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A BIOLINGUISTIC APPROACH TO LANGUAGE DISORDERS: TOWARDS A PARADIGM SHIFT IN CLINICAL LINGUISTICS

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ABSTRACT

Current clinical typologies may be not the best tool for properly categorising, describing, and explaining the origin and nature of language disorders. This shortcoming may reduce the effectiveness of the therapeutic approaches aimed to ameliorate the impact of these conditions. Here we claim for a biolinguistic approach to this problem. Biolinguistics aims to gain a confident knowledge of the biological underpinnings of human language. We will argue that clinical linguistics will benefit from a shift of focus in the line of the ongoing evo-devo turn in biolinguistics: instead of relying in the analysis of the phenotype in the adult state, more attention should be paid to developmental processes. Moreover, an approach to language disorders which heavily relies on concepts like canalization, developmental plasticity, robustness, evolvability or adaptive landscapes will surely help clinical linguists in clarifying, understanding, and explaining what they observe in their practice. We also expect that this fresh approach allows us to identify better endophenotypes of the disorders that can be used as confident hallmarks for an earlier and more accurate diagnosis.

Keywords: biolinguistics, clinical linguistics, development, evo-devo, language disorders

1. CLINICAL LINGUISTICS: A MESSY SCENARIO

On paper clinical categories like dyslexia or specific language impairment (SLI) refer to cognitive disorders in which only language becomes impaired and that can be distinguished from other similar categories at all levels of analysis (phenotypic, cognitive, neurobiological, genetic, etc.). For example, people suffering from dyslexia have difficulties to read texts and to spell words (Lyon *et al.* 2003). These problems are thought to be caused by the dysfunction of the phonological component of the working memory (Shaywitz *et al.* 1998). In turn, the brain of dyslexics show anomalies that are both structural (Galaburda *et al.* 1985, Deutsch *et al.* 2005) and functional (Shaywitz *et al.* 1998, Maisog *et al.* 2008) and which concern to many of the brain areas involved in reading and spelling in the non-affected population (see Démonet *et al.* 2004 for review). Finally, most of the several candidate genes for dyslexia identified to date regulate axonal growth and neuronal migration in the cortex, plausibly accounting for the structural and functional anomalies attested in the brains of dyslexics (see Benítez-Burraco 2010 for review).

Nonetheless, for clinical linguists things are usually less clear cut and more difficult to handle. To begin with, patients commonly show symptoms that are compatible with more than one disorder (linguistic or not linguistic by nature), to the extent that

comorbidity is a frequent outcome of clinical practice. Using again dyslexia as an example, reading difficulties are observed in many cognitive disorders. Actually, dyslexia is frequently comorbid with other language disorders, including SLI (Smith *et al.* 1996; Catts *et al.* 2005) and speech-sound disorder (SSD) (Smith *et al.* 1996; Shriberg *et al.* 1999; Stein *et al.* 2004), but also with attention deficit hyperactivity disorder (ADHD) (Purvis and Tannock 1997; Shaywitz 1998). Secondly, people affected by one disorder generally display linguistic abilities (and are endowed with a linguistic competence) that are quite variable. In order to apprehend this variability different subtypes of the same disorder need to be posited, in which one (among several) specific aspect(s) of language becomes more impaired. However, variation is also observed throughout development, to the extent that affected children can switch from one subtype to another of the same disorder as they grow (Botting and Conti-Ramsdem 2004). As Karmiloff-Smith and Mills point out (2006: 585) “one cannot simply assume that deficits in the phenotypic outcome are the same as those apparent in the infant start state” (Karmiloff-Smith and Mills 2006: 585). Importantly, deficits in performance may arise from cognitive deficits in a non-direct fashion. This circumstance substantially increases the observed variation at the symptomatic/clinical level. Obviously, it substantially complicates the categorization of disorders. As Karmiloff-Smith puts it (2008: 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)”. In fact, “sometimes equivalent behavioral scores camouflage very different cognitive processes” (Donnai and Karmiloff-Smith 2000: 167).

On the whole, it seems that different disorders (or different subtypes of the same disorder) may result from the same (broad) cognitive deficit, which can manifest differentially in different populations and/or environmental conditions (hence the alleged heterogeneity and/or comorbidity). At the same time, different deficits (that may be or may be not specifically linguistic) can contribute to the same disorder, this implying that clinical categories may be well construed as conglomerates of several cognitive deficits, yet characterised by substantially similar symptomatic profiles. Moreover, the diverse subtypes of a particular disorder may represent conditions in which one of such deficits prevails. Ultimately, in other different population and/or environment any of these underlying deficits can manifest as a different disorder (hence the purported heterogeneity and/or comorbidity). For instance, the dysfunction of the phonological component of working memory gives rise not just to dyslexia, but also to SLI (Bishop 2002) and SSD (Shriberg *et al.* 1999). Conversely, several other deficits have claimed to contribute to dyslexia, including problems with categorical perception (Serniclaes *et al.* 2004), difficulties for correctly processing (and discriminating between) brief acoustic impulses (Temple *et al.* 2000), cerebellar dysfunctions (Nicolson and Fawcett 2006), problems with visual processing (Lovegrove *et al.* 1980), or a dysfunction of the magnocellular pathway (Livingstone *et al.* 1991; Stein and Walsh 1997).

Finally, it is frequently observed that problems with language in the affected people concern to quite broad aspects of language, to the extent that the attested deficits do not normally match the units, levels, or operations that underlie linguistic theory (Newmeyer 1997). As a consequence, clinical typologies are not always acceptable under a linguistic lens. For instance, some speech therapists claim that three basic subtypes of SLI do exist: phonological, expressive and expressive-receptive (e.g. Rapin and Allen 1983; American Psychiatric Association 1994). Similarly, a syntactic-pragmatic subtype is also included in some classifications (Rapin and Allen 1983). However, these are separate levels in most usual accounts of language.

Comorbidity, heterogeneity, and variability are observed at the neurobiological level too. Hence, the affected regions (structurally or functionally) in one disorder may well be impaired in people suffering from other different condition. Consider, for instance, the ventral portion of the occipito-temporal region. This area contains one of the two processing subsystems needed for reading that are located in the posterior region of the left hemisphere. Not surprisingly, this area is underactive in dyslexics during reading tasks (Horwitz *et al.* 1998; Shaywitz *et al.* 1998; Paulesu *et al.* 2001). However, this area seems to be involved as well in the recognition of faces and it has been linked to prosopagnosia too (Sorger *et al.* 2007; Dricot *et al.* 2008). At the same time, similar (abnormal) neurobiological profiles can be observed in different clinical conditions. For instance, an increase of the gray-matter density in the perisylvian cortex has been documented in ADHD, Williams syndrome, and fetal alcohol syndrome (Toga *et al.* 2006). All these conditions have diverse aetiologies and different neurocognitive profiles, but all of them encompass language deficits (Mervis and Becerra 2007; Rapport *et al.* 2008; Wyper and Rasmussen 2011) and may be comorbid (O'Malley and Nanson 2002; Rhodes *et al.* 2011). Overall, it is not clear whether the involved regions are multifunctional by nature or perform instead some basic process that is recruited for language and for other cognitive abilities. Moreover, it is frequently observed that affected regions may give rise to mixed symptoms. Lastly, it commonly occurs that their boundaries are located differently in different individuals.

Finally, things are not easier to interpret at the molecular level. Different candidate genes and risk factors for different language disorders have been identified to date. However, as we have seen in the case of dyslexia, it is not one but many genes that usually contribute to each disorder (*polymorphism*). Typically, several pathogenic variants of each candidate gene have been identified. At the same time, other polymorphisms may contribute to the language abilities of the non-affected population. Importantly, the same mutation in the same gene may cause the disorder in some individuals, but not in others (*variable penetrance*). Conversely, pathogenic variants of a gene may be well absent in people affected by the disorder (*phenocopy*). Moreover, the same mutation can give rise to different disorders in different populations, to the extent that candidate genes for a particular disorder may be found mutated in other conditions (*pleiotropy*). Ultimately, mutations in genes encoding proteins that are functionally related to one particular candidate (i.e. they belong to the same interactome) may give rise to different disorders in different subjects or environments. *FOXP2* (the famous “language gene”) and some of its functional partners nicely illustrate this complex scenario. The linguistic (and the cognitive) profile of people bearing the well-known mutation R553H (KE family) is not homogeneous (Vargha-Khadem *et al.* 1995; Watkins *et al.* 2002). Moreover, several other pathogenic mutations of the gene, entailing diverse linguistic and cognitive deficits, have been identified thus far (Vargha-Khadem *et al.* 1995; Watkins *et al.* 2002; Vargha-Khadem *et al.* 2005; Shriberg *et al.* 2006; Roll *et al.* 2010). Additionally, unlike *FOXP2* itself, the mutation of *CNTNAP2* (one of its physiological targets) give rise to canonical SLI (Vernes *et al.* 2008), but also to stuttering (Petrin *et al.* 2010), language and mental delay (Sehested *et al.* 2010), and autism (Alarcón *et al.* 2008; Bakkaloglu *et al.* 2008). However, some polymorphisms of *CNTNAP2* also affects to language development (Whitehouse *et al.* 2011) and language processing in adult healthy people (Whalley *et al.* 2011). Conversely, the mutation of *SRPX2* (another of *FOXP2*'s targets) (Roll *et al.* 2010) gives rise to rolandic (or sylvian) epilepsy with speech dyspraxia (Roll *et al.* 2006) or to bilateral perisylvian polymicrogiria with dysarthria and mild mental retardation (Roll *et al.* 2006).

2. UNSATISFACTORY EFFORTS (THOUGH STILL WORTH TRYING)

To some extent, the problem reviewed above should benefit from the improvement of the diagnostic tools currently used in clinical linguistics. Specifically, it is important to maximize the linguistic nature of the experimental tasks used for the diagnosis in order to only evaluate specific components/operations of language that may be selectively impaired in language disorders. However, this is not easy to achieve. Actually, it may well be impossible if other cognitive devices besides language itself are involved in passing from competence to performance (Newmeyer 1997). At the same time, diagnostic tests should evaluate real neurolinguistic entities only. As we discuss below, in some cases the linguistic features, units, categories, rules, or computations under analysis (phonological features, agreement patterns and the like) may be not compatible with the sort of computations the brain is able to make in real time. A related concern is the reliability and relevance of the parameters under evaluation in the tests. For instance, a shortfall in repeating pseudowords or in generating inflected verbal forms have been proposed as core psycholinguistic markers for SLI (Bishop *et al* 1996). Nonetheless, the former deficit is also a relevant hallmark of children with dyslexia (Mayringer and Wimmer 2000, Quaglino *et al.* 2008), or with Down syndrome (Jarrold *et al.* 2000). At the same time, pseudoword reading could actually be a misinforming measure if either phonological processing ability or phonological awareness are to be evaluated in children below 4 years (Thomson *et al.* 2006). Concerning inflection, because different computational processes are involved in agreement, lower scores in tests evaluating inflectional morphology can be actually due to diverse underlying deficits.

Moreover, several diagnostic tests may exist for the same disorder. If they follow different criteria, it can be (erroneously) concluded that the condition is caused by different underlying deficits, and/or is co-morbid with other language impairments (and/or other cognitive disorders), or (quite typically) that several subtypes of the disorder actually exists. A related concern is the fact that disorders are commonly diagnosed categorically (you have it or you haven't). As a consequence, people having one disorder usually show symptoms that are not homogeneous (this ultimately explains why different subtypes of a disorder are frequently postulated; see above). In practice, clinical categories are cover terms for pathological groups that are diverse both symptomatically and aetiologically (see Parisse and Maillart 2009 on SLI). Because their definition commonly entails some sort of homogenization of the observed data, it is important to always rely on properly normalised statistical procedures when establishing them. Nowadays language disorders are usually characterised as continuous variables, that is, as specific intervals within a continuum also encompassing the linguistic competence of the non-affected population. However, we should always wonder about the biological reliability and significance of the clinical frontiers we may eventually draw (between the affected and the non-affected populations, across different disorders, or those delimiting different subtypes of the same disorder) (see Shaywitz *et al* 2008 on dyslexia for a discussion).

Similarly, we need to improve the confidence and the resolution of the neuroimaging devices used for analysing the disordered brain. Current techniques do not allow us to always discern whether multifunctional areas are composed or not of different neuronal populations performing different kind of computation. Moreover, functional neuroimaging just provide us with (low-resolution) images of the physiological changes (in terms of blood flux, electrical activity, and so on) elicited by the experimental tasks used for the diagnosis. However, these pictures cannot be equated to the representations and computations that are important for language (and for linguistic theory). As Poeppel

(2012) puts it, mapping is not explaining. In order to explain what we observe we first need to address two important shortcomings of current neurolinguistic studies. First, “[l]inguistic and neuroscientific studies of language operate with objects of different granularity” (Poeppel and Embick 2005: 105). Neurolinguistics makes broad conceptual distinctions (syntax vs. semantics, morphology vs. syntax, etc.), which usually involve the admixture of multiple components or processes of diverse nature. Second, “the fundamental elements of linguistic theory cannot be reduced or matched up with the fundamental biological units identified by neuroscience” (Poeppel and Embick 2005: 105). Overall, we first need to spell language (and language deficits) “in computational terms that are at the appropriate level of abstraction (i.e. can be performed by specific neuronal populations)” (Poeppel and Embick 2005: 106) (we will return to this problem in section 3). Ultimately, if other cognitive systems besides language are compulsorily involved in passing from competence to performance, we should not expect that neuroimaging techniques provide us with ‘sharp’ images of language at the neural level.

Finally, it is necessary as well to optimise the tools employed for analysing the molecular underpinnings of language disorders. Of course, the concerns raised above (in particular, the way in which clinical subjects are diagnosed) are also important at this level. However, these tools have different caveats and shortcomings. For example, approaches based on quantitative trait loci cannot properly detect highly polymorphic loci. As a consequence, it may be wrongly concluded that the disorders is caused by the mutation of a few principal genes. Similarly, positional cloning just renders statistical correlations between specific phenotypes and genes. Nonetheless, this needs to be validated in other populations and environments. Finally, genome-wide analysis (GWAs) allows for identifying candidate genes across the whole genome, but strong statistical corrections needs to be implemented.

On the other hand, we need to optimize as well current typologies of the disorders, both those based on symptoms and those based on their aetiology. Concerning typologies based on symptoms, because disorders usually show a continuous distribution, it may be worth taking into account the severity of the symptoms (see Monfort and Monfort 2012 for a discussion). However, this may be not enough. Actually, we should expect that clinical categories still have different aetiologies. Moreover, some of them (or some of their subtypes) may be unreal if they merge units, levels, or operations of language. With regards to aetiological classifications, it has been suggested that clinical approaches to disorders may greatly benefit if different kind of data are considered: genetic, neurobiological, cognitive, and even evolutionary. However, as we reviewed above, the same dysfunctional pieces may be shared across disorders that have distinctive symptomatic profiles.

3. EXPLORING NEW AVENUES

The strategies reviewed above will surely contribute to a better understanding and handling of language disorders. Nonetheless, they could be not enough. (Some kinds of) clinical linguistics still relies on naïve approaches to the biological underpinnings of language and of language disorders. Hence, as we highlighted in the first section of the paper, gene mutations are expected to affect to brain areas involved in language processing only, and ultimately, to give rise to linguistic deficits only (e.g. Falcato *et al.* 2008). Similarly, language disorders are expected to be homogeneous categories (at all levels of analysis) across populations and throughout development. And this is not the case. In our opinion, we need an improved approach to language disorders in the spirit of

the Biolinguistic turn in language sciences (see Boeckx and Benítez-Burraco 2014a for a review). Eventually, a change of focus (or a paradigm shift) may be also needed.

At the very least, it is urgent to take both linguistics and biology seriously when analysing language disorders. On the Linguistics side, language disorders should be construed (and examined) in terms of the primitives (units and computations) that are central in current linguistic theories (of course, only of those that can be computed by the brain in real time). On the Biology side, some key lessons about the way in which living beings are organized and develop should be taken into account. To begin with, genes are not blueprints. Non-genetic factors also play a key role in controlling development. At the same time, development (and this is particularly true of the brain) is not fully predetermined before birth, since it also depends of environmental factors. As a consequence, the phenotype is always indirectly related to the genotype.

Let us examine this problem in some detail. Genes just codify biochemical products (either proteins or non-coding RNAs [ncRNAs]) that perform specific functions inside or outside the cell. However, genes are not able to do this by themselves (not to mention to give rise to phenotypic traits!). Genes are transcribed into RNA and (some of them) are subsequently translated into proteins by a complex biological machinery. In conjunction with gene regulatory regions, this machinery determines when, where, and how much a gene is expressed, and which functional products are going to be synthesised (several functional products can be synthesised from the same gene, which will affect to different traits). We are just about getting a rough idea about the intricacy of this regulatory machinery. But we have recently learnt that gene expression heavily relies on ncRNAs and not only in DNA sequences and regulatory proteins (Mattick *et al.* 2009; Mattick 2011). Additionally, it seems that development more depends on the transcriptional state of the cell than on genetic sequences themselves (Mattick *et al.* 2009). We are used to regard DNA mutations as the major aetiological factor of inherited language disorders. However, we have found that DNA is widely epigenetically modified, that is, it is modified to modulate how regulatory factors interact with it). Importantly, these modifications are inheritable too (Isles and Wilkinson 2000) and they have been linked to basic brain processes (such as neural proliferation and differentiation, and particularly, to neural plasticity), and eventually, to key cognitive abilities for language acquisition and processing, such as learning and memory (Levenson and Sweatt 2006; Gräff and Mansuy 2008; Mehler 2008). Of course, many internal (proteins, hormones, chemiotactic factors, etc.) and external (environmental cues) may affect the transcriptional (and epigenetic) state of a gene. Overall, we now believe that development (and ultimately, the emergence of pathological traits) more depends on the transcriptional state of the cell than on genetic sequences themselves (Mattick *et al.* 2009).

Nonetheless, even if a gene is expressed in the proper place, time window and amount, a direct link with a particular phenotype is not granted. Gene products usually undergo posttranscriptional and/or posttranslational modifications, rendering different transcripts and/or diverse proteins or non-coding RNAs (ncRNAs). Very frequently these molecules need to be assembled in multimolecular complexes. Importantly, gene products usually interact in the form of intricate regulatory networks (Geschwind and Konopka 2009). These complex interactions make the phenotype linked to the mutation of a particular gene pretty variable and hardly predictable. This explains why the mutation of one of these genes can give rise to different language and/or cognitive deficits and disorders, as we pointed out in section 1. Likewise, other diverse factors influence (the variability of) the trajectories ultimately followed by developmental processes. For example, viscoelasticity or differential diffusion and oscillation (acting in combination with basic properties of the cell like polarity or differential adhesion) modulate the way

in which all the involved elements (proteins, ncRNAs, hormones, etc.) behave, interact, and function. This ultimately affects to basic dimensions of tissue development and organization, such as regionalization patterns, and eventually, to phenotypic traits (Newman and Comper 1990; Goodwin 1994; Newman *et al.* 2006). Lastly, developmental processes are, to some extent, stochastic phenomena. This is why “identical developmental processes [and consequently, identical gene sequences] in identical environments produce different outcomes” (Balaban 2006: 320).

When it comes to the brain it is important to notice that this complex regulatory mechanism does not give rise to neural devices that are fully operative. On the contrary, additional changes in neural architecture are needed. They usually result from feedback effects from other brain regions or from external stimuli. Consequently, a direct link between language and the brain should be never expected. Actually, as we pointed out in the previous section, the neural devices resulting from development cannot be directly equated to (the neural substrate of) linguistic features or operations. On the contrary, “differently structured cortical areas are specialized for performing different types of computations, and [...] some of these computations are necessary for language but also for other cognitive functions” (Poeppel and Embick 2005: 112). This is why the impairment of any of these areas may affect to more than one cognitive functions and ultimately, give rise to symptoms that are suggestive of more than one (co-morbid) disorders. Incidentally, this disqualifies language from being a module in the Fodorian sense. On the contrary, language is a cross-modular cognitive function, resulting from the interface of diverse neuronal devices performing basic functions (Hauser *et al.* 2002; Balari and Lorenzo 2013; Boeckx and Benítez-Burraco 2014b). Such cognitive modules (as Griffiths 2007 calls them) are always the outcome of major changes in the brain architecture and function occurred during development under environmental cues, although their basic wiring is achieved before birth under genetic instructions (see Karmiloff-Smith 2010 for discussion). Consequently, we cannot go on construing disorders as static entities. On the contrary, we should expect that the phenotypic profile of the affected people (and the biological and cognitive machinery supporting their linguistic abilities) is different at different stages of development. As pointed out by Karmiloff-Smith (2009: 58): “to understand developmental outcomes, it is vital to identify full developmental trajectories, to assess how progressive change occurs from infancy onwards, and how parts of the developing system may interact with other parts differently at different times across ontogenesis” (Karmiloff-Smith 2009: 58). Moreover, similar cognitive profiles can rely on different brain architectures. As Karmiloff-Smith (2010: 182) puts it: “the same behaviour may be subserved by different neural substrates at different ages during development” (Karmiloff-Smith 2010: 182). Because there may be more than one way of implementing a (more or less) functional faculty of language at the term of growth (see Hancock and Bever 2013 for discussion), we (urgently) need a good developmental account of language disorders.

4. A NEW PARADIGM

The improved biological (or biolinguistic) account of language and of language disorders outlined in the previous section is more in line with how biologists think about development and evolution, how neuroscientists think about the brain and how psychologists think about cognitive development (and even how most linguists outside Chomskyan circles think about language). Nonetheless, further evidence suggests that we may actually need a new theoretical framework in clinical linguistics if we want to properly understand (and deal with) language disorders.

We do believe that developmental processes are the key to understand what we observe in the adult state. As we noted in the previous section variation pervades language (and language disorders) at all levels, from genes to molecules to brain networks to psycholinguistic measures. However, it is crucial to note that variation is quite constrained too. And the same holds for developmental disturbances. In truth, the developing brain is able to compensate many kind of damage, to the extent that quite preserved linguistic abilities can be achieved in spite of many kind of mutations, brain anomalies and severe cognitive impairments (Sirois *et al.* 2008). Interestingly, while some aspects of language are nearly never disturbed or are always compensated (for example, basic phrasal rules), others are impaired in many (if not all) disorders (for example, verbal inflection). Ultimately, the number of language disorders is far smaller than the number of aetiological factors involved. Moreover, we observe that although disorders show specific symptomatic profiles, their prevalent symptoms usually result from the impairment of low-level, more generalized processes. Actually, we find in them diffuse effects on the brain architecture and function, and on different cognitive capacities (this being compatible with a greater impairment of certain functions). This is why disorders are better described in terms of the juxtaposition of impaired and preserved modules than as the outcome of anomalous associations across domains. Ultimately, as we have already noted, the linguistic profile of affected people changes from one group to another, and from one developmental stage to the next. Overall, quite preserved linguistic capacities can be achieved in spite of deeper cognitive impairments relying on different (and changing) brain architectures and cognitive abilities. At the same time, there are not so many ways of implementing language at the brain level.

Our main point is that this messy scenario (as we called it in section 1) is easier to interpret if we move to a new theoretical paradigm, namely, an evo-devo account of disorders, which builds on the evo-devo theories that interpret the deep links between development and evolution in biology. Actually, what we observe in language disorders (to a greater degree than in the normal population) is that language is both sensitive to environmental changes (that is, *plastic*) and resistant to environmental perturbations (that is, *canalized*), both prompted to evolve (that is, *evolvable*) and resistant to modification (that is, *robust*). Whenever canalization fails to cope with developmental perturbations (deleterious gene mutations, brain damage, and the like), certain cognitive deficits arise, certain linguistic abilities are not properly achieved and/or certain developmental milestones are not reached or its acquisition becomes delayed because they are achieved via compensatory mechanisms. Plausibly, these anomalies only concern to neural networks that are endowed with less robust compensatory mechanisms because of their evolutionary novelty (Toro *et al.* 2010). Conversely, the components of language (genetic, physiological, or cognitive) that are more resistant to damage (and that are not affected in disorders) have a long evolutionary history. According to Gibson (2009), decanalization explains the high prevalence of complex diseases (including language disorders) among human populations. We believe that specific mutations, demographic bottlenecks and cultural changes caused a phase transition from ape cognition to human cognition that prompted the emergence of language as a result of the interface among basic cognitive blocks which are particularly robust after millions of years of stabilizing selection (Balari and Lorenzo 2013; Boeckx and Benítez-Burraco 2014). However, this transition uncover cryptic variation which increased the prevalence of language disorders. Moreover, because of its evolutionary novelty and the less resilience of the networks they rely on, these new interfaces are very sensitive to damage. Plausibly, this explains why the same deficits are found in nearly all disorders and why they usually concern to

morpho-phonology and to the most demanding tasks in computational terms (e.g. agreement).

Importantly, we also believe that the limited set of pathological conditions characterised by clinical linguists may be the only possible set of phenotypes resulting from the combination of the diverse factors regulating the development of the brain. In evo-devo theories these limited set of phenotypes are usually characterised as morphospaces or adaptive landscapes (McGhee 2006). We think that this fresh account of disorders may be of great interest for clinical linguistics. Accordingly, we should expect that each disorder is placed in a different place of the language morpho-space (which also includes the language faculty of the non-affected population). What we need then is to find the best parameters defining the language morpho-space. For example, we might rely on (aberrant) gene expression profiles in the cell to define the stable states attracted through development (remember that we expect pathological instances to be also stable ontogenetic states, but endowed with idiosyncratic, less functional properties). Another promising possibility is the kind of networks resulting from the measurement of the syntactic relationships between words (or morphemes) in the utterances produced by speakers in real conversations. This approach accurately characterizes language growth in the child as phase transitions in the syntactic complexity of her discourse. Different disorders are expected to show different, disorder-specific profiles in terms of the topographical features of these networks and the timing of the transitions (if any) between different kinds of networks, to the extent that they may work as robust endophenotypes or early clinical hallmarks of the disorders (see Barceló-Coblijn *et al.* submitted for details).

Nonetheless, a better candidate for properly defining the morpho-space of language growth in the species (either pathological or not) are brain rhythms. Brain rhythms are primitive components of brain function and we expect them to be connected to some computational primitives of language, allowing to understand (and not just to localize) brain functions. For example, basic operations in Minimalism, like ‘Spell Out’ or ‘Unify’ (that is, the regulation of Merge by means of its interfacing with, or its embedding inside, the cognitive systems responsible for interpretation and externalization) (Jackendoff 2002; Hagoort 2005), may be interpreted as the embedding of high frequency oscillations inside oscillations operating at a slower frequency (see Benítez-Burraco and Boeckx 2014 for details). Similarly, some rhythmic features of speech have been related to specific brain oscillations (Giraud and Poeppel 2012). Importantly, the hierarchy of brain oscillations has remained remarkably preserved within mammals during evolution (Buzsáki *et al.* 2013). Consequently, we should expect that the human pattern of brain activity is a slight variation of the pattern observed in other primates. Interestingly, different cognitive disorders have probed to correlate with specific profiles of brain activity (Buzsáki and Watson 2012). We believe that these anomalous patterns may correspond to different points within the adaptive landscape of the language faculty. If we succeed in this translation, we may be able to diagnose language disorders earlier and in a more accurate way, because each disorder is expected to result from a selective, disorder-specific alteration of the same brain oscillation grammar. Importantly also, these brain rhythms are expected to be highly quantifiable and heritable traits and thus, good endophenotypes of the disorders.

5. FUTURE PROSPECTS

The paradigm shift in clinical linguistics we advocate for is not easy to achieve. If we really want to gain a better characterization (and understanding) of language disorders

and also to optimize our therapeutic tools, we need to improve our current understanding of the biological underpinnings of the language faculty (disordered or not). In this we can rely in recent achievements of biolinguistics which is moving from a naïve account of the biology of language to more biologically-grounded views of language facts (see Boeckx and Benítez Burraco 2014a for review). Concerning language disorders and the new account we have argued for in this paper, we should persevere in several lines of research: i) disentangle the molecular mechanisms that channel (and fail to channel) variation at all levels, ii) improve evo-devo-friendly depictions of the modularization of the disordered brain; iii) optimize current models of the linguistic ontogeny of people with disorders; and iv) pay attention to emergent properties (and to properties that fail to emerge), since language is surely a complex system (Deacon 2005).

REFERENCES

- Alarcón, M., Abrahams, B. S., Stone, J. L., Duvall, J. A., Perederiy, J. V., Bomar, J. M., Sebat, J., Wigler, M., Martin, C. L., Ledbetter, D. H., Nelson, S. F., Cantor, R. M. and Geschwind, D. H. (2008) Linkage, association, and gene-expression analyses identify *CNTNAP2* as an autism-susceptibility gene. *Am. J. Hum. Genet.* 82: 150–9
- American Psychiatric Association (1994) *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)*. Washington: American Psychiatric Association.
- Bakkaloglu, B., O’Roak, B. J., Louvi, A., Gupta, A. R., Abelson, J. F., Morgan, T. M., Chawarska, K., Klin, A., Ercan-Sencicek, A.G., Stillman, A. A., Tanriover, G., Abrahams, B. S., Duvall, J. A., Robbins, E. M., Geschwind, D. H., Biederer, T., Gunel, M., Lifton, R. P. and State, M. W. (2008) Molecular cytogenetic analysis and resequencing of contactin associated protein-like 2 in autism spectrum disorders. *Am. J. Hum. Genet.* 82:165-73.
- Balaban E. (2006) Cognitive developmental biology: history, process and fortune’s wheel. *Cognition* 101: 298-332.
- Balari, S., and Lorenzo, G. (2013). *Computational Phenotypes*. Oxford: Oxford University Press.
- Barceló-Coblijn, Ll., Benítez-Burraco, A. and Irutzun, A. (submitted) Syntactic networks as endophenotypes of language disorders: an Evo-Devo approach to clinical linguistics. *Front. Lang. Sci.*
- Benítez-Burraco, A. (2010) Neurobiology and neurogenetics of dyslexia. *Neurología* 25: 563-581
- Benítez-Burraco, A. and Boeckx, C. (2014) Universal Grammar and biological variation: an EvoDevo agenda for comparative biolinguistics. *Biol. Theor.* 9: 122–134.
- Bishop, D. V. M. (2002). Motor immaturity and specific speech and language impairment: Evidence for a common genetic basis. *Am. J. Med. Genet.* 114: 56–63
- Bishop, D. V. M., North, D., and Donlan, C. (1996) Nonword repetition as a behavioural marker for inherited language impairment: evidence from a twin study. *J. Child Psychol. Psychiatry* 36: 1-13
- Boeckx, C. and Benítez-Burraco, A. (2014a) Biolinguistics 2.0. In *The Design, Development and Evolution of Human Language: Biolinguistic Explorations* (K. Fujita, N. Fukui, N. Yusa & M. Ike-Uchi, eds). Tokyo: Kaitakusha, pp. 8-30.
- Boeckx C. and Benítez-Burraco A. (2014b) The shape of the human language-ready brain. *Front. Psychol.* 5: 282

- Botting, N. and Conti-Ramsden, G. (2004) Characteristics of children with specific language impairment. In *Classification of Developmental Language Disorders* (L. Verhoeven and H. Van Balkom, eds.). Mahwah: Erlbaum, pp. 23-38.
- Buzsáki, G., Logothetis, N., and Singer, W. (2013) Scaling brain size, keeping timing: evolutionary preservation of brain rhythms. *Neuron* 80: 751-64.
- Buzsáki, G. and Watson, B. O. (2012) Brain rhythms and neural syntax: implications for efficient coding of cognitive content and neuropsychiatric disease. *Dialogues Clin. Neurosci.* 14: 345-67.
- Catts, H. W., Adlof, S. M., Hogan, T. P. and Weismer, S. E. (2005) Are specific language impairment and dyslexia distinct disorders? *J. Speech Lang. Hear. Res.* 48: 1378-1396.
- Deacon, T. W. (2005) Language as an emergent function: some radical neurological and evolutionary implications. *Theoria* 54: 269-286
- Démonet, J.-F., Taylor, M. J. and Chaix, Y. (2004) Developmental dyslexia. *Lancet* 363: 1451-1460
- Deutsch, G. K., Dougherty, R. F., Bammer, R., Siok, W. T., Gabrieli, J. D., and Wandell, B. (2005) Children's reading performance is correlated with white matter structure measured by diffusion tensor imaging. *Cortex* 41: 354-363
- Donnai, D. and Karmiloff-Smith, A. (2000) Williams syndrome: from genotype through to the cognitive phenotype. *Am. J. Med. Genet.* 97: 164-71.
- Dricot, L., Sorger, B., Schiltz, C., Goebel, R., and Rossion, B. (2008) Evidence for individual face discrimination in non-face selective areas of the visual cortex in acquired prosopagnosia. *Behav. Neurol.* 19: 75-79.
- Falcaro, M., Pickles, A., Newbury, D. F., Addis, L., Banfield, E., Fisher, S. E., Monaco, A. P., Simkin, Z., Conti-Ramsden, G., and the SLI Consortium (2008) Genetic and phenotypic effects of phonological short-term memory and grammatical morphology in specific language impairment. *Genes Brain Behav.* 7: 393-402.
- Galaburda, A. M., Sherman, G. F., Rosen, G. D., Aboitiz, F., and Geschwind, N. (1985). Developmental dyslexia: four consecutive patients with cortical anomalies. *Ann. Neurol.* 18: 222-233
- Geschwind, D. H. and Konopka, G. (2009) Neuroscience in the era of functional genomics and systems biology. *Nature* 461: 908-915
- Gibson, G. (2009) Decanalization and the origin of complex disease. *Nature Rev. Genet.* 10:134-140
- Giraud, A. L. and Poeppel, D. (2012) Cortical oscillations and speech processing: emerging computational principles and operations. *Nat. Neurosci.* 15: 511-7.
- Goodwin, B. (1994) *How the Leopard Changed its Spots. The Evolution of Complexity.* New York: Charles Scribner's Sons
- Gräff, J. and Mansuy, I. M. (2008) Epigenetic codes in cognition and behaviour. *Behav. Brain Res.* 192: 70-87
- Griffiths, P. E. (2007) Evo-Devo meets the mind: towards a developmental evolutionary psychology. In *Integrating Evolution and Development: Form Theory to Practice* (R. Brandon and R. Sansom, eds). Cambridge: MIT Press, pp. 195-225.
- Hagoort, P. (2005). On Broca, brain, and binding: a new framework. *Trends Cogn. Sci.* 9, 416-23.
- Hancock, R. and Bever, T. G. (2013) Genetic factors and normal variation in the organization of language. *Biolinguistics* 7:75-95
- Hauser, M. D., Chomsky, N., and Fitch, W. T. (2002) The faculty of language: what is it, who has it, and how did it evolve? *Science* 298: 1569-1579.

- Horwitz, B., Rumsey, J. M., and Donohue, B. C. (1998) Functional connectivity of the angular gyrus in normal reading and dyslexia. *Proc. Natl. Acad. Sci. U.S.A* 95: 8939-8944.
- Isles, A. R. and Wilkinson, L. S. (2000) Imprinted genes, cognition and behaviour. *Trends Cogn. Sci.* 4: 309-318
- Jackendoff, R. (2002) *Foundations of Language: Brain, Meaning, Grammar, Evolution*. Oxford: Oxford University Press.
- Jarrold, C., Baddeley, A. D., and Hewes, A. K. (2000) Verbal short-term memory deficits in Down syndrome: a consequence of problems in rehearsal? *J. Child Psychol. Psychiatry* 41: 233-244
- Karmiloff-Smith, A. (2008) Research into Williams syndrome: the state of the art. In *Handbook of Developmental Cognitive Neuroscience* (C. A. Nelson and M. Luciana, eds.), Cambridge, MIT Press, pp. 691-700.
- Karmiloff-Smith, A. (2009) Nativism versus neuroconstructivism: rethinking the study of developmental disorders. *Dev. Psychol.* 45: 56-63
- Karmiloff-Smith, A. (2010) A developmental perspective on modularity. In *Towards a Theory of Thinking* (B. M. Glatzeder, V. Goel, and A. von Müller, eds.) Berlin: Springer-Verlag, pp. 179-187
- Karmiloff-Smith, A. and Mills, D. L. (2006) Williams Syndrome. In *Encyclopedia of Language and Linguistics* (K. Brown, ed.). Vol. 13. Oxford: Elsevier, pp. 585-589.
- Levenson, J. M. and Sweatt, J. D. (2006) Epigenetic mechanisms: a common theme in vertebrate and invertebrate memory formation. *Cell. Mol. Life Sci.* 63: 1009-1016.
- Livingstone, M. S., Rosen, G. D., Drislane, F. W. and Galaburda, A. M. (1991) Physiological and anatomical evidence for a magnocellular defect in developmental dyslexia. *Proc. Natl. Acad. Sci. U. S. A.* 88: 7943-7947
- Lovegrove, W. J., Bowling, A., Badcock, D. and Blackwood, M. (1980) Specific reading disability: differences in contrast sensitivity as a function of spatial frequency. *Science* 210: 439-440
- Lyon, G., Shaywitz, S. y Shaywitz, B. (2003) A definition of dyslexia. *Ann. Dyslexia* 53: 1-14.
- Maisog, J. M., Einbinder, E. R., Flowers, D. L., Turkeltaub, P. E., and Eden, G. F. (2008) A meta-analysis of functional neuroimaging studies of dyslexia. *Ann. N. Y. Acad. Sci.* 1145: 237-259
- Mattick, J. S. (2011) The central role of RNA in human development and cognition. *FEBS Lett.* 585: 1600-1616
- Mattick, J. S., Taft, R. J., and Faulkner, G. J. (2009) A global view of genomic information: moving beyond the gene and the master regulator. *Trends Genet.* 26: 21-28.
- Mayringer, H. and Wimmer, H. (2000) Pseudoname learning by German-speaking children with dyslexia: evidence for a phonological learning deficit. *J. Exp. Child Psychol.* 75: 116-33.
- McGhee, G. R. (2006) *The Geometry of Evolution, Adaptive Landscapes and Theoretical Morphospaces*. Cambridge: Cambridge University Press.
- Mehler, M. F. (2008) Epigenetic principles and mechanisms underlying nervous system functions in health and disease. *Prog. Neurobiol.* 86: 305-341
- Mervis, C. B. and Becerra, A. M. (2007) Language and communicative development in Williams syndrome. *Ment. Retard. Dev. Disabil. Res. Rev.* 13: 3-15.

- Monfort, I. and Monfort, M. (2012) Utilidad clínica de las clasificaciones de los trastornos del desarrollo del lenguaje. *Rev. Neurol.* 54: S147-S154.
- Newman, S. A. and Comper, W. D. (1990) 'Generic' physical mechanisms of morphogenesis and pattern formation. *Development* 110: 1-18
- Newman, S. A., Forgacs, G., and Müller, G. B. (2006) Before programs: the physical origination of multicellular forms. *Int. J. Dev. Biol.* 50: 289-299
- Newmeyer, F. J. (1997) Genetic dysphasia and linguistic theory. *J. Neurolinguistics* 10: 47-73.
- Nicolson, R. I. and Fawcett, A. J. (2006) Do cerebellar deficits underlie phonological problems in dyslexia? *Dev. Sci.* 9: 259-262.
- O'Malley, K. D. and Nanson, J. (2002) Clinical implications of a link between fetal alcohol spectrum disorder and attention-deficit hyperactivity disorder. *Can. J. Psychiatry* 47: 349-354
- Parisse, C. and Maillart, C. (2009) Specific language impairment as systemic developmental disorders. *J. Neurolinguistics* 22: 109-122
- Paulesu, E., Demonet, J.-F., Fazio, F., McCrory, E., Chanoine, V., Brunswick, N., Cappa, S. F., Cossu, G., Habib, M., Frith, C. D., and Frith, U. (2001) Dyslexia: cultural diversity and biological unity. *Science* 291: 2165-2167.
- Petrin, A. L., Giacheti, C. M., Maximino, L. P., Abramides, D. V., Zanchetta, S., Rossi, N. F., Richieri-Costa, A., and Murray, J. C. (2010) Identification of a microdeletion at the 7q33-q35 disrupting the CNTNAP2 gene in a Brazilian stuttering case. *Am. J. Med. Genet. A* 152A: 3164-3172.
- Poepfel, D. (2012) The maps problem and the mapping problem: Two challenges for a cognitive neuroscience of speech and language. *Cogn. Neuropsychol.* 29: 34-55.
- Poepfel, D. and Embick, D. (2005) Defining the relation between linguistics and neuroscience. In *Twenty-first Century Psycholinguistics: Four Cornerstones* (A. Cutler, ed.). Hillsdale: Lawrence Erlbaum, pp. 103-120.
- Purvis, K. L. and Tannock, R. (1997) Language abilities in children with attention deficit hyperactivity disorder, reading disabilities, and normal controls. *J. Abnorm. Child Psychol.* 25: 133-144
- Quaglino, V., Bourdin, B., Czternasty, G., Vrignaud, P., Fall, S., Meyer, M. E., Berquin, P., Devauchelle, B., and de Marco, G. (2008) Differences in effective connectivity between dyslexic children and normal readers during a pseudoword reading task: an fMRI study. *Neurophysiol. Clin.* 38: 73-82..
- Rapin, I. and Allen, D. A. (1983) Developmental language disorders: Nosologic considerations. In *Neuropsychology of Language, Reading and Spelling* (U. Kirk, ed.). New York: Academic Press, pp. 155-184
- Rapport, M. D., Alderson, R. M., Kofler, M. J., Sarver, D. E., Bolden, J., and Sims, V. (2008) Working memory deficits in boys with attention-deficit/hyperactivity disorder (ADHD): the contribution of central executive and subsystem processes. *J. Abnorm. Child Psychol.* 36: 825-37
- Rhodes, S. M., Riby, D. M., Matthews, K., and Coghill, D. R. (2011) Attention-deficit/hyperactivity disorder and Williams syndrome: shared behavioral and neuropsychological profiles. *J. Clin. Exp. Neuropsychol.* 33: 147-56.
- Roll, P., Rudolf, G., Pereira, S., Royer, B., Scheffer, I. E., Massacrier, A., Valenti, M. P., Roedel-Trevisiol, N., Jamali, S., Beclin, C., Seegmuller, C., Metz-Lutz, M. N., Lemainque, A., Delepine, M., Caloustian, C., de Saint Martin, A., Bruneau, N., Depétris, D., Mattéi, M. G., Flori, E., Robaglia-Schlupp, A., Lévy, N., Neubauer, B. A., Ravid, R., Marescaux, C., Berkovic, S. F., Hirsch, E., Lathrop, M., Cau, P.,

- and Szepetowski, P. (2006) *SRPX2* mutations in disorders of language cortex and cognition. *Hum. Mol. Genet.* 15: 1195-1207.
- Roll, P., Vernes, S. C., Bruneau, N., Cillario, J., Ponsolle-Lenfant, M., Massacrier, A., Rudolf, G., Khalife, M., Hirsch, E., Fisher, S. E., and Szepetowski, P. (2010) Molecular networks implicated in speech-related disorders: *FOXP2* regulates the *SRPX2/uPAR* complex. *Hum Mol Genet* 19: 4848-4860.
- Sehested, L. T., Møller, R. S., Bache, I., Andersen, N. B., Ullmann, R., Tommerup, N., and Tümer, Z. (2010) Deletion of 7q34-q36.2 in two siblings with mental retardation, language delay, primary amenorrhea, and dysmorphic features. *Am. J. Med. Genet. A* 152A: 3115-3119.
- Shaywitz, S. E. (1998) Dyslexia. *N. Engl. J. Med.* 338: 307-312
- Shaywitz, S. E., Shaywitz, B. A., Pugh, K. R., Fulbright, R. K., Constable, R. T., Mencl, W. E., Shankweiler, D. P., Liberman, A. M., Skudlarski, P., Fletcher, J. M., Katz, L., Marchione, K. E., Lacadie, C., Gatenby, C. and Gore, J. C. (1998) Functional disruption in the organization of the brain for reading in dyslexia. *Proc. Nat. Acad. Sci. U.S.A.* 95: 2636-2641.
- Shaywitz, S., Morris, R., and Shaywitz, B. (2008) The education of dyslexic children from childhood to young adulthood. *Annu. Rev. Psychol.*, 59, 451-475
- Shriberg, L. D., Ballard, K. J., Tomblin, J. B., Duffy, J. R., Odell, K. H., and Williams, C. A. (2006) Speech, prosody, and voice characteristics of a mother and daughter with a 7,13 translocation affecting *FOXP2*. *J. Speech Lang. Hear. Res.* 49: 500-525.
- Shriberg, L. D., Tomblin, J. B. and McSweeney, J. L. (1999) Prevalence of speech delay in 6-year-old children and comorbidity with language impairment. *J. Speech Lang. Hear. Res.* 42: 1461-1481.
- Sirois, S., Spratling, M., Thomas, M. S., Westermann, G., Mareschal, D., and Johnson, M. H. (2008) Précis of neuroconstructivism: how the brain constructs cognition. *Behav. Brain Sci.* 31: 321-331
- Smith, S. D., Gilger, J. W. and Pennington, B. F. (1996) Dyslexia and other specific learning disorders. In *Principles and practice of medical genetics* (D. L. Rimoim, J. M. Connor and R. E. Pyeritz, eds.). New York: Churchill Livingstone, pp. 1767-1789.
- Sorger, B., Goebel, R., Schiltz, C., and Rössion, B. (2007) Understanding the functional neuroanatomy of acquired prosopagnosia. *Neuroimage* 35: 836-852.
- Stein, J. and Walsh, V. (1997) To see but not to read: the magnocellular theory of dyslexia. *Trends Neurosci.* 20: 147-152
- Temple, E., Poldrack, R. A., Protopapas, A., Nagarajan, S., Salz, T., Tallal, P., Merzenich, M. M. and Gabrieli, J. D. E. (2000) Disruption of the neural response to rapid acoustic stimuli in dyslexia: evidence from functional MRI. *Proc. Nat. Acad. Sci. U.S.A.* 97: 13907-13912.
- Thomson, B., Crewther, D. P., and Crewther, S. G. (2006) Wots that werd? Pseudowords (non-words) may be a misleading measure of phonological skills in young learner readers. *Dyslexia* 12: 289-99.
- Toga, A. W., Thompson, P. M., and Sowell, E. R. (2006) Mapping brain maturation. *Trends Neurosci.* 29: 148-159.
- Toro, R., Konyukh, M., Delorme, R., Leblond, C., Chaste, P., Fauchereau, F., Coleman, M., Leboyer, M., Gillberg, C., and Bourgeron, T. (2010) Key role for gene dosage and synaptic homeostasis in autism spectrum disorders. *Trends Genet.* 26: 363-72
- Vargha-Khadem, F., Gadian, D. G., Copp, A., and Mishkin, M. (2005) *FOXP2* and the neuroanatomy of speech and language. *Nat. Rev. Neurosci.* 6: 131-138.

- Vargha-Khadem, F., Watkins, K. E., Alcock, K. J., Fletcher, P., and Passingham, R. E. (1995) Praxic and nonverbal cognitive deficits in a large family with a genetically transmitted speech and language disorder. *Proc Natl Acad Sci USA* 92: 930-933.
- Vernes, S. C., Newbury, D. F., Abrahams, B. S., Winchester, L., Nicod, J., Groszer, M., Alarcón, M., Oliver, P. L., Davies, K. E., Geschwind, D. H., Monaco, A. P., and Fisher, S. E. (2008) A functional genetic link between distinct developmental language disorders. *N. Engl. J. Med.* 359: 2337-45.
- Watkins, K. E., Dronkers, N. F., and Vargha-Khadem, F. (2002) Behavioural analysis of an inherited speech and language disorder: comparison with acquired aphasia. *Brain* 125: 452-64.
- Whalley, H. C., O'Connell, G., Sussmann, J. E., Peel, A., Stanfield, A. C., Hayiou-Thomas, M. E., Johnstone, E. C., Lawrie, S. M., McIntosh, A. M., and Hall, J. (2011) Genetic variation in *CNTNAP2* alters brain function during linguistic processing in healthy individuals. *Am. J. Med. Genet. B Neuropsychiatr. Genet.* 156: 941-948
- Whitehouse, A. J., Bishop, D. V., Ang, Q. W., Pennell, C. E., and Fisher, S. E. (2011) *CNTNAP2* variants affect early language development in the general population. *Genes Brain Behav.* 10: 451-456
- Wyper, K. R. and Rasmussen, C. R. (2011) Language impairments in children with fetal alcohol spectrum disorders. *J. Popul. Ther. Clin. Pharmacol.* 18: e364-376