Understanding Delay in Developmental Disorders

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ABSTRACT—*Researchers in developmental disorders frequently refer to abilities that are in line with mental age as simply delayed.* The qualifier simply *might imply an existing theory of developmental delay that is well understood and uninteresting (perhaps because it is an exaggerated form of individual differences, the responsibility of other researchers). In this article, I argue that the notion of* delay *can be separated into descriptive and explanatory versions. The descriptive version is often used too coarsely to be helpful. Instead, we need an approach based on developmental trajectories to separate types of descriptive delay, which may then have different underlying causes. The explanatory version is poorly articulated in developmental theory. One useful way to deepen our understanding of delay is by building computational models that simulate development in large populations of individuals and explicitly implementing factors that cause variations in development. Finally, I suggest that dividing research among separate investigators of typical development, individual differences, and developmental disorders may be counterproductive if the underlying mechanisms recognize no such distinction.*

KEYWORDS—*developmental disorders; delay; trajectories; computational modeling; intervention*

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When researchers who investigate the causes of developmental disorders examine an impaired cognitive ability in a given disorder, they frequently ask whether the ability is atypical or simply delayed. Why attach the qualifier *simply* to the notion of delay? The implication is twofold: Researchers already have a theory of developmental delay, and then find it uninteresting. In this article, I express reservations with these implications and argue that the concept of delay is both poorly characterized and lacking explanation at a mechanistic level. The assumption of delayed development—that abilities will catch up eventually—is usually wrong.

In the first part of the article, I argue that characterizing development in terms of trajectories provides a richer vocabulary to describe delay, and I offer examples of these methods. In the second part, I consider explanations of delay and argue that computational modeling allows us to focus on the mechanisms that influence rates of development. I also provide examples from language development and show how these models may provide a basis to inform intervention. I finish with a look ahead at areas for research.

DELAY AS A DESCRIPTIVE TERM

The notion of delay, which originated in a clinical context, identifies when a child fails to show age-appropriate developmental milestones or falls quantitatively below certain thresholds on standardized tests of core domains, such as motor skills, speech and language, personal–social cognition, or daily living (1). Implicit in the quantitative definition is the recognition that in typical children, early trajectories of development vary. Variation below a certain range is deemed problematic. The label *delay* may also serve as a temporary placeholder in the dialog between clinicians and parents, indicating that development is not on track; a specific diagnosis may became apparent when the child is older (1). The clinical notion of delay is frequently criticized for failing to recognize that in many cases, a slower rate of development is also associated with long-term impairment rather than resolution into the normal range (2, 3).

The concern here is when the clinical label of delay is imported into theoretical explanations of developmental deficits.

The term is used often in describing children's behavioral profiles. Delay is identified when a child with a developmental disorder gets scores or displays error patterns on a behavioral measure that are similar to younger typically developing (TD) children. Sometimes, the terminology is associated with a particular form of experimental design. A group of individuals with a given disorder is matched with two separate TD control groups, with one matched on chronological age (CA) and the other matched on mental age (MA). MA is derived from a standardized test deemed relevant to the cognitive ability under consideration (e.g., a vocabulary test for language ability). If children in the disorder group are impaired when compared to children in the CA-matched group but resemble the MA-matched group, individuals with the disorder are considered delayed in this ability. In contrast, if children in the disorder group are impaired when compared to the MA-matched control group, they are said to deviate developmentally or be atypical for this ability (4, 5).

Researchers (6) have pointed to two disadvantages to the matching design: First, age is eliminated from the analysis at the design stage, with variations in age collapsed within the group for both the TD and the disorder groups. Given that development is intrinsically a process concerning change across age, downplaying age in this way seems counterproductive. Second and more seriously, the notion of delay that emerges is a blunt one. In studies of language and visuospatial skills in disorders such as autism, Down syndrome, and Williams syndrome, researchers have argued for using *developmental trajectories* that trace changes in ability over time, and comparing these trajectories between typical and atypical groups. Using trajectories to study developmental disorders originated in growth curve modeling (7–9) and in the wider consideration of the shape of change in development (10, 11).

When trajectories are constructed using even simple linear methods, abilities can be delayed in their onset, in their rate of development, or in both. Plotting trajectories this way also allows researchers to identify other descriptors that can differentiate TD and disorder groups, including earlier or lower ceiling performance, differential relationships between abilities, and the possibility of differently shaped (nonlinear) functions in the disordered group.

Trajectory approaches have limits. Cross-sectional trajectories are frequently constructed in the first instance, but these confound individual differences (and for disorders, differences in severity) with developmental change. Longitudinal studies are preferable, especially for informing causality, although practical barriers to large-scale longitudinal studies are well-known. Researchers (6) have advocated a compromise of initially constructing cross-sectional trajectories, which should then be validated by following up longitudinally with the younger participants. Both cross-sectional and longitudinal methods for investigating disorders should then be complemented by intervention studies, which are the most direct way to test causal models.

Although building functions that link ability to CA can identify different types of descriptive delay, the more theoretically informative trajectories are those that link different abilities, which researchers term *developmental relations* (6). Trajectories can be constructed that link a target ability to a child's MA on standardized tests, or that directly link the development of different abilities. Figure 1 illustrates one heuristic used with this method, comparing data for a TD group and a disordered group with trajectories plotted against CA or MA on a task-relevant standardized measure. If impaired task performance in the disorder group is in line with the standardized measure, then plotting the disorder group's data according to each participant's MA should normalize the atypical trajectory—that is, move it to the top of the TD trajectory, as shown in Figure 1.

To illustrate this approach, in one study (12), toddlers with autism spectrum disorder (ASD) were shown pictures of faces while electrophysiological measures of neural response were collected. The electrophysiological responses were plotted against CA and social scores, revealing delayed relations with CA, but normal relations with social scores. The authors inferred that slower development of the face-processing system in ASD may be related to reduced self-directed expected experience with faces in early development.

The study of developmental relations taps an implicit assumption researchers frequently hold: that the cognitive system develops in integrated blocks or domains (e.g., verbal, nonverbal, spatial). Abilities that develop in harness are considered to be related causally or subject to the same causal factors. The lack of an expected relation between an ability and a given MA measure might indicate the absence of the integrated block in the disorder; the presence of an unexpected MA predictor might indicate atypical blocks or developmental contingencies (13). Therefore, fully understanding delay requires tracking many abilities, not a single ability.

DELAY AS AN EXPLANATORY TERM

Explanatory accounts of developmental delay focus on the mechanisms by which cognitive systems change over time. However, these accounts frequently lack explicit detail at the level of mechanism. Maturational views of delay have been formulated via the analogy to biological growth, with variations in rate of growth reflecting differences in (putative) genetically controlled timing mechanisms (see 14, 15, for proposals with respect to language development). Experience-dependent views of delay are less frequent, but presumably entail either a cognitive system that receives fewer learning experiences or a learning system that is less malleable so more experience is needed to change behavior (16). These views are not mutually exclusive: A cognitive system may receive fewer or less rich experiences because the providers of those experiences think the child's cognitive system is less malleable.
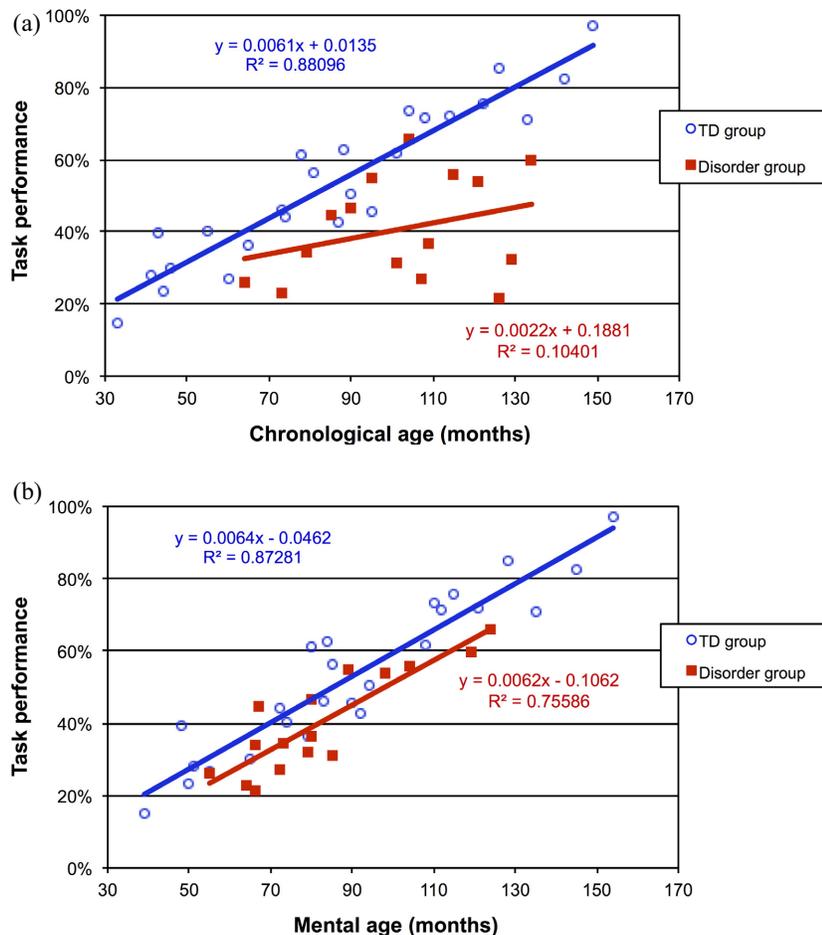
(a)

$y = 0.0061x + 0.0135$
$R^2 = 0.88096$

$y = 0.0022x + 0.1881$
$R^2 = 0.10401$

Task performance

Chronological age (months)

○ TD group
■ Disorder group

(b)

$y = 0.0064x - 0.0462$
$R^2 = 0.87281$

$y = 0.0062x - 0.1062$
$R^2 = 0.75586$

Task performance

Mental age (months)

○ TD group
■ Disorder group

Figure 1. Illustrative data from the trajectories method comparing task performance in typically developing (TD) and disorder groups. (a) Cross-sectional trajectories linking performance to chronological age. (b) Trajectories linking performance to a mental age (MA) on a task-relevant standardized test. If ability A is developing at the same rate as some other (also delayed) ability B, then plotting a trajectory between performance on A and the MA on ability B will normalize the developmental pattern.

COMPUTATIONAL MODELING AS A TOOL TO STUDY MECHANISMS OF DELAY

Computational modeling of development is a useful way to focus on explicit mechanisms. Such models have been used to investigate language and cognitive development, often using artificial neural network architectures (ANNs; 17, 18). ANNs are computational systems based loosely on the principles of neural information processing. As such, they are positioned at a level of description above biological neural networks, but aim to explain behavior on the basis of the same style of computations as the brain. ANNs can learn from data by progressively altering the strengths of the connections in their networks, and therefore can explain the mechanisms underlying behavioral change in cognitive development. Changes in behavior are the result of experience-dependent alterations in the network that result from its interaction with a structured learning environment. A model has intrinsic constraints that affect its learning ability and rate of development, such as the number of artificial neurons, the pattern of connections between units, the network's plasticity, and the way external or environmental inputs are encoded for processing. The richness of environmental information can also be manipulated. However, in contrast to child development, in a model these constraints are known precisely and can be manipulated by the modeler to observe changes to the developmental trajectory and the learning outcome (19, 20).

MODELING DEVELOPMENT IN POPULATIONS OF CHILDREN

Recently, researchers have used the computational approach to simulate developmental processes in large populations of children, and to include intrinsic (neurocomputational) and extrinsic (environmental) factors that interact to produce variability in developmental trajectories (21, 22). Population models simulate a sufficiently large number of individuals to approximate variation in the whole group. Population modeling contrasts

with the approach of building a single model to represent typical development and a single altered model to represent a given disorder.

To understand why population modeling is a necessary step to investigate the notion of delay, consider one reason researchers studying disorders may consider delay to be of less theoretical interest (and so refer to it as simple). The *developmental theoretical perspective* on intellectual disabilities proposes two causes of disability (23). First, disability can arise from underlying organic dysfunction, producing specific deficits in cognitive functioning and qualitatively atypical cognitive development. Second, disability may reflect individuals at the extreme lower end of the distribution of normal functioning who show the same overall qualitative pattern of development as nonimpaired individuals. For this latter group, understanding disordered development would amount to an extension of individual-differences theory, not the responsibility of researchers who study disorder. If we accept this two-group approach, then to bring computational methods to bear on developmental disorders, we need to simulate the normal range of variation in typical development in a population of children (the familiar bell curve) as a backdrop against which to consider the possibility of atypical variation.

POPULATION MODELING AND DELAY

Population modeling of development is illustrated in research (24) that investigated the effects of differences in socioeconomic status (SES) on language acquisition. My colleagues and I created an ANN to simulate children's acquisition of inflectional morphemes, which change a word to fit with its grammatical context (e.g., in English, adding -*ed* to form the past tense). This work sought to capture SES effects observed in a sample of three hundred 6-year-olds (25) via a simulated population of 1,000 children. Intrinsic variability was built into the simulated population through small variations in a number of different neurocomputational parameters within the ANN, such as the number of artificial neurons in the network, the density of connectivity between neurons, and the plasticity of the network in responding to experiences. Extrinsic variation due to SES was implemented as a variation in level of cognitive stimulation, captured by altering the information content of the environment to which the simulated children were exposed. Figure 2 shows the range of trajectories that the model simulated, in this case, for acquiring the past tense of irregular English verbs. Individual trajectories are shown in Figure 2a, the population mean in Figure 2b, and cross-sections in Figure 2c.

The model captured qualitative patterns within the children's behavioral data. It also demonstrated how gene–environment interactions, such as resilience effects, might emerge. For example, in low-SES environments, differences in intrinsic ability were suppressed, while in high-SES environments, differences between individuals were exaggerated (26). Moreover, the model predicted that high SES would predict reliably whether children fell in the top 10% of the population, but low SES would not predict reliably whether children fell in the bottom 10% of the population. This novel prediction was subsequently supported by empirical evidence. In the model, SES was a stronger predictor of strong performance than weak performance because there are few ways to succeed but many ways to fail.

We can now apply this valid model of population development to the question of atypicality versus delay in disorders, as was done in a study of language delay (27). Diagnosing language delay early allows intervention at a time of greater plasticity and minimizes the impact on the child. Delay can be identified at 3–4 years based on vocabulary size and parental reports. However, of children diagnosed with delay at this age, between half and two-thirds fall within the normal range 2 years later and do not need intervention, making intervening early inefficient in terms of cost. How do children for whom delay resolves differ from those for whom it persists? Are those with persistent delay qualitatively different and atypical (15, 28), or do resolving language delay and persisting language delay fall on a continuum of severity and involve variation in the same mechanisms (29, 30)?

In a study (27) that used the population model (24) to identify early signs of delay in the simulated children, researchers followed the trajectories of these networks across development. Just as with the real children, in the model, about two-thirds of the early-delay networks later fell within the normal range, while in a third, the delay persisted. However, the researchers had designed this simulation so that the variation in developmental mechanisms was *continuous* across the population. The networks with persisting delay varied in neurocomputational parameters primarily affecting processing capacity and the quality of the signal inside the networks, such as the number of units in the network or the level of processing noise; those with resolving delay varied in parameters that primarily affected plasticity, such as the extent to which connections changed their strength following experience with language. Capacity and plasticity effects produced similar behavioral profiles early in development. Again, the model generated an empirical prediction: SES should reliably predict the final level of performance in the resolving delay group, but not in the persisting delay group (the environment being the limiting factor in the former, the processing capacity being the limiting factor in the latter). Once more, empirical data supported this novel prediction (27). In short, persisting delay appeared as a behaviorally discrete (atypical) subgroup despite a continuum of variation in the underlying mechanisms across the population.

The population model above considered only a continuum of mechanistic variation in a population of developing children. Other work has addressed the interaction of that continuum with atypical variation, for example, in cases where a subset of individuals in the population have neurocomputational parameters that vary outside the normal range, either due to a genetic muta-
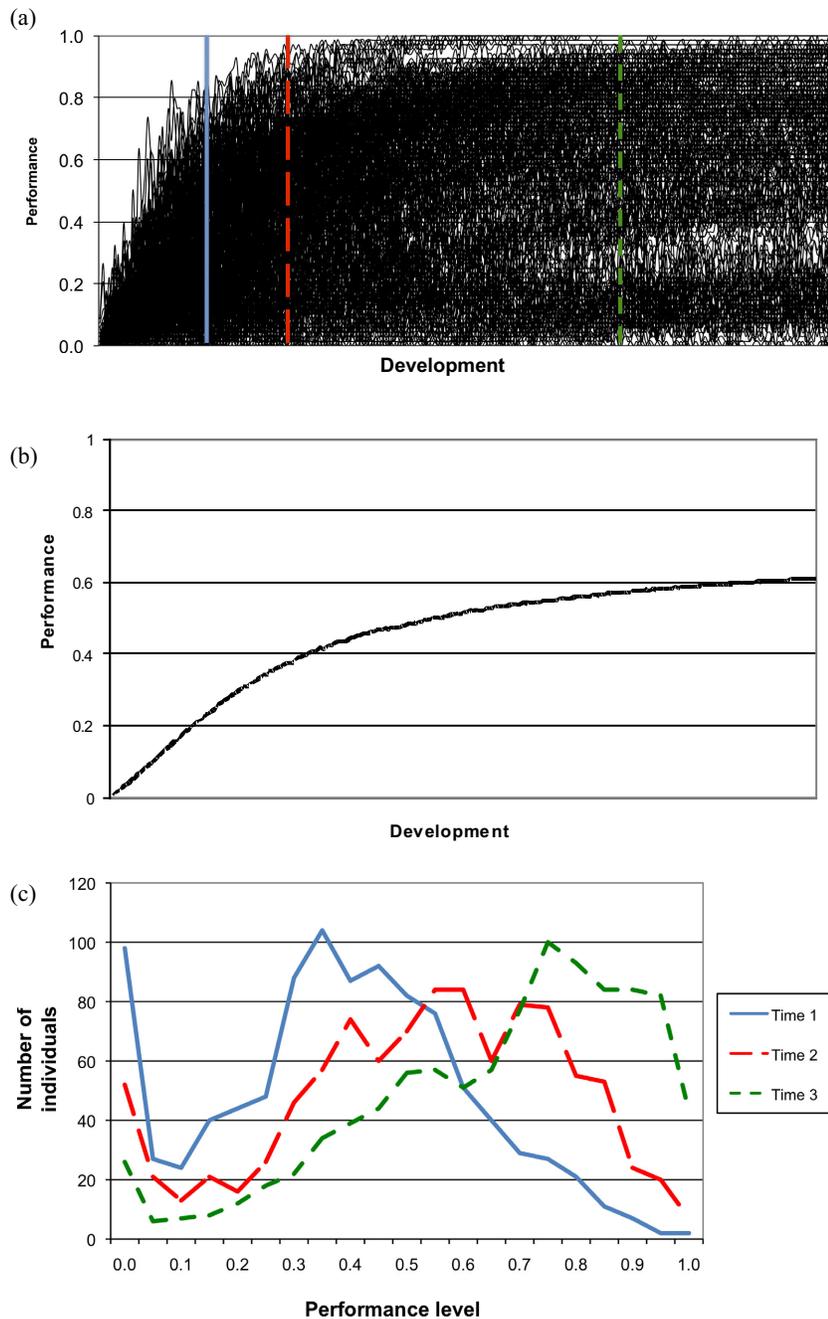
Figure 2. (a) Developmental trajectories simulated by a computational model of child development for a population of 1,000 children, with variations in the quality of learning mechanisms and the environment producing different trajectories. (b) A development view of these data, considering change over time for the average child. (c) An individual-differences view of these data, plotting the frequency distribution of performance levels for cross-sections at different time points.

tion or an accumulation of common genetic variants conveying risk. In one model (31, 32), researchers investigated whether ASD is caused by overpruning of brain connectivity, an exaggeration of an otherwise-normal phase of brain development. These simulations demonstrated that the effects of a pathological mechanism can interact with population-wide individual differences. Three parameters were included that captured individual variation in pruning of network connectivity: its onset, its rate,

and its severity. As with other parameters, all three were allowed to vary by small amounts in the typical population. On their own, each had little impact on developmental trajectories. However, when the severity of pruning was increased to atypical levels, impairing the functioning and further development of the network, the effects of the same individual differences in onset and rate of pruning were exaggerated, leading to identifiably different subtypes of atypical development. Researchers linked

these different trajectories to putative early onset, late onset, and regressive subtypes of ASD. Together, both the model of language delay and the model of ASD (27, 31, 32) suggest that subgroups at the behavioral level are artifacts, generated by interactions between continuous variation in many underlying mechanisms (Figure 3).

FROM DEFICIT TO INTERVENTION

Population modeling provides a framework to develop and test theories about mechanisms underlying delay and atypicality in disorders, specified in terms of influences on developmental trajectories. Constructing models of this type provides a platform to investigate the mechanisms by which behavioral interventions alleviate developmental deficits. This platform allows us to address why children's response to intervention often varies widely. This avenue of computational modeling is relatively unexplored (see 33, for a notable exception). Recently, my colleagues and I demonstrated that computational models of developmental deficits (this time, in the domain of productive vocabulary development) can be created that are tailored to the profile of individual children, which can then be used to predict which type of behavioral intervention each child will best respond to (34). Tests of the predictions can then be used to refine the model, harnessing intervention data to guide mechanistic theories. This research is in its early stages, but it may help us build bridges between a theoretical, mechanistic understanding of variations in developmental outcomes and the practice of clinical, therapeutic interventions to remediate developmental impairments.

LOOKING AHEAD

We can progress further in investigating the underlying causes of developmental delay when we think in terms of developmental trajectories and the factors that modulate such trajectories, both across abilities and across children. We need adequate description of those trajectories using the appropriate statistical methods and more fully articulated mechanistic explanations of the relevant causal factors. Computational models of development can aid in this enterprise.

Advances in understanding will be accelerated by studying individual differences, of a typical or atypical nature, within a developmental framework. This contrasts with a frequent division of labor among researchers into the separate fields of typical development, individual differences, and developmental disorders. However, the underlying mechanisms may not reflect the distinction between these three fields. To highlight this point, Figure 2a illustrates the trajectories that can be generated in a model system where we have a full understanding of the species universal mechanisms of development, as well as the constraints that cause modulations of trajectories across individuals. The development of the average child can be characterized (as shown in Figure 2b) or explanations can be sought for the rank order of performance in cross-sections of the population (as shown in Figure 2c). But both of these represent restricted, partial views of the same system.

The use of population-level models is consistent with the latest methods in genetics research. In that field, variations in cognitive development are viewed as the combination of many small effects, detectable only with large sample sizes. Results
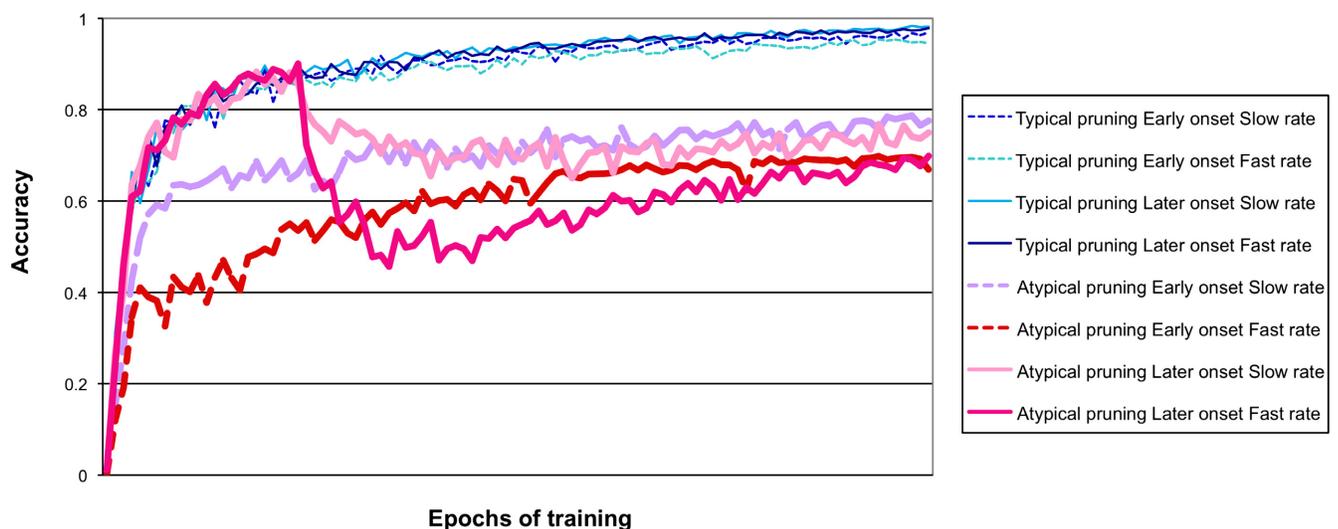

Figure 3. Simulated developmental trajectories from a computational model of autistic spectrum disorder (ASD; 32). The model evaluated the hypothesis that ASD might be caused by overpruning of brain connectivity. This plot demonstrates the feasibility of the idea that apparent subgroups within ASD— early onset, late onset, and regressive subtype—might be caused by the interaction of a single pathological mechanism (overpruning) with preexisting, population-wide individual differences in other neurocomputational parameters (in this case, the onset and rate of pruning). Atypical trajectories (thick lines) show divergent profiles. These individual differences produce otherwise-negligible effects on trajectories in the typically developing group (thin lines). Reproduced with permission from (32).

from behavior genetics encourage the view that, aside from cases of overt genetic mutations, the causes of atypical variation lie on a mechanistic continuum with individual differences in the TD population (35). This is consistent with the computational results considered in this article, where apparently atypical subgroups emerged from continuous variations in developmental mechanisms across a population.

Even if causal explanations are formulated most optimally at a population level, diagnosis and intervention ultimately concern individual children. Given the increasing emergence of datasets at the population level, using results to connect meaningfully to individual children—for instance, to remediate cases of developmental delay—is a challenge for the future.

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