

Do autism spectrum disorders involve a generalized object categorization and identification dysfunction?

Chris Fields

815 East Palace Ave. #14, Santa Fe, NM 87501 USA

Telephone: +1 505-995-9859

Email: fieldsres@gmail.com

Abstract: Experience-dependent learning of feature-based object categories, including entry-level categories such as “human being” and more specialized categories such as “family member”, “pet” or “toy”, is required to support correct object re-identification over time and hence to support social bonding, language learning, and the development of general life skills. It is hypothesized that activity imbalances between motion-analyzing and feature-analyzing components of the visuo-motor system resulting in hyper-activation of parahippocampal cortex relative to perirhinal cortex during the initial period of experience-dependent category learning in early infancy could lead to the construction of object categories dominated by trajectory information as opposed to feature information. It is shown that the deployment of trajectory-dominated object categories would disrupt normal object re-identification and produce developmental outcomes consistent with both the recognized symptoms and experimentally characterized cognitive-behavioral phenotypes of autism spectrum disorders. Further experiments to test the hypothesis and its potential clinical relevance are discussed.

Keywords: Vision, Category Learning, Medial Temporal Lobe, Parietal Lobe, Default Network, Mirror Neuron System

Introduction

Despite intensive study motivated by increasing prevalence and apparently-increasing underlying incidence (reviewed by Fombonne, 2009; Leonard *et al.*, 2010; Matson & Kozlowski, 2011), both the etiology and pathology of autism spectrum disorders (ASDs; APA, 2000; reviewed by Volkmar, Chawarska & Klin, 2005; Dawson, 2008; Geschwind, 2009) remain substantially unclear. Dozens of gene- or chromosomal-level variants, both inherited and *de novo*, have been implicated in ASDs (reviewed by Abrahams & Geschwind, 2008; Geschwind, 2008; Betancur, 2011; Miles, 2011a). Fetal over-exposure to testosterone (Knickmeyer & Baron-Cohen, 2006; Auyeung *et al.*, 2009), maternal and infant vitamin D deficiency (Cannell, 2008; 2010), fetal and/or infant autoimmune response (Patterson, 2009), *Toxoplasma* infection (Prandota, 2011), pathological variants in the gut microbiome (Gonzalez

et al., 2011), and a host of other environmental (DeSoto, 2009; Currenti, 2010; Kinney *et al.*, 2010) and neonatal (Gardener, Spiegelman & Buka, 2011) factors have also been implicated. These presumptive etiological factors result, in most patients, not in evident focal or systemic neuropathology, but rather in systemic disruption of normal neuroarchitectural development, with considerable evidence indicating increased local and medium-range interneural connectivity at the expense of longer-range connectivity, especially between frontal and temporal lobes (reviewed by Geschwind & Levitt, 2007; Wass, 2011). It has been hypothesized that these alterations in connectivity reflect disruptions of activity-dependent brain maturation due to early-developmental dysfunction of the highly-distributed “social brain” network (reviewed by Pelphrey *et al.*, 2011).

The relationships between the etiology and pathology of ASDs and the functional and behavioral phenotypes that provide diagnostic criteria are similarly unclear. Social interaction, language, and goal directed motor behavior – the three areas of function in which disruptions are jointly diagnostic of ASDs – are themselves complex outcomes of coordinated, systemic neurocognitive activity that are at best characterized endophenotypically at the resolution of functional MRI studies of small study populations engaged in relatively simple and often contrived activities. The diagnostic categories composing the ASDs are now commonly regarded as broad phenotypic clusters covering many distinct underlying etiologies and neurodevelopmental variants or pathologies that may result in multiple distinct endophenotypes (Happé, Ronald & Plomin, 2006 ; Rajendran & Mitchell, 2007; Geschwind, 2008; Tager-Flusberg, 2010; Miles, 2011b). Why the three diagnostic behavioral manifestations of ASDs should appear in conjunction remains unknown. Two of the deepest mysteries of the autism spectrum are the appearance of savant abilities in otherwise dysfunctional individuals (Happé & Frith, 2009), and the apparent continuity of the severity spectrum from debilitating mental retardation to highly-functional and in some cases highly-successful hyper-systemizers (Baron-Cohen *et al.*, 2003; Fitzgerald & O'Brien, 2007; Baron-Cohen, 2008; Fields, 2011a).

The present paper offers a hypothesis that the diagnostic manifestations of ASDs may be consequences of a single endophenotype: the construction through experience-dependent learning of aberrant or variant categories of objects with a consequent, reinforcing failure to correctly re-identify individual objects, including individual human beings, across gaps in observation. On this hypothesis, any combination of etiological factors that jointly results in a disruption or significant alteration of the neurotypical dominance of feature information over motion information in the token or category level representations of objects is predicted to result in symptoms of ASD, with severity dependent on the extent to which the influence of motion information on object token or object category construction is increased relative to normal development. This hypothesis offers a neurodevelopmental explanation of both the known neuropathology and the functional-behavioral phenomenology of ASDs. Experimental designs to test the hypothesis and its clinical relevance if correct are discussed.

The Hypothesis: ASDs involve disrupted object categorization and identification

That everyday objects persist through time and can be re-identified from one perceptual encounter to the next is such a basic human assumption that it is often explicitly regarded as innate (e.g. Baillargeon, 2008). One can ask, however, how the ability to re-identify objects through time is implemented by the brain, and how this implementation develops prenatally and postnatally. One can also ask how the ability to re-identify objects as persistent individuals depends on the ability to recognize individual objects as members of both general and specific categories, and how this categorization ability develops. The present hypothesis is that ASDs involve early-developmental functional disruptions of the object categorization and identification network that result in pervasive difficulties in the re-

identification of individual people or other objects over time. On this hypothesis, the “normal” early-childhood world of easily-identifiable persistent individuals – parents, teachers, friends, relatives, pets, toys and other items of the home or school environments – is replaced by a world in which many if not most perceptual encounters are encounters with novel and often mis-categorized objects. As described below, the most straightforward neurofunctional model of this hypothesis is that ASDs are the functional-behavioral expression of quantitative disinhibition of motion information in the object categorization and identification network.

Disrupted suppression of motion information in object files disrupts normal category formation

The categorization and identification of perceived objects has been most thoroughly studied in the case of vision; the current hypothesis and neurofunctional model are, therefore, developed on the basis of visual-system data. Six decades of experimental studies of within-scene visual object tracking indicate both that specific characteristics of the trajectories of moving objects dominate featural information in determining within-scene object identity for infants, children and adults, and that the ability to recognize these identity-specifying trajectory characteristics develops progressively during the first two years (reviewed by Treisman, 2006; Flombaum, Scholl & Santos, 2008; Gerhardstein *et al.*, 2009; Fields, 2011b). The short-term memory (STM) resident “object file” that provides the initial representation of a perceived object on which downstream processes including object categorization and identification operate is, therefore, dominated by motion information; indeed Gao & Scholl (2010) demonstrate that object files are constructed and processed normally even in the complete absence of static feature information. Normal human object categories such as “animal,” “person,” “toy,” or “teddy bear,” however, are not based on the trajectories that objects follow when moving, but on the static or motion-independent features of such objects: in the case of visual categorization, on what they look like. Only categories in which static features dominate trajectories enable the categorization of perceived objects regardless of whether and how they are moving. Normal human object categorization and normal human experience-dependent category learning both require, therefore, that the scene-specific trajectory information that dominates the initial object file representation be suppressed relative to static feature information. It has been suggested that this suppression of scene-specific motion information is implemented by downward inhibition of specific trajectory representations by the more general, abstracted trajectory representations that are incorporated as categorical motion constraints into normal, feature-dominated categories (Fields, 2011b), and hence that trajectory information is suppressed relative to feature information when object files are bound to category representations, as illustrated in Fig. 1.

Fig. 1 about here

Any etiologic factor or factors that consistently disrupted the suppression of trajectory information in object files during the first two years of life – the period during which object-file construction reaches mature levels of performance – would be expected to result in two mutually-reinforcing consequences. First, the detailed trajectory information that enables such early-developing skills as trajectory prediction and grasping would be expected to be incorporated into the long-term memory (LTM) resident object tokens (Zimmer & Ecker, 2010) that represent newly-encountered, novel objects; indeed, detailed trajectory information would be expected to dominate such object tokens just as it dominates object files. Second, the process of abstracting across similar object tokens to construct or extend the scope of object categories would be expected to result in categories that grouped objects based on similarities in their observed trajectories instead of on similarities in their static features.

Categories dominated by or even significantly incorporating detailed trajectory information would be aberrant compared to normal human categories; an individual deploying such categories to interpret the visual world would consider objects that moved in the same way to be the same kind of thing, but would categorize single objects capable of moving in many different ways as many different kinds of things based on their different motion patterns. Such a disruption of normal category formation would be expected, in particular, to lump normally distinguishable but similarly moving kinds of inanimate objects together, while splitting autonomous agents capable of varied behavior – such as human beings and family pets – into multiple kinds of things, each of which exhibit one categorized kind of motion. The severity of this disruption of category formation would be expected to depend on the extent to which the suppression of trajectory information in object files was disrupted. Such aberrant object categorization would be expected to have pathological functional-behavioral consequences; in particular, it would be expected to disrupt the ability to correctly re-identify individual people or other objects through time.

Disrupted categorization disrupts object re-identification

The maintenance of individual object identity across extended gaps in observation – in some cases, over years of non-observation – is known to involve four distinct but coupled processes: (1) categorization of a currently-perceived object both to general “entry-level” type (e.g. “human being”) and to a specific subtype (e.g. “middle-aged, medium-height, blond European woman”); (2) retrieval of episodic memories of contexts that include object tokens representing individuals of that specific categorical subtype; (3) selection of object tokens from the retrieved episodic memories that are good feature matches to the currently perceived object; and (4) use of both both categorical and context-dependent causal constraints to determine whether a remembered object that is a good feature match to the currently perceived object could undergo the featural and contextual changes required to be the very same individual entity as the currently perceived object (reviewed by Rips, Blok & Newman, 2006; Eichenbaum, Yolinas & Ranganath, 2007; Zimmer & Ecker, 2010; Fields, 2012). The anatomical implementation of these processes as derived from functional imaging, lesion and other data (Martin, 2007; Nassi & Callaway, 2009; Ranganath, 2010; Zimmer & Ecker, 2010; Kiefer & Pulvermüller, 2011; Pelphrey *et al.*, 2011) is sketched in cartoon form in Fig. 2. The temporal-lobe systems shown in Fig. 2 carry out the first three of the above-listed processes; with input from frontal and cingulate cortices, the parietal-lobe systems carry out the fourth process. Successful execution of these four processes whenever a particular individual is encountered is essential to the maintenance of a coherent remembered history of that individual – whether it is a person, animal, or an inanimate object – as its features and contexts of observation change through time. Correctly updating the object token representing the individual in LTM following re-identification is, in turn, essential for correct future re-identification of the individual following further featural or contextual changes.

Fig. 2 about here

The encoding of trajectory-dominated categories, or even of categories that encode detailed and hence overly-narrow categorical motion constraints, would be expected to disrupt each of the four processes required for individual re-identification. First, the currently-perceived object may be mis-categorized – compared to normal, feature-based categorization – or be uncategorizable on the bases of its currently-observed motion. Second, events may be retrieval from episodic memory based on the salient motions they involved instead of the kinds of objects they involved. Third, object tokens from remembered contexts may be matched to the current object token using primarily motion instead of primarily feature

information. Fourth, the use of overly-specific and hence narrow categorical causal constraints to simulate changes in context may rule out re-identifications of objects that would with more abstract, broader causal constraints be successfully re-identified. It is hypothesized that such disruption of object re-identification across extended gaps in observation, if initiated in early infancy, would be sufficient to cause the functional and behavioral phenotypes commonly associated with ASDs, including dysfunction or difficulties with attentional focus, language-learning and the formation of appropriate social-emotional relationships with caregivers and other individual people. On this hypothesis, infants with ASDs do not experience the world of familiar, persistent entities that neurotypicals experience and that is implicitly assumed by human languages and social arrangements; instead they experience a world of sometimes-familiar motions executed by often- or mostly-unfamiliar entities. If this hypothesis is correct, infants with ASDs fail to develop normal social abilities, in particular, because they fail to coherently identify the basic elements of the social world: persisting individual people with multifaceted personalities and complex behavior.

Neurofunctional model: Quantitative variation in motion suppression is sufficient for ASDs

A variety of underlying pathologies could, in principle, cause the formation of aberrant, trajectory-dominated categories and the consequent failure of individual re-identification described above. The most parsimonious model, however, requires only quantitative variation in the activity of the motion-representing components relative to the feature-representing components shown in Fig. 2. On this model, any etiological factor or combination of factors having the net effect of increasing activity in PHC relative to PRC would drive object-token encoding and hence object category construction and experience-dependent category learning toward an emphasis on scene-specific trajectory information at the expense of scene-nonspecific feature information. Subsequent failure to construct categories adequately representing autonomous agents capable of diverse behaviors would, in turn, suppress activity in IPL relative to SPL, driving episodic memories toward a further emphasis on how objects move at the expense of why they move. Because many geometrically complex motions – including biological motions such as walking or communicative gestures – are recognized as abstractions over particular instances executed in different ways by different individuals, failure to coherently categorize such individuals would also disable learning and hence recognition of complex motions. This positive feedback loop, if established during the initial phase of experience-dependent category learning, would be expected to affect the functional architecture of the object categorization and identification network systemically, and hence to alter category formation and object re-identification throughout life.

Explanatory power of the hypothesis

The present hypothesis and the quantitative model of its realization in the developing brain are motivated not by the symptomatology and phenomenology of ASDs, but rather by an analysis of expected failure modes in experience-dependent category learning (Fields, 2011b, 2012). The ability of the hypothesis and model to explain the symptomatology and phenomenology of ASDs, including the characteristics highlighted by social-cognitive models of autism and the results of experimental studies, therefore constitutes evidence in their favor. The present hypothesis predicts that ASDs will present symptomatically during the initial phase of experience-dependent category learning, from roughly 3 to 18 months of age, with the age of onset dependent on the severity of disruption of feature-based category formation. Evidence increasingly indicates an onset of ASDs prior to age two, that more-severe cases are more reliably diagnosed at early ages, and that early interventions lead, at least in some cases, to improved outcomes (reviewed by Dawson, 2008; Martínez-Pedraza & Carter, 2009; Chuthapisith & Ruangdaraganon, 2011; Dixon *et al.*, 2011). A recent prospective study of siblings of

children ASD, for example, indicates onset between six and twelve months of age with marked declines in social-communication abilities specifically following onset (Ozonoff *et al.*, 2010). Assuming a quantitative neurofunctional model, the present hypothesis predicts a range of severity from effective inability to construct feature-based categories and identify individuals other than by motion-oriented cues to a relatively benign phenotype in which motion information has enhanced salience both perceptually and cognitively. The apparently quantitative spectrum from severe autism to normally-functional “systemizing” (Ring *et al.*, 2008) is consistent with this expectation.

The present hypothesis provides an explanation for why deficits in language learning and social-emotional interaction would present in conjunction with the narrowly-focused attention and repetitive motor behavior that is typical of ASDs: all of these presentations can be expected to result from early formation of motion- as opposed to feature-based categories. Among the earliest detectable signs of ASD are failures to follow gaze or pointing fingers that indicate a distant object. However, both adults and children with ASDs are capable of both imitating and inferring the goals of actions that involve visible manipulations of objects (Hamilton, Brindley & Frith, 2007; Southgate, Gergely & Csibra, 2009; Marsh & Hamilton, 2011). One interpretation of these results is that in ASD, objects are salient only when they are moving, a condition characteristic of visual perception in early infancy (Flombaum, Scholl & Santos, 2008). The ability to distinguish static objects from the local background on the basis of features alone, i.e. in the absence of motion-based cues, appears to depend on the construction of feature-based categories from moving exemplars (Fields, 2011b), an ability that is on the present hypothesis disrupted in ASD. As categorization confers salience through attentional amplification (reviewed by Chun, Golomb & Turk-Browne, 2011), motion-dependent salience is, on the other hand, an expected result of motion-based categorization.

Early development of motion-based categories with a concomitant failure to develop feature-based categories is, moreover, expected to favor categorizations based on simple trajectories, as complex patterns of motion can only be generalized given a feature-based categorization that identifies the kind of object that is moving. Both children and adults with ASD tend to focus on motion details at the expense of gestalt, and exhibit deficits or delays in the recognition of biological motion, the canonical complex motion of the human environment (reviewed by Freitag *et al.*, 2008; Simmons *et al.*, 2009). Neurotypical children prefer biological to non-biological motions, as assayed with point-light walkers, from birth (Simion, Regolin & Bulf, 2008); by two years of age, children with ASD prefer predictable non-biological motions to biological motions (Klin *et al.*, 2009), a difference that corresponds in older children to alterations in the activities of STS, fusiform gyrus, amygdala and prefrontal areas (Kaiser *et al.*, 2010). Consistent with the relative decrease in activity of fusiform gyrus that would be expected if categories are primarily motion based, children and adults exhibit deficits or neglect in face perception (reviewed by Weigelt, Koldewyn & Kanwisher, 2012) and the recognition of facial expressions (reviewed by Harms, Martin & Wallace, 2010). Decreased activity in LFG and particularly the fusiform face area (FFA) also predicts decreased functional connectivity between LFG and amygdala with a concomitant decrease in feature-driven emotional responses, as is observed (Dziobek *et al.*, 2010; Herrington *et al.*, 2011).

Enhanced salience of simple motions over complex biological motions or motion-independent features can be expected to impact the development of language as well as non-linguistic communicative abilities such as pointing or gaze following. Common nouns in human languages name feature-based categories; with a few exceptions such as “orbit,” verbs refer to actions by agents, not to motions characterized by geometric trajectories. The construction of motion-dominated categories in infancy would, therefore, be expected to directly disrupt learning of common nouns, and disrupt learning of proper names and verbs through subsequent effects on the ability to represent complex motions of

identified autonomous agents. Deficit language comprehension and production are observed in ASD from the initiation of language use, with substantial variation in severity (Luyster, *et al.*, 2008; Tek *et al.*, 2008; Weismer, Lord & Esler, 2010) as would be expected on a quantitative model. Over developmental time, an attentional preference for simple motions, failure to develop feature-based categories, and consequent failure to develop categories that recognize complex, multi-entity events can be expected to result in insensitivity to the gestalt structure or conventionalized meaning of complex scenes. The characterization of ASD in terms of weak cognitive coherence (Happé & Frith, 2006) captures the detail orientation and neglect of gestalt that a focus on simple motions and a tendency to categorize objects in terms of their motions would enforce.

Unlike a “broken mirrors” hypothesis that ASDs involve systemic dysfunction of the mirror system and hence of action interpretation in general (Williams *et al.*, 2001; Iacoboni & Dapretto, 2006; Oberman & Ramachandran, 2007), the present hypothesis does not predict generalized disruption of mirror-system function; it rather predicts that the mirror system is only effectively activated by observed actions that produce evident motion in one or more target objects. Communicative, affiliative and other social actions are typically not intended to and typically do not produce evident kinematic outcomes; hence on the current hypothesis, such social actions would not activate the mirror system and hence would not be perceived as “actions” but rather as uninterpreted complex motions. Understanding and interpreting social actions requires both an ability to represent their emotional or social goals and intentions in the context of a representation of the self as a social agent and an ability to associate these intention and goal representations with the object token representing the observed agent of the social action. Both of these abilities require functional connections between the mirror network and components of the extended fronto-parietal “theory of mind” (ToM) network, including representations of long-term goals and other mental states of both the self and others (Saxe, 2006; Singer, 2006; Hamilton, 2009; Waytz *et al.*, 2010). As emphasized by Pelphery *et al.* (2011), early disruption of this network would be expected to generate the social and emotional deficits observed in ASD. On the present hypothesis, one would expect systemic hypo-development of the ToM network due to lack of activation from the mirror system, a prediction consistent with the reduced autonotic “sense of self” in ASD (reviewed by Lombardo & Baron-Cohen, 2010). As has often been noted, a selective “mind blindness” (Frith, 2001) that affects the perception and interpretation of social intentions but spares the perception and interpretation of intentions to physically manipulate visible objects is consistent with the “extreme male” cognitive orientation typical of high-functioning ASD (Baron-Cohen, 2002; 2008).

Markram, Rinaldi & Markram (2007) have characterized the experience of ASD as an “intense world” of unfamiliarity and unpredictability from which physical and emotional withdrawal are natural responses. A world of salient motions carried out by unidentifiable and often uncategorizable objects would, for a developing mind, be terrifying. Even a slight chronic over-emphasis on similarity of motions relative to similarity of features during category learning can be expected to lead to categorization conflicts and hence confusion about object identity when a previously-familiar individual executes a novel action, or even when a previously-familiar inanimate object is moved in a novel way by a third party. Conflict between motion-driven and feature-driven categorizations of a single object has recently been offered as an explanation of adverse “uncanny valley” emotional responses to actions carried out by human-like robots (Saygin *et al.*, 2011); even high-functioning individuals with ASD may find themselves in an “uncanny valley” during most interactions with other human beings.

While the core symptoms of ASDs are expected to result from early-developing quantitative variations away from normal activity within the temporal and parietal lobes as discussed, the consequences for global connectivity and activation patterns of these relatively local changes would be expected to

develop over the course of childhood and adolescence (Johnson & Munakata, 2005; Fair *et al.*, 2008; Supekar, Musen & Menon, 2009). Decreased activity in IPL, in particular, would be expected to decrease formation of relatively long-range frontal-parietal and cingulate-parietal connections. As coherent long-range activity facilitates activity-dependent pruning of short-range connections (Supekar, Musen & Menon, 2009), decreased activity in IPL would be expected to result, by late childhood, in the decreased long-range and increased temporal and parietal short-range connectivity that is observed in ASD (Wass, 2011) as previously suggested (Pelphery *et al.*, 2011). Both IPL and TPJ are significant posterior components of the default network (Buckner, Andrews-Hanna & Schacter, 2008); decreased long-range default network activity would therefore be expected in ASD, as is observed (Monk *et al.*, 2009; Weng *et al.*, 2010). Decoupling within the default network would explain ToM deficits as previously mentioned; it may also explain, at least in part, the decreased recruitment of language during internally-directed thought implied by the “thinking in pictures” cognitive style in high-functioning ASD (Kunda & Goel, 2011).

Testing the hypothesis

The primary prediction of the current hypothesis and quantitative neurofunctional model is that a common endophenotype of ASDs is an early-developing activity imbalance, relative to neurotypical development, between PRC and PHC. The model therefore predicts that genetic and other etiological factors that have been identified are either singly or in combination sufficient to generate this endophenotype without causing other, more debilitating and hence more evident comorbid phenotypes. While changes in the structure or expression of neurodevelopmental genes would be expected to affect architecturally-similar systems across the cortex, the present model requires that such changes be functionally evident primarily in the connection patterns within the medial temporal lobe (MTL) made or refined within the first 18 months of life. Why MTL would be particularly vulnerable to subtle variations in activity during this period is not addressed by the model, but the requirement that this object-encoding area develops rapidly while maintaining exceptional plasticity in the face of early experience renders such vulnerability plausible. Without considerable refinement, current functional imaging methods are unlikely to be capable of detecting the predicted endophenotype directly in infants or toddlers. However, high-resolution studies of older children, adolescents or adults engaged in learning novel object categories in the presence or absence of object motion would be predicted to reveal PHC dominance over PRC in ASD but not in neurotypicals, both when constructing initial object tokens on first exposure and during later categorization of previously-learned novel objects. The novel tool re-use protocol (Casler & Kelemen, 2007; Phillips, Seston & Kelemen, 2011) may be applicable for testing the prediction of motion-based category formation with novel objects.

The present model predicts that individuals with ASD will display systematic deficits in feature-based re-identification of individual people, animals or objects following gaps in observation. A comparison of ASD to neurotypical performance using change blindness protocols (Simons & Ambinder, 2005; Simons & Resnick, 2005) designed to assay sensitivity to featural changes in or between unfamiliar individual people or objects would be expected to reveal such deficits; modified change-blindness protocols that assay for sensitivity to changes in motion patterns of unfamiliar objects would be expected to show enhanced motion sensitivity and motion-dependent categorization in the ASD group. The individual re-identification protocol of Gutheil *et al.* (2008), which requires preschoolers to identify a plush toy with which they have interacted in the presence of identically-featured competitors, may also provide a useful assay of normal versus ASD object re-identification abilities. Protocols testing the re-identification of either previously-familiar or previously-unfamiliar individual people or objects without feature changes but with variably-significant changes in context would be expected to

reveal a greater tendency for re-identification across contexts, accurate or not, in neurotypicals compared to ASD, with the difference in performance increasing with the significance of the contextual change.

The role of the mirror system in ASD remains controversial, with different experimental protocols yielding different degrees of apparent intention understanding in different, mostly high-functioning, ASD groups (Gallese *et al.*, 2011). The protocol of Boria *et al.* (2009), which employed still images of manipulations of familiar tools, suggests that in ASD observed actions are assigned intentions consistent with the outcomes of similar actions observed in the past, a suggestion in line with the current prediction of mirror-system activation only in the presence of evident kinematic outcomes. Further experiments testing intention understanding that explicitly contrasted manipulative actions with kinematic outcomes against social actions with facial expressions or other social indicators as outcomes would be a sensitive test of the current hypothesis, especially if the protocols used were able to probe the source in memory of the assigned intentions.

A further type of investigation suggested by the proposed hypothesis is the retrospective or experimental analysis of category learning in individuals diagnosed with high-functioning ASD or displaying an unusually high orientation toward systemizing. Parental reports or other family records of early word usage, interests, or toy preferences may be useful in this regard. The protocol of Sobel *et al.* (2007), which examines the ability to attribute hidden mechanical causes to the behavior of inanimate objects in young children, may be a useful positive assay for a salience bias toward mechanical motions, while variations on point-light walker displays would provide a contrasting negative assay.

Conclusions

The present hypothesis reconceptualizes ASDs away from either atypical mental retardation or a pervasive social-emotional disability and toward a broad-spectrum agnosia with quantitatively variable severity. This reconceptualization is significant in two distinct ways. First, agnosias tend to appear and be treated against a background of normal intelligence. Viewing ASDs as variably-severe presentations of an underlying agnosia acknowledges that individuals with ASDs perceive the world differently from neurotypicals, and may be bringing normal intelligence to the task of functioning within that differently-perceived world. Second, the hypothesized agnosia is traced to the categorization and object identification functions organized around MTL. This area has been intensively studied, but with a focus on normal function and on late-life dysfunctions in dementias rather than on infancy and early development. The integration of sensory and motor capabilities around the implicit assumption of object persistence is, however, arguably the most significant outcome of the first few months of experience-dependent learning. If the present hypothesis is correct, a better understanding of the developmental genetics and developmental neurobiology of both MTL and its wide-ranging connectivity to other areas is critical to understanding the etiology of ASDs.

While intensive interventions focused on social-interaction skills show promise in ameliorating ASDs, the relatively low significance levels achieved in controlled trials suggest that either additional methods or improvements in protocol-level uniformity are required (reviewed by Ospina *et al.*, 2008; Rogers & Vismara, 2008; Odom *et al.*, 2010). If the present hypothesis is correct, measures designed to assess category learning, object categorization and object re-identification, interventions designed to explicitly teach “normal” object categories and facilitate the correct re-identification of individual human beings and inanimate objects over time, and efforts to facilitate the re-identification by patients of personnel in

particular across episodes of interaction may be useful additions to comprehensive early treatment of ASDs.

Statement regarding conflict of interest

The author states that he has no financial or other conflicts of interest relevant to the reported research.

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Figure Captions

Fig. 1: Sketch of the hypothesized mechanism leading to ASD symptoms. Trajectory information dominates feature information in the transient object files that provide the initial representation of objects involved in on-going visual scenes. Feature information dominates normal object categories. Binding of category information to an object file to generate a categorized object token representing a familiar kind of object requires the suppression of trajectory information relative to feature information (“Neurotypical Outcome” at upper right). Failure of this suppression mechanism would generate an object token dominated by trajectory information (“Hypothesized ASD Outcome” at lower right); the downstream processing of such aberrant, motion-dominated object tokens is hypothesized to result in symptoms of ASDs.

Fig. 2: Simplified cartoon showing major components and connections in the visual object categorization and identification network, including the sources of “what,” “where,” “how” and “why”

information incorporated by hippocampus (HC) into episodic memories of objects in context. The dorsal visual-processing stream via medial temporal area (MT) constructs motion representations for perceived objects in medial temporal gyrus (MTG) and superior temporal sulcus (STS) for inanimate objects and agents respectively; the ventral stream via visual area four (V4) constructs feature representations for perceived objects in medial (MFG) and lateral fusiform gyrus (LFG) for inanimate objects and agents respectively. Feature and motion information are consolidated into “what” and “where” components of object tokens by perirhinal cortex (PRC) and parahippocampal cortex (PHC) respectively. Motion-dependent “how” and “why” information is consolidated by superior (SPL) and inferior (IPL) parietal lobule – temporal-parietal junction (TPJ) respectively. New exemplars are integrated into categories via the anterior temporal pole (ATP); emotional response to objects is generated by amygdala (AMG). Solid lines indicate primary information flow; dashed lines indicate temporal and parietal lobe boundaries. Both lateral and top-down modulatory connections are suppressed for clarity.



