The Logical Problem of Language Acquisition Revisited: Insights from Error Patterns in Typical and Atypical Development[1]

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Abstract
A major impetus for understanding and building theories of language acquisition is the fact that children's grammars often deviate from adult-state grammars in intriguingly systematic ways, before converging on a grammatical system that is equivalent to that of the local linguistic community. This paper will focus on error patterns in children's non-adult structural configurations, particularly those found in a subpopulation of children diagnosed with Specific Language Impairment and Autism Spectrum Disorders. Data from language disorders may provide a prolonged window into primitives of grammar and suggest a mapping of certain genes to higher-level cognitive modules such as language. However, the heterogeneity along the developmental paths highlights the significance of the process of ontogenetic development, ultimately demonstrating that the relationship between genotype (the genetic code, i.e. the material encoding heritable traits) and phenotype (the expression of the genetic code, i.e. the observable characteristics or behavior) is quite indirect.

1. Introduction

It is astonishing that every typically-developing (henceforth TD) child acquires a natural language without formal instructions or scaffolding in the form of progressively sequenced linguistic input. Children thus

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converge on a grammatical system parallel to that of the local linguistic community, in the face of significant variability in the linguistic input (Crain, 1991, p. 597). Considering how hard it is even for trained linguists to discern grammatical principles, it is remarkable that research on language acquisition has demonstrated that young children know them, often by the age of three.

At its core, generative grammar aims to understand the (finite) combinatorial system of rules that underlie the (infinite) range of possible sentences in the world's languages (past, present and future). The logical problem of language acquisition refers to the idea that the data that children are exposed to underdetermine what they wind up knowing about their native language, as there may not be conclusive evidence for it in the linguistic input, i.e. what is known as *poverty of the stimulus*. This raises the question of what exactly the language-acquiring child brings to this induction task (Crain, 1991; Crain and Pietroski, 2001; Thornton, 1990).

Brown (1973: 156) concludes that errors in language acquisition are "triflingly few". This paper will focus on grammatical, primarily syntactic, errors in typical and atypical language development. The errors discussed are "grammatical" in the sense that they conform to both grammar (i.e. language-specific rules) and Grammar (i.e. the underlying grammatical system common to all languages), contra Kizach's (this volume) interpretation. A further distinction needs to be clarified: This contribution will not deal with grammatical *mistakes*; in contrast to errors, a mistake is made by a learner who knows a language-specific grammatical rule, but neglects to employ it, due to performance-related or extralinguistic factors.

Language is a like an organism, a biological system, and the methods linguists use when we study it ought to reflect this. Investigations into the biology of language typically draw upon empirical data from either language acquisition, language breakdown (e.g. Broca's aphasia and Wernicke's aphasia), neuroscience (in relation to neurologically intact individuals, using fMRI, EEG, MEG, etc.) or molecular biology (scrutinizing the relation between gene expression and language). By focusing on genetic developmental disorders in language-acquiring children, this contribution combines data from all four areas. Investigations into the grammatical nature of these language disorders have previously tended to be descriptive and not rely on theoretical linguistic principles. This limits their interdisciplinary potential, as explorations of the grammatical phenomena at the interface between e.g. linguistics and neurobiology require hypotheses built on underlying principles that can be tested (or

falsified). One of the aims of this paper is to highlight a new avenue of evidence and point to a theoretical platform that can integrate language disorders into the theory of the biological underpinnings of language, as the errors made by children with Autism Spectrum Disorders (ASD) and Specific Language Impairment (SLI) are so systematic in their deviance from the target that they may reflect universal properties of grammatical structure (e.g. Universal Grammar).

Just like the TD peers, children with language disorders such as SLI and ASD are in a linguistic learning environment that is characterized by idiosyncrasies and finiteness, but even though their grammars may never reach full convergence with those of the surrounding speech communities, their productions nonetheless exhibit universal properties in the face of selective and underdetermined input.

This contribution is a review of the state-of-the-arts in language acquisition research as it relates to language disorders. It is primarily a theoretical story that calls for extensive future research on language disorders, employing the fine-grained theoretical apparatus provided by decades of research in theoretical linguistics. Section 2 discusses constraints on the hypothesis space in language acquisition, including a probabilistic model, while Section 3 and 4 examine typical development in order to put the language disorders (ASD and SLI) in a relevant context. Two linguistic arguments for innate constraints (the phenomena of medial-*wh* and structure-dependence) are summarised and evaluated in the process. Based on these considerations, section 5 goes into depth with the syntactic profiles of SLI and ASD and discusses the extent to which the phenomena found here supports the idea of innate constraints in language acquisition. Finally, section 6 debates the implications of the data for neural networks and whether it is appropriate to map the linguistic phenotypes found in SLI and ASD with specific genotypes.

2. Constraints on the hypothesis space in language acquisition

According to Chomsky (1965, 1986), children are born equipped with Universal Grammar (henceforth UG), i.e. innate, biologically determined information about language. UG is envisaged as "a distinct system of the mind/brain" (Chomsky, 1986, p. 25), separate from general intelligence, and it is typically regarded as a two-tier system present ab initio: In the Principles & Parameters framework, restrictions on the learning space in language acquisition consist of both a hard-wired basic layer of universal *principles*, applicable to all languages, and a second layer, only partially

wired-in and subject to parametric variation, referred to as *parameters,* to which structural variation between languages are to a large extent attributed. However, the innate knowledge cannot be information about any particular language, because babies can learn all natural languages with equal ease: A Danish baby brought up in England will learn English just as easily as an English baby in Denmark will learn Danish!

Innate constraints are negative in the sense that they sanction certain constructions and hence restrict the hypothesis space that children have to contend with. Hence, rapid language acquisition would not be surprising. It is widely believed that the data for grammar construction available to the child does not include negative evidence (information about which sentences are unacceptable or ungrammatical). Negative evidence could be used by a child to avoid constructing an overly general grammar, but parents usually do not correct their children's errors, and when they do, their feedback is typically disregarded, as illustrated by Vikner's (2005, p. 3) interaction with his 5-year-old son:

(1) Child: Ved du hvor meget jeg drikkede?
Know you how much I drink-ED
Parent: Nej, hvor meget drak du?
No, how much drank you
Child: Først drikkede jeg en hel kop te og så drikkede jeg et glas juice, og så ...
First drink-ED I an entire cup tea and then drink-ED I a glass juice, and then
Parent: Drak du så meget?
Drank you that much
Child: Ja, så meget drikkede jeg
Yes, so much drink-ED I

Genuinely conservative item-based learning in the sense of MacWhinney (2004) would result in children simply parroting back what they hear, and not making the classic errors found cross-linguistically with irregular verbs where children overgeneralize the regular tense-marking, as in the example above with Danish *drikkede*. Thus, "children generalize along some dimensions but not others" (Pinker, 2004, p. 951), but given innate constraints, positive evidence should suffice for language acquisition. Even children with language disorders go through the logical stages of language acquisition, even if they do not attain full linguistic competence by adulthood (see e.g. Gernsbacher, Morson & Grace, 2015). Hence, the

grammatical errors made in e.g. SLI and ASD are not unique to those specific populations, but generally follow a trajectory similar to that found in typical development, qualitatively if not quantitatively.

2.1 The interplay between constraints and statistical learning

The relative contributions of biological endowment and learning in the process of language acquisition is a controversial issue. Chomsky's UG is a theory that relates to the part of language acquisition that hinges on the biological endowment. Infants have been demonstrated to employ statistics in language acquisition (Saffran et al., 1996), and these findings have been employed by Tomasello (2000, 2003) to argue against innateness. However, there is no inherent opposition between the existence of UG and the use of statistical learning (demonstrably based in part on transitional probabilities), as an effective learning algorithm requires a proper representation of the relevant learning data (cf. Yang, 2004, p. 451).

There has been a general consensus in the generative literature that parameter setting proceeds on the basis of "triggering", such that the grammar of the child (or learner, for that matter) is identified with specific parameter values, which are then modified by the input (see e.g. Gibson & Wexler, 1994). However, this "triggering" model faces problems on multiple counts: First, because the linguistic evidence that children encounter in the process of language acquisition is so variable, there is a theoretical possibility that convergence on the target grammar of the local speech community might not happen. Second, one would expect abrupt changes to the child's syntactic production when she switches between grammars. However, this is not what the empirical data suggest; instead, children appear to settle on a specific parameter quite gradually (Yang, 2004, p. 453).

This led Yang (2004) to suggest an account in which the idea of innateness is combined with a model of probabilistic learning, which he calls *the variational model* and, based on a hypothesis space built on UG-defined grammars, principles and parameters, it proceeds as follows (from Yang, 2004, p. 453):

(a) For an input sentence, *s*, the language-acquiring child:
  (i) with probability P_i selects a grammar G_i,
  (ii) analyzes s with G_i,
  (iii) if successful, rewards G_i by increasing P_i, otherwise punishes G_i by decreasing P_i.

In this model, there is selectionist competition between grammars, and only the grammar that best fits the target grammar will survive, eventually eliminating all the other possible grammars made available by UG. How long it takes for a specific parameter value to become dominant is related to the incompatibility of its competitors with the input data, its "fitness value" (Yang, 2004, p. 454). Hence, Yang argues that the triggering model of children's language development must be abandoned and replaced with an account that conjoins the domain-specific space of UG's principles and parameters with domain-general probabilistic mechanisms.

To sum up, according to Yang's (2004) account of the basic mechanisms in language acquisition, variational learning hinges on the cumulative effect of language input on the one hand and UG constraints on the hypothesis space on the other. In addition, his probabilistic approach to parameter setting can be extended to account for mechanisms in language change, as the latter typically proceeds gradually diachronically and offers a foundation for variation synchronically. Thus, Yang's (2004) model comes with the added benefit of accounting for Labov's (2001) "enigma" in sociolinguistics, namely that speakers tend to display great uniformity in the structural aspects of language (including the error patterns), while varying greatly when it comes to other levels of linguistics.

3. UG-compatible errors in typical language development

There is general agreement about the necessity of innate constraints but not about their exact nature and source (Crain, 1991, p. 597). One proclaimed source of evidence for innateness is based on children's non-adult (but UG-compatible) question formation. Crain and Pietroski (2002, pp. 177-182) consider this type of phenomenon a genuine poverty of the stimulus argument. Employing an elicited production task, Thornton (1990) found that about one-third of the 3-4-year-old English-speaking children she studied consistently inserted an "extra" *wh*-word in their long-distance questions, as illustrated in (2):

(2a) What do you think what pigs eat? (Object WH)
(2b) Who did he say who is in the box? (Subject WH)

The emergence of the medial-*wh* in the language of children learning English cannot be explained as a response to the input, as English-speaking adults (who provide the primary linguistic data to children) do not produce medial-*wh* constructions. Although these constructions are not

grammatically well-formed in English, structures like (3) are attested in colloquial (adult) German (see also Müller, 2000, p. 54; Thornton & Crain, 1994):

(3) Wer glaubst du wer nach Hause geht? (Subject WH)
Who think you who to house went
"Who do you think went home?"

The acquisition-related data in (2) and the variational example in (3) are viewed as evidence supporting the idea of successive-cyclic movement, the "stopping over" of a filler undergoing long-distance movement at the left edge of the clause. This is assumed to be a universal property of language, a basic computational principle (Chomsky, 1973, 1986). As suggested by Thornton (1990) and Crain and Thornton (1998), the extra *wh*-phrase in children's questions may be an overt manifestation of a process that appears in French when extraction occurs from subject position. In French, the alternation from *que* to *qui* takes place in subject relative clauses and subject extraction questions. An example of a subject relative demonstrating the necessity of a *qui* complementizer is given in (4) from Rizzi (1990, p. 56):

(4) L'homme que je crois *que/qui viendra (Subject REL)
The man who I think who will come
"The man who I think will come"

The complementizer *que* and its alternating form *qui* both also function as *wh*-words in French. This fact is important in the account given by Crain and Thornton (1998) because their claim is that the medial-*wh* in Child English is also a complementizer, although it is similar to a *wh*-phrase in appearance (see Rizzi, 1990 for a full analysis of the phenomenon in French, and Crain & Thornton, 1998 for an analysis of the English language acquisition data).

The similarity of Child English to a foreign language extends even further. Investigation has shown that lexical (full) *wh*-phrases cannot be repeated in the medial position for both adult Germans and English-speaking children. Finally, children never employed a medial-*wh* when extracting from infinitival clauses, so they never asked questions like (5), and it is not permissible in languages that allow the medial-*wh* either (Thornton, 1990):

(5) # Who do you want who to win?

This complex pattern of linguistic behaviour suggests that many children of English-speakers go through a stage at which they speak a language that is like adult English in many respects, but also one that is analogous to other languages in allowing for the medial-*wh* (cf. Crain and Pietroski, 2002, pp. 177-182). As pointed out by Crain and Pietroski (2001, p. 179), "similarities between child-English and adult-German are as unsurprising as similarities between cousins who have never met". Children acquire a native language by testing wide range of the linguistic options that exist in human languages. However, they do not appear to entertain syntactic structures that would violate the constraints enforced by UG. This is known as the Continuity Hypothesis (cf. Crain, 1991; Crain and Thornton, 1998; Pinker, 1984). English-speaking children make grammatical errors that may exhibit German or Romance syntax in the absence of any evidence for these structures in the primary linguistic data.

These systematic mismatches between child and target adult language are at the core of the theoretical backbone of the stimulus poverty argument and may be the strongest argument for UG, as they demonstrate that children do not simply parrot their input or are inductively determined by it, but instead project beyond their linguistic data. Relating these data to Yang's (2004) variational model, the presence of non-target grammars in the hypothesis space ensures a gradual syntactic development before children settle on specific parameter settings, and this would explain why children appear to only make "principled" errors that correspond to potential grammars (i.e. UG-compatible), such as medial-*wh* in Child English.

A growing body of research suggests that there are many parameter-driven plateaus in domains of syntactic development, apart from the medial-*wh* constructions (see Pierce, 1991 for an overview). In a certain sense, then, children's errors should not merely be viewed as failures to match the target language; at any given time, they are in effect speaking a foreign language (cf. Crain and Pietroski, 2001, pp. 178-181), or a possible, natural human language, rather like the interlanguage in foreign language acquisition. The same appears to apply to children with language disorders (see section 5). These systematicities across typical development and language disorders are not only consistent with the theory of UG but may in fact be considered evidence for it.

4. The non-occurrence of UG violations

At all levels of language, it is hierarchically organized, and the fact that syntactic structure operates on specific types of linguistic representations, namely constituents and phrases, rather than linear strings of words is a classic argument for the innateness of language. Chomsky (1995) proposed that the operation Merge was the Basic Property of language (Berwick & Chomsky 2016), at the core of the formation of linguistic structures. It is basically a principle of recursion, in that it combines two linguistic units, *x* and *y*, forming the composite (*x*, *y*), which may in turn merge with *z* producing ((*x*, *y*), *z*), a hierarchical structure. However, logically speaking, even if recursive Merge is indeed a Basic Property of language, this does not mean that it is necessarily employed as an option in all languages. The theory of UG predicts that language-acquiring children do not make errors that violate innate principles and parameters, and it is a basic tenet of UG that grammatical rules are structure-dependent (cf. Chomsky, 1971, p. 1975). The structure-dependence constraint demands that syntactic derivations operate on hierarchical structure (not linear order) and hence it restricts the hypothesis space of language-acquiring children (cf. Crain and Thornton, 1998, p. 165).

Thus, one of the strongest cases of learning from inadequate evidence discussed in the literature concerns verb-initial positioning in *yes/no*-questions, e.g. *Er han tysker?* "Is he German?", the *yes/no*- question corresponding to the declarative *Han er tysker* "He is German". The formation af such sentence structure is structure-dependent, as it hinges on hierarchical relations: the finite verb (auxiliary or main verb *be*) in the matrix clause is assigned initial position. Chomsky (1971, pp. 29-33) gives these examples:

(6a) The dog in the corner is hungry
(6b) Is the dog in the corner hungry?
(6c) The dog that is in the corner is hungry
(6d) Is the dog that is in the corner hungry?
(6e) *Is the dog that in the corner is hungry?

When transforming the declarative in (6a) into an interrogative question, (6b), main verb *be* is placed in sentence-initial position. Two hypotheses regarding the formation of *yes/no*-questions can be formed on this basis: one, the first (finite) verb in the declarative is fronted, and two, the first

(finite) verb in the matrix clause is fronted. The first hypothesis would incorrectly yield (6e) on the basis of the declarative clause in (6c), while the second hypothesis, based on the structure-dependence constraint, would result in (6d) (cf. Pullum and Scholz, 2002, p. 36).

Employing an elicited production technique, Crain and Nakayama (1987) tested children's knowledge of the structure-dependence constraint. If the structure-dependence constraint is not part of children's innate knowledge and their ungrammatical productions instead constitute misgeneralizations of a structure-independent hypothesis, their errors would be expected to be random. This turns out not to be the case, and the conclusion reached by Crain and Nakayama (1987) was that children's questions provide no evidence that can be incontrovertibly employed as evidence representing violations of the structure-dependence constraint, which they thus assume to be part of UG (cf. Crain and Thornton, 1998, pp. 171-175).

If children initially formed a structure-independent hypothesis when encountering complex examples like (6c), positive evidence would not suffice to prohibit non-local movement, as it could co-exist alongside the local movement option in children's grammars (cf. Yang's variational model). Nonetheless, every language – irrespective of impairments – appears to be imposed with a restriction on non-local movement of the heads of phrases (cf. Travis' 1984 Head Movement Constraint, cf. Crain and Pietroski, 2001, p. 166). Structure-dependence is thus likely an innate constraint, a negative principle that bars certain structures (both in comprehension and production), and children do not appear to adopt grammatical analyses that are not made available by UG.

In sum, a number of language acquisition studies indicate that language-acquiring children do not make errors relating to a range of syntactic structures and dependency relations, not just structure-dependence (Crain, 1991), but also Subjacency (Newmeyer, 1991; Pinker & Bloom, 1990), *that*-trace effects (Chomsky & Lasnik, 1977), the Empty Category Principle (Chomsky, 2001), inter alia (however, see MacWhinney, 2004 for a critical review). In the presumed absence of sufficient evidence in the child's input (the poverty of the stimulus), these linguistic phenomena might hence be assumed to be innate principles. What might be perceived as even more remarkable is the fact that in a variety of language disorders, children make systematic error patterns that match the performances of TD children at an earlier stage in the process of language acquisition.

5. Language impairments in ASD and SLI

The errors that language impaired children make are not random, but are constructed in a manner that appears to follow the basic architecture of the language system (see Fromkin, 1997). Thus, Levi and Kavé (1999, p. 138) suggested that language deficits may be regarded as "a natural laboratory in which linguistic theories may be tested". The performance data that we can gather from genetic developmental language disorders such as those found in SLI and ASD may provide an extended window into both the neurobiological and computational system of language (perhaps even UG), by reflecting primitives of grammar and some of its core properties, and they have the potential of revealing important aspects of syntactic representations in the brain. In addition, data from this field can advance concepts in learnability.

According to the *Diagnostic and Statistical Manual of Mental Disorders 5* (DSM-V, American Psychiatric Association [APA] 2013), ASD and SLI, also known as developmental dysphasia, share the diagnostic trait of poor communication skills. However, in SLI, linguistic deficits and delays are at the core of the symptomatology, whereas language-acquiring children with ASD exhibit immense variability in their language abilities, ranging from absence of functional verbal abilities to fluent speech (cf. Lord et al., 2006). Pragmatic impairments, however, are ubiquitous in ASD and are thus found at both ends of the spectrum (Tager-Flusberg, 2004). Roberts, Rice & Tager-Flusberg (2004), Kjelgaard & Tager-Flusberg (2010) Zebib et al. (2013) have suggested that a subset of ASD children exhibit grammatical impairments that are reminiscent of those found in SLI.

5.1 The selective nature of (morpho)syntactic errors in ASD and SLI

The exact nature of the grammatical impairments in ASD in general is largely undetermined. Early speech production-based studies carried out by Bartak, Rutter & Cox (1975) and Pierce & Bartolucci (1977) indicated that the grammatical competencies of ASD children are parallel to those of typically developing (TD) peers when the two groups are matched on mental age (see Durrleman & Delage, 2016, p. 362). However, later work (e.g. Roberts, Rice & Tager-Flusberg 2004; Zebib et al. 2013) has revealed domain-specific grammatical impairments in the ASD population that appear to be independent of domain-general cognitive deficits. SLI

is a heterogeneous family of language impairments which affects 7% of children (Lely & Pinker, 2014). Recently, the claim has been put forth that a subset of ASD children have a syntactic profile akin to that found in SLI (see e.g. Kjelgaard & Tager-Flusberg, 2010 and Zebib et al., 2013).

Children tend to leave out and/or substitute bound inflectional morphemes in SLI (Levi & Kavé, 1999) and ASD (Tager-Flusberg, 2002); speech in SLI (Leonard, 1995) and ASD (Bartolucci, Pierce & Streiner, 1980) is also characterized by omissions of free function words (e.g. articles, auxiliary verbs and conjunctions). Sentence length and complexity may also be reduced in SLI and ASD (Tager-Flusberg et al., 1990). All of these error types have parallels in typical language development, e.g. as described by Radford (1990) for English, and in Broca's aphasia (Grodzinsky, 2000). Overall, then, children with SLI and ASD (and individuals with other types of language disorders) mirror typically developing children in terms of the error patterns that they exhibit.

Lely (1996) identified a subtype of SLI relating specifically to certain aspects of syntax, morphology and phonology. She termed it Grammatical-SLI (henceforth G-SLI) and Lely & Pinker (2014, p. 586) define it as having "greater impairments in 'extended' grammatical representations, which are non-local, hierarchical, abstract, and composed, than in 'basic' ones, which are local, linear, semantic, and holistic". Lely & Pinker (2014) suggest that G-SLI is related to abnormalities in the left hemisphere. This would fit recent models of the neurobiology of language making a distinction between dorsal and ventral processing streams. As the name suggests, G-SLI does not affect language globally, but locally (or specifically) in certain properties of language, while leaving others intact (Pinker & Lely, 2014, p. 586). More specifically, it has been found cross-linguistically that children with G-SLI have both production and comprehension problems relating to syntactic dependencies in hierarchical structures, e.g. *wh*-questions, relative clauses, passive structures and syntactic embedding, especially if they involve non-canonical word orders (Lely and Battell, 2003; Hamann, 2006). In addition, they omit tense-marking on verbs (Bishop, 1979). In what Lely & Pinker (2014, p. 587) term Basic syntax (or lexical semantics), words are "inserted directly from the lexicon", whereas they have to be "computed by operations such as movement and feature checking" in Extended syntax (see Lely & Pinker, 2014, p. 587 for an extensive overview of studies that have found children in G-SLI having a contrast in performance between Extended syntax and Basic syntax).

Interestingly, a subgroup of ASD children with language impairment exhibit the same pattern, as can be gleaned from the following spontaneous productions of an 11-year-old boy with language delay and low-functioning autism from the Østergaard corpus on the *Child Language Data Exchange Systems* (CHILDES, 2015, see Østergaard, 2016 for more details):

(7) *ASD: Anker (...)skyde tungen derover op i fryseren.
Anker shoot tongue overthere up in freezer-the
*ASD: <og så> [//] indtil da, <så blev> [/] så blev savl frysede.
and then until then then became then became saliva froze
*ASD: og så sidde tunge fast.
and then sit tongue stuck
*ASD: <og så er det nemlig sådan at så har de ehm> [//] og så er det
and then is it right so that then have they uhm and then is it
that they have got an uhm
<at de har fået en ehm>
[//] <at de så> [//] <at ham> [//] at kommer snart med en bil.
that they then that him that comes soon with a car

As exemplified in (7), this ASD child has consistent problems with irregular tense-marking (e.g. *frysede* used as a past participle instead of "frosset"). The infinitival forms *skyde* and *sidde* appear to be inserted directly from the lexicon and are uninflected for past tense (targets would be the irregular forms *skød* and *sad*). In addition, this child does not produce any structures with non-canonical word-order in this example (characteristic of his syntactic profile) and he encounters serious problems with embedding, as is evident from his multiple retracings of the complementizer *at* "that" and the fact that he ends up omitting the subject. The example in (7) is just an illustration of the (morpho)syntactic profile discussed, but it certainly warrants further investigation into the parallels between SLI and ASD (see Nyvad, 2016 for more details), as only a few studies have examined this.

Among these, Riches et al. (2010) found that adolescents with ASD and SLI perform significantly less accurately than TD peers in a sentence-repetition task involving subject and object relatives, such as:

(8a) The thief that ___ robbed the granny (Subject REL)
(8b) The granny that the thief robbed ___ (Object REL)

Both groups (ASD and SLI) tended to make performance errors because they wanted to avoid structures with non-canonical word-order (complex and part of Extended syntax in Lely and Pinker's 2014 sense). In (8a), there is canonical word-order, as the subject of the main verb *robbed* precedes the direct object *the granny*, whereas it follows it in (8b), resulting in increased syntactic complexity. Complex (morpho)syntax requires more processing capacity as it involves more working memory load. For instance, in the object relative in (8b), the filler (the relative element) has to be held in working memory longer than is the case for the subject relative in (8a). However, when matched with a control group in terms of working memory capacity, individuals with G-SLI still appear to experience more problems relating to Extended syntax, according to Lely and Pinker (2014) (see Tager-Flusberg, 1981 and Van der Lely, 1996).

However, an asymmetrical pattern in the performance on subject and object relatives, cf. (8), is by no means unique to SLI and ASD. A great variety of individuals with language impairment have been demonstrated to have a better comprehension of sentences with canonical word-order than those where elements have been displaced. This is also true for Broca's aphasia (Grodzinsky, 2000), Wernicke's aphasia (Bastiaanse & Edwards, 2004), Alzheimer's disease (Grober & Bang, 1995), Down's syndrome (Ring & Clahsen, 2005) and for children who sustain focal brain damage (Dick et al., 2004), especially when it is localized in the left hemisphere (Dennis & Whitaker, 1976) (see Penke, 2015 for an excellent overview).

The neural organization of language can be gleaned through new technologies such as functional magnetic resonance imaging (fMRI), electroencephalography (EEG) and magnetoencephalography (MEG), and so far they indicate that the neural networks supporting Extended syntax is different from the ones that form the basis of Basic syntax. New models of the neural organization of language outlined in Lely & Pinker (2014) offer a more fine-grained picture by transcending the basic distinction between Broca's and Wernicke's areas. Three distinct fronto-temporal networks appear to be related to the processing of syntax. A dorsal pathway seems to be particularly related to Extended syntax, namely one that connects Broca's area (specifically Brodmann area 44) to Wernicke's area (in the posterior superior temporal gyrus) via the arcuate fasciculus. This neural pathway does not mature fully until the child reaches the age of approximately 7. As pointed out by Lely & Pinker (2014, p. 590), "the dorsal pathways in human brains differ substantially from those in other primates, suggesting that phylogenetic changes to the dorsal pathway may have been a key driver of the evolution of language".

SLI and ASD are thought of as separate disorders with distinct aetiologies (cf. Bishop, 2003, p. 214). However, impairments in Extended syntax appear to be common to a subpopulation of both groups, and the dissociation between language and cognition is also found in SLI, which has led a number of researchers to consider whether SLI and ASD are on a continuum (Tager-Flusberg, 2004; Bishop, 2003, 2010).

5.2 Are the linguistic deficits in SLI and ASD on a continuum?

Interestingly, SLI and ASD were considered mutually exclusive diagnoses on the DSM-IV, but they no longer are on the DSM-V (see Durrleman & Delage, 2016, p. 361). This illustrates how the linguistic impairments of SLI and ASD may be considered deficits on a continuum of severity, such that milder cases would only involve problems in syntax whereas both syntax and pragmatics are affected in more severe cases. However, such a view would predict that pragmatic deficits should be manifest in those with the most severe syntactic impairments, and this prediction does not square with the facts: Pragmatic difficulties are ubiquitous in ASD, whereas syntactic deficits are only present in a subgroup (cf. Kjeldgaard & Tager-Flusberg, 2000). In other words, there is a double dissociation between syntax and pragmatics in the two types of language disorder: In ASD, syntax is not uncommonly unaffected while pragmatics is impaired, and in SLI, syntax may be impaired and pragmatics unaffected. This lack of logical dependency between the two levels of linguistics in SLI and ASD suggests that they have "distinct neurological bases" (Bishop, 2003, pp. 219-220). In other words, if you view SLI and ASD as being on the same spectrum, you have to do so for each linguistic level of description in isolation, as the deficits in pragmatics phenotypically are not continuous with the observed impairments in syntax (cf. Bishop, 2003, p. 224).

The symptom-overlap in terms of parallels in syntactic impairment in SLI and ASD may hint at a shared aetiology, but these surface correspondences become even more striking viewed against the backdrop of genetic studies involving relatives of people with autism for whom it is common to exhibit subthreshold symptomatology which resembles SLI (Bishop, 2003, pp. 218-219). The neurological bases for ASD and SLI can thus be envisaged as being distinct, but common aetiological factors may be implicated. Bishop (2003, p. 222) further proposes that there may be genes that "disrupt processes of neuronal migration, leading to abnormal brain structure". The effect of this disruption will be dependent upon which neural networks are involved, and the correlations found in

the symptomatologies of SLI and ASD may thus reflect overlaps in the implicated neural networks. The specificity found in the syntactic profiles of ASD and SLI, reviewed in this section, may be a reflection of an underlying division in the neural and genetic substrates of language (cf. Lely & Pinker, 2014, p. 586).

6. On the mapping of genotypes and linguistic phenotypes

Chomsky argues for the biological model of language development. Given that only humans can acquire grammatical rules, language must partly derive from the genome. Indeed, genetics appear to be able to interfere with language (e.g. genetic region SPCH1, chromosome 7q31). Smith & Tsimpli (1995, p. 31) suggested that the human mind "is equipped with a body of genetically determined information specific to Universal Grammar", and Chomsky (2012) proposes that human language originates from a single genetic mutation (and hence, that it did not evolve gradually through natural selection). However, this theory is complicated by the fact that hundreds of genes (out of a total of approximately only 24,000 in humans) contribute to the development and functioning of the neural substrate of language (Benítez-Burraco, 2009).

It is a truism that our genes "code for a brain that can learn language" (Karmiloff-Smith et al., 2002, p. 312), but the suggestion that there are "grammar genes" in the sense that information specific to the domain of grammar is pre-wired in the genes is highly contentious. The temptation to map genes and cognitive modules 1:1 largely comes from genetic developmental disorders such as SLI, where syntax can be selectively impaired, while other domains of language are seemingly preserved, as described above. However, based on recent research, one might need to be wary of suggesting that there is a gene or even a set of genes for e.g. syntax. The relation between genotype and phenotype is far too indirect and complex, and mapping between specific genes and higher-order cognitive modules such as language in general or grammar in particular is (still) untenable. Ontogenetic development (e.g. biochemical, nutritional and social experience) plays a crucial role at this complex interface, and it may sometimes lead to SLI, sometimes to ASD, and sometimes to an intermediate clinical picture (cf. Bishop, 2003, p. 224). In fact, Karmiloff-Smith et al. (2002, p. 318) point out that, even the discovery of "a gene (*y*) for *x*" (where absence of gene *y* correlates with phenotype *x* in a developmental disorder) does not entail that the absence of *y* is the sole

cause of phenotype *x*. More likely, according to Karmiloff-Smith et al. (2002, p. 318), gene *y* is a component part of a collection of genes coding for molecular processes responsible for constructing the brain.

So far, the development of SLI has been associated with at least four candidate genes and it is believed to be exceedingly heritable, as is the case for ASD. These facts suggest that research into language disorders such as SLI and ASD may provide information about the intricate relationship between nature and nurture on the one hand and the biological underpinnings of language on the other. However, a simple 1:1 mapping with the phenotypic outcome is implausible, because the genes in question may have a number of both cognitive and physical effects (see Karmiloff-Smith et al., 2002). Further, several of these so-called "language genes" are polymorphic in the sense that they may or may not lead to language impairment, depending on which variant is present in the genome. In addition, the same pathogenic allele can lead to different developmental disorders (cf. Benítez-Burraco & Boeckx, 2014). This heterogeneity (both with respect to genetic make-up and symptomatology), also found in connection with ASD, has led Lely & Pinker (2014, p. 586) to recommend that instead of trying to find a direct link between genotype and linguistic phenotype, it would be more fruitful to search for links between "genetic variants with alterations in the neural substrates of subcomponents of language processing". Anatomically speaking, the neural underpinnings of language are difficult to pinpoint, as the functional areas of the brain vary from person to person. These variations are astounding in light of the fact that, in the normal population, and to a more limited extent in the impaired population, they converge phenotypically on the same grammatical system, and the errors made along the developmental path adhere to universal principles.

Talk of a linguistic genotype that is equated with UG conflates nativism with geneticism (cf. Benítez-Burraco & Boeckx, 2014). What is striking, nonetheless, is that language pathologies such as SLI and ASD (but also Broca's aphasia) do not hit random areas of the linguistic system. Quite systematically, they appear to affect inflectional morphology and complex syntax. It may be the case that the neural network implicated in these processes (the neural substrate and the dorsal pathway that supports it) is so spread-out and complex that its sheer intricacy makes it vulnerable, as any disturbance in the system would break it down (for an analysis, see e.g. Nyvad, 2018). These dorsal pathways may, however, also be engaged in computations relating to other high-level cognitive functions. Thus, for

example, Broca's area and its vicinity may be specialized for computations of a hierarchical, complex nature, but these computations may not only be relevant for syntax, but also other cognitive functions. As pointed out by Benítez-Burraco & Boeckx (2014, p. 6), "it seems that it is only their basic architecture that is genetically encoded, while their functional specificities are environmentally driven". The functional variability is, however, fairly confined. As pointed out by Grodzinsky (2010), the areas of the brain that are activated when processing language appear to be relatively uniform across individuals. Benítez-Burraco (2009) states that, at the molecular level, "a core set of genetic cues" are responsible for "the initial wiring of the linguistic brain…in all subjects" (Benítez-Burraco & Boeckx, 2014, p. 6). The specific parallels in linguistic phenotypes can thus emerge from quite diverse brain architectures and genotypes.

To sum up, only certain structures appear in grammars, be they normal or impaired, delayed or broken down. This parallels what is found in language variation and change where only particular elements of the grammar are subjectable to variability. All of this points to the existence of a *genotype grammar* (an underlying grammar, common to all languages, referred to as UG) which leads to *phenotypic grammars* (the observable variations in grammars – typical or atypical - in the world's languages). The study of language disorders is a new frontier of research which can be a powerful tool to help us understand the biological underpinnings of language. The linguistic description of SLI has been advanced significantly by theories grounded in UG, and the next step is to carry out investigations into ASD while applying the same theoretical apparatus. As pointed out by Lely & Pinker (2014, p. 593), future research has to take an interdisciplinary approach that takes full advantage of the fine-grained analyses offered in linguistics, instead of coarsely mapping genotype (genetic variants) directly unto phenotypes (overall language impairment), as is largely the case in the extant literature on language disorders.

7. Conclusion

Gene expression and experience (both linguistic and non-linguistic) interact in the development of grammar. By adulthood typically-developing language users have mastered a rich and complex linguistic system. However, while adhering to e.g. structure-dependence, children's grammars deviate from adult grammars in intriguingly systematic and constrained ways – both in typical and atypical development, which would

be surprising if there are no underlying restrictions in the hypothesis space forming the patterns. Any theory of language acquisition, be it relating to typical development or language disorders, should thus provide an account of why children project beyond their experience in certain ways but not in others. While these deviances from adult grammar and the selective nature of syntactic impairments across language pathologies is not indisputable evidence for the existence of UG, it is surely a strong case for it. Especially in light of the fact that certain genetic mutations in language disorders are associated with these specific linguistic patterns.

That children use statistical information should be viewed against backdrop of them knowing what linguistic units are relevant and important for cracking the code of the specific language that they are acquiring (be it stress patterns, segments, word classes, syntactic dependency relations, etc.). An explanation of the data from ASD and SLI in terms of statistical learning in the absence of innate constraints would have to (implausibly) assume that these children (and adults) have a deficiency in their ability to extract knowledge from statistical regularities in their data.

There is a continuum concerning the degree to which individuals make errors in performance that cuts across the divide between impaired and unimpaired language (cf. Penke, 2015). It thus appears that the grammatical errors in language disorders like SLI and ASD are gradable and not qualitatively different from those found in typically-developing children. Without taking away the importance of UG, the variability in the performance data from language disorders may in part be explained with reference to processing capacities, such that limitations on working memory or short-term memory can impede the extraction of (morpho-) syntactic information when the latter is complex. This type of account may be able to capture the gradience in performance within and across language disorders (as well as within and across typical language), as variability in processing load may engender variability in performance. In addition, we expect people with e.g. ASD to make significantly more mistakes than neurotypical children, simply due to cognitive constraints that are domain-general, rather than domain-specific.

However, while this may help explain the gradience observed, it cannot answer for the systematicity in the error patterns without appealing to syntactic representation and perhaps ultimately innate constraints. The characteristics of the errors found in the performance of language-acquiring children and individuals with a wide range of acquired and developmental deficit syndromes (like ASD and SLI) all appear to involve the uppermost

projections of the syntactic tree (i.e. the left periphery of the clause). The latter has a crucial role in the comprehension and production of syntactically complex sentences with e.g. non-canonical word-order or embedding. If G-SLI and the subset of ASD children exhibiting syntactic impairments have a shared aetiology, the linguistic phenotype would be expected to be more or less identical. Demarcating what exactly it comprises might not only lead to improvements in training methods, but more fundamentally strengthen our understanding of how genes affect brain circuitry in healthy and pathological language profiles. Today, a lot of research is dedicated to integrating the formal/theoretical approach with behavioral/experimental studies, and future research must employ a fine-grained analysis of how distinctive linguistic components correlate with anatomical and functional aspects of the human brain. Whether there is an underlying neural deficit that manifests itself in this relative uniformity across disorders is still an open question.

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