farth 1982; Rendall et al. 1999). In habituation-dishabituation experiments that asked listeners to make a same-different judgment between calls, subjects assessed calls based on their meaning, not just their acoustic properties (Cheney & Seyfarth 1988; Zuberbuhler et al. 1999). The parallels with children's perception of words cannot be ignored (see Zuberbuhler 2003 for review).

Indeed, it is now clear that although primates' production of vocalizations is highly constrained, their ability to extract complex information from sounds is not (Seyfarth & Cheney 2003b). Upon hearing a sequence of vocalizations, for example, listeners acquire information that is referential, discretely coded, hierarchically structured, rule-governed, and propositional (Bergman et al. 2003; Cheney & Seyfarth, in press). These properties of primates' social knowledge, although by no means fully human, bear striking resemblances to the meanings we express in language, which are built up by combining discrete-valued entities in a structured, hierarchical, rule-governed, and open-ended manner. Results suggest that the internal representations of language meaning in the human brain initially emerged from our prelinguistic ancestors' knowledge of social relations, as exhibited in the information they acquire from vocalizations (Cheney & Seyfarth 1997; in press; Worden 1998).

Nonhuman primate vocalizations also exhibit parallels with human speech in their underlying neural mechanisms. Behavioral studies of macaques suggest that the left hemisphere is specialized for processing species-specific vocalizations but not other auditory stimuli (Hauser & Anderson 1994; Petersen et al. 1978). Lesion results demonstrate that ablation of auditory cortex on the left but not the right hemisphere disrupts individuals' ability to discriminate among acoustically similar call types (Heffner & Heffner 1984). Most recently, Poremba et al. (2004) measured local cerebral metabolic activity as macaques listened to a variety of auditory stimuli. They found significantly greater activity in the left superior temporal gyrus as compared with the right, but only in response to conspecific vocalizations. These and other results (e.g., Wang et al. 1995; see Hauser [1996] and Ghazanfar & Hauser [2001] for review) suggest that Arbib is wrong to assume that primate vocalizations "appear to be related to non-cortical regions" (sect. 1. 2, para. 3). They further suggest that the neurophysiological mechanisms underlying human speech processing evolved from similar mechanisms in our nonhuman primate ancestors.

In sum, research demonstrates a striking number of continuities – in behavior, perception, cognition, and neurophysiology – between human speech and the vocal communication of nonhuman primates. Nonhuman primate vocal communication does not qualify as language, but it does exhibit many of the characteristics that one would expect to find if human language had evolved from the vocal communication and cognition of the common ancestor of human and nonhuman primates.

Arbib cites none of this research. As a result, his presentation is strongly biased in favor of his own view that the emergence of human speech "owes little to nonhuman vocalizations" (target article, Abstract). To accept the convoluted hypothesis that spoken language evolved from vocalizations, through gestures, then back to vocalizations again, one must first have good reason to reject the simpler hypothesis that spoken language evolved from prelinguistic vocal communication. A substantial body of data argues against such a rejection.

Making a case for mirror-neuron system involvement in language development: What about autism and blindness?

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Abstract: The notion that manual gestures played an important role in the evolution of human language was strengthened by the discovery of mirror neurons in monkey area F5, the proposed homologue of human Broca's area. This idea is central to the thesis developed by Arbib, and lending further support to a link between motor resonance mechanisms and language/communication development is the case of autism and congenital blindness. We provide an account of how these conditions may relate to the aforementioned theory.

Arbib presents a strong argument in favor of a link between mirror neurons (MN), imitation, and the development of human language. We endorse his thesis that a protolanguage based on manual gestures was a precursor to human language as we know it today. Additional support for this claim comes from two seemingly different conditions: autism and congenital blindness.

Autism. Language and communication deficits are one of the defining features of autism spectrum disorders (ASD; American Psychiatric Association 1994) and are core elements of their diagnosis and prognosis (Herbert et al. 2002; Ventner et al. 1992). Particularly relevant is the fact that these impairments are more prominent in pragmatic speech associated with social communication (Tager-Flusberg 1997). Interestingly, individuals with ASD also display well-documented deficits in imitative behavior (e.g., Avikainen et al. 2003). Recent magnetoencephalographic data suggest that an abnormal mirror-neuron system (MNS) may underlie the imitative impairment observed in individuals with ASD (Nishitani et al. 2004). That study reported imitation-related abnormalities in Broca's area and its contralateral homologue, the human equivalent of monkey area F5, where most MN are found (Rizzolatti & Craighero 2004).

The idea that imitative abilities, and possibly language impairments, are related to basic MN dysfunction in ASD was recently investigated in our laboratory. In line with the MN hypothesis of ASD (Williams et al. 2001), motor cortex activation during the observation of simple finger movements was found to be significantly weaker in individuals with ASD compared to matched controls (Théoret et al. 2005). The MNS/language disorder hypothesis is also supported by the fact that individuals with autism display structural abnormalities in Broca's area (Herbert et al. 2002). Other symptoms that may be associated in some way with MN dysfunction in ASD include abnormal eye gaze, theory-of-mind deficits, the use of other's hands to communicate or demand, hand mannerisms, repetitive behaviors, and echolalia.

Taken together, these data support Arbib's main argument that a simple action observation/execution matching mechanism anchored in area F5 (Broca's area in humans) may have evolved into a complex system subserving human language. Consequently, a pathological, congenital dysfunction of the mirror-cell system in humans would be expected to dramatically affect social interactions and language/communication as a result of gesture/speech interpretation and acquisition. This appears to be the case in ASD. As mentioned by Arbib, Broca's is not the only area making up the human language and MNS. It is thus possible that other regions within the MNS underlie some intact language skills in some ASD individuals (e.g., grammar and syntax), which could in turn partly account for the heterogeneity of the symptoms across individuals.

To that effect, the case of individuals with ASD and normal IQ is particularly relevant to the argument put forth by Arbib. In that population, it is the social and pragmatic aspects of language that are usually impaired, with some individuals displaying normal abilities in, for example, vocabulary and syntax. It appears that ab-

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normal imitation-related cortical activations in ASD with normal IQ are located mostly within the inferior frontal gyrus as opposed to the superior temporal sulcus and inferior parietal lobule (Nishitani et al. 2004). In light of these neurophysiological results, it may be that modularity of the MNS can account for differential language symptomatology, with Broca's area being the principal component of the system.

Blindness. Another pathological condition that may add some insight into the perspective offered by Arbib is congenital blindness. It has been suggested that congenitally blind individuals display autism-like characteristics (Hobson & Bishop 2003). For example, visually impaired children perform at lower levels than normal subjects on theory-of-mind tasks (Minter et al. 1998), and blind children are at an increased risk of meeting diagnostic criteria for autism (Brown et al. 1997). Interpretation of these data as suggesting a causal link between sensory deprivation and autism-like characteristics has been challenged (Baron-Cohen 2002), but they nevertheless bring to mind interesting questions regarding ASD, MN function, and language impairment.

Some blind children display fewer periods in which they direct language towards other children and are generally impaired in the social and pragmatic aspects of language (Hobson & Bishop 2003), reminiscent of individuals with ASD. In blind individuals, lack of visual input would derail the normal mechanism matching action perception and execution within the visual system. A motor resonance mechanism could still operate through the auditory modality (Kohler et al. 2002), but in an obviously limited manner due to lack of visual input.

Mechanisms of disorder. We have tried to describe two pathological conditions that offer insight into the role of the MNS in language/communication. We have showed that a breakdown in MN function may be associated with specific language impairments, most notably pragmatic speech. In contrast to the theory put forth by Arbib, these examples speak to the ontogeny, rather than the phylogeny, of language. Nevertheless, they share a striking similarity: the necessity of an adequately "evolved" (as Arbib puts it) MNS to develop the unique ability of human language. Although still speculative, the two conditions we have described suggest different mechanisms that may lead to MNS impairment and associated language deficits.

In the case of blindness, it may be that loss of visual input impairs the normal development of a motor resonance system, thereby leading to language/communication deficits. In that sense, it is an environmental factor that hinders adequate development of the MNS. In ASD, where genetic factors are an important part of the etiology, individuals may be born with a dysfunctional MNS, preventing normal language and social behavior. In that regard, it is tempting to look at the Forkhead box P2 (FOXP2) gene, located on chromosome 7q, which is believed to be implicated in the acquisition of language (Lai et al. 2001) and may be involved in the human properties of the MNS (Corballis 2004). Most evidence argues against a direct link between autism and FOXP2 (e.g., Newbury et al. 2002), but the idea that MN development may be genetically determined is an intriguing possibility that requires further investigation.

In summary, this commentary highlights the need to test Arbib's theory against various pathological conditions, either those specific to language (e.g., aphasia) or those which may be associated with MN dysfunction (autism, schizophrenia, Williams' syndrome). For example, one of the co-morbidities of specific language impairment (SLI) is motor impairment (Hill 2001), suggesting yet another association between motor skill and language dysfunction. It seems obvious to us that specific predictions of Arbib's model need to be tested this way, as direct evidence in support of some aspects of the theory is lacking.

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Language is fundamentally a social affair

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Abstract: Perhaps the greatest evolutionary advantage conferred by spoken language was its ability to communicate mentalistic concepts, rather than just extending the vocabulary of action already served by an imitation function. An appreciation that the mirror-neuron system served a simple mentalising function before gestural communication sets Arbib's theory in a more appropriate social cognitive context.

It may not be an obvious question to ask why spoken language should evolve from gestural communication, but it is an important one. Simply put, if gesture can be used to communicate effectively, why evolve speech? Why didn't we just evolve a complex gesturing language that did not require changes to the larynx? Arbib has presented a theory of language evolution but has omitted to discuss the selection pressures involved.

According to the Machiavellian intelligence hypothesis (Byrne & Whiten 1988; Whiten & Byrne 1997), the human brain evolved because of the selection pressure to develop cognitive capacities that facilitate social manoeuvring. This would also suggest that language evolved through the need to communicate mental states. The evolution of language would be driven primarily by the need to discuss matters such as loyalty, betrayal, forgiveness, and revenge. Arbib uses few examples to illustrate the content of the language he is discussing; he mentions gestures used to describe flying birds, hunters directing each other, the tastes of food, and the use of fire to cook meat. His argument seems to assume that speech and gesture are used to discuss the physical activities of daily living, rather than to express feelings, desires, or intentions, or to consider the thoughts of conspecifics.

Also, Arbib derives his model of imitation from that proposed by Byrne and Russon (1998) following their observations of leaf-folding by mountain gorillas. This is an imitative task that requires replicating the structural organisation of an action, rather than the mental states driving it. Communicating the knowledge inherent to this skill is a relatively straightforward matter using action demonstration, whereas to describe it using only speech would be more difficult. Conversely, communication concerning invisible mental states may lend itself more to speech than descriptive gesture. Consider for example, "John wrongly thinks that Bob is jealous of me," or, "you distract John whilst I plot revenge against Bob." It may be that in the discussion of invisible mental states, speech can add a valuable modality of communication, which may even supplant manual and facial gesture.

Arbib does not mention the possible role of the mirror-neuron system in mentalising, or the importance of this mentalising function in imitation. Imitation involves incorporating a novel action into a pre-existing behavioural repertoire (Whiten et al. 2004). It follows that for this to occur, the observed behaviour must be compared with the existing knowledge of the behaviour. Therefore, imitation requires more than remembering and then replicating the components and organisational structure of an action sequence. Rather, imitation requires that the observer draw on his or her own knowledge of an action exhibited by a model. This includes the observer's knowledge of the action's relationships to causes, beliefs, goals, desires, effects, and agency. Only then can the observer understand the role of the action in the model's behaviour.

Actions are therefore vehicles for the thoughts that shape them, in that thoughts are carried by actions from mind to mind. Both imitation and "simulation theory of mind" involve observing actions or behaviours from a stance of using self-knowledge to predict the mental states behind them (Meltzoff & Decety 2003). This means that both "theory of mind" and imitation depend on relating perceived actions to their motor counterparts (Meltzoff & Prinz 2002). The mirror-neuron system is the prime candidate to serve this

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