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THE ORIGINS OF AUTISM: A COMMENTARY ON MURATORI & MAESTRO'S "AUTISM AS A DOWNSTREAM EFFECT OF PRIMARY DIFFICULTIES IN INTERSUBJECTIVITY INTERACTING WITH ABNORMAL DEVELOPMENT OF BRAIN CONNECTIVITY"

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ABSTRACT. Muratori & Maestro (2007, this issue) provide a fascinating investigation into the possible origins of autism and how it may begin to show itself during the first year of life. However, one could hope for greater clarity on how the variety of hypotheses that they consider relate to each other and to the origins and development both of autism and of the dialogical self. In this commentary I support a neurological hypothesis on the origin for autism and provide an information processing account of the development of autistic difficulties in social interactions and in forming a conception of the dialogical self.

Muratori & Maestro (2007, this issue) investigate possible origins of autism by studying signs of this disorder that begin to emerge during the first year of life. Because there is as yet no way to assess autism at birth, it takes time to determine whether an infant or toddler has autism, and it is only in the third or fourth year that autism can be clearly identified. However, through retrospective analysis of videos taken of infants who eventually get assessed as exhibiting autism, Muratori and Maestro, and others, have been able to discover possible early signs of autism even during the first year of infancy. And there are now some prospective studies of infants, who are siblings to autistic individuals and later diagnosed with autism (Bryson, et al., 2007; Zwaigenbaum, et al., 2005) that provide some complementary support for findings from these retrospective studies.

The question arising from these studies that is the focus of this article is what is the original cause of autism. A variety of fascinating hypotheses are considered; however, it isn't always clear how these hypotheses relate to each other and to the origins and development both of autism and of the dialogical self. In particular, the psychological and neurological hypotheses that they cover appear to be at odds with each other. On the one hand, they hypothesize that neurological events that happen during the latter part of the first year are downstream effects of earlier deficits in primary intersubjectivity that are the original cause of autism, while on the other hand

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they sometimes hypothesize that a neurological disorder is the original cause of autism. Because the arguments presented in favour of a neurological origin to autism are more compelling to me than those for a psychological origin, in what follows I will present a unified neurological account and point out how the social psychological hypotheses that are presented fail to deal with issues satisfied by a neurological account.

According to the main neurological hypothesis that is presented there is a neurological basis to autism that makes its first serious appearance in the latter part of the first year when there is a great neural expansion in the frontal regions of the brain that tend to be involved in social functions. Through some genetic or epigenetic neural disorder, possibly associated with the formation of glial cells, the development of "long-distant" cells is inhibited relative to the development of "short-distant" cells in the prefrontal cortex, cerebellum and other cortical areas. Some of the correlates that may emerge during the second half of the first year due to this neural dysfunctional are problems in attention, particularly in joint attention with others, which is important for developing secondary intersubjectivity.

Although not directly considered as such in the present article, this problem could also account for the neural deficiency in the mirror neuron system (MNS) as well as the "default state"[1] system that is found in older children with autism, both of which have implications for social information processing. The MNS, by which one interprets the actions of others through matching one's own action plans with the perception of action in others, requires closely timed integration of information from distant regions of the brain in order to be operative, and is unlikely to be innately tuned to deal with these diverse sources of information, so they are likely to emerge in development with the expansion of the long-distant neural system. This neural disorder could also cause abnormalities in the default state system found in older autistic individuals (Kennedy, Redcay, & Courchesne, 2004).

There are two important issues that arise with respect to this neurological hypothesis, both of which conflict with the social psychological hypothesis that Muratori and Maestro propose. The first is whether autism is fundamentally a problem in the social domain or whether it is a general information-processing problem that affects most importantly the social domain. With respect to this issue the evidence so far suggests that both social and non-social problems are likely to be associated with these unusual neurological developments. Zwaigenbaum, et al. (2005), have found that between the 6th and 12th month, siblings of autistic children who will eventually be

[1] The term "default state" is used to indicate the activation state of brains of participants in imaging studies during baseline or "resting" conditions, when no stimulus is presented. The brain is surprisingly active during this state, particularly in the medial frontal region. This default activation state has been hypothesized to indicate reflective self-consciousness as well as social consciousness and it seems likely that this system is affected when neural development becomes disordered in autism.

diagnosed with autism show, relative to other siblings without autism, an increasing inability to "disengage" attention from one stimulus, when a new interesting stimulus appears. This attention disengagement problem seems likely to be one of the consequences of lack of long-distant neural connections and the density of short-distant connections in the frontal region (Courchesne & Pierce, 2005). This inability to shift attention would naturally have an enormous impact on the development of shared attention, because of the necessity to shift attention between another person and objects of attention in triadic interactions, which are the basis of secondary intersubjectivity. However, it would produce other consequences that are also autistic but are not particularly social, for instance, the tendency to focus on one and only one activity to the exclusion of others and the tendency to engage in repetitive actions. So it seems possible on this neurological hypothesis that disturbances in social interactions associated with autism may represent only the major outcome of problems of a more general information-processing sort – in particular, of integrating information from multiple modalities or information sources. Both integrating these sources into complex unities and shifting attention with respect to dynamic social phenomena involving multiple sources of information would then lead to special problems in the social domain.

There is also a second consequence of this neurological hypothesis that also opposes the psychological hypothesis. It is that the infant who will eventually acquire autism need not have any problem in the earlier phases of development, during that period of primary intersubjectivity prior to the latter half of the first year. Indeed, it is not usually until the latter part of the first year that retrospective analyses of videos of infants begin to show clear signs predictive of autism (e.g., Palomo, Belinchòn & Ozonoff, 2006), a result that has been confirmed in a projective study (Bryson, et al., 2007). Yet, on the other hand, and on the basis of their psychological hypothesis, Muratori and Maestro suggest that there is a problem in primary intersubjectivity and the motivation for social interaction during the first-half year in these infants, which is evidenced by differences in social attention in videos taken before 6 months in their own studies (Maestro, et al., 2005; Maestro et al., 2006). Indeed, they suggest that the neurological consequences that occur during the latter part of the first year may be due to these earlier disturbances in primary intersubjectivity.

The hypothesis that these neurological symptoms are the consequence of disturbances in primary intersubjectivity and social motivation early in the first year strikes me as implausible. For instance, it doesn't seem likely that early psychological phenomena of this sort could account to the great expansion in head size during the latter part of the first year found in a large proportion of infants later diagnosed as autistic, or for the disturbances in development of glial cells during that period (Courchesne & Pierce, 2005). More likely is the possibility that an incipient phase of this neurological disorder already displays itself in early differences in social attention

found in videos of infants later diagnosed as having autism, and especially in cases of early onset autism, as opposed to what the authors call regressive autism in Maestro, et al. (2006).

Let's assume, then, that autism has a neurological basis that may exhibit itself during the first year in some cases by affecting primary intersubjectivity in the form of reduced social attention during the first half of the first year, and in many more cases affecting secondary intersubjectivity, through the complex information processing involved in joint attention and other triadic interactions that usually develop during the latter part of the first year. How might these disturbances in intersubjectivity relate to disturbances in the origins and development of the dialogical self?

Near the beginning of their article, when arguing in favour of their psychological hypothesis on the origin of autism, Muratori and Maestro (this issue) assert that "a very early lack of the desire to share experiences, activities, and feelings with other persons" (p. 97) is at the core of the autistic disorder and that problems that arise in dialogical competencies are downstream effects of this original problem. However, toward the end of their article, and more in agreement with the neurological hypothesis, they suggest that it is a "genetically based dysfunction" in "contingency detection" (p. 108) that impairs dialogical activities with others. They go on to discuss the hypothesis that neurological deficits in the MNS and the prefrontal default system in autism may affect the representational system applicable to self and other and that this may "represent a core deficit in the development of dialogical self and in the achievement of primary and secondary intersubjectivity" (p. 109). In partial agreement with the later neurological account, rather than with the earlier motivational account, I believe that problems in contingency matching and dialogical competencies are due to the neurological disorder associated with glial activity and the development of long-distance neural connections and that it is this disorder, which produces deficits in both the MNS and the default system along with contingency matching.

In order to support this hypothesis, I would like briefly to describe a psychological hypothesis that Chris Moore and I proposed some time ago (Barresi & Moore, 1996), and which we have recently described in neurological terms (Barresi & Moore, in press). We hypothesized that in typically developing infants, dyadic and triadic interactions with others provide contexts in which infants share intentional activities with others and become able to match and integrate the first-person information that they have about their own activities with third-person information about these same activities in others. For instance, in shared attention, the infant will match the first-person information of their own attention to an object with the third-person information that they have of the attention of the other individual. The net result is a representation of shared attention that integrates both how attention of a person to an object appears from a first-person point of view and how it appears from a third-person point of view. A later outcome of sharing such experiences with others is the

development of a capacity to imagine the first-person perspective of another person in an intentional activity when not currently also engaged in that activity. It also results in a capacity to become reflectively aware in imagination of one's own activity from a third-person point of view. It is this later capacity, in particular, that is most relevant to the dialogical self and which is disturbed in autism.

It was our suggestion that because of the information processing difficulties associated with autism involving multi-modal information that they fail to form integrated representations of intentional activities of self and other that include both first- and third-person aspects of those activities. Instead, they develop separate "first-person" and "third-person" representations, which they apply to self and other. More recently, we have related these ideas to disturbances in the MNS and prefrontal areas associated with the default system (Barresi & Moore, in press). The basic idea is that autistic individuals develop a behavior-based third-person model of people that is formed by observing others, but not penetrating to their inner states. On the other hand, they also have an internally based first-person model of people that is peculiarly egocentric and does not take into account how other people might have a different internal perspective. The net result is that they fail to form uniform representations applicable to self and other with both internal and external aspects of intentional activities.

I believe that it is here where the failure to form a dialogical self occurs, and it has its origins, as Muratori and Maestro suggest, in the failure to engage successfully in dyadic and triadic intersubjective activities during the first year of life. However, as I see it, the problem is not originally one of motivation, but one of information processing. The inability to enjoy social interaction is the result of the inability to process contingent matching between self and other and to interpret dialogical encounters with others. Furthermore, since it is through these dialogical encounters that we typically gain an understanding of both self and other, the decrease in capacity of autistic individuals to make sense of these encounters in terms of a uniform model of self and other, eventually leads to a failure in the development of a conception of the dialogical self.

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