (2010). *Language, usage, and cognition*. New York: Cambridge University Press.

Chang, S.-E., & Guenther, F. (2020). Involvement of the cortico-basal ganglia-thalamocortical loop in developmental stuttering. *Frontiers in Psychology*, *10*, 3088.

Chomsky, N. (2007). Approaching UG from below. In U. Sauerland & M. Gaertner (Eds.), *Interfaces + recursion = language?* (pp. 1–30). New York: Mouton de Gruyter.

Christiansen, M., & Chater, N. (2008). Language as shaped by the brain. *Behavioral and Brain Sciences*, *31*(5), 489–558.

Christiansen, M., & Chater, N. (2016). The now-or-never bottleneck: A fundamental constraint on language. *Behavioral and Brain Sciences*, *39*.

Civier, O., Bullock, D., Max, L., & Guenther, F. H. (2013). Computational modeling of stuttering caused by impairments in a basal ganglia thalamo-cortical circuit involved in syllable selection and initiation. *Brain and Language*, *126*(3), 263–278.

Conway, C. M. (2020). How does the brain learn environmental structure? Ten core principles for understanding the neurocognitive mechanisms of statistical learning. *Neuroscience and Biobehavioral Reviews*, *112*, 279–299.

Culicover, P. (2013). *Explaining syntax: Representations, structures, and computation*. New York: Oxford University Press.

Darwin, C. (1859). *On the origin of species*. London: John Murray.

Darwin, C. (1862). *On the various contrivances by which orchids are fertilized by insects (1862)*. Chicago, IL: University of Chicago Press.

Dehaene, S., & Cohen, L. (2007). Cultural recycling of cortical maps. *Neuron*, *56*(2), 384–398.

Dick, F., Bates, E., Wulfeck, B., Utman, J., Dronkers, N., & Gernsbacher, M. A. (2001). Language deficits, localization and grammar: Evidence for a distributive model of language breakdown in aphasics and normals. *Psychological Review*, *108*(4), 759–788.

Diessel, H. (2017). Usage-based linguistics. In *Oxford research encyclopedia of linguistics*.

Dominey, P. F., & Boussaoud, D. (1997). Encoding behavioral context in recurrent networks of the fronto-striatal system: A simulation study. *Cognitive Brain Research*, *6*(1), 53–65.

Dronkers, N. (2011). The neural architecture of the language comprehension network: Converging evidence from lesion and connectivity analyses. *Frontiers in Systems Neuroscience*, *5*, 1.

Dronkers, N., Wilkins, D., Van Valin, R., Redfern, B., & Jaeger, J. (2004). Lesion analysis of the brain areas involved in language comprehension. *Cognition*, *92*(1–2), 145–177.

Edelman, G. (1987). *Neural Darwinism: The theory of neuronal group selection*. New York: Basic Books.

Elman, J. L., Bates, E., Johnson, M., Karmiloff-Smith, A., Parisi, D., & Plunkett, K. (1996). *Rethinking innateness*. Cambridge, MA: MIT Press.

Evans, J. L., & MacWhinney, B. (1999). Sentence processing strategies in children with expressive and expressive-receptive specific language impairments. *International Journal of Language and Communication Disorders*, *34*(2), 117–134. https://doi.org/10.1080/136828299247469.

AU: Please provide editor names, publisher city name, publisher name and page range for reference Diessel, 2017.

Fisher, S. E., & Scharff, C. (2009). FOXP2 as a molecular window into speech and language. *Trends in Genetics*, *25*(4), 166–177.

Fodor, J. (1983). *The modularity of mind: An essay on faculty psychology*. Cambridge, MA: MIT Press.

Freudenthal, D., Pine, J. M., Aguado-Orea, J., & Gobet, F. (2007). Modeling the developmental patterning of finiteness marking in English, Dutch, German, and Spanish using MOSAIC. *Cognitive Science*, *31*(2), 311–341.

Frigerio-Domingues, C., & Drayna, D. (2017). Genetic contributions to stuttering: The current evidence. *Molecular Genetics and Genomic Medicine*, *5*(2), 95–102.

Galton, F. (1883). *Inquiries into human faculty and its development*. London: Dent.

Goldberg, A. (2006). *Constructions at work: The nature of generalization in language*. Oxford: Oxford University Press.

Goldstone, R., & Rogosky, B. J. (2002). Using relations within conceptual systems to translate across conceptual systems. *Cognition*, *84*(3), 295–320.

Goodwin, C. (2013). The co-operative, transformative organization of human action and knowledge. *Journal of Pragmatics*, *46*(1), 8–23.

Gow, D. W. (2012). The cortical organization of lexical knowledge: A dual lexicon model of spoken language processing. *Brain and Language*, *121*(3), 273–288.

Grodzinsky, Y. (2000). The neurology of syntax: Language use without Broca's area. *Behavioral and Brain Sciences*, *23*(1), 1–21.

Guenther, F., & Vladusich, T. (2012). A neural theory of speech acquisition and production. *Journal of Neurolinguistics*, *25*(5), 408–422.

Gupta, P., MacWhinney, B., Feldman, H., & Sacco, K. (2003). Phonological memory and vocabulary learning in children with focal lesions. *Brain and Language*, *87*(2), 241–252. https://doi.org/10.1016/s0093-934x(03)00094-4.

Hagoort, P. (2013). MUC (memory, unification, control) and beyond. *Frontiers in Psychology*, *4*, 1–13.

Hart, B., & Risley, T. R. (1995). *Meaningful differences in the everyday experience of young American children*. Baltimore, MD: Paul H. Brookes.

Hauser, M., Chomsky, N., & Fitch, T. (2002). The faculty of language: What is it, who has it, and how did it evolve? *Science*, *298*(5598), 1569–1579.

Hebb, D. (1949). *The organization of behavior*. New York: Wiley.

Hernandez, A. E., Claussenius-Kalman, H. L., Ronderos, J., Castilla-Earls, A. P., Sun, L., Weiss, S. D., & Young, D. R. (2019). Neuroemergentism: A framework for studying cognition and the brain. *Journal of Neurolinguistics*, *49*, 214–223.

Hickok, G. (2009). The functional neuroanatomy of language. *Physics of Life Reviews*, *6*(3), 121–143.

Hickok, G., Bellugi, U., & Klima, E. S. (2001). Sign language in the brain. *Scientific American*, *284*(6), 58–65.

Hickok, G., & Poeppel, D. (2004). Dorsal and ventral streams: A framework for understanding aspects of the functional anatomy of language. *Cognition*, *92*(1–2), 67–99.

Hymes, D. (1962). The ethnography of speaking. *Anthropology and Human Behavior*, *13*(53), 11–74.

Johnson, M. (2011). Interactive specialization: A domain-general framework for human functional brain development? *Developmental Cognitive Neuroscience*, *1*(1), 7–21.

Just, M., & Carpenter, P. (1992). A capacity theory of comprehension: Individual differences in working memory. *Psychological Review*, *99*(1), 122–149.

Kemmerer, D. (2015). *The cognitive neuroscience of language*. New York: Psychology Press.

Kohonen, T. (2001). *Self-organizing maps* (3rd ed.). Berlin: Springer.

Korniol, R. (1995). Stuttering, language, and cognition: A review and model of stuttering as suprasemental sentence plan alignment (SPA). *Psychological Review*, *117*, 104–124.

AU: Please provide page range for reference Lashley, 1951.

Lashley, K. (1951). The problem of serial order in behavior. In L. A. Jeffress (Ed.), *Cerebral mechanisms in behavior*. New York: Wiley.

Li, P., Legault, J., & Litcofsky, K. A. (2014). Neuroplasticity as a function of second language learning: Anatomical changes in the human brain. *Cortex*, *58*, 301–324.

Maclay, H., & Osgood, C. E. (1959). Hesitation phenomena in spontaneous English speech. *Word*, *25*(1), 19–44. Retrieved from http://search.ebscohost.com/login.aspx?direct=true&db=mzh&AN=1959001726&site=ehost-live.

MacWhinney, B. (2004). A multiple process solution to the logical problem of language acquisition. *Journal of Child Language*, *31*(4), 883–914. https://doi.org/10.1017/s0305000904006336.

MacWhinney, B. (2005a). The emergence of grammar from perspective. In D. Pecher & R. A. Zwaan (Eds.), *The grounding of cognition: The role of perception and action in memory, language, and thinking* (pp. 198–223). Mahwah, NJ: Lawrence Erlbaum Associates.

MacWhinney, B. (2005b). Language evolution and human development. In B. Ellis & D. Bjorklund (Eds.), *Origins of the social mind* (pp. 383–410). New York: Guilford.

MacWhinney, B. (2014). Item-based patterns in early syntactic development. In T. Herbst, H.-J. Schmid, & S. Faulhaber (Eds.), *Constructions collocations patterns* (pp. 33–70). Berlin: de Gruyter Mouton.

MacWhinney, B. (2019). Neuroemergentism: Levels and constraints. *Journal of Neurolinguistics*, *49*, 232–234. https://doi.org/10.1016/j.jneuroling.2018.04.002.

MacWhinney, B. (2021). The competition model: Past and future. In J. Gervain (Ed.), *A life in cognition*. New York: Springer Nature.

MacWhinney, B., & Bates, E. (Eds.). (1989). *The crosslinguistic study of sentence processing*. New York: Cambridge University Press.

MacWhinney, B., Feldman, H., Sacco, K., & Valdes-Perez, R. (2000). Online measures of basic language skills in children with early focal brain lesions. *Brain and Language*, *71*(3), 400–431. https://doi.org/10.1006/brln.1999.2273.

MacWhinney, B., Malchukov, A., & Moravcsik, E. (Eds.). (2014). *Competing motivations in grammar and usage*. New York: Oxford University Press.

Mainela-Arnold, E., Evans, J. L., & Coady, J. A. (2008). Lexical representations in children with SLI: Evidence from a frequency-manipulated gating task. *Journal of Speech, Language, and Hearing Research*, *51*(2), 381–393.

McClelland, J. (2015). Capturing gradience, continuous change, and quasi-regularity in sound, word, phrase, and meaning. In B. MacWhinney & W. O'Grady (Eds.), *The handbook of language emergence* (pp. 53–80). New York: Wiley.

McClelland, J., Mirman, D., & Holt, L. (2006). Are there interactive processes in speech perception? *Trends in Cognitive Sciences*, *10*(8), 363–369.

McDonald, J., & MacWhinney, B. (1989). Maximum likelihood models for sentence processing research. In B. MacWhinney & E. Bates (Eds.), *The crosslinguistic study of sentence processing* (pp. 397–421). New York: Cambridge University Press.

Meaburn, E., Dale, P. S., Craig, I., & Plomin, R. (2002). Language-impaired children: No sign of the FOXP2 mutation. *NeuroReport*, *13*(8), 1075–1077.

Mill, J. S. (1859). *System of logic, rationative and inductive; being a connected view of the principles of evidence and the methods of scientific investigation. Chapter VI: Of the composition of causes*. New York: Harper and Brothers.

Naganathan, A. N., & Muñoz, V. (2005). Scaling of folding times with protein size. *Journal of the American Chemical Society*, *127*(2), 480–481.

Newell, A. (1990). *A unified theory of cognition*. Cambridge, MA: Harvard University Press.

Nozari, N., & Hepner, C. R. (2019). To select or to wait? The importance of criterion setting in debates of competitive lexical selection. *Cognitive Neuropsychology*, *36*(5–6), 193–207.

Nozari, N., & Novick, J. (2017). Monitoring and control in language production. *Current Directions in Psychological Science*, *26*(5), 403–410.

Ochs, E. A., Schegloff, M., & Thompson, S. A. (1996). *Interaction and grammar*. Cambridge: Cambridge University Press.

O'Grady, W. (2005). *Syntactic carpentry*. Mahwah, NJ: Lawrence Erlbaum Associates.

Olszewska, A. M., Gaca, M., Herman, A. M., Jednoróg, K., & Marchewka, A. (2021). How musical training shapes the adult brain: Predispositions and neuroplasticity. *Frontiers in Neuroscience*, *15*, 204.

Pavlik, P., & Anderson, J. (2008). Using a model to compute the optimal schedule of practice. *Journal of Experimental Psychology: Applied*, *14*(2), 101–117.

Pinker, S. (1994). *The language instinct*. New York: William Morrow.

Pulvermüller, F. (2003). *The neuroscience of language*. Cambridge: Cambridge University Press.

Ratcliff, R., & McKoon, G. (2008). The diffusion decision model: Theory and data for two-choice decision tasks. *Neural Computation*, *20*(4), 873–922.

Recanzone, G., & Merzenich, M. (1993). Functional plasticity in the cerebral cortex: Mechanisms of improved perceptual abilities and skill acquisition. *Concepts in Neuroscience*, *4*, 1–23.

Rice, M., & Wexler, K. (1996). Toward tense as a clinical marker of specific language impairment in English-speaking children. *Journal of Speech and Hearing Research*, *39*(6), 1239–1257.

Roberts, J. E., Rosenfeld, R. M., & Zeisel, S. A. (2004). Otitis media and speech and language: A meta-analysis of prospective studies. *Pediatrics*, *113*(3), e238–e248.

Roelofs, A. (2011). Modeling the attentional control of vocal utterances: From Wernicke to WEAVER+. In J. Guendozi, F. Loncke, & M. Williams (Eds.), *The handbook of psycholinguistic and cognitive processes: Perspectives in communication disorders* (pp. 189–208). New York: Psychology Press.

Rosenbaum, D. (2015). *It's a jungle in there: How competition and cooperation in the mind shape the brain*. New York: Oxford University Press.

Schlichting, M. L., & Preston, A. R. (2015). Memory integration: Neural mechanisms and implications for behavior. *Current Opinion in Behavioral Sciences*, *1*, 1–8.

AU: Please provide page range for reference MacWhinney, 2021.

Schmahmann, J., Pandya, D., Wang, R., Dai, G., D'Arceuil, H., de Crespigny, A., & Wedeen, V. (2007). Association fibre pathways of the brain: Parallel observations from diffusion spectrum imaging and autoradiography. *Brain*, *130*(3), 630–653.

Shiell, M. M., Champoux, F., & Zatorre, R. J. (2015). Reorganization of auditory cortex in early-deaf people: Functional connectivity and relationship to hearing aid use. *Journal of Cognitive Neuroscience*, *27*(1), 150–163.

Thompson, C. K., Meltzer-Asscher, A., Cho, S., Lee, J., Wieneke, C., Weintraub, S., & Mesulam, M. (2013). Syntactic and morphosyntactic processing in stroke-induced and primary progressive aphasia. *Behavioural Neurology*, *26*(1–2), 35–54.

Valiant, L. G. (2014). What must a global theory of cortex explain? *Current Opinion in Neurobiology*, *25*, 15–19.

van der Lely, H. (1994). Canonical linking rules: Forward vs. reverse linking in normally developing and Specifically Language Impaired children. *Cognition*, *51*(1), 29–72.

van der Lely, H. (2005). Domain-specific cognitive systems: Insight from Grammatical-SLI. *Trends in Cognitive Sciences*, *9*(2), 53–59.

van der Lely, H., & Christian, V. (2000). Lexical word formation in children with grammatical SLI: A grammar-specific versus an input-processing deficit? *Cognition*, *75*(1), 33–63.

Van Essen, D. C., Felleman, D. F., DeYoe, E. A., Olavarria, J. F., & Knierim, J. J. (1990). Modular and hierarchical organization of extrastriate visual cortex in the macaque monkey. *Cold Spring Harbor Symposium on Quantitative Biology*, *55*, 679–696.

Vargha-Khadem, F., Gadian, D. G., Copp, A., & Mishkin, M. (2005). FOXP2 and the neuroanatomy of speech and language. *Nature Reviews Neuroscience*, *6*(2), 131–138.

Vargha-Khadem, F., Watkins, K., Alcock, K., Fletcher, P., & Passingham, R. (1995). Praxic and nonverbal cognitive deficits in a large family with a genetically transmitted speech and language disorder. *Proceedings of the National Academy of Sciences of the United States of America*, *92*(3), 930–933.

Vernes, S., Newbury, D., Abrahams, B., Winchester, L., Nicod, J., Groszer, M., … Fisher, S. (2008). A functional genetic link between distinct developmental language disorders. *New English Journal of Medicine*, *359*, 1–9.

Webb, A. R., Heller, H. T., Benson, C. B., & Lahav, A. (2015). Mother's voice and heartbeat sounds elicit auditory plasticity in the human brain before full gestation. *Proceedings of the National Academy of Sciences*, *112*(10), 3152–3157.

West-Eberhard, M. J. (2003). *Developmental plasticity and evolution*. Oxford: Oxford University Press.

Whitehurst, G., & Vasta, R. (1975). Is language acquired through imitation? *Journal of Psycholinguistic Research*, *4*(1), 37–59.

Wilson, S. M., & Saygin, A. P. (2004). Grammaticality judgments in aphasia: Deficits are not specific to syntactic structures, aphasic syndromes, or lesion sites. *Journal of Cognitive Neuroscience*, *16*(2), 238–252.

Wong, P. C., Morgan-Short, K., Ettlinger, M., & Zheng, J. (2012). Linking neurogenetics and individual differences in language learning: The dopamine hypothesis. *Cortex*, *48*(9), 1091–1102.

Woollett, K., & Maguire, E. A. (2011). Acquiring "the knowledge" of London's layout drives structural brain changes. *Current Biology*, *21*(24), 2109–2114.

Zerilli, J. (2022). *The adaptablel mind: What neuroplasticity and neural reuse tell us about language and cognition*. New York: Oxford University Press.