To appear in Ian G. Roberts (ed.), *The Oxford Handbook on Universal Grammar*. Oxford: Oxford University Press.

Language Pathology

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Universal Grammar (UG) denotes the species-specific faculty of language, presumed to be invariant across individuals. Over the years, it has shrunk from a full-blown set of *principles and parameters* to a much smaller set of properties, possibly as small as just containing the linguistic structure-building operation *Merge*, which in turn derives the uniquely human language property of *recursion* (Hauser et al., 2002). UG qua human faculty of language is further assumed to constitute the "optimal solution to minimal design specifications" (Chomsky, 2001: 1), a perfect system for language. Unfortunately, the human system or physiology does not always run perfectly smooth in an optimal fashion. There are malfunctions, misformations, and other aberrations throughout. The language system is no exception. This chapter will present language pathology from the perspective of the underlying system: What can non-intact language tell us about UG?

19.1 Introduction

Pathology has long played an important role in the study of language in general, and the human faculty of language in particular. Modern linguistics has benefited greatly from insights gained through investigating language breakdown, for example, supporting the original theory of movement and theta theory with evidence from agrammatic aphasia (trace deletion: Grodzinsky, 1986, 1990, 1995) or employing some postulated mechanics to first language acquisition (truncation/tree pruning: Ouhalla, 1993; Rizzi, 1994; Friedmann and Grodzinsky, 1997; Friedmann, 1998, 2002). That is, theoretical implementations into analyses of impaired language utilized and thereby buttressed linguistic theory; and at the same time, the types of impaired language found in different pathologies across languages and the patterns attested—as well as those not attested, often ruled out by the theory—were taken to be the direct workings of what is possible in human language, and what is not (UG, principles and parameters, and so on). In other words, the linguistic study of language pathologies has long held one constant: the invariant human faculty of language.

Working on this assumption, data fed theory and vice versa. However, it is now time that the leverage of investigations of pathological language and cognition be raised. Future research could perhaps steer the focus of its potential contribution to finding out more about the underlying language faculty. We take the central questions about such a contribution of research on language pathology to research on Universal Grammar to be the following:

(A) Can pathology affect the core of language abilities (i.e. the use of universal operations and primitives) or does pathology restrict language-specific properties (giving rise to optionality or variability)? (In other words, is there any evidence that language operations such as external or internal Merge are unavailable or otherwise impaired in any population with language pathology?)

(B) Does language pathology affect linguistic competence (in language-specific options) or does the variability in language use depend on accessing this knowledge due to affected mechanisms mediating language use (such as working memory resources)?

(C) Does language pathology affect language use differently depending on whether we are dealing with an acquired or a developmental language disorder? (Associated with this question: Could we distinguish between language use vs. language knowledge issues as a function of acquired vs. developmental language disorders?)

(D) Can we disentangle the contribution of language-external factors in human cognition such as executive control or in the environment, such as input frequency of a particular token, type, or structure from language-impaired performance in developmental or acquired language disorders?

We will address each of these questions as we go along. The chapter is structured as follows. Section 3 surveys research on developmental language disorders, with emphasis on specific language impairment, where we will try to find some answers to questions (A) and (D) in particular. The study of linguistic savantism, introduced in section 4, addresses the interplay of language and cognition in the context of language pathology, focusing on question (D). Section 5 singles out the most widespread acquired language disorder, aphasia, which allows us to address all of (A)–(D). Finally, section 6 sketches paths towards a comparative biolinguistics through phenotypic comparisons, which also provides an outlook for future research, especially on language pathology as a window into variation in UG and the human faculty of language, respectively. The chapter will be briefly concluded in section 7. But first we provide some background on linguists' conceptions of the human faculty of language, complementing many other contributions to this handbook, and relating these to impaired language; this section also contains suggestions for further investigations that are beyond the scope of this chapter.

19.2 Impaired language and the invariance of the language faculty

Theoretical assumptions regarding UG, and whether or how it is affected in developmental or acquired language impairment, have also raised issues about the links or dissociations among speech, language, and communication disorders. This is because human communication can include speech (the sensorimotor system: the spell-out of communication and the input to language perception), language (the elementary tools: words, syntactic rules, sentence meaning), and pragmatics (appropriateness: why a particular communication/social act happens at this time and place). The distinction among the three terms is fundamental in diagnosis, intervention, and prognosis for clinical practice and research, although the tacit assumption is that impairment on speech, language, and communication is rarely selective, affecting more than one of the three abilities (Bishop and Adams, 1989; Bishop, 1990, and much research since, recently summarized by Benítez-Burraco, 2013). For example, specific language impairment is often co-morbid with dyslexia (Smith et al., 1996; Catts et al., 2005), speech-sound disorder (Shriberg et al., 1999), or autism spectrum disorders (Norbury, 1995; Tager-Flusberg, 2006).

It is thus usually the case that when speech impairment is diagnosed as the main area of deficit, communication problems ensue. Similarly, when problems in expressive language are identified, impairment will also affect communication as well, albeit not as the prime cause. Considering a diagram such as the one depicted in figure 1, it is obvious that interactions between various components of speech, language, and communication are not only possible but perhaps inevitable as well.

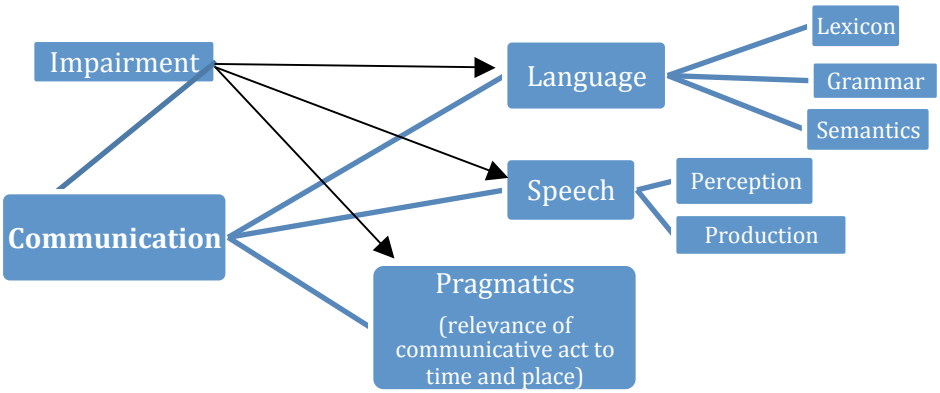

Figure 1: Impairment of communication for speech, language, and pragmatics

In view of the general question we seek to address in this chapter, namely the link between the contributions that a UG-based framework can make to language pathology, theory, and practice (and vice versa), we suggest that, with request to questions (A)–(D) above, two main issues arise from the picture in figure 1. The first has to do with the status of UG and its assumed location in this diagram, which the remainder of this section will largely deal with. The second issue concerns the possibility of 'selective' impairment in speech, language, and communication in already known types of language disorders given the intricate, and inevitable, interactions among them in the process of language production and processing (linguistic performance), the topic of the following sections. To complicate the picture further, linguists have successfully shown that 'language' is more of an umbrella term, including various levels of linguistic analysis such as morphology, phonology, semantics, and syntax which in turn are fed into by a lexicon viewed as an interface component *par excellence*: Abstract feature specifications of a morphosyntactic or semantic nature interface with input from the phonological, semantic, syntactic, and encyclopedic levels allowing pragmatics, speech, and verbal communication to contribute (or be contributed to).

Thus, in language impairment, both developmental and acquired, dissociations are found among levels of linguistic analysis, too—not only among speech, language, and communication as such. For instance, lexical abilities dissociate from morphosyntax in individuals with specific language impairment (cf. van der Lely and Stollwerck, 1997; Tomblin and Zhang, 2006) or with aphasia, exhibiting word-finding problems but not morphosyntactic problems and vice versa (cf. Grodzinsky, 2000; Mendez et al., 2003). Similarly, in types of dementia, lexical access problems are attested with no or minimal morphosyntactic problems (Hodges et al., 1992). In autistic individuals, problems with phonetics–phonology dissociate from problems with grammar (morphosyntax) and communication deficits (pragmatics). Specifically, large variation is found in individuals with autism spectrum disorders insofar as language impairment is concerned (Kjelgaard and Tager-Flusberg, 2001), distinguishing between Autism with Language Impairment and Autism with Normal Language (Bishop, 2002; De Fossé et al., 2004; Hodge et al., 2010; Tomblin, 2011), while phonetics and phonology are usually spared. On the other hand, impaired pragmatics and social skills are assumed to be universal characteristics of autistic individuals, evincing a communication impairment. (We concentrate on lexical and morphosyntactic issues and do not cover the relevance of semantics for language pathologies from a UG perspective in this chapter, but for a current review of relevant issues and additional references, see e.g. Hinzen, 2008 as well as Tsoulas, this volume; likewise, for details on phonology and UG, see Miller et al., this volume.)

This complex picture of dissociations in language pathology in seemingly diverse populations has been used to argue against a UG-approach to language: The complex interactions between different levels of linguistic analysis with communication and speech are claimed to illustrate the non-autonomous nature of language, arguing against a mental organ with that name. What we hope to show, however, is that these complex and highly intricate patterns of linguistic behavior in developmental and acquired language disorders allow us to disentangle the locus of UG-driven problems from other, interacting components (speech and pragmatics on one hand, non-verbal cognition on the other) which lead to impaired communication skills in diverse populations.

When asking question (A), then, whether a given pathology can affect the core of language abilities, one important assumption must be kept in mind: The study of human language as set out from the earliest days of generative grammar (Chomsky, 1955, 1956, 1957)—whether it involves a Language Acquisition Device (Chomsky, 1965), a Faculty of Language (Chomsky, 1975), or Universal Grammar (Chomsky, 1981a) to capture the species' tacit, underlying 'knowledge of language' (Chomsky, 1981b, 1986)—can only be successful if we assume invariance across the species. If we permit for healthy, typically developing individuals to acquire language with the help of different faculties, then there is no constant against which differences and, possibly more importantly, similarities observed in the acquisition and subsequent development process could ever be evaluated (see also the chapters by Guasti, Huang and Roberts, Lasnik and Lidz, Lohndal and Uriagereka, and Rizzi, this volume, as well as many discussions in Norbert Hornstein's *Faculty of Language* blog at http://facultyoflanguage.blogspot.com).

Looking back on the past six decades of generative research and the core assumptions that have been put forth concerning UG and the language faculty, it becomes clear that such tacit, underlying *knowledge of language* is indeed a significant consideration. Take the quote that starts off the chapter by Rizzi (this volume), the opening sentence of Chomsky (1973: 232): "From the point of view that I adopt here, the fundamental empirical problem of linguistics is to explain how a person can acquire knowledge of language" (republished in Chomsky 1977: 81). This conceptualization predates Chomsky's (1986) five questions on the knowledge of language, the first three of which already appeared in Chomsky (1981b), which we will return to presently. And it ties in with longer held assumptions about language acquisition:

> Having some knowledge of the characteristics of the acquired grammars and the limitations on the available data, we can formulate quite reasonable and fairly strong empirical hypotheses regarding the internal structure of the language-acquisition device that constructs the postulated grammars from the given data. (Chomsky, 1968: 113)

Specifically, UG is "a hypothesis about the initial state of the mental organ, the innate capacity of the child, and a particular grammar conforming to this theory is a hypothesis about the final state, the grammar eventually attained" (Lightfoot, 1982: 27). UG is thus understood as "the biological system that accounts for the different individual grammars that humans have", though "not to be confused with the theory of grammar itself; rather UG is an object of study in the theory of grammar" (Ludlow, this volume). More explicitly, UG "is taken to be a characterization of the child's pre-linguistic initial state" (Chomsky, 1981a: 7) and—especially in the Principles-and-Parameters era of generative grammar (Chomsky, 1981a, 1986; cf. Chomsky and Lasnik, 1993)—consists of "a system of principles with parameters to be fixed, along with a periphery of marked exceptions" (Chomsky, 1986: 150–151).

In this context, the distinction between 'core grammar' and 'peripheral grammar' is introduced. While the latter "is made up of quirks and irregularities of language," "core grammar entails a set of universal principles, which apply in all languages, and a set of parameters which may vary from language to language" (Gentile, 1995: 54). As Gentile continues, "[t]he theory of UG must observe two conditions" (p. 54):

> "On the one hand, it must be compatible with the diversity of existing (indeed possible) grammars. At the same time, UG must be sufficiently constrained and restrictive in the options it permits so as to account for the fact that each of these grammars develops in the mind on the basis of quite limited evidence … What we expect to find, then, is a highly structured theory of UG based on a number of fundamental principles that sharply restrict the class of attainable grammars and narrowly constrain their form, but with parameters that have to be fixed by experience." (Chomsky, 1986: 3–4)

To the extent that the core/periphery distinction is still valid, one way of approaching language pathology might be to separate impaired competence and performance into these categories, and to then focus on core grammar as a window into a possibly compromised UG. Having said that, though, it becomes immediately obvious that it is often in the grammatical periphery that we see revealing problems. For example, discourse-driven structures such as topicalization or dislocation go hand in hand with subtle interpretative differences which might not be picked up by individuals who experience problems in the pragmatics component. Preempting our brief presentation of the architecture of grammar below, this might indicate impairment of the syntax–pragmatics interface, for example, going well beyond core grammar. Approaches such as the Interpretability Hypothesis (Tsimpli, 2001) and others are sensitive to this aspect of impaired language, and more recent research ties such problems to other cognitive deficits (addressing questions (B) and (D)); unfortunately, this has not yet been systematically investigated across the board (therefore not allowing a definite answer to (C)).

Another way to tackle questions (A)–(D) would proceed along the aforementioned 'five questions' familiar from current perspectives on the field of biolinguistics, the study of the 'biological foundations of language' (Lenneberg 1967). Expanding on Jenkins (2000), Boeckx and Grohmann (2007) connected Chomsky's (1986) five questions on 'knowledge of language' to Tinbergen's (1963) four questions on 'the aims and methods of ethology'. Boeckx (2010) is a more recent attempt to flesh out this research program in (text)book length, and the five questions have each been dubbed specific 'problems', denoting their possible intellectual origin (from Leivada 2012: 35–36):

(1) What is knowledge of language? (Humboldt's problem; Chomsky, 1965)
(2) How is that knowledge acquired? (Plato's problem; Chomsky, 1986)
(3) How is that knowledge put to use? (Descartes's problem; Chomsky, 1997)
(4) How is that knowledge implemented in the brain? (Broca's problem; Boeckx, 2009)
(5) How did that knowledge emerge in the species? (Darwin's problem; Jewett, 1914)

There is a direct relationship between the five questions and language pathology. While (1) is usually investigated from the vantage point of healthy, adult speakers, impaired language may give us further clues as to which aspects of language belong to the realm of tacit, underlying knowledge of language. Likewise, looking at developmental language impairments may tell us more about (2), in addition to studying typical language development. The exploration of (3) would gain tremendously from the examination of

language-impaired populations, especially for a yet outstanding unified competence–performance model of language, possibly one that also encompasses the above-mentioned aspects of speech and communication. (4) is a very obvious concern for language pathology, especially impaired language that is the result of damage to the brain. And (5), lastly, which has recently been redubbed 'Wallace's problem' (Berwick and Chomsky, 2016), is the question that not only opens up so much room for speculation in normal language; it also concerns language pathology that arises from genetic malfunction, for example. However, each of these questions merits a chapter of its own with respect to language pathology; moreover, the relationship to UG as such may be more indirect at this point and not as clear-cut as one would wish for. So, rather than pursuing this route (briefly returned to in section 6), we will proceed with more direct consequences of the study of impaired language for UG.

19.3 Developmental language impairments

Although generative researchers admit that some variation in language abilities can be found among monolingual healthy adults, particularly in areas of language that belong to the periphery or are primarily affected by education levels (larger vocabularies, more complex structures), the core of language knowledge is supposed to be shared by native speakers of the same language (Chomsky, 1955). Continuing along the lines sketched above, developing knowledge of language is the outcome of typical first language acquisition which shows overwhelming similarities across children and across languages, despite variation in the pace of development within predictable time limits (Chomsky, 1981a).

Based on research of typical first language acquisition, researchers have concentrated on different stages of development looking for triggers in the input, as well as poverty of the stimulus arguments which downplay the role of the input and appraise the role of the innate endowment (Lightfoot, 1991; Poeppel and Wexler, 1993; Crain and Thornton, 1998; Anderson and Lightfoot, 2002; Han et al., 2016; see also the chapters by Guasti, Lasnik and Lidz, and Rizzi, this volume). Since Lenneberg (1967), generative research on first language acquisition has explicitly assumed that during the preschool years, the core of grammatical knowledge has been acquired by the typically developing child, and that this is to a certain extent determined by human genetics. The special status of the faculty of language as a partly autonomous computational system in the human brain was thus associated with a developmental pattern that was specific to that system, biologically determined and unique to the species. The time frame in which language development takes place is consistent with this innate predisposition for language (Guasti, 2002).

Since the early 1980s, the generative approach to language acquisition inspired and was inspired by studies on developmental language disorders, with particular interest focusing on a type of language disorder revealing a strong asymmetry between an unexpectedly delayed or deviant profile of language development in the absence of any hearing loss, cognitive, emotional, social, or neurological damage (Leonard, 1989, 2014; Levy and Kavé, 1999). Although this type of developmental language disorder has been referred to under different names, specific language impairment (SLI) is the term mostly used in the literature. In early work on SLI, emphasis was placed on the considerable delay in the emergence of language use as well as in lexical and morphosyntactic development both in comprehension and production. A debate concerning the domain-specific (i.e. language-specific) or a domain-general deficit in SLI has been central in the literature, usually creating a boundary between generative and non-generative approaches to SLI (cf. Clahsen, 1989; van der Lely and Stollwerck, 1996; Leonard, 1998; Bishop et al., 2000; van der Lely et al., 2004; Ullman and Pierpont, 2005; Bishop et al., 2006; Archibald and Gathercole, 2006; Leonard et al., 2007).

In the extensive research on SLI that has been conducted since, the question about the domain-specific vs. domain-general deficit has become considerably more refined in view of the heterogeneity of SLI and the variation that can be found across subgroups in terms of the areas of language that seem to be more or less affected. For instance, van der Lely et al. (2004) argued for a particular type of SLI, referred to as G(rammatical)-SLI, for children who unlike other SLI groups show a deficit in grammatical ability and core domains of grammar such as hierarchical structures and computations of dependencies (van der Lely 1998, 2004). Other generative approaches argued for a deficit or a delay in the acquisition of or access to specific grammatical features, agreement processes, or computational complexity of the syntactic derivation (Jakubowicz et al., 1998; Wexler et al., 1998; Tsimpli 2001; Clahsen 2008; Rothweiler et al., 2012). Some of these proposals may in fact be taken to support an affirmative answer to question (A)—for example, that the operation Move is impaired—but more recent considerations suggest an interplay of different factors, moving towards the direction of question (B), such as the role of working memory resources on the syntactic computation. Regardless of whether we are dealing with an acquired or a developmental language disorder, disentangling the contribution of language-external factors, then, has become one principal issue of experimental concern (cf. (D)).

Assuming that there are indeed discrete sub-types of SLI (van der Lely, 2005; Friedmann and Novogrodsky, 2008), such as lexical SLI, phonological SLI, or syntactic SLI, these sub-types ideally signal an impairment at the relevant interface level. Thus, viewed against a typical minimalist architecture of the sort displayed in figure 2, PhonSLI would indicate some impairment on the interface between language-internal Phonetic Form (PF) and language-external sensorimotor system (SM), SemSLI and perhaps PragSLI as well between Logical Form (LF) and the conceptual-intentional system (CI), LexSLI between the mental storage unit and the computational component, and so on.

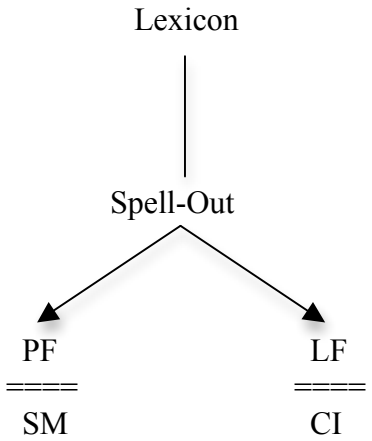

Figure 2: A minimalist architecture of the grammar

Arguably the most significant advantage of an interface approach is the potential to tease apart language-internal (linguistic) from -external effects (cognitive, motor, etc.), and SLI sub-types are predestined to provide the relevant evidence. More detailed investigations are in order, though, which might also bear on the distinction between the faculty of language in the broad vs. the narrow sense (cf. Fitch, 2009; Balari and Lorenzo, 2015; and the more programmatic Barceló-Coblijn et al., 2015), something we will return to below. After all, it is generally assumed that PF and LF form part of the language faculty (first-factor properties), while SM and CI refer to corresponding neural structures and operations in the brain (relating to the third factor).

Of course, the devil lies in the detail. Coming from a separationist perspective such as Distributed Morphology (Halle and Marantz, 1993; Marantz, 1997; Arregi and Nevins, 2012), for example, one could hold that there is no discrete component "Lexicon" as such; in that case, the impaired interface could denote the link of Vocabulary and/or Encyclopedia with the computational system. The point is that conceptual and empirical knowledge could work better hand in hand, across disciplines, and inform *conceptual* theory (confirming the architecture, for example, or requiring revisions)—just as we apply theoretical advances to impaired language data and the other way around.

An important aspect of SLI research which illustrates the mutually beneficial relation between linguistic theory and the study of developmental language disorders is the large number of studies investigating lexical, grammatical, semantic, and pragmatic properties of different languages in the performance of children with SLI. The motivation is both theoretical and clinical. Efforts to identify the language-specific, vulnerable domains in atypical language development can lead to the evaluation of alternative approaches to SLI—but also to the evaluation of typological generalizations favored by linguistic theory concerning the nature and status of features, structures, and derivations. From a clinical perspective, identifying the vulnerable domains for each language which induce extensive delays in the language development of children with SLI helps speech and language therapists isolate diagnostic markers and design appropriate interventions.

The cross-linguistic study of children with SLI has revealed that the patterns of acquisition which distinguish among typically-developing monolingual children also distinguish among children with SLI speaking those languages. For instance, the earlier acquisition of finiteness by French compared to English monolingual children is also attested in French- and English-speaking children with SLI (Rice and Wexler, 1996; Conti-Ramsden et al., 2001; Jakubowicz and Nash, 2001; Thordardottir and Namazi, 2007; Hoover et al., 2012). Similarly, pronominal object clitics have been used as diagnostic markers for French-speaking children with SLI as they are particularly prone to omission (for recent summary, see Varlokosta et al., 2015), while in Cypriot Greek enclitics do not seem to be problematic for children with the same diagnosis (Petinou and Terzi, 2002; Theodorou and Grohmann, 2015). This distinction parallels the difference in the precocious development of enclitics in Cypriot Greek-speaking typically developing children compared with French monolingual L1 data (Grohmann et al., 2012). The value of cross-linguistic studies on children with SLI also extends to the comparison between children with SLI speaking different varieties of the same language. Thus, for children with SLI speaking Standard Modern Greek, a language with proclisis in finite contexts, the evidence is conflicting, with many studies showing clitic omission in production or processing as well as problems with clitic comprehension and some with no omission problems in production (cf. Tsimpli and Stavrakaki, 1999; Tsimpli, 2001; Tsimpli and Mastropavlou, 2008; Stavrakaki and van der Lely, 2010; Manika et al., 2011; Chondrogianni et al., 2015).

More recently, research on SLI has included bilingual or dual language children with the aim of identifying similarities and differences between the developmental patterns and outcomes of typically developing bilingual learners from monolingual and bilingual children with SLI (Paradis, 2007). This research is also largely driven by two opposing views: the domain-specific account of SLI, whereby language is the primary domain of the deficit, and the domain-general account, according to which general processing and conceptual deficits are responsible for problems in non-verbal cognition and for the profound language problems in children with SLI (Leonard et al., 1992; Miller et al., 2001; Kohnert and Windsor, 2004). For the latter account, learning a second language is expected to cause some type of double-delay in children with SLI (e.g. Steenge, 2006; Orgassa and Weerman, 2008), according to which bilingual children with SLI will be outperformed both by typically developing

monolingual and bilingual children but also by monolingual children with SLI. In recent years, many studies on different combinations of languages in bilingual children with SLI have been carried out and shed light on the debate between the domain-specific and the domain-general nature of SLI as well as on the question of whether bilingualism affects in either a positive or negative way the phenotypic profile of SLI.

Many recent studies on bilingual children with SLI converge in showing that bilingualism either causes no additional deficits in their linguistic performance or improves some aspects of it (Paradis et al., 2003; Paradis, 2010; Armon-Lotem, 2012; Kambanaros et al., 2013, 2014; Tsimpli et al., 2016). One example, based on research on the lexicon in a multilingual child with SLI, revealed that there was no marked difference in lexical retrieval abilities across her three spoken languages (Kambanaros et al., 2015). Although research on bilingual children with SLI is still limited, the findings concerning the absence of further deficits in their language ability not only supports a domain-specific approach to SLI but also suggests that whatever language deficit is involved in children with SLI, it does not seem to be so severe that it prevents children from developing a second language. From the generative perspective, this conclusion implies that SLI does not affect the basic properties and operations of the language faculty itself (i.e. a negative answer to question (A)), but instead delays development so extensively that maturational instructions associated with sensitive or critical periods for language development can, ultimately, become inoperative (Rice et al., 2009).

Even if our current state of knowledge about SLI does not allow us to offer insights into the precise structure of UG, there is an obvious theoretical import that concerns the interface-driven architecture of grammar and the minimalist program of linguistic research. Furthermore, the study of developmental language disorders in relation to bilingualism and in relation to the co-morbidity they present with other processing, cognitive, or socio-emotional deficits enriches our knowledge by directing research to the investigation of the (behavioral and neurological) links between language and other aspects of human cognition.

19.4 Linguistic savantism

When concentrating on language impairment and, in particular, developmental language disorders from the perspective of a UG-based approach, we ask questions about the levels, operations, features, or derivations that are affected, leading to deviant output. At the same time, we ask questions about the contribution of non-linguistic, cognitive systems (memory, executive functions, general intelligence) to language use and deviant language outputs (cf. question (D) above). The underlying motivation for both sets of questions is the *asymmetry* observed: An individual with SLI appears to have impaired language in the absence of other, major cognitive deficits in general intelligence, emotional development, or social skills. Thus, language impairment in individuals with SLI reflects an asymmetry between language ability and other (non-linguistic, cognitive) abilities. The special status of language as a mental organ, autonomous and encapsulated from other aspects of cognitive abilities, can also be examined from asymmetries with the opposite pattern: impaired communication, emotional, and executive control abilities with language being unaffected or, more strikingly, a *talent*. This asymmetrical pattern has been extensively studied in the case of a polyglot-savant, Christopher (Smith and Tsimpli 1995; Smith et al., 2010).

To appreciate the status of a language as a talent in an individual with otherwise impaired cognition, we will briefly present *savantism*: a profile built on an atypical and strongly asymmetrical pattern of behavior (Tsimpli, 2013). In savants, verbal and non-verbal performance is at or below the lower end of the average scale, and communication skills are severely deficient. In contrast, savants excel in some special skill; music, mental arithmetic

and calendrical calculations, drawing or sketching (Hermelin, 2002). Importantly for the status of language, as noted by Howlin et al. (2009), savants are more frequently found in individuals with autism spectrum conditions than in any other group. Moreover, the majority of savants reported in the literature is autistic.

Autism does not present a single profile; it is a spectrum of communication disorders associated with impairments in three major domains: social interaction, social communication and imagination, and a restricted repertoire of activities and interests (Wing, 1997). Deficient social interaction is perhaps the defining feature of individuals with autism spectrum disorders (ASD). As suggested in the previous section, problems in social interaction can dissociate from linguistic and cognitive abilities. Linguistic abilities show considerable variation across individuals with ASD (Kjelgaard and Tager-Flusberg, 2001), ranging from non-verbal individuals to those with normal language skills. Of the autistic children with some but not normal language ability, many exhibit language impairments affecting both vocabulary and morphosyntax.

There are different cognitive approaches to autism, some of which offer alternative explanations and others complementary ones. One of the dominant views of autism involves the notion of *mind-blindness* (Baron-Cohen, 1995), which views autism as a deficit in theory of mind. Theory of mind can be understood as the ability to interpret other people's intentions, beliefs, and attitudes through interpreting discourse, context, facial expressions, and body language. The theory of *weak central coherence* (Frith, 1989) views autism as a deficit in the integration of relevant pieces of information drawn from perceptual evidence and/or from previous knowledge, preventing the autistic individual from seeing the 'big picture' and allowing him to focus on the detail. Thus, the typical autist shows excellent attention to detail in terms of input processing and memory of detail and patterns. The more recent version of weak central coherence suggests that this attention to detail is due to a local processing bias. A third approach to autism suggests that the deficit lies in executive functions leading to limited or no cognitive flexibility (Ozonoff et al., 1991). Among other things, individuals with ASD show an inability to abandon decisions or misguided interpretations of actions as a result of this executive dysfunction.

All three approaches concentrate on the deviance of autistic behavior from the neurotypical individuals and attempt to account for it on the basis of some deficit: in theory of mind, in weak central coherence, or in executive functions. Linking these accounts to language impairment, we expect weak central coherence to be closer to an account of communication, and pragmatics in particular, as making sense of the global picture allows individuals to interpret non-literal and figurative language, among other things. However, weak central coherence would not necessarily account for the co-morbidity of autism and language impairment in a mild or a severe form. Executive dysfunctions would also predict problems with communication and coherence both at the level of language but also of non-verbal cognition. Variation in the patterns of language impairment in individuals with autism still remain largely unpredictable on this type of account. A deficit in theory of mind would account for social and communication skills, problems with figurative and non-literal language, and problems with social interaction. Although it has been suggested that good theory of mind presupposes a certain level of language development, such as lexical abilities (Happe, 1995) or subordination (de Villiers, 2007), the fact that complex theory of mind skills may be missing even when language abilities have reached the expected threshold of abilities indicate that language is necessary but not sufficient.

Overall, all three approaches seek to account for the main cognitive cause of autism and the way in which individuals with ASD diverge from the norm. Nevertheless, the fact that savantism and autism appear to frequently co-occur challenges the above theories: What is the link between 'talent' and autism? Baron-Cohen et al. (2009) analyze the excellent

attention to detail, a characteristic of individuals with ASD regardless of whether they are high- or low-functioning, as *strong systemizing*. Systemizing is a higher cognitive ability which helps the extraction of patterns, rules, and generalizations from perceptual input. Autistic individuals are good at extracting repeated patterns in stimuli, while strong systemizing is also evident in their actions and overall (verbal and non-verbal) behavior.

Baron-Cohen et al. (2009) suggest that savants exhibit a stronger version of the common autistic trait of strong systemizing: sensory hypersensitivity, hyper-attention to detail, and hyper-systemizing. Sensory hypersensitivity concerns low-level perception, aspects of which (tactile, visual, auditory) seem to be enhanced in savants. Hyper-attention to detail is necessary for extracting the detailed ingredients of an association; these enter into *modus-ponens* ($p \rightarrow q$) patterns from a complicated and highly integrated input. Hyper-systemizing constitutes a basic inferential ability which, if meticulously applied to perceptually accessible systems, leads to excellent knowledge of the system's operations and categories. A memory system dedicated to the domain of hyper-systemizing in the savant enables the build-up, storage, and retrieval of a highly sophisticated and detailed system of knowledge in this cognitive domain. This is reminiscent of Ericsson and Kintsch's (1995) proposal of a long-term working memory for experts in a specific field, such as professional chess players; long-term memory is an ability that "requires a large body of relevant knowledge and patterns for the particular type of information involved" (*ibid.*, p. 215). It would seem appropriate to characterize hyper-systemizing as a possible development of a trait in typical individuals with high expertise in a particular domain. Music, arithmetic, and fine arts (drawing, sculpture) are highly complex systems appropriate for the savant hyper-systemizing mind. Most savants presented in the literature excel in one of these systems. The question now turns to language.

The complexity of human language is usually based on operations such as recursion and discrete infinity which characterize other aspects of human cognition, such as music and mathematics (Berwick and Chomsky, 2011, 2016). At the same time, language enjoys a privileged position in the human mind as a vehicle of thought and, secondarily, communication. Since autism typically involves a deficit in social interaction and communication but also a deficit in thought processes, language is not expected to be a domain of hyper-systemizing in savants (cf. Howlin et al., 2009). Christopher is a unique case of a polyglot-savant (Smith and Tsimpli, 1995; Smith et al., 2010, and references therein).

Christopher is unable to look after himself, and he has thus been institutionalized in his adult life. He is severely apraxic with visuo-spatial deficits, very poor drawing skills, and poor motor coordination. His working memory ability is poor but, at the same time, unusual in terms of his delayed consolidation phase: His memory of details appears to improve, rather than deteriorate, with time. His behavior shows autistic traits in the limited use of facial expression and intonation, in the avoidance of eye contact, and in the resistance to social, verbal, and non-verbal interaction. His performance on theory of mind tasks is inconsistent, as he usually fails versions of the Sally-Ann task and passes the Smarties tasks. However, when varying these tasks in terms of certain variables, such as accessibility of encyclopedic knowledge ("Smarties tubes contain Smarties"), Christopher's performance deteriorates (Smith et al., 2010). Turning to language, Christopher's profile is very different.

Christopher speaks English as his native language. He understands, speaks, and judges English sentences of varying lexical and syntactic complexity like a native speaker of English. He can appropriately judge long-distance *wh*-questions, island violations, sequence-of-tense phenomena, morphosyntactic agreement patterns, negative inversion, and other 'core' phenomena of English grammar in a fast and determined way. The only phenomena where Christopher behaves unlike native English speakers are those that rely on the

processing of information structure, such as topicalization and dislocation structures. In addition, he fails to recover from garden-path violations, indicating that his ability to inhibit a first, incorrect parse is rather deficient (Smith and Tsimpli, 1995; Smith et al., 2010 and references therein). Consistent with his autistic behavior is Christopher's inability to interpret irony and metaphor, metalinguistic negation, and figurative language in general.

At the same time, Christopher has learned around twenty languages, most of which he learned without spoken interaction. Through his 'obsessive' interest in written language, he has been able to develop large vocabularies in most of these languages, knowledge of derivational and inflectional morphology (with overgeneralizations evident in many of the 'second' languages in which he has achieved a good level of proficiency), and, to some extent, syntactic variation. At the same time he reads for comprehension, as he has clearly gathered considerably encyclopedic information on a variety of topics ranging from football and politics to history and meta-language. Christopher was taught an invented language, Epun, from mostly written input. Epun had both natural and unnatural rules of grammar, the latter based on linear rather than hierarchical ordering, hence not UG-constrained. Unsurprisingly from a generative perspective, Christopher was unable to discover the unnatural rules but was able to learn Epun's natural, UG-based rules of grammar (Smith et al., 1991; Smith and Tsimpli, 1995). His learning of Epun illustrates a learner profile compatible with the autonomous status of a language faculty, which in Christopher's case remained intact (cf. question (A)), despite a seriously challenged general cognitive profile.

Teaching Christopher British Sign Language (BSL) was a risk; his limited eye-contact, insensitivity to facial expression, and the fact that BSL lacks a written form were all reasons to predict failure. Nevertheless, Christopher's learning of BSL showed a strong asymmetry between comprehension and production—in favor of comprehension. Moreover, his inability to develop good knowledge of BSL classifiers stood in sharp contrast with his learning of other domains of morphosyntax (agreement, negation, questions), where he performed similarly with the control group of adult, good language learners. The learner profile Christopher revealed in his BSL development was interesting precisely due to the asymmetries it involved: His inability to learn classifiers, but not other aspects of BSL grammar, indicates that classifiers draw heavily on representations mediated by visuo-spatial cognition, which is deficient in Christopher's case (Smith et al., 2010). As such, his poor performance indicates the role of language modality (signed vs. spoken) on language acquisition and the role of interfaces in each. For instance, the form of classifiers in BSL seems to involve an interface between the visuo-spatial processor and language, while in spoken languages this interface does not seem relevant, as shown by Christopher's unproblematic acquisition of spatial prepositions in spoken languages (Smith et al., 2010).

We could thus conclude that Christopher's unique savantism in language (itself an obvious answer to question (C)) might be attributed to his hyper-attention to linguistic detail, hyper-sensitivity to written language input, and hyper-systemizing in the discovery of rules, patterns, and generalizations that make languages similar—and at the same time different from each other. Crucially, Christopher's case demonstrates multiple dissociations among language, thought, and communication, pointing to a certain degree of autonomy of the language faculty itself.

19.5 Acquired language disorders

Every human being uses language to communicate needs, knowledge, and emotions. In everyday life, talking, finding the right words, understanding what is being said, reading and writing, and making gestures are all part of using language effectively to communicate. If, as a result of brain damage, one or more parts of language use stop functioning properly, this

brings on aphasia. The word *aphasia* comes from the ancient Greek *aphatos* meaning without speech, but this does not mean that people who suffer from aphasia are speechless. In theoretical and clinical terms, aphasia is a linguistic impairment due to brain injury (usually a stroke in the left hemisphere), which results in difficulties with both comprehension and production of spoken and written language.

Aphasia is classified into different types according to performance on production, comprehension, naming, and repetition tasks. The different aphasia types and associated brain lesioned areas are reported in table 1.

Aphasia type	**Fluency**	**Comprehension**	**Naming**	**Repetition**	**Lesion (BA)**
Broca's	non-fluent	preserved	impaired	impaired	BA 44, 45
Wernicke's	fluent	impaired	impaired	impaired	BA 22, 37, 42
Anomic	fluent	preserved	impaired	preserved	BA 21, 22, 37, 42
Conduction	fluent	preserved	impaired	impaired	BA 22, 37, 42
Global	non-fluent	impaired	impaired	impaired	BA 42, 44, 45

Table 1: Classification of aphasias based on fluency, language understanding, naming, and repetition abilities, and lesion site (BA=Brodmann area).

Overall, the salient symptoms of aphasia are interpreted as disruptions of normal language processing and production, affecting multiple levels of linguistic description (lexicon, morphology, phonology, semantics, and syntax). Yet, individuals with aphasia form a highly heterogeneous group with large individual differences in post-stroke linguistic profiles, severity of aphasia type, and recovery patterns. Cognitive disorders such as working memory limitations and/or executive function deficits may also influence the impact of—and recovery from—aphasia (Lazar and Antoniello, 2008).

When focusing on acquired language disorders from the perspective of a UG-based approach, a number of questions about the mechanisms mediating language use after language breakdown in relation to linguistic knowledge (semantic, syntactic, phonological, pragmatic) become pertinent, such as the possibility of impaired core operations (cf. question (A) above) or the distinction between language use vs. linguistic knowledge (cf. question (B)). Furthermore, whether linguistic competence *per se* is impaired or whether language difficulties are the consequence of a breakdown in non-linguistic, cognitive systems (e.g., working and/or short-term memory, executive functions) affecting language use merits deciphering (also relating to question (D)). Moreover, whether language pathology affects language use differently, depending on how or when in life the language breakdown occurred, deserves further exploring as well (cf. question (C)). We will address each question area below.

Let us start with question (B), language use vs. language knowledge in aphasia. For the aims of this chapter and due to space restrictions, our focus lies on describing the effects of a specific language deficit (for words, syntactic rules, sentence meaning, etc.), such as agrammatic Broca's aphasia, on the language faculty. Agrammatic aphasia is very well studied, and characterized within and across modalities (comprehension and production) by impaired and preserved syntactic abilities. Individuals with post-stroke agrammatic aphasia show no major cognitive deficits in general intelligence or any severe impairment in activities of daily living and social skills. In this case, we have impaired language and communication with cognitive and social abilities generally intact.

The theoretical implications for analyses of impaired and spared grammatical abilities in agrammatic aphasia is the extensively reported asymmetry in grammatical knowledge and use for grammatically complex structures on the one hand, and between comprehension and

production on the other. We will briefly present recent research on the topic as it contributes to the discussion of issues debated in current syntactic theory suggesting a resulting breakdown of language, not of UG. What these theories have in common is the same culprit: Linguistic processing complexity is responsible for the agrammatic profile in light of generally intact linguistic representations. Nevertheless, the notion of agrammatism *per se* remains widely open to cross-linguistic investigation, and not yet resolved.

One large body of studies has shown that comprehension of sentences with canonical word order remains intact in agrammatic aphasia (e.g., active sentences, subject *wh*-questions, subject relative clauses, and subject clefts), while comprehension of sentences with non-canonical word order is impaired (e.g., passives, object *wh*-questions, object relative clauses, and object cleft sentences). Several accounts have been postulated to account for the underlying deficit in comprehension of canonical vs. non-canonical word order, which can be grouped as computational, syntactic, compositional, movement-derived, or as related to processing effects (see Varlokosta et al., 2014, and references therein). Also, the frequency of certain attested complex constructions—that is, how often the construct appears in a language (e.g., verb movement or transitivity)—does not seem to impact negatively on comprehension of word order (Bastiaanse et al., 2009).

A second body of work on agrammatism has revealed that a deficit in producing inflectional morphemes is selective. For example, tense-marking has been found to be more impaired than subject–verb agreement across morphologically distinct languages (such as Arabic, Hebrew, German, Greek, and Korean, to name but a few; see e.g. Friedmann et al., 2013; Varlokosta et al., 2014). It has been suggested that this finding may be accommodated within a general hypothesis about impairment of interpretable features (Fyndanis et al., 2012), motivated by the framework of the Minimalist Program (Chomsky, 1995, 2000), such as the Interpretability Hypothesis (Tsimpli, 2001). This could mean that for agrammatic aphasia, there is a loss of interpretable features (tense), while uninterpretable features (agreement) are preserved.

There is also increasing work on the production of negation markers across languages to explain the linguistic deficit in agrammatism. Several different hypotheses have been postulated to predict the findings, including hierarchical, syntactic, and computational accounts. The reader is referred to Koukoulioti (2010) for an excellent review of current predictions of the different hypotheses on the basis of published findings on use of negation cross-linguistically by agrammatic speakers. Here we would just like to mention that one way of addressing question (A) might be adopting the 'weak syntax' approach of Avrutin (2006), possibly in terms of a "*slow syntax* characterization", according to which the operation Merge is "critically delayed in the agrammatic system" (Burkhardt et al., 2008: 122f.). However, since 'slow syntax' refers to a temporal delay, it is not at all clear that this would constitute an answer to (A) after all; the hypothesized limited processing capacity rather points to (B), affected mechanisms mediating language use, if not even (D), language-external factors.

What remains fundamental to most accounts for either comprehension and production deficits of grammatically complex structures in agrammatism is that the overall language disorder reflects processing limitations or underspecified syntactic representations in terms of some features for grammatically complex structures—and *not* a breakdown in core linguistic knowledge (Kolk, 1998; Grillo, 2009).

A second part to question (B), thereby also touching on (D), concerns the exact relationship between language use and the memory systems: short-term memory (STM) and working memory (WM) after language breakdown. Many studies have highlighted the strong connection between language and STM impairments in aphasia by using tasks that require a spoken response (e.g., digit repetition) and for tasks that are non-verbal (e.g., pointing span). Similarly, WM impairments have been argued to correlate with language impairments (word-

level and sentence-level processing) in aphasia. The reader is referred to Salis et al. (2015) for a recent update of the literature on the close link between aphasia and STM/WM impairments. Given the focus in this chapter on UG, it is fair to say that very little has been done to evaluate the relationship between WM and linguistic complexity in aphasia. One preliminary study (Wright et al., 2007) reported a syntactic WM deficit (measured by n-back tasks) for patients with different types of aphasia (Broca, conduction, and anomic) who showed the most difficulty comprehending complex non-canonical word order vs. canonical word order. The authors claim that language-specific WM deficits are a promising avenue of research with regards to the specific type of processing required (syntactic, semantic, phonological) for sentence comprehension (see also Gvion and Friedmann, 2012). Yet, the main controversies in relation to WM and aphasia are (i) whether a deficit in WM necessarily leads to a deficit in language understanding and (ii) the nature of WM for different language processes.

With regards to question (C), we will focus on lexicon-based research coming out of our own work using the noun-verb distinction in production to probe lexical retrieval deficits in SLI and anomic aphasia. Both SLI and anomic aphasia share a number of characteristics in relation to their linguistic profiles (e.g., word-finding problems, fluent speech) but differ significantly in other areas (e.g., aetiology). Aphasia, for example, may range from mild to severe, but it almost universally affects the ability to find words, a condition known as anomia. Patients diagnosed with anomic aphasia often struggle in structured confrontation naming tasks and in conversational speech when retrieving the target word. In their attempt to produce the correct response, they often generate a wide range of errors including semantically or phonetically related words, descriptions of the word, unrelated or jargon words, or no response at all.

Similarly, lexical SLI has been proposed as a separate sub-type of SLI by Friedmann and Novogrodsky (2008) based on their description of Hebrew-speaking children, who after rigorous testing met the criteria for lexical SLI; they failed a naming test, a word-to-definition task, and two verbal fluency tasks, but showed preserved abilities on grammatical (e.g., relative clause comprehension and production) and phonological testing (e.g., repetition of words and non-words). In our research (Kambanaros, 2010; Kambanaros et al., 2014; Kambanaros and van Steenbrugge, 2013), we have shown that children with SLI and adults with anomic aphasia display a similar pattern of verb-noun performance in relation to context-effects (picture naming vs. connected speech). In fact, the verb-specific naming deficit observed in both language impaired groups during a picture confrontation task involving concrete verbs (and nouns) did *not* resurface in connected speech (Kambanaros and van Steenbrugge, 2013).

Overall, there seems to be no direct, one-to-one relationship between the production of verbs/nouns in connected speech and the retrieval and naming of these word types in isolation. Children with SLI and adults with anomic aphasia have intact comprehension for verbs and nouns but show a differential retrieval performance according to context, with poorer naming than word retrieval in connected speech. It is conceivable that multiple factors may influence greater retrieval success for verbs in connected speech. These can include the lexical characteristics of the target words (e.g., lexical-semantic heaviness or specificity), the interactions among activations at different linguistic levels (i.e. semantic, phonological), and the cognitive demands of the experimental tasks on verb use (e.g., contextual influences on lexical retrieval). Nevertheless, verb deficits cannot serve as a distinctive characteristic for either language-impaired group but only as a clinical descriptor in the context of naming.

The bigger picture concerns the classic competence–performance distinction. We suggest to use insights from such work on the lexical–syntactic interface and transform competence-based research into a performance-sensitive model: It is not an issue of being

able to name a particular action with a single verb which is well known—it is a matter of activating and retrieving the concept speedily and successfully within the human language faculty and its interface with the performance systems for which there is a model. Put simply, in the current minimalist framework (Chomsky, 2000 *et seq.*), there is a well defined pathway from the lexicon to the conceptual-intentional interface (vocabulary, syntax, semantics) with the add-on component of sound (phonological structure, instructions to the articulatory-perceptual/sensorimotor system), roughly as depicted in figure 2 above. In the research just cited (see also Kambanaros and Grohmann, 2015), we show that language pathology can affect language use in a similar pattern, at least for the lexicon and for our populations under investigation, irrespective of whether the language impairment is developmental (e.g., SLI) or acquired (e.g., aphasia).

In closing this section on aphasia, it is important to note that little is known about the relationship between aphasia treatment and the pattern of language impairment (speech, understanding, reading, writing), individual patient profiles (age, gender, education, languages), and stroke (severity, location, and time since onset). Unfortunately, these gaps in knowledge limit the accuracy of the diagnosis, the design of optimum rehabilitation interventions, and the precision of prognosis for people with aphasia following stroke, their families, and the health and social care professionals working with them.

19.6 Language pathology within comparative biolinguistics

In addition to the five foundational questions (Chomsky, 1986) addressed above, Chomsky (2005) suggested three factors that are crucially involved in the design of language: genetic (UG), epigenetic (experience), and third-factor considerations (not language-specific). As McGilvray (this volume) puts it, the final state of a child's linguistic development is "the result of 1) genetic specification (biology), 2) 'input' or 'experience', and 3) other non-genetic natural constraints on development, such as constraints on effective computation", that is, "Chomsky's 'three factors'" (see also the chapters by Huang and Roberts, Lohndal and Uriagereka, Newmeyer, and Rizzi, this volume).

The null hypothesis about UG is that it, qua faculty of language, is invariant across the species (see section 2). This perspective has recently been challenged in a very interesting proposal by Balari and Lorenzo (2015), who argue for a *gradient* view of language, "an aggregate of cognitive abilities" (p. 8), rather than a language *faculty* as such (see also Hernández et al., 2013; Kambanaros and Grohmann, 2015; Grohmann and Kambanaros, 2016):

> Developmentally speaking, the resulting gradient view purports that language is not circumscribed to a particular compartment of our mind/brain, but spreads on a complexly interactive system of bodily capacities subject to the impact of a correspondingly complex array of developmental influences, both endogenous and exogenous. Such a picture makes very unlikely the idea of a faculty of language as an epitome of sorts, from the point of view of which impaired, lessened or even enhanced variants must be deemed exceptionally deviant. (Balari and Lorenzo, 2015: 34)

Such a conceptualization of the biological factors underlying language fits well into the current debates concerning the size of UG; see, for example, many discussions on Norbert Hornstein's *Faculty of Language* blog (http://facultyoflanguage.blogspot.com). These, in turn, were fed by Noam Chomsky himself opening up the possibility that a faculty dedicated to language need not be unique to humans; what is is the particular combinatorial system

required for computing and interfacing language. This is Hauser et al.'s (2002) distinction between 'Faculty of Language in the Broad Sense' (FLB) and 'Faculty of Language in the Narrow Sense' (FLN); for useful follow-up, see in particular Fitch et al. (2005), Fitch (2009), Miyagawa et al. (2013), Berwick and Chomsky (2016), and Miyagawa (to appear).

What this means is that the question of how language pathologies may inform UG and vice versa receives a new twist, too new as to provide a learned overview. But it certainly gives rise to interesting new questions. One such question concerns the neurobiological basis of language (see e.g. Schlesewsky and Bornkessel-Schlesewsky, 2013 and other chapters in Boeckx and Grohmann, 2013). Addressing this question is obviously relevant for directly brain damage-induced language pathologies such as aphasias, independently of whether we assume a full-fledged faculty of language in the traditional sense ('big UG'), a highly reduced one ('small UG'), or the distinction between FLB and FLN. However, one very important barrier to investigating the relationship of language breakdown and UG in this case is what Embick and Poeppel (2006) dubbed the 'granularity mismatch problem' (see also Poeppel and Embick, 2005), "a serious problem for those aiming to find brain correlates for the primitives of [the human faculty of language]" (Hornstein, 2009: 7, fn. 14):

> *The Granularity Mismatch Problem* (Poeppel and Embick, 2005: 104–105)
> Linguistic and neuroscientific studies of language operate with objects of different granularity. In particular, linguistic computation involves a number of fine-grained distinctions and explicit computational operations. Neuroscientific approaches to language operate in terms of broader conceptual distinctions.

That is, if we understand language pathology to inform researchers on UG, or the other way around, then trying to find the neural correlate for a possibly impaired Merge or Move operation will not be easy, to put it mildly (notwithstanding the exciting recent findings reported in Ding et al., 2015). As Grohmann (2013: 343) points out, this issue is also addressed by Boeckx (2010: 159), who actually turns the tables: "[U]ntil neurolinguists try to look for units that match what theoretical linguists hypothesize [note with references omitted—KKG], the conundrum we are in will not go away".

Regardless of the outcome of these developments, UG viewed from the perspective of language pathology may open new windows into the human faculty of language as conceived today, windows that may not have been available in earlier stages of theoretically informed language research. By highlighting some relevant linguistic profiles across selected language pathologies, one route for future research could be to refine these, collect more data, tie in impaired language more closely with current linguistic theorizing (and vice versa). However, there is also a broader, larger message behind the above. A particular avenue of research that investigates more closely the commonalities behind genetic, developmental, and acquired language pathologies may be couched within what Benítez-Burraco and Boeckx (2014) refer to as *comparative biolinguistics*. Looking at variation in language pathology allows us to consider in more depth "inter- and intra-species variation that lies well beneath the surface variation that is the bread and butter of comparative linguistics" (Boeckx 2013: 5–6, see also Samuels, 2015; Boeckx et al., this volume).

This is a larger research enterprise (for a first sketch, see Grohmann and Kambanaros, 2016, which parts of this sections draw on). The primary aim is to obtain distinctive linguistic profiles regarding lexical and grammatical abilities, for example, concomitant with the goal to develop cognitive profiles across a range of genetically and non-genetically different populations who are monolingual, multilingual, or somewhere in between as well as populations with or without co-morbid linguistic and/or cognitive impairments as part of their genotype. While individual variability is clinically crucial, population-based research can

advance cognitive–linguistic theory through behavioral testing that acknowledges the brain bases involved. This will offer a unique opportunity to researchers to collaborate in fields as different as (but not restricted to) genetic biology, neurobiology of the brain, cognitive neuroscience, cognitive and developmental psychology, speech and language therapy or pathology, psycho-, neuro-, and clinical linguistics, and language development—as well as theoretical linguistics. In addition, it may inform better about the underlying faculty(s) involved, of particular concern, of course, the role of UG in pathology. Some recent work goes in this direction, if only partially, such as emergent perspectives on autism phenotypes (Bourguignon et al., 2012), the biological nature of human language and the underlying genetic programs (Di Sciullo et al., 2010), genetic factors in the organization of language and the role of the two cerebral hemispheres (Hancock and Bever, 2013), and the idea that syntactic networks may constitute an endophenotype of developmental language disorders (Barceló-Coblijn et al., 2015).

One concrete research project could involve child populations with developmental language disorders that are language-based (SLI and beyond), behavior-based (such as ASD), or the result of a genetic syndrome (not discussed in this chapter). It would investigate language competence and performance with a range of tools (from lexical to syntactic) as well as non-verbal, cognitive tasks (such as executive control); additional information will be yielded from comparing mono- and bilingual participants. Ideal groups could include the following:

- Specific Language Impairment (SLI): SLI is considered a language disorder in children exhibiting difficulties acquiring grammar, phonological skills, semantic knowledge, and vocabulary, despite having a non-verbal IQ within the normal range.
- Developmental Dyslexia (DD): Children with DD experience problems learning to read, write, and spell below their chronological age, despite having a non-verbal IQ within the normal range.
- Autism Spectrum Disorders (ASD): Children with a high-functioning ASD, such as Asperger's, have problems with language and communication; they also show repetitive and/or restrictive patterns and thoughts of behavior, despite having a non-verbal IQ within the normal range.
- Down Syndrome (DS): DS is caused by three instead of the normal two copies of chromosome 21; children present with language and cognitive deficits, though differently from WS.
- Williams Syndrome (WS): Individuals with WS are missing around 28 genes from one copy of chromosome 7; children present with language and cognitive deficits, though differently from DS.
- Fragile X Syndrome (FXS): In FXS, a particular piece of genetic code has been multiplied several times on one copy of the X chromosome; children present with language and cognitive deficits.

19.7 Conclusion

In a sense, the purpose of this chapter was two-fold. First, we wanted to provide a rough-guide overview of the present state of the art concerning linguistic accounts of some pertinent language pathologies. We believe that much can be gained from an interface approach that looks at the interaction of different components of linguistic analysis. The second goal, however, is less graspable and directly links the study of language pathology to the theme of this handbook: What can non-intact language tell us about UG? Looking at explicit accounts in the current literature, one could reach the conclusion that, despite decades of linguistically

informed investigations into impaired language, not much progress has been made to address the fundamental, underlying issue: the nature of the human *faculty of language* (UG) and its role in genetically damaged language, impaired language development, or acquired language disorders.

It is fair to say that most of the discussion on language pathology suggests a resulting *breakdown of language* (possibly alongside speech and/or communication), *not of UG*. In the past, allusions to some deeper breakdown may have been made. However, to our knowledge no study had been conducted in order to tease apart direct connections between language breakdown and UG; when UG principles were evoked, it was typically as an epiphenomenal attempt of explaining independent findings. An additional contributing factor seems to be the confusion that surrounds the notion of 'UG', especially in more recent years—in particular, whether the language faculty should be considered FLB or FLN (Hauser et al., 2002) and whether we assume a 'big UG' or a 'small UG' (Clark, 2012) (but see Fitch, 2009 for clarifications on both), and perhaps whether it is static or gradient (Balari and Lorenzo, 2015). More recent advances in biolinguistics (Boeckx and Grohmann, 2013) also provide new ways of thinking about, and investigating, the links between typical and impaired knowledge of language, language acquisition, language use, and its implementation in the brain (and organism at large). These are exciting, new starting points for dedicated research into the relationship between language pathologies and UG.

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