



## A verbal strength in children with Tourette syndrome? Evidence from a non-word repetition task



Cristina D. Dye<sup>a,\*</sup>, Matthew Walenski<sup>b</sup>, Stewart H. Mostofsky<sup>c</sup>, Michael T. Ullman<sup>d,\*</sup>

<sup>a</sup> Centre for Research in Linguistics and Language Sciences, Newcastle University, United Kingdom

<sup>b</sup> The Roxelyn and Richard Pepper Department of Communication Sciences and Disorders, Northwestern University, United States

<sup>c</sup> Kennedy Krieger Institute, Johns Hopkins University, United States

<sup>d</sup> Brain and Language Lab, Department of Neuroscience, Georgetown University, United States

### ARTICLE INFO

#### Article history:

Received 8 December 2015

Revised 6 June 2016

Accepted 17 July 2016

### ABSTRACT

Tourette syndrome (TS) is characterized by motor and vocal tics, and frontal/basal-ganglia abnormalities. Whereas cognitive strengths have been found in other neurodevelopmental disorders, less attention has been paid to strengths in TS, or to verbal strengths in any neurodevelopmental disorder. We examined whether the finding of speeded TS production of rule-governed morphological forms (e.g., “slipped”) that involve composition (Walenski, Mostofsky, & Ullman, 2007) might extend to another language domain, phonology. Thirteen children with TS and 14 typically-developing (TD) children performed a non-word repetition task: they repeated legal phonological strings (e.g., “naichovabe”), a task that taps rule-governed (de)composition. Parallel to the morphology findings, the children with TS showed speeded production, while the two groups had similar accuracy. The results were not explained by potentially confounding factors, including IQ. Overall, the findings suggest that rule-governed grammatical composition may be speeded in TS, perhaps due to frontal/basal-ganglia abnormalities.

© 2016 Elsevier Inc. All rights reserved.

### 1. Introduction

Previous research on neurodevelopmental disorders has, not surprisingly, focused on the various impairments and weaknesses found in the disorders, and their underlying causes. However, studies have recently also begun to examine whether individuals with these disorders might have not only weaknesses, but also strengths, relative to typically-developing (TD) individuals. For example, research has revealed possible enhancements in auditory and visual perception or attention in individuals with autism spectrum disorder, as compared to TD controls (Mottron, Dawson, Soulie, & Burack, 2006; Remington, Swettenham, & Lavie, 2012). Similarly, strengths in visuo-spatial processing have been suggested for developmental dyslexia (Diehl et al., 2014; Schneps, Brockmole, Sonnert, & Pomplun, 2012). However, some other neurodevelopmental disorders, including Tourette syndrome (TS), have been less well studied with regard to potential strengths. In addition, there has been relatively little research suggesting

strengths in the verbal domain in any neurodevelopmental disorder (Just, Cherkassky, Keller, & Minshew, 2004; Järvinen-Pasley, Wallace, Ramus, Happé, & Heaton, 2008; Walenski, Mostofsky, Gidley-Larson, & Ullman, 2008; Walenski, Mostofsky, & Ullman, 2014), including in TS (Walenski, Mostofsky, & Ullman, 2007). Here we address these gaps by probing for potential verbal strengths in TS.

TS is a neurodevelopmental disorder characterized by multiple motor tics and at least one vocal tic that are not explained by medications or another medical condition (American Psychiatric Association, 2013). Tics, which can be expressed as simple or complex motor movements or vocalizations, are characteristically fast, abrupt, recurrent, and semi-voluntary (American Psychiatric Association, 2013). The tics are thought to be caused by disturbances of the basal ganglia and closely connected regions of cortex, especially motor and cognitive regions of frontal cortex (Albin & Mink, 2006; Müller-Vahl et al., 2014). Such disturbances are reflected in both structural and functional abnormalities of the basal ganglia and frontal cortex, and their connecting circuits (Müller-Vahl et al., 2009; Tremblay, Worbe, Thobois, Sgambato-Faure, & Feger, 2015), as well as in abnormal levels of dopamine, a crucial neurotransmitter in frontal/basal ganglia circuits (Buse, Schoenefeld, Munchau, & Roessner, 2013). These frontal/basal ganglia abnormalities, including hyperactive circuitry, have been posited to result in increased disinhibition of frontal activity, leading

\* Corresponding authors at: Newcastle University, Centre for Research in Linguistics and Language Sciences, Newcastle NE7 1RU, United Kingdom (C.D. Dye) and Brain and Language Lab, Department of Neuroscience, Georgetown University, United States (M.T. Ullman).

E-mail addresses: [cristina.dye@ncl.ac.uk](mailto:cristina.dye@ncl.ac.uk) (C.D. Dye), [michael@georgetown.edu](mailto:michael@georgetown.edu) (M.T. Ullman).

to tics and a hyperkinetic behavioral profile (Johannes et al., 2002; Ziemann, Paulus, & Rothenberger, 1997).

Although most research has focused on delineating the impairments and weaknesses of TS (Murphy & Eddy, 2013), some studies have begun to examine strengths in the disorder. Thus far, strengths have been found in somewhat disparate domains and functions, and have not been consistently observed, or have been reported in a small number of studies.

First, some studies have reported TS advantages in what have been characterized as executive functions such as volitional control and task switching. For example, children with TS have been found to perform faster and/or more accurately than TD controls on mixed-trial anti-saccade tasks, which involve repeatedly switching between pro-saccade and anti-saccade trials, i.e., between eye movements towards vs. away from a target (Jackson, Mueller, Hambleton, & Hollis, 2007; Jung, Jackson, Nam, Hollis, & Jackson, 2014; Jung, Jackson, Parkinson, & Jackson, 2013; Mueller, Jackson, Ranu, Sophia, & Hollis, 2006). Speeded reaction times have also been observed on an analogous task involving hand instead of eye movements (Jackson et al., 2011). It has been argued that these findings may be explained by children with TS developing improved control abilities due to their constant efforts at tic suppression (Mueller et al., 2006). Some evidence has also been taken to suggest TS strengths at working memory updating (Thibault et al., 2008). However, other tasks probing executive functions, including working memory, have shown normal or even mildly impaired performance in TS, weakening the hypothesis that the disorder is associated with strengths in this domain (Channon, Gunning, Frankl, & Robertson, 2006; Mostofsky, Lasker, Singer, Denckla, & Zee, 2001; Murphy & Eddy, 2013).

Second, we are aware of four studies that have reported strengths related to motor function or knowledge. One study found that children with TS showed faster movements than TD controls on a finger sequence task (Avanzino et al., 2011). This was found when performing the task both with the right hand only and bimanually, though the right hand condition was also associated with decreased accuracy. The results were taken to indicate altered organization of inter-hemispheric connections in TS. Another study found that individuals with TS showed faster and more force-efficient performance than controls in some (though not all) goal directed movements, with no accuracy differences (Georgiou, Bradshaw, Phillips, Cunningham, & Rogers, 1997). This was taken to reflect abnormal motor asymmetry due to abnormal basal ganglia. Yet another study found superior motor performance in combination with reward in TS, which was explained in terms of overactive dopamine transmission (Palminteri et al., 2011). A fourth study found that TS participants were faster (but not more accurate) than controls at naming manipulated objects such as *hammer*, but not non-manipulated objects such as *elephant* (Walenski, Mostofsky, & Ullman, 2007). Since knowledge of manipulated objects seems to involve motor skill knowledge learned in procedural memory (in contrast to non-manipulated objects, which may primarily involve conceptual knowledge learned in declarative memory; Ullman, 2007), the findings were attributed to atypically fast processing of knowledge learned in the procedural memory brain system.

Third, two studies have reported strengths in other domains. One study found superior TS performance (in both speed and accuracy) on one of four conditions in a time processing task (Vicario et al., 2010). The authors suggested that the results were consistent with enhanced cognitive control abilities. Another study reported enhancements in mental rotation (more correct responses in a 5 min. period, thus reflecting a combination of speed and accuracy) in females with TS relative to female TD controls, though it was impaired in males with TS relative to male controls (Alexander &

Peterson, 2004). The results were discussed with regard to potential associations between elevated prenatal androgen levels and brain masculinization.

Overall, there seems to be little if any functional coherence to the TS strengths reported thus far. However, these strengths may all be related *neurobiologically*, in particular because all the types of tasks and functions for which TS strengths have been reported appear to depend on frontal and/or basal ganglia structures, and in most cases dopamine as well: control, task switching and other executive functions (Ravizza & Ciranni, 2002); working memory (Smith & Jonides, 1998); motor function and knowledge (Swinnen et al., 2010), including for naming manipulated objects (Ullman, 2007); procedural memory (Ullman, 2004); time processing (Ivry & Spencer, 2004); and mental rotation (Cohen et al., 1996). As discussed in greater detail in the Discussion, we suggest that the underlying neurobiology of TS that leads to rapid motor and verbal tics and a hyperkinetic behavioral profile may also lead to the speeded performance (and potentially greater accuracy as well) of a range of other processes that depend on the neurobiological substrates affected in TS, in particular frontal/basal ganglia circuits and related dopaminergic systems. In other words, the abnormal rapidity found in the clinical symptom of tics may extend to other functions that are not part of the clinical manifestation, due to their common reliance on abnormal frontal/basal ganglia circuits. We refer to this neurobiological account as the Clinical Extension Hypothesis.

If previously observed strengths may be best explained not by cognitive or functional accounts, but by the neurobiological abnormalities of frontal/basal ganglia circuits in TS, we would expect that other functions that depend on these circuits may show analogous strengths. A substantial body of work has linked aspects of language, in particular grammar, to frontal/basal ganglia structures and circuitry (for reviews, see Teichmann et al., 2015; Ullman, 2004, 2016). Our mental grammar underlies the rule-governed sequential and hierarchical combination of complex (compositional) linguistic representations, across grammatical subdomains, including syntax (in computing phrases and sentences; e.g., “the” + “cat”, noun phrase + verb phrase), morphology (in computing complex words, for example, in regular inflection; e.g., “walk” + “-ed”) and phonology (in novel word forms, whose phonological elements must somehow be combined according to the phonotactics of the language, i.e., according to the phonological grammar of the language). In particular, evidence suggests that grammatical rules whose learning depends on the basal ganglia are eventually processed in frontal structures, especially in Broca’s area and nearby cortex (e.g., Brodmann’s areas (BA) 44 and 6 Ullman, 2004, 2016). Thus grammatical processing – across syntax, morphology, and phonology – might also be expected to be speeded in TS.

Surprisingly, there has been very little research examining grammar in TS (a handful of studies have investigated other aspects of language in TS, though none have reported strengths; Legg, Penn, Temlett, & Sonnenberg, 2005). We are aware of no studies of either syntax or phonology and only one of morphology. Consistent with our hypothesis, this study reported speeded performance (Walenski et al., 2007). Children with TS and age- and sex-matched TD control children (aged 8–17) were tested on a past tense production task, in which participants were presented with verb stems (e.g., *walk*, *dig*), and were asked to produce their past tenses as quickly and accurately as possible. The verbs were of several types, including regulars (both existing and novel, e.g., *slip*, *rick*), and irregulars (both existing and novel, e.g., *dig*, *spling*). The group with TS was significantly faster than the TD group at producing regular past tenses (for both existing and novel regulars), which evidence suggests undergo rule-governed composition by the mental grammar (e.g., “slip” + “-ed”) and

depend on frontal/basal ganglia circuits (Pinker & Ullman, 2002; Ullman, 2004, 2016). In contrast, there were no group differences in response times in the production of irregular past tense forms, which evidence suggests are retrieved from (*dug*) or processed in (*splung*) associative memory (Pinker & Ullman, 2002; Ullman, 2004). Likewise, there were no group differences in “inconsistent” regulars (e.g., *squeezed*), which, like irregulars, appear to be retrieved from memory. There two groups did not differ in accuracy on any verb type. The authors argued that the speeded performance of only the rule-governed combined morphological forms (*slipped*, *ricked*) may reflect a broader pattern of speeded rule-governed grammatical composition in TS. Moreover, since independent evidence indicates that grammar, in particular rule-governed composition, is learned and processed in the procedural memory system, which is rooted in frontal/basal ganglia circuits (Ullman, 2004), it was suggested that the observed pattern might reflect speeded processing of knowledge learned in procedural memory more generally, that is, of both linguistic and non-linguistic knowledge. Indeed, the speeded performance observed at naming manipulated (and not non-manipulated) objects described above was found in the same participants (Walenski et al., 2007), underscoring the plausibility of this account.

The current study aims to advance the investigation of speeded grammar in TS by examining whether the speeded processing of morphology extends to another key domain of grammar: phonology, that is, the sound structure of words. In particular, if children with TS are faster at rule-governed grammatical combination in general, they should show not only speeded combination of morphological units, that is, of morphemes (“walk” + “-ed”), but also of phonological units, that is, phonemes or syllables.

In order to test whether the findings suggesting speeded TS composition in regular morphology might extend to phonology, we probed TS and TD participants on a non-word repetition task, in which participants hear and repeat aloud phonological sequences such as /teivɔɪtʃaɪg/. These sequences do not exist as words in the language, and have no meanings (hence the term ‘non-words’), but generally follow the phonological grammar (phonotactics) of the language, in this case American English. The dependent measure in this task is typically accuracy.

The non-word repetition task taps language-related functions in the domain of phonology that seem to be analogous to those involved in morphology in the past-tense production of regulars (Walenski et al., 2007). Perhaps most importantly, like the production of regular past tenses, which appears to involve the rule-governed composition of morphemes (Pinker & Ullman, 2002; Ullman, 2004, 2016), the non-word repetition task also seems to involve rule-governed (de)composition, though in this case in the manipulation of phonological segments (e.g., phonemes or syllables). Participants do not simply repeat the novel sequence of phonemes as an unanalyzed whole. Rather, several lines of evidence suggest that they generally break it down into smaller phonological units, and then attempt to reconstruct the phonological sequence in their output, according to the phonotactics of the language.

First, evidence suggests that in non-word repetition individual segments (e.g., phonemes, diphones, or syllables) are accessed. In particular, studies have found that properties of individual segments affect task performance, which would not be expected if the string were simply repeated as an unanalyzed whole. For example, accuracy of repetition is modulated by phoneme type (e.g., coronal stops vs. coronal fricatives, Cleary, Dillon, & Pisoni, 2002), the presence of singleton consonants vs. consonant clusters (Gathercole & Baddeley, 1989) syllable type (real-word or not; e.g. BATHesis versus FATHesis; Dollaghan, Biber, & Campbell, 1995), and phoneme, diphone, and syllable frequency (Coady & Aslin, 2004; Coady, Evans, & Kluender, 2010; Tremblay,

Deschamps, Baroni, & Hasson, 2016). Evidence also suggests that the position of a segment within the non-word string modulates performance at the task (Coady & Evans, 2008; Tremblay et al., 2016). Furthermore, Barry, Hardiman, and Bishop (2009) provided electrophysiological evidence of sensitivity to individual segments in a non-word repetition strings, while a recent fMRI study demonstrated neural sensitivity to syllable frequency in non-word strings in the task (Tremblay et al., 2016).

Second, evidence suggests that in non-word repetition individual phonological segments are not just accessed, but also stored in short-term/working memory. Non-word repetition accuracy correlates with accuracy at traditional measures of phonological short-term memory, such as digit span (which clearly involves individual units) (Adams & Gathercole, 2000). Additionally, length effects have been commonly reported for the non-word strings (i.e., strings with more segments are repeated less accurately than those with fewer segments), which has been interpreted as suggesting that longer strings constitute a greater load on short-term memory, due to the greater number of individual segments (Gathercole & Adams, 1994). Also, patients with auditory short term memory impairments have shown poor performance on the task, especially with longer items (Baddeley, Gathercole, & Papagno, 1998). Finally, the segments in the non-words show the same type of primacy and recency effects that are encountered in the serial recall of individual words or numbers, suggesting that they are maintained as individual units (Archibald & Gathercole, 2007b; Gupta, 2005).

Third, evidence suggests that individual segments are not only accessed and stored, but also manipulated. Error analyses of responses have revealed that migration errors, which entail re-ordering segments, suggesting the manipulation of phonemes, are common errors, as are addition, deletion, and substitution errors, also consistent with phoneme manipulation (Archibald & Gathercole, 2007a; Jones & Witherstone, 2011; Nakeva Von Mentzer et al., 2015). Furthermore, Archibald and Gathercole (2007a) found that migrations were more common in non-word repetition than in the repetition of syllable lists (matched to the non-words on syllable length and content), further supporting phoneme manipulation in non-word repetition.

Finally, evidence suggests that the re(composition) of the individual segments in non-word repetition is influenced by phonotactic rules. For example, at least in typically-developing children and adults, the output (i.e., the produced form) is modulated not only by the input sequence, but also by phonotactic rules in the speaker’s language (Coady & Evans, 2008). Along the same lines, non-words that follow the speaker’s phonotactic rules are repeated more accurately than those that do not, consistent with the task being sensitive to the phonotactics of the language (Munson, Kurtz, & Windsor, 2005; Nakeva Von Mentzer et al., 2015; Vitevitch, Luce, Charles-Luce, & Kemmerer, 1997).

Thus, overall, multiple lines of evidence suggest that non-word repetition generally undergoes decomposition and then recomposition of phonological units, following the phonotactic rules of the language.

The similarity between non-word repetition and the production of regulars in past-tense production extends beyond their mutual dependence on rule-governed composition. In addition, both tasks probe rule-governed processing in oral production, even though they differ in the extent to which the stimulus is included in the target (fully in non-word repetition, and only partially in past tense production, in which participants need to add an *-ed* suffix). Moreover, both tasks seem to tap short-term memory. As we have seen above, evidence suggests that non-word repetition involves temporarily maintaining the disassembled phonological units in short-term memory. Similarly, recent evidence suggests that the morphemes seem to be retained in short term memory in the

processing of regular past tense forms (Walenski, Prado, Ozawa, Steinha, & Ullman, submitted for publication). Finally, more direct evidence also suggests that past tense production and non-word repetition are related, since performance at the two tasks has been found to correlate in typically-developing children (Christensen & Hansson, 2012).

The non-word repetition paradigm not only shows important similarities and overlap with the production of regulars in the past-tense production task, but also has the advantage of being widely used, particularly with neurodevelopmental disorders. Thus it is well understood, and allows for comparability among disorders. Performance at the task seems to be impaired in most neurodevelopmental disorders in which it has been examined, including specific language impairment/dyslexia (Melby-Lervåg, Lyster, & Hulme, 2012), autism spectrum disorder (Tager-Flusberg & Joseph, 2003) and Down syndrome (Comblain, 1999). It has been less well studied in other developmental disorders. We are aware of one study in Attention Deficit Hyperactivity Disorder (ADHD), which did not find group differences (Redmond, Thompson, & Goldstein, 2011), and a handful of studies in Williams syndrome, which reported performance worse or similar to that of controls (Grant, Karmiloff-Smith, Berthoud, & Christophe, 1996). We are not aware of any studies examining the task in either Obsessive Compulsive Disorder (OCD) or Tourette syndrome. Importantly, there has been very little research on the task in neurodevelopmental disorders examining response time (in addition to accuracy) as a dependent measure, as was done in the present study (we are aware of only one study, which examined children who stutter, and found no group differences compared to typically-developing children; Sasisekaran & Byrd, 2013).

Two of the most widely used non-word repetition tests are the Children's Test of Non-word Repetition (CNRep; Gathercole, Willis, Baddeley, & Emslie, 1994) and the Non-word Repetition Test (NRT; Dollaghan & Campbell, 1998). Although seemingly similar, these differ in certain important respects. Of particular interest here, the characteristics of the NRT seem to be better suited for probing phonological combination. First of all, in the CNRep, quite a few items pose particular articulatory difficulties; as a result, performance may be especially affected by articulation, which is not of interest here, and could confound the results. This could be particularly problematic in TS, which has been associated with articulatory and fluency difficulties (De Nil, Sasisekaran, Van Lieshout, & Sandor, 2005). Specifically, the CNRep contains some items with complex consonant clusters (/ˈblɒntəˈsteɪpɪŋ/), and most items have weak syllables (which have reduced vowels; e.g., /ˈhæmpɒnt/, /ˈtæfləst/); both of these seem to pose articulatory difficulties that can affect non-word repetition performance (Coady & Evans, 2008). In contrast, the NRT contains no items with either complex consonant clusters or weak syllables. Rather all items contain simple CV(C) syllables, and have equal stress on each syllable – which has the added benefit of making the items acoustically salient. Second, in the CNRep many items include phonological sequences that correspond to existing words or morphemes (e.g., 'pen' in /ˈpenl/ or '-ing' in /ˈblɒntəˈsteɪpɪŋ/). This is problematic for testing phoneme (de)composition and manipulation, since these sequences could be treated as chunks rather than broken into individual phonological units. In contrast, the items in NRT were specifically selected not to contain any such phonological sequences.

The present study, which examined whether fast grammatical processing in TS might extend from morphology to phonology, tested children with TS and age- and sex-matched TD children on the NRT.

**Table 1**  
Participant information.

	TS children (n = 13)	TD children (n = 14)
Age	Mean 11.87, SD 2.5	Mean 11.31, SD 2.1
Full Scale IQ (FSIQ)	Mean 101.46, SD 11.2	Mean 120.86, SD 10.6
Right handed	9	13
Male sex	12	12
Comorbidities	ADHD 2; OCD 3	None
Psychoactive medication	7 taking medication	None

## 2. Methods

### 2.1. Participants

Thirteen children diagnosed with TS, aged 8–16 years (Table 1), were recruited through the Child Neurology Outpatient Clinic at the Johns Hopkins Hospital. All met the diagnostic criteria defined by the Tourette syndrome Classification Group (1993), confirmed by a child neurologist (author S.H.M.). ADHD and OCD are common in TS (American Psychiatric Association, 2013), so children with these comorbidities were not excluded. Two of the children with TS met criteria for ADHD, as determined by the DICA-IV criteria (Reich, Welner, & Herjanic, 1997), and three met criteria for OCD, also determined by the DICA-IV criteria (one child had both ADHD and OCD). Seven of the 13 children with TS were taking psychoactive medication at the time of testing: one was taking haloperidol and fluvoxamine, 1 aripiprazole, 1 lamotrigine and fluvoxamine; 1 dextroamphetamine and clonidine; 1 methylphenidate and clonidine; 1 sertraline hydrochloride; 1 risperidone; and 1 pimozone and clonidine. The age range of the participants was determined by the age range in the past tense production article (Walenski et al., 2007), as well as patient availability. All children with TS were tested at the Kennedy Krieger Institute (KKI).

In addition, 14 typically-developing control children aged 8–15 were tested (Table 1). They were group-matched to the children with TS on age, handedness, and sex ( $ps > 0.5$ ); full scale IQ differed between the groups ( $p < 0.05$ ); these and other variables were considered as covariates in all analyses (see below). The control group was recruited through local schools and community-wide service groups. Five of the controls were tested at KKI and 9 at the Georgetown University Medical Center (GUMC). No control children had any known developmental or psychiatric disorders, and none had any history of tics; this assessment was based on parental report and the DICA-IV criteria (Reich et al., 1997) for children tested at KKI, and parental report and the Child Behavioral Checklist (Achenbach & Dumenci, 2001) for children tested at GUMC. Absence of ADHD was further confirmed with the Conners ADHD parent scale (Conners, 2008) and the DuPaul ADHD parent scale (DuPaul, Power, Anastopoulos, & Reid, 1998). None of the control children were taking psychoactive medication.

All children were monolingual native American-English speakers from the Washington- Baltimore area. Both right and non-right handed children were included (Oldfield, 1971). Full scale IQ was assessed with the Wechsler Intelligence Scale for Children – III (Wechsler, 1991), except for the 9 controls tested at GUMC, who were administered the Kaufman Brief Intelligence Test, second edition (Strauss, Sherman, & Spreen, 2006). The scores for these two tests are highly correlated (Strauss et al., 2006). The Institutional Review Boards of Johns Hopkins University and GUMC provided approval for this study. All participants provided written consent (caregivers) and assent (children) before testing, and received a copy of the consent form.

## 2.2. Non-word Repetition Task

All children were tested on a modified version of the Non-word Repetition Test (NRT; [Dollaghan & Campbell, 1998](#)). In summary, the task consists of 16 test items that are phonetically legal in American English: four non-words at each of four syllable lengths (one, two, three, and four syllables), presented in order of increasing length. That is, the two one syllable non-words are presented first, followed by the two syllable non-words, and so on. All non-words begin and end with a consonant and contain no consonant clusters. Thus, 1-syllable non-words are consonant-vowel-consonant (CVC) sequences; 2-syllable non-words CV.CVC; 3-syllable non-words CV.CV.CVC; and 4-syllable non-words CV.CV.CV.CVC. The 16 items were as follows (represented in IPA): /naɪb/, /vɒʊp/, /taʊdʒ/, /dɔɪf/, /teɪvək/, /tʃɔʊvæɡ/, /væʃtʃaɪp/, /nɔɪtaʊf/, /tʃɪnɔɪtaʊb/, /naɪtʃɔʊveɪb/, /dɔɪtaʊvæb/, /teɪvɔɪtʃaɪɡ/, /veɪtatʃaɪdɔɪp/, /dæʊvɔʊnɔɪtʃɪɡ/, /natʃɔɪtaʊvub/, /tæʊvətʃɪnaɪɡ/. For full details regarding the items, see [Dollaghan and Campbell \(1998\)](#). In addition to the 16 test items, 3 practice items (/bæθrʌs/, /rɛfɪlɪt/, /træʃɪmʌs/) are presented to ensure participants understand the task. If the child makes an attempt to repeat at least 2 of the 3 practice non-words, the experimenter continues to the test items.

Whereas [Dollaghan and Campbell \(1998\)](#) collected accuracy data alone, given the research questions in the current study we additionally needed response time data. To this end, an E-Prime version of the task was created, using the original recordings of the practice and test items (produced by a trained female native American-English speaker). All stimuli were presented aurally to the child through Sony MDR-XD200 Stereo headphones using E-prime version 1.1 on a PC computer. The children were tested individually in a quiet room, and were told: “Now you’re going to hear some made-up words. As soon as each word is finished please repeat exactly what you’ve heard. If you aren’t sure, just do the best you can.” Presentation of the non-word initiated a software timer, which was terminated by the child’s oral response (via a microphone connected to the computer). The RT dependent measures (see below) was computed as the time from the end of the presented stimulus (the end of the non-word) to the beginning of the response. To advance to the next stimulus, the experimenter clicked the mouse button. The entire session was audio-recorded.

Responses were transcribed phonemically from the audio recording, independently by two transcribers trained in phonological transcription. The transcribers were not aware of the study hypotheses regarding TS, though they were not blind to diagnosis. There was less than 5% disagreement (4.83%) between the two transcribers. The rare disagreements were independently resolved by a third trained transcriber. Responses were coded as either correct or incorrect. Following other studies of non-word repetition ([Archibald & Gathercole, 2007a](#)), a response was coded as correct only if it precisely matched the sequence of phonemes in the stimulus. Thus, any phoneme deletions, additions, or substitutions resulted in a categorization of the response as incorrect.

## 2.3. Analysis

The two dependent variables were (1) accuracy, that is, whether the first response was correct or incorrect (as a binary variable, scored for each item for each participant), and (2) response times (RTs) to correct first responses. A logit-link function (for binary outcome data) was used for accuracy analyses (SAS 9.2 proc glimmix). Response times were analyzed with SAS 9.2 proc mixed. For response time analyses, response time errors (response times of 0, indicating a response before the end of the stimulus; 2.7% of all responses) as well as incorrect first responses (18%) were excluded. RTs were natural-log transformed to reduce the skewness of the distribution. Both dependent variables were analyzed in SAS (ver-

sion 9.2) using restricted maximum likelihood (REML) in mixed-effects regression models with crossed random effects for subjects and items on the intercept and a fixed effect of participant group (TS vs. TD control). The models were fit with an unstructured covariance matrix for each random effect.

Both the accuracy and RT analyses were performed (and are reported below) in two ways: once *without* any covariates, and a second time *with* the inclusion of potentially confounding subject- and item-level variables, to examine whether the effects were reliable when analyzed with the influence of covariates removed ([Walenski et al., 2014](#)). We considered 11 such variables. The 7 subject-level covariates consisted of subject FSIQ, age, sex, and handedness (binary variable indicating whether or not a subject was right-handed), and whether or not the child was taking psychotropic medications, had co-morbid ADHD, or had co-morbid OCD. The 4 item-level covariates consisted of the following. First, we included a measure indicating the phonological length of an item (number of syllables), which is likely to affect response times because longer words may require more time for syllabication and articulatory planning ([Levelt, Roelofs, & Meyer, 1999](#)); note that whether item length is counted as syllables or phonemes does not affect analysis outcomes since syllable and phoneme length are fully collinear for the items in the NRT. We also included two variables that describe the type of sound at the beginning of an item, as this can affect computer-recorded response time measurements: one variable coding whether or not the initial sound was a fricative; and another coding whether it was a plosive ([Dye, Walenski, Prado, Mostofsky, & Ullman, 2013](#)). Finally, we included a variable reflecting whether or not an item had the same phonological length as the previously presented item ([Dye et al., 2013](#)).

For the analyses without any covariates, none of the above 11 variables were included in either the accuracy or RT models. For the analyses with covariates, all 11 variables above were initially included in both the accuracy and RT models. However, those variables that were not significant at a 0.2  $\alpha$ -level ( $ps > 0.2$ ) were subsequently removed from the final models (following [Walenski et al., 2014](#)). For accuracy analysis, the covariates that were retained in the final model were FSIQ ( $\beta = 0.03$ ,  $t(22.7) = 1.4$ ,  $p = 0.17$ ), age ( $\beta = 0.28$ ,  $t(22.3) = 2.4$ ,  $p = 0.02$ ), and phonological length ( $\beta = -1.5$ ,  $t(14.4) = 4.88$ ,  $p = 0.0002$ ). For RT analysis, the only variable that was retained as a covariate in the final model with covariates was phonological length ( $\beta = 0.14$ ,  $t(14.9) = 4.3$ ,  $p = 0.0007$ ).

Below, we report type III F tests for the results of the fixed effect of group, with significance assessed at  $\alpha = 0.05$ . All  $p$  values are reported as two-tailed. In all analyses, degrees of freedom were computed using the Satterthwaite approximation.

## 3. Results

The children with TS did not differ from the TD control children on the accuracy of their responses in the Non-word Repetition Task; this result also held both without and with covariates included. See [Table 2](#).

In contrast, analysis of response times indicated that the children with TS were significantly faster at producing non-words than the control children. This held in analyses both without and with covariates. See [Table 3](#).

## 4. Discussion

This study tested children with TS and TD children on the Non-word Repetition Task (NRT) ([Dollaghan & Campbell, 1998](#)). The children with TS were faster than the TD children at initiating their responses in non-word repetition. This result held whether or not

**Table 2**  
Accuracy results from the Non-word Repetition Task.

	Means		Group comparison: Analysis without covariates	Adjusted Means		Group comparison: Analysis with covariates
	TS	TD		TS	TD	
Accuracy	82.2% (2.81)	81.7% (4.02)	$F(1,22.7) = 0.00, p = 0.999$	2.79 (0.49)	2.34 (0.53)	$F(1,20.4) = 0.49, p = 0.493$

Note. Mean accuracy (and standard errors in parentheses) are shown for each group. Mean percent correct scores shown here were computed for each subject and then averaged over all subjects in each group. See text for the covariates contributing to the adjusted means in the analysis with covariates. Adjusted means and standard errors from the logistic regression are presented as untransformed values. These can be transformed into probabilities of correct responses with the equation  $y = 1/(1 + e^{-x})$ , where  $x$  is the adjusted mean. Transformed probabilities are not shown here because standard errors are not transformable.

**Table 3**  
Response time results from the Non-word Repetition Task.

	Means		Group comparison: Analysis without covariates	Adjusted Means		Group comparison: Analysis with covariates
	TS	TD		TS	TD	
lnRTs	5.98 (0.04)	6.44 (0.06)	$F(1,24.9) = 5.00, p = 0.035$	5.99 (0.16)	6.48 (0.15)	$F(1,24.9) = 5.03, p = 0.034$

Note. Mean natural-log (ln) transformed RTs (and standard errors in parentheses) are shown for each group. Means were computed for each subject and then averaged over all subjects in each group. Untransformed means: TS RTs: 442 ms (SD 41); TD RTs: 903 ms (SD 210). See text for the covariates contributing to the adjusted means in the analysis with covariates.

covariates were included in the analyses. No group differences were found on accuracy.

The analyses with covariates revealed that this pattern was likely not explained by a number of potentially confounding subject-level variables (sex, age, full scale IQ, handedness, medication, ADHD diagnosis, OCD diagnosis) or item-level variables (phonological length, the type of sound at the beginning of an item, and whether or not an item had the same phonological length as the previously presented item). Additionally, a speed-accuracy trade-off between the two groups cannot account for the results, since the two groups differed in speed but not in accuracy.

The results suggest that one or more processes that are involved in the NRT are speeded in TS. Based on previous research on non-word repetition, and the NRT in particular, both the (de)composition and maintenance of phonological elements in short-term memory appear to be particularly important processes in the task (see Section 1).

Of these processes, maintenance in short-term memory does not seem a likely candidate to underlie the observed speeded performance, since it is not clear how the enhancement of this capability would lead to this outcome. Indeed, better short-term memory maintenance or increased capacity might rather be expected to lead to increased accuracy, which was not observed. The pattern of speeded production also does not seem likely to be explained by various other factors. For example, it is not clear how group differences in articulation would underlie the results, since the NRT stimuli were specifically designed to minimize articulatory demands (see Section 1). Moreover, our covariate analyses considered certain variables that reflect aspects of articulation and speech production (phonological length, and the type of sound at the beginning of an item). Additionally, though some evidence suggests that long-term lexical knowledge can affect certain non-word repetition tasks, this seems to hold particularly for those tasks whose stimuli contain chunks of segments that are also found in real words, such as the CNRep, rather than the NRT examined here (Coady & Evans, 2008).

Thus the findings may be largely explained by a speeding in TS of the other main processes involved in non-word repetition, and the NRT in particular, namely phonological decomposition and/or composition (i.e., concatenation). Indeed, as discussed in the Introduction, research on non-word repetition strongly implicates rule-governed (de)composition of phonological segments in the task. Importantly, a role for (de)composition in the finding of speeded

non-word repetition is also consistent with the previous finding that children with TS show speeded processing of morphological forms that appear to be combined (*slipped, ricked*), but not those that seem to be retrieved or processed in memory (*dug, splung, squeezed*) (Walenski et al., 2007). Moreover, because the morphological task in that study involved composition but not decomposition (verb stems, such as *slip* or *rick*, constituted the input in that task), only composition seems to be common to both the morphology (past tense production) and phonology (non-word repetition) tasks showing speeded performance in TS. This suggests that composition in the output might be the locus of speeded processing in TS, or at least an important contributing factor. Note that the finding that the *initiation* of the responses was fast in TS is consistent with speeded composition, since composition seems to take place prior to articulation, as suggested by evidence and models of articulatory planning and speech production (Indefrey & Levelt, 2004). Nevertheless, the evidence from the present study is also consistent with speeded decomposition in non-word repetition, perhaps in addition to speeded composition.

If composition, i.e., concatenation, is indeed speeded in TS, then the observed TS speed advantage should be greater, as compared to TD, with more concatenation. We therefore performed exploratory analyses on the effects of non-word length. First of all, whereas increasing syllable number (i.e., non-words of increasing length, from one to four syllables) led to increasing RTs in the TD group ( $F(3,12.8) = 8.8, p = 0.002$ ), consistent with previous findings (Coady & Evans, 2008), there was no effect of syllable number on RTs in the TS group ( $F(1,24.9) = 5.96, p = 0.186$ ). This is consistent with the view that an increasing difficulty with syllable number is offset in the TS group by an increasing RT advantage due to more concatenation. Additionally, even though the TS group was significantly faster than the TD group on non-words of all four lengths (1 syllable:  $t(30.3) = 2.06, p = 0.049$ ; 2 syllable:  $t(30.3) = 2.49, p = 0.019$ ; 3 syllable:  $t(30.8) = 2.35, p = 0.025$ ; 4 syllable:  $t(39.6) = 2.88, p = 0.006$ ), the group difference seems larger for 4 syllable than 1 syllable non-words. However, the group (TS, TD) by syllable number (1–4) interaction was not significant ( $F(3,312) = 1.03, p = 0.382$ ). Note that these analyses were performed with no covariates, and an almost identical pattern was obtained with the appropriate covariates (see above) included. It remains to be seen whether the hints at a greater TS speed advantage with more concatenation might be borne out with future studies with more power.

Why might composition be speeded in TS? One possibility is that speeded performance is specific to composition in grammar, including both morphology and phonology. However, the same TS participants who showed fast production of morphologically composed forms were also fast at naming manipulated (but not non-manipulated) objects, which was taken to suggest that children with TS may be fast at processing knowledge learned in procedural memory, including both linguistic and motor knowledge (Walenski et al., 2007). Although a TS account of speeded processing of knowledge learned in procedural memory seems plausible, an underlying neurobiological account is still needed. Moreover, procedural memory may not easily explain speeded performance in TS in tasks designed to probe various other aspects of cognition (see Section 1).

As discussed briefly in the Introduction, here we suggest that a neurobiological account may explain most if not all of the findings of speeded performance in TS, including those reported in the present study. In particular, we propose the neurobiologically motivated Clinical Extension Hypothesis. This posits that the speeded performance found for phonology (reported here) and morphology (Walenski et al., 2007), as well as the speeded performance (and perhaps increased accuracy; see below) observed in other tasks in TS (see Section 1), may be largely explained by neurobiological mechanisms that also result in clinical symptoms, in particular in the rapidity of the motor and verbal tics that characterize the disorder. That is, the rapidity of tics may *extend* to other functions that are not part of the clinical symptomology, in particular functions that depend on the neurobiological substrates that are abnormal in TS and lead to tics.

As stated in the Introduction, a key characteristic of tics is their rapidity (American Psychiatric Association, 2013). Interestingly, it is less clear whether or not and to what extent tics are voluntary (Leckman, Bloch, Sukhodolovsky, Scahill, & King, 2013). Although, not surprisingly, some evidence suggests that tics are different from voluntary movements (Obeso, Rothwell, & Marsden, 1981), recent research suggests that they are at least partially voluntary, being carried out to relieve sensory urges (Leckman et al., 2013; Puts et al., 2015). Thus, it is plausible that at least certain other processes might also be rapid in TS, even if they do not show evidence of the apparently involuntary nature of tics. Tics may therefore simply be the clinical tip of the iceberg of rapidity.

We suggest that any motor or cognitive processes that depend on the neurobiological substrates that underlie tics, in particular that underlie their rapidity, could potentially also be speeded. Although these neurobiological substrates are not fully elucidated, they are beginning to be understood. As discussed in the Introduction, abnormalities of frontal/basal ganglia circuits and dopamine are clearly implicated in the disorder, including in tics. Indeed, an important model of TS posits an imbalance between the direct and indirect pathways within the basal ganglia that leads to the increased disinhibition of frontal (and other regions) to which the basal ganglia project, via the thalamus, accounting for the tics and hyperkinetic profile of the disorder (Albin & Mink, 2006; Mink, 2006). Various lines of evidence support this view (Heise et al., 2010; Kalanithi et al., 2005; Kataoka et al., 2010; McCairn, Bronfeld, Belevovsky, & Bar-Gad, 2009). Additionally, individuals with TS may show enhanced structural and functional connectivity both in frontal/basal ganglia circuits and within frontal cortices, with the abnormalities associated with increased frontal activity and the hyperkinetic/tic profile (Wang et al., 2011; Worbe et al., 2012, 2015).

As indicated in Section 1, all of the functions and tasks for which speeded (or in some cases more accurate) performance, has been observed, including morphology and phonology, depend on frontal and/or basal ganglia structures, and in many cases dopamine as well. Importantly, this also holds for non-word repetition and

related tasks (Kalm & Norris, 2014; Liegeois, Morgan, Connelly, & Vargha-Khadem, 2011; Papoutsi et al., 2009; Peeva et al., 2010; Strand, Forssberg, Klingberg, & Norrelgen, 2008). Moreover, children with TS appear to have faster speech rates than TD children (De Nil et al., 2005), and speech production also depends on these structures (Munhall, 2001). Overall, these data lend support to the hypothesis that the frontal/basal ganglia and dopaminergic abnormalities in TS may underlie not only the tics and hyperkinetic profile, but also the rapid processing reported for a variety of tasks in the disorder, including non-word repetition.

Importantly, we do not expect that all tasks and functions that depend on frontal/basal ganglia circuits should show speeded processing. First of all, for a process to be speeded it would presumably have to have the potential to be speeded (e.g., unlike working memory capacity), and depend on the frontal/basal ganglia circuits in such a way that enable speeded processing (the specifics of which remain to be determined). Note that increased accuracy may also be expected in at least some cases: greater accuracy can result from speeded processing, since accuracy is generally determined with a time cutoff, so slow responses are also often counted as incorrect. Conversely, it is also possible that in some cases in TS the disinhibition of frontal structures might result in actual errors, as has been suggested for patients with Huntington's disease, for example in their production of multiply affixed regular past tense forms such as *slippeded* (Ullman et al., 1997). It is not clear why such errors have not been observed in TS; future studies may elucidate this.

Second, not all portions of frontal cortex and the basal ganglia are abnormal. One might expect only those functions that depend on frontal/basal ganglia "loops" whose underlying frontal and/or basal ganglia circuitry is abnormal to be affected. Motor and premotor regions, as well as Broca's area, have all been found to be abnormal in TS and linked to tics (Müller-Vahl et al., 2014; Stern et al., 2000; Wang et al., 2011), suggesting that various aspects of motor and language function, including procedural memory (Ullman, 2004), might be speeded in TS.

Indeed, most of the speeded behaviors found in TS could in principle be due to just a few underlying functions, including potentially procedural memory. Speeding of skills learned in this system could explain observed findings of rapid morphological and phonological processing, as well as the speeding of naming manipulated objects, complex motor tasks, goal-directed movement, and mental rotation (Ullman & Pierpont, 2005). Note that while some studies have suggested that *learning* in procedural memory may be impaired in TS (Keri, Szlobodnyik, Benedek, Janka, & Gadoros, 2002; Marsh et al., 2004), other studies have found no differences, or even enhanced learning (Nemeth et al., 2015). Even if procedural learning turns out to be impaired in TS, this does not preclude the possibility of speeded *processing* of the knowledge learned in this system. Indeed, given the disinhibition of frontal structures in TS, together with evidence suggesting that whereas learning skills in procedural memory relies heavily on the basal ganglia, once learned they are processed in frontal structures (Ashby, Turner, & Horvitz, 2010; Doyon et al., 2009), it is not unreasonable that only processing of well-learned skills in this system would be speeded.

The present findings and proposed neurobiological account for speeded behaviors in TS have various implications and potentially open new avenues of research. First of all, the finding of speeded non-word repetition and other speeded tasks in TS may also be observed in other disorders, especially those that are comorbid with TS, involve frontal and/or basal ganglia abnormalities, and are associated with symptoms that may be related to tics (e.g., stereotypies, obsessions, or impulsivity) – for example, autism spectrum disorder, OCD, and ADHD (Bradshaw, 2001). Indeed, some evidence suggests speeded processing in some of these disor-

ders (Just et al., 2004; Mavrogiorgou et al., 2002; Morault, Bourgeois, Laville, Bensch, & Paty, 1997; Walenski et al., 2014). For example, children with high functioning autism have been found to show the same pattern of speeded morphology as children with TS (Walenski et al., 2014). Interestingly, evidence also suggests speeded behaviors in Huntington's disease (Beste, Humphries, & Saft, 2014), consistent with the hyperkinetic profile of the disorder.

Second, more generally, the Clinical Extension Hypothesis may warrant investigation in other disorders for characteristics beyond rapid processing. According to this neurobiological hypothesis, the characteristics of any symptom in any disorder could potentially extend to other functions that are not part of the clinical manifestation of the disorder, but depend on the neurobiological substrates that are abnormal in the disorder and lead to the clinical symptoms. Although in many cases this might simply lead to more subtle (non-clinical) deficits in other functions or domains, in some cases it might lead to unexpected behaviors, such as speeded processing, and perhaps even strengths.

Third, the present study addresses certain gaps in the non-word repetition literature. To our knowledge, this is among the first studies to examine response times (not just accuracy) in non-word repetition in neurodevelopmental disorders, and the first to examine the task in any form in TS. It also seems to be the first to identify an apparent strength (speeding) in the non-word repetition task in any disorder. It may behoove future studies to examine response times in this and related tasks in a variety of neurodevelopmental and other disorders. More generally, the findings underscore the usefulness of investigating response times, and suggest that accuracy may sometimes be too crude a measure to detect atypical language processing (Walenski et al., 2014).

Fourth, the findings may have clinical implications. In particular, given that performance on non-word repetition seems to be impaired in most other neurodevelopmental disorders (at least as measured by accuracy), this test could potentially be used as an early diagnostic or predictor of TS in at-risk children (e.g., those with TS in their family) – especially since the onset of tics is usually later (around the age of 6 years of age; Singer, 2013) than the age at which non-word repetition abilities have been tested (as young as two; Coady & Aslin, 2004). Note that non-word repetition relies on imitation, a natural behavior even in infants.

Fifth, the results warrant examination in other domains, both in language (e.g., syntax), as well as other functions, including those that might rely on procedural memory, or whose functional anatomy overlaps that of the abnormalities seen in TS.

Finally, future work should investigate whether the findings reported here could translate into actual advantages for children or adults with TS, including in everyday life. After all, speeded language (or motor or cognitive functions) do not necessarily provide benefits. Some work in this direction has already been carried out for strengths in other disorders (Abraham, Windmann, Siefen, & Daum, 2006), though not in TS.

Of course, the present study also has limitations. First, the non-word repetition task does not have a control condition (i.e. real word repetition), and we did not assess neurobiological parameters of this task. Second, although in the present study the children with TS were faster than the TD children at initiating their responses, it remains to be seen whether or not the children with TS also took less time to complete their responses. Third, at least some studies show no RT or accuracy advantage in TS patients in tasks that involve basal ganglia circuits and could potentially be speeded, such as fine motor skills (Bloch, Sukhodolsky, Leckman, & Schultz, 2006). The hypothesis suggested here therefore needs to be treated with caution and tested in future research.

In conclusion, this study has presented evidence of speeded phonological processing, perhaps specifically in phonological com-

position, in children with TS. This reveals a new area of potential strength in TS, which may also warrant examination in related disorders. We have proposed an underlying neurobiological account for these and other speeded behaviors in TS. This account, the Clinical Extension Hypothesis, posits that the rapidity of tics may extend to other functions, due to their common reliance on abnormal frontal/basal ganglia circuits. Although the hypothesis clearly requires further investigation, it provides a promising theoretical framework for the study of strengths in TS, and possibly in related disorders.

## References

- Abraham, A., Windmann, S., Siefen, R., & Daum, I. (2006). Creative thinking in adolescents with attention deficit hyperactivity disorder (ADHD). *Child Neuropsychology*, 12(2).
- Achenbach, T. M., & Dumenci, L. (2001). Advances in empirically based assessment: Revised cross-informant syndromes and new DSM-oriented scales for the CBCL, YSR, and TRF: Comment on Leguna, Sadowski, Friedrich, and Fisher (2001). *Journal of Consulting and Clinical Psychology*, 69(4), 699–702.
- Adams, A. M., & Gathercole, S. (2000). Limitations in working memory: Implications for language development. *International Journal of Language & Communication Disorders*, 35(1), 95–116.
- Albin, R. L., & Mink, J. W. (2006). Recent advances in Tourette syndrome research. *Trends in Neurosciences*, 29(3), 175–183.
- Alexander, G. M., & Peterson, B. S. (2004). Testing the prenatal hormone hypothesis of tic-related disorders: Gender identity and gender role behavior. *Development and Psychopathology*, 16(2), 407–420.
- American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders: DMS-5* (5th ed.). Washington, D.C.: American Psychiatric Association.
- Archibald, L., & Gathercole, S. (2007a). Nonword repetition in specific language impairment: More than a phonological short-term memory deficit. *Psychonomic Bulletin & Review*, 14(5), 919–924.
- Archibald, L., & Gathercole, S. (2007b). Nonword repetition and serial recall: Equivalent measures of verbal short-term memory? *Applied Psycholinguistics*, 28, 587–606.
- Ashby, F. G., Turner, B. O., & Horvitz, J. C. (2010). Cortical and basal ganglia contributions to habit learning and automaticity. *Trends in Cognitive Sciences*, 14(5), 208–215.
- Avanzino, L., Martino, D., Bove, M., De Grandis, E., Tacchino, A., Pelosin, E., ... Abbruzzese, G. (2011). Movement lateralization and bimanual coordination in children with Tourette syndrome. *Movement Disorders*, 26(11), 2114–2118. <http://dx.doi.org/10.1002/mds.23839>.
- Baddeley, A., Gathercole, S., & Papagno, C. (1998). The phonological loop as a language learning device. *Psychological Review*, 105(1), 158–173.
- Barry, J. G., Hardiman, M. J., & Bishop, D. V. M. (2009). Mismatch response to polysyllabic nonwords: A neurophysiological signature of language learning capacity. *PLoS One*, 4(7), e6270. <http://dx.doi.org/10.1371/journal.pone.0006270>.
- Beste, C., Humphries, M., & Saft, C. (2014). Striatal disorders dissociate mechanisms of enhanced and impaired response selection - Evidence from cognitive neurophysiology and computational modelling. *NeuroImage: Clinical*, 4, 623–634.
- Bloch, M., Sukhodolsky, D. G., Leckman, J. F., & Schultz, R. T. (2006). Fine-motor skill deficits in childhood predict adulthood tic severity and global psychosocial functioning in Tourette's syndrome. *Journal of Child Psychology and Psychiatry*, 47(6), 551–559.
- Bradshaw, J. L. (2001). *Developmental disorders of the frontostriatal system*. Hove, East Sussex, Great Britain: Psychology Press.
- Buse, J., Schoenefeld, K., Munchau, A., & Roessner, V. (2013). Neuromodulation in Tourette syndrome: Dopamine and beyond. *Neuroscience and Biobehavioral Reviews*, 37(6), 1069–1084. <http://dx.doi.org/10.1016/j.neubiorev.2012.10.004>.
- Channon, S., Gunning, A., Frankl, J., & Robertson, M. M. (2006). Tourette's syndrome (TS): Cognitive performance in adults with uncomplicated TS. *Neuropsychology*, 20(1), 58–65.
- Christensen, R. V., & Hansson, K. (2012). The use and productivity of past tense morphology in specific language impairment: An examination of Danish. *Journal of Speech, Language, and Hearing Research*, 55(6), 1671–1689.
- Cleary, M., Dillon, C., & Pisoni, D. (2002). Imitation of nonwords by deaf children after cochlear implantation: Preliminary findings. *Annals of Otology, Rhinology, and Laryngology. Supplement*, 189, 91–96.
- Coady, J., & Aslin, R. N. (2004). Young children's sensitivity to probabilistic phonotactics in the developing lexicon. *Journal of Experimental Child Psychology*, 89(3), 183–213.
- Coady, J., & Evans, J. (2008). Uses and interpretations of non-word repetition tasks in children with and without specific language impairments (SLI). *International Journal of Language & Communication Disorders*, 43(1), 1–40.
- Coady, J., Evans, J., & Klueder, K. (2010). Role of phonotactic frequency in nonword repetition by children with specific language impairments. *International Journal of Language and Communication Disorders*, 45(4), 495–509.
- Cohen, M. S., Kosslyn, S. M., Breiter, H. C., DiGirolamo, G. J., Thompson, W. L., Anderson, A. K., ... Belliveau, J. W. (1996). Changes in cortical activity during

- mental rotation: A mapping study using functional magnetic resonance imaging. *Brain*, 119, 89–100.
- Comblain, A. (1999). The relevance of a nonword repetition task to assess phonological short-term memory in individuals with Down syndrome. *Down's Syndrome Research and Practice*, 6(2), 76–84.
- Conners, K. (2008). *Conners manual* (3rd ed.). Pearson Publishers.
- De Nil, L. F., Sasisekaran, J., Van Lieshout, P. H., & Sandor, P. (2005). Speech disfluencies in individuals with Tourette syndrome. *Journal of Psychosomatic Research*, 58(1), 97–102.
- Diehl, J. J., Frost, S. J., Sherman, G., Mencl, W. E., Kurian, A., Molfeese, P., Pugh, K., ... Pugh, K. R. (2014). Neural correlates of a language and non-language visuospatial processing in adolescents with reading disability. *Neuroimage*, 101, 653–666. <http://dx.doi.org/10.1016/j.neuroimage.2014.07.029>.
- Dollaghan, C., Biber, M., & Campbell, T. (1995). Lexical influences on nonword repetition. *Applied Psycholinguistics*, 16, 211–222.
- Dollaghan, C., & Campbell, T. (1998). Nonword repetition and child language impairment. *Journal of Speech Language and Hearing Research*, 41, 1136–1146.
- Doyon, J., Bellec, P., Amsel, R., Penhune, V. B., Monchi, O., Carrier, J., ... Benali, H. (2009). Contributions of the basal ganglia and functionally related brain structures to motor learning. *Behavioural Brain Research*, 199, 61–75.
- DuPaul, G. J., Power, T. J., Anastopoulos, A. D., & Reid, R. (1998). *ADHD rating scale – IV: Checklists, norms, and clinical interpretation*. New York, NY: Guilford Press.
- Dye, C. D., Walenski, M., Prado, E. L., Mostofsky, S. H., & Ullman, M. T. (2013). Children's computation of complex linguistic forms: A study of frequency and imageability effects. *PLoS One*, 8, e74683.
- Gathercole, S., & Adams, A. M. (1994). Children's phonological working memory: Contributions of long-term knowledge and rehearsal. *Journal of Memory and Language*, 33(5), 672–688.
- Gathercole, S., & Baddeley, A. (1989). Evaluation of the role of phonological STM in the development of vocabulary in children: A longitudinal study. *Journal of Memory and Language*, 28, 200–213.
- Gathercole, S., Willis, C., Baddeley, A., & Emslie, H. (1994). The children's test of nonword repetition: A test of phonological working memory. *Memory*, 2(2), 103–127.
- Georgiou, N., Bradshaw, J. L., Phillips, J. G., Cunningham, R., & Rogers, M. (1997). Functional asymmetries in the movement kinematics of patients with Tourette's syndrome. *Journal of Neurology, Neurosurgery and Psychiatry*, 63(2), 188–195.
- Grant, J., Karmiloff-Smith, A., Berthoud, I., & Christophe, A. (1996). Is the language of people with Williams syndrome mere mimicry? Phonological short-term memory in a foreign language. *Cahiers de Psychologie Cognitive*, 15(6), 615–628.
- Gupta, P. (2005). Primacy and recency in nonword repetition. *Memory*, 13(3–4), 318–324.
- Heise, K. F., Steven, B., Liuzzi, G., Thomalla, G., Jonas, M., Müller-Vahl, K. R., ... Hummel, F. C. (2010). Altered modulation of intracortical excitability during movement preparation in Gilles de la Tourette syndrome. *Brain*, 133(2), 580–590.
- Indefrey, P., & Levelt, W. J. M. (2004). The spatial and temporal signatures of word production components. *Cognition*, 92(1–2), 101–144.
- Ivry, R. B., & Spencer, R. M. (2004). The neural representation of time. *Current Opinion in Neurobiology*, 14(2), 225–232.
- Jackson, G., Mueller, S., Hambleton, K., & Hollis, C. (2007). Enhanced cognitive control in Tourette syndrome during task uncertainty. *Experimental Brain Research*, 182(3), 357–364.
- Jackson, S., Parkinson, A., Jung, J., Ryan, S. E., Morgan, P., Hollis, C., & Jackson, G. (2011). Compensatory neural reorganization in Tourette syndrome. *Current Biology*, 21(7), 580–585.
- Järvinen-Pasley, A., Wallace, G. L., Ramus, F., Happé, F., & Heaton, P. (2008). Enhanced perceptual processing of speech in autism. *Developmental Science*, 11(1), 109–121.
- Johannes, S., Wieringa, B. M., Nager, W., Müller-Vahl, K. R., Dengler, R., & Munte, T. F. (2002). Excessive action monitoring in Tourette syndrome. *Journal of Neurology*, 249(8), 961–966.
- Jones, G., & Witherstone, H. L. (2011). Lexical and sublexical knowledge influences the encoding, storage, and articulation of nonwords. *Memory and Cognition*, 39(4), 588–599.
- Jung, J., Jackson, S. R., Nam, K., Hollis, C., & Jackson, G. M. (2014). Enhanced saccadic control in young people with Tourette syndrome despite slowed pro-saccades. *Journal of Neuropsychology*, 9(2), 172–183.
- Jung, J., Jackson, S. R., Parkinson, A., & Jackson, G. M. (2013). Cognitive control over motor output in Tourette syndrome. *Neuroscience and Biobehavioral Reviews*, 37(6), 1016–1025.
- Just, M. A., Cherkassky, V. L., Keller, T. A., & Minshew, N. J. (2004). Cortical activation and synchronization during sentence comprehension in high-functioning autism: Evidence of underconnectivity. *Brain*, 127, 1811–1821.
- Kalanithi, P. S., Zheng, W., Kataoka, Y., DiFiglia, M., Grantz, H., Saper, C. B., ... Vaccarino, F. M. (2005). Altered parvalbumin-positive neuron distribution in basal ganglia of individuals with Tourette syndrome. *Proceedings of the National Academy of Sciences (USA)*, 102(37), 13307–13312.
- Kalm, K., & Norris, D. (2014). The representation of order information in auditory-verbal short-term memory. *Journal of Neuroscience*, 34(20), 6879–6886.
- Kataoka, Y., Kalanithi, P. S., Grantz, H., Schwartz, M. L., Saper, C., Leckman, J. F., & Vaccarino, F. M. (2010). Decreased number of parvalbumin and cholinergic interneurons in the striatum of individuals with Tourette syndrome. *Journal of Comparative Neurology*, 518(3), 277–291.
- Keri, S., Szlobodnyik, C., Benedek, G., Janka, Z., & Gadoros, J. (2002). Probabilistic classification learning in Tourette syndrome. *Neuropsychologia*, 40(8), 1356–1362.
- Leckman, J. F., Bloch, M., Sukhodolosky, D., Scahill, L., & King, R. (2013). Phenomenology of tics and sensory urges. In D. Martino & J. F. Leckman (Eds.), *Tourette syndrome* (pp. 3–25). Oxford, UK: Oxford University Press.
- Legg, C., Penn, C., Temlett, J., & Sonnenberg, B. (2005). Language skills of adolescents with Tourette syndrome. *Clinical Linguistics & Phonetics*, 19(1), 15–33.
- Levelt, W. J., Roelofs, A., & Meyer, A. S. (1999). A theory of lexical access in speech production. *Behavioral and Brain Sciences*, 22(1), 1–38 [discussion 38–75].
- Liegeois, F., Morgan, A., Connelly, A., & Vargha-Khadem, F. (2011). Endophenotypes of FOXP2: Dysfunction within the human articulatory network. *European Journal of Pediatric Neurology*, 15(4), 283–288.
- Marsh, R., Alexander, G. M., Packard, M. G., Zhu, H., Wingard, J. C., Quackenbush, G., & Peterson, B. S. (2004). Habit learning in Tourette syndrome, a translational neuroscience approach to a developmental psychopathology. *Archives of General Psychiatry*, 61, 1259–1268.
- Mavrogiorgou, P., Juckel, G., Frodl, T., Gallinat, J., Hauke, W., Zaudig, M., ... Hegerl, U. (2002). P300 subcomponents in obsessive-compulsive disorder. *Journal of Psychiatric Research*, 36(6), 399–406.
- McCairn, K. W., Bronfeld, M., Belevsky, K., & Bar-Gad, I. (2009). The neurophysiological correlates of motor tics following focal striatal disinhibition. *Brain*, 132(8), 2125–2138.
- Melby-Lervåg, M., Lyster, S. A., & Hulme, C. (2012). Phonological skills and their role in learning to read: A meta-analytic review. *Psychological Bulletin*, 138(2), 322–352. <http://dx.doi.org/10.1037/a0026744>.
- Mink, J. W. (2006). Neurobiology of basal ganglia and Tourette syndrome: Basal ganglia circuits and thalamocortical outputs. *Advances in Neurology*, 99, 89–98.
- Morault, P. M., Bourgeois, M., Laville, J., Bensch, C., & Paty, J. (1997). Psychophysiological and clinical value of event-related potentials in obsessive-compulsive disorder. *Biological Psychiatry*, 42(1), 46–56.
- Mostofsky, S., Lasker, A., Singer, H. S., Denckla, M., & Zee, D. (2001). Oculomotor abnormalities in boys with Tourette syndrome with and without ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry*, 40(12), 1464–1472.
- Mottron, L., Dawson, M., Soulie, I., & Burack, J. (2006). Enhanced perceptual functioning in autism: An update, and eight principles of autistic perception. *Journal of Autism and Developmental Disorders*, 36(1), 27–43.
- Mueller, S. C., Jackson, G. M., Ranu, D., Sophia, D., & Hollis, C. P. (2006). Enhanced cognitive control in young people with Tourette's syndrome. *Current Biology*, 16(6), 570–573.
- Müller-Vahl, K. R., Grosskreutz, J., Prell, T., Kaufmann, J., Bodammer, N., & Peschel, T. (2014). Tics are caused by alterations in prefrontal areas, thalamus and putamen, while changes in the cingulate gyrus reflect secondary compensatory mechanisms. *BMC Neuroscience*, 15, 6.
- Müller-Vahl, K. R., Kaufmann, J., Grosskreutz, J., Dengler, R., Emrich, H. M., & Peschel, T. (2009). Prefrontal and anterior cingulate cortex abnormalities in Tourette syndrome: Evidence from voxel-based morphometry and magnetization transfer imaging. *BMC Neuroscience*, 10(47).
- Munhall, K. G. (2001). Functional imaging during speech production. *Acta Psychologica Amst*, 107(1–3), 95–117.
- Munson, B., Kurtz, B., & Windsor, J. (2005). The influence of vocabulary size, phonotactic probability, and wordlikeness on nonword repetitions of children with and without specific language impairment. *Journal of Speech, Language, and Hearing Research*, 48(5), 1033–1047.
- Murphy, T., & Eddy, C. M. (2013). Neuropsychological assessment in Tourette syndrome. In D. Martino & J. F. Leckman (Eds.), *Tourette syndrome* (pp. 439–465). Oxford University Press.
- Nakeva Von Mentzer, C., Lyxell, B., Sahlén, B., Dahlström, Ö., Lindgren, M., Ors, M., ... Uhlén, I. (2015). Segmental and suprasegmental properties in nonword repetition – An explorative study of the associations with nonword decoding in children with normal hearing and children with bilateral cochlear implants. *Clinical Linguistics and Phonetics*, 29(3), 216–235.
- Nemeth, D., Kákács, Á., Chezán, J., Élrető, N., Kóbor, A., Tárnok, Z., ... Ullman, M. T. (2015). *Probabilistic sequence learning and consolidation in Tourette syndrome*. Paper presented at the UCL Institute of Neurology Computational Psychiatry Course London.
- Obeso, J. A., Rothwell, J. C., & Marsden, C. D. (1981). Simple tics in Gilles de la Tourette's syndrome are not prefaced by a normal premovement EEG potential. *Journal of Neurology, Neurosurgery and Psychiatry*, 44(8), 735–738.
- Oldfield, R. C. (1971). The assessment and analysis of handedness: The Edinburgh inventory. *Neuropsychologia*, 9(1), 97–113.
- Palminteri, S., Lebreton, M., Worbe, Y., Hartmann, A., Lehericy, S., Vidailhet, M., et al. (2011). Dopamine-dependent reinforcement of motor skill learning: Evidence from Gilles de la Tourette syndrome. *Brain*, 134, 2287–2301.
- Papoutsis, M., de Zwart, J. A., Jansma, J. M., Pickering, M. J., Bednar, J. A., & Horwitz, B. (2009). From phonemes to articulatory codes: An fMRI study of the role of Broca's area in speech production. *Cerebral Cortex*, 19(9), 2156–2165.
- Peeva, M. G., Guenther, F. H., Tourville, J. A., Nieto-Castanon, A., Anton, J. L., Nazarian, B., & Alario, F. X. (2010). Distinct representations of phonemes, syllables, and supra-syllabic sequences in the speech production network. *Neuroimage*, 50(2), 626–638.
- Pinker, S., & Ullman, M. T. (2002). The past and future of the past tense. *Trends in Cognitive Sciences*, 6(11), 456–463.
- Puts, N. A., Harris, A. D., Crocetti, D., Nettles, C., Singer, H. S., Tommerdahl, M., ... Mostofsky, S. H. (2015). Reduced GABAergic inhibition and abnormal sensory

- processing in children with Tourette syndrome. *Journal of Neuropsychology*, 114(2), 808–817.
- Ravizza, S. M., & Ciranni, M. A. (2002). Contributions of the pre-frontal cortex and basal ganglia to set shifting. *Journal of Cognitive Neuroscience*, 14, 472–483.
- Redmond, S. M., Thompson, H. L., & Goldstein, S. (2011). Psycholinguistic profiling differentiates specific language impairment from typical development and from attention-deficit/hyperactivity disorder. *Journal of Speech, Language, and Hearing Research*, 54(1), 99–117.
- Reich, W., Welner, Z., & Herjanic, B. (1997). *Diagnostic interview for children and adolescents-IV (DICA-IV)*. Toronto: Multi-Health Systems.
- Remington, A. M., Swettenham, J. G., & Lavie, N. (2012). Lightening the load: Perceptual load impairs visual detection in typical adults but not in autism. *Journal of Abnormal Psychology*, 121(2), 544.
- Sasisekaran, J., & Byrd, C. (2013). Nonword repetition and phoneme elision skills in school-age children who do and do not stutter. *International Journal of Language & Communication Disorders*, 48(6), 625–639.
- Schneps, M. H., Brockmole, J. R., Sonnert, G., & Pomplun, M. (2012). History of reading struggles linked to enhanced learning in low spatial frequency scenes. *PLoS One*, 7(4), e35724.
- Singer, H. S. (2013). Motor control, habits, complex motor stereotypies, and Tourette syndrome. *Annals of the New York Academy of Sciences*, 1304(1), 22–31.
- Smith, E. E., & Jonides, J. (1998). Neuroimaging analyses of human working memory. *Proceedings of the National Academy of Sciences of the United States of America*, 95(20), 12061–12068.
- Stern, E., Silbersweig, D. A., Chee, K. Y., Holmes, A., Robertson, M. M., Trimble, M., ... Dolan, R. J. (2000). A functional neuroanatomy of tics in Tourette syndrome. *Archives of General Psychiatry*, 57(8), 741–748.
- Strand, F., Forssberg, H., Klingberg, T., & Norrelgen, F. (2008). Phonological working memory with auditory presentation of pseudo-words—An event related fMRI Study. *Brain Research*, 1212, 48–54.
- Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). Kaufman brief intelligence test (K-BIT). In *A compendium of neuropsychological tests: Administration, norms, and commentary*, pp. 164–168. Oxford: Oxford University Press.
- Swinnen, S. P., Vangheluwe, S., Wagemans, J., Coxon, J. P., Goble, D. J., Van Impe, A., ... Wenderoth, N. (2010). Shared neural resources between left and right interlimb coordination skills: The neural substrate of abstract motor representations. *Neuroimage*, 49(3), 2570–2580.
- Tager-Flusberg, H., & Joseph, R. M. (2003). Identifying neurocognitive phenotypes in autism. *Philosophical Transactions of the Royal Society of London. Series B, Biological sciences*, 358(1430), 303–314.
- Teichmann, M., Rosso, C., Martini, J. B., Bloch, I., Brugieres, P., Duffau, H., ... Bachoud-Levi, A. C. (2015). A cortical-subcortical syntax pathway linking Broca's area and the striatum. *Human Brain Mapping*, 36(6), 2270–2283.
- Thibault, G., Felezou, M., O'Connor, K. P., Todorov, C., Stip, E., & Lavoie, M. E. (2008). Influence of comorbid obsessive-compulsive symptoms on brain event-related potentials in Gilles de la Tourette syndrome. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 32(3), 803–815.
- Tourette Syndrome Study Group (1993). Definitions and classification of tic disorders. *Archives of Neurology*, 50, 1013–1016.
- Tremblay, P., Deschamps, I., Baroni, M., & Hasson, U. (2016). Neural sensitivity to syllable frequency and mutual information in speech perception and production. *Neuroimage*, 16.
- Tremblay, L., Worbe, Y., Thobois, S., Sgambato-Faure, V., & Feger, J. (2015). Selective dysfunction of basal ganglia subterritories: From movement to behavioural disorders. *Movement Disorders*, 30(9), 1155–1170.
- Ullman, M. T. (2004). Contributions of memory circuits to language: The declarative/procedural model. *Cognition*, 92(1–2), 231–270.
- Ullman, M. T., Corkin, S., Coppola, M., Hickok, G., Growdon, J. H., Koroshetz, W. J., & Pinker, S. (1997). A neural dissociation within language: Evidence that the mental dictionary is part of declarative memory, and that grammatical rules are processed by the procedural system. *Journal of Cognitive Neuroscience*, 9(2), 266–276.
- Ullman, M. T. (2007). The biocognition of the mental lexicon. In M. G. Gaskell (Ed.), *The Oxford handbook of psycholinguistics* (pp. 267–286). Oxford, UK: Oxford University Press.
- Ullman, M. T. (2016). The declarative/procedural model: A neurobiological model of language learning, knowledge and use. In G. Hickok & S. A. Small (Eds.), *The neurobiology of language* (pp. 953–968). San Diego: Elsevier.
- Ullman, M. T., & Pierpont, E. I. (2005). Specific language impairment is not specific to language: The procedural deficit hypothesis. *Cortex*, 41(3), 399–433.
- Vicario, C. M., Martino, D., Spata, F., Defasio, G., Glacche, R., Martino, V., ... Cardona, F. (2010). Time processing in children with Tourette's syndrome. *Brain and Cognition*, 73(1), 28–34.
- Vitevitch, M. S., Luce, P. A., Charles-Luce, J., & Kemmerer, D. (1997). Phonotactics and syllable stress: Implications for the processing of spoken nonsense words. *Language and Speech*, 40(1), 47–62.
- Walenski, M., Prado, E. L., Ozawa, K., Steinhauer, K., & Ullman, M. T. (2016). The compositionality and storage of inflected forms: Evidence from working memory effects. *Cognition*. [under revision]
- Walenski, M., Mostofsky, S. H., Gidley-Larson, J. C., & Ullman, M. T. (2008). Brief report: Enhanced picture naming in autism. *Journal of Autism and Developmental Disorders*, 38(7), 1395–1399.
- Walenski, M., Mostofsky, S. H., & Ullman, M. T. (2007). Speeded processing of grammar and tool knowledge in Tourette's syndrome. *Neuropsychologia*, 45(11), 2447–2460.
- Walenski, M., Mostofsky, S. H., & Ullman, M. T. (2014). Inflectional morphology in high-functioning autism: Evidence for speeded grammatical processing. *Research in Autism Spectrum Disorders*, 8(11), 1607–1621. <http://dx.doi.org/10.1016/j.rasd.2014.08.009>.
- Wang, Z., Maia, T. V., Marsh, R., Colibazzi, T., Gerber, A., & Peterson, B. S. (2011). The neural circuits that generate tics in Tourette's syndrome. *American Journal of Psychiatry*, 168(12), 1326–1337. <http://dx.doi.org/10.1176/appi.ajp.2011.09111692>.
- Wechsler, D. L. (1991). *Wechsler intelligence scale for children* (3rd ed.). San Antonio, TX: The Psychological Corporation.
- Worbe, Y., Malherbe, C., Hartmann, A., Pelegrini-Issac, M., Messe, A., Vidailhet, M., ... Benali, H. (2012). Functional immaturity of cortico-basal ganglia networks in Gilles de la Tourette syndrome. *Brain*, 135(6), 1937–1946.
- Worbe, Y., Marrakchi-Kacem, L., Lecomte, S., Valabregue, R., Poupon, F., Guevara, P., ... Poupon, C. (2015). Altered structural connectivity of cortico-striato-pallido-thalamic networks in Gilles de la Tourette syndrome. *Brain*, 138(2), 472–482.
- Zieman, U., Paulus, W., & Rothenberger, A. (1997). Decreased motor inhibition in Tourette's disorder: Evidence from transcranial magnetic stimulation. *American Journal of Psychiatry*, 154(9), 1277–1284.