On why simulated developmental disorders don't predict real ones

Laurent Mottron[1] and Jacob A. Burack[2]

1. *Clinique spécialisée de l'autisme and University of Montreal, Canada*
2. *McGill University, Canada*

Mathematical deductive models cannot replace inductive descriptions of developmental disorders. The utility of the application of artificial neural network models (ANNM) to developmental disorders is too limited to substantiate the revolutionary ambitions of the authors. ANNM are simply mathematical metaphors for learning and threshold effects in pattern recognition. They are used as an analogy (mathematics, regardless of its internal consistency, can be used as an analogy) and are only related by the tentative notion of biological plausibility to the domains of reality that they pretend to model. For example, the claim that the manipulation of the gain parameter in ANNM for schizophrenia is *directly* motivated by a specific neurochemical deficit (Cohen & Servan-Shreiber, 1992) appears exaggerated since the same manipulation of the network is made for all the cognitive systems and the pathologies modelled by ANNM.

As models, ANNM do not provide a close representation of reality but create a new reality in a deductive mode. ANNM simulations do not yet apply to any particular clinical entities. They predict *possible* disorders, but are unable to predict why only *real* ones, and not other possible ones, exist. An optimistic account of these limitations is that these models are still in their infancy. However, our position is that the limitations are

Address for correspondence: Laurent Mottron, Département de Psychiatrie de l'Université de Montréal, 7070 boulevard Perras, Montréal, Québec, Canada, H1E1A4; e-mail: mottronl@istar.ca

intrinsic, as mathematical *simulation* models cannot *explain* behavioural levels. More generally, causality involving only one level (e.g. biochemical or genetic) cannot account for the results of interactions among multiple and heterogeneous levels of causality.

At a general level, the case for ANNM is based on the false assumption that developmental cognitive neuropsychology (DCNP) can be *replaced* by ANNM. However, disciplines at different levels of consistency cannot replace one another: mathematics does not replace physics, which cannot replace biology etc. Furthermore, Marxism cannot replace history, and Marxism, but not history, may be rejected! In this case, ANNM are simply consistent and homogeneous mathematical programmes, and therefore cannot replace the more comprehensive and diversified DCNP in which diverse domains such as brain anatomy, behavioural description and genetics are integrated.

This multicomponent DCNP is criticized by Oliver *et al.* for the use of discrete and 'static' terminology, such as syndromes, specific cognitive functions and functional brain specialization. Nevertheless, discrete concepts used in DCNP are necessary to the vocabulary of description of cognitive functions and developmental syndromes, even if they must be constantly modified. For example, the static use of clinical entities (such as the set of DSM-IV criteria for autism, regardless of age of diagnosis) needs to be questioned, but within the context of typical and recognizable clinical pictures.

The emphasis on continuity, as opposed to discreteness, diminishes the contribution of ANNM to an understanding of the patterns of characteristics that are evident among specific atypical populations. One, the notion of continuity is contradicted by the obvious similarities in behavioural profiles, and why autism, for example, looks the same in any part of the world, regardless of problems in defining the limits of the syndrome. Two, unlike DCNP, ANNM are unable to produce a taxonomy of descriptors to account for discrete objects. ANNM are learning algorithms that can account for the stages of learning in general but are unable to label the evolution of the model in specific cases. For example, a mathematical calculator can be used to add 2 and 3 to make 5, but cannot inform about 5 of 'what'. With these limitations, ANNM (or any other type of mathematical model) cannot replace observations and labelling of phenomenal regularities among patients and experimental assessments of cognitive differences in the study of clinical symptoms.

This lack of specificity of the ANNM infers the untenable position that all disorders can be treated similarly with respect to the level of temporal or taxonomic boundaries. As there are both discrete clinical entities (e.g. depression) and disorders or deficits that permeate virtually all aspects of the individual's life (e.g. mental retardation), there is a need to differentiate among these types of disorders. This difference in level of delineation is intrinsic to even the most 'static' diagnostic tools. For example, in the DSM-IV (American Psychiatric Association, 1994), the discrete clinical entities of Axis 1 are in contrast to the permanent and non-delineated aspects of Axis 2. Similarly, in DCNP, both non-modular (e.g. planning) and modular (e.g. pitch processing) functions are studied. This is consistent with the study of functional brain anatomy in which there is a gradient of discreteness from very localized functions (e.g. primary visual processing) to poorly localized ones (e.g. visual semantic processing).

Oliver *et al.*'s arguments against the existence, importance and genetic predetermination of functional specialization of brain areas are mitigated by empirical evidence of species-typical architectures at certain developmental stages. The functional gain of brain specialization cannot be used as a general argument against either the importance of topographic organization or the existence of syndromes that arise from lesions/impairments in specific regions. The theoretical emphasis on diffuse lesions (as simulated by ANNM) is not a strong argument against the reality of focused brain lesions which may, in some cases, mimic autism to the extent that it is indistinguishable from autism *sine materia* (Mottron *et al.*, 1997). Brain plasticity may be modelled by dynamic, continuous models at the microscopic level, but at a higher level of organization it requires compensation among modules. For example, early visual impairment does not yield a general compensation but a specific one of regions that are devoted to auditory processing.

In sum, ANNM reflect a thoughtful and innovative framework for thinking about developmental disorders, but do not challenge the utility of DCNP. ANNM's unique focus on developmental transformations and continuity is not relevant to discussions of their accuracy, just as the unique focus of any theory on a specific phenomenon does not ensure its veracity or continued prominence. The proclamation of ANNM as superior to DCNP for understanding developmental transformations and brain plasticity in developmental disorders is not substantiated. Despite the authors' claims, ANNM are clearly neither the unique nor mandatory frameworks for integrating development and psychopathology. Rather, the integrated study of shared and specific deficits of modular and non-modular functions on the one hand, and developmental maturation of these modules on the other, provides comprehensive pictures of the similarities and differences

among developmental cognitive disorders (Pennington & Ozonoff, 1996).

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