# Plotting the causes of developmental disorders

**Understanding Developmental Disorders: A Causal Modelling Approach** by John Morton, 2004, Blackwell. (300 pp.) £55.00 ISBN 0 631 18757 X (hbk)/ £19.99 ISBN 0 631 18758 8 (pbk)

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What causes autism? Is it a genetic problem? Is it due to a brain abnormality, or is it a result of a viral infection? Could it be a consequence of low intelligence? Or is autism a more specific difficulty, perhaps with developing normal executive functions, or with forming coherent high-level conceptual representations, or with reasoning about mental states? Is it a difficulty with the false-belief task? How might these various 'explanations' be related? In fact, could all of them be right?

John Morton's recent book, *Understanding Developmental Disorders*, presents a diagramming technique to pull apart competing causal models of developmental disorders. It is the crystallization of many years' experience of research in this area. Morton illustrates the technique using several topical disorders, including autism, dyslexia, attention deficit–hyperactivity disorder and conduct disorder. He goes on to demonstrate the implications of such causal theories for issues such as diagnosis and individual variability.

Here's a simplified overview of how Morton's technique works. Take a piece of paper. Split it into three horizontal bands. Label them 'Biology', 'Cognition', and 'Behaviour'. Draw a vertical line down the left-hand side, and to the left of the three bands, write 'Environment'. Now, add boxes with the various facts you know about the disorder, placing them in the appropriate bands. For example, for autism, brain abnormalities would be placed in a box in the Biology band, reasoning about mental states ('mentalizing') would go in the Cognition band, and performance on the false-belief task and on intelligence tests would go in two separate boxes in the Behaviour band. Now draw causal arrows linking these boxes to represent your theory. For example, a given brain abnormality might cause the problem with mentalizing. This in turn might produce poor performance on the false-belief task but not be the cause of poor performance on intelligence tests. Causal relations are allowed between levels of description or between boxes within levels. This causal modelling notation builds on a previous technique proposed by Morton called Developmental Contingency Modelling [1], where one plots out the processes and information necessary for normal development. Each contingency model suggests ways that normal development can break down to produce a disorder, which in turn can be represented with a separate causal model.

The aim of causal modelling is to clarify the relationships between competing theories, and draw out potential implications, predictions and inconsistencies within them. The book is not an attempt to argue for specific theoretical accounts of, say, autism and dyslexia (although, naturally, Morton has his preferences). Instead, it is an attempt to offer a productive tool for theorizing about causal explanation. To facilitate this, the notation offers extensions to deal with interactions and compensation, as well as protective and precipitative factors. However, the notion of cause is a thorny one. Morton's notation places emphasis on certain levels of description as representing causally 'privileged' steps in producing a developmental disorder. For example, he is not particularly concerned with the details of how cognitive mechanisms might work (e.g. the computational operations that a system has to learn for it to perform mentalizing); and he is happy to live with explanatory gaps between his levels of description (e.g. the lack of an explanation for how a set of gene variants actually produces the atypical development of the neurocomputational system involved in mentalizing). Hence, at times it seems sufficient to place any brain anomaly within the Biology band, without the need to establish that a viable causal pathway could link this anomaly with a proposed cognitive deficit.

Moreover, the idea of a 'privileged' step in a causal chain cannot be had for free. By way of illustration, it is sometimes said that '*it is not guns that kill people, it is other people*'. However, if one is seeking to explain a given bullet-related death, what is the justification for identifying the intention to pull the trigger as a causally privileged step over, say, the manufacture of the gun, its acquisition by the owner, the gun's successful operation in this instance, or the passage of the bullet? All were *necessary* steps in producing the outcome. The answer is that people's intentions are given a privileged status in service of a goal: in the above statement, in service of the (debatable) proposal that it is easier to change people's motivations to shoot each other than it is to eliminate all guns or make everyone wear a bulletproof jacket.

In a similar way, Morton's notation privileges certain causal steps with particular (laudable) goals in mind

for improving the study of developmental disorders. His primary goal is to stress that the cognitive level of description must intervene when linking biology to behaviour. The cognitive level is sometimes omitted in naïve attempts to link genes to disorders. His secondary goal is to help theorists avoid a range of pitfalls in constructing their explanations. Here are three: (i) Don't mistake descriptions of behaviour for cognitive mechanisms; (ii) Do distinguish between deficits at the cognitive level that have direct biological causes and those that are the indirect effect of prior cognitive deficits; (iii) Do remember that most cognitive mechanisms and most behaviours have multiple causes, and therefore can fail to develop normally for multiple reasons.

On the whole, Morton's notation is very successful in helping theorists towards these goals. Morton readily admits that final accounts must close the explanatory gaps, but the final accounts are some way off. Every notation has its strengths and weaknesses: for a given notation, there will be some theories that are not easy to represent and some questions that are not easy to ask. What are the weaknesses in Morton's causal modelling notation? Curiously, it has a problem depicting the process of development itself: it collapses the dimension of time into a single atemporal causal diagram, and leaves little scope for partially formed cognitive mechanisms, precursor states, atypical versions of cognitive mechanisms or atypical behaviours. Such issues are effectively de-emphasized by the difficulty of capturing them in the formalism. For example, Morton suggests that disorders that exhibit different atypical states at different ages could be depicted using a different causal model to capture each stage. This sidesteps the question of the mechanisms by which the transition between stages takes place. It may be that other formalisms, such as computational modelling, are more appropriate to investigate the idea of atypical developmental change (for example, see [2]). Meanwhile, Morton's lucid and highly readable book offers an excellent tool to clarify the field of developmental disorders as it stands and to point the way to the future.

**Acknowledgement**

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**References**

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2 Thomas, M.S.C. and Karmiloff-Smith, A. (2002) Modelling typical and atypical cognitive development. In (Goswami, U., ed.), *Handbook of Childhood Development*, pp. 575–599, Blackwell

## Author's postscript: How could all the explanations in the introductory paragraph be right?

"Is it a genetic problem? Is it due to a brain abnormality, or is it a result of a viral infection? Could it be a consequence of low intelligence? Or is autism a more specific difficulty, perhaps with developing normal executive functions, or with forming coherent high-level conceptual representations, or with reasoning about mental states? Is it a difficulty with the false-belief task? How might these various 'explanations' be related? In fact, could all of them be right?"

Well, consider the following hypothetical scenario: the individual could have a genetic vulnerability to a viral infection. The viral infection then causes an anomaly in brain development, causing a structural abnormality [all at Morton's *biological* level]. The result is a set of atypical neurocomputational constraints changing aspects of the development of perception and of 'social' circuits, and impacting on the development of frontal circuits. The latter might lead at first to poor development of executive functions and problems with forming high-level conceptual representations. When combined with the anomalous social circuits, the frontal problems could also then lead to difficulties in reasoning about mental states [all at Morton's *cognitive* level]. One consequence of the problems forming high-level conceptual representations would be low performance on (abstract) tests of general intelligence. One consequence of problems with reasoning about mental states would be difficulty with the false belief task [both at Morton's *behavioral* level].

The point, here, is not that this scenario is necessarily the correct one (in fact, there is no current evidence implicating viral infection as a cause of autism). Rather, the example is intended to demonstrate that, theoretically, a whole set of apparently competing causes could turn out to be mutually *inclusive* when they are placed at their appropriate levels of description and in the correct temporal order.