



Reinforcement learning in young adults with developmental language impairment

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ABSTRACT

The aim of the study was to examine reinforcement learning (RL) in young adults with developmental language impairment (DLI) within the context of a neurocomputational model of the basal ganglia-dopamine system (Frank, Seeberger, & O'Reilly, 2004). Two groups of young adults, one with DLI and the other without, were recruited. A probabilistic selection task was used to assess how participants implicitly extracted reinforcement history from the environment based on probabilistic positive/negative feedback. The findings showed impaired RL in individuals with DLI, indicating an altered gating function of the striatum in testing. However, they exploited similar learning strategies as comparison participants at the beginning of training, reflecting relatively intact functions of the prefrontal cortex to rapidly update reinforcement information. Within the context of Frank's model, these results can be interpreted as evidence for alterations in the basal ganglia of individuals with DLI.

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1. Introduction

1.1. Linguistic and non-linguistic deficits in developmental language impairment (DLI)

Individuals with DLI represent a heterogeneous group of people who have substantial difficulty acquiring language despite normal hearing, normal nonverbal intelligence, appropriate social functioning, and no obvious signs of brain injury (Bishop, 1997). One of the defining characteristics of DLI¹ is the impaired acquisition of rule- or pattern-based components in language, such as morphology, syntax, and some aspects of phonology (Leonard, 1997). This commonly used definition leads to early hypotheses regarding the etiology of DLI that an impaired language-specific learning mechanism underlies language development and disorders (Clahsen, 1989; Rice, Wexler, & Cleave, 1995; van der Lely, 2005).

However, recent studies have shown that deficits in individuals with DLI are not limited to language but include general cognitive functioning, such as phonological working memory (Archibald & Gathercole, 2006), long-term memory (Lum, Conti-Ramsden, Page, & Ullman, in press; Tomblin, Mainela-Arnold, & Zhang, 2007), speed of processing (Miller, Kail, Leonard, & Tomblin, 2001), music processing (Jentschke, Koelsch, Sallat, & Friederici, 2008), and sta-

tistical learning (Evans, Saffran, & Robe-Torres, 2009). These findings indicate that poor language learning in general and morphosyntax in particular shown in individuals with DLI may be a manifestation of impaired domain-general cognitive mechanisms that go beyond the language system. As noted above, some of the studies have pointed to a domain-general impairment in statistical learning (e.g., Evans et al., 2009). In this research tradition, neither the nature of the learning system nor the underlying neural mechanisms have been clearly elucidated. The current study expands the line of inquiry concerning statistical learning deficits in DLI to consider reinforcement learning (RL) as a potential learning mechanism supporting language development, due to its reliance on the corticostriatal loops, the basal ganglia in particular.

1.2. A neurocognitive approach to the understanding of DLI

Procedural learning has been suggested to be important for language learning (Gupta & Cohen, 2002; Gupta & Dell, 1999; Nicolson & Fawcett, 2007; Ullman, 2001, 2004). More recently, Ullman and Pierpont (2005) proposed the Procedural Deficit Hypothesis to explain a wide array of behavioral and neurophysiological findings for individuals with DLI. According to this hypothesis, a fundamental, but not exclusive, cause of DLI, particularly the grammatical deficits, can be attributed to impaired or less efficient procedural memory. Procedural memory is mediated, at least in part, by the corticostriatal loops connecting the basal ganglia with the cerebral cortex (Alexander & Crutcher, 1990; Alexander, DeLong, & Strick, 1986; Eichenbaum & Cohen, 2001; Gabrieli, 1998; Seger, 2006). The basal ganglia play a particularly important role in this system for two reasons. First, anatomically, the basal ganglia connect to almost all regions of the cortex, and therefore are in an ideal

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¹ In this paper, we will use the term *developmental language impairment* (DLI) rather than *specific language impairment* (SLI) to avoid the suggestion that individual differences in our research participants are only present in language behavior.

position to influence a wide range of behaviors mediated by the corticostriatal loops. Second, it is becoming well understood that the basal ganglia, especially the striatum, not only play an important role in motor function, but they also support a broad array of incremental and implicit cognitive learning, most notably procedural learning (Poldrack et al., 2005; Seger, 2006, 2009; Shohamy, Myers, Grossman, Sage, & Gluck, 2005; Shohamy, Myers, Kalanithi, & Gluck, 2007) and RL (Doya, 1999; Frank, Seeberger, & O'Reilly, 2004; Niv, 2009; O'Doherty, Dayan, Friston, Critchley, & Dolan, 2003).

In the literature, several studies have shown poor procedural learning in individuals with DLI (Hedenius et al., *in press*; Kemeny & Lukacs, 2009; Lee & Tomblin, *submitted*; Lum, Gelgec, & Conti-Ramsden, 2010; Lum et al., *in press*; Tomblin et al., 2007; but see Gabriel, Maillart, Guillaume, Stefaniak, & Meulemans, 2011). These findings provide empirical support for the Procedural Deficit Hypothesis. Because RL also shares the same basal ganglia system as procedural learning, we might predict that performance on RL would also be relatively poorer in individuals with DLI than in comparison participants; however, no studies have examined RL in DLI. Therefore, in the current study, we used an RL paradigm developed by Frank, Seeberger, and O'Reilly (2004) to further test the Procedural Deficit Hypothesis by examining how individuals with DLI implicitly extract reinforcement history from the environment by trial and error. We believe that the findings can shed light on the neurocognitive underpinnings of DLI, and by extension the role of RL and the basal ganglia in language development in general.

1.3. RL in a nutshell: the past and the present

The beginning of RL research originated from the area of artificial intelligence and machine learning that deals with how an agent (e.g., a robot) learns to make decisions through an incremental, trial-and-error process (Sutton & Barto, 1998). The ultimate goal of RL is to maximize the likelihood of rewards while minimizing the occurrence of punishments without explicit instructions in the learning process (c.f., supervised learning). In the early 1960s when the contemporary study of language development began, RL was rejected as a plausible mechanism for language learning (Chomsky, 1959). This state of affairs has remained so, despite considerable reconsideration of Chomsky's original argument.

During the last 30 years, the reinforcement theory has been substantially revised. One of the key features of this advancement has been the discovery that the striatal dopaminergic systems play a role in calculating probability of future reward and punishment that come from an agent's current actions in an environment (e.g., Dayan & Niv, 2008; Frank, Moustafa, Haughey, Curran, & Hutchison, 2007; Pizzagalli et al., 2008). Researchers found that phasic changes in dopamine levels are based upon prior experiences of interacting with the environment. On the one hand, phasic dopamine increase (a.k.a. dopamine burst) primarily results from positive reinforcing experiences and occurrence of unanticipated rewards, which strengthens synaptic plasticity in D1 dopamine receptors in the "Go" neural pathway and therefore supports learning of the behavior. On the other hand, phasic dopamine decrease (a.k.a. dopamine dip) is the result of negative reinforcing experiences and omission of an expected or predicted reward, which strengthens synaptic plasticity in D2 dopamine receptors in the "NoGo" neural pathway and therefore leads to avoidance of this behavior in the future. All of the reinforcement-related values converge to the substantia nigra (SN), the dopamine synthesizing region within the basal ganglia. As a result, while the cortical regions are responsible for information processing and representation storage, the basal ganglia select, via thalamic pathways, which of the numerous possible representations in the cortex are appro-

priate to execute under different circumstances (Frank, Loughry, & O'Reilly, 2001; Redgrave, Prescott, & Gurney, 1999).

Recent research in the role of the dopaminergic system in RL invokes interest among cognitive scientists for at least two reasons. First, RL algorithms can be instantiated in biologically plausible mechanisms, and therefore provide a direct means to uncover important insights on human decision-making behaviors (Dayan & Niv, 2008). Second, findings regarding the role of dopamine in RL bring about clinical implications in both degenerative neurological disorders (e.g., Parkinson's disease and Huntington's disease) and developmental disorders (e.g., attention deficit/hyperactivity disorder (ADHD) and autism spectrum disorder (ASD)) (Bradshaw, 2001). In addition, understanding the nature of these disorders helps reconstruct the path from the brain to individual differences in behaviors. The corticostriatal loops, in particular the striatum of the basal ganglia, are one of the primary recipients of dopaminergic projections, and therefore is considered as an obvious candidate neural system for RL (Niv, 2009).

1.4. The probabilistic selection task: an RL paradigm based on a biologically grounded computational model

Recently, Frank and his colleagues developed a neurocomputational model of the basal ganglia-dopamine system, which was built upon a large body of earlier theoretical work on the role of dopamine in RL (see Cohen & Frank, 2009, for a review). According to their model, the basal ganglia are conceptualized as a gating system, which strengthens the pattern of neural firing in the frontal cortex that is related to appropriate actions, while suppressing those that are less appropriate. The dopaminergic system, the D1 and D2 dopamine receptors to be more specific, regulate the gating function of the basal ganglia (Wickens & Arbutnot, 2010). Both types of dopamine receptors are highly concentrated in the caudate nucleus and the putamen, while D1 receptors have much higher density in the prefrontal cortex (PFC) than D2 receptors (Hall et al., 1994; Meador-Woodruff, 1994). This dopaminergic modulation of cortical input to the basal ganglia is conceived to be the primary mechanism of RL: it leads to correct feedback or reward by sending positive learning signals via D1 dopamine receptors, while simultaneously reducing the probability of incorrect or non-rewarding behaviors by sending negative learning signals via D2 dopamine receptors (Frank, 2005, 2011; Frank, Moustafa, et al., 2007; Frank, Santamaria, O'Reilly, & Willcutt, 2007; Frank, Seeberger, & O'Reilly, 2004).

To further test this biologically plausible model, Frank, Seeberger, and O'Reilly (2004) designed an RL paradigm, and made several verifiable predictions with respect to human behavioral responses during RL. The RL paradigm is a probabilistic selection task. During the acquisition phase of the task, participants learn to choose the most frequently reinforced stimulus from each training pair (e.g., choosing A from the AB pair) based on either positive or negative feedback on their decisions. It should be noted that the feedback is probabilistic: the feedback received is not always the same for each choice, and therefore it is impossible to always make the right decision. During the acquisition phase, all participants are expected to learn to choose Stimulus A over B, given that Stimulus A is the most frequently rewarded symbol whereas Stimulus B is the least. However, learning to choose A over B can be achieved by learning that (1) Stimulus A leads to positive feedback, (2) Stimulus B leads to negative feedback, or (3) both. Therefore, to distinguish the choose-A and avoid-B learning strategies, a test phase follows immediately. During the test phase, participants are presented with the original stimuli in novel pairings in order to evaluate whether participants have a bias for choosing more reinforced stimuli and/or for avoiding less reinforced stimuli. This sheds light on individual differences in basal ganglia dopamine

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