CHILD DEVELOPMENT

Child Development, July/August 2009, Volume 80, Number 4, Pages 946–951

Introduction to a Special Section of Developmental Social Cognitive Neuroscience

Rebecca R. Saxe
Massachusetts Institute of Technology

Kevin A. Pelphrey
Yale University

The articles in this special section offer a sample of the state of the art in an exploding new field. Noninvasive neuroimaging techniques, including functional magnetic resonance imaging (fMRI), electroencephalograms (EEG), and functional near-infrared spectroscopy (fNIRS), are increasingly being used to study development in infants, children, adolescents, and adults. Applying these tools to the study of *social* cognitive development, in particular, is a very new development. Consequently, in this commentary, we wish to step back and consider the new intersection of neuroscience and social cognitive development. What do these new fields have to offer each other, theoretically and pragmatically? What pitfalls can we expect to encounter along the way?

Even in adults, a neuroscience of social cognition is a new invention. Traditional neuroscientific techniques were applied to the brains of nonhuman animals, to study how brains see, hear, feel, move, and remember. Uniquely human cognitive capacities, such as language and social cognition, could not be studied in nonhuman animals. When neuroimaging techniques were first applied to human adults, the key test of these technologies was replication, in humans, of functions known from nonhuman animals. Thus, early neuroimaging focused on the human homologues of known regions from other primates: early visual cortex, the motion perception region (MT), early sensory, and motor cortices.

Soon, though, human neuroimaging led to fascinating novel discoveries. The human brain contains many cortical regions, previously unknown or little studied, which have apparently social functions. The fusiform face area (FFA) is involved in perceiving human faces (Kanwisher, McDermott, & Chun, 1997). The extrastriate body area is involved in perceiving human bodies (Downing, Jiang, Shuman, & Kanwisher, 2001). The right posterior superior temporal sulcus (pSTS) is involved in perceiving and analyzing human actions (Pelphrey, Viola, & McCarthy, 2004). The right temporo–parietal junction (RTPJ) is involved in reasoning about people's thoughts (Saxe & Kanwisher, 2003). The medial precuneus and posterior cingulate and medial prefrontal cortices are involved in other aspects of higher level social cognition (Amodio & Frith, 2006). With the exception of the pSTS (Perrett et al., 1985), these regions were unknown from the studies of nonhuman animals.

The discovery of these regions provides an exciting challenge for neuroscientists, and a role for developmental psychology. For neuroscientists, the key questions are: How are high-level social functions implemented in neurons? How do brain regions with these functional roles arise, phylogenetically and ontogenetically? Ideas, experimental paradigms, and data from developmental psychology may help to pose and to address these questions. For example, neuroimaging studies of action representation in the pSTS have been modeled on studies of infants' action perception (Vander Wyk, Hudac, Carter, Sobel, & Pelphrey, in press); and most early studies of the RTPJ's role in thinking about thoughts used versions of the false belief paradigm originally developed for studying children (e.g., Fletcher et al., 1995; Gallagher et al., 2000). In the future, we expect that neuroscientists will continue to benefit greatly from the theoretical concepts and paradigms generated within developmental psychology.

We express our deepest gratitude to the outstanding reviewers who generously gave of their time in order to help us select the articles for this special section. Kevin Pelphrey and Rebecca Saxe are each supported by awards from the John Merck Scholars Fund and grants from the Simons Foundation. Additionally, a Career Development Award from the National Institute of Mental Health supports Kevin Pelphrey.

Correspondence concerning this article should be addressed to Rebecca Saxe, Department of Brain and Cognitive Science, MIT 46-4019, 77 Massachusetts Ave., Cambridge, MA 02139 or to Kevin Pelphrey, Child Study Center, Yale University, 230 South Frontage Rd., New Haven, CT 06520. Electronic mail may be sent to saxe@mit.edu or to kevin.pelphrey@yale.edu.

In turn, as neuroscientists begin to link specific brain regions, and their interactions, to particular social cognitive processes, this information may enhance theory building within developmental psychology. In one example, developmental psychologists have debated for decades whether the child's mind contains special-purpose mechanisms for learning specific contents (domain specificity) or whether the structure of the mind is the outcome of domain-general learning of regularities in experience. The existence of neural regions with highly specific response profiles—for example, the FFA response to faces and the RTPJ response to thinking about thoughts—provides strong prima facie evidence for the corresponding domain-specific cognitive mechanisms.

Neuroscientific results can also contribute to developmental theory by documenting the continuity of common mechanisms across developmental stages. For example, does the perception of goal-directed action in 6-month-old infants depend on the same cognitive mechanism as the perception of goal-directed action in adults? **Yoon and Johnson** (p. 1069) provide evidence that infants perceive the structure of actions presented as point-light walkers, and even follow the gaze of the point-light person. **Lloyd-Fox and colleagues** (p. 986) strengthen the impact of this evidence for common mechanisms across development but provide initial evidence that perception of biological motion in infants depends on the same brain region as in adults—the pSTS.

A similar approach could make an enormous theoretical impact on our understanding of theory-of-mind development. The paradigmatic test of children's ability to think about other's thoughts is the false belief task: For example, children are asked to predict where a person will look for an object that she left in one location but that has since been transferred to a new location. On the traditional version of this task, 3-year-olds predict that the character will look for the object in the new (true) location; by contrast, 5-year-old children (like adults) predict that the character will look for the object in the old location, where she falsely believes it to be (**Sabbagh, Bowman, Evraire, and Ito,** p. 1147; Wellman, Cross, & Watson, 2001). The 3-year-olds who fail the traditional false belief task are not performing at chance; they make systematically below-chance predictions, with high confidence (Ruffman, Garnham, Import, & Connolly, 2001).

Nevertheless, a rapidly expanding literature has recently revealed that much younger children—as young as 15 months old—can pass the very same false belief task if their understanding is measured in terms of looking time (looking longer to unexpected actions) instead of action prediction (e.g., **Scott & Baillargeon,** p. 1172). What accounts for this long developmental lag? One account suggests that young infants have a mature understanding of false beliefs but cannot express this knowledge in the traditional action prediction format. An alternative view is that infants and adults are using qualitatively different mechanisms for understanding other minds; for example, the infants may have only an ''implicit'' understanding of false beliefs, whereas adults use an explicit, and therefore more flexible and powerful, conception of beliefs. Importantly, these accounts make clearly distinct predictions for the corresponding neural signatures. If infants and adults are using the same cognitive mechanism to think about false beliefs, then the same brain regions should be recruited in the infant and adult versions of the false belief task. This prediction has not yet been tested. We hope it will be very soon.

Neuroscientific evidence does not always have such straightforward implications for developmental theories. In particular, whereas continuity across development is fairly easy to interpret, developmental change in neural response patterns is not. There are a few basic options for patterns of developmental change in a cognitive neuroscience experiment. If the experiment uses a metabolic measure of brain function (e.g., fMRI or fNIRS), then younger participants could show less activation in the same regions as older participants, or more activation in the same regions, or less activation in the same regions and more activation in distinct regions. If the experiment uses an electromagnetic measure of brain function (EEG or magnetoencephalogram [MEG]), then younger participants could show a slower profile of response to the same stimulus, or a faster response, or could be missing a ''component'' of the response, or show a different ''component'' not present in the older participants. In all of these cases, these differences could be present along with behavioral differences in performance of the task, or in spite of matched task performance across groups.

What do these different patterns of developmental change mean? Unfortunately, there is no simple and universal rule. Instead, there are at least three options in each case. First, it is possible that the observed change reflects a purely cognitive difference between groups. Younger and older participants' brains may be organized and structured in the same ways—that is, the same neurons perform

the same computations in the same places—but the young children perform the task using different strategies, or perceive different value in the rewards. In this case, the neural evidence could be used to make a ''reverse inference'' about children's strategies or evaluations. (In a ''reverse inference,'' the engagement of a cognitive process is inferred from the activation of a particular brain region; D'Esposito, Ballard, Aguirre, & Zarahn, 1998; Poldrack, 2006.)

Second, it is possible that the observed change reflects a purely neural difference between groups. Younger and older participants' may use exactly the same strategies, but over time the brain may reorganize, so that, for example, neurons involved in a common function are closer together. Or there may even be no changes in the electrophysiological changes in neural activity, but there may be purely physical changes in the structure, density, or vascularization of the brain, causing changes in the observable neural signature (Colonnese, Phillips, Kaila, Constantine-Paton, & Jasanoff, 2008).

If so, these groups of neurons would become more visible as a focal activation in metabolic measures, and a stronger component in electromagnetic measures, even though the computations involved remained exactly the same.

Finally, the observed change may reflect a change in how the child's mind and brain are representing or computing the task; that is, neuroscientific tools could provide a window on developing cognitive mechanisms and their neural mechanisms. In many cases, we suspect, developmental social cognitive neuroscientists hope that they are studying this latter kind of developmental change. Differentiating these three causal models of change thus poses a key challenge for many of the articles in this special section.

Our own article in this issue illustrates this challenge (**Saxe, Whitfield-Gabrieli, Scholz, & Pelphrey,** p. 1203). We report an increase in the selectivity of the response of the RTPJ to talk about the contents of other people's minds with age in school-age children. However, without additional information, it is impossible for us to specify the precise mechanism underlying this finding. On the one hand, the children in our study might process the stories using the same cognitive mechanisms, across ages. From this perspective, the differences in selectivity would simply reflect maturational differences driving brain reorganization. Alternatively, the findings might reflect changes in the cognitive strategies used by children at different ages. The RTPJ, from this perspective, would remain organized in the same fashion across the observed age range, but younger and older children would use different cognitive strategies (e.g., spontaneously inventing mental states for the characters). Finally, our result could truly reflect changes across the school-age years in how both mind and brain are performing this theory-of-mind task. That is, our neuroimaging finding could reflect development of the cognitive and neural mechanisms for theory of mind.

A similar challenge faces the three articles in this issue that investigate changes over adolescence, in the medial frontal activation during self-reflection (**Guyer, McClure-Tone, Shiffrin, Pine, & Nelson,** p. 1000; **Pfeifer et al.,** p. 1016; **Ray et al.,** p. 1239). Are adolescents responding to the stimuli differently, cognitively, or emotionally, using ''mature'' neural systems? Or are the neural systems for self-reflection undergoing maturation? Converging evidence will be necessary to disentangle these two possibilities.

How might researchers work their way out of this kind of dilemma? We suggest that linkages between carefully constructed behavioral tasks (inside and outside of the magnet) and multimodal imaging data (i.e., data concerning functional and anatomical changes) collected in the context of longitudinal studies of young children will prove essential. The article by **Sabbagh and colleagues** (p. 1147) illustrates the power of combining neuroimaging data and careful behavioral tasks. They found that individual differences in resting function in the RTPJ (among other regions) are associated with the performance on false belief tasks outside the scanner. These data provide the best evidence to date that changes in the structure of the brain are related to the development of social cognition.

Although such a cross-sectional study is an excellent first step, there is a great need for longitudinal studies of social brain development in infants, children, and adolescents. Indeed, it is regrettable that none of the studies in our special section are longitudinal in design. For future research, there are straightforward methodological reasons to adopt longitudinal designs. Functional neuroimaging data are inherently noisy (particularly in children) because individual brains are different from one another. This is acknowledged, and it is part of the reason why almost all fMRI studies are conducted within subjects. A longitudinal design is the only way to study developmental processes and have the power of within-subject statistics, thereby making it much more likely to visualize actual developmental changes with reasonable sample

sizes. It also makes it more likely to detect relations between different developmental changes, and thus provides stronger inferential leverage upon which to build lasting theoretical contributions.

In addition to the challenges for data interpretation mentioned earlier, we think there are theoretical barriers to clear the communication between neuroscientists and developmental psychologists (Sur, 2008). For example, developmental psychologists are often interested in investigating whether a concept (or group of concepts) is innate or learned. By contrast, neuroscientists generally assume that all neural systems are shaped by both biological preparation and the structure of experience. The details of these interactions are often fascinating but do not translate easily into the theoretical language of developmental psychology. Two of the articles presented in this special section (**Corina & Singleton,** p. 952; **Moulson, Westerlund, Fox, Zeanah, & Nelson,** p. 1039) tackle the issue of how unusual environments and biological factors interact to generate different developmental pathways. We believe that these articles portray clear paths forward and highlight the value of mechanistic developmental analyses that consider equally biological and environmental influences.

To further illustrate this point, consider this example from the field of neuroscience. Ferret brains contain separate channels by which auditory information from the ears and visual information from the eyes are normally sent to the brain. By a very precise surgery in a developing ferret, the nerves from the eye can be induced to replace the normal auditory channel, so that primary auditory cortex—the region that normally receives auditory signals—receives purely visual information throughout development. When they are mature, these ferrets are taught to look for food near a red light but not a green light. Later if early visual cortex is lesioned, in a way that would normally cause total blindness, these ferrets continue to perform normally on this visual task, relying exclusively on their rewired ''auditory'' region. Only if the ''auditory'' cortex is also lesioned do the ferrets become functionally blind.

Critically, it is possible to then study, in microscopic detail, what happened to the auditory cortex that was rewired during development. In a normal ferret or human brain, visual cortex is organized in an intricate grid depending on the location, orientation, contrast, and color of the visual stimulus; auditory cortex is laid out in elongated stripes ordered by pitch. So what would auditory cortex look like if it received only visual inputs? Like auditory cortex or like visual cortex? The answer is a mixture of the two. Like visual cortex, rewired auditory cortex contains patches of neurons with preferences for horizontal versus vertical lines, and objects at the center of the visual field are represented at one end of the region whereas objects at the periphery are at the other end. On the other hand, rewired auditory cortex also retains elements of auditory structure, like long parallel lines of neurons with similar properties, and the grid of visual properties is more loosely packed than in real visual cortex (Von Melchner, Pallas, & Sur, 2000).

The details of this example defy easy characterization in the traditional theoretical vocabulary of developmental psychology. The division of sensory input into an auditory and visual stream is clearly biologically driven. However, the organization of early auditory cortex is partly experience driven, partly experience expectant, and partly resistant to changes in experience.

Similar, and equally fascinating, results are currently emerging from investigations of the neural basis of language. In human adults, linguistic functions typically depend dominantly on left hemisphere temporal and frontal regions. Damage to the left hemisphere in adulthood often causes deep and persistent deficits in language use and comprehension, whereas after damage to the same regions in the right hemisphere, language is often unaffected (see Goulven & Tzourio-Mazoyer, 2003, for a review). The first few studies to use functional neuroimaging in human infants suggest that this hemispheric asymmetry emerges early in brain development and is present even in very young infants (e.g., Dehaene-Lambertz, Dehaene, & Hertz-Pannier, 2002; see also Witelson & Pallie, 1973). These data suggest that the left hemisphere is structurally better ''prepared'' to process linguistic information. Nevertheless, children who suffer very early damage to their left hemisphere appear to acquire language almost normally (Bates, 1999).

Taken together, these examples suggest that neuroscientific data may not fit neatly into the preexisting theoretical categories of developmental psychology. Instead, we think both fields will change and adapt in response to their marriage, and new theoretical questions will arise that are of interest to both groups of scientists. We are very excited to see where these questions will lead, in our specific field of developmental social cognitive neuroscience, and more generally.

Of course, neuroscientific measures have pragmatic as well as theoretical benefits. Direct access to brain development could be used to provide

critical diagnoses and/or earlier predictors of long-term outcomes, including for neurodevelopmental disorders such as autism and schizophrenia. The inclusion of three articles examining various aspects of autism in this special section (**Akechi et al.,** p. 1134; **Van Hecke et al.,** p. 1118; **White, Hill, Happé, & Frith,** p. 1097) is a reflection of the intense interest in this neurodevelopmental disorder within the field of developmental social neuroscience. Properly acquired neuroimaging data are very reliable, inherently quantitative, and offer unparallel density and measurement precision. Neural measures are presumably more proximal to the genetic and neural causes of autism, and therefore could provide earlier and more sensitive markers of disease onset. Also, the density of neural data may allow for better differentiation of subgroups within heterogeneous populations that are lumped together by standard behavioral diagnostic criteria.

For example, one could adopt the fNIRS approach taken by **Lloyd-Fox and her colleagues** (p. 986) to study the response to biological versus nonbiological motion in very young children with and without autism, or even infants at increased risk for developing autism. A neurobiological marker for individual differences in social cognitive abilities would be important for improving early identification. It could also offer advantages in terms of guiding interventions. Targeted treatments could be designed to target the earliest markers, which might then guarantee the most effective course of intervention possible. Neuroimaging data could be used both to identify treatment targets and to evaluate treatment outcomes. In particular, neuroimaging data could help to distinguish different mechanisms of treatment effectiveness, including whether posttreatment improvements in social cognition correspond to rehabilitating normal neural mechanisms for social cognition, or to the construction of novel compensatory mechanisms.

We are also optimistic about a new trend in developmental social cognitive neuroscience: combining neural data with genetic markers (Hariri & Weinberger, 2003). These studies would provide for the empirical realization, in humans, of a ''transactional'' approach to the study of mechanisms for developmental changes in social cognition (Gottlieb, 1997; Sameroff & Chandler, 1975). Such a perspective emphasizes the necessity to characterize the development of social cognition as an emergent property reflecting transactions occurring across levels of an active, developing organism in its environment. The goal of theory building is then to specify mechanisms through the identification of critical transactions between two, three, or more levels of analysis (e.g., gene ↔ brain ↔ behavior transactions over a specific developmental period). We embrace this as a way to generate exciting and deeply influential theoretical advances in our field.

In sum, we stand at the beginning of a golden era for work at the interface of developmental psychology and social cognitive neuroscience. This existence of elegant behavioral techniques for studying change at the cognitive level of analysis and the explosion of neuroimaging techniques for studying brain development in even the very youngest children have provided the opportunity for studies of social cognitive development, and particularly individual differences in developmental pathways, which span multiple levels of analysis from molecules to mind. We hope you enjoy the articles in this special section as much as we have enjoyed working with the individual authors, the Editor-in-Chief of *Child Development*, Dr. Jeffrey J. Lockman, and the Managing Editor, Ms. Detra Davis, to make them available to you.

References

Amodio, D. M., & Frith, C. D. (2006). Meeting of minds: The medial frontal cortex and social cognition. *Nature Reviews Neuroscience*, *7*, 268–277.

Bates, E. (1999). Language and the infant brain. *Journal of Communication Disorders*, *32*, 4.

Colonnese, M. T., Phillips, M. A., Kaila, K., Constantine-Paton, M., & Jasanoff, A. (2008). Development of hemodynamic responses and functional connectivity in rat somatosensory cortex. *Nature Neuroscience*, *11*, 72–79.

Dehaene-Lambertz, G., Dehaene, S., & Hertz-Pannier, L. (2002). Functional neuroimaging of speech perception in infants. *Science*, *298*, 2013–2015.

D'Esposito, M., Ballard, D., Aguirre, G. K., & Zarahn, E. (1998). Human prefrontal cortex is not specific for working memory: A functional MRI study. *NeuroImage*, *8*, 274–282.

Downing, P., Jiang, Y., Shuman, M., & Kanwisher, N. (2001). A cortical area selective for visual processing of the human body. *Science*, *293*, 2470–2473.

Fletcher, P. C., Happé, F., Frith, U., Baker, S. C., Dolan, R. J., Frackowiak, R. S., et al. (1995). Other minds in the brain: A functional imaging study of ''theory of mind'' in story comprehension. *Cognition*, *57*, 109–128.

Gallagher, H. L., Happé, F., Brunswick, N., Fletcher, P. C., Frith, U., & Frith, C. D. (2000). Reading the mind in cartoons and stories: An fMRI study of ''theory of mind'' in verbal and nonverbal tasks. *Neuropsychologia*, *38*, 11–21.

Gottlieb, G. (1997). *Synthesizing nature-nurture: Prenatal roots of instinctive behavior*. Mahwah, NJ: Erlbaum.

Goulven, J., & Tzourio-Mazoyer, N. (2003). Review: Hemispheric specialization for language. *Brain Research Reviews*, *44*, 1–12.

Hariri, A. R., & Weinberger, D. R. (2003). Imaging genomics. *British Medical Bulletin*, *65*, 259–270.

Kanwisher, N., McDermott, J., & Chun, M. (1997). The fusiform face area: A module in human extrastriate cortex specialized for the perception of faces. *Journal of Neuroscience*, *17*, 4302–4311.

Pelphrey, K. A., Viola, R. J., & McCarthy, G. (2004). When strangers pass: Processing of mutual and averted gaze in the superior temporal sulcus. *Psychological Science*, *15*, 598–603.

Perrett, D. I., Smith, P. A. J., Potter, D. D., Mistlin, A. J., Head, A. S., Milner, A. D., et al. (1985). Visual cells in the temporal cortex sensitive to face view and gaze direction. *Proceedings of the Royal Society of London: B. Biological Sciences*, *223*, 293–317.

Poldrack, R. A. (2006). Can cognitive processes be inferred from neuroimaging data? *Trends in Cognitive Sciences*, *10*, 59–63.

Ruffman, T., Garnham, W., Import, A., & Connolly, D. (2001). Does eye gaze indicate knowledge of false belief: Charting transitions in knowledge? *Journal of Experimental Child Psychology*, *80*, 201–224.

Sameroff, A. J., & Chandler, M. J. (1975). Reproductive risk and the continuum of caretaker casualty. In F. D. Horowitz (Ed.), *Review of child development research* (Vol. 4, pp. 187–245). Chicago: University of Chicago Press.

Saxe, R., & Kanwisher, N. (2003). People thinkingabout thinking people. The role of the temporo-parietal junction in ''theory of mind.'' *NeuroImage*, *19*, 1835–1842.

Sur, M. (2008). The emerging nature of nurture. *Science*, *322*, 1636.

Vander Wyk, B. C., Hudac, C. M., Carter, E. J., Sobel, D. M., & Pelphrey, K. A. (in press). Action understanding in the superior temporal sulcus region. *Psychological Science*.

Von Melchner, L., Pallas, S. L., & Sur, M. (2000). Visual behaviour mediated by retinal projections directed to the auditory pathway. *Nature*, *404*, 871–876.

Wellman, H. M., Cross, D., & Watson, J. (2001). Meta-analysis of theory-of-mind development: The truth about false belief. *Child Development*, *72*, 702–707.

Witelson, S. F., & Pallie, W. (1973). Left hemisphere specialization for language in the newborn: Neuroanatomical evidence of asymmetry. *Brain*, *96*, 641.