CHAPTER 2

Explaining Developmental Communication Disorders

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A hallmark of Larry Shriberg's work has been a commitment to the notion that the subtypes or dimensions of developmental speech sound disorders are organized on the basis of their etiology, and that the surface features of these disorders will reflect this multiple causal organization. Certain key points of this perspective are particularly prominent.

First, etiology is key to the study of developmental communication disorders. This statement may sound trite now. But in the 1970s and to some degree even today, some argued that etiology is not relevant, particularly for clinical intervention in behavioral disorders such as developmental speech sound disorders. Shriberg's early research was concerned with psychodynamic factors in speech sound disorders and expanded to include auditory (otitus media), motor (developmental apraxia of speech), and familial/genetic genetic factors. Each of these represented a subtype of developmental speech sound disorder.

A second feature of his work is his emphasis on development and developmental history in understanding the different forms of speech sound disorders. Presumably, different causal factors shape different developmental trajectories and thus, developmental course must be considered.

Finally, Shriberg is known for his concern for careful measurement and description of behavior. Just as etiology is revealed in development, it should be revealed in the fine structure of the behavior of the speaker.

Running through these ideas is the notion that we must develop comprehensive accounts of development and behavior that are grounded in causal explanations. Thus, advancement in our study of human communication and disorders should not merely focus on simple surface accounts of communication behaviors. In this chapter we accept many of Shriberg's key principles as we consider how we might construct a framework for research on developmental communication disorders that is rooted in

explanatory sources for individual differences. In so doing we hope to demonstrate that there are alternatives for proceeding with this, and these alternatives have strong roots in several branches of philosophy. Some of the philosophical choices that we will take diverge from those found in Shriberg's work. Consistent with Shriberg, we intend to show that our research rests upon assumptions about communication behavior, development, and individual differences. Additionally, Shriberg's influence can be seen in our adherence to multiple causal systems and developmental history. We will cast these themes into an account that we hope will further scholarship in this field, much as Shriberg has enriched it.

The Challenge of Explanation in Developmental Communication Disorders

Much of science is concerned with generating explanations for observations in the world. And yet for centuries, philosophers have debated the nature of scientific explanation. Within this paper we draw upon some of this literature to present a rather comprehensive framework for conducting scientific research on developmental communication disorders. Our use of the term "developmental communication disorder" is intended to encompass all forms of communication disorders that arise during development, including developmental speech sound disorders which have been the focus of Shriberg's research. Although the factors explaining speech sound disorders may differ from those involving language or fluency, the framework for these explanations is the same. Explaining all forms of developmental disorders is challenging because the affected individual will not previously have had an ambiguous unaffected state. Furthermore, most developmental disorders represent variants in developmental trajectories, and thus they blend in with normal developmental patterns. These are challenges common to characterizing and explaining developmental speech sound disorders, developmental language disorders, and stuttering.

Explanations in science can take various forms, but most focus on either asking how something works or **why** it works as it does (Chater & Oaksford, 1999). In contemporary scientific research, the most common question posed is the "how" question. For example, we can ask how humans produce vocalizations. Our coherent explanation: we use mechanisms of respiration, airflow and pressure, vocal fold dynamics, and acoustic resonance. Note this account does not explain why humans vocalize or why the human larynx is positioned in the vocal tract the way it is. Asking "why" questions of this sort takes us into the realm of teleology (Mayr, 2004) Why questions produce explanations about the role of a system, process or object in serving some end state or goal. Mayr (2004) and Mundale and Bechtel (1996) argue that although one can attempt to generate mechanistic answers to how questions without considering why questions, understanding how mechanisms work ultimately also addresses the systems' functional roles. Therefore why questions become entailed in the how questions. For instance, explaining how the larynx functions in the account above will ultimately incorporate the why as well—the place of the larynx in the vocal tract and the importance of this position in human vocal performance, resulting in a teleologic answer involving evolution (e.g., Lieberman, 2007). Within this paper we aim to address both forms of these explanations as we attempt to answer why and how it is that some chil-

dren are said to have a developmental communication disorder. The key is that we need to be clear when we are addressing why questions versus how questions.

A Starting Point for Explanation of Developmental Communication Disorders

As we present our model for explanatory research on communication disorders, it may be helpful to establish a prototype of current scientific practice. As with all models, this one aims to capture essential features and highlight the underlying premises. It is unlikely that this model exemplifies the thinking of any particular researcher. Thus, we might view this as an initial model of research practice from which we can build our particular model.

This model begins with the notion that variations in communication development, and resulting communication function, comprise classes of different types of abnormal communication and that each abnormal form of variation contrasts with a normal form of communication. Each class of abnormal communication development is formed by a flaw in the operation of a mechanism necessary for attaining the normal state. The abnormal operations of each these flawed mechanisms will result in distinctive properties (markers) in the communication behaviors (phenotypes) of the affected individuals. Although not often explicitly stated, it seems that the normal state is viewed as largely invariant—or if there is variation in the normal state, it is just the result of noise in the causal system. Scarr (1992) described this view as a platonic conception of normality wherein "individual differences . . . are considered to be unimportant variations on the ideal type" (p. 1). Within this platonic framework, an organism's healthy state incorporates no variation from the ideal other than functionally unimportant noise. Following from this, illness represents a situation in which the variations are important. Furthermore, presumably the disruptions in the causal systems are no longer noise but rather dysfunctional (broken) systems. Ill health then arises out of these dysfunctional flaws in the causal systems, and ill health can be understood by discovery of these broken systems. Health and ill health, then, represent very different kinds of individual differences.

This perspective, just summarized, is largely consistent with a viewpoint which the philosophy of medicine referred to as neutralism or descriptivism (Boorse, 1977, 1987). This position claims that the constructs of health and ill health refer to natural properties of organisms and not statements reflecting social/cultural values. Within this view it is expected that there are flaws in the causal system that result in disease. In this account, the explanation of ill health requires only one type of explanation that rests primarily on how it is that the processes that give rise to the healthy state are flawed.

Toward an Alternative Explanatory Account

For a number of years, one of us (Tomblin, 2006) has argued against the neutralist model described above and instead advocated a weak normativist position. This model (Figure 2–1) recognizes two somewhat independent systems (biobehavioral and cultural) that each play a role in the explanation of communication disorders. This requirement of two different kinds of explanation is an important distinction between our alternative account and the standard account just described. At the interface of

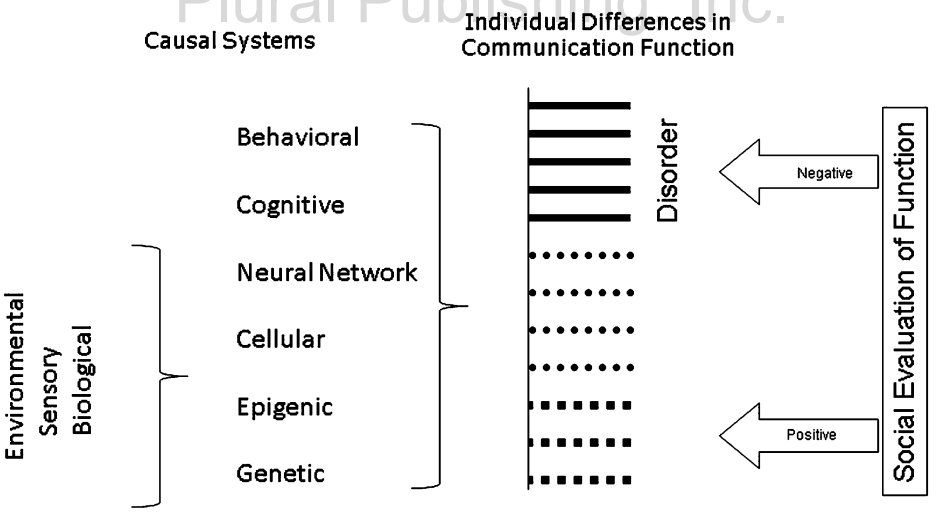

Figure 2–1. Component sources for explanation of communication disorder where individual differences are explained by multiple interactive systems and individual differences are differentially valued via social value systems.

these two explanatory systems are individual differences in communication development and function. A full explanation of a developmental communication disorder requires the use of both the biobehavioral and cultural aspects of this model. The explanatory role of these two domains in relation to individual differences is, we contend, different.

Explaining Disorder via Social Values

On the right-hand side of Figure 2–1, we show that social values are applied to individual differences in communication in such a way that some levels or some forms of communication function have greater negative consequences to the individual than others. The role of social/cultural values in a comprehensive explanation of developmental communication therefore, provides an explanation for why we claim that a given child presents with a communication disorder. Within our normativist model, we would answer this "why" question by stating "because the child's communication skills place this child at risk for culturally based disvalue." Put simply, the child's differences are likely to result in undesirable outcomes for the child. A normative account accepts the position that concepts of health and illness are inherently value judgments arising from cultural beliefs and the goals societies have for their members. Thus, we can consider health as a state in which one functions within the social expectations of our society; ill health represents an inability to meet these expectations. A developmental communication disorder is a status assigned to the individual by either the individual herself or by others in society based on cultural values. We immediately can see that any explanation of communication disorder must incorporate cultural values and pro-

vide statements regarding the relationships between communication performance and the capacity of an individual to participate in socially valued roles.

It can be argued that communication is a universal characteristic of humans, much like bipedalism and opposable thumbs, and thus cultural values are not necessary to claim that limitations in these traits represent ill health. However, if we envision a world without gravity, legs as we know them are of no use. It could be possible that a society in such a world would no longer view the absence of legs as a condition of poor health. Likewise, we might have a world where speech and hearing are not necessary for communication, and therefore their absence is no longer viewed as unhealthy. This last example is not hypothetical, but rather is very well exemplified in the Deaf community where speech and hearing are in some circles disvalued, and lack of hearing and speech do not constitute ill health.

How and why cultural values carve up individual behavioral differences into more or less desirable states is an interesting problem that draws on principles and theories of sociology, particularly social deviance (Erikson, 1962). Thus, construing health and ill health as the normative product of human culture is not dismissive of science. Instead, it points to the necessity of constructing scientific theories that incorporate both why values are applied to behaviors and also why societies would do this. It is most likely that societies will disvalue behaviors that are deleterious to the well-being of the society. If so, as communication becomes important to a society, individual differences in communication skill will become the target of value-based judgments. This implies that the way in which a social group carves up individual differences in communication—along with the functions served by such communication—will differ not only between societies at a point in time but also within a particular society across time. Today technologic changes have greatly affected our daily lives regarding the amount and import of communication. The bases for social evaluation of behaviors are not arbitrary, and explanations should elucidate the dynamics that produce and support social systems.

Thus, a full explanation of human communication requires understanding how communication functions are directly and indirectly associated with cultural values. The most obvious of these is the importance of communication for interpersonal social interaction. Theorists such as Searle (1989) and Grice (1975), who have broadly addressed facets of pragmatics, have provided an outline of these functions with regard to interpersonal communication settings. Communication also serves to support many other functions in our lives. Indeed, communication is an important means by which we establish and maintain our cultural membership. Thus, it is not just a vehicle for social interaction, it is also a principal means for establishing cultural and subcultural identity. Since communication also serves as the basic tool for instruction and learning, it is a tool of acculturation. We have considerable evidence that children's language and communication abilities are very strongly associated with school outcomes (Bishop & Edmundson, 1987; Hall & Tomblin, 1978; Nation & Aram, 1980; Snowling et al., 2001; Stothard et al., 1998; Young et al., 2002). In fact for children with milder forms of communication disorder, the impact of communication on learning may provide the strongest case for considering these as forms of ill health. Interestingly the distinction made by Shriberg (Chapter 1) between *Speech Delay* and *Speech Errors* highlights this point by noting that the latter is much less likely to be associated with comorbid language and/or learning problems.

This perspective leads to an important shift in how we study communication disorder. As we noted previously, the prominent strategy has been to assume that by careful study of communication systems, we can identify classes of disorder. That is, the diathesis between "normal" and "abnormal" will be contained in the behavioral system itself; furthermore, this abnormality will extend to the causal systems that affect these behaviors. We contend that the locus of the disorder in a communication disorder will not be found in the characteristics or behavior of the individual, but rather in the cultural context. Thus, the scientific theories used to explain how and why some aspects of communication comprise disorder need not be the same as those used to explain communication function and its individual differences. Thus, we now shift our focus to the second explanatory component in our account, specifically, the factors that give rise to individual differences.

Individual Differences and Communication Disorder

Figure 2–1 shows that individual differences play a central role in our model. Furthermore, we argue that individual differences do not come in two types—one representing abnormal variation based on a defect and the other simply due to noise. Instead, we would argue that all individual differences have the same explanatory basis and the same value within the natural system.

Within an evolutionary perspective, individual differences are the means by which species form, adapt, and survive. Monocultures are well known to be highly vulnerable to the survival of the strain. Furthermore, as future environments cannot be foreseen, all forms of this variation have equal potential value regarding survival. Thus, there is no place in nature to carve out forms of individual differences that are inherently normal versus disordered. By extension, we should not expect to discover the nature of a communication disorder by simply examining patterns or characteristics or flaws in systems that produce individual differences. In our account, these individual differences become disvalued, and thus disorder exists only in the context of a culture.

We also noted earlier that individual differences within the normal range are often ignored or considered noise. This viewpoint has been particularly prominent with respect to speech and language: it is assumed that full speech and language competence is attained by nearly all adults across languages and that the only individual differences in adulthood are found in those representing speech and language impairments. A common tenet within modern linguistic theory is that spoken language is a universal human trait. Thus, most adult speakers and listeners are treated uniformly with regard to language unless the language system is impaired. This assumption of uniformity of adult language status has then been used to claim that individual differences found during development in typically developing children are temporary and will be resolved by the time the child reaches adulthood. That is, these individual differences represent noise in developmental trajectories. If the differences are not resolved by adulthood when uniformity is expected then they must represent a disordered system. But is this assumption valid? In fact, a vast amount of data in psycholinguistic research has shown that adults do vary on lexical abilities and grammatical processing skills, even among the fairly homogenous population of college-aged adults (see, for instance, Gernsbacher & Faust, 1991; Pearlmutter & Macdonald, 1995).

We have followed a large group of children from kindergarten through age 16—an age most would view as the terminus of child language development. During this time the children's language abilities were measured at five time points: kindergarten, second, fourth, eighth, and tenth grade. After converting the raw scores into Rasch ability scores (Meslevy & Bock, 1990), we examined the growth trajectories of the children who represented a wide range of ability when initially sampled. Figure 2–2 shows that the pattern of language growth over time is remarkably similar regardless of the children's ability level. Individual differences are found at each age level, and there is no evidence that all children within the normal range converge on a common point —leaving individual differences at maturity to those who were initially language impaired. Furthermore, the language ability among children is stable, demonstrating that the variance in language abilities among individuals is not simply measurement error, but rather is systematic and persistent across both development and communication tasks. This stability of relative performance in a coherent behavioral domain is often used as evidence of a behavioral trait. Scarr (1992) for example differentiated between behavioral traits that are enduring and stable across many situations and years versus contextual and situational behaviors that are specific to time and context. Scarr refers to the former stable traits as phenotypes.

We can see now that many individual differences in the development of communication behavior are systematic and therefore should be open to the kind of explanation that draws upon biology, neuroscience and

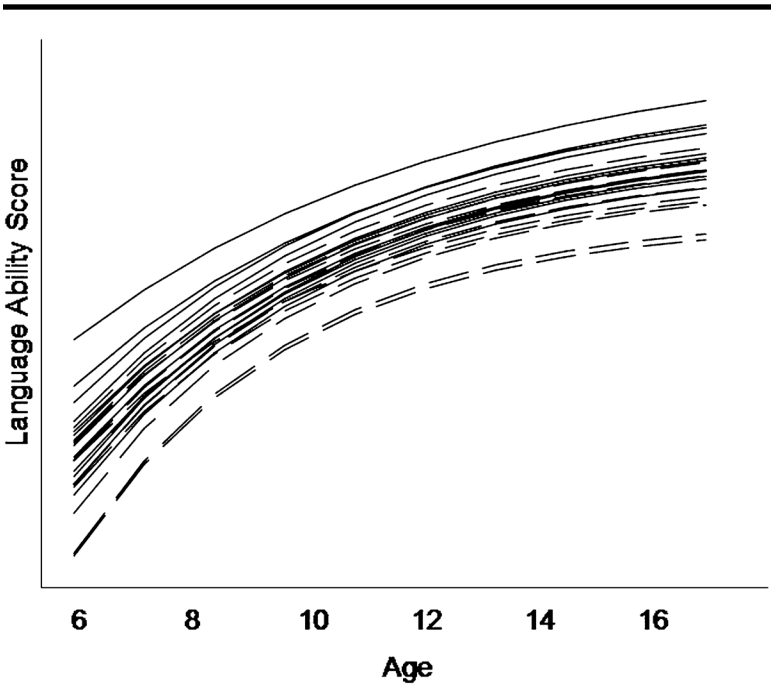

Figure 2–2. A series of language growth trajectories for a random sample of individual during the school years.

cognitive science. The explanatory accounts will not be reserved for explaining developmental communication disorder, but rather will simply explain individual differences in communication development in general. By highlighting the importance of understanding individual differences as potential sources of communication disorders, researchers concerned with such disorders can incorporate theoretical accounts of individual differences into the fields of natural science rather than describing them as ill health. It is to this end that we now consider how such a general theory spanning biology, neuroscience and cognitive science might provide an explanatory framework for individual differences in communication development.

Explanations of Individual Differences That Incorporate Multiple Systems

In the last decade, we have experienced a remarkable change in our ability to ask—and to some degree answer—questions concerning the nature of several systems important to the development and use of language. Cognitive science offers crucial tools involving computational modeling and theories ranging from those that are linguistically and symbolically based to those that are more distributed and statistically based. These tools and theories have resulted in a vibrant discussion of the possible nature of language development, processing, and representation. In addition, a field of cognitive neuroscience has emerged. It is now possible to measure brain function in language usage tasks: event related potentials (ERPs), functional magnetic resonance imaging (fMRI), and recently near-infrared spectroscopy (NIRS). These techniques allow us to examine spatial and temporal properties of brain activity associated with language usage tasks. This ability to study brain function—coupled with continued advances in structural brain imaging such as diffusion tensor imaging—has allowed us to move from merely speculating about brain systems and language to forming data-driven theory. Advances in molecular genetics and biology have also resulted in research concerned with the discovery of genetic bases for cognition, particularly cognitive development and disorders (see also Chapter 3). One cannot ask about genetics and behavior without also asking about cultural and biological environmental influences, which may bring us back to theories of communication development such as usage-based theories (Tomasello, 2003) and exemplar-based approaches (Barsalou, 1990) that are now emphasizing experiential input.

We can see that contemporary scholars in communication sciences and disorders have a rich array of research methods, data, and theory from which to build an account of the origins of individual differences in communication development. In fact, rather than having a shortage of potential explanatory theories we now have an ever expanding array of mechanisms and processes that we can incorporate into a theoretical account of individual differences. The challenge then is how to deal with the complexity that results from this richness of what could be viewed as multiple scientific fields of study. One starting point has been to consider each of these theories as focusing on different explanatory levels or systems for the ultimate product of communication performance. We may conceive of these different methodological and theoretical domains as forming a system comprising levels of explanation as shown in Figure 2–2. In this figure, we hypothesize that there may be at least 7 levels that can be used to study and explain individual differences in communication development.

To begin, we have identified one level that operates in parallel with all the others. This level concerns the sensory, physical, and biological environment of the child. The sensory environment provides the child with stimuli concerning objects and events in the world, including social artifacts such as speech and language. The physical and biological environments comprise a wide range of inputs ranging from gravity to nutritional substances to toxins and even "foreign" beneficial and deleterious flora and fauna that live within us. The genetic level is concerned with heritable biological information contained within DNA sequences as well as transcription and translation of this information into RNA and protein. The epigenetic level is concerned with heritable changes in DNA transcription. The cellular level is concerned in our case with biological functions (metabolism, axon growth, modification synaptic spines, etc.) operating within the neuron. The network level concerns the connectivity of neurons to form the brain, and within these networks the behavior of these neural networks beginning with the synapse. The cognitive level represents mental states and processes that involve such things as learning, memory, recall, intent, representations, etc. The behavioral level concerns all those coherent behavior patterns that yield communication, as well as the change in these patterns that come with development.

Reductionist Explanations of Individual Differences

Once we have identified these levels, we need to determine how each level relates to the others as we attempt to explain individual differences. Some philosophers of science use theory reduction to account for multiple levels of scientific theories and methods. Theory reduction assumes that the scientific statements and data at each level in the hierarchy can independently explain the behavior of the system, but that lower levels provide more explanatory detail than the higher levels, and that theories at higher levels can be translated into comparable theories at more basic levels using bridging laws (Bechtel, 1994); (Mayr, 2004). Thus, all characterizations at a higher level can be also accomplished at a more basic level. This means that each level can explain a phenomenon, but that more basic levels will provide more detail.

The use of theory reduction is called reductionism. For example, we can use any of various levels to describe individual differences in speech production. We can describe speech production broadly using the vocabulary of transcriptional phonetics, finer acoustic descriptions, descriptions of articulatory gestures or even at a more basic level the kinematic features of gestures. Each level is viewed as being concerned with speech sound production, and it should be possible to relate any one level to another. The differences have to do with the grain size of the description, and the choice of level may be determined by the needs of the research question.

An important feature of theory reduction is the implication that a theory at one level can be developed independently of another level. In the prior example regarding speech, it has been common to differentiate phonological theories, transcriptional phonetics, acoustic phonetics, and physiological phonetics. Scholars have been very comfortable working in one of these areas, but with the belief that bridging laws will connect their work to that of others.

Thus, we see a common approach to situations in which there are multiple scientific theories regarding a complex function: identify a level that seems to provide theoretical and methodological strength, and then develop theory through bridging laws to

account for a higher level. We see this practice quite clearly in much of the research on developmental communication disorders—linguistic or cognitive data and theory are used to explain speech and language behaviors and development of children with developmental communication disorders. Such a research strategy allows investigators to work within their respective research domain free of the constraints of the other levels. Thus, we can have studies of neural structure, connectivity, and network function associated with individual differences in communication development. Likewise, we can have research on the genetics of these individual differences. However, this approach assumes that a common functional system spans these levels. Since the functional system in this case is the process of speech and language development, we would conclude that there are brain systems for speech and language development and that there might be speech and language genes whose functions can be bridged to brain systems. In fact, this very picture of language development is represented in nativist accounts of language development (Hauser et al., 2002; Pinker, 1994). Reductionism, however, has been broadly and extensively criticized as inadequate either as a description or prescription for scientific inquiry of most complex systems in nature, and biological systems in particular (Hull, 1974).

Mechanistic Explanations of Individual Differences

Many of those who have argued against reductionism in biological and cognitive systems have advocated an alternative approach referred to as mechanistic explanation (Salmon, 1984). A mechanistic explanation, like theory reduction, accepts that complex systems or mechanisms have parts that can even be described in terms of levels (Bechtel, 1994). Bechtel has characterized a mechanism as

> . . . a structure performing a function in virtue of its component parts, component operations, and their organization. The orchestrated functioning of the mechanism is responsible for one or more phenomena. (Bechtel & Abrahamsen, 2005, p. 423)

Thus, mechanisms perform functions and have parts. Each part or submechanism can be considered as having a function as well; however, the functions of submechanisms will not be the same as those of the whole mechanism (Craver, 2001). This was not true with our account of reductionism. Within the context of communication, the function of a gene that in some way influences language development is not the same as the function of cognitive systems. Thus, there will not be bridging rules that allow translation from one level to another, and we would be advised not to talk about genes being language genes or neurons being language neurons (Fisher, 2006).

Much of mechanistic explanation is used to address the "how" questions concerning a system. However, the function of any submechanism must be considered within the context of the broader system (Craver, 2001). The function of vocal folds can be to protect the lungs or to produce the acoustic source for speech, depending on whether we are explaining swallowing and respiration or speech production. The fact that the subsystems can have different roles depending on explanatory accounts leads us to realize that fundamental to explaining how a complex system generates a particular function requires also considering why certain functions come to be. Thus, even in

mechanistic accounts of "how" we can be led to consider "why" in order to produce a complete explanation of, for instance, laryngeal function in human vocalization.

The layered structure of complex systems that are often the object of mechanistic accounts allow for the interactions of the functions of different underlying mechanisms. The importance of interactions of components yielding the behavioral functions of the mechanism can be seen in Glennan's definition of a mechanism.

> A mechanism for a behavior is a complex system that produces that behavior by the interaction of a number of parts, where the interactions between parts can be characterized by direct, invariant, change-relating generalizations. (Glennan, 2002, p. 344)

These are not simply additive, such that the mechanism is an aggregation of the parts. Rather, the interactions are nonlinear and often involve cooperative or competitive interactions (Craver, 2001). Thus, mechanisms in this sense produce emergent behaviors.

Using the mechanistic perspective to explain individual differences in communication, we will discover that our way of doing research will change. We cannot assume that there will be simple and predictable ways to move from one level of explanation to another and that each level is concerned with the same thing. Individual differences may not arise from some part being essentially flawed and thus disordered. Rather, the manner in which some subsystem contributes to the variation in communication function is likely to be a feature of a complex interaction, and the "flaw" emerges out of interactions in the system. Sickle cell anemia is associated with a point mutation in the recessive β-globin gene. Although homozygotes with this mutation are prone to abnormal deformation of the red blood cells (sickling) in situations with low oxygen, this mutation is actually beneficial to heterozygotes exposed to malaria. Thus, the functional characteristics of this mutation depend on (1) the interaction of one allele of the β-globin gene with the homologous allele and (2) whether the person is exposed to malaria. Thus, in our framework, the individual differences in red blood cell function that caused the socially disvalued state of pain and illness are emergent properties arising from system level interactions.

It would be easy at this point to conclude that complex systems defy explanation or that these explanations cannot incorporate any analytic methods or accounts, but rather they can be only global holistic descriptions of complex functions with few details. Admittedly, our explanations will be complex. It is also likely that these explanations may often be incomplete. But it is still possible to functionally decompose a complex system and develop an explanation for the emergence of complex behavior that is grounded on well understood mechanisms. Following is one example of such an explanation.

Long-Term Potentiation, BDNF and Listening Comprehension—A Case Example

A common communication function is that of listening to a passage and later recalling this information. We often describe this as a form of comprehension. In order to accomplish this task, many processes and subprocesses must occur. One of these has to do with the retention of the information that was heard. There is considerable evidence that the hippocampal learning system

plays a role in this. This learning system is widely believed to contribute to declarative learning—learning facts or recalling specific events (Squire, 1992). Learning within the hippocampus has been studied extensively within the context of a particular type of neural plasticity called long-term potentiation (LTP) (Bliss & Collingridge, 1993). LTP represents a change in the efficiency of transmission between two neurons at a synaptic site as a product of simultaneous high-frequency firing of the synapse. Thus, it is an example of Hebbian learning. This change in synaptic efficiency can have short term or longer term persistence. In order for this learning to persist over hours and days structural changes must occur in the postsynaptic spine in which the spine actually enlarges. A part of this structural change in response to synaptic activity is dependent upon the actions of proteins. One of these proteins is BDNF which is coded by the *BDNF* gene. Recently, Soule and colleagues (2006) have summarized a model of BDNF action in LTP. In brief, activation of the post synaptic site results in BDNF being secreted where it then docks on both pre and postsynaptic receptors involved in the activity. Within the postsynaptic neuron, this docking triggers an activity-dependent cascade within the neuron that results in increases in another protein called ARC. ARC concentrations in the active synaptic site contribute to the production of actin proteins at the postsynaptic site, resulting in the enlargement of the postsynaptic spine, in turn resulting in consolidation of learning. Thus, secretion of BDNF plays a mechanistic role in memory consolidation. This case about BDNF so far has focused on mechanisms at the neuron (cell) and gene level. The case, however, does move to higher levels in the mechanistic hierarchy. It is well known that in the listening comprehension task described earlier, some people perform better than others. Recent research has shown that these individual differences in comprehension are associated with allelic variation in the *BDNF* gene (Egan et al., 2003)—a genetic level characteristic. Individuals with one form of the *BDNF* gene secrete less BDNF than those with another form—a cellular characteristic. Those individuals with the BDNF variant associated with less secretion also show lower levels of comprehension than those with the form that is secreted at higher levels. Thus, secreting less BDNF has functional consequences in this cascade from the neuron, to the neural network, through cognitive processes to communication behavior. The effects are not directly upon the language behavior or on a system identified as a language system. Thus, we can't bridge the gene function to language; rather language behaviors and the individual differences therein emerge from the complex mechanistic cascade across these levels. This story about BDNF demonstrates that explanation can be accomplished within a complex system.

Implications for Future Research on Developmental Communication Disorders

This mechanistic approach to explanation of individual differences shows that the manner in which research is conducted in the field of communication disorders will need to change. We can no longer be comfortable reducing the problem to one level of the system and assuming that, in time, we can translate our findings to either higher or lower levels of the causal chain. Rather, we will need to become familiar with theories that span multiple levels of a complex system in a truly interdisciplinary fashion (e.g., Christiansen & Kirby, 2003; Mareschal

et al., 2006, see p. 8). Furthermore, we cannot develop research strategies that assume we can work sequentially through these levels as though peeling an onion. Rather, ideally, we should study these systems at multiple levels in parallel. Given that research is often hampered by limited resources, this objective of full parallelism in our research is perhaps not likely. Researchers will have to choose aspects of the problem and prioritize their inquiry based on the availability of resources. This process is likely similar to an artist drawing a picture where a rough sketch of the whole system is developed and then certain local details filled in. In this regard, a researcher may pick a particular level at which the multilevel system is entered. The choice of the entry point should be grounded on good theoretical evidence that the mechanisms at that level are likely to contribute to individual differences in communication development. But another factor is the technologic advances enabling the researcher to obtain data regarding a particular mechanistic level. This latter issue certainly explains why recent advances in neural imaging and molecular genetics have become so prominent. A major constraint on our research in speech and language development is that it can be observed only in humans, thus many molecular and cellular biological techniques cannot be used. However, if we assume that language does not arise from mechanisms that are unique to humans and human language behavior, we can open up our options to the use of animal models as was exemplified by the *BDNF* research.

We also hope to have shown that explanatory accounts of developmental disorders may need to be multifaceted. The explanation of individual differences that utilize the natural sciences of genetics, cognitive neuroscience, and language science will be insufficient for a full account of developmental communication disorder. We need to recognize that an entire additional domain of explanation coming from the social sciences will be needed to understand the social mechanism involving conditions of health and illness. Most importantly, we hope we have shown that all of our research is couched in an extensive network of assumptions and logical arguments that rest on sound philosophical foundations. We can wish that it were simpler and we can try to ignore the metatheoretical context of our research, but we do so at the cost of the important insights that come from seeing the forest in which our small tree of research exists.

We began by noting that the scope of Larry Shriberg's research program was truly ambitious. His research focused on multiple sources for explanations of developmental speech disorders. In this regard, his work has anticipated the likely directions future scholars—as he is indeed a scholar—will need to travel. These new scholars will have at their service the advantages of new and more informative technologies, but they will also face the challenges of incorporating this information into explanatory narratives that respect and exploit the fundamental complexity of speech and language development and disorders.

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