CHAPTER

19 Modularity and Developmental Disorders

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Abstract

In this chapter, we consider the origin of uneven cognitive profiles in individuals with developmental disorders, including accounts from cognitive, brain, and genetic levels of description. We begin by introducing the main types of developmental disorder. We then discuss what is meant by the idea of modularity and the key issues surrounding it, outlining how modularity has been applied to explain deficits in developmental disorders. We illustrate competing theoretical positions by contrasting two developmental disorders, Williams syndrome (WS) and specific language impairment (SLI). In the first case, we consider cognitive deficits in WS with reference to modularity at a cognitive level and evidence from behavioral studies. In the second case, we consider SLI with reference to modularity at the neural level and evidence from brain imaging studies. We finish by considering the future for modular theories of atypical development, including the implication of recent findings from genetics and from computational modeling.

Key Words: modularity; developmental disorders; cognitive profiles; emergentism; Williams syndrome; specific language impairment; autism; dyslexia; brain imaging; genetics; computational modeling

Introduction

When cognitive development goes wrong, the outcome can either be a child demonstrating global impairment across all cognitive abilities, or a child in whom some abilities are more impaired than others, thereby producing an *uneven cognitive profile*. While an atypical learning environment can produce an uneven cognitive profile—if you never encounter written text, you will have a selective disability in reading—uneven cognitive profiles can emerge across development even when the children are exposed to an apparently normal physical and social environment. The idea of *modularity* has been used to explain the origin of uneven cognitive profiles in developmental disorders. This proposal remains controversial for several reasons. These include ongoing debates concerning when modular cognitive functions appear in development, how cognitive modules are implemented in the brain, and the extent to which genetic variation (implicated in many disorders) can plausibly affect only particular modular functions. In this chapter, we consider each of these questions in more detail. We first introduce the main types of developmental disorder. We then discuss what is meant by the idea of modularity and the key issues surrounding it, outlining how modularity has been applied to developmental disorders. We next illustrate these theoretical positions by contrasting two developmental disorders, Williams syndrome and specific language impairment, the first with reference to modularity at a cognitive level, the second with reference to modularity in the brain. We finish by considering the future for modular theories of atypical development.

Developmental Disorders

In this section, we identify the developmental disorders to which the notion of modularity has

been applied. These can be broadly distinguished into two groups: disorders with a known genetic cause and disorders defined on behavioral grounds. In the first group are disorders caused either by chromosomal abnormalities, missing genetic code, duplicated genetic code, or single gene mutations. Down syndrome (DS) is caused by three instead of the normal two copies of chromosome 21. Turner syndrome is a disorder where females are missing all or part of one of the normal two copies of the X chromosome. In Williams syndrome (WS), around 28 genes are missing from one copy of chromosome 7, while in Fragile X, a particular piece of genetic code has been repeated multiple times on one copy of the X chromosome. Phenylketonuria (PKU), associated with frontal cortex dysfunction, is caused by the mutation of a single gene on chromosome 12, while a mutation of the FOXP2 gene on chromosome 7 has been associated with a particular profile of speech and language difficulties in one British family. The majority of these disorders (DS, WS, Turner syndrome, Fragile X, untreated PKU) are associated with general learning disabilities and a low overall IQ. However, some of these disorders additionally show uneven cognitive profiles. For example, as we shall see, in WS language and face recognition are viewed as relative strengths, while visuospatial cognition is a relative weakness.

Some developmental disorders are identified purely on behavioral grounds, where one or more cognitive abilities are failing to develop at age-appropriate levels. These include dyslexia (problems with reading), autism (problems with social skills and communication, as well as stereotyped and repetitive behaviors), specific language impairment (SLI; problems with language development), dyscalculia (problems with acquiring numeracy skills), prosopagnosia (problems with recognizing faces), attention-deficit/hyperactivity disorder (problems with sustained attention and behavioral control), and Tourette syndrome (problems with controlling behavioral initiation). Sometimes by virtue of their definition, these disorders entail that other cognitive abilities and indeed overall intelligence falls within the normal range, as assessed by standardized (intelligence) tests[1]. In most of these cases, risk for the disorder runs in families, implying that the disorder is at least partly of genetic origin. However, typically the genes have not yet been identified (for disorders such as autism and dyslexia, this is an area of intense research, with candidate genes regularly being reported).

The current state of our understanding regarding the genetic causes of such behaviorally defined disorders is as follows (see, e.g., Plomin, DeFries, McClearn, & McGuffin, 2008). Predominantly, the disorders are not caused by new genetic mutations but by versions of genes that are present and perhaps common in the normal population. Particular versions of such genes may carry an elevated risk for a given disorder (of, say, 0.5% per gene). Certain families accrue greater number of risk versions across generations, increasing the chance that offspring will develop the disorder (so, say, if you inherit the riskier versions of 100 of the genes, each contributing a risk of 0.5%, your chance of developing this disorder would be 50%). However, disorders are not all or none but vary on continua, with intermediate numbers of risk alleles associated with milder versions of the disorder. Even with a large number of the riskier versions of contributory genes, an environmental stressor may still be required to produce the full disorder, explaining why identical twins don't always both exhibit a given developmental disorder. Moreover, variations that carry a risk for one disorder might also carry a risk for another disorder (see, e.g., Vernes et al., 2008, for a report of a variant carrying elevated risk for both autism and SLI, and Keller & Miller, 2006, for discussion of the genetic causes of serious mental disorders). This state of affairs has rendered it difficult to identify the genes that "cause" a given behavioral disorder because, for example, it may be different genes contributing to the disorder in different children, and gene versions on their own contribute little risk.

Modularity

The concept of modularity is inspired by the idea that a system can comprise a set of parts, each of which has a different specialized function. The interaction between the specialized parts produces the behavior of the whole system. The idea of specialized components is familiar from designed artifacts (e.g., a bicycle has a frame, wheels, gears, a chain, handlebars, brakes, etc.). However, it is also an influential concept in biology (e.g., a body has arms and legs, there are specialized internal organs such as hearts and livers, and so forth). In 1983, the philosopher Jerry Fodor argued that the use of such specialized parts seemed to be a sensible way for a cognitive system to work (Fodor, 1983). Moreover, he identified a set of properties at least some of which he expected these "modules" to possess: modules would be specific to cognitive domains; they would work quickly, unconsciously, and

automatically; their computations would employ a dedicated knowledge base of facts about this specific cognitive domain; they would use dedicated neural architecture; they would be innate; and they would exhibit a characteristic pattern of breakdown (so that a restricted set of behaviors would be impaired when the module was damaged). Fodor conceived of these components as accounting for low-level perceptual and motor skills, in the manner of cognitive reflexes.

The concept of modularity has subsequently caused a great deal of debate (see, e.g., Barrett & Kurzban, 2006; Coltheart, 1999; Fodor, 2000, 2005; Pinker, 2005). In part, this is because Fodor's definition of a module constituted only a set of *likely* rather than *necessary and sufficient* properties. It was therefore hard to test empirically: absence of a given property or dissociation of any two properties could not demonstrate that the idea of modularity was wrong. For instance, one might identify that fast unconscious processing can be found in a skill that is learned rather than innate, such as reading or driving a car. However, this would not falsify Fodor's claim that modules tend to be fast, unconscious, and innate, because the claim is only for a tendency of the association of these two properties. As such, modularity became more a way of talking about behavioral data rather than a falsifiable scientific hypothesis about how the cognitive system worked. Subsequently, several theorists sought to revise the notion of modularity, either emphasizing particular properties from the original set to pin down a concrete definition or to widen its scope of application. For example, Coltheart (1999) emphasized domain-specificity as the key property, while Fodor (2000) emphasized the dedicated knowledge base that the module would use (sometimes called encapsulation of information), and Barrett and Kurzban (2006) focused on functional specialization. Some researchers argued that modularity wasn't only applicable to low-level cognitive processes, but that high-level cognitive skills could also be modular, an idea called "massive modularity" (see Fodor, 2000, for discussion). Stressing the property of innateness, some researchers additionally viewed high-level modules as the product of natural selection, connecting modularity with evolutionary psychology (e.g., Cosmides & Tooby, 1992, 2008; Pinker, 1997).

Within biology, a network of interactions within a system is called modular if it is subdivided into relatively autonomous, internally highly connected components (Wagner, Pavlicev, & Cheverud, 2007). As before, the idea is predicated on the assumption that the system has parts, and is formulated in terms of restricted causal scope[2]. In biology, the concept of modularity is used both for causal relations (e.g., protein–protein interactions, gene regulatory networks) and to summarize statistical relationships, such as traits that are observed to vary together in species versus those that vary independently (Wagner, Pavlicev, & Cheverud, 2007).

When the idea of modularity is applied to psychology, there is an additional complication. Our current research paradigm views the study of the mind as intimately connected with the study of the brain. Modularity as originally conceived by Fodor involves the cognitive system, including domains such as perception, motor action, language, memory, and attention. Somehow these cognitive systems must be implemented in the brain. However, it is not clear whether the brain adheres to the same principles of modularity as the cognitive system (if indeed it does so at all). Does the brain have specialized parts? Do these line up with the specialized parts identified in the cognitive system? Or do several specialized parts of the brain in concert produce cognitive-level modules?

Modularity and Development

If the cognitive system has specialized parts, where do they come from? One view is that the components are innate, already present in some primitive form in infancy. This view is typically associated with evolutionary psychology, where the components of the mind are considered to be the product of natural selection. The aim of evolutionary psychology is to explain psychological traits in terms of adaptations to ancestral environments (Buss, 2005). Within this framework, the results of psychological adaptations are cognitive modules. The idea is to discover modules by reverse engineering (e.g., Pinker, 1997). This is a means of discovering the design of a mechanism by considering the task that it performs. Evolutionary psychology uses this method in an attempt to reconstruct the mind by considering the adaptive challenges faced by our ancestors and then proposing the modules that evolved to meet them (see Cosmides & Tooby, 2008; Tooby & Cosmides, 1992).

Evolutionary psychologists have traditionally deemphasized development in favor of *design*, because design usually pertains to the endpoint of development. It is the behavior generated by the outcome of development that presumably leads the individual to have more or less offspring; the

behavior of the mature organism is therefore the target of natural selection. Thus adaptations are defined in terms of the outputs of an array of mature modules, each evolved to solve a particular problem. One well-known example of the evolutionary psychology approach is Cosmides and Tooby's (1992) hypothesis that there is an innate module that serves to detect *cheating behavior*. This is based on the notion that the evolution of reciprocal altruism (Trivers, 1971), where individuals provide benefits to each other without expecting immediate recompense, would create the opportunity for cheats to prosper by accepting benefits without returning them. In evolutionary terms, there would be a selection advantage for cheating, to the cost of those expecting their altruism to be reciprocated. In turn, there would be a selection advantage for *detecting* cheaters and excluding them from social interactions.

Of course, one can have an evolutionary perspective without committing to a modular notion of cognition, instead shifting the emphasis to development. For example, Jay Belsky and colleagues (Belsky, 2005; Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007) argue that phenotypic *plasticity* can be an adaptation, given the uncertainty of the environment: this year's behavioral adaptation might be next year's disadvantage. Therefore it can be advantageous to allow the environment to help optimize behavior. On this view, plasticity (or heightened sensitivity to the negative effects of poor environments and to the beneficial effects of supportive environments) is partly innate, but an *interaction* of plasticity/susceptibility and environment determines the phenotypic outcome. Other related adaptations might include variation in amount or timing of myelination, amount of gray matter, gross brain weight, and cortical thickness—properties of the brain that increase the scope and capacity of cognitive development.

Other researchers accept modularity but view it as an outcome of the developmental process, rejecting the idea that the cognitive system starts out with preformed modules in respect of high-level cognition. This position, known as *emergentism*, views development as a process of experience-dependent self-organization (Elman et al., 1996; Karmiloff-Smith, 1992; Smith & Thelen, 2003). The cognitive system begins with components with preferential inputs and outputs. Components may even have biases about the sorts of computations that they are more efficient at performing. However, there is initially lots of cross-connectivity between components, and components are poorly tuned to carry out processes relating to particular cognitive domains. The greater information flow around the system means that early in development, more components contribute to generating behavior. Depending on the environment to which the individual is exposed, the overall system organizes itself so that the components become highly tuned for particular functions, pruning away the over-connectivity and committing to a particular functional structure. This picture of emergent modularity is informed by recent theories on the origins of functional specialization in the cortex of the brain, such as Johnson's interactive specialization account (Johnson, 2005).

A reconciliation between the evolutionary and developmental positions has been proposed that views natural selection as operating on developmental systems (Barrett & Kurzban, 2006). That is, evolutionary causes are instantiated by genes and environments during the developmental process. In this view, the set of human modules fashioned by selection reliably emerge in individuals *so long as the normal process of development takes place*, which in turn depends on the child being raised in a normal environment. Thus, one might develop a specialized system for recognizing faces so long as one was exposed to a world containing lots of faces, but not otherwise. Under this view, evolution would have selected for the mechanisms that reliably lead to the development of a specialized face-recognition module when exposed to a world containing faces.

Modularity and Developmental Disorders

The mechanisms by which modular outcomes are delivered remain sketchy, particularly with regard to the way in which genetic causes serve to shape such outcomes. The detail is important, because we are faced with some puzzling questions. Here is a good example. By the time we are adults, many of us have acquired a specialized cognitive system for reading written text. This system can even be selectively damaged in adults who experience brain damage (e.g., Coltheart, 2001). Some people experience problems in learning to read, and this developmental dyslexia can run in families. But how can one *inherit* a *specific* deficit for reading, affecting no other cognitive abilities? Reading is a relatively recent cultural invention; there is insufficient time for this skill to have been the target of natural selection and for the design for a "reading module" to have been encoded on the human genome (and faults in that design passed down through families). If the genes that produce poor reading development

are not *genes for reading*[3], then why are other cognitive abilities not affected?

Modular explanations of developmental deficits have, however, been widely deployed. These accounts work as follows. If in a given disorder there is an uneven cognitive profile, with some abilities scoring in the normal range but one (or more) showing deficits, then perhaps this is the result of one (or more) cognitive modules failing to develop properly, while the other modules follow the normal developmental pathway. In the case of reading, one might argue that a module that contributes to reading performance has not developed normally, while the rest of the cognitive system has nevertheless done so. If the developmental disorder is genetic (and we have seen that most have genetic contributions), then maybe the uneven profile is due to genetic variation in the genes normally responsible for the development of the impaired module(s). Examples of this form of proposal include the idea that indeed, a module crucial for reading, in this case processing phonology, fails to develop properly in dyslexia (Frith, 1995), the idea that a theory-of-mind module may fail to develop properly in autism (Leslie, 1992), a module for syntax may fail to develop properly in some types of SLI (van der Lely, 2005), and a module for editing intentions may fail to develop properly in Tourette syndrome (Baron-Cohen, 1998a).

There are a number of problems with the simplest application of the idea of modularity to apparent selective behavioral deficits in developmental disorders. These stem from the fact that development is an adaptive process, in which individuals are attempting to optimize their interactions with their physical and social environments. Behavioral impairments that emerge across development must therefore be construed in terms of the developmental process itself (Karmiloff-Smith, 1998). This means that explanations of developmental deficits should include several key concepts that characterize developmental theories (Thomas, 2005a). These include *plasticity*, that the cognitive system changes its structure in response to experience; *interactivity*, the idea that cognitive components interact with each other across development; *redundancy*, the idea that a given task can be accomplished equally adequately by more than one system; and *compensation*, the idea that certain behaviors can be delivered in different ways (perhaps not as well) by alternative systems. In other words, in the case of a disorder, the cognitive system has a goal of developing adaptively, but the altered constraints on development mean that it is not successful. The appropriate explanation of the behavioral deficits therefore involves identifying how the constraints on development have been altered by the disorder (Bishop, 1997; Karmiloff-Smith, 1997, 1998; Temple, 1997; Thomas, 2008; Thomas & Karmiloff-Smith, 2002). In particular, any explanation of an uneven cognitive profile in modular terms must answer two questions (Thomas, 2005b): (1) Why can't the normally developing modules compensate for the initially impaired module(s) across development, thereby attenuating the effects of the initial deficits? (2) Since the normal development of modules involves interactions with other modules (if only to share information), why hasn't the initial impairment to one/some module(s) spread to other modules across development, thereby exaggerating the initial deficits? If answers to these questions have been omitted in a given proposal, then that proposal is incomplete, because it means we do not understand the details of the developmental account.

Four different perspectives on the application of modularity to developmental disorders can currently be discerned. The first view is that most developmental disorders can be characterized in terms of the normal set of high-level cognitive modules in individuals of equivalent age, but with one or more modules underdeveloped (or potentially, in the case of savant abilities, overdeveloped). This position places perhaps the least emphasis on development. For example, Temple (1997) offered explanations for a range of developmental disorders (including dyslexia, prosopagnosia, and dyscalculia) with reference to modular theories of normal function originally conceived to explain intact and impaired performance in *adults*. On this account, behavioral deficits correspond to focal impairments to selected modules. Although minimizing the role of development, theories of this type are nevertheless claimed to be inspired by behavioral observation. As Tager-Flusberg (2000) remarked, in most developmental disorders, there appear to be behaviors that look normal—at least, normal for the overall mental age of the individuals concerned. This leads to the inference that in most cases, the disorders are characterized by largely normal cognitive architectures, with individuals varying from the normal pattern in only certain circumscribed ways. However, much debate surrounds whether this behavioral observation is apparent or real. Moreover, if some behaviors are delayed in a disorder (i.e., at mental-age level rather than chronological-age level), it remains puzzling what the mechanistic cause of such delay could be.

A second position, called "minimalist innate modularity," accommodates a greater role for development. In this view, the modules observed in adults are built from smaller, more basic innate modules across development (Baron-Cohen, 1998b). Where a selective deficit is observed in an adult module, this may arise from the failure of one or more basic innate modules that contribute to its development. For instance, the proposed theory-of-mind module for reasoning about mental states may be constructed from lower-level systems for detecting eye gaze, for detecting emotions or intentions, or for sharing attention with others. Impairments in any of these low-level modules might cause developmental problems in learning to reason about other people's mental states, as for instance is observed in autism.

The third position is similar but argues that the low-level factors that contribute to impairments in the development of high-level skills are in fact more general than the domain in which they appear. It is the developmental process that creates the illusion of greater specificity. As we will later see, there are proposals that high-level deficits in grammar in SLI are caused by an initial deficit in processing or maintaining information about speech sounds (Gathercole & Baddeley, 1990; Joanisse & Seidenberg, 1998). This is because the speech sounds in question turn out to be key for carrying certain grammatical information in a sentence, producing higher-level deficits in comprehending and producing sentences. There are even more general accounts of SLI, for instance that its root cause may be a limitation in *processing capacity* (e.g., Bishop, 1994) or an underlying deficit of the *procedural memory system*, which is particularly important for acquiring and performing skills that involve sequences (Ullman & Pierpoint, 2005).

The fourth position places a still greater emphasis on development. This position is aligned with emergentism and is sometimes called *neuroconstructivism* (Karmiloff-Smith, 1998; Mareschal et al., 2007). The "neuro" prefix here reflects a school of thought that argues that theories of cognitive development should be informed by the way the brain develops. Neuroconstructivism proposes that if modules are a product of development and development has gone wrong, it is unlikely that only one module will be impaired and that the rest will have developed normally (termed "residual normality"; see Thomas & Karmiloff-Smith, 2002, for discussion). These researchers are therefore more cautious about the nature of uneven cognitive profiles, and argue that the selectivity of some of these behavioral deficits may be overstated. Scores on some behavioral tests that fall in the normal range may mask subtle differences in the nature of the underlying cognitive processes. The focus of this approach is to use more sensitive measures of processing to probe areas of apparent strength in individuals who exhibit uneven cognitive profiles, as well as areas of weakness. Moreover, neuroconstructivists argue that uneven profiles may change over developmental time. For example, Paterson and colleagues (1999) reported that individuals with WS and DS showed different respective abilities in language and numeracy in toddlers compared to adults. Toddlers with the two disorders showed no difference in a language task, but by adulthood, individuals with WS were superior to those with DS in language skills. Toddlers with WS were better at a numeracy task than toddlers with DS, but by adulthood, it was individuals with DS who showed stronger numeracy skills.

Let us summarize where we have reached thus far. We have encountered the idea of a module—broadly a specialized functional component—and seen how it has been deployed by some researchers to explain the uneven cognitive profiles observed in some developmental disorders. However, we have seen that those disorders can be split into different types (known genetic vs. behaviorally defined); we have seen that the notion of modularity has been used in different ways by different researchers; we have seen that for psychology, an additional problem arises of how cognitive modularity relates to brain modularity; and we have seen several competing theoretical stances that place different degrees of emphasis on the involvement of developmental processes in producing the uneven cognitive profiles. In the next two sections, we work through two examples in greater detail, making contact with the behavioral and brain imaging data that have been used to advance our understanding of the respective disorders. Our first example takes a known genetic disorder, WS, and examines modularity from a cognitive standpoint. Our second example takes a behaviorally defined disorder, SLI, and examines modularity from a brain standpoint.

Modularity and Disorders from a Cognitive Perspective: The Example of WS

WS is a rare developmental disorder, with a prevalence estimated to be around 1 in 20,000 (Morris, Dempsey, Leonard, Dilts, & Blackburn, 1988). The disorder is associated with learning disability, with IQs usually falling in the range between 50 and 70. It is also associated with an uneven cognitive profile.

Despite particular difficulties with visuospatial and numerical cognition, language abilities appear less impaired in individuals with WS (Ansari, Donlan, Thomas, Ewing, Peen, & Karmiloff-Smith, 2003; Donnai & Karmiloff-Smith, 2000; Farran & Jarrold, 2003; Howlin, Davies, & Udwin, 1998). Published findings that language is a relative strength in WS (e.g., Bellugi et al., 1990; Bellugi, Lichtenberger, Jones, Lai, & St. George, 2000) have been interpreted by some researchers as evidence that people with WS have a language module that has developed normally. The disorder has therefore been used as evidence in support of the claim that language is modular in typically developing individuals (e.g., Anderson, 1998; Bellugi, Marks, Bihrle, & Sabo, 1988).

For example, following initial investigations into WS, Pinker (1991, p. 534) wrote: "Although their IQ is measured at around 50, older children and adolescents with WS are described as hyperlinguistic with selective sparing of syntax, and grammatical abilities are close to normal in controlled testing. This is one of several kinds of dissociation in which language is preserved despite severe cognitive impairments, suggesting that the language system is autonomous of many other kinds of cognitive processing." A few years later, Pinker (1999) amplified this modular view by contrasting WS with SLI, a disorder where language development is impaired but measures of nonverbal skills fall in the normal range. Pinker argued that WS and SLI together represent a "genetic double dissociation": if both are disorders of genetic origin, and the development of language and nonverbal cognition can be separately impaired, then perhaps the genes influencing the development of language and nonverbal cognition are independent.

Fifteen years of research into WS followed these early claims. In 2007, Brock published a review of the subsequent findings into language capabilities in WS. He concluded that there is little compelling evidence that individuals with the disorder perform better on tests of syntax, morphology, phonology, or pragmatics than predicted by nonverbal measures. The unevenness of their cognitive profile had been overstated. However, one explanation of this exaggeration was that there was one language skill where individuals with WS appeared particularly strong, receptive vocabulary (e.g., Bellugi et al., 1990; Brock, Jarrold, Farran, Laws, & Riby, 2007; Jarrold, Baddeley, & Hewes, 1999), and one nonverbal skill where individuals with WS appeared particularly weak, visuospatial cognition (Donnai & Karmiloff-Smith, 2000). Juxtaposing these two skills maximizes the unevenness of the profile.

For disorders where there is a background of learning disability, one must be cautious with terminology. For a skill described as a relative strength in individuals with WS, this is in relation to their mental age (i.e., their general stage of developmental progression), not their chronological age. Few if any skills would be at the same level as a typically developing individual of the same chronological age. For example, a 12-year-old child with WS might have receptive vocabulary skills at the level of an 8-year-old but visuospatial skills of a 3-year-old. Claims of independent language and cognition in WS are often based on studies employing mental-age–matched controls, because this choice of control implicitly assumes a predictive relationship between language and cognition (Karmiloff-Smith & Thomas, 2003). For our example child with WS, one might say his overall mental age is the average of his verbal and nonverbal abilities ([8 + 3] / 2 = 5.5 years) and remark that his verbal ability exceeds his overall mental age—yet his verbal ability is still 4 years behind chronological age expectations. As Karmiloff-Smith (1998) pointed out, the use of IQ scores can be particularly misleading with reference to language abilities. It may be striking to reveal that an individual has fluent language despite an IQ of 50, but less so to say that an individual with a mental age of 6 has fluent language; most typically developing 6-year-olds have fluent language.

Special Language Abilities in WS?

There is one often-cited aspect of WS language that has been used to assert particular communicative sophistication. The reported use of low-frequency words by individuals with WS is often used as evidence that language is special or precocious for people with the disorder (e.g., Bellugi et al., 1990; Bellugi, Wang, & Jernigan, 1994; Udwin & Dennis, 1995; see Thomas, Dockrell, Messer, Parmigiani, Ansari, & Karmiloff-Smith, 2006, for a review). For instance, it is striking to hear an individual who is struggling to complete a simple puzzle talk of later going to visit his "associates" rather than his "friends." This use of unusual words was investigated by Thomas and colleagues (2006) with a speeded picture-naming task in order to assess whether such use resulted from an atypical criterion used to access lexical items for speech production. When we name pictures, people tend to take longer to produce low-frequency words than high-frequency words, and longer to name pictures

of actions than objects. By measuring how quickly individuals with WS named pictures, it was possible to see whether frequency was encoded in the same way in their lexicons. The results indicated that frequency and semantic category effects were identical to those of a receptive vocabulary-matched control group. Where individuals with WS used rare words, this was not therefore due to a malfunction of their mental dictionaries. Instead, Thomas and colleagues suggested the use was deliberate, and that unusual words may be a social engagement device.

People with WS are sometimes described as having a *hypersocial* personality profile (e.g., Gosch & Pankau, 1997; Jones, Bellugi, Lai, Chiles, Reilly, Lincoln, & Adolphs, 2000) and their conversational speech includes frequent pragmatic conversational devices in attempts to engage the attention of listeners (Reilly, Losh, Bellugi, & Wulfeck, 2004). For example, one individual with WS said the following, while inventing a story to accompany a set of pictures: "And he said '*Hey*, frogs! We're all together!' The end! *That was great, wasn't it?*" (engagement devices in italics). Although some individuals with WS are reported to make use of devices such as clichés, idioms, and figurative language, this language is often misapplied or out of place in the particular social context (Bertrand, Mervis, Armstrong, & Ayers, 1994; Udwin & Yule, 1990). Udwin and Yule (1990) found that around a third of a sample of 43 children with WS met the criteria for "cocktail party speech," characteristics of which include well-developed articulation, intonation, and stress patterns, but a poverty of communicative content and a tendency to relate irrelevant personal experiences, along with liberal use of stereotypical social phrases and conversational fillers.

This inappropriate use of language may reflect a poor underlying knowledge of its meaning. In support of this notion, a small study by Bertrand and colleagues (Bertrand, Mervis, Armstrong, & Ayers, 1994) showed that individuals with WS find it very difficult to explain the meanings of proverbs and metaphors, with only 3 out of 14 participants able to provide an explanation for any of 12 test proverbs. Most participants' responses focused on surface elements of the proverbs without any reference to the figurative message. For example, responses to "Strike while the iron is hot" included "Don't touch it" and "Iron clothes." Performance was somewhat better for metaphors, with eight participants able to offer an explanation for at least one of seven metaphors; the highest number of metaphors explained was four. In a similar fashion, individuals with WS also find it very hard to distinguish lies from jokes, tending to judge both as lies and justifying their interpretation simply by recounting the narrative, even when they demonstrated an understanding of second-order theory of mind and all the requisite knowledge for successful task performance (Sullivan, Winner, & Tager-Flusberg, 2003).

One possibility, then, outlined by Thomas and colleagues (2010) is that individuals with WS might use figurative language as a kind of "frozen" vocabulary: each phrase is invariant and retrieved from memory rather than involving any kind of "online" process (Annaz, van Herwegen, Thomas, Fishman, Karmiloff-Smith, & Runbland, 2008). Use of frozen language by people with WS might give rise to overestimations of language abilities by peers, teachers, and caregivers, in turn leading to people with WS facing language that they do not understand. Clearly, any difficulty in understanding language would be highly likely to contribute to the social difficulties experienced by children and adolescents with WS (e.g., Gosch & Pankau, 1997). However, perhaps the most important reason to consider figurative language is that, far more than vocabulary, it captures something of day-to-day communication and seems a good representative of language as a whole.

Investigating Figurative Language in WS

Although traditionally viewed as unusual linguistic ornaments that complement literal language, recent research indicates that metaphor is common in everyday language (e.g., Graesser, Long, & Mio, 1989; Pollio, Barlow, Fine, & Pollio, 1977). For instance, Graesser and colleagues (1989) found that speakers on television programs used approximately one unique metaphor for every 25 words uttered. Moreover, various strands of linguistic evidence suggest that metaphor is important for communicating about abstract concepts and may even be the only way of reasoning about them (e.g., Gibbs, 1994; Lakoff & Johnson, 1980). Understanding metaphorical language involves proficiency in both cognition and language, relying on several component abilities, including an understanding of communicative pragmatics, semantic knowledge processing capacity, and metalinguistic skill (see Vosniadou, 1987a, 1987b).

To understand metaphor, for example, one must perceive a similarity between two terms, and also realize that those terms belong to different conventional categories (e.g., Bowdle & Gentner, 2005). Therefore, an initial step in investigating children's

ability to understand metaphor at a given stage in development is to assess their ability to understand nonliteral similarity statements, which necessitates knowing that items falling in different semantic or conceptual categories can nevertheless be similar in some sense.

Thomas and colleagues (2010) administered a simple picture-based categorization task to children and adults with WS, typically developing children aged between 4 and 11, and typically developing adults. The study employed a developmental trajectories approach, in which a function is constructed linking task performance to changes in verbal mental age (see Thomas, Annaz, Ansari, Scerif, Jarrold, & Karmiloff-Smith, 2009). This permits developmental change to be compared across the typically and atypically developing groups. A key benefit of employing trajectories that link performance on a task to a mental-age measure is that they can be used to examine whether that performance is commensurate with the developmental state of other measures of cognitive ability. One asks: Is ability *X* where you would expect it to be, given the developmental state of the rest of the cognitive system, even if ability *X* is not at age-appropriate levels?

In this categorization task, both perceptual and functional nonliteral similarity were investigated. Nonliteral similarity is similarity that crosses category boundaries: the sun and an orange are different things, but both are of similar shape and color. The ability to understand nonliteral similarity is a key component of using figurative language (e.g., "the sun is an orange in the sky"). In a paradigm adapted from Vosniadou and Ortony (1983), participants were required to complete comparison statements and categorization statements (e.g., "The sun is like . . . ?" or "The sun is the same kind of thing as . . . ?") by choosing one of two words. The pairs of words were formed from items that were literally, perceptually, or functionally similar to the target word, or else anomalous (e.g., "moon," "orange," "oven," or "chair," respectively). Justifications of responses were also recorded to gain an insight into how participants selected their responses. The logic of the study (after Vosniadou & Ortony, 1983) was that selecting literally similar pairs in the categorization task was evidence of category knowledge (e.g., the sun is the same kind of thing as the moon, rather than an orange). Selecting perceptually or functionally similar items in the comparison task was evidence of nonliteral similarity (e.g., the sun is like an orange, rather than the moon).

The results indicated that understanding of nonliteral similarity emerges in a similar way in individuals with WS as it does in typically developing individuals. However, there was an important group difference: while typically developing individuals expressed an emerging preference for functional similarity with increasing age (e.g., the sun is like an oven, rather than an orange), the WS group maintained a preference for the simpler perceptual similarity. Adult usage of figurative language tends to depend on more abstract types of nonliteral similarity (e.g., the metaphor "boiling mad" does not mean hot but about to explode into action). The results of this study are consistent with the notion that where individuals with WS do use adult figurative language, they may do so without fully understanding the abstract mappings that underpin it (cf. Bertrand et al., 1994).

Thomas and colleagues sought to investigate further the nature of any disparity between language use and underlying knowledge in WS. This time, Purser, Thomas, Snoxall, and Mareschal (2009) investigated lexical semantic knowledge using a task where individuals were asked to sort animals into semantic categories. The particular concern was to tap underlying knowledge while avoiding the metacognitive demands inherent in the more usual definitions task. If one asks an individual to define the word "elephant," the implicit request is to list the salient and diagnostic features of the category "elephant" in decreasing order of salience and diagnosticity—a fairly challenging task. By contrast, the individual may know that elephant and zebra should be grouped together, and horse and cow should be grouped together, which would indicate underlying knowledge of typical habitat. Individuals were given both an animal sorting task and a definitions task to compare performance. Focusing on the domain of animals gave the individuals with WS the best chance to succeed, since individuals with WS as young as 10 have been shown to have unimpaired basic knowledge in this area relative to verbal mental-age–matched controls (Johnson & Carey, 1998).

Analyzing the trajectories of development, the performance of a group of adolescents and adults with WS on the definitions task began as if it were in line with their verbal mental age (as measured by a receptive vocabulary task). However, with increasing verbal ability, the typically developing group improved more steeply than the WS group. In the categorization task, the WS group's performance developed at a similar rate to that of

the typically developing group, but was markedly poorer on average than predicted by verbal mental age. This pattern of results indicated two things. First, the task of defining animals was indeed hard for the individuals with WS, particularly at the more sophisticated levels expected of older individuals (cf. Benelli et al., 1988). Second, as assessed by the sorting task, individuals with WS have poorer lexical semantic knowledge than expected given their level of receptive vocabulary, a skill for which this population is noted. Despite the disparity in absolute level, semantic knowledge, indexed by categorization task performance, develops at a similar rate to that found in typical development. Taking the results of the Thomas and colleagues and Purser and colleagues studies together, it can be seen that apparent linguistic competence in WS does not necessarily reflect normally developing underlying processes. The hypersocial personality profile of people with WS, along with good vocabulary learning ability, appears able to overcome weaker aspects of cognition and language to give an appearance, at least in some cases, of relatively advanced language skills, illustrated by the presence of rare words and figurative language in their speech.

A final point to emphasize in relation to figurative language comprehension is that understanding nonliteral or abstract relations is but one necessary part of real-world figurative comprehension; another is to interpret the intention of the speaker, recognizing that the statement is not intended to be taken literally. As outlined above, Bertrand and colleagues (1994) asked participants with WS to explain metaphors, and those participants tended to focus on surface features. A range of *conservation* tasks was also administered, testing conceptual understanding of the physical properties of the world: of the five participants who demonstrated any understanding of conservation, four also performed above chance on the figurative language tasks. Bertrand and colleagues pointed out that, like conservation tasks, the successful comprehension of figurative language involves integrating several sources of information (words, intonation, context, gesture), and the ability to integrate different sources of information appears to cut across both cognitive and language domains. This serves to illustrate that notions of monolithically preserved language are likely to be misguided, simply because language involves so many components of ability, only some of which are likely to be unique to that system.

Fractionation

We have reviewed a line of investigation into whether the language system *as a whole* develops normally in WS, against a background of poor nonverbal (and particularly visuospatial) cognition. Is WS a case where the module of language develops normally? The answer appears to be no. In this section, we consider whether more fine-grained selective deficits can be found within the language or cognitive systems of individuals with WS, and whether these deficits can inform theories of how the normal systems are structured. Can WS "fractionate" cognition?

Taking language ability to start with, the first problem that is encountered is that as performance is examined at finer levels of detail, fractionations appear within those finer levels. For example, Grant, Valian, and Karmiloff-Smith (2002) investigated productive grammar in WS, and found that although syntax was generally in line with overall level of cognitive functioning, their participants with WS performed worse than typically developing children when required to repeat sentences with complex relative clauses, often simplifying the sentence structure by omitting a verb or verb phrase, or an entire clause. This was despite the fact that the WS group had a higher mean verbal mental age than the typically developing participants. Should one conclude from this that people with WS have an impaired complex relative clause module within the syntax module?

If the answer is no—because the number of modules we have to postulate risks becoming so large—how should this level of fractionation be explained? Neuroconstructivists have attempted to offer an account (Karmiloff-Smith, 1998; Karmiloff-Smith & Thomas, 2003; Mareschal et al., 2007). Thomas (2006) used an example from outside the domain of language, that of face recognition, to illustrate how neuroconstructivism might explain fine-grained cognitive fractionations. Recognizing faces is another area in which people with WS show a relative strength, performing better than would be predicted by most other measures of cognition and even scoring at chronological-age levels in some tasks. One might view this as part of the hypersocial personality profile observed in this syndrome. Individuals with WS tend to be very interested in faces from an early age (Jones et al., 2000). Thus, people with WS are likely to be widely exposed to faces, and find it more intrinsically rewarding than usual to recognize faces and engage with people.

One could interpret the relative strength in recognizing faces as evidence that a face-processing module has been developing normally in this disorder (e.g., Bellugi, Wang, & Jernigan, 1994). However, research has shown that face recognition in WS doesn't work in quite the same way. Highly skilled face recognition in typical development involves a reliance on recognizing combinations of facial features and their exact arrangement in the face. In WS, however, skilled recognition is delivered by processing that relies more on individual facial features, such as the eyes or the mouth, rather than configurations of features (Annaz et al., 2009; Karmiloff-Smith, 1997; Deruelle et al., 1999; though see Tager-Flusberg, Boshart, & Baron-Cohen, 1998). Indeed, the ability of individuals with WS to process configurations of features might be as weak as their general ability to process visuospatial information (Karmiloff-Smith et al., 2004).

How does one reconcile the strengths and the weaknesses? Thomas (2006) gives the following possible neuroconstructivist account: genetic effects during brain development in WS generate initial cortical structures with different neurocomputational properties from the corresponding structures in typically developing individuals. Although overall processing is poorer, these neural structures are better able to process isolated featural information than configurations. The atypical visuospatial system is exposed to many faces via a socioemotional reward mechanism operating in a (functionally) separate brain structure. The visuospatial system subsequently develops atypically, but the system is able to perform acceptably because many faces can be identified on the basis of individual features and because faces are a very common stimulus. However, the system cannot develop the same neural organization, specialization, and localization that the system would typically possess for a given level of proficiency in face processing. A lot of practice enables a suboptimal system to reach performance in the normal range on a standardized test that is relatively insensitive to the way that this performance is being achieved.

Modularity and Shared Resources

The situation we have considered so far is that individual modules might develop atypically while others develop normally. However, there are more sophisticated ways that modular theories might accommodate uneven cognitive profiles. For example, instead of uneven profiles arising purely from specific deficits to specialized cognitive apparatus, it could be that difficulties lie at a level of processes shared by different cognitive specializations. As Barrett and Kurzban (2006, p. 637) comment, "it seems uncontentious that some computational resources are shared by multiple systems and that the use of such a shared resource at a given time precludes its simultaneous use by another. Arguments about modularity do not turn on this feature of cognition." Our final example from WS contrasts short-term memory ability with that found in DS. Once again, this is a domain in which early reports talked in terms of dissociations between specialized abilities.

Although the evidence for relatively strong language in individuals with WS has proved mixed, it is certainly clear that these individuals have a marked difficulty in visuospatial cognition (e.g., Farran & Jarrold, 2003). In this sense, DS offers an interesting contrast. The language abilities of individuals with DS are more impaired than their nonverbal skills, relative to their general level of cognitive functioning (e.g., Chapman, 1995; Fowler, 1990). The short-term memory abilities of each disorder appear to echo their different profiles of verbal and nonverbal abilities. Wang and Bellugi (1994) directly compared verbal and visuospatial short-term memory abilities in WS and DS with digit span and Corsi span tasks. In the digit span task, a list of numbers must be repeated back by the participant in the correct serial order, with lists increasing in length. The Corsi span task is analogous but uses the visuospatial domain. Participants watch an experimenter tap out a sequence of spatial locations across a board of pegs, then attempt to reproduce that sequence (Corsi, 1972, cited in Milner, 1971). Wang and Bellugi found that individuals with DS showed significantly poorer digit recall than individuals with WS, but reliably better performance on the Corsi task. The authors argued that this double dissociation constitutes neurogenetic evidence for two different cognitive systems, one specialized for short-term storage of verbal information, one specialized for short-term storage of visuospatial information. It should be noted, in passing, that the logic of double dissociation evidence is not straightforwardly extended to developmental disorders, for several reasons. For example, performance differences at one point in development do not imply that a stable double dissociation will be found at a later or earlier point (Karmiloff-Smith, Scerif, & Ansari, 2003; see Dunn & Kirsner, 2003, for wider discussion).

Several other studies have shown that individuals with DS have either superior (Jarrold & Baddeley, 1997; Jarrold, Baddeley, & Phillips, 2002; Laws, 2002) or similar (Brock & Jarrold, 2005; Jarrold, Baddeley, & Hewes, 1999; Numminen, Service, Ahonen, & Ruoppila, 2001) Corsi spans relative to matched control participants, while demonstrating relatively poorer digit spans (although see Seung & Chapman, 2000; Vicari, Carlesimo, & Caltagirone, 1995). Jarrold and colleagues (1999) also replicated the above finding that individuals with WS are impaired in Corsi recall, but not digit span, relative to learning-disabled controls. However, among other caveats, Jarrold and colleagues pointed out that, rather than necessarily showing *specific short-term memory deficits*, the pattern of results could reflect more general verbal processing problems in DS, or visuospatial processing difficulties in WS. As we saw with face processing, good task performance cannot necessarily be taken as evidence of a normally functioning system. The related point here is that poor performance on a given task cannot *prima facie* be taken as evidence of dysfunction of the systems primarily ascribed to afford good task performance for that task (in this case, short-term memory systems).

Instead, analysis of task demands can suggest that, rather than specific deficits of specialized cognitive apparatus, difficulties could lie at a level of processes shared by different cognitive specializations. For example, Brock and Jarrold (2005) suggested that a common mechanism might underlie both poor verbal short-term memory and speech production difficulties in DS. In typical development, there is evidence from neuroimaging studies indicating that both serial recall and speech production rely on the same cortical areas (see Gupta & MacWhinney, 1997). Furthermore, errors in speech production have been successfully modeled by computational models of serial memory (Vousden, Brown, & Harley, 2000).

In contrast to Baddeley's (1986) working memory model, several researchers have formulated models in which verbal short-term memory is not distinct from language systems, but instead reflects the transient storage of linguistic representations. For example, Monsell (1987) has presented a number of logically possible relations between speech input and output pathways and argued that verbal short-term memory data require the existence of two phonological buffers, possibly located in these separate speech input and output pathways. Evidence from typical development supports this notion: Shallice, McLeod, and Lewis (1985) showed that there is relatively little interference between detecting a name in a stream of words (processing input phonology) and reading aloud (processing output phonology), but there is severe interference between shadowing one stream of words while detecting a name in another (each of which requires processing input phonology).

The evidence outlined above could be interpreted in a framework in which speech production and digit span require some *common sequencing process*. One clear link between expressive language and verbal short-term memory is that both rely on the ability to successfully sequence verbal material. Purser and Jarrold (2005), using a modified verbal probed recall task, found that individuals with DS performed significantly worse than typically developing controls in a task where both item and order memory were necessary to afford good recall. However, when only item memory was required, the two groups performed similarly. This suggests that, to the extent that individuals with DS have a verbal short-term memory deficit, it might be a dysfunction of order memory. In support of this idea, Brock and Jarrold (2004) showed that individuals with DS performed worse on short-term verbal order memory task than predicted by their performance on a test of phonological item memory.

WS: Implications for Modularity

The take-home message from this work at the cognitive level is that although we may begin by identifying broad patterns of cognitive strengths and weaknesses in a disorder and formulating a simple explanation in terms of typically and atypically developing modules, the picture that emerges from more detailed research is more complex in at least three ways. The pattern of behavioral fractionation turns out to be much more fine-grained than whole cognitive domains; performance in the normal range may be generated by highly practiced skills that nevertheless rely on atypical underlying cognitive processes; and apparently selective impairments may arise from reliance on (impaired) resources that are shared across some but not all cognitive processes.

Modularity and Disorders from a Brain Perspective: The Example of Specific Language Impairment

In contrast to WS, SLI is a behaviorally defined developmental disorder. As the name suggests, the language abilities of children with SLI are

particularly affected. Despite having a nonverbal IQ within the normal range, children with SLI exhibit particular difficulties in the acquisition of grammar. These children also have poor phonological skills, and may also have poor semantic knowledge, and vocabulary (Van der Lely, 2005; Webster & Shevell, 2004). These difficulties also result in children with SLI experiencing problems learning to read, write, and spell, and in some cases affect their ability to learn mathematical skills such as performing arithmetic (Cowan, Donlan, Newton, & Lloyd, 2005). Although SLI is considered to be a disorder of language, it is also sometimes accompanied by nonlinguistic deficits, which include problems with working memory, impairments in motor skills that require sequencing, timing, and balance (Hill, 2001), and sequential auditory processing (Hill, Hogben & Bishop, 2005; McArthur & Bishop; 2005). This profile of linguistic and nonlinguistic deficits in SLI is highly variable and differs from child to child, so that SLI is usually characterized as a heterogeneous disorder. Although young children with SLI do go on to develop functionally complex language, pervasive underlying difficulties may still be detected through the use of cognitive tasks such as nonword repetition, where prior knowledge cannot be called upon to compensate for the linguistic deficit (Newbury, Bishop, & Monaco, 2005).

While SLI does not have a clearly defined genetic basis, its heritability attests to genetic involvement (Smith, 2007). The familial link with SLI has influenced research, with studies investigating differences in brain structure that are common to children with SLI and their families (Clark & Plante, 1998; Jackson & Plante, 1996; Plante et al., 1991). In recent years genetic researchers have begun the search for potential candidate genes that may contribute to the SLI phenotype (behavioral profile). These studies have used nonword repetition—a task that children with SLI find particularly difficult—as a phenotypic marker for identifying chromosomal anomalies associated with language impairment (Newbury, Bishop, & Monaco, 2005; SLI Consortium, 2002, 2004; Stromswold, 1998, 2001). Furthermore, previous studies of a British family in whom a single genetic mutation appeared to produce speech and language deficits supports the notion that there may also be a genetic link for SLI; in the KE family, as they are known, members inheriting the mutated FOXP2 gene exhibit a marked expressive language and articulation disorder (Lai, Fisher, Husrt, Vargha-Khadem, & Monaco, 2001).

Theories regarding the potential cause(s) of SLI at the cognitive level are predominantly domain-specific, focusing on a root deficit in a particular processing system. Grammar-specific hypotheses are a particularly prevalent feature of the literature to the extent that a grammar-specific subtype of SLI known as grammatical or G-SLI has been reported (van der Lely, 2005; van der Lely & Christian, 2000), and theories have been put forward regarding the nature of the faulty component. For example, van der Lely (2005) proposed the Computational Grammatical Complexity Hypothesis; Rice (2000) put forward the Extended Optional Infinitive Account, in which children with SLI are claimed to have a delayed and protracted period of grammatical development. Once more, these grammar-specific hypotheses are predicated on certain assumptions about how development works—in this case, that a grammar-specific processing module exists at the onset of language development, and that this module develops independently from other components of the language system.

Other domain-specific accounts suggest that SLI may be caused by a deficit in phonological processing—for instance, an impairment in phonological short-term memory (Gathercole & Baddeley, 1990) or phonological discrimination (Joanisse & Seidenberg, 1998). Difficulty in processing basic units of speech known as phonemes results in a degraded representation of initial speech information. The behavioral profile of SLI emerges as a result of the degraded quality of this phonological information upon which subsequent components of the language network (such as the semantic system and grammatical processing) rely.

Conversely, there are also theories of SLI that propose that the behavioral profile of the disorder is caused by a general rather than a specific processing deficit—for instance, a limitation in processing capacity (Bishop, 1994; Ellis Weismer et al., 1996, 1999), or an underlying deficit of the procedural memory system, which is particularly important for acquiring and performing skills that involve sequences (Ullman & Pierpoint, 2005; see Thomas, 2005b, for discussion). These theories suggest that the root cause of SLI is not domain-specific, affecting predominantly a single processing module independently of others, but either has a more general impact across multiple processing modules or requires an account that avoids the notion of modules altogether. Specific impairments appear in those areas that rely on a particular sort of information

(phonological) or process (sequencing) that has developed atypically.

Proposals for the causes of SLI at the cognitive level are in advance of our understanding of the processing systems and regions affected at the neurological level. The Procedural Deficit Hypothesis (PDH) does attempt to identify the brain regions that may be affected in SLI (Ullman & Pierpoint, 2005). These regions include the caudate nucleus (part of the basal ganglia), the temporal cortices, Broca's area, and the cerebellum, which are associated with a range of cognitive processes, such as working memory, language, rapid temporal processing, and dynamic mental imagery. According to the PDH hypothesis, the profile of impairment in SLI for any given child is dependent upon how severely these regions are affected, and how effectively the declarative memory system is able to compensate for the defective procedural system.

In this section we begin by considering how modules may be represented at the brain level. We then summarize recent studies investigating the brain basis of SLI, and discuss how atypical brain structure and function may explain different aspects of the behavioral profile of the disorder and provide an indication as to the potential cause(s). We also provide an example of how functional differences may be tested within a cognitively motivated theoretical framework using brain imaging. Finally, we discuss the implications of current differences in brain structure and function in SLI in terms of the broader modular perspective.

The Concept of Modularity at the Brain Level

If one were to adopt a literal view of how modularity might apply at the brain level, one might hypothesise that a domain-specific processing system would be localized within a single anatomically defined brain region. This is perhaps most clearly illustrated within classic cognitive neuropsychology, where damage to Broca's area (inferior frontal gyrus) causes a deficit in speech production, while damage to Wernicke's area (posterior region of the superior temporal cortex) causes a deficit in speech comprehension (Goodglass, 1993). Perhaps these regions house modules for production and comprehension, respectively. However, our current understanding of functional anatomy reveals a more complex picture. For example, while syntactic processing frequently engages the inferior frontal gyrus (Caplan et al., 1999, 2001; Cooke et al., 2001), functional studies have also indicated the involvement of temporal brain regions (e.g., Friederici et al., 2003; Humphries et al., 2005). Furthermore, the inferior frontal gyrus is activated by tasks that do not involve syntactic processing (e.g., Wagner et al., 2001). On the basis of this evidence, the syntactic processing system is likely to consist of a network of inferior frontal and temporal regions, which may also be engaged in other tasks, and whose involvement is unlikely to represent dedicated neural architecture. This brain-based description of the syntactic system violates the cognitive concept of modularity in two ways. *Multiple brain regions* are typically engaged in a specific cognitive task, and each region is frequently involved in *multiple tasks*.

In the following section we summarize current findings regarding the brain basis of SLI. We then follow by considering how these findings fit within a modular perspective in reference to potential underlying causes of the disorder.

The Brain Basis of SLI

As the brain is the substrate of cognition, the assumption is that deviations from typical cognitive ability may be due to underlying structural differences in brain anatomy, and/or functional differences in processing information. Therefore, a disorder may be characterized in terms of differences in brain structure or function in comparison to the typical population. Differences in brain structure can be examined through postmortem examinations, or through the analysis of detailed anatomical images such as those taken in magnetic resonance imaging (MRI) scanners. Structural differences may include cellular abnormalities, cortical malformations, differences in brain volume, or differences in the composition of brain tissue. Differences in brain function require recordings of task-related activity, for instance taking images of brain activity using an MRI scanner when an individual is carrying out a specific task such as listening to speech or reading words presented on a screen. In the context of disorders, functional differences may take the form of over- or under-activation of the same brain regions used to perform the task by the typical population, or the activation of one or more different brain regions not used by the typical population while performing the same task. In this subsection we summarize present structural and functional differences detected in imaging studies of SLI.

Structural Differences

Structural studies of SLI have revealed less pronounced differences in comparison to structural

studies of disorders such as WS (Thompson et al., 2005). However, the structural investigation of SLI has been limited in approach, with structural studies of SLI typically involving taking measurements of the size of different brain regions from detailed anatomical scans acquired through MRI. These studies have detected several features of the SLI brain that differ from those usually seen in normal development. The most consistently occurring finding is increased symmetry of the *planum temporale*. This area is an area located posterior to Heschl's gyrus encompassing superior temporal regions and is associated with the temporal processing of speech sounds and acoustic information (Griffiths & Warren, 2002), and it is normally observed to be larger on the left than the right side of the brain. These findings are consistent with an earlier postmortem study carried out by Cohen et al. (1989) on the brain of a child diagnosed as having SLI. However, the finding has not been consistently replicated (De Fossé et al., 2004; Preis et al., 1998), and differences in findings may in part be due to discrepancies in how the region of the planum temporale is anatomically defined, as well as differences in age and handedness of subjects, which can influence planum asymmetry (Eckert & Leonard, 2000). Increased variability in the asymmetry of the planum temporale is consistent with postmortem studies of developmental dyslexia (Galaburda, Kemper, et al., 1979; Galaburda et al., 1985; Humphreys et al., 1990), although here again, subsequent studies have not been entirely consistent in terms of the direction of the difference (Eckert & Leonard, 2000).

Other anatomical differences detected in SLI include abnormal patterns of asymmetry and symmetry in prefrontal and parietal cortex (Jernigan et al., 1991); a smaller pars triangularis (an inferior frontal region) and a narrower right hemisphere (Gauger et al., 1997); a smaller left Heschl's gyrus—an auditory processing region in the temporal lobes (Leonard et al., 2002); reduced total brain volume (Preis et al., 1998); and differences in cortical morphology in the form of gyral patterns (Clarke & Plante, 1998; Jackson & Plante, 1996). Regarding the last of these, gyral patterns in the brain are defined prenatally, which may bring researchers closer to a genetic link. In their evaluation of morphological patterns of the perisylvian language regions in the brains of parents of children with SLI and children with SLI in comparison to controls, Jackson and Plante (1996) found one particular pattern (out of four possible classifications of gyral patterns) that was more common in families with a history of SLI. An investigation of the inferior frontal gyrus (Clark & Plante, 1998) also found differences in patterns of morphology associated with a familial history of SLI. Affected individuals were more likely to have an extra sulcus in this region. This latter finding is consistent with an earlier postmortem investigation identifying polymicrogyra (small-scale structural malformations) in the brain of a child with SLI (Cohen et al., 1989).

These anatomical studies have provided an indication as to the cortical regions that may be affected. However, there are limitations associated with the methodology adopted by these studies. Most notably, these studies typically focus on particular brain regions selected in advance of examining the brain structure of individuals with SLI. The regions are selected as those likely to be affected by the disorder, for example only regions most commonly associated with language processing such as inferior frontal and temporal areas. As a consequence, there may be other regions outside these areas that also show anatomical differences yet remain undetected. A further disadvantage is that these broad regional studies do not provide a precise localization of these differences in the brain.

The introduction of a statistical analysis technique called *voxel-based morphometry* (VBM; Ashburner & Friston, 2000) for the analysis of structural brain images provides a more precise localization of differences between atypical and typical populations. This technique can be used to compare the brains of controls and disorder groups in order to identify precise locations where there is a difference between the two groups in the amount of gray or white matter (brain cells and connectivity, respectively). A recent study carried out by Soriano-Mas and colleagues (in press) using VBM investigated differences in gray and white matter volume in younger (5 to 11 years) and older (up to 17 years of age) children with SLI in comparison to controls. This study found that the overall amount of gray and white matter in young children with SLI was *higher* in comparison to controls. Overall, children with SLI had increased gray matter volume in the right perisylvian region of the left middle occipital gyrus. More differences were observed in younger children with SLI, in which several regions showing an increase of gray matter were detected. These areas included the entorhinal area, the temporopolar cortex, the caudate nucleus, the motor/precentral cortex, and the precuneus of the left hemisphere. Areas of white matter increase were also observed in these children, located in the

right medial frontal cortex and bilaterally in the middle temporal gyrus. However, the findings of this study contrast with an earlier study of young children with SLI carried out by Jancke and colleagues (2007), which detected regional *decreases* in white matter rather than increases. These decreases were detected in a network of left hemisphere regions comprising the motor cortex, the premotor cortex, and the superior temporal gyrus.

In sum, present studies indicate that SLI is associated with differences in brain structure, which may be linked to deviations from the typical trajectory of cortical development. Differences have been observed in multiple brain regions, and although at present there are too few studies from which to draw any consensus, there is the suggestion that these differences may be more prominent in young children with SLI rather than older children. This may be because older children with SLI are beginning to overcome their language disorder and are beginning to compensate for their linguistic difficulties, which may bring about associated changes in local brain structure. In the following section we discuss how functional studies of SLI tentatively reveal differences in how language is processed in SLI.

Functional Differences

To date, there are only two published functional imaging studies that investigate language processing in SLI. This small number of studies in comparison to the number of functional studies of other developmental disorders may be due to the heterogeneity of SLI, combined with the practical difficulties associated with scanning young children. The first published functional study of SLI, carried out by Hugdahl and colleagues (2004), investigated language processing in five Finnish family members with SLI in comparison to an age-matched sample of six controls. Participants carried out a passive listening task in which they listened to real words, vowel sounds, and pseudo words. Participants with SLI showed smaller and weaker patterns of activation in left hemisphere language regions in comparison to controls. Specifically, activations were concentrated within the upper posterior region of the superior temporal gyrus. There was no significant activation in the superior temporal sulcus or the medial temporal gyrus as seen in control participants. Friederici (2006) has suggested that this reduced activation in these regions that are typically engaged in speech perception may be related to the difficulties individuals with SLI have in decoding the phonological structure of linguistic information.

The second study, carried out by Ellis Weismer and colleagues (2005), investigated language and working memory processing in a group of adolescents with SLI. The task performed by participants involved an encoding task during which they listened to sentences, and a recognition task using final words from previously presented sentences. The results of this study also demonstrated that adolescents with SLI activated the same language regions in the left hemisphere as controls, but tended to show reduced activation in some of these regions. Specifically, hypoactivation was observed in the parietal region and the precentral sulcus during the encoding task and in the inferior frontal gyrus during the recognition task. Additional analysis carried out in this study also indicated that the coordination of activation of these brain regions during the recognition process was different from that observed in controls.

In sum, although the number of functional studies investigating SLI is limited, the two published studies show convergence. Individuals with SLI appear to activate the *same language regions in the left hemisphere*, but within this network, show regions that are *activated to a lesser extent* than in controls.

An Example of Testing Cognitive Theories at the Brain Level

At present, theories of the cause of SLI that purport a single underlying modular deficit are divided in terms of the nature of this deficit, which according to cognitive theory may be either grammar-specific or phonological. This divergence in cognitive theory can be tested using functional brain imaging. The design involves localizing brain regions that are involved in processing a particular type of sentence, namely *reversible sentences*. In these sentences, the subject and object of the sentence may be swapped or reversed and still produce a meaningful sentence—although the exact meaning of the sentence is changed as a result. For example, in the sentence "the leopard chases the lion," the subject and object of the sentence may be swapped to form the sentence "the lion chases the leopard," which remains a meaningful sentence. These sentences are harder to process than nonreversible sentences, for which swapping the subject and object of the sentence results in a semantically incoherent sentence. For example, swapping the subject and object of the sentence "the mouse eats the cheese" results in a semantically incongruent sentence, "the cheese eats the mouse."

Typically developing children and adults alike find reversible sentences harder to process than nonreversible sentences (Herriot, 1969; Kemper & Catlin, 1979; Slobin, 1966; Turner & Rometveit, 1967), and individuals with SLI show a pronounced difficulty in processing these sentences (van der Lely & Harris, 1990). By comparing functional activations produced while comprehending reversible and nonreversible sentences, it is possible to identify regions specific to the processing of reversible sentences. These are then candidate regions for deficits that would produce impairments in SLI. Are these regions involved in grammar or in phonology?

Richardson and colleagues (2010) succeeded in identifying a region that showed increased activation during the processing of reversible sentences in both typically developing children and adults. This region was on the left temporal-parietal boundary, which bridges a lateral region of the left posterior superior temporal gyrus and the neighboring inferior parietal region. Through the use of additional tasks carried out by the same participants, Richardson and colleagues were then able to localize this activation as being associated with phonological processing. In contrast to expectations, there was no additional activation for reversible sentences in comparison to nonreversible sentences in the left inferior frontal gyrus, which is typically associated with syntactic processing (Caplan et al., 1999, 2001; Cooke et al., 2001). The authors suggest that in the normal case, the processing of reversible sentences places additional demands upon phonological working memory. The finding implies that developmental anomalies in this area would provide one causal pathway that would explain why children with SLI experience exaggerated difficulties with reversible sentences. Of course, in SLI, these difficulties may have a syntactic or semantic cause; the current data are only suggestive that a temporal-parietal anomaly affecting phonological processing might be sufficient to explain sentence-level difficulties in SLI.

Structural and Functional Differences in SLI: Implications for Modularity

The multiple structural differences identified in SLI may at first suggest that a modular causal theory is unlikely. However, although multiple differences in brain structure have been detected, it is important to consider that some of these differences may be a consequence of the disorder rather than being causes of it. For instance, differences in gray matter or white matter in a given region may be due to the use and subsequent development of alternative processing systems in an attempt to compensate for initial difficulties. Alternatively, these differences may represent initial anomalies that normalize as children with SLI begin to overcome their disorder. The familial link between differences in cortical morphology observed when comparing individuals with SLI with either unaffected family members or typical individuals suggests that structural deviations of this type may be causally related, since the morphological patterns of gyri and sulci in the brain develop prenatally. However, not all individuals who have deviations in cortical morphology have SLI. Therefore, these differences may indicate a risk factor for language impairment, which when combined with other abnormalities impairs the functional processing of linguistic information. Structural differences in the asymmetry of the planum temporale may reflect an initial difference, but variations in this region may also be the outcome of developmental processes. In short, current studies of brain structure in SLI suggest that more than one structural abnormality may be associated with the disorder. However, more research is needed to identify the degree to which these abnormalities are linked with the behavioral profile of the disorder.

Although current functional imaging evidence in SLI is sparse, there is agreement between studies that individuals with SLI use the same language network as typically developing individuals. However, this network is underactive in individuals with SLI. This reduced pattern of functional activation may be linked to underlying structural abnormalities, but may also just reflect the fact that children are performing more poorly on the language tasks. Therefore, further studies that match for both chronological age and ability are required in the functional imaging of developmental disorders to establish whether this is the case. For comparison, a study with developmental dyslexics that used chronological age- and ability-matched participants did reveal hypoactivation in comparison to controls even when behavioral performance was controlled for (Hoeft et al., 2007). Reduced activation in dyslexia may therefore be more strongly associated with the disorder itself rather than due to poorer behavioral performance. If this held for SLI, it would suggest that *a network of functional regions is operating suboptimally*. Even if this is the case, it is then necessary to address whether each of these regions is impaired, or whether each is struggling with the same impoverished input (e.g., a deficit in phonological working memory would have negative consequences for other language processing systems

that rely upon this resource). Here we see echoes of the same issues we encountered at the end of the section on cognitive-level theories of modularity.

In sum, to determine whether a modular causal account is applicable to SLI, it is essential to establish how differences in brain structure and function are associated with increasingly typical or atypical behavioral performance. At present, too little is known about the brain basis of SLI in order to establish whether the behavioral profile of this disorder has a single cause or multiple underlying causes. However, given the heterogeneous nature of this disorder, it may be difficult to envisage a single underlying cause that accounts for both linguistic and nonlinguistic characteristics, although this may still be possible if parts of the language processing system affected in SLI are also engaged in nonlinguistic processing. Where multiple regions are held to account for SLI, we must account for why the deficits should co-occur, and how they are linked to the genetic basis of the disorder.

The Future

The future of modularity in the study of developmental disorders depends, to some extent, on which sense of the term prevails: the weaker sense, implying little more than functional specialization of processing components, or the stronger sense, implying as much as domain-specific, encapsulated, high-level, innate cognitive mechanisms. What is most often lacking in the field at present is explanation of the nature of the developmental process even if the cognitive system is postulated to contain modules. To understand how development can go wrong so that it produces uneven cognitive profiles, we must also understand how development works in the normal case.

We saw that the idea of modularity is deployed in several fields, but that in psychology, there are additional complications. The mind is implemented in the brain: Do the mind and brain exhibit the same type or degree of modularity? There is, in fact, a further complication, one that we have alluded to but skirted around: What is the relation of genes to the brain level? Kovas and Plomin (2006) identified three possible relations. First, a given set of genes may be involved in the development and/or regulation of one brain network, and the brain network may be involved in producing one cognitive process. This is the chain that makes it seem most likely that genetic variation or mutation could target a single cognitive process. Second, a given set of genes may be involved in the development and/or regulation of one brain network, but that brain network is then involved in producing multiple cognitive processes. Last, a given set of genes may be involved in the development and/or regulation of multiple brain networks, and each brain network may be involved in producing multiple cognitive processes. The last of these possibilities would make it least likely that there could be very specific cognitive outcomes via discrete genetic differences. Kovas and Plomin (2006) argued that on current evidence, it is indeed the last of these three options that seems most likely to relate genes and brain. For example, our review of the brain basis of SLI pointed to the involvement of multiple brain areas. Moreover, the candidate genes that have been implicated to date in the causes of developmental disorders such as dyslexia and SLI do not appear to have particularly specific functions but to be involved in the developmental placement of neurons, the general functioning of neurons, the signaling between these neurons, or producing experience-dependent change in connectivity. Although to date we have relatively little definitive knowledge about the domain specificity of genetic influences on the development of cortex, the mechanisms able to deliver innate modularity in high-level cognition have yet to be found (see, e.g., Marcus & Rabagliati, 2006, and Plomin & Kovas, 2006, for discussion on this point). If the mechanisms aren't forthcoming, the likelihood of innate high-level modules will have to be reduced, whatever the superficial plausibility of the evolutionary accounts supporting their origin, and accounts of developmental deficits that invoke such modules will have to be reconsidered.

We are left with a conundrum of sorts. Some developmental disorders do exhibit uneven cognitive profiles. That is, in many cases, individuals with disorders will possess some skills that fall within the normal range for their chronological age, or in cases of learning disability, in the normal range for the individual's overall mental age. Conversely, even where all abilities fall behind chronological-age expectations, some abilities may be more affected than others. Where does the unevenness come from?

Before this question can be answered, we need to know exactly what degree of unevenness needs to be explained. We saw in the section on WS that the degree of unevenness within abilities can be greater than expected, with fractionation continuing down inside modules as traditionally constructed. We saw that areas of apparent strength may be delivered by atypical underlying processes. And we saw that

uneven cognitive profiles may change across development. Similarly, we are not yet at a stage where we know the degree of atypicality of underlying brain structures in disorders, let alone their consequences for function. In the section on SLI, we saw that multiple structural differences have been identified in this heterogeneous disorder, but that language function appeared to involve underactivation of the same network of brain areas as in normal development. However, little work had been done to explore the brain activity associated with behavior in areas of apparent strength in SLI, such as nonverbal skills.

On the plus side, it is increasingly apparent what questions we need to have answers to, and the methods required to answer them. For a given disorder, we need sensitive measures of cognition across both areas of strength and areas of weakness, and these data must span development. We need similar data corresponding to the activity of functional brain networks. We need to know how genes influence the initial functional differentiation of the brain in the development of the fetus, how much the initial functions of the regions change as they acquire content through experience, and how much scope regions have to change their eventual specialization through experience. We need to know what impact genetic mutations or genetic variation have on this pattern of initial differentiation and experience-dependent specialization, the level at which these constraints operate (sensorimotor vs. high-level cognition), and the specificity of genetic influences to the content of each cognitive domain. For example, for SLI, do genetic influences target language-specific computational operations or generic computational resources in regions with input/output connectivity appropriate to integrate auditory, motor, and multimodal information? We need a better understanding of developmental interactions between different cognitive and brain systems that permit compensation or lead to deficit spread. We need to understand what differences lead to deficits compared to delays in particular cognitive abilities. And perhaps most importantly, for each disorder, we need to know the developmentally important moments to target intervention in order to stand the best chance of deflecting developmental trajectories toward more adaptive outcomes.

Today, we already have many of the scientific methods required to answer these questions. We have considered two of these methods in some detail, behavioral and brain imaging techniques. Others we have considered in less detail (genetics) or not at all. Two important complementary methods for understanding uneven cognitive profiles are developmental neurobiology and computational modeling. Here are a couple of examples. Developmental neurobiology has revealed how in the developmental disorder PKU, insufficient tyrosine in the blood has a differential impact on executive functions despite the fact that the entire brain receives too little tyrosine. Tyrosine is one of the 20 amino acids that are used by cells to synthesize proteins. The dopamine neurons that project to prefrontal cortex (the brain area responsible for executive functions) have higher rates of dopamine turnover than other brain regions and, because tyrosine is a metabolic precursor of dopamine, frontal systems are sensitive to modest reductions in tyrosine that are too small to affect the rest of the brain (Diamond & Amso, 2008). Second, using mouse models, Matzel, Babiarz, Townsend, Grossman, and Grumet (2008) found that a NRCAM gene knockout mouse that was unable to produce neuronal cell-adhesion molecule (NRCAM) showed impairments in only one of five learning tasks tested compared to normal mice (namely passive avoidance but not Lashley maze, odor discrimination, spatial water maze, or fear conditioning). Despite the widespread involvement of NRCAM in cell adhesion and migration, axonal growth, guidance, target recognition, and synapse formation across the whole brain, once more the result was an uneven profile. In this mouse model, NRCAM did not appear to play a central role in the regulation of general cognitive abilities, only in regulating impulsivity.

Computational models are also an essential tool to further our understanding of how atypical processing constraints can alter developmental trajectories and functional specialization. Implementation is required for the implications of (atypical) development in complex systems to become clear. For example, Baughman and Thomas (2008) used a dynamic systems framework to capture how an initially focal deficit might spread across a cognitive system during development, depending on the degree of modularity present in the architecture of the system. Thomas and Karmiloff-Smith (2003) investigated the precise processing constraints that would reproduce patterns of developmental deficits in a particular aspect of language development. And Thomas and Richardson (2006) explored the computational conditions that might lead to the emergence of atypical modular structures in distributed processing systems.

Together these additional methods have produced at least two possible answers to why most developmental disorders exhibit some similarities to the typical developmental profile. First, some brain systems (and cognitive domains that supervene upon them) may be differentially sensitive to certain genetic factors even though the genetic influences are brain-wide. Some domains may indeed be (initially) less impaired than others. Second, the genes that influence the development of the largest-scale constraints in the brain, such as the broad wiring pattern of the cortex that involves its inputs and outputs, may not vary in disorders that produce viable embryos (see Brock, 2007, and Thomas & Richardson, 2006). If the inputs and outputs to the systems are the same, the information that shapes the development of mental representations will be similar. Cognitive profiles may share similarities because in most cases, typically and atypically developing children are trying to master the same sorts of tasks in their subjective physical and social environments using the same sorts of information, even if the details of the mechanisms with which they try to solve these problems may differ.

We finish by returning to a puzzle that we raised earlier, concerning the origin of the uneven cognitive profile observed in dyslexia. How is it possible for a selective deficit for reading to be inherited when reading itself is a recent cultural invention? A sketch of an answer to this question will serve to give a flavor of what future explanations of developmental disorders might look like (see Mareschal et al., 2007, for more details of this account).

The reading system in adults is evidence that practice in the life of an individual can wire together a new high-level cognitive system, in this case one that integrates an appropriate substrate from the visual system with the spoken language system. The emergence of the reading system relies on integration between multiple systems, including the processing of spoken forms of words, written form of words, and word meanings. Weaknesses in the development of any these skills could in principle make it harder to learn to read, so we might expect there to be different varieties of reading disability, consistent with the observed heterogeneity in this disorder. Most children with dyslexia appear to have problems representing the component sounds of words (that is, breaking words up into phonemes). While this problem may be the initial cause, subsequently there are likely to be complex interactions with the environment: children who are finding it hard to read tend to read less, and so gain less practice in learning to read.

In all likelihood, the genes responsible for this type of cognitive variation have been in the gene pool for a long, long time. If you can only represent words in larger chunks of sound, this won't particularly affect your oral skills. Maybe you will name pictures a few milliseconds more slowly, a subtle difference at best. Before the invention of reading, individuals with these genes would to all intents and purposes have appeared to have an *even cognitive profile*.

It is problematic to have these chunky phonological representations only when you need to link the component sounds of words to component written letters (or groups of letters). And it is particularly problematic only when the relationship between component sounds and letter clusters is a tricky one, like it is in English. Where the relationship is simpler, like in Italian, no particular problem in learning to read emerges in these individuals. In other words, the uneven cognitive profile observed in dyslexia is the result of cultural innovation to learn particular tasks (reading English) for which some proportion of the population won't have the right sorts of speech representations. They won't have the right sorts of representations under normal circumstances, but this does *not* rule out the possibility that the right behavioral intervention at the right age may provide them with phonological representations appropriate for learning to read—genetic predispositions do not inevitably determine behavioral outcomes. One solution would be to simplify the spelling of English. If our nostalgia for the language of Shakespeare could be overcome (among other obstacles), the influence of the "genes for dyslexia" on our reading behavior would disappear and the even cognitive profile of individuals with these genotypes would be restored.

Summary

- Developmental disorders may be split into disorders of a known genetic origin and disorders that are defined on behavioral grounds. Uneven cognitive profiles can be found in the presence of learning disability (e.g., WS) or in its absence (e.g., SLI).
- Modularity is the idea that a system is made up of functionally specialized parts. It has been proposed that uneven profiles may be explained in

terms of damage to one or more specialized parts of the cognitive system.

• For developmental disorders, this idea is controversial, because it is unclear when functional specialization occurs in development. Explanations are complicated by the fact that the notion of modularity may apply differently to cognitive and neural levels of description.

• Four types of modular explanations of developmental deficits can be discerned, which place different degrees of emphasis on the developmental process, and which predict more or less selective behavioral deficits.

• WS is an example of a disorder of known genetic origin demonstrating an uneven cognitive profile as well as learning disability. Research has demonstrated no straightforward modular explanation of the behavioral deficits found in the disorder. We considered language development and face recognition as examples of recent cognitive-level explanations in WS.

• SLI is an example of a behaviorally defined disorder, where some researchers argue that the language system alone (or some subcomponent within it) fails to develop normally. Other researchers appeal to more domain-general processing deficits that exert a greater eventual impact on language.

• SLI offers an opportunity to consider how the idea of modularity might apply at the brain level, via recent findings from structural and fMRI. Brain-based views of functional specialization are inconsistent with cognitive views in two ways: multiple brain regions are typically engaged in a given cognitive task, and each brain region is frequently engaged by multiple tasks.

• Individuals with SLI show subtle and inconsistent differences in the structure of their brains, but there is as yet limited evidence on functional differences. Current evidence indicates that similar language regions become active, but to a reduced extent.

• The future study of developmental disorders involves convergent evidence from multiple methodologies and disciplines, including behavioral testing, brain imaging, genetics, and computational modeling.

Questions for Future Research

• Are uneven cognitive profiles in behaviorally defined disorders on a continuum with the strengths and weaknesses found in typically developing children?

• What are the genetic effects on early brain development that contribute to uneven cognitive profiles?

• To what extent does the subsequent process of cognitive development change the nature of the even profile, by spreading deficits via interactions or attenuating them via compensation?

• To what extent can exposure to particular environments (such as interventions) remove weaknesses in uneven profiles or build on strengths?

• How will an increasing understanding of brain function from neuroimaging methods inform our understanding of the origins of developmental deficits?

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Notes

1. A standardized test is a psychological test that has been given to a large number of children and adults so that the range of performance at each age can be established. The performance of any given individual can then be compared to the standardization sample to see whether this individual falls within the normal range for his or her age. Intelligence tests are usually made up of a battery of standardized subtests that examine different abilities (e.g., language, memory, attention, visuospatial skills, motor skills, and so forth).

2. The idea that modularity is a *restriction on causal scope* works as follows. At the finest grain of functional elements in the system, some clusters of elements interact only locally with nearby elements, whereas other elements also interact with more distant elements. The clusters of only locally interacting elements are viewed as the modules, while the elements with longer causal links are viewed as the connections between modules. This formulation allows for a graded concept of modularity rather than an all-or-none concept, where the degree of modularity depends on the distribution of ranges of causal interactions that the elements exhibit.

3. The terminology "a gene for *X*," where *X* is some behavior, has become increasingly common in the media, but also to some extent in behavioral genetics and cognitive neuroscience. Within science, the term is intended as shorthand and its meaning is fairly specific. It means that having versus not having some particular gene variant explains a statistically significant amount of the variation in behavior *X*. The amount of variation explained, although statistically reliable, is typically rather small, meaning that many other genes must also be involved in producing variation in behavior *X*, as well as environmental factors.

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