Relying on brain rhythms for improving the diagnosis of dyslexia

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Abstract

Background. Developmental dyslexia is a pervasive child condition whose characteristic phenotypic profile precludes an early diagnosis. Different early markers of the risk for dyslexia have been posited over time, but neither of them seems totally reliable. *Aims and main contribution.* In this paper we argue for a diagnostic test based on the abnormal oscillatory signature of the dyslexic brain which builds on recent, robust oscillation-to-phonology links.

Keywords:

Dyslexia, early diagnosis, frequency bands, oscillatory phase-locking, phonology

Introduction

Developmental dyslexia, a neurobiological condition characterized by reading problems and reduced phonological awareness, is the most common learning disability in children, with a prevalence of around 10% (Darki, Peyrard-Janvid, Matsson, Kere, & Klingberg, 2012; Shaywitz, 1998). Dyslexic children typically show a reduced ability for attending to, judging, and manipulating, speech sounds, which ultimately impairs phoneme-to-grapheme and grapheme-to-phoneme correspondences (Cohen, Jobert, Le Bihan, & Dehaene, 2004; Martin, Kronbichler, & Richlan, 2016; Wagner & Torgesen, 1987). Nonetheless, some of them can also exhibit broader deficits, like reduced working memory and attentional problems (Hari & Renvall, 2001; Laasonen et al., 2012; Vidyasagar & Pammer, 2010), or broad sensory and motor deficits (Facoetti, Lorusso, Paganoni, Umiltà, & Mascetti, 2003; Nicolson, Fawcett, & Dean, 2001; Stoodley, Harrison, & Stein, 2006; see Goswami, 2014 for a review).

As expected, dyslexia is usually diagnosed once the child starts to read. However, the diagnosis is often hindered by its comorbidity with other cognitive conditions which result from similar underlying deficits and/or entail deficits that exacerbate the dyslexic phenotype. Among these comorbid conditions, one finds mathematic disorder (Willcutt et al., 2013), attention deficit/hyperactivity disorder (ADHD) (Sexton, Gelhorn, Bell, & Classi, 2012), developmental coordination disorder (Kaplan, Wilson, Dewey, & Crawford, 1998), and types of language disorders entailing an abnormal phonological processing, like speech-sound disorder (SSD) (Pennington & Bishop, 2009) and specific language impairment (SLI) (Bishop & Snowling, 2004). Since an early diagnosis of developmental cognitive disorders usually results in better outcomes at later ages, particularly because it allows intervention aimed to prevent rather than remediate reading difficulties, there is a great interest for

identifying early markers of dyslexia. Predictors of developmental dyslexia in preliterate children include i) behavioural and language measures, such as language delay, problems with naming, difficulties for recognizing and manipulating rhymes, and/or attention/comprehension problems (Flax, Realpe-Bonilla, Roesler, Choudhury, & Benasich, 2009; Puolakanaho et al., 2008; Vanvooren, Poelmans, De Vos, Ghesquière, & Wouters, 2017); ii) neurobiological signatures, such as changes in brain connectivity, alterations of grey matter volumes, or abnormal patterns of brain activity (Noordenbos, Segers, Wagensveld, & Verhoeven, 2013; Raschle, Chang, & Gaab, 2011; Skeide et al., 2015; van Zuijen, Plakas, Maassen, Maurits, & van der Leij, 2013); and iii) genetic data, like mutations in candidate genes for the condition, many of which typically contribute to the development and wiring of brain areas involved in reading (Marino et al., 2012; Paracchini et al., 2011, Paracchini, Diaz, & Stein, 2016). Nonetheless, neither of these measures and signatures is expected to be specific to dyslexia and they might well fail to predict the onset of the disease (see Lyytinen, Erskine, Hämäläinen, Torppa, & Ronimus, 2015; Ozernov-Palchik & Gaab, 2016; Thompson et al., 2015 for a discussion).

The quest for an early diagnosis of dyslexia

Let us examine this problem in some more detail. At the surface, although wrong phoneme-to-grapheme correspondences are a hallmark of the disease, no unique, disorder-specific cognitive deficit has been identified to account for the attested symptoms. The phonological deficit theory is the most widely accepted view of the origins of dyslexia. Under this view, dyslexia results from an impaired access to phoneme representations from auditory cues (Boets et al., 2013; Ramus, 2014), and/or from the impairment of specific higher-order language brain networks (Molinaro, Lizarazu, Lallier, Bourguignon, & Carreiras, 2016).

Nonetheless, phonological problems have been claimed to account as well for other comorbid conditions, like SSD (Shriberg, Tomblin, & McSweeney, 1999) or SLI (Bishop, 2002). Moreover, one should expect that problems with reading at different ages result from different cognitive deficits. As stressed by Karmiloff-Smith (2008, p. 693), “to understand any developmental syndrome, it is essential to distinguish between the behavioral phenotype (based on scores from standardized tests of overt behavior) and the cognitive phenotype (based on in-depth analyses of the mental processes underlying the overt behavior)”. Also, it is worth considering that speech processing in preliterate children may differ from speech processing in their literate peers (Havron & Arnon, 2017), as also suggested by the differences in brain activation patterns observed between illiterate and literate adults during phonological processing (Castro-Caldas, Petersson, Reis, Stone-Elander, & Ingvar, 1998). Eventually, these circumstances may preclude the finding of useful early markers of the disease. For instance, impaired non-speech auditory processing at a pre-reading age is regarded a risk-factor for dyslexia but it is not a predictor (Plakas, van Zuijen, van Leeuwen, Thomson, & van der Leij, 2013).

Regarding their brains, dyslexic people display structural and functional differences respect to neurotypical subjects in areas important for reading, including the auditory-phonological, the visuo-magnocellular, and the motor/cerebellar subsystems (see Jiménez-Bravo, Marrero, & Benítez-Burraco, 2017 for a review). Nonetheless, most, if not all, of these brain anomalies are not dyslexia-specific either, because they can be found in other clinical conditions. For instance, dyslexics are reported to exhibit an increase of grey matter density in the right perisylvian cortex (Barrick et al., 2005). However, this feature has been documented as well in Williams syndrome (Thompson, 2005). Likewise, a robust neurobiological marker of dyslexia is said to be the underactivation of the temporoparietal

and occipitotemporal regions in the left hemisphere during reading tasks (together with a compensatory overactivation of the same regions in the right hemisphere) (Brunswick, McCrory, Price, Frith, & Frith, 1999; Paulesu, Danelli, & Berlinger, 2014; Pugh et al., 2001). However, the ventral portion of the occipito-temporal region is also found impaired in people with prosopagnosia, hence its alleged role in the recognition of faces too (Dricot, Sorger, Schiltz, Goebel, & Rossion, 2008; Sorger, Goebel, Schiltz, & Rossion, 2007). Overall, although neuroimaging facilities have enhanced our understanding of the dyslexic brain, in terms of its anatomical distinctive features and the way in which it processes sounds and visual cues during reading tasks, most of the purported brain differences between dyslexic and normal reading individuals are difficult to replicate, particularly, because they result from small-scale studies; in fact, meta-analyses and large-scale studies have found that the only robust structural difference between groups is a smaller brain volume in dyslexics (Ramus, Altarelli, Jednoróg, Zhao, & Scotto di Covella, 2017).

At the bottom, genetic linkage and association studies have identified several loci related to dyslexia, as well as additional loci that confer susceptibility to the disease. Mutations in classic candidate genes for this condition (*DYX1C1*, *DCDC2*, *ROBO1*, and *KIA0139*) are hypothesised to result in altered neuronal migration and/or abnormal neuronal interconnection patterns in brain areas related to dyslexia, ultimately accounting for the processing deficits typically observed in this condition (see Benítez-Burraco, 2010 for a review). For instance, in rats, RNA interference of the dyslexia candidate *Dyx1c1* impairs the normal migration of the neurons and gives rise to ectopias that resemble the structural anomalies observed in the brain of dyslexics (Rosen et al., 2007). It also affects the animals' ability to process complex auditory stimuli (Threlkeld et al., 2007). Nonetheless, neither of these genes is specific to dyslexia. For example, *ROBO1* has been also associated to retinal

angiogenesis and lymphangiogenesis (Huang et al., 2009, Yang et al., 2010). Next generation sequencing facilities have significantly increased the number of candidate genes for dyslexia (see Paracchini et al., 2016 for the most recent review). Although many of them cluster into key pathways that drive the disease, many others without an obvious connection to dyslexia are expected to increase the risk of suffering this condition because of the high degree of interconnection between gene regulatory networks (Boyle, Li, & Pritchard, 2017). In truth, the involved pathways are not dyslexia-specific either. KEGG and Panther analyses of published candidates for dyslexia and SLI via Enrichr (<http://amp.pharm.mssm.edu/Enrichr/>) reveal that although these two conditions are enriched in different pathways, some shared pathways still exists, like the Rap1 signalling pathway (KEEG) and the ionotropic glutamate receptor pathway (Panther), among the 5 top-scoring pathways. Likewise, under the “omnigenic” view of complex disorders (Boyle et al. 2017), whole-brain transcriptomic profiles should help distinguish among disorders with a complex etiology, identifying shared and distinct biological processes across disorders. However, as noted by Parikshak and colleagues (2015), molecular profiling and network approaches to complex cognitive diseases like dyslexia face major challenges, including the complexity of molecular phenotypes (i.e. multiple cell types are involved and the brain exhibits a noteworthy spatial and temporal heterogeneity), together with our scarce knowledge of the best intermediate phenotypes to measure. Additionally, it is difficult to infer causality in molecular networks with a systems genetics approach (Chen et al., 2014; Civelek & Lusis, 2013).

According to Port and colleagues (2015, p. 26), “for a biomarker to be effective, it must be sensitive and specific to the disorder/dysfunctional domain, scale with severity, be simple to implement, and demonstrate robustness.”. At present, our understanding of the etiopathogenesis of dyslexia hinders promoting any of the biological hallmarks of dyslexia to

effective biomarkers of the disease, particularly when pursuing for an early diagnosis. For achieving this, we first need to bridge the gap that still exists between the gene variants found in dyslexics, the abnormal development of their brains, the altered brain functions observed in selected brain areas, the cognitive deficits these people exhibit, and the problems they experience for reading and processing phonemes. As also pointed by Parikshak and colleagues (2015, p. 441), “understanding the role of specific genetic variants in the brain involves dissecting a functional hierarchy that encompasses molecular pathways, diverse cell types, neural circuits and, ultimately, cognition and behaviour”. We believe that this is possible only if a systems biology approach to dyslexia is adopted, aimed to characterise, from a holistic perspective, the complex interactions among myriads of biological components that take place within the dyslexic brain, as well as the emergent properties resulting from such interactions. We expect that this approach also enables to identify effective biomarkers of the disease. In our opinion, the atypical neuronal oscillations found in dyslexics might be the most promising biomarker of dyslexia at our current state of knowledge, because they allow for establishing robust, causal links between the genes and phonological/reading disabilities, particularly, in response to linguistic stimuli, as we show below.

Why brain rhythms

Underlying all behavioural and cognitive processes we find arrays of brain rhythms resulting from the oscillatory activity of neurons over a wide range of spatial and temporal scales (see Buzsáki, 2006 for a review). Neuronal oscillations are rhythmic modulations of local excitability through which neuronal populations phase-synchronize their spike output as well as their sensitivity to inputs (Thut, Miniussi, & Gross, 2012). This phase synchronization

results in a hierarchical cross-frequency coupling of different frequency bands which allows information to be processed accurately and flexibly at multiple temporal scales. Importantly, not only stimulus processing are mediated by neuronal oscillations, but also internal aspects of brain function, like interactions across brain areas, memory formation, or attentional selection (Fries, Fernández, & Jensen, 2003; Canolty et al., 2006; Womelsdorf et al., 2007; Lakatos, Karmos, Mehta, Ulbert, & Schroeder, 2008).

Specifically, language functions are hypothesised to result from complex interactions between close and distant brain areas in which the coupling of brain oscillations at different frequency bands plays a key role, ultimately explaining, and not just mapping, how language emerge from neural activity. In a nutshell, computational and representational properties of language can be decomposed into generic processes that interact in dynamic ways and can be implemented via neural oscillations in terms of a specific multiplexing algorithm (see Murphy, 2015a, 2015b, 2016a for details). Noteworthy examples are the entrainment of left-cortical delta to hierarchical linguistic structures from syllables to sentences (Ding, Melloni, Zhang, Tian, & Poeppel, 2016), the coupling of delta phase-amplitude with both theta and beta involved in phrasal construction (Murphy, 2016b), or the linking of reduced parieto-occipital alpha to increased semantic predictability (Wöstmann, Herrmann, Wilsch, & Obleser, 2015).

As with other aspects of language processing, neuronal oscillations are responsible for segmenting the continuous sound stream and parsing the temporal components of speech at different levels (Giraud & Poeppel, 2012). Abundant evidence suggests that neural oscillations have a strong causal-explanatory power with respect to abnormal speech processing, and particularly, to the deficits exhibited by dyslexic people, in contrast with the lack of specificity of the anatomical and cognitive findings (see Jiménez-Bravo et al. 2017 for

details). In brief, dyslexic children show abnormal oscillation patterns in the auditory cortex, including the theta, delta, and alpha bands, which hinder the correct parsing of the slow-rate dynamic features of speech. Besides, abnormal cortical oscillations in the gamma range result in undersampled or oversampled phonemic representations of speech, which negatively impact on grapheme-to-phoneme matching.

Interestingly, language-specific and script-specific differences among dyslexics are better understood under this oscillatory view. For instance, differences in low frequency temporal features, like the theta band at 4 Hz, seem to exist between dyslexic children using syllable-timed rhythmic languages like Spanish and children using stress-timed languages like English. Accordingly, Spanish children with dyslexia show stronger synchronization of theta at 4 Hz in the auditory cortex (Lizarazu et al., 2015) compared to English dyslexics (Lehongre, Morillon, Giraud, & Ramus, 2013). This suggests that dyslexics using syllable-timed languages rely to a greater extent on phonemic information to compensate for an impaired right specialization of the gamma band.

Brain rhythms as biomarkers of dyslexia

Because oscillopathic approaches on dyslexia draw more coherent pictures of the brain dysfunctions that are characteristic of this condition and of the symptoms typically found in dyslexics, we believe that they should help find better and earlier biomarkers of the disease. Table 1 summarises current findings of abnormal brain responses at different frequency bands to linguistics and non-linguistic stimuli by dyslexic children. We regard the delta and gamma bands, as measured in the auditory cortex, as the most confident markers for evaluating the risk for dyslexia in preliterate children. Accordingly, we expect an abnormal response of the delta band, which is involved in tracking the syllabic features of speech, in

the right auditory cortex. Likewise, we expect an abnormal response of the gamma band, which is responsible for encoding the phonemic information, either bilaterally or in the right auditory cortex.

Frequency Band	Location	Stimuli	Reported Anomalies	Analytic Tool	Dyslexic Subjects	References
Delta	Right auditory cortex	Rhythmic speech using syllable [ba] repeated at 2 Hz	Decreased phase-synchronization	EEG/MEG	N = 11 Ages = 13–14	Power et al., 2013
Delta	Right auditory cortex and left inferior frontal gyrus	Short spoken sentences in Spanish	Decreased phase-synchronization	MEG	N = 10 Ages = 9–11	Molinaro et al., 2016
Delta	Right supramarginal gyrus	Amplitude-modulated white noise at 2 Hz	Increased hemodynamic response	fNIRS	N = 18 Ages = 11–14	Cutini et al., 2016
Theta	Both auditory cortices	Amplitude-modulated white noise at 4 Hz	Absence of rightward lateralization and stronger bilateral phase-synchronization	EEG/MEG	N = 20 Ages = 8–14	Lizarazu et al., 2015
Gamma	Right auditory cortex	Amplitude-modulated white noise at 80 Hz	Smaller response amplitudes	EEG/MEG	N = 34 Age = 5 (at risk for	Vanvooren et al., 2014

					dyslexia)	
Gamma	Right auditory cortex	Amplitude-modulated white noise at 30 Hz	Increased phase-synchronization	EEG/MEG	N = 20 Ages = 8–14	Lizarazu et al., 2015
Theta	Both auditory cortices	Amplitude-modulated white noise at 4 Hz	Reduced activity in non-synchronized surrounding frequencies 3-5 Hz			
Beta	Both auditory cortices	Amplitude-modulated white noise at 20 Hz	Increased phase-synchronization and larger response amplitudes	EEG	N = 32 Ages 14–15	De Vos et al., 2017a
Alpha	Both auditory cortices	Amplitude-modulated white noise at 10 Hz	Decreased phase-synchronization and smaller response amplitudes			
Alpha	Temporal, parietal, and occipital sites	Resting state brain activity	Decreased response amplitude	EEG	N = 26 Age = 11	Babiloni et al., 2012

Table 1. Potential oscillatory biomarkers for an early detection of dyslexia in preliterate children. The most confident biomarkers are shadowed in grey.

A test for detecting dyslexia in preliterate children based on brain oscillations

As noted in Table 1, atypical oscillations in dyslexic children have been mostly observed in response to non-linguistic stimuli. Nonetheless, because most of the problems

that dyslexic children experience are linguistic in nature, we believe that a good diagnostic test based on brain oscillations aimed to detect dyslexia at an early age should include linguistic stimuli, and mostly consist of linguistic tasks as closer as possible to the kind of stimuli and tasks that dyslexic children will have to struggle with during reading. Because we already have quite robust bridging hypotheses linking abnormal brain oscillations and reading problems in dyslexics (see Jiménez-Bravo et al., 2017 for a proposal), such a test is not an impossibility (see Table 2).

In our opinion, three tasks would be particularly informative. First, minimal-pair processing. Children would be asked to listen to pairs of words and pseudowords that differentiate in only one sound in the same position, like *goat* vs. *coat* (which is a minimal pair in English), and *soat* vs. *toat* (which is not a minimal pair in English, just two strings of sounds without meaning resembling real words and contrasting in their initial sounds). Because fast acoustic fluctuations underlying minimal pairs are typically parsed by high oscillatory frequencies bilaterally or with a left bias in the auditory cortex, we expect to find an abnormal response of the gamma band within this area when performing this task. It is true that the temporal sampling framework (Goswami, 2011) predicts that the gamma band is not impaired in children with dyslexia before they learn to read. It is also true that gamma phase-synchronization has been reported to be normal before reading onset (De Vos et al., 2017b). However, preliterate children at risk for dyslexia have been found to exhibit an atypical lateralization for this oscillatory frequency band (Vanvooren et al., 2014). Phoneme discrimination has proven to be a confident criterion for differentiating poor and normal reading abilities (Hämäläinen et al., 2009, Brunner, Bäumer, Rosenauer, Scheller, & Plinkert, 2010; Groth, Lachmann, Riecker, Muthmann, & Steinbrink, 2011; Steinbrink, Klatter, & Lachmann, 2014), and in fact, this is an ability that emerges very early during language

acquisition (Kuhl, 1979; Shahidullah & Hepper, 1994; Cheour-Luhtanen et al., 1995).

Second, processing of consonant-vowel transitions. Children would be asked to listen to trios of simple open syllables, like *ba-da-ga*, in which a voiced consonant is followed by a vowel, thus involving quick, specific changes in sound frequency (*formant transitions* in phonetics) and to which the auditory nerve fibres respond strongly (Delgutte & Kiang, 1984). Poor codification of consonant-vowel transitions has been reported for 4-year-old dyslexic children in the inferior colliculus (IC), which is the primary auditory nucleus of the midbrain (White-Schwoch & Kraus, 2013). Most neurons in the IC phase lock to speech stimuli in the range of 100-1000 Hz (Liu, 2006). Therefore, because subcortical timing is time-linked to the auditory cortex during speech processing, and because cortical asymmetry to speech sounds is predicted by the temporal encoding of speech in auditory brainstem (Abrams, Nicol, Zecker, & Kraus, 2006; Wible, Nicol, & Kraus, 2004), we expect to find an atypical gamma lateralization in the auditory cortex of preliterate children at risk for dyslexia, together with poor codification in the auditory brainstem.

Third, rhythmic entrainment to the envelope of a speech stretch. Children would be asked to listen to a set of familiar nursery rhymes whose amplitude envelope will be controlled to maximize the contrasts in stress and syllabic patterns. Phonological problems in dyslexia have been claimed to result in part from impaired neural oscillatory phase-locking to slow amplitude modulation patterns in the speech envelope, which precludes capturing the full spectro-temporal complexity of speech over multiple timescales (Leong, & Goswami, 2014). Because this component of speech is typically processed at low frequencies in the right auditory cortex and is thought to result from poorer or impaired oscillatory synchronization between theta oscillations (syllable rate) and delta (stress rate) oscillations, we expect to find reduced synchronization in the delta range within this area in children at

risk for dyslexia. We also anticipate to observe atypical synchronization in the theta band in these children, which is expected to be stronger in children speaking syllable-timed languages as Spanish, as previously observed by Lizarazu et al. (2015), because stress-timed languages like English typically contain more complex and varied syllabic onsets and codas (Dauer, 1983; Auer, 1991), as well as shorter unstressed vowels (Ramus, Nespor & Mehler, 1999).

Ultimately, we expect that the reliability of our test will improve if the entire range of frequencies is examined, instead of relying on frequency bands as defined in previous literature, (and) particularly if time-scales (corresponding to utterances, phonic groups, stress patterns, syllables, etc.) are extracted from statistical regularities in the speech material. As recent research shows, this approach offers more reliable data and enables to overcome the high inter-subject variability usually found in studies using pre-defined bands (Keitel, Gross,, & Kayser, 2017; Segaert, Mazaheri, & Hagoort, 2017). Likewise, we envisage to perform a wavelet analysis of the relevant EEG activity aimed to established whether the oscillatory activity across the spectrum of frequencies increases or decreases relative to a certain event, and ultimately, to uncover the strictly phase-locked (or evoked) activity to the event of interest (see Grimaldi 2017 for discussion).

Task	Nature	Example of stimuli	Frequency band	Location	Expected finding in children at risk
Minimal pairs/pseudo-minimal pairs	Perception	goat / coat	Gamma	Left and right auditory cortices	Altered lateralization pattern
		soat/toat			
Consonant-vowel transition	Perception	[ba]-[da]- [ga]	Gamma	Inferior colliculus and left and right	Reduced synchronization and

				auditory cortices	altered lateralization
		I'm Yertle the Turtle! /			
		Oh, marvelous me! /	Delta and	Right auditory	Reduced
Speech rhythm	Perception	For I am the ruler / of	theta	cortex	synchronization
		all that I see!			

Table 2. Draft of a detection test of dyslexia in preliterate children based on brain oscillations.

As a final note, we wish to highlight that, as stressed by Anderson (2016), a genetic network underlies any neural network, whereas a chemical gradient overlies it. In our past research we have shown that the relationships between genes, oscillations, and linguistic computations are particularly robust and specific to language (Murphy & Benítez-Burraco, 2017) and that this is also true for cognitive disorders involving language deficits, like autism-spectrum disorder (Benítez-Burraco & Murphy, 2016), schizophrenia (Murphy & Benítez-Burraco, 2016), and certainly, dyslexia (Jimenez-Bravo et al., 2017). In our opinion, this circumstance gives additional support to our claim that relying on brain oscillations enables to implement diagnostic tools that are more accurate than those using other potential biomarkers of the disease.

Conclusions

In conclusion, it seems feasible (and desirable) to build on current research on the oscillopathic profile of young dyslexics to construct robust oscillatory biomarkers of dyslexia, and ultimately, to design diagnostic tests than can be applied to preliterate children

at risk for dyslexia in order to achieve an earlier and more confident diagnosis of this condition.

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