

DISCUSSION

DOUBLE DISSOCIATIONS IN DEVELOPMENTAL DISORDERS? THEORETICALLY MISCONCEIVED, EMPIRICALLY DUBIOUS

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Single dissociations are the bread and butter of neuropsychologists, and double dissociations their chocolate cake! So it is unsurprising that developmental psychologists working on genetic disorders find the concept of double dissociations an attractive one. The following quotations bear eloquent witness to this:

“... the study of mental retardation would profit from the application of the framework of cognitive neuropsychology” ... In cognitive neuropsychology, one key question running through the investigator’s mind is «Is this process or mechanism intact or impaired in this person?”. (Baron-Cohen, 1998, p. 335)

“...overall, the genetic double dissociation is striking ... The genes of one group of children [SLI] impair their grammar while sparing their intelligence; the genes of another group of children [WS] impair their intelligence while sparing their grammar” (Pinker, 1999, p. 262).

We argue that the use of the double dissociation method in *developmental* disorders is not only inappropriate theoretically, but also erroneous empirically, often based on a dubious choice of control groups. It rests on a false assumption: that the brain of an infant with a genetic disorder comprises a pattern of neatly segregated, “intact/impaired cognitive modules”. In doing so, it neglects one vital factor: the actual process of ontogenetic development (Karmiloff-Smith, 1998). The effects of a genetic mutation during embryogenesis and postnatal brain growth are likely to be widespread across the developing system. Some domains may be more affected than others due to the features of their particular problem space, but in-depth studies reveal subtle impairments in domains that originally seemed “intact” (Karmiloff-Smith, 1998; Karmiloff-Smith et al., 2002). As Dunn and Kirsner (2003, this issue) highlight, the notion of “intact” performance depends on the sensitivity of the measurement scale. In sum, an uneven pattern in the cognitive profile of a genetic disorder cannot be taken for granted to imply impaired versus totally preserved abilities. All domains may be impaired, some more subtly than others. The case is different for adult neuropsychology patients. In their previously normal brains, specialisation and localisation of function had already stabilised, so selective impairments might emerge if pure cases exist.

But what about the double dissociation claimed above for Specific Language Impairment (SLI) and Williams syndrome (WS)? Empirical data show that *absolute* statements about “sparing” of intelligence or grammar should actually

be *relative* statements. True, people with WS are better at language than at non-verbal tasks, but their language is far from «intact». Many studies reveal that WS language follows an atypical developmental trajectory (Laing et al., 2002; Nazzi et al., in press; Singer Harris et al., 1997). The same applies to face processing. Although individuals with WS score in the normal range on some face processing tasks, the cognitive processes by which they achieve their superficially «normal» behaviour are different from those of normal controls (Deruelle et al., 1999; Karmiloff-Smith, 1998). This is not only true at the cognitive level; it holds equally for the brain processes sustaining the behaviour (Grice et al., 2001). Similar arguments can be made with respect to SLI where intelligence is claimed to be «spared». Many studies now point to subtle impairments in the intelligence of children with SLI that cannot be explained away by the linguistic component of tasks (Chiat, 2001). So, WS and SLI fail to present a neat double dissociation.

Furthermore, claims about genetic disorders tend to take for granted that the disorder manifests a similar pattern in infancy as in adulthood. Yet our studies of toddlers with WS and Down syndrome reveal that the early profile can be very different from the adult one (Paterson et al., 1999). For example, toddlers with WS and DS both show equal language delay, despite the WS adult language being significantly better than the DS adult's. Moreover, toddlers with WS resemble normal CA- and MA-matched controls in their sensitivity to changes in number, whereas toddlers with DS are worse than MA-matched controls. However, by adulthood, those with DS perform significantly better than people with WS on a battery of number-relevant tasks (Paterson et al., 2002). Therefore, a difference in performance across clinical populations at any point in development does not permit the inference of a stable double dissociation at a later or earlier time. Such claims must be supported by empirical evidence drawn from the developmental trajectories themselves. In sum, the adult phenotypic outcome is affected not only by genomic variation, but also crucially by the process of ontogenetic development and the regulation of gene expression over time.

Another caveat concerning claims of developmental dissociations lies in conclusions drawn from comparisons of typically and atypically developing groups or across different clinical populations. A control group matched on, say, Mental Age can mean that the participants with the genetic disorder are many years older than the control group. However, when the clinical population shows equivalent behaviour to the control group in domain A but not in B, claims of single dissociations abound. A more accurate account of such results is that both domains are very delayed (with B more than A), not that A is normal and B selectively impaired! When exploring an atypically developing *system*, we need to understand the difference between simple delay and complex deviance. To dismiss delayed performance as irrelevant carries the assumption that the representational processes under investigation do not interact with others throughout developmental time. Without knowledge of the developmental trajectory in each case, we cannot establish the true sources of *both* differences and similarities between developmental disorders.

So, does the adult neuropsychological concept of double dissociations have any use for the study of developmental disorders? At best, it is useful in our

view for generating testable hypotheses. However, without a developmental account of the underlying mechanisms, it is impossible to account for performance differences within and between developmental disorders in terms of selectively spared or impaired modules. In conclusion, the claims that emerge from borrowing the double dissociation method for the study of developmental disorders often turn out to be empirically unfounded and, in any case, for ontogenetic development the enterprise is theoretically misguided.

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