Causal Modeling: A Structural Approach to Developmental Psychopathology

JOHN MORTON and UTA FRITH

Nel mezzo del cammin di nostra vita mi ritrovai per una selva oscura che la diritta via era smarrita

DANTE: INFERNO, CANTO 1

Dante's poetic words very aptly express our position at the beginning of this chapter. We are lost in a dark wood in the middle of life where no straight paths exist. How can we find our way in the crisscrossing paths of developing abilities that forever seem to change direction? Like Dante, we have a vision of a distant hill. If we had a view *from* such a hill, we could discern the paths and draw a map.

In this chapter, we survey a few of the paths that others have already traced, and we show which directions might be fruitful, if followed. The problem we are addressing is the creation of a framework within which *different* views of developmental disorders can be modeled. We can clarify matters by introducing two major distinctions. First, we distinguish among various *levels of discourse.* Most commonly, when defining disorders or conditions, workers in developmental psychopathology have concerned themselves with the biological level and the behavioral level. People coming from a medical background have tended to stress the biological, and those coming from a psychological or psychodynamic background have tended to focus on the behavioral. The problem of relating these levels is great and, usually, there have been no more than gestures toward it. We have pointed out elsewhere (Frith, Morton, & Leslie, 1991) that an understanding of autism, at least, requires a third level between the biological and the behavioral. This third level we have called the cognitive level. We will rehearse the arguments and the scope of the terms in the next section.

The second general distinction we make is between *descriptive* models and *causal* models. Descriptive models are especially useful for the stable state, and description can be at any level—biological, cognitive, or behavioral, whichever is most appropriate for the current question. Indeed, it is sometimes argued that unless the current question is explicitly that of mapping between levels, descriptive models should always be at one level only (Mehler, Morton, & Jusczyk, 1984). When discussing development, descriptive models can sometimes be like snapshots of a moving scene. Causal models, on the other hand, have the principle of change over time built in, and will often require all levels to be represented. This is the case for autism, for example, where the very definition of the condition requires an intervening cognitive level between the biological and behavioral levels. The choice of levels here is an empirical issue rather than depending on what question is being asked. We would expect to find conditions where there is a direct link between the biological and behavioral levels without cognitive intervention. Tics in Tourette's syndrome are a possible example.

This chapter is concerned mainly with exploring and developing a framework within which *causal* models can be expressed. It is important to note that the framework of itself makes no empirical claims about any condition nor does it commit the user to any particular theory about anything. This feature makes the framework a neutral forum for the comparison of alternative or even contradictory theories. Our aim is that any coherent theory about developmental psychopathology—even one considered to be wrong—should be expressible within the framework. For example, the consequences of a claim about a single biological cause of a particular condition can be mapped out over different levels and compared against the consequences following from a claim about multiple biological causes of that condition. Empirical data can then be brought to bear on those points that emerge as critical, when the two competing theories are represented in a directly comparable fashion.

In almost all cases of developmental disorders, there is continuing controversy as to what constitutes the unique and universal features. These are usually contrasted with what are called associated features or secondary features. Diagnostic schemes generally strive toward defining disorders in terms of presumed primary features. Indeed, in some cases, the notion of primary features seems to have been constrained in terms of what could be defined within a particular diagnostic structure. We will see later that, when we want to explain rather than describe a condition, the so-called secondary features are as vital as the core features in constraining a theory.

In this chapter, we first review our approach to autism, the area for which the framework was first developed. While reviewing the development of our notation, we draw most of our examples from a particular causal theory for autism—a theory postulating a specific deficit at the cognitive level, with its origin at the biological level. It is important to note that we are not concerned with arguing the merits of one over other causal accounts of autism, although our preferences will certainly reveal themselves. Our own causal theory (Frith, Morton, & Leslie, 1991; Morton, 1989b) proposes a computational fault in a particular mechanism. We use the framework to show how it contrasts with others, but we do not review empirical work; such a review would only result in replicating information provided in other chapters of this volume.[1]

After establishing the framework, we explore its properties. In particular, we show how the notation reveals three distinct patterns of diagnostic practice. We work through a few examples of diagnostic categories of current interest and show how, in each case, a potentially useful definition of the category can be achieved. We then discuss some major problems in diagnosis, such as the variability of symptoms and the action of compensatory and protective factors. We present the concept of psychosocial pathways, along with the related technique of developmental contingency modeling. This is a modeling technique devised to enable us to represent the normal cognitive prerequisites of any particular behavior. Finally, we apply the framework to dyslexia in order to review the causal modeling approach.

A NOTE ON TERMINOLOGY

To start with, *levels* are not to be confused with *domains* (a term used to distinguish between, for example, cognitive and affective functions), which are placed at the same level in the present framework. The interactions among different domains, and especially the interactions of emotions and rational thinking, have been of concern to theorists in psychology from the time of Aristotle, in about 300 B.C., and these interactions would certainly be amenable to being represented inside the cognitive level.

We are also aware that our levels of discourse—*biological, cognitive,* and *behavioral*—could give rise to problems of definition.

At any stage in development, the biological component includes extensive contributions from the environment. Any reference to the biological origins of a particular problem should, then, be taken to include the appropriate environmental and social factors. Johnson and Morton (1991) discussed this issue at some length.[2] Such considerations, as well as our need to refer to the genetic contributions to psychopathological disorders, rule out use of the term *neural* or *organic* in place of *biological.*

Cognitive has a variety of meanings. We will try to make clear the scope of the word as we intend it. On one hand, the term is contrastive with *biological* and *behavioral.* For example, when we use *cognitive,* we do not necessarily imply conscious constructions, although that is still the way the term is used in such environments as cognitive therapy, where "cognitions" are always conscious. The other problem for our use of the term comes from the contrast that has been made, since Kant, between *cognitive* and *affective.* We have already pointed out that this contrast involves domains, not levels.

In our framework, affects and cognitions occur at the same level, intervening between biology and behavior. Affects in other frameworks might well be defined simply at the biological level, as physiological responses. In this case, it would still be necessary to bring in a special process capable of interpreting these responses so that they could exert an influence over mental activities. This process would have to be placed within the cognitive level of discourse. In yet other frameworks, affects might be defined simply at the behavioral level, for instance, as facial expressions, voice modulations, body language, and so on. Again, these outward behavioral expressions of affect need to be related to internal processes that interpret feelings in the same way as in the former case. It seems that affects can be defined, described, and discussed at all three levels, but the cognitive level is the one that explains how affects can have meaning.

It should be clear from the above that we use the term *cognitive* in a general sense of referring to the functional description of the brain's activity. We know of no other term suitable for this purpose, except perhaps *psychological.* This term is confusing, however, because it is commonly used to refer to behavior as well as to cognitive activity. These two levels must be kept distinct from each other.

GROUND RULES OF CAUSAL MODELING

The rules of causal modeling of a developmental disorder are informally well established but rarely made explicit. We propose

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[1] The preceding chapter by Baron-Cohen presents a detailed discussion of autism and of the particular cognitive deficit that we propose in order to account for this disorder. Here, we merely summarize the facts that are essential to the understanding of our general framework.

[2] While accepting that no clear partition can be made of the contribution of genetic factors, Johnson and Morton (1991) argued that it is still useful to make distinctions along a continuum. They proposed that the term *genetic* should be reserved for referring to the genome, and interactions of the genome with the internal environment should be termed *innate.* They then made a major distinction between the Species-Typical Environment and the Individual-Specific Environment. Where development is facilitated through interaction with nonspecific aspects of the Species-Typical Environment, they proposed the term *primal.*

here a short but expandable list of the most important maxims, which we shall illustrate later with examples. The maxims are:

1. *Start with biology.* Let the causal chain start with the biological origins.[3]
2. *Build causal chains.* The causal chain should be specified, or at least sketched, from the origin to behavior.
3. *Give full account.* All signs and symptoms of the disorder must be accounted for.[4]
4. *Specific over general.* A distinction between specific and general conditions must be made.[5] Features that can be accounted for as part of a general condition need not be accounted for within the causal theory for the specific condition.
5. *Correlation is not causation.* Do not confuse correlation with cause.

Violation of all these rules for causal modeling has been common in the history of autism.

Maxim 1: Start with Biology

The biological origin of autism has often been ignored. Some time ago, in the absence of evidence, this was understandable. Autism manifested itself most obviously in terms of social isolation, and it was easy for people to believe that social problems must have a social cause. Purely nonbiological psychosocial causes of autism can no longer be invoked. The evidence for a genetic origin, for example, is accumulating rapidly (Bolton & Rutter, 1990).

Unfortunately, belief in a psychosocial origin of autism still lingers on, much to the detriment of autistic people and their families. They suffer unnecessarily from self-blame and often follow punishing and entirely unproven regimes assumed to reverse or ameliorate supposedly disastrous interpersonal relationships.

Focus on the behavioral level alone has, for a while, been the hallmark of intervention programs. This has led to practical benefits in many cases, because behavior modification has been a

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[3] The precise biological origins of developmental disorders are very rarely known. This does not mean, however, that we should ignore biology. This is true even where we might want to explicitly rule out any biological component explicitly.

[4] For our causal model, the quality of the information on these matters is of vital importance. We try to use the most up-to-date epidemiologically based evidence, as well as clinical practice as laid down in DSM-III, DSM-III-R, and DSM-IV (American Psychiatric Association, 1980, 1987, 1994) and ICD-10 (WHO, Geneva, 1992), but we must leave open the possibility that received wisdom as to the symptomatology will be overturned by new research.

[5] The developmental disorders we are concerned with here are all specific disorders; that is, there is a particular deficit (identifiable at some level—biological, cognitive, or behavioral) that has particular consequences for development and that may occur in a pure form (with all other psychological functions intact). The pure form may be rare; more often, there are associated problems of a variable kind. If the original damage was large, then many functions are affected and this may lead to general deficits. Specific deficits have to be demonstrated over and above general deficits.

successful tool for the management of "difficult" children in general. On the negative side, such programs have also allowed unrealistic hopes about restoration of normality.

It is not advised to start with the behavioral level and work backward. The same behavior can be shown for different reasons. Autistic children who are emotionally aloof may, on the surface, look like children who avoid social contact because of extreme shyness. Children who superficially show fluent reading may be severe dyslexics with excellent compensatory skills.[6] We would expect to reveal the compensation by use of suitable tests (such as dual tasks) and measures (such as reading time rather than errors).

Maxim 2: Build Causal Chains

A claim such as the following is unhelpful: Limbic system abnormality causes social impairment. Even if the statement were true, in that it pinpointed correctly a critical brain structure, we would still have an insufficient account of social impairment. A single causal statement is not the same as a causal model. We need plausible links in the causal chain from origin to signs and symptoms.

Maxim 3: Give Full Account

A number of early cognitive explanations of autism suffered from a problem that could be caricatured as ring-around-a-rosy. When the job is to explain all the symptoms, it is hazardous to choose one of them as the most important, suggest an underlying process fault for this single symptom, and ignore the other symptoms or assume that they derive from the same underlying psychological dysfunction. The remaining symptoms are in as much need of causal explanation as the supposed primary ones. It is tempting to relegate all problems not accounted for in the primary explanation to the position of secondary consequences. For example, in a social deficit account of autism, the social deficit would be assumed to result in poor learning of language; conversely, in a language-based account of the disorder, the language deficit would be assumed to result in poor social relations.

Maxim 4: Specific over General

Mental retardation is an example of a general deficit that, to varying degrees, is present in a large proportion (often estimated at around 75%) of autistic individuals. Fombonne and du Mazaubrun (1992), in a study of the prevalence of autism in four French regions, found that more than 66% of the autistic group were severely or profoundly retarded. Only 13% showed no intellectual retardation. Mental retardation affects almost all cognitive functions and is manifest in a very wide range of behavior. It implies brain abnormalities of the type that would affect the basic efficiency of biological and of cognitive information processing.

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[6] Different underlying representations can give rise to the same behavior by the same individual at different developmental stages (Karmiloff-Smith, 1984). This is to be distinguished from the observation of the same behavior shown by different individuals on different developmental paths.

The effect of such damage on development is grave. Delays and capacity limitations in many cognitive areas are common in autism. These have to be taken into account separately from specific deficits. After controlling for developmental level (MA), degree of mental retardation (IQ), and chronological age (CA), many supposedly typical symptoms were found to be neither unique nor universal to autism (Hermelin & O'Connor, 1970). They can be attributed to the general condition of mental retardation, and therefore should not be accounted for in the theory of the specific condition of autism.

Maxim 5: Correlation Is Not Causation!

We have included this maxim partly as a reminder to ourselves, because the temptation to interpret correlation as causation is ever present. Examples of using correlational evidence as causal proof abound in all areas of psychology. It would be a violation of the previous maxim to single out autism researchers for specific blame.

A CAUSAL ACCOUNT OF AUTISM

The Biological Origin of Autism

It has now been generally accepted that there must be a biological origin to autism (see chapters in Gillberg, 1989, and in Schopler & Mesibov, 1987). There are several reasons for this acceptance. First, it is known that people with autism have a greatly increased chance of having diverse medical conditions as background factors. Second, the majority of autistic individuals show direct signs of brain dysfunction. This shows through MRI scans, cerebral spinal fluid investigations, brain stem auditory potentials, presence of epilepsy, and many other factors (Gillberg, 1992; Steffenburg, 1991). Third, there are indirect indications of brain damage in that autism is strongly associated with mental retardation. As one includes progressively more retarded samples of children, the likelihood of autism increases (Smalley, Asarnow, & Spence, 1988; Wing & Gould, 1979). One simple model for this pattern is that a specific brain system is necessary for normal development, disturbance of which leads to autism. Brain damage that results in general intellectual retardation could be seen as caused by randomly distributed lesions. The more the damage, the higher the probability that the critical brain system will be affected.

Even in high-functioning autistic people, in whom we can assume that the general level of damage is low, MRI techniques have revealed abnormalities in the cerebellar vermis (Courchesne, Yeung-Courchesne, Press, Hesselink, & Jernigan, 1988). Autopsy studies have suggested neuronal disruption in a number of brain areas, particularly in the limbic system (Bauman & Kemper, 1985), and recent reviews of the literature conclude that there is cortical as well as subcortical involvement (Dawson & Lewy, 1989a). However, such studies have not yet separated causal and correlative relationships between these various kinds of brain damage and autism. Given the range of possible brain damage in autistics, we cannot be sure which particular kind of damage is actually responsible for the autism.

The damage that causes the autism also has a cause. The latter causal factor could give rise to other damage that is unrelated to the autism. In Figure 13.1, the flat, unfilled arrow is the symbol we have chosen to represent the causal relationship. A horizontal line is used to separate levels. A hypothesized single origin, O, could be a particular genetic condition, for example, that gives rise to two consequences in the brain during the course of development. We have called these br_1 and br_2. We have supposed that br_1 is solely responsible for the cluster of behavioral signs S_1 to S_3, which, in our special case, are the main diagnostic criteria for autism. However, in this not implausible scenario, the kind of damage we have termed br_2 will always be found, but will, by the terms of the scenario, have no causal role in the autism. This, then, illustrates the difference between correlation (br_2) and cause (br_1). It is possible, for example, that the cerebellar damage found in many autistic people has no causal effect on the defining symptoms of autism. The symbol S is used for signs and symptoms at the behavioral level. We have indicated subcategories S_1 to S_3 without specifying them. They would be easily formed, for instance, by using the distinction between positive and negative signs, or between signs and symptoms. Signs are the objectively observable behaviors. They are positive if they are abnormal by their presence and negative if they are abnormal by their absence. Symptoms we only know about through the patient's self-report or through indirect inference. For instance, if one saw a person talking and listening intently when no one is there, one might infer that the person hears "voices."

We have mentioned a variety of kinds of brain damage that have been associated with autism. These correspond to kinds of damage found at the end of an early occurring developmental process and are thought to persist throughout life. The adult autistic brain is just as good a source for data about these abnormalities as that of the child. However, the cause of the damage is located in the developmental history of the autistic individual. As to such biological origins of autism, genetic factors have recently received particularly strong confirmation (Rutter et al., 1990). The results from both twin and family studies suggest that the genetic causes account for more than 80% of the phenotypic variance and are themselves likely to be heterogeneous (Szatmari & Jones, 1991). Genetic factors may well be the major cause of the majority of
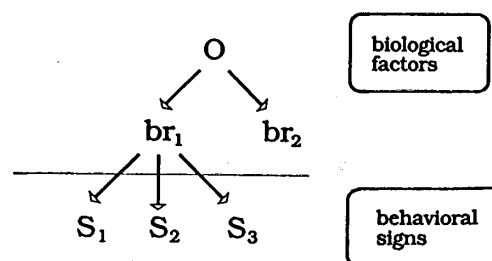
Figure 13.1 A hypothetical relationship between biological origin and autistic signs. S_n refers to signs and symptoms; br_n refers to conditions in the brain. The origin has two consequences in the brain, called br_1 and br_2. The former is responsible for the autism syndrome, and the latter gives rise to correlated disorders.
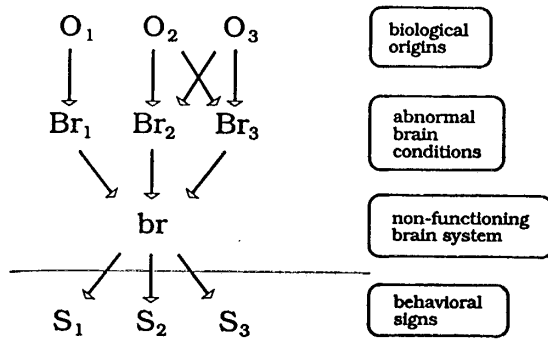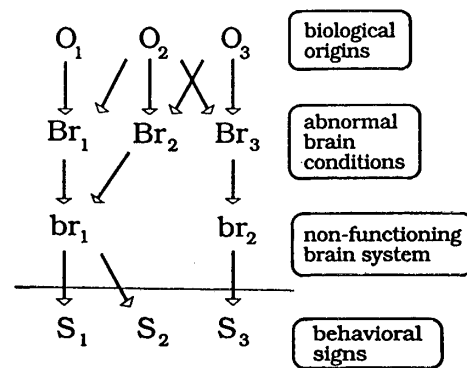
Figure 13.2 A hypothetical summary statement representing the theory that autism is always caused by a particular nonfunctioning brain system, which could be caused by any one of a number of possible origins.


Figure 13.3 A representation of the hypothesis that two brain systems are involved in autism rather than the single one represented in Figure 13.2.

autistic disorders, but other causal factors, such as viral disease, are still being considered as additional, independent causes (Nunn, Lask, & Cohen, 1986; Tsai, 1989).

At the biological level, then, a variety of prime causes is possible. The effect of these is always autism, and, therefore, we suppose they will all eventually be shown to affect the same brain system in some way. By brain system we mean that part of the brain that performs a particular identifiable function. The possible causal effects include destruction of the system, disconnection, or malfunction. In the absence of more precise information, we can still discuss the available biological evidence in terms of a causal analysis.

In Figure 13.2, we represent the material we have just summarized. The items at the top level, O_1 to O_3, represent the postulated biological origins—genetic and other factors. This portion of the diagram represents a summary statement, ideally across the full range of the disorder; for any one child, only one of the biological origins will usually apply. From these origins come a variety of abnormal conditions over the course of development, in a way as yet to be established. Such nonspecific brain conditions we have symbolized as Br_n.[7] Having identified the place in the model for the autism-critical brain condition, we can go a step further. The kinds of general brain abnormality (Br_n) required for a causal account will be those that affect a common system, here called br. Damage to the specific system br or interruption of the functioning of system br causes autism. In Figure 13.2, then, we have added another link to the causal chain on the biological level. Note that we could do this even though very little is as yet known about the anatomical and physiological, let alone the molecular biological facts. Having added the link, we immediately see an interesting consequence: it may turn out that two or even more identifiable cortical systems are responsible for the autistic behavioral complex.

We have illustrated this possibility in Figure 13.3, where we propose two biological systems, br_1 and br_2, responsible for dif-

ferent aspects of the behavioral manifestations of autism. This exercise illustrates what it means to say that different symptoms, even though they co-occur, are not necessarily traceable to the same underlying fault.

How would we know when this was the case? We would see occasional dissociations between otherwise related behaviors. An example will be considered in Figure 13.5, in the next section.

We have claimed elsewhere that the causal chain between candidate biological factors and the resulting behavioral impairment requires an intervening *cognitive* level (Frith et al., 1991). The final form of our diagram, then, must include the cognitive level, and, within it, a number of further links in the causal chain. The general form of this causal model is shown in Figure 13.4. For purposes of simplified exposition, we show just a single link in the cognitive level and with the biological level. The hollow, struckout C symbolizes a cognitive system that is found in normal development but is missing in autism, the abnormal developmental path being described.
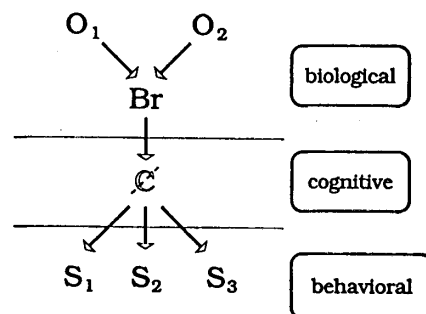
Figure 13.4 Introduction of a cognitive level between the biological and behavioral levels. The general hypothesis expressed is that autism can be seen as a cognitive deficit with a biological origin. The hollow, struck-out C indicates that a particular cognitive function, which is present in all normal children, is absent in autistics by virtue of the brain condition Br, which, in turn, could be caused by one of two different origins, genetic or nongenetic.

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[7] We use Br to refer to general conditions and br to refer to specific conditions.

All the primary elements of the causal model are now in place. We may have given the impression, in the preceding paragraphs, that outcome in the causal model is determined. As a matter of empirical fact, all outcomes of developmental abnormalities appear to be probabilistic, and the causal diagrams for conditions as a whole should be interpreted in this way.

The Behavioral Picture

The history of autism has been fraught with problems of diagnosis. For any causal account of a condition, it is vital to be able to agree on what one wishes to account for. "Is there such a thing as autism?" is a question that continues to be asked when there is uncertainty about the behavioral as well as the biological level of description. "Every autistic child is different" is a frequent cry from bewildered caregivers. And yet, over the past 50 years, the consensus about autism as a clinical entity with special needs and a predictable course has grown steadily.

Where do we start? Despite continuing controversy about the precise diagnostic criteria,[8] there is agreement that there are different variants of autism. A matter for debate is whether the variants are to be described merely in terms of degree of severity (i.e., "spectrum") or in terms of qualitative differences. From the point of view of this chapter, the outcome of this debate would only result in variation in the detail of the causal model. For simplicity, let us take the concept of the *autistic spectrum*. Disorders of this spectrum have in common three core features (Wing, 1988):

1. *Impairment in socialization*—specific impairment in the quality of reciprocal interactions, ranging from aloof to passive to odd.

2. *Impairment in communication*—delay in language acquisition and poor use of verbal and nonverbal means of communication.

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[8] DSM-III (American Psychiatric Association, 1980, pp. 87–90) gave the following criteria for Infantile Autism:
- Pervasive lack of responsiveness to other people;
- Gross deficits in language development;
- Peculiar speech patterns;
- Bizarre responses to various aspects of the environment, e.g., resistance to change, peculiar interest in or attachments to animate or inanimate objects.

DSM-III-R (American Psychiatric Association, 1987, pp. 38–39) specified the following criteria for Autistic Disorder:
- Qualitative impairment in social interaction;
- Qualitative impairment in verbal and nonverbal communication, and in imaginative activity;
- Markedly restricted repertoire of activities and interests.

DSM-IV (American Psychiatric Association, 1994, pp. 70–71) requires:
- Qualitative impairment in social interaction;
- Qualitative impairment in communication;
- Restricted repetitive and stereotyped patterns of behavior, interests, and activities.

3. *Impairment in imagination*—lack of understanding make-believe.[9]

These three features were first found as a triad of impairments in the course of epidemiological research (Wing & Gould, 1979). They were discovered to form a syndrome, that is, to be closely associated in the same individual and to manifest themselves in a variety of behaviors according to age and ability. Although the triad was found in children with classic autism it was also present in children who had not been so diagnosed. Furthermore, it was found not only in children who suffered at the same time from general intellectual retardation but also, occasionally, in those of average intelligence.

Even if there were no argument about the definition of the core features of autism, there are usually many more problems to be considered in each individual autistic child, in addition to any core features. We frequently find specific language impairments, motor coordination problems, and general learning disability. Other features often seen in autism may include anxiety and bewilderment as well as slow learning and lack of generalization. Impairments in attention, memory, and perception have also been described in children within the spectrum of autistic disorders (Gillberg, 1992). The range of signs and symptoms is indeed great, and the problem of accounting for all of them is still wide open.

The Role of Cognition in Defining Autism

According to our maxims, a causal analysis of autism will have to account for all of the *core* features (maxim 3) and all of what have been called the *associated* features. Specific accounts, however, are only developed if general accounts do not suffice (maxim 4). Thus, we do not have to account for mental retardation and its consequences in the same way that we account for the triad of impairments and its consequences. That is to say, although any account of the biological origins of autism must also explain the accompanying mental retardation, it is not appropriate to attempt a single cognitive account of the two. We explore this concept in Figure 13.5, where we show two specific consequences, br_1 and br_2, of one and the same general damage, Br. They have different consequences at the cognitive level: C_1 leads to impaired intellectual function and C_2 leads to the criterial signs of autism. In any child who suffers from this general type of brain damage, we will always find autism in conjunction with severe mental retardation.

According to maxim 3, the analysis must also allow for the principled explanation of all the features, including secondary ones. It is often a matter of controversy as to what constitute primary and secondary features. As an example, let us take anxiety, a frequent symptom in autism. People who regard the anxiety as primary see it as springing directly from a malfunction of the arousal mechanism, which, in turn, is seen as directly caused by the biological conditions underlying the disorder. Others see the anxiety as a secondary and not a primary feature.

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[9] The phrasing of this particular impairment in Wing and Gould's (1979) epidemiological study was "repetitive activities in place of imaginative symbolic interests" (p. 26).
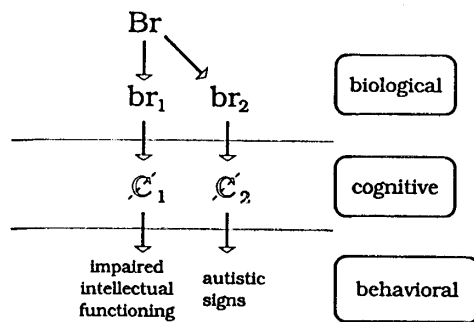
Figure 13.5 The frequent association of intellectual retardation with autism can be explained as resulting from a single brain condition, Br, with two separate consequences, br_1 and br_2.

Thus they would derive it as a result of the autistic child's incomprehension of features of the environment. In other words, they believe that autistic anxiety is a normal (and possibly functional) response.

One particular claim that we wish to make is that pathological features involving a component of the environment in their causal chain will always be secondary and not core features. (This is explored later, in the context of Figure 13.10.) Such subchains could be broken by appropriate manipulation of the environment at the appropriate time.

We also need to allow for the components that are identified with the large individual differences in the clinical picture of autistic people. These include, in particular, processes of *compensation*, and changes over time, attributable either to internal factors such as maturation or to external factors such as teaching.

From our brief review (above) of current work on the biology of autism, we concluded that autism has no single biological origin. Nevertheless, autism, or rather the developmental disorder underlying the spectrum of autistic features, continues to be seen as a single diagnostic category. If there is no single origin nor any single kind of damage that can be used as criterial, what, then, justifies the application of a single label?

The triad of impairments and the already envisaged spectrum that encompasses different degrees of autism seem, at first glance, to provide justification enough. However, the triad, or any similarly defined behavioral phenotype, is only a collection of symptoms that happen to co-occur with sufficient regularity to convince us that this co-occurrence is meaningful and has implications at other levels. Therefore, although the term *behavioral phenotype* has descriptive value, it has no explanatory value.

As we have already pointed out, the common constellation of signs and symptoms points to an underlying single cognitive deficit (Rutter, 1983). Clinical experience and the history of autism itself suggest that there is such a diagnostic entity and that it is much more than a chance constellation. Despite proven heterogeneity at the biological and behavioral levels, we assume that it is possible to justify a single label in a causal account and propose that what all autistic people have in common is a single *cognitive* deficit. This deficit gives rise to the core symptoms in the course of development.

To define the specific deficit that underlies the triad of impairments, we have to draw attention to a particular aspect of normal development that has been explored only recently (Astington, Harris, & Olson, 1988; Butterworth, Harris, Leslie, & Wellman, 1991; Whiten, 1991): the development of theory of mind (Premack & Woodruff, 1978), or "mentalizing"—our ability to predict and explain the behavior of other humans in terms of their mental states. Our ability to mentalize is revealed in our use and understanding of such words as *believe, know, wish, desire, intend,* and *pretend,* which are acquired remarkably early (Wellman, 1990). A central feature of our proposal is that autistic children lack this ability (Baron-Cohen, Leslie, & Frith, 1985; Leslie, 1987).

The term *theory* in the phrase *theory of mind* can lead to certain misunderstandings of our position. In particular, it can lead to a belief that we are talking about conscious constructions concerning not only other individuals, but also other minds in general. This is most certainly *not* what we—or, indeed, Premack and Woodruff (1978), who coined the term—intend to imply. Human beings do develop explicit theories concerning other minds, and autistic people are notably deficient in such development; however, this is not the difference we consider basic. The ability we are talking about, mentalizing, is primarily unconscious or implicit. It is a property of our cognitive apparatus that comes into action when triggered by particular stimuli, and it "makes sense" of other people's and our own behavior fully automatically.

What Is Mentalizing?

The hypothesis is that the ability to mentalize is dependent on a primal[10] mechanism and cannot be explained by learning. The ability manifests itself gradually in behavior over the first 5 years. By age 1, infants already internally represent many physical states of the world; that is, they can remember and manipulate in their heads what they perceive in the world. These are first-order representations.[11] From some time in their second year (or arguably, even earlier), children have at their disposal second-order representations and can represent mental states as well as physical states (Leslie, 1987).

What is the difference between first- and second-order[12] representations? We know that <ducks are fowl> and can represent that idea in memory as a first-order representation. At the same time, we can represent the idea <ducks are fish>, as long as we ascribe it to someone else, in a form such as "Some monks believed

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[10] *Primal* is defined by Johnson and Morton (1991) as the interaction of the genotype with the internal environment and the nonspecific component of the Species-Typical Environment (see note 2).

[11] Note that when we talk about representations, we mean *mental* representations, not pictures.

[12] We use "first- and second-order representation" because these two terms make a clear and unambiguous contrast. In other places, Leslie has used "meta-representations" when referring to what we have called second-order. Unfortunately, there are other meanings of "meta-representation," and the theory of mind literature has become very confused through lack of distinction among the various senses of this term. In response to this confusion, Leslie (Leslie & Thaiss, 1992) has started to use "M-representation" in this context.

that <ducks are fish>." Second-order representations—in this case, the representation of someone's belief—can be used to predict people's behavior. For instance, monks could eat ducks on Fridays if they believed them to be fish. In this way, we can establish relationships between external states of affairs and internal states of mind.

We postulated that the cognitive cause of autism is damage to a particular part of the mentalizing mechanism, termed EXPRAIS.[13] This theory enabled us to explain difficulties with pretend and, more importantly, to predict special problems with other mental state computations. The first test of our theory was as follows: Will autistic children, taking into account CA and MA, have special problems with understanding a false belief about something, as opposed to understanding a real state of affairs? This was confirmed by the Sally-Anne experiment (Baron-Cohen et al., 1985).[14]

The Sally-Anne task tested whether the child would know that Sally will look for her toy in the place she hid it, in spite of the child's knowing that the toy has been moved elsewhere. If so, then the child can represent Sally's false belief as well as the true state of things. Normal children have no problems with this sort of task from about 4 years of age. Down syndrome children with a mental age of 5 or 6 can also answer correctly. However, of a group of 20 autistic children, with a mean mental age of 9 years, 16 failed the task in spite of being able to answer correctly a variety of questions of fact about what happened. They knew where Sally had put the toy, and they knew that Anne had moved it and that Sally had not seen the move. Their problem did not lie in perception, in memory, or in language. The autistic children just could not conceptualize the possibility that Sally believed something that was not true.

We believe that the mentalizing deficit goes some way toward explaining the three core features in the autistic spectrum. It does not explain certain other criterial features of autism, such as "restricted, repetitive, and stereotyped patterns of behavior, interests and activities" (DSM-IV, American Psychiatric Association, 1994; also in ICD-10, World Health Organization, 1992). It may well be that the autism spectrum demands explanation in terms of more than one cognitive deficit. Some symptoms whose precise status as part of the behavioral phenotype is as yet unclear, notably those associated with repetitive actions, special skills, and so-called frontal signs, may need additional explanation. These would also be mediated through the cognitive level but could require a second component at the biological level, as in Figure 13.3.

Changes over Time

In all studies reported so far, there is a minority of autistic children who perform mentalizing tasks correctly. We ourselves have now tested over 50 able autistic children on the false belief and
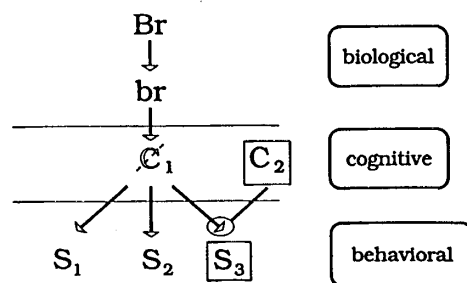
Figure 13.6 Compensation in a causal model. This diagram is intended to indicate that whereas S_3 is normally the consequence of the absence of C_1, this sign is attenuated in the presence of another cognitive factor, C_2. This outcome is more easily thought of in positive terms. If we think of S_3 as the absence of a particular piece of behavior which is normally mediated by C_1, this piece of behavior is now mediated by C_2 instead.

related tasks. So far, we have found that, for autistic children to succeed on a mentalizing task, they have to have a much higher chronological and mental age than is the case in normal development. On the whole, those autistic subjects who pass theory of mind tasks are teenagers with a mental age in excess of 8 years (Frith et al., 1991). Could the successful autistic children have acquired some mentalizing ability after all? If so, how? We consider two possibilities under the headings of *compensation* and *residual deficits*.

In Figure 13.6, we use a special symbol to represent compensation in the causal model. We have the usual range of behavioral signs, S_1–S_3, arising from the cognitive deficit, the hollow, struck-out C_1. The behavior S_3 would occur in a set of conditions that the autistic child cannot understand or respond to as a normal child would. The supposition is that the individual develops knowledge or skill in response to these environmental conditions in order to cope with the problem. We symbolize such knowledge or skill with C_2, and show a connection from C_2 to S_3 with an elliptical surround to the $C_1 \rightarrow S_3$ causal arrow. This indicates that C_2 provides a compensation for the missing or dysfunctional C_1 in respect of S_3.

Next, we will consider residual deficits. This possibility would assume that all individuals in the autistic spectrum initially share the same underlying deficit but that, for some, mentalizing ability eventually develops because the innate mechanism matured late. Normally, children structure their experience of others, from a very early age, taking into account mental states. A child who spends extra years without the benefit of this ability will have established habit patterns and interpretive frameworks whose influence will be difficult to eradicate.[15] Another example, from the area of vision, is that a squint uncorrected in a young child means that the individual lacks stereopsis. Correction of the squint in adulthood does not correct the residual deficit.

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[13] EXPRAIS is short for "expression raiser." This, in Leslie's 1987 paper, is the single most crucial computational process in forming representations of other people's beliefs, desires, and intentions.

[14] A number of experimenters have now carried out similar studies worldwide, confirming that autistic children have a specific impairment with beliefs. (See Happé & Frith, in press, for a recent review.)

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[15] The converse explanation would apply in schizophrenia, where the already established good habit patterns serve to sustain the patients' understanding of mental states, even though the critical mechanism has ceased to function (C. Frith, 1992).
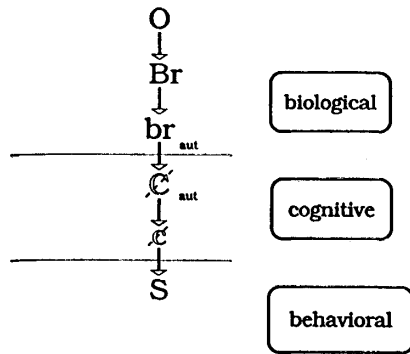
Figure 13.7   The essential components of a causal model for autism.

Competing Causal Accounts of Autism

We are concerned with providing a notation that enables alternative accounts of the cause of developmental disorders to be satisfactorily represented. As we have seen, such a notation requires, at least, that biological, cognitive, and behavioral levels be represented. Most of the current competing accounts have the same general structure as the ones already discussed. However, as we shall see, the formalism of the present framework helps to focus on the crucial differences between these accounts.[16]

Nearly all researchers now agree that a biological disorder is at the root of autism. Reducing the causal model down to its essence, we arrive at Figure 13.7, which indicates that there is some origin, O, of the condition with one of a set of possible biological consequences, Br. This has a specific effect, br_{aut}, affecting a particular brain system, which underlies the cognitive function C_{aut}.[17] Damage to C_{aut} affects the development of the set of cognitive functions, c, which, in turn, leads to a variety of signs and symptoms, S. The only theories that do not subscribe to this general account—the pure psychoanalytic account and the ethological account (Tinbergen & Tinbergen, 1983)—require the causal diagram to represent the role of agents outside the child. We have done this in Figure 13.8, where the dotted vertical line divides internal from external effects. The external circumstance—let us suppose for the moment that these are family circumstances, P—are seen as affecting the cognitive function, C_{aut}. The role of genetic factors in this case would need specification. The logic of the formalism allows one possibility: that both factors, damage to br_{aut} and external factor P, are required for autism to occur. To indicate this conjunction, we have used the symbol &. Whenever this symbol is used it is to be understood as saying that both causal factors have to be present for the effect to occur. If only one of the two factors is present, then the condition will not arise. The theory expressed in Figure 13.8 would next be
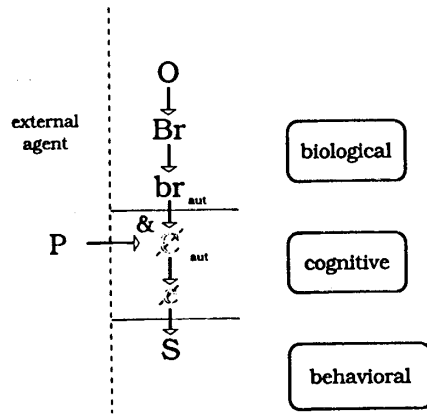
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[16] We will, in any case, only mention a few of the current accounts of autism. Furthermore, new versions are bound to be created in the future.

[17] We identify C_{aut} with the Expression Raiser—the function required to create second-order representations. Other theories will identify C_{aut} differently.

Figure 13.8   A theory of autism where family circumstances, P, interact with biological factors. The dotted vertical line divides internal from external effects.

obliged to specify, or at least to indicate, the nature of C_{aut} such that there is (or, at least, that it is plausible that there might be) a brain state that maps onto it. Such a constraint would, for example, rule out knowledge states (such as a belief or a feeling about mother) as candidates for C_{aut}.

A more plausible scenario, perhaps, is shown in Figure 13.9. This diagram represents a scenario where the autistic child behaves in some way so as to provoke a particular kind of response in the caregivers. This, in turn, changes the child's cognitive states to create the autistic spectrum of behavior. In the abstract, then, the biologically based cognitive condition, C_{aut}, leads to behavior that disrupts parental bonding. The behavior is not indicated in the diagram because it is not (ex hypothese) a part of the autistic spectrum. However, the parental response, in turn, feeds back into the child to create a change at the cognitive level. This leads to behavior which, by virtue of being part of the autistic spectrum, has to be represented in the diagram.

Psychodynamic accounts emphasize the role of interpersonal factors as both cause and effect of conditions such as autism.
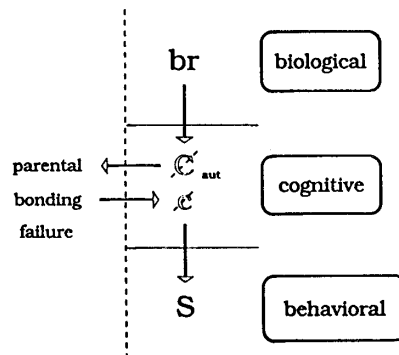

Figure 13.9   A hypothesis that autism is caused by a disruption of parental bonding.

Interpersonal factors clearly exist, but their causal role is not clear. In our own account of autism, we would want to allow a place for secondary problems that were the reaction of the child to the parent's response to the manifestation of the child's basic disorder. In previous articles on this subject (Frith et al., 1991; Morton, 1989b), we have represented the social level as equivalent to the other three but causally related to the behavioral level only. This is not the only option. It is also possible to represent the social environment as interacting with the individual at any of the three levels. For instance, we can understand family variables, from psychiatric conditions to stress and deprivation, as affecting the cognitive level rather than the behavioral level; that is, behavioral change would take place by virtue of the cognitive change and not independently.

A different kind of possibility involving the environment would occur where one wished to postulate that some external event created a changed brain state. This is shown in Figure 13.10. The first parts of the biological chain represented in previous diagrams is missing from this figure because the claim in question would relate to a child without genetic or other developmental abnormality. An example would be the occasional report that a child developed normally until the age of 3 or so but then had a viral condition leading to brain damage, a rapid regression of language gains, and subsequent autistic symptoms.

Rogers and Pennington (1991) proposed that the root cognitive cause of autism is an inability to imitate. They pointed to "the potential power of an early deficit in imitation to disrupt other early developing interpersonal processes" (p. 137). While accepting that there is no evidence of any deficit in autistic children during the first year of life, Rogers and Pennington pointed to deficits in imitation skills in older autistic children. Note, however, that it is unlikely that early imitation skills are mediated by the same mechanisms as later skills (they differ in the intentional component, for example). In addition to deficits in imitation, Rogers and Pennington supposed that autistic infants also lack the ability of emotion sharing. These defects "would greatly affect the baby's ability to organise *social* information concerning other people by depriving the baby of primary sources of social data" (p. 147, original italics). These two deficient skills, together with the

theory of mind deficit that emerges later, are viewed as "increasingly complex expressions of the ability to form and coordinate certain representations of self and of another and to use these representations to guide the planning and execution of one's own behavior" (p. 150). Such functions are, apparently, supposed to be mediated by biological circuits involving the prefrontal cortex and the limbic system.

The underlying deficit in this theory as currently expressed is "impaired formation/coordination of specific self-other representations." This is clearly a description of the outcome of a variety of information processing operations spread out over time. In this formulation, there is nothing corresponding to a deficit in any single function that could find its way into a cognitive model. The theory could not, then, be represented formally in the same way as our own theory, with a number of possible biological origins converging onto a single cognitive core, as in Figure 13.4. Rather, the core impairment seems to be at the biological level. Rogers and Pennington depicted their own model in the form shown in Figure 13.11. The triad of impairments, shown as part of a causal chain, are all being affected by the core impairment. We are inclined, then, to represent the Rogers and Pennington model inside our framework in the form shown in Figure 13.12.

This is a much more complex model than one in which deficit in imitation is the primary cognitive cause. The disadvantage of the latter formulation is that imitation in the older child is a complex cognitive skill and cannot be readily mapped onto a biological substrate. Thus, it would be necessary to specify the elements of this skill in such a way as to make the possibility of a biological mapping plausible. This is much the same kind of problem as would arise with our own model if the underlying deficit were seen simply as an inability to create a theory of mind. This complex, cognitive, conscious activity is clearly the result of a good deal of cognitive development and interaction with the environment, and not at all the kind of factor that one would want to introduce into a developmental theory. The mentalizing theory, however, reduces to a core computational ability—the ability needed to create second-order representations. This order of things could well correspond to a simple biological deficit that
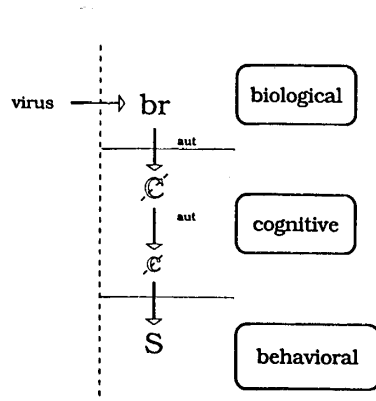
Figure 13.10   An example of an external influence operating at the biological level.
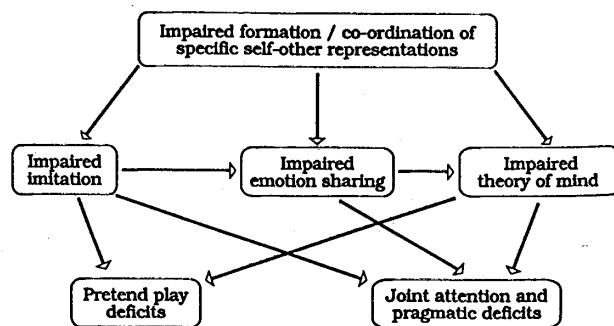
Figure 13.11   Adaptation of the Rogers and Pennington model. From "A Theoretical Approach to the Deficits in Infantile Autism" by S. J. Rogers and B. F. Pennington, 1991, *Developmental Psychopathology, 3,* p. 152. Copyright 1991 by Cambridge University Press. Adapted by permission.
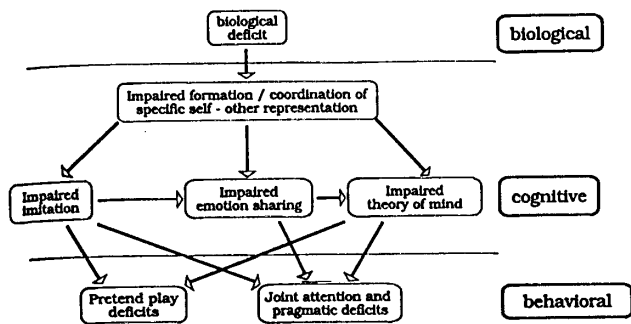
Figure 13.12 Our interpretation of the Rogers and Pennington model. Compare with the version in Figure 13.11.

might have no clearly visible signs over the first year of life (Johnson, Siddons, Frith, & Morton, 1992). If ability to imitate were to be fitted into a causal, developmental, multilevel representation, then it would require breaking down in a similar way.

A theoretical explanation that is close to our own and that postulates a cognitive deficit was proposed by Mundy, Sigman, and Kasari (1993). Predating Rogers and Pennington's approach, they postulated a seemingly simple and early appearing cognitive function as faulty, and focused on joint attention and the component of shared affect in this particular cognitive function. Now, it seems plausible to analyze deficits in joint attention as early manifestations of deficits of second-order representational skills (Mundy, Sigman, Ungerer, & Sherman, 1986). However, Mundy, Sigman, and Kasari (1993) rejected this "in the light of developmental data indicating that gestural joint attention skills emerge prior to meta-representational skills in normal development" (p. 126, n. 3). This represents a confusion between an underlying ability and the manifestation of that ability. Baron-Cohen et al. (1985) initially demonstrated the specific mentalizing deficit with a task that normal children could not do until they were around 4 years old. But that task also requires a number of other abilities, and it is those other abilities that are not developed until age 4. The underlying computational process, EXPRAIS, is supposed to be innate and will operate in other situations earlier. The Mundy and Sigman view (Mundy & Sigman, 1989; Mundy, Sigman, & Kasari, 1993) of our theory (e.g., Frith, 1989; Frith, Morton, & Leslie, 1991) is given in Figure 13.13, and the model
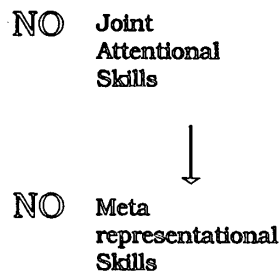
Figure 13.13 The Sigman and Mundy view of the Frith/Morton/Leslie theory of autism diagrammed using our notation.
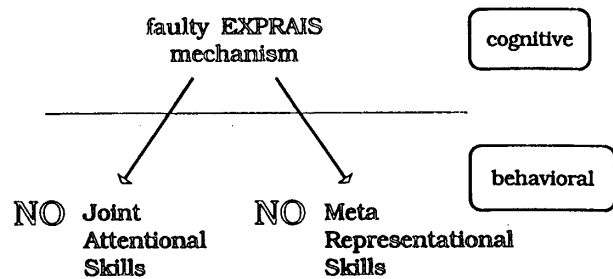
Figure 13.14 Our view of the Frith/Morton/Leslie theory of autism. Compare with Figure 13.13.

we actually support is shown in Figure 13.14. Let us reiterate that performance on tasks such as the Sally-Anne task is a manifestation of the core ability in interaction with a good deal of knowledge together with a variety of cognitive skills, including language. Within the theory we espouse, there is no early limit for the manifestation of mentalizing. We would, for example, be happy to include joint attention skills such as pointing (Baron-Cohen, 1991; Leslie & Happé, 1989), eye-gaze engagement, and teasing (Reddy, 1991), all of which occur within the first year.[18]

We turn now to the alternative proposed by Hobson (1989, 1990, 1993; Leslie & Frith, 1990). Hobson supposed that the primary deficit in autism is a disturbance of affective contact. He would accept that there is a biological origin to this problem—this much he has in common with us. He would also agree that it would be necessary to provide a causal chain from the affective disorder to the full range of autistic symptoms. The problem then arises as to how to represent the core affective disorder in the framework we have created.[19] The account by Dawson and Lewy (1989b) also emphasized the role of an impairment in very early socioemotional interaction.

At first glance, it might seem as though there is a clear contrast between Hobson's *affective* theory and our *cognitive* theory. But when we diagram Hobson's theory as a causal model, we can see that what Hobson calls the affective level has the same relationship to biological and behavioral levels as does what we call the cognitive level. That is, Hobson would not want to say that the affective problem he alludes to was either biological or behavioral. In terms of the contrast we have been using, Hobson's theory is of the same kind as all the other cognitive models.

This puts us into something of a representational dilemma. Hobson will also want to specify cognitive consequences of this affective, or, rather, interpersonal disorder, as well as its behavioral consequences. The affective and the cognitive are different domains but belong at the same level of description. The most convenient way to represent Hobson's theory, then, is as shown in Figure 13.15.

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[18] See the previous chapter, by Baron-Cohen, for links between mentalizing and early eye-gaze detectors.

[19] Hobson suggests that there is "an interpersonal psychological system that is impaired as the final common pathway in autism" (personal communication, June 18, 1992).
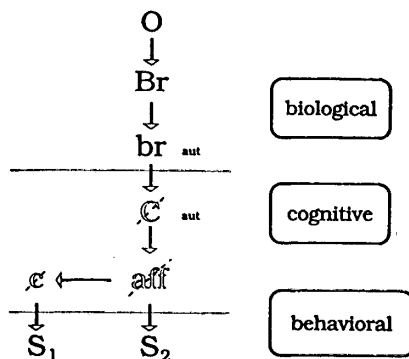
Figure 13.15 A simplified view of Hobson's theory of autism. The cognitive deficit, c, is seen as deriving from an affective disorder rather than being directly consequent on a biological deficit.
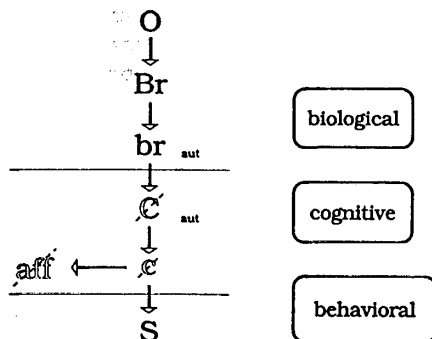

Figure 13.16 An affective disorder shown as a secondary consequence of a cognitive disorder.

In the expansion of our own theory, we would interpret the affective disorders as secondary consequences of the child's interaction with the environment. One way of doing this is shown in Figure 13.16, where the affective disorder, aff, is seen as a consequence of the cognitive disorder, c, at the same level.

PROBLEMS OF DIAGNOSTIC PRACTICE

Now that we have defined and explored the properties of the notation, we can begin to put it to use. First, we will consider the way pathological conditions are diagnosed. Conditions—that is, syndromes or disease entities—are, in practice, defined at all of the biological, cognitive, and behavioral levels. In many cases, the levels are mixed haphazardly and, in the case of the cognitive level, intuitive psychology often takes over from scientific concepts.

Basically, the definition of an entity can have a strong form or a weak form. The strong form corresponds to a singularity in the causal nexus. This would be revealed in our causal diagram by the existence of three types of causal model, resembling, respectively, the shapes of the letters A, X, and V (see Figure 13.17). Each of these letter shapes shows a convergence of paths at a single point: the top, the middle, and the bottom, respectively. These
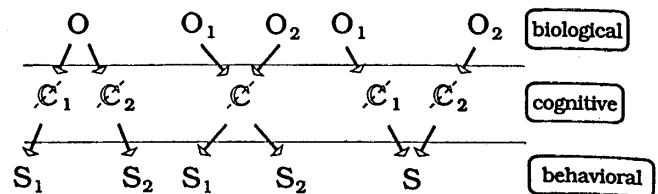

Figure 13.17 Idealized diagnostic types, resembling the letters A, X, and V.

three locations of "pinch-points" correspond to biological, cognitive, and behavioral levels. The convergence or pinching means that there is just one feature at that level or sublevel. In the weak forms of the three types, there would be two or more nodes at the pinch-point. In the illustrations that follow, we will restrict ourselves to the strong forms.

A typical example of a strong biologically defined syndrome would be a specific genetic defect, such as phenylketonuria (PKU). This would be an A-shape definition, a form of which is shown in Figure 13.18, where C_1 and C_2 represent the cognitive consequences of a single defect that is of genetic origin. In PKU, there appear to be global cognitive impairments as well as emotional disorder (Taylor, 1991). S, as usual, represents signs and symptoms, the behavioral manifestations.

Two aspects of Figure 13.18 need to be strongly marked. First, as we have already stressed, we do not intend the notation to imply that there is a deterministic relationship between origin at one end and signs and symptoms at the other. The notation is descriptive, not prescriptive, and causal theories of any degree of complexity can be represented to any required level of detail. Thus, one might want to represent the fact that the presence of the particular genetic defect characteristic of PKU is not sufficient for the full cognitive and behavioral expression of the disorder, but that certain other biological, environmental, or cognitive preconditions would be necessary as well. Thus, PKU children's difficulties depend strongly on how well the prescribed diet is kept to. To give a dramatic example of the amazing diversity following from a single major gene disorder, we could take neurofibromatosis. Manifestations in a single family may vary from a few skin blemishes, requiring an expert to note them, to grotesque multiple nerve tumors, causing gross deformity.

The second aspect is related to the first. The diagram could be seen either as the description of an individual or as the
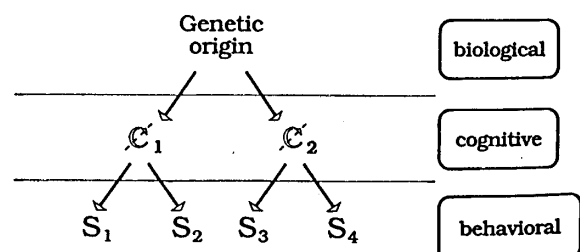
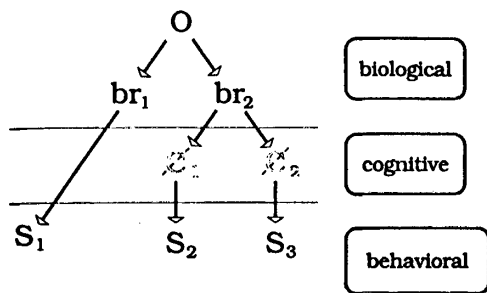**Figure 13.18** The archetypal A-type causal model.

Figure 13.19   A variant on the A-type causal model.

Figure 13.21   The archetypal V-type causal model.

description of a type. If we are describing an individual, then we enter only those elements that are germane for that individual, and we represent the specific determinants of the signs and symptoms for that individual. The causal links would then be seen as clinically real. On the other hand, we could use the diagram for the description of a type in order to answer the question: What is the range of clinical picture possible for PKU? For this kind of question, the causal connections should be seen as probabilistic rather than deterministic.

Note that a number of variations are possible in this general form of the diagram. One example is that the primary biological defect has a number of biologically defined consequences. This would be represented by a causal nexus entirely within the biological level. A second variant is that a biological defect may directly lead to behavioral consequences without cognitive mediation. Thus, dopamine deficiency in Parkinson's disease (in interaction with other factors) can lead to tremor. The way in which these two examples would be incorporated into our diagram is shown in Figure 13.19. In this case, br_1 and br_2 are biological consequences of the primary deficit; br_1 is shown as directly causing the behavioral consequences S_1; br_2 has consequences C_1 and C_2 which, together, have a variety of signs and symptoms, S_2 and S_3. All these variations would still roughly conform to the A-shape.

An example of a cognitively defined syndrome is that of autistic spectrum disorder, which we have already briefly outlined above. This would be represented as an X-shape, as shown in Figure 13.20. A variety of possible biological causes, O_1 to O_3, all lead to the same cognitive deficit. The single cognitive deficit then results in (some subset of) a large number of signs and symptoms. Again, variations at all three levels are possible, as we have already shown in our discussion of competing theories. All would maintain a relative convergence at the cognitive
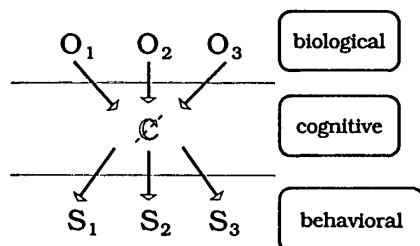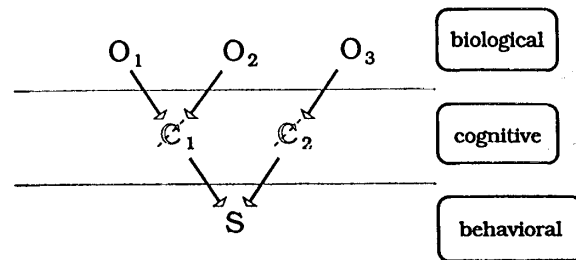
level. Even if it were necessary to postulate two or three separate cognitive deficits for a full explanation of the condition, the causal analysis would still roughly conform to the X-shape.

Note that the links do not at the moment make a distinction between conjunctive and disjunctive causation; that is, the preceding diagram is to be interpreted as saying that C can be caused by O_1 or by O_2 or by O_3. It will be necessary in a full causal account to allow for restrictions such as that C is caused by the conjunction of O_2 and O_3. This is equivalent to the "additional preconditions" discussed in the context of Figure 13.18.

Finally, we can represent the behaviorally defined syndrome, which would have the general form of a V-shape, as shown in Figure 13.21. S represents the defining behavior or behavior pattern which, in the example, can be caused directly or through cognitive mediation. We use the example of hyperactivity, which is defined as a diagnostic entity at the behavioral level.

Hyperactivity: An Example of a V-Type Causal Model

The diagnosis of hyperactivity is contentious. Not only are there major differences between U.S. and European practice (as represented in DSM-IV and ICD-10; see Taylor, 1986, for other differences) but also there appear to be differences within each of the communities with respect to the status of the diagnosis. It is not our intention to attempt to legislate in these debates. We can, however, represent the position of at least some of the protagonists.

Hyperactivity is a "pattern of restless, inattentive, and impulsive behavior in childhood" (Schachar, 1991, p. 155); such a pattern is what is called a *diagnostic entity*. There is much concern with the face validity of diagnostic entities. Rutter (1978) suggested that, to be *valid*, a diagnostic entity must differ in etiology, course, characteristics, or treatment response from those of other child psychiatric entities as well as from normality. At the moment, there is concern over the validity of the distinction between hyperactivity and conduct disturbance (Hinshaw, 1987). We discuss the implications of such a concern below.

Essentially, hyperactivity and associated disorders have been defined behaviorally. This is confirmed by a glance at the DSM-III and the DSM-III-R (American Psychiatric Association, 1980, 1987) criteria. In DSM-III (1980), the diagnosis of attention deficit disorder with hyperactivity (ADDH) required inattentiveness, impulsiveness, and overactivity. These had to continue for more than 6 months, starting before the age of 7. If

Figure 13.20   The archetypal X-type causal model.

a child presented with inattentiveness and impulsiveness but without overactivity, the diagnosis of attention deficit disorder without hyperactivity (ADD/WO) was applied in the belief that this combination delineated a distinct syndrome (see Lahey, Schaughency, Hynd, Carlson, & Nieves, 1987). DSM-III-R introduced a further category of attention deficit hyperactivity disorder (ADHD). To qualify for this category, a child had to exhibit 8 symptoms from a menu of 14 symptoms of hyperactivity, inattention, and impulsiveness. Schachar (1991) pointed out that this created two new subcategories of hyperactivity, one characterized by overactivity and impulsiveness but without inattention, and the other by inattention and overactivity but without impulsiveness. These have now been introduced in DSM-IV.

The ICD-9 criteria differed in a number of ways, as described in some detail by Schachar (1991). One feature that he focused on was the treatment of comorbid psychopathology. He commented:

> The syndrome of hyperactivity is viewed, for the most part, as an epiphenomenon or non-specific correlate of various forms of psychopathology that carries no particular etiological significance. Consequently, when hyperactivity occurs as part of a mixed presentation, the clinician is encouraged to diagnose the underlying condition. (p. 158)

In this case, then, the focus of diagnosis was to be either at the cognitive or the biological level rather than at the behavioral level. Schachar commented that "a diagnosis of hyperkinetic syndrome is usually limited to a presentation uncomplicated by co-morbid psychopathology" (p. 158). There was clearly potential for enormous variability of symptoms that might or might not lead to distinct subgroups.

We have chosen the example of hyperactivity because it is specified at the behavioral level. What can we say about the rest of the causal tree? We can surmise that the intentions of the two diagnostic systems are different from each other in some respects. In particular, DSM-III (1980) permitted diagnosis of ADDH when the symptoms occurred at school but not at home, or vice versa. In contrast, the ICD-10 diagnosis of hyperkinetic syndrome requires that the behavior be reported consistently in several situations. One might surmise that ICD-10 regards it as being more of an endogenous problem. DSM-III-R (1987), on the other hand, with its explicit "some people . . . show signs of the disorder in only one setting, such as at home or at school" (p. 50) suggested a more temporary and exogenously caused problem. The statement clearly indicated that the behavior, rather than the precipitating circumstances, was important. In this classification, the notion of cause seemed to be submerged under the behavioral criterion.[20] In DSM-IV the behavioral emphasis is less marked.

Cantwell (1977), on the basis of studies of response to stimulant drugs, follow-ups, and neurological and neurophysiological

studies, concluded that all of these techniques indicate that hyperactive children are a heterogeneous group. In an earlier paper examining the genetics of hyperactivity, Cantwell (1975) concluded that "if there is a genetic component to the syndrome, it is operating in one sub-group of these children; or there may be several genetically distinct sub-groups" (p. 264). We can see, then, that there are a number of causal possibilities in the definition of hyperactivity. All of these can be represented in the V-shape.

Taylor (1986) also considered pharmacological effects as a means of establishing diagnostic categories. The underlying principle is that patients who respond in the same way to drugs belong to the same biologically defined category. We can illustrate this principle using the causal notation. In Figure 13.22, we suppose two subgroups of children with the same sign, S. The groups differ in that they have different abnormal brain states, br_1 and br_2, which have the same cognitive consequence, C. Whether the operation of a drug may be useful in helping to define subgroups will depend on the level at which it interacts with the causal tree. If the drug is operating at the biological level in the causal tree and the response of two patients is the same, then we would be justified in classifying the two as being in the same group. On the other hand, if two patients have different responses to such drugs, then they would be classified as coming from different subgroups. Thus, $drug_1$ could operate selectively on br_1. Patients in this group would no longer suffer the cognitive dysfunction and would not, then, exhibit the characteristic signs. Patients with damage to br_2, on the other hand, would remain unchanged by the drug. In contrast, $drug_2$ could operate selectively on br_2, abolishing the signs in that group but not in the first one. Use of these two drugs, then, will serve to distinguish the two subgroups of children. However, another drug, $drug_3$, might operate by suppressing the behavior without otherwise interacting with the causal tree. In this case, the two groups of children would be responding in the same way to $drug_3$ in spite of their underlying biological differences. The existence of a common response to $drug_3$ would demonstrate nothing about the homogeneity of the groups but would be equivalent to suppressing the signs by behavior modification techniques or even simple physical restraint.

Taylor's (1986) review of childhood hyperactivity and, even more, his review (1991) of child neuropsychiatry in general allude to many of the problems that explanations of developmental

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[20] It would be more correct to talk about *reported* behavior rather than *actual* behavior, because much of the research in this area has been done with teacher-based checklists rather than observation.
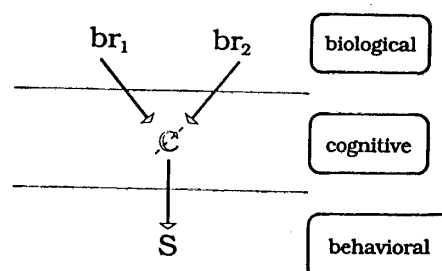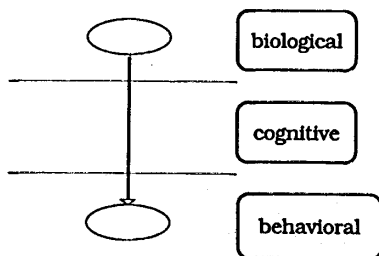

Figure 13.22 A simple causal model to help understand whether reaction to drugs can decide diagnostic categories (see text).
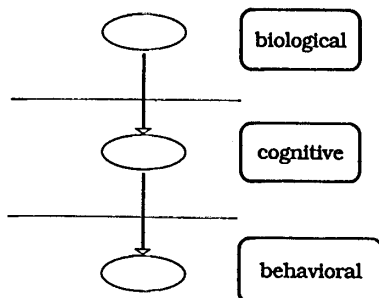
disorders encounter. It is precisely for these reasons that we are suggesting the present framework.

The approach followed by Sonuga-Barke, Taylor, and Heptinstall (1992) and Sonuga-Barke, Taylor, Sembi, and Smith (1992) suggests that it would be fruitful now to consider an X-shape of causal modeling with a hypothesized underlying cognitive deficit, such as *impulsiveness* or *inability to delay reward*. This approach is possible because of the convergence of clinical, epidemiological, and neuropsychological studies, which (just as in the case of autism) together strongly point to hyperactivity as a valid diagnostic entity, not in terms of behavior but in terms of underlying causes. Like autism, hyperactivity is likely to be defined as a cognitively based and biologically caused developmental disorder. If this happens, however, it is essential that a separate diagnostic category be created for children with the same behavior but different cognitive cause.
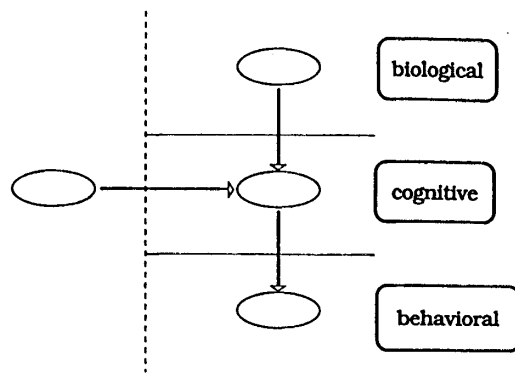
As yet, we cannot go further in the X-shape causal model of hyperactivity because we lack a psychological theory of hyperactivity that would relate the phenomena to normal control of movement, as well as to impulsivity and attention deficits. We can show in simplified causal diagrams how various causal theories can be represented (Figures 13.23–13.28). Taylor (1986) suggested two possibilities: (a) genetic or other biological factors may affect activity levels directly (Figure 13.23) or (b) the biological effects are mediated through cognitive factors (Figure 13.24). In either case, the condition could be exacerbated by psychosocial factors that, for example, increase levels of stress. In Figure 13.25, we have indicated the environmental factors as in-

Figure 13.25 A schematic representation of environmental factors interacting with genetic factors to produce hyperactivity symptoms.
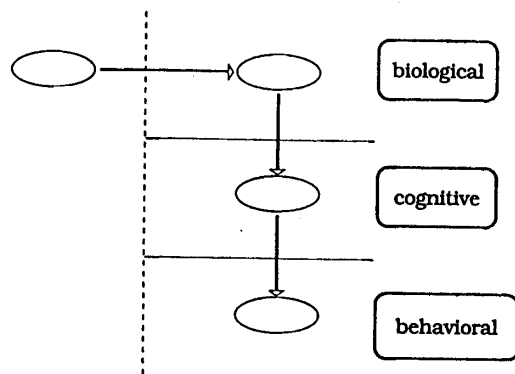
Figure 13.26 The effects of the environment operating at the biological level.

teracting with some genetic factor to precipitate a state not describable at the biological level. The theory that hyperactivity is caused by an environmental toxin of sorts (suggestions include lead intake and dietary additives) is illustrated in Figure 13.26, where we diagram the possibility of an external cause operating at the biological level. A factor originating in the environment need not operate at the biological level in the causal chain. It may affect general attitudes toward schooling, while interacting with other factors. This possibility is represented in Figure 13.27.

At the behavioral level, all of these diagrams refer to the same behavioral complex. Thus, we can put them all together into a single causal diagram, as in Figure 13.28. There is, in all cases, some degree of freedom left for specification of the involvement of alternative constructs at the cognitive level.

Variability

One of the irritating as well as fascinating problems in developmental psychopathology is the variability among individuals. If selection of a group of people is based on their identity by some definition, then it will inevitably be discovered that they are
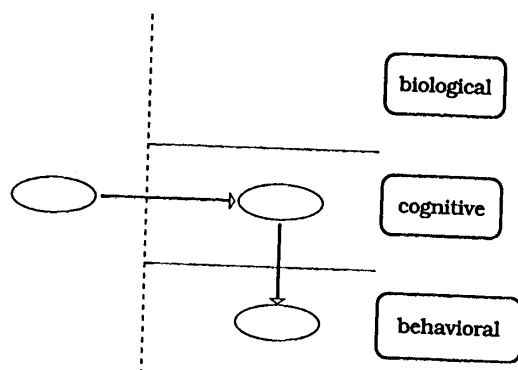

Figure 13.23 A schematic representation of direct causation of hyperactivity from biological factors.

Figure 13.24 A schematic representation of biological cause of hyperactivity being mediated through cognitive factors.

Figure 13.27   The effects of the environment operating directly at the cognitive level with no biological involvement.
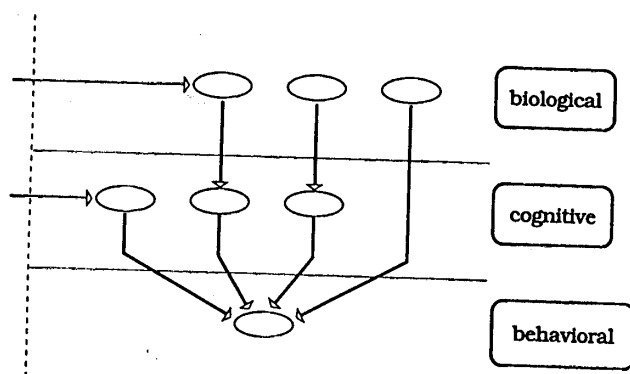
Figure 13.28   A compilation of Figures 13.23–13.27 showing, schematically, a variety of causal pathways for the hyperactivity behavioral phenotype. This is a classic V-shaped causal model.

different, sometimes wildly different, by some other definition, particularly if this definition is at another level. Thus, a group of Fragile X children will display a range of severity of symptom from the mildly hyperactive to full-blown autism. Such a phenomenon is not the same as convergence, which has already been alluded to. Convergence is what happens when a group of autistics, presenting as a single diagnostic entity, are discovered to manifest a variety of biological causes. This is not a problem when one considers any system that develops with multiple contingency and when there are no inconsistencies to unravel.

Variability can be handled in a number of ways. Sometimes, we have embarrassments such as the DSM II-R definition of hyperactivity, where having any 3 out of 8 symptoms qualifies a child. Such pragmatics—categories at all costs—have little scientific content and could even be thought of as antiscientific. In some accounts of hyperactivity, there are signs of uneasiness with this approach and indications that more consistency might be found through redefining the concept at a cognitive or biological level (see above). In other cases, symptoms might be ordered in terms of severity. The ideal patient will show all the signs from the mildest up to the most severe for that patient. What will not
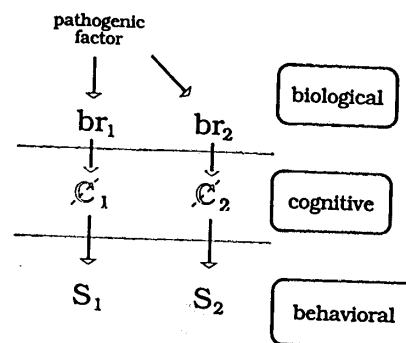
Figure 13.29   In this figure and in Figure 13.30, we illustrate one theory concerning the variability of the expression of a particular condition. Here, the condition has full expression.

be found is a scattering of symptoms of different severity, with some medium severity symptoms missing. The underlying model for the concept of variability would then be that of a quantitative disorder, either at the biological or cognitive level. At the biological level, one could imagine gauging the severity of the affliction in terms of the amount of tissue damaged or the level of a particular neurotransmitter. Another way of referring to this would be in terms of the genetically mediated penetrance of the disorder. At the cognitive level, the diagnostic tasks could be ordered in terms of their information processing demands. Patients would then be located on this continuum as a function of some cognitive measure—for example, the available working memory.

Let us look at one simpler example of variability, a genetic condition where in some cases we find signs S_1 and S_2, but in others we find only S_1. How can we conceptualize this condition? Of the possibilities available, let us illustrate a couple where we examine explanations in terms of a second biological factor that can be either protective (in which case, only S_1 results) or precipitating. In Figure 13.29, we model the assumption that the pathogenic condition would normally give rise to two brain abnormalities, br_1 and br_2. These cause the nondevelopment of the two cognitive systems, C_1 and C_2, which give rise to the symptom complex in its full-blown form. In Figure 13.30, we see how the
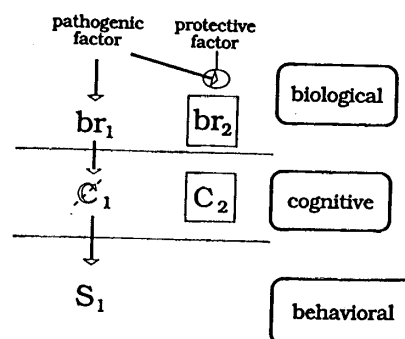
Figure 13.30   In this variant on Figure 13.29, a protective factor mitigates the condition so that only some signs and symptoms are found.
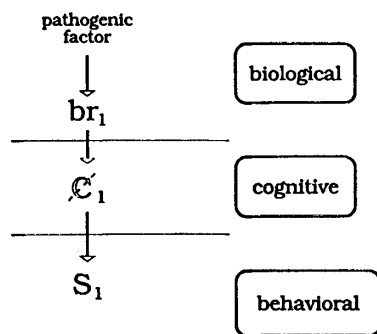
Figure 13.31 An alternative theory of variability of expression of a particular condition. C_2 is not shown because it is not affected by the pathogenic factor. Individuals will not show the group symptom S_2 found in Figure 13.32.

extra factor serves to counteract the effect of the condition on br_2, which will then develop normally. The cognitive structure, C_2, will then be intact (symbolized by the box around it). In this case, the extra factor can be seen as protective.

A second possibility is illustrated in Figures 13.31 and 13.32, where the assumption is that the extra factor works in a negative way. By itself, as shown in Figure 13.31, the pathogenic factor only gives rise to br_1. In such a case, br_2 is not found unless the extra, precipitating factor is present, as shown in Figure 13.32. The two factors are seen as acting in concert—symbolized by the & sign—in producing the br_2 condition. The extra factor here can be seen as making the individual vulnerable to the effects of the genetic condition.

In many cases, we suspect that the source of variation will be hidden at a level well below the one at which the causal model is operating. Johnson and Morton (1991) discussed the sense in which genetic and environmental effects could be equivalent. Thus, differences between individuals can be brought about by "normal" variations within the genotype. Such differences give rise to the range of color, the body type, and some characteristics of the central nervous system that lead to differences in speed of processing. This kind of variation is usually referred to
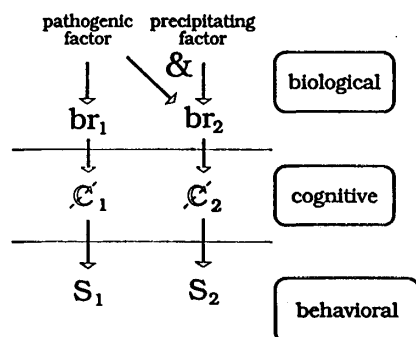
as *individual differences*. "Normal" variations in lower-level environments (especially the in utero environment) would be functionally equivalent here. Thus, maternal variations in diet shift the average height without affecting the genotypic variation. Another source of variation discussed by Johnson and Morton is traceable to abnormalities within the genotype. Down syndrome would be one such variation. Another example is maternal rubella, which, in combination with genotypic factors, can lead to autism in the child (Chess, Korn, & Fernandez, 1971).

Finally, we can review cases where the condition has a known biological origin. For this, we move outside the developmental area and consider a case of a left middle cerebral artery infarct. The variability in symptoms following a hemorrhage will surprise no one and will call for no special principles of explanation. Lesions will differ in their extent as a function of both the exact location of the problem and the individual variation in distribution of the artery. In addition, the effects of two roughly equivalent lesions can be widely different because of the differences in the way that particular psychological functions have become implemented in a particular region of the cortex. All patients who have suffered from a left middle cerebral artery infarct will have symptoms that are related. All will have some form of aphasia and most, if not all, will have some form of dyslexia. At one time, such a description might have been thought sufficient to justify the single diagnostic category, but work in cognitive neuropsychology has established a number of clear categories of dyslexia with different patterns of symptoms and different possibilities of compensatory strategies. The variation, then, is accountable in terms of the cognitive models of the reading process (Patterson, 1981; Shallice, 1981).

PSYCHOSOCIAL PATHWAYS

The causal model, as we have developed it, has a similarity with the psychosocial pathway developed by Rutter (1989) and his colleagues. The main difference is that pathways represent, broadly, trends over time for groups of individuals. They show the contingent relationships among events in the lives of individuals. The pathways are variously termed "chains of adversity" or "chains of circumstance." Usually, these pathways are constructed from correlation or contingency tables. Causality is implied, not explicitly claimed. In these cases, as in our own model, the causal links are not determinate. Where two events are linked by an arrow:

$$x \rightarrow y$$

One is to understand that in those cases where x is found, there is an increasing likelihood of y occurring later. Note that y could occur for other reasons, and that pathway analysis seems to concentrate more on the forward implications. Figure 13.33, based on a study of Gray, Smith, and Rutter (1980), gives a simplified pathway from poor schooling to poor job success, controlling for other variables such as the individual's measured intelligence and social circumstance. It seems to focus more on a series of forward questions such as:

Figure 13.32 A variant on Figure 13.31, where an extra factor works in a negative way, for example, making the individual especially vulnerable. In this case, C_1 and C_2 are both defective.
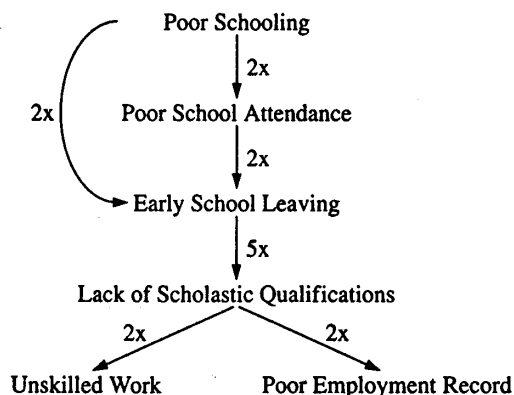
"What are the consequences of poor schooling?"

rather than backward ones such as:
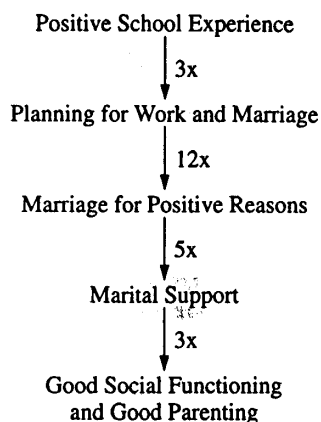
"What are the causes of poor employment records?"

Figure 13.33 indicates comparative rates of traversing each subpath. We see that children with poor schooling are twice as likely to have poor school attendance (compared to children with good schooling). The next stage of the pathway, rather than being the continuation of the first part (that is, using only children with poor schooling) is actually recomputed from scratch, using all children with poor school attendance. Thus, the relationship of poor school attendance and early school leaving is a twofold increase over the group with good school attendance. Remember that this calculation ignores the quality of the schooling. In fact, we are also told that the poor schooling group is only twice as likely to leave school early. This means that we can infer that poor attendance with good schooling has a *worse* outcome than poor attendance with poor schooling. Poor schooling might then be seen as a mitigating circumstance for early school leaving in the poor attendance group!

In the rest of the pathway, we are not given the necessary data to see whether the same complex contingency is a factor. However, it is possible that, although a child with poor schooling was more likely to lack scholastic qualifications, a child who lacked scholastic qualifications because of poor schooling was less likely to end up in unskilled work than someone without qualifications who had good schooling.

Rutter (1989) described the figure from Gray et al. (1980) as "simplified," and in this form the figure may be less than helpful for thinking about some of the problems. Rutter also brought together a number of examples of how the psychosocial pathway was used to show different outcomes. One example, shown in Figure 13.34, comes from a study by Quinton and Rutter (1988) on the

Positive School Experience
↓ 3x
Planning for Work and Marriage
↓ 12x
Marriage for Positive Reasons
↓ 5x
Marital Support
↓ 3x
Good Social Functioning
and Good Parenting

Figure 13.34 The psychosocial pathway: the outcome for institutionally reared girls. From "Pathways from Childhood to Adult Life" by M. Rutter, 1989, *Journal of Child Psychology and Psychiatry, 30,* p. 33. Copyright 1989 by the Journal of Child Psychology and Psychiatry. Reprinted by permission.

outcome of institutionally reared girls, a third of whom manifested parenting breakdown. More detailed analysis showed that this was not caused by the institutionalization per se, but rather by the quality of the family home. If, on leaving the institution, the child returns to a discordant family in adolescence, the likelihood of an early marriage with poor prospects is increased. This is associated with increased risk of poor social functioning which, in turn, is associated with an increase in breakdown of parenting. Girls who return from the institution to a harmonious home during adolescence are much more likely to end up as adequate parents. It is tempting to see such intergenerational transmission as inevitable, but there is at least one other major factor, the quality of the schooling. Children's homes tend to distribute the children in their care around the local schools. This leads to a variety of school experience which has a major impact on outcome, as indicated in Figure 13.34.[21]

We have analyzed these examples in some detail as a means of contrasting them with the causal model. One clear difference that emerges from the above analysis is that, although *cause* propagates down the causal model, it does not propagate down the psychosocial pathway as usually constructed. This is not accidental, we feel; rather, it expresses a view of life-span development that is gaining currency. By this view, adverse early life events are no longer seen as *determining* later outcomes (Clarke & Clarke, 1992). Instead, such events place the child in a disadvantaged situation from which it is more difficult to achieve a satisfactory outcome at the next stage. They cause more vulnerability to bad outcomes from later setbacks. The adverse early events certainly are a disadvantage, but this is not the same as life-span determination.[22] This analysis may all look dangerously close to a social

Poor Schooling
↓ 2x
Poor School Attendance
↓ 2x
Early School Leaving
↓ 5x
Lack of Scholastic Qualifications
2x ↙   ↘ 2x
Unskilled Work   Poor Employment Record

(2x curved arrow from Poor Schooling to Early School Leaving)

Figure 13.33 The psychosocial pathway from poor schooling to poor job success. From "Pathways from Childhood to Adult Life" by M. Rutter, 1989, *Journal of Child Psychology and Psychiatry, 30,* p. 31. Copyright 1989 by the Journal of Child Psychology and Psychiatry. Reprinted by permission.

[21] Note, however, that there could well be contingencies between family background and the ability to profit from school, even when intelligence and other individual variables are taken into account.

[22] The fault ". . . is not in our stars but in ourselves . . ." (Shakespeare).

philosophy rather than a scientific analysis, but cause and responsibility are not to be equated.[23] In summary, it is possible to say that a particular behavioral sign is caused by a genetic abnormality at the beginning of the causal chain. However, we are not on so good a ground in saying, for example, that someone's poor employment record is caused by his or her poor schooling.

Another, related difference can be found between psychosocial pathways and causal models. In the causal models, we can represent the full causal path for the condition for any affected individual or, alternatively, for all individuals in a defined group with the full variety of possibilities. With respect to the time dimension, we can note that the psychosocial pathway is restricted to the historical events, whereas the causal model may or may not represent temporal sequence. A notation that has temporal sequence at its core is Developmental Contingency Modeling (Morton, 1986). This notation also is more natural when discussing the preconditions for normal development.

DEVELOPMENTAL CONTINGENCY MODELING

To give an example, let us start with the following question: "What is the cause of the development of pretend play?" A question of this form seems distinctly odd because it has to do with the relative uniqueness of A in a claim that *A caused B.* The uniqueness takes these forms:

1. There should not be many other examples of x for which it would be the case that *x caused B.*
2. Condition A should not be common in the population.
3. There should not be too many people for whom A is true but B is not.

Notice that this use of *cause,* which is the way it is used in ordinary language (including in medicine), is very much weaker than the notion of implication: if *A implies B,* then there would be no cases of A without B. *Cause,* in the developmental domain (and, indeed, in the human domain as a whole), is not nearly so strict, for reasons we will explore below.

The above constraints on *cause* mean that we cannot (that is to say, we normally do not) talk about the cause of a normal condition. In particular, too many things would count as causes to make the notion worthwhile. Instead, we might refer to the *preconditions* for a particular aspect of normal development. For such reasons, Morton (1986) developed the Developmental Contingency Model (DCM), which allows one to trace the normal developmental preconditions for a particular process or skill. We will illustrate DCMs in the area for which this theoretical device was developed: the emergence of mentalizing in normal development. This brings us back to the question on the cause of the development of pretend play, posed at the beginning of this section. Here, instead of asking about the *cause* of pretend play, we are asking about the *preconditions* of pretend play, and the related abilities. In fact, we will focus on only one of the many necessary preconditions.

Figure 13.35 is a developmental contingency model, not a flow chart or an information processing model. The symbol on the lines in Figure 13.35 is to be read as "(normally) requires the (pre) existence of." Thus, pairs of connected elements are related developmentally, and each such pair effectively represents a hypothesis about developmental contingencies. We have choices regarding which elements to represent and their relationship.

The particular form of Figure 13.35 was driven as follows. We wanted to separate representations having to do with things from those having to do with people. We called these representations material (MAT) and individual (IND), respectively. IND constitutes those representations that have to do with individual people. MAT refers to all other representations. This division is based solely on content; we are *not* postulating the existence of two different memory stores. In Figure 13.35, then, MAT is to be understood as the *ability* to create representations with respect to objects. IND is to be understood as the *ability* to create representations with respect to individual people. We have separated IND from MAT because there will be prerequisites for IND that are not shared by MAT. These will relate to the differentiation of individuals from each other, an ability not necessarily tied to the differentiation of objects from each other. It may
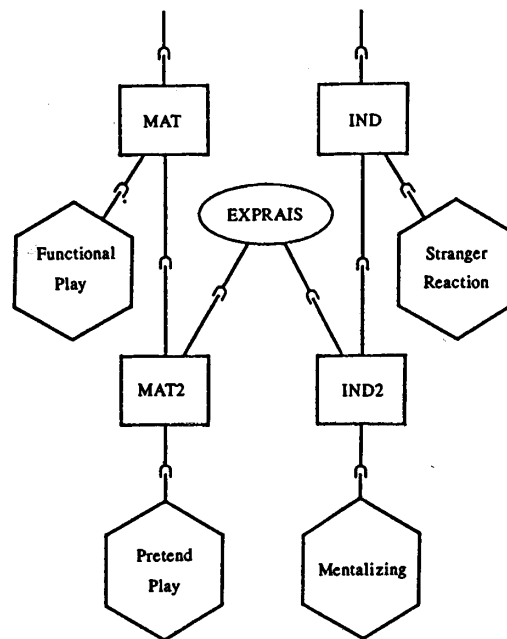
Figure 13.35 A developmental contingency model (DCM) relating to autism. From "Developmental Contingency Modelling: A Framework for Discussing the Processes of Change and the Consequence of Deficiency" by J. Morton, 1986, in P.L.C. van Geert (Ed.), *Theory Building in Developmental Psychology* (p. 148). Copyright 1986 by Elsevier Science Publishers. Reprinted by permission.

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[23] An extreme example of the separation is given in a guidebook to Bali. The traveler is warned of the consequences of being involved in a traffic accident. Irrespective of the cause of the accident, it is claimed, the tourist will be deemed responsible because if the tourist had not visited the island, the accident would not have taken place.

also be important to separate MAT and IND for the reason that the understanding of individuals as agents and the understanding of objects as physical causes seem to be separate developmental achievements (Carey, 1985; Gelman, 1990).

MAT and IND are first-order representations. The equivalent second-order representations are created by the action of a specific mechanism, first called expression raiser (EXPRAIS) by Leslie (1987). EXPRAIS operates on first-order representations. Thus, a first-order representation can be decoupled from its job of representing a real state of affairs ("It is raining.") and used inside a phrase (She thinks, "It is raining."). We call the resulting ability to create second-order representations MAT2 and IND2. A single knowledge fragment, or "thinks," could include all four kinds of representation. The following fragment is an example:

Daddy is on his brown chair (which I am pretending is my space machine) and he pretends I'm an alien.

In this fragment, the four kinds of representation are as follows:

MAT: the chair is brown
MAT2: the chair is a space machine
IND: Daddy is on the chair
IND2: Daddy pretends I'm an alien.

We maintain the separation among these four classes of knowledge because we believe that some people cannot create some of them and because they do not appear to develop at the same time (Leslie, 1987). In Figure 13.35, the relationship between MAT and MAT2 should be understood as follows: the development of the ability to form MAT2 representations is contingent on having previously developed the ability to form MAT representations.

In Figure 13.35, we have represented a number of claims made with respect to mentalizing and other skills. Thus, the existence of pretend play presupposes the existence of MAT2 representations. In the same way, mentalizing (the ability to attribute mental states) requires the ability to create IND2 representations. The contrast is made, on the one hand, with so-called functional play, which only requires first-order MAT representations, and, on the other hand, with the stranger reaction, which only requires first-order IND representations. If someone cannot mentalize, it is because that person cannot form IND2 representations. The diagram makes it obvious that an inability to form IND2 representations could arise for two reasons. First, if the EXPRAIS was missing, there could be no IND2. This was our original supposition for autism. The resulting inability to mentalize would be accompanied by a lack of pretend play, which also relies on the existence of EXPRAIS. At the same time, the ability to represent objects and individual agency would be presumed intact.

Second, there may be an absence of IND representations, or the ability to create IND representations may be computationally insufficient to allow the creation of IND2 representations. Such an individual would fail the Sally-Anne experiment but would be able to pretend play—so long as EXPRAIS existed. This is the case in the normally developing 2- to 3-year-old child.

One important feature in developmental contingency modeling is that there are always unexpressed contingencies. We only consider here the developing abilities and the kinds of knowledge that are germane to the particular theory of development being represented.

The Properties of the Notation

In the DCM shown in Figure 13.35, there is the objective that contingencies should be traced back to the biological givens.[24] A second presupposition is that such biological givens will be buried deep with respect to behavior. Each one will be implicated in a wide range of activities, and the absence of any one will have far-ranging consequences. A third presupposition is that no special environmental conditions are required for the normal fruition of the givens. That is not to say that there is no learning, but only that the learning is effort-free. The child learns about language, objects, family, causality, number, and so on, in an effort-free way because what is happening in the course of such learning is that the givens are being used. There is almost a teleological element about this process. The processing machinery and the innate structures are constructed in the way they are in order that the goals shall always be reached. This is the achievement of evolution. The child has no choice in the matter; its "learning" is under the control of its processes. A child can choose not to speak but it cannot choose not to learn its native language. The biological givens that subsume language learning make sure of that.

In the DCM framework, the focus is on the prerequisites for the emergence of a particular process or structure. Such properties of the infant brain form "elements" in a DCM model. Although our direct evidence for the existence of such an element will be behavioral, our primary focus will be on the elements and not on the behavior. There are two main reasons for this. First, an element may be present without being visible in behavior. Thus, a profoundly deaf infant who has no experience of sign language still has the innate component of the language learning apparatus. The presence of this component becomes revealed as soon as signing starts. Before this point, the component is not able to exert any significant influence on behavior.

Second, a particular piece of behavior could be mediated by a variety of means. For example, autistic children may learn to have exchanges of utterances with adults. However, in the majority of cases, such exchanges would not, on close analysis, be confusable with the conversations that normal children have. Normally, conversations are driven by IND2 representations (among other things) and are intrinsically "reinforcing" for normal children. The autistic child would only slowly learn that they were appropriate modes of behavior.

In the preceding paragraphs, we indicated why the focus of the DCM method is on the elements of the child's cognitive apparatus rather than on behavior. We should now look at the elements more closely. Elements are either primitive or not. By "primitive" we mean innate and irreducible. Trivially, either a particular element E can only emerge if some specific element D has already emerged (to some level of specification), or E is a primitive. The development of nonprimitives depends on the prior functioning of particular primitives plus exposure to specific kinds of stimuli.

[24] This is, of course, the first of our maxims of causal modeling.

Primitives require, at most, a minimal environment. Primitives need not be present from birth; they can arise in the course of maturation.

In practice, there will be a variety of patterns of contingencies. Thus, one can imagine a skill whose emergence is a function of a late maturing structure but which also depends on the prior existence of other processes or knowledge. We would want to be able to represent all such contingencies. The general form of the contingency model is that of elements connected in a directed graph. The elements can be of a variety of kinds—processes, structures, knowledge, perceptual or other experiences, or biological elements. The symbols on the connecting lines have temporal/causal implications.

The Relation between DCMs and Causal Models

We noted in the previous section that it doesn't make much sense to talk about the *cause* of normal development. For this reason, we introduced the Developmental Contingency Model (DCM). The relation between the two is quite straightforward. Suppose we are interested in the development of an ability, A—say, speech. We establish that the normal development of A depends on the prior development of at least two features, X and Y, ears and mouth. The DCM for this development is shown in Figure 13.36.

From what we have already said, it should be clear that the normal development of A (speech) will be prevented or impeded if there is delay of development or malfunction of either X (ears) or Y (mouth). Thus, there will be two different causal models of malfunction of A, depending on whether hearing or producing speech sounds is affected. The two causal models are shown in Figure 13.37.

In case (i), it is possible that feature Y (producing sounds) is still intact; in case (ii), it is possible that feature X is still intact. Let us examine case (i). The first step in establishing that feature Y is functioning would be to test some other ability, B, which would normally also depend on the existence of feature Y. Thus, a more complete DCM would be as shown in Figure 13.38.

Children categorized under case (i) would show ability B; children under case (ii) would not. We would, however, expect case (ii) children to show other abilities that depend on feature
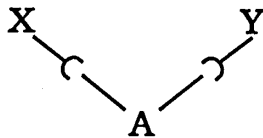

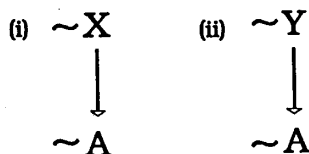**Figure 13.36**   A simplified developmental contingency model.

Figure 13.37   The causal implications of DCM in Figure 13.36: no X leads to no ability A; no Y also leads to no ability A.
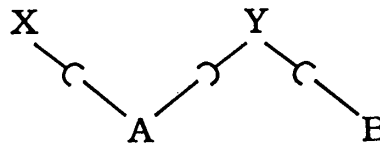

Figure 13.38   A DCM that slightly elaborates the one shown in Figure 13.36: two abilities, A and B, presuppose Y. Ability A also presupposes X.
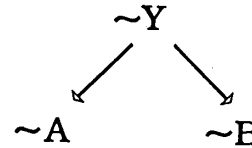

Figure 13.39   A causal model related to the DCM in Figure 13.38: no Y leads to double disability A and B.
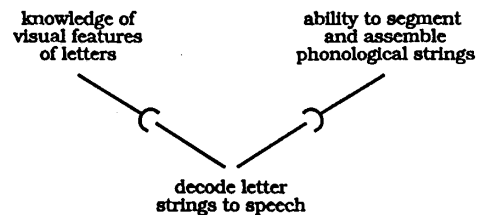

Figure 13.40   A fragment of a DCM for acquisition of reading.

X.[25] The more complete causal model for case (ii) would take into account both abilities A and B, as shown in Figure 13.39.

We can illustrate this principle with an example from the area of literacy development. One component of mature reading is the ability to decode letter strings into speech. Many factors are prerequisites for this skill. We will select two: (a) knowledge of the visual features of letters and (b) the ability to segment and assemble phonological strings. The relationships among these abilities are shown in the DCM fragment in Figure 13.40. If a child has failed to acquire either the letter knowledge or the phonological skills, then this child will not be able to acquire the decoding skill. These simple causal relationships are shown in Figure 13.41. If we only know that a particular child has no decoding skills, we cannot tell whether this is because the child lacks one or another of the prerequisites (not to mention other possibilities). However, the phonological abilities will reveal themselves in other simple tasks, such as an "I spy" game.[26] This dependency is represented in

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[25] The prediction of the absence of an ability depends on the child's being unable to develop the ability through a compensatory strategy. As we shall see, this is not always the case; a case (ii) child might not demonstrate ability B because of the absence of Y, but may exhibit ability A through compensation. Thus, a deaf child can learn to speak via lip reading, even though the normal route for doing so is blocked.

[26] The "I spy" game involves one person saying, "I spy, with my little eye, something beginning with b" (or some other letter name). The other person has to guess the object in question.
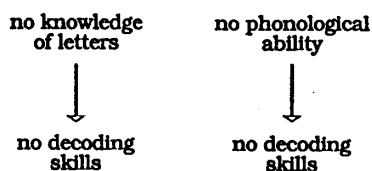
Figure 13.41 The causal implications of the DCM in Figure 13.40. Absence of either of the two abilities in Figure 13.40 will lead to an inability to decode letter strings into speech.
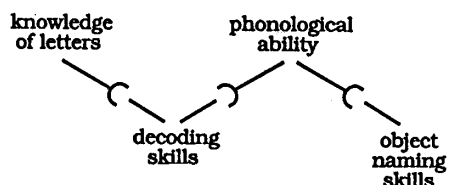
Figure 13.42 An example of the DCM in Figure 13.38. This indicates that phonological ability is necessary not just for decoding but also for object-naming skills.
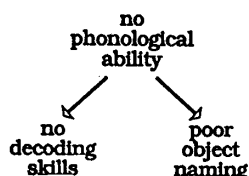
Figure 13.43 The causal model implications of the DCM in Figure 13.42.

Figure 13.42. The causal consequences of an absence of phonological skills can now be extended, as in Figure 13.43. Other things being equal, we can tell the difference between a child who cannot decode simply because letter knowledge is absent and a child who lacks the requisite phonological skills. In the next section, we look at performance on the "I spy" game and on a letter-naming game, using dyslexia as a way of illustrating the generality of the notation that we have been developing.

A CAUSAL ANALYSIS OF DYSLEXIA

The Dyslexia Debate: Is There Such a Thing as Dyslexia?

The first distinction we have to make is between *true dyslexia* and reading difficulty of the type that is sometimes called the garden variety (Gough & Tunmer, 1986). This is not as easy a task as might initially appear (Stanovich, 1988). What we are seeking to know is the difference between those children who are delayed for reasons stemming from the relationship between the child and the educational process, and those children who have some clear and specific cognitive deficit that gives rise to a reading problem. There appear to be major differences in the literature with respect

to the delineation of these groups. On the one hand, we have the biological school, exemplified by Galaburda and his team. The advance publicity information for a conference cosponsored by the New York Academy of Sciences, in September 1992, and organized by Galaburda, contained the following (Tallal, 1988):

> Despite normal intelligence some 10% of our school children have great difficulties in learning to read and write. This severe handicap, which is often combined with developmental language impairment (dysphasia), seems to have a neurological etiology.

The implication is that all 10% are to be seen as neurologically impaired. The basis of the claim is work comparing the brains of dyslexics and of normals with respect to certain parameters (Galaburda, 1989; Galaburda, Rosen, & Sherman, 1989; see also Hynd & Semrud-Clikeman, 1989, for a review).

This position contrasts with that of educationalists such as Marie Clay, who see reading difficulties as educational problems from a variety of causes, all of which have educational solutions (Clay, 1979). Clay's work in New Zealand involved retesting children after a year's reading instruction. Those children in the bottom 10–20% in reading attainment, irrespective of the apparent cause, were given remedial teaching, individually designed and delivered by highly trained specialists, half an hour a day for 12–20 weeks. Clay reported that 70–90% of these children responded to this special teaching by attaining class-appropriate performance (Clay, 1987). The children who did not respond included some who arrived at satisfactory performance levels the following year, without further intervention, and some children who turned out to be significantly handicapped. The implication is that less than 1% of the original population had a permanent, specific handicap.

How is one to reconcile these two sets of ideas and facts? There are a number of possibilities:

1. Educationalists tend to use a behavioral definition of reading difficulties and are concerned only with children at primary school age. Neurologists tend to consider only cases that evidence a lifelong handicap in reading and reading-related skills, regardless of systematic improvement over time.

2. Educationalists might argue that the Galaburda team sampled a set of extreme dyslexics with brains not typical of the reading-impaired population (the sample were self-referred by willing their brains for scientific research). The vast majority of dyslexics, by this argument, would not be expected to show any brain abnormalities.

3. Neurologists might argue that all people with developmental reading difficulties have some kind of cortical abnormality but only in a minority of cases is the difficulty not remediable. This account also requires a particular assumption: that most cases of reading difficulty are remediable if caught in time, or are only remediable if they are treated by a method that includes a feature contained in the Clay method.

We will attempt to take these possibilities into account in the discussion that follows.

The Discrepancy Definition of Specific Reading Disability

It is well known that there are great difficulties in defining reading failure as opposed to generally low academic achievement. The pioneering work in studying the total population of a particular age group in the Isle of Wight (Rutter, Tizard, & Whitmore, 1970; Rutter, Tizard, Yule, Graham, & Whitmore, 1976) showed that it was hard to establish clear-cut differences on the behavioral level between children who were specifically reading disabled and those who were merely backward. Their reading patterns were equivalent. To identify the target subpopulation of dyslexics, intelligence had to be taken into account. Using a discrepancy definition based on the regression between reading test scores and intelligence test scores, a group of underachievers could be identified who had "specific" difficulties—they were unexpectedly failing to become literate at the pace of their peers (Rutter & Yule, 1975).

Ultimately, this method, which seemed very promising as a basis for identifying dyslexia, was defeated by its own strength: its behavioral descriptive basis. In the measures available, no consistent and meaningful neurological correlates could be found that would allow the delineation of a biologically based syndrome. On the contrary, the *backward* group exhibited neurological symptoms such as clumsiness, deafness, visual problems, and epilepsy. Particularly damaging was the later claim that the originally proposed "hump" in the normal distribution of discrepancy scores was the result of a statistical artifact (van der Wissel & Zegers, 1985). What was actually shown was that the hump *could have been* an artifact. In any case, it is very difficult to demonstrate the presence of two different distributions when one of them is a very small subsample.

The next step, which proved irresistible to critics, was to deny that there is such a thing as dyslexia (Bryant & Impey, 1986; Prior, 1989; Treiman & Hirsh-Pasek, 1985). However, this is a step too far (Miles & Haslum, 1986). There is good evidence that reading and spelling problems can be caused by genetic factors (Pennington, 1991). On the other hand, the discrepancy definition of dyslexia does not map on to such a concept, as is already apparent in the Isle of Wight studies. How could this state of affairs come about?

The behavioral definition of reading difficulties is not the same as a cognitive definition. A discrepancy definition is a definition at the behavioral level and can only distinguish between two broad categories:

1. There are children who show no discrepancies. Their poor scores on reading tests could be a consequence of general developmental delay, general learning disability, or adverse external circumstances, such as lack of reading experience.

2. There are children with poor reading test scores relative to IQ. This could be a sign of a specific cognitive deficit, but not necessarily.

Some children may be found to be reading-disabled relative to their IQ for rather tangential reasons: they may not speak the language, or they may have missed out on schooling because of illness, bullying, or poor teaching. Some children may appear to be specifically reading-disabled simply because they are growing up in a culture where schooling and literacy are not valued. All these children would be picked up in big sweeps of educational tests and would look worse off even than true dyslexics, in terms of their reading test scores. They may be shown to be dyslexic in terms of a discrepancy definition; however, they are only pseudo-dyslexic. The Clay sweep at the end of the first school year gathers up the bottom 15 + % of readers to place in a remedial program, and includes these types of children. One would expect particularly high success rates if these children were taught individually and might be given their first real chance at learning to read.

Current reading test scores do not identify particular types of specific disability. Therefore, the category of specific underachievement necessarily lumps together children whose deficit could also be caused by, say, visual impairment. Not all children with an unexpected reading failure suffer from a specific underlying cognitive deficit. Let us stress the causal asymmetry here. We agree that if there is an underlying cognitive problem, then there will be behavioral signs. However, the presence of such behavioral signs does not necessarily imply an underlying cognitive problem. There is another reason for dissatisfaction with a discrepancy definition of dyslexia.[27] Within the framework we have set up, we can readily make the required distinctions within the area of specific reading difficulties. The notation introduced in the previous section can help us solve the problem of representing true dyslexia as well as representing different causes for dyslexia, or subtypes of dyslexia. We can also separate out pseudo-dyslexia, the reading failure originating from external and often reversible causes.

Toward a Cognitive Definition

Beginning with the Isle of Wight studies, a large body of work on specific reading difficulty has succeeded in sharpening the distinction and in laying the foundations of genetic studies of a particular type of dyslexia that seems to run in families and to be more frequent in boys than in girls (Critchley, 1970; Pennington, 1989). The main outcome from the genetic studies (twin studies and family pedigree studies) is that the phenotype for the disorder is a phonological processing problem (Olson, Wise, Conners, & Rack, 1990; Olson, Wise, Conners, Rack, & Fulker, 1989; Stevenson, Graham, Fredman, & McLoughlin, 1987). This outcome was independently arrived at in a wide variety of psychological studies comparing dyslexic readers and reading-age-matched normal readers. We shall focus on this particular condition as a prototype of dyslexia and will model it in an X-type causal diagram. We make no theoretical claim other than those implicit in the position we are modeling.
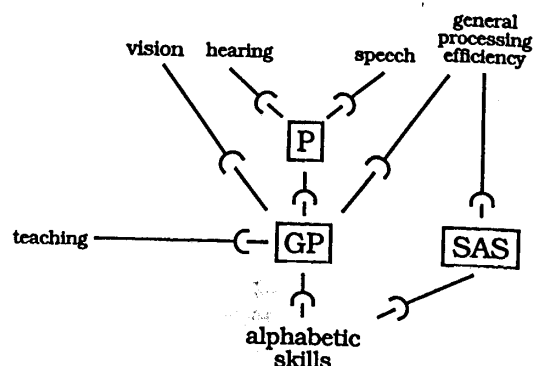
Why is there continuing disagreement as to the existence of such a prototype? It has been suggested that a simple yet sensitive

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[27] A further reason for not trusting the behavioral discrepancy definition is that it excludes dyslexics who are compensated sufficiently to be accurate readers but still read very slowly and with effort.

behavioral measure that will distinguish dyslexics with phonological problems is performance on nonword reading and spelling (Rack, Snowling, & Olson, 1992; Siegel, 1989; Snowling, 1987). Ordinary reading tests that consist of real word recognition and text comprehension are likely to camouflage the problem because, by sheer rote learning, a child may acquire a large sight vocabulary, and, by sheer intelligence, may be able to use sentence context to guess the gist of the message.[28] The question that fires the debate is: What is special about dyslexic readers that is not also shown by garden variety poor readers or indeed by young normal children before they have learned to read? In other words, are there differences among these groups at the cognitive level? Before we can address these questions, we need to look at the prerequisites for the normal development of literacy. We do this because what applies to deficits also applies to development.

Contingencies in Literacy Acquisition

In Figure 13.44, we diagram some of the main contingencies for learning to become literate in an alphabetic script. One important internal prerequisite is a minimum of general processing efficiency.[29] Furthermore, we need to assume that there is adequate vision and hearing—the normal input channels for the skill to be learned. (Children with impairments in these channels would have to be acknowledged separately.) In addition to these very basic prerequisites, which figure in a great many developmental contingencies, we require two cognitive capacities that are specifically relevant: (a) a normally developing phonological system, P, which many researchers have proposed is damaged in true dyslexics, and (b) a normally developing Supervisory Attentional System (SAS), as proposed by Shallice (1988). This general SAS needs to function efficiently if any formal learning is to take place, and we assume that, when this SAS is immature or damaged, the acquisition of any taught skills, including reading, would be difficult or impossible, even if all other prerequisites were available. This might be the case with certain types of attention disorder (see the section on Hyperactivity, above). External input—specifically, teaching—will be required for any progress in learning to read. If all of these internal and external conditions are fulfilled, then an automated system for handling grapheme-phoneme (GP) correspondence will be established, and, as a result, alphabetic skills will be evident.[30] Only after achieving a certain degree of proficiency with alphabetic skills will the child go on to become an orthographically skilled reader (Frith, 1985; Morton, 1989a).
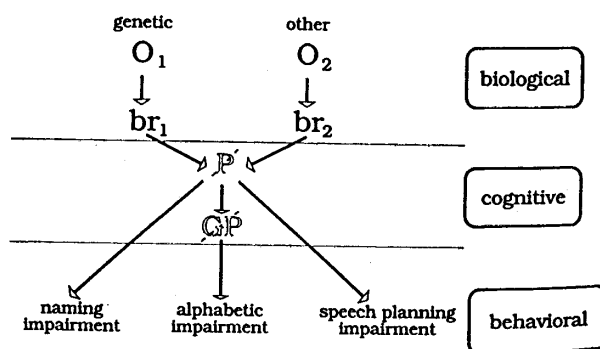
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[28] That the vocabulary score correlates very highly with all other reading measures, including those that stress comprehension at the most abstract level, is another problem. The correlation is valid only for the normal population. Where there is a cognitive deficit and the child has adopted a compensatory strategy, normal population statistics are no longer applicable.

[29] The concept of general processing efficiency is discussed extensively by Anderson (1992).

[30] The alphabetic skills will, of course, depend on more than GP. Thus, the ability to segment at the level of onset/rime enables children to use spelling patterns in familiar words (beak) when decoding unfamiliar words (peak) (Goswami, 1986, 1990).

Figure 13.44   A minimal set of contingencies for learning to become literate in an alphabetic script.

An X-Type Causal Model of Dyslexia

As with autism, enough facts are available at both biological and behavioral levels of description to suggest that there is a diagnostic entity. This occurs even though (a) the biological origins of dyslexia are unknown and are likely to be multiple, and (b) the signs and symptoms are extremely variable and have not yet been sorted out into core symptoms, additional associated problems, and secondary consequences.

The causal model for dyslexia that seems to be well supported by the weight of the evidence is given in Figure 13.45. As with autism, the model includes alternative biological origins, a single, defining cognitive deficit, and a variety of core and other signs and symptoms. We would not be surprised, however, if, in the future, different or additional deficits were

Figure 13.45   A schematic causal model for dyslexia, involving all three levels of description. The defining condition is a cognitive deficit, an absence of the cognitive structure P. This leads to absence of structure GP, involving automated routines that relate sounds and letters. The biological causes may be of different types, genetic and other. Behavioral consequences include problems other than lack of decoding skills.

considered necessary for a full explanation of the many varieties of reading problems.

Biological Factors

As yet, the biological basis of dyslexia is a matter of speculation. Galaburda (1989) reviewed the anatomical evidence from 8 brains analyzed up to that time. The brains all showed an absence of ordinary asymmetry in the planum temporale, a particular area of the temporal lobes that is thought to subserve speech and language. Exactly what this implies in terms of developing psychological functions is unknown. ,

More recently, Livingstone, Rosen, Drislane, and Galaburda (1991) reported further anatomical findings, this time concerning the visual cortex, in particular, V1. Here, too, abnormalities were found in dyslexic brains, specifically in the magnocellular system, which is responsible for low-contrast, high-speed visual processing. Experimental evidence for a deficit in this process has been provided by Lovegrove, Garzia, and Nicholson (1990). Although a visual cognitive deficit underlying reading failure is a perfectly reasonable option (see the models in Figures 13.49 and 13.50, below), we cannot jump to the conclusion that there is a deficit that is different from our previously assumed P structure. We shall have to await further evidence before we can talk about contrasting biological causes that may or may not be connected with contrasting cognitive deficits.

The genetic findings (Olson et al., 1989; Smith, Kimberling, Pennington, & Lubs, 1983; Stevenson et al., 1987) all agree that there is an identifiable, single component underlying dyslexia, namely, a deficit in phonological processing. Scarborough (1990) studied preschool children whose parents were dyslexic. Of these children, 65%, a remarkably high level, were later themselves diagnosed dyslexic. These children had tended to show deficits in certain speech processes by age 3. In a recent theory concerning brain lateralization, Annett (1992) went one step further, postulating a single gene for left hemisphere specialization, which enables efficient phonological processing. Thus, in the normal population there will be wide variation in the extent to which speech is supported by dedicated neural systems. Individuals at the extremes of the normal variation may be at risk for dyslexia (Annett & Kilshaw, 1984).

It is notable that scarcely any theories of the origins of dyslexia have implicated other than genetic factors. A possible exception is the somewhat speculative Geschwind-Galaburda hypothesis explaining abnormalities in brain asymmetry that might be relevant to dyslexia in terms of complex interactions of intrauterine environments and sex of fetus (Geschwind & Galaburda, 1985). The biological level in our causal model will have only one node, corresponding to genetic disorders, although it is anticipated that a number of different types of genetic disorder will be identified. Bishop (1990), who provided a critical evaluation of both Annett's and Geschwind's theories of cerebral lateralization and dyslexia, concluded:

[T]here is little support for the theory that individual differences in the direction and degree of laterality of language representation are the basis for developmental dyslexia. (p. 129)

Cognitive Factors

The consensus of the best available research is that a proportion of poor readers—garden variety as well as true dyslexics—is deficient in the formation of the particular cognitive structure that is responsible for grapheme-phoneme correspondence (Frith, 1985; Johnston, 1982; Snowling, 1991; Snowling, Stackhouse, & Rack, 1986). This can have a number of different causes. In true dyslexia, as we have described it in our X-type model, the cause is a deficient P structure. This deficit results in not only a faulty GP structure and poor alphabetic skills but additional impairments as well. Some agreement has been reached as to the crucial impairments (Catts, 1989; Pennington, 1989). These all concern problems in the phonological processing of spoken language and are termed by Pennington: "name retrieval," "verbal short-term memory," and "speech production." It will be apparent from this description that the deficit proposed by such theorists to underlie dyslexia will reveal itself well before the normal onset of literacy.[31]

There is widespread agreement that the developmental deficit in system P, which we assume characterizes dyslexia (Olson et al., 1989; Shankweiler, Liberman, Mark, Fowler, & Fischer, 1979; Snowling, 1987; Stanovich, 1988), can be indexed by a variety of phonological tests. Such tests include alliteration and rhyming (Bradley & Bryant, 1983; Bryant, Maclean, Bradley, & Crossland, 1990), nonword reading (Rack, Snowling, & Olson, 1992), and rhyme matching (Lenel & Cantor, 1981), all of which involve the use of certain components of phonological skills.[32] Many studies have shown that the critical component P is also absent in normal children below school age (see Figure 13.46), presumably because of a relatively late process of maturation (Byrne & Fielding-Barnsley, 1989; Liberman, Liberman,
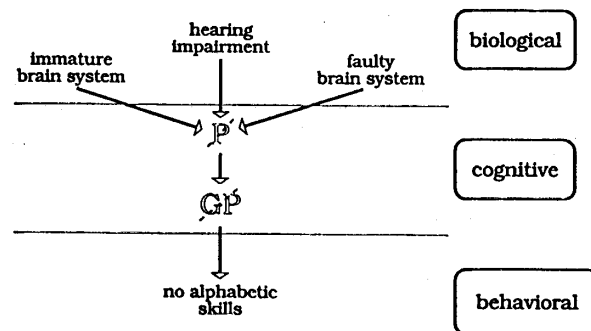
Figure 13.46 Failure of structure P can come about either through a fault at the biological level or, with young children, because of immature biological structures. In the latter case, the "dyslexia" is purely temporary.

[31] It also follows that the deficit underlying dyslexia in English-speaking children should be found in all cultures, including nonliterate cultures and nonalphabetic cultures.

[32] At the moment, it is unclear how this developing process might be characterized. Its identification at the cognitive level is a major goal for researchers in the field.

Mattingly, & Shankweiler, 1980; Lundberg, Frost, & Peterson, 1988).[33] A third reason for the lack of development of the critical component, P, could be hearing impairment, including intermittent impairments such as otitis media with effusion (OME).

### Difficulties of the Beginning Reader

The reciprocal relationship between literacy acquisition and phonological skills (Cataldo & Ellis, 1990; Ehri, 1984; Perfetti, 1991; Stuart & Coltheart, 1988) means that clear causal pathways for reading failure are difficult to establish. For instance, subsyllabic segmentation and blending skills appear simultaneously with alphabetic reading skills. However, it seems likely that different components of phonological processes are evoked when typical phoneme awareness tasks are given to a person who is literate and when given to a preliterate child (Morais, 1991).

There is a clear correlation between the emergence of P-indexed skills in preschool children and their subsequent smooth and early entry into an alphabetic system, but there is also evidence from training studies (Lundberg et al., 1988) to strengthen the notion of a causal relationship. Wimmer, Landerl, Linortner, and Hummer (1991) showed that prereaders with poor P-indexed skills (e.g., "mis-repetition," not being able to substitute one particular vowel for another vowel in a single word) could be divided into two groups after the first school year: (a) those whose P component was simply delayed compared to their peers but who subsequently became competent readers, and (b) those who remained persistently poor readers, with the possible explanation that their P component was faulty.

In its simplest form, this hypothesis supposes that, at preschool age and on standard tasks, an immature P system is not distinguishable from a faulty P system. The implication of this theory is that the brain systems involved in Figure 13.46 should be the same in the case of fault and delay. The study by Scarborough (1990) of preschool children at risk for dyslexia suggested, however, that a careful analysis of speech processes may pick out a dysfunctional from a merely immature P system even at this early stage. On this view, one would expect the biological components in the causal chains of the two to be different from each other. We would also expect remediation in the case of delay to be much more straightforward than in the case of deficit.

Behavioral Signs and Symptoms

There are other reasons for not developing a GP system, and we have already alluded to several other prerequisites apart from the phonological system in Figure 13.44. In other words, specific evidence is needed in order to draw the conclusion that a child is truly dyslexic—which we equate here with a faulty P component. Figure 13.47 is a diagram of the possibility of different types of
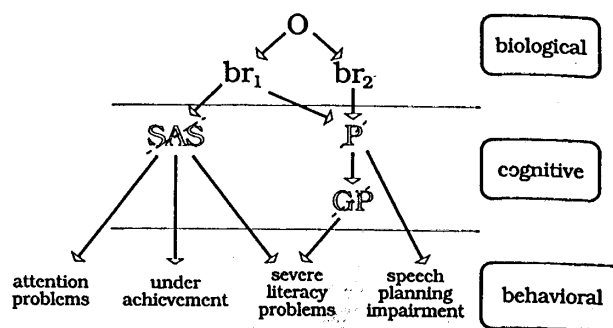
Figure 13.47   A hypothesis of aggravated dyslexia where a biological deficit can cause not only a lack of the P system but also a deficit in the supervisory attentional system (SAS).

biological causes, one of which affects both the P component and the SAS component. This would be the case for an individual suffering from a severe learning difficulty affecting not only literacy, but also any other type of formal learning. Figure 13.48 illustrates a case where only the SAS component is directly affected by a biological malfunction. This could occur with attentional deficit disorder. When certain aspects of the SAS system are affected, absence of a GP system would result. The solid box around the P system indicates that it is intact.[34]

As our maxim 4 of causal modeling demands, we must distinguish between general and specific deficit. If a deficit is explicable in terms of a general deficit, then we need have no recourse to specific accounts. Poor reading achievement can often be explained as part and parcel of general mental retardation or social disadvantage. We would not want to talk about specific reading problems in such circumstances.

Associated and Secondary Problems

A number of authors have reported that children with severe reading problems have more attentional deficits than would be expected by chance (Taylor, 1986). It remains to be seen whether these children would be classified as dyslexic by virtue of a faulty P component. If they are, then there would be two ways in which the association could come about, each of which would have a particular representation in a causal model. One possibility is that the attentional problem is secondary—that is, it is caused by the effects of being backward in reading on the child's attitude toward himself and toward the process of education (Stanovich, 1986). The other account of the attentional problem involves the SAS component, and the behavior would be classified as associated. Fergusson and Horwood (1992) claimed to have evidence for the latter account and not for the former. An association between reading problems and conduct disorder has also long been known

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[33] There is reason to believe that another component of phonology is derivative on alphabetic skills and only develops in individuals who had a minimum of experience with alphabetic scripts. Thus, Morais, Cary, Alegria, and Bertelson (1979) showed that adult illiterates found it difficult to understand, for instance, what was meant by the request to say "Ted" without the "tuh." Because letters are visible manifestations of the artificial concept phoneme, it is not surprising that so-called phoneme awareness is dependent on knowing letters.

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[34] We note here that there exist other children who are hyperlexic in the presence of potentially severe SAS problems, and, indeed, general intellectual impairments. In these cases, it has been shown that a GP system is fully operative, presumably in the presence of a normal P structure (Cossu & Marshall, 1990; Frith & Snowling, 1983; Seymour & Evans, 1992).
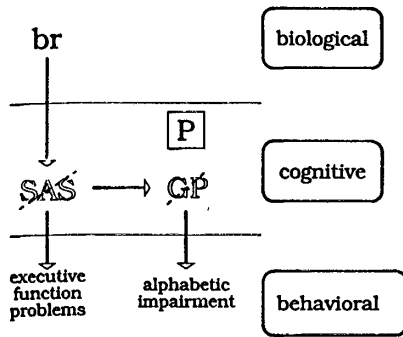
Figure 13.48 A hypothesis relating a deficit in SAS to subsequent dyslexic symptoms in the presence of an intact P system. The setting up of the GP structure is assumed to be dependent on an intact attentional structure. Hence, GP would be absent despite intact P.

(Sturge, 1982; Yule & Rutter, 1985) and might receive a similar explanation.

Competing Theories of Dyslexia

There are, of course, challenges to the particular causal model of dyslexia that we have adopted. Clay (1987), for instance, argued that children learn to become learning-disabled and that a biological basis may be an unsound assumption to start with. Another challenge is that we focused on a phonological deficit when there may be others (e.g., visual deficits) that underlie at least a subtype of dyslexia. Our notation enables us to represent competing theories of dyslexia, and, just as in the case of autism, the potential to compare theories on neutral ground can be demonstrated by some examples.

Figures 13.49 and 13.50 illustrate a specific visual impairment as a cause of dyslexia. Such a cause has been suggested by several researchers, notably Stein (1991) and Lovegrove, Martin, and
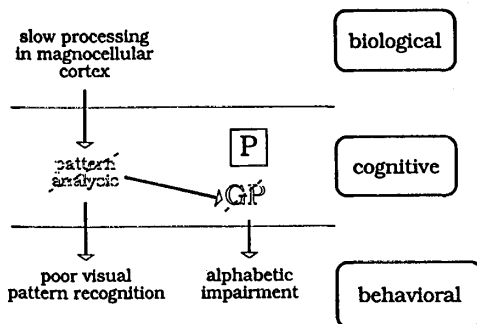
Figure 13.49 A representation of a hypothesis implicating a visual deficit caused by a biological problem which, in turn, leads to a dyslexic syndrome in the presence of an intact P system. GP is absent or diminished because the visual component of letter sound routines (letter analysis) is deficient.
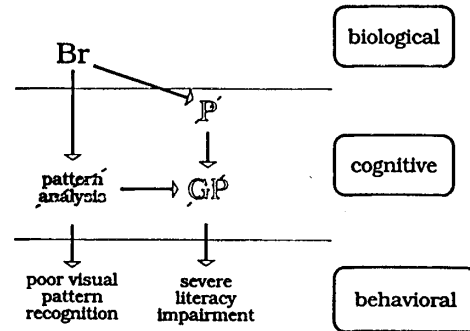

Figure 13.50 A representation of a hypothesis of a biological deficit that affects both visual and phonological systems. This illustrates a case that could be termed "can't read" rather than "won't read."

Slaghuis (1986). We expect that, in some cases at least, a cognitive component will be identified whose absence, quite separately from the P component, will inhibit the development of alphabetic skills. In Figure 13.49, we propose *pattern analysis* as such a component. The biological cause could be as Livingstone et al. (1991) suggested: a defect within the magnocellular system. Cases of dissociation between phonological and visual skills exist (Seymour, 1990) and strongly suggest the possibility of subgroups that can be defined at the cognitive level. Figure 13.50 illustrates the possibility of a more complex picture: a double deficit, at the cognitive level, both in phonological skills and in visual pattern analysis. This double deficit may nevertheless be caused by a single biological fault (Stein, 1991).

Nonbiological Causes

Figures 13.51, 13.52, and 13.53 show examples of externally generated causes of poor reading—lack of language knowledge, poor teaching/learning environment or severe emotional resistance to school, and test performance brought about by negative external influences. Sometimes, these causes directly affect the GP component (Figure 13.51). More often, the SAS component is affected
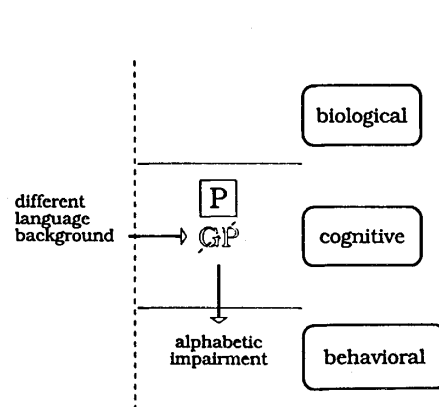
Figure 13.51 Reading problems caused by virtue of a different language background.
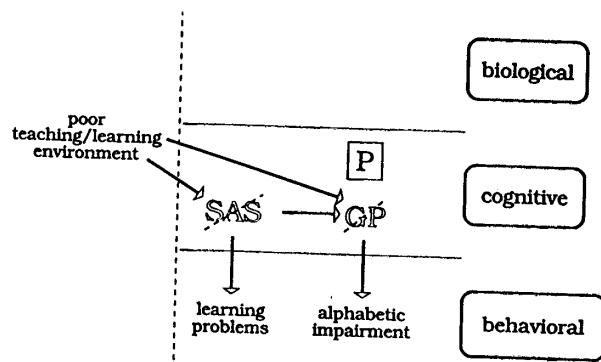
Figure 13.52 Reading problems caused by problems in an attentional component, which is, in turn, caused by external factors (poor teaching). Here, the external influences have impeded the establishment of the GP system. The P system is intact. The deficit in SAS will give rise to additional learning problems (and possibly conduct disorder).

(in the form of attentional problems), as illustrated in Figures 13.52 and 13.53.

A "normal" variant of externally motivated causes that accidentally impair reading acquisition is the effect of a child's being newly confronted by a different language and phonology, or simply not getting instruction because he or she is growing up in a nonliterate culture. In such cases, we would have no reason to suppose that process P is dysfunctional; rather, we could point to incomplete knowledge of the phonology of the particular language that the child is still in the process of acquiring. Figure 13.51 illustrates such a cultural factor.

Figures 13.52 and 13.53 show the effects of a different class of factors—a disturbed home life, a hostile relationship with a teacher, or, simply, misguided teaching (all of which have been claimed to inhibit reading acquisition). These external causes may primarily affect the SAS component; that is, they have potentially
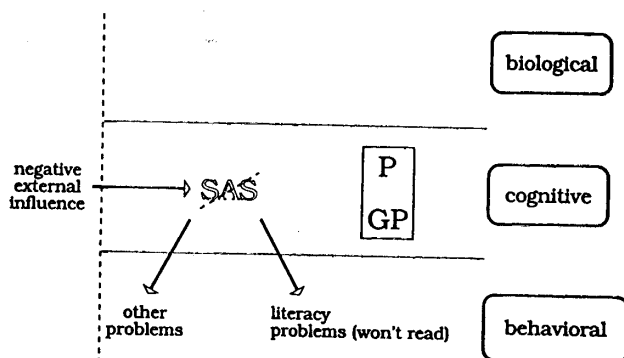
more widespread effects than mere reading difficulties. We have indicated that behavioral disorders would be expected in this group, given the association of conduct disorder and reading difficulty, delinquency, and illiteracy. These externally caused reading problems (part of the large garden variety group) may be mediated either through an effect on the GP system (as in Figure 13.52, where we would expect poor performance specifically in reading and spelling nonwords) or only at the behavioral level (Figure 13.53).[35] Figure 13.53 presents the case of a poor reader who won't read rather than can't read. We are suggesting that the child's not wanting to read can and should be treated as a cognitive link in the causal chain. An external cause that is not mediated by the SAS system and yet has a specific and detrimental effect on setting up a GP system might be an impenetrable orthographic writing system. English, with its complex orthography, is a possible candidate (Wimmer, 1993).

In another version of the causal theories just discussed, the damage done has its effect solely at the behavioral level. For instance, counterproductive reading strategies may have been induced by incompetent or misguided teaching. Such patterns of behavior are established instead of the normal reading strategies, which generally function as self-teaching mechanisms. This is in contrast to the first version illustrated above, where the effect occurs at the level of cognitive structures and the acquisition of a well-functioning GP system is prevented.

Other Biological Causes of Reading Failure

We can imagine a case of reading failure attributable to a cognitive deficit and yet not of biological origin. We would claim that this condition, if it exists, is pseudo-dyslexia. True dyslexia, in contrast, is defined by a cognitive deficit (whether in a visual or in a phonological system) and has a biological origin. The question that we now need to ask is whether all biological causes of reading failure are connected with a specific cognitive deficit and therefore qualify as true dyslexia.

The case of a blind person (who has not learned Braille) illustrates the answer to this question (Figure 13.54). There would undoubtedly be a biological origin that we would want to call the cause of the person's being unable to read. We would not, however, want to link this through the cognitive factor P, in spite of the fact that the blind person (lacking the relevant teaching) has no grapheme-phoneme system. The immediate/local cause of absence of a GP system, then, is the absence of Braille stimuli and a teacher, not the absence of P. It would be more sensible to stick with the remote cause and to show the behavioral problem, lack of alphabetic skills, as being caused (remotely) by the blindness, but not by a specific cognitive problem. A similar case might be made for some types of hearing impairment. However,

Figure 13.53 Reading problems caused via external influences acting on the attentional systems to create reading difficulties in the presence of an intact P system and an intact GP system. This might be termed "won't read," as opposed to "can't read."

[35] The effect of lead in the environment has repeatedly been shown in lower scores on reading tests (Fulton et al., 1987; Silva, Hughes, Williams, & Faed, 1988). We would suggest that this sort of external cause of reading impairment may act in the same way as certain psychosocial causes. Thus, there would be an effect on the behavioral level (alphabetic skills), which would be mediated through the attentional (SAS) component rather than the phonological (P) component.
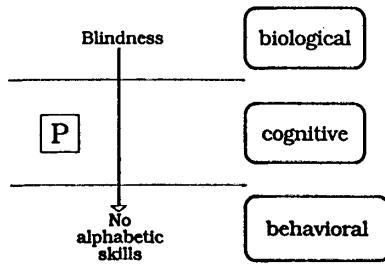
Figure 13.54   A scarcely necessary causal model of the relationship between blindness and inability to read. We do not show the absence of a GP system as being causal in the lack of decoding ability because it is preempted by the visual impairment.

in the absence of a clear theory of the normal development of the P system, we feel such a case would have to be heavily qualified.

CAUSE AND CONTINGENCY: FUTURE DEVELOPMENT

We have introduced a method of transcribing causal theories into a graphical notation. In doing so, we have made the case that, at least for higher cognitive functions, the route from biology to behavior must be through cognition. This is the only substantive theoretical claim we have made in this chapter. The justification for this position is that, without the cognitive level, we cannot account for patterns of breakdown within individuals, nor can we account for the nature of the variability of breakdown in the population. We have used existing work on autism as an example. The essential aspect of this work is the claim that the vast range of symptoms found in autistics, which include disorders of communication, socialization, and imagination, can be accounted for on the basis of a single cognitive deficit. Such a unification requires, of course, theories of cognitive development in the normal population as well as in autistics. The theories of cognitive development enable us to account for the variability in the population. Matters of intelligence, other individual sources of variability (including the extent of brain damage), and environmental considerations will interact in complex ways. This complexity is not manageable if one only looks at the behavior, because the invariant core of autism and most of the interactions between the variables that affect the behavioral manifestations can only be described at the cognitive level.

In addition to our account of autism, we have analyzed developmental dyslexia in the same terms. There are many other candidate pathologies. Laszlo's (1990) approach to the development of motor skills is similar to the one we have used in this chapter. Instead of talking about *cognition* and *behavior,* she referred to the "process-oriented approach" and the "task-oriented approach." She pointed out that, with the task-oriented methods:

> . . . assessment results in a list of tasks the child can perform at a certain age. However, confounding variables, such as motivation,

opportunity and interest, will have a decisive influence on the types of skills the child has practised and can perform well. If, on the other hand, developmental progression in underlying processes, which contribute to learning and performance, can be established—performance-oriented approach—it should be possible to assess which tasks the child is ready to acquire and whether the child is functioning according to his ability level or below it.

> Secondly, for a child presenting motor difficulties, task-oriented assessment can only reconfirm the list of tasks the child cannot perform adequately. The process-oriented diagnosis can establish the reason or reasons why the child cannot master some of the skills expected of him. That is, process dysfunction can be diagnosed and, following causal diagnosis, focal therapy, aiming at improvement of the defective process can be given. (Laszlo, 1990, p. 281)

We do not claim that all invariance is at the cognitive level. There will be many cases where the invariance is at the biological level. An example is Lesch-Nyhan syndrome, where a diverse set of signs—mental retardation, self-injury involving the upper limbs and mouth, and gout—co-occur simply because there is a missing enzyme, hypoxanthine-guanine-phospho-ribosyl-transferase. The converse of this case is represented by the example of reading disability, where a variety of kinds of breakdown at the biological level may give rise to the same functional breakdown. This functional breakdown is properly described at the cognitive level. We reiterate that "cognition" is just the label that we give to the middle level of our model, and that, where affective factors enter into the causal chain, they will generally do so at the same level as cognitive factors. For example, we have shown that affective disturbance in autism, if postulated as the root cause, will lead to a causal model very similar to the one based on the cognitive causes discussed earlier in the chapter.

Much as we champion consideration of cognitive causes of developmental disorders, we would readily agree that interest in a pathological condition does not have to be focused on the cognitive level, nor indeed on the point of convergence, at whatever level this point may be. Much will depend on the type of question that is being asked. For example, questions of prevention will almost always involve consideration of the biological level. On the other hand, the practicalities of management and teaching devolve on the behavioral level. Thus, to someone whose job involves managing severely brain-damaged autistic individuals, the technical details of the relationship between high-ability autism and Asperger's syndrome will be irrelevant. On the other hand, for a variety of activities, including the design and evaluation of new tests or therapeutic methods, the cognitive approach is essential. Irrespective of the focus of the particular work, however, a multilevel causal model will provide a framework that allows the relationship between different approaches to emerge.

The method we have described in this chapter is not tied to any theory, although in our illustrations we have been partisan. Roughly speaking, if you can say it, then it can be represented in the causal notation. One major advantage of the notation is that it is explicit with respect to the causal chain from its biological, social, or other origin, to the signs and symptoms. The pathway itself will be explicit even if some of the intervening links lack detail. Once the causal chain is made explicit, two things follow:

1. What is missing will be more apparent. Thus, in our discussion of dyslexia, we are inevitably drawn to considering the precise nature of the process that, in the normal child, is a required factor in making the phonological system available in the construction of a grapheme-phoneme system.
2. The notation simplifies the task of distinguishing among core behaviors and those that are secondary or merely associated through correlated defects at the biological levels. This is illustrated in a general fashion in Figure 13.55.

Equipped with nothing more than the five maxims of causal modeling, it is possible to make sense of topics that are of current concern for developmental psychopathology. Looking at diagnostic practice in a variety of examples, we have identified three diagnostic types, which we have termed the A-type, the X-type, and the V-type. Each will likely be appropriate in particular circumstances, but it has seemed to us important to establish which type is being discussed at any time. Again, the notation is a powerful way of revealing the underlying structure of a diagnostic concept.

Some Future Prospects of Causal Modeling

The Problem of Variability

Some readers may suspect that the phenotypic causal model makes a deterministic claim. This would be a grave error. The model is neutral in that respect; the facts are overwhelmingly against. Just because the individuals in a group have a particular genetic abnormality, it does not follow that they manifest the same abnormal brain condition. The likelihood of their behaving in the same way is even more remote. The facts of variability are undeniable, and we have to be able to indicate in our notation, at least in principle, what the sources of variation might be. We have already mentioned protective factors and precipitating factors as being two such possibilities, acting at the biological level. Such factors might be seen as variables, with values that are within the normal range of variability; that is, by itself, such a value would be unremarkable—only in combination with something else do we

find abnormal development.[36] We could take as an example a mild phonological difficulty, within the "normal" range, and undetectable preliterately or where the individual belongs to a community with a nonalphabetic script (such as Kannada). When faced with learning to read English, for example, this individual could be revealed to be dyslexic.

If we look at the relationship between the cognitive and the behavioral levels, there are numerous important sources of variability. Frith (1992) analyzed both autism and dyslexia in this respect. She pointed to motivation, compensation, experience, and maturation, each of which could have a positive or negative role. These factors all intervene between stable cognitive structures and behavior.

The Problem of Comorbidity

In clinical practice, it is often very difficult to know whether a particular constellation of features in a few individuals represents a diagnostic entity or a chance combination. Furthermore, we have to rule out the possibility that correlated symptoms represent the secondary consequences of a particular core symptom. The causal modeling approach offers a method and a solution to these difficulties. We propose that the definition of a diagnostic entity could be validated by the existence of a single causal chain.

We have occasionally touched on the problem of explaining overlapping or superimposed disorders, for example, when we considered the association of attention disorder and reading disability. Any rigorous account of comorbidity must avoid many pitfalls. As Caron and Rutter (1991) pointed out, fully representative epidemiological data are vital in order to assess comorbidity. In many cases, this information is not yet available. In this relatively new field of diagnostic refinement, there will certainly be a need for a tool to enable the comparison of alternative accounts.

Changing Patterns of Diagnosis

Often, the most obvious aspects of developmental abnormality, as with disease, are the outward manifestations. It is natural for a clinician, upon noticing a new pattern of signs and symptoms, to classify the individuals together as a group and to refer to the signs and symptoms as a syndrome. This behaviorally defined group would give rise to a V-shaped causal model. For some aspects of management and treatment, this might be sufficient, but, in most cases, there seems to be a process of search for the underlying cognitive or biological condition. This would lead to an X-shaped causal model or, if a single biological cause were identified, to an A-shaped causal model. The way of thinking about autism, for example, has changed, largely to keep pace with the advance of its scientific study. The current debate rests on the precise nature of the underlying cognitive deficit, rather than on behavioral description. It remains to be seen whether a
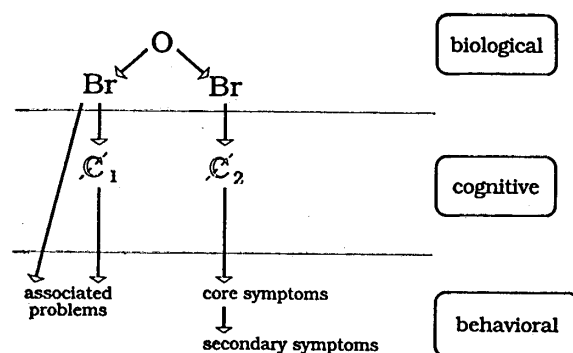
Figure 13.55   A generalized schematic causal model showing how the core signs and symptoms can be distinguished from secondary symptoms and from associated problems.

[36] It is, of course, possible to find two variables, either of which could take particular values without disturbing the developmental trajectory, but which, in combination, could lead to some developmental abnormality. Such conditions would, in any case, strain any attempt to precisely define "abnormal." Because of such limiting conditions, we would not want to abandon analysis of clear cases of pathology.

division of autism into subgroups as a function of their biologi-
cal origin will be useful.

Latent Developmental Disorders

A further point arises in terms of the relationship between be-
havior and cognition. Let us take again the example of autism.
From a behavior-based view, the search for early indicators re-
duces solely to a question of increasing refinement of the detec-
tion instruments. From a cognitive developmental viewpoint,
which assigns the root of autism to the cognitive level, the pur-
suit of earliest signs has natural limitations. It therefore re-
mains entirely possible that there will be no behavioral index of
autism earlier than 18 months (Johnson et al., 1992; Lister-
Brook, 1992). The particular cognitive deficit that we postulate
in autism may have no visible manifestations in behavior for the
first year of life. As a matter of fact, our particular theory
leads us to suspect that autism can only be detected at the
age when certain critical kinds of behavior would normally be
expected to emerge. Delay between root and manifestation ap-
plies to almost all genetically based conditions. The first man-
ifestation may occur as late as adulthood (e.g., Huntington's
chorea and certain kinds of schizophrenia).

Finally, we would like to stress that the notation gives us a
ready method of relating abnormal development, as expressed
with a causal model, with normal development, as expressed with
a contingency model. Only by keeping this relationship firmly in
mind shall we be able to follow the straight as well as the crooked
pathways.

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