COMMENTARY ON "MOTION PERCEPTION IN AUTISM
(E. MILE, J. SWETTENHAM, & R. CAMPBELL)

Cross-syndrome, cross-domain comparisons of development trajectories

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Introduction

In their interesting review of motion perception and the autistic spectrum disorder (ASD), Milne, Swettenham and Campbell (henceforth MSC) focus on the details of the visual system and on studies of static snapshots of children and adults with high-functioning ASD, whom they compare to other individuals with non-autistic disorders and low intelligence. In this commentary, we highlight the need for tracing cross-syndrome and cross-domain comparisons of full developmental trajectories. In our view, it is only in this way that the important question of domain-specific versus domain-general development can be properly addressed.

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Is the deficit domain specific?

The focus on a specific domain, such as visual perception, as MSC's article does, limits our ability to understand whether a deficit is domain specific or domain general. In the case of the magnocellular and parvocellular processing systems, it is becoming increasingly clear that *both* visual and auditory perception call on these streams (Beer & Roder, 2004). Indeed, the overall map of cortical areas involved in auditory processing seems to be organised in a similar way to the visual system, with a dorsal stream for sound location and a ventral stream for sound identification (Poremba et al., 2003). Rama and collaborators (2004) using fMRI have also pinpointed the separation of dorsal and ventral auditory processing streams during the recognition of human voices versus their location in space. Moreover, Beer and Roder (2004) have shown that attention to motion enhances processing of *both* visual and auditory stimuli. If this is the case, and if one wants to argue that the deficit in autism is rooted in the magnocellular/dorsal stream, then one prediction should be that deficits should occur not only in visual perception but in auditory perception in autism. If it turns out that auditory perception is not impaired, then the explanation of visual motion deficits becomes more complex than simply implicating the magnocellular processing stream.

Is the deficit syndrome specific?

MSC report that difficulties in visual motion perception have been found not only in autism but also in individuals with FragileX, Williams syndrome and mental retardation in general. Thus, problems with motion perception may not be syndrome specific at all, but related more generally to mental retardation and to other deficits found early on in developmental disorders such as processing low or high spatial frequencies (e.g. Deruelle et al., 2004), poor saccadic eye movement planning (Brown et al., 2003), attention/inhibition problems (Scerif, Cornish, Wilding, Driver, & Karmiloff-Smith, 2004) or impairments in forming global percepts (Farran, 2005). Moreover, the magnocellular system is thought to reach full maturation later than the parvocellular system, and it is known that later-developing systems are more vulnerable than earlier ones to developmental impairment (Mitchell & Neville, 2004). Thus, one would actually expect most disorders to yield greater magnocellular than parvocellular impairment. All of these points highlight the need to study developmental disorders at their earliest starting point rather than in middle childhood or adulthood.

The importance of tracing developmental trajectories

Much of the thrust of MSC's article stems from the adult neuropsychological perspective. For instance, while it is true that one can argue for double dissociations in motion impaired adult patients of the perception of first- versus second- order motion (Vaina, 1998; Vaina & Cowey, 1996), this segregation in *adults* does not entail the automatic assumption that first- and second-order perception is segregated at the start of either normal or atypical development (Karmiloff-Smith 1997, 1998). Moreover, when it comes to developmental studies, the double dissociation methodology is in our view both theoretically and empirically questionable (Karmiloff-Smith, Scerif & Ansari, 2003; Annaz, Thomas, Karmiloff-Smith, & Johnson, in prep.). In fact, some studies suggest that both magnocellular and parvocellular pathways contribute early on to all processing, with their segregation only happening gradually as development proceeds (Parrish, et al., 2005). Double dissociations are very unlikely in early development because, as the work of Rakic (1988) and Mitchell & Neville (2002) has clearly shown, the infant cortex starts out with its regions highly interconnected and it is only with progressive development that regions become increasingly specialised and localised (see, also, Johnson, 2004) or what we have termed “progressively modularised” (Karmiloff-Smith, 1992). In the case of developmental disorders of genetic origin, the brain may remain more interconnected with less pruning and specialisation over time than is the normal case, making pure dissociations very unlikely.

Although scientists still do not know which genes are causal in autism, twin studies make it clear that there is a genetic contribution to the disorder. Recall, however, that specific genes are rarely if ever expressed in a single brain area, and therefore genetic mutations are likely to be widespread across the heavily interconnected atypical brain, even if the phenotypic effects of these mutations are subtler in some areas than others. Even a very tiny abnormality early on can have cascading but differential effects on subsequent development, making the outcome *seem* domain-specific although it may have originated in a domain-general impairment (Karmiloff-Smith, 1997, 1998; Karmiloff-Smith, Thomas, Annaz et al., 2004). Hence the importance of tracing full developmental trajectories. All these ontogenetic factors have to be taken into account when considering any domain of typical or atypical development.

Concluding thoughts

In our view, notions such as "spared"/"preserved", which stem from the adult neuropsychological literature, hinder rather than help the study of the dynamics of atypical development. Indeed, when a brain has developed normally and results in specialised, localised functions then, if there is brain damage, yet one of those functions continues to operate normally in the adult patient, one can deem it to be "spared". But development is very different. "Spared" implies that a function has *developed* totally normally from infancy through childhood to adulthood. However, given the interconnectivity of the infant brain, this is unlikely to be the case in developmental disorders, even when individuals display good behavioural scores (Karmiloff-Smith, 1998; Karmiloff-Smith, Thomas, Annaz et al., 2004). It is indeed crucial to differentiate between “normal” scores at the behavioural level from the cognitive and brain processes underlying them.

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