exchanges of the same vocalization, and vocal exchanges between paired individuals and other nearby pairs. Mirror neurons may play a role in such systems but become functional only after experience.

A possible role for mirror neurons in human speech has been raised by others (e.g., Hickok 2010). Here, I propose that mirror neurons (MNs) may play a role in other forms of vocal communication, specifically wherever imitative or responsive vocalization is found. In humans, examples would include contagious laughing or crying, and synchronized singing in duets or choral groups. In more general terms, I wish to raise the possibility that mirror-like neurons exist to foster vocal communication between individuals, where rapid exchange of information is required. Communication of this type exists in nature in duetting birds and mammals, and in rapid vocal exchanges between two individuals, where the appropriate response to a particular vocalization is a vocal response given after a brief interval. I further propose that neurons that serve this function are part of inherited neural circuitry that mediates a communication system, but become active only through experience.

There are numerous species of birds and mammals that engage in rapid and frequent vocal exchanges. These may be referred to as "duetting," "antiphonal calling," or simply vocal exchanges. In birds and some primates, duetting seems to serve to maintain or strengthen the pair-bond. In some cases, the duetting pair call in such rapid synchrony that it is often difficult to determine which partner is performing which part (e.g., Geissmann 2002; Müller & Anzenberger 2002).

Such synchronization obviously requires a neural system that can manage this behavior, and, presumably, also mediate the learning processes that lead up to the performance of this behavior (Brenowitz et al. 1985. I propose that a "mirror-neuron–like" system may be central to the performance of this behavior. Such an ensemble of neurons would, in each partner, monitor both the immediately ensuing component and trigger the expected output. How such an ensemble becomes activated in an individual may depend on associative learning, but there also must be a genetically derived program that puts together the components of the ensemble in the first place.

While Cook et al. propose that experimentation in the lab is needed to demonstrate the essential role of associative learning in MN formation, most of the behaviors I am addressing are probably not likely to occur in lab settings. However, some primates widely used in laboratory settings engage in vocal exchanges that can be observed while they are in captivity, and for which an established history in behavioral neuroscience exists. In squirrel monkeys (*Saimiri*), females who are affiliative partners (as determined by their close association during foraging and rest periods) produce "chuck" vocalizations in rapid exchanges of no more than a few hundred msec (Newman & Bernhards 1997). Each affiliative partnership exchanges chucks with each other, and each individual makes chucks that are sufficiently distinct acoustically so that their partner can recognize them on the basis of their vocalizations alone. Experimentation has shown that one particular part of the chuck (referred to as the "flag") is essential for accurate recognition and response (Soltis et al. 2002). This behavior starts out during development as a more general "contact call," in which young females respond indiscriminately to the chucks of other females. Gradually, over a year or two, a female learns to distinguish, and differentially respond to, the chucks of its mother and other affiliating females (McCowan & Newman 2000). The genetic component of this behavior is in the tendency to make chucks in the first place. The associative learning component comes from a young female gradually learning to restrict her chuck responses to the chucks of affiliative partners. While MN-like populations of neurons need not be necessary for this behavior, I propose that ensembles that regulate the production, monitoring, and subsequent response (and hence "MN-like") would be more efficient (and likely favorably selected for) over populations of neurons that might be engaged in a variety of behavioral activities.

Another primate vocal communication system that might be mediated by a MN-like system is antiphonal calling in the common marmoset (*Callithrix jacchus*). Here, members of a mated pair exchange loud "phee" calls with other mated pairs within an interval of 5 sec or less (Miller & Wang 2006). This behavioral system requires that a vocalizer recognizes when its partner calls, and when a member of another pair calls, and responds only to the latter with less than a 5-sec delay. No one knows how the brain mediates this behavior, but a study (Miller et al. 2010) using immunocytochemistry of c-fos gene expression has identified areas of the cortex that show neural activity to hearing a phee, to producing a phee, and to the production and hearing of a respondent's phee. No one, as yet, has recorded from single units in the identified frontal cortical areas in the marmoset, so it would be interesting to learn if MN-like activity was found there. Such specialized activity would likely occur both when the vocalizer called and when there were responses.

To summarize, ensembles of neurons that possess mirror-neuron–like properties are likely to exist in a wide range of birds and mammals, so that a correspondingly broad approach to identifying and learning more about these systems is needed. Cook et al. suggest that experiments in the lab would be needed to reveal the MNs emerging during associative learning. My suggestion that MN-like systems exist widely in nature implies that behavioral tests performed in a lab setting would be insufficient to explore the full extent of their role in communication. Some species are too vulnerable in nature or otherwise protected from invasive experimentation, but, with the improving technology to record neural activity in freely moving animals, it might be possible to study these systems. Some work being done on European Starlings (*Sturnus vulgaris*) has potential here (George et al. 2010).

ACKNOWLEDGMENT

This research was supported (in part) by the Intramural Research Program of the NIH, NICHD.

NOTE

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Associative learning alone is insufficient for the evolution and maintenance of the human mirror neuron system

doi:10.1017/S0140525X13002422

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Abstract: Cook et al. argue that mirror neurons originate from associative learning processes, without evolutionary influence from social-cognitive mechanisms. We disagree with this claim and present arguments based upon cross-species comparisons, EEG findings, and developmental neuroscience that the evolution of mirror neurons is most likely driven simultaneously and interactively by evolutionarily adaptive psychological mechanisms and lower-level biological mechanisms that support them.

In the target article, Cook et al. suggest that the evolutionary origins and maintenance of the mirror neuron system (MNS) lie in "domain-general processes of associative learning in the course of individual development, and, although they may have psychological functions, their psychological functions do not necessarily have a specific evolutionary purpose or adaptive function" (target article, Abstract). We agree that the excitement surrounding the discovery of mirror neurons (MNs) has led to an inordinate focus on their role in social-cognitive functions and how these functions might play a role in the evolution of the MNS. However, we strongly disagree with the authors' claims that the known social-cognitive roles the MNS plays in primate cognitive and behavioral functioning have not and do not affect the MNS in an evolutionary context and that the associative account "separates questions about their origin and function" (sect. 1, para. 4).

The target article describes a lower-level biological mechanism (associative learning) that, as Cook et al. argue, fully accounts for the phylogenetic and ontogenetic development of mirror neurons. We assert that the initial evolution, further evolution, and evolutionary maintenance of the MNS is likely jointly influenced by such lower-level biological mechanisms *and* by the well-documented role that the MNS plays in social-cognitive functions. One example of this joint influence can be observed in individuals with an intact versus an impaired MNS who are more able to attract reproductive partners, reproduce, and protect and provide for their offspring within the complex social structures of primate societies (e.g., Howlin & Moss 2012).

We agree that associative learning is likely a critical mechanism for both the development and the evolution of mirror neurons. However, given that associative learning mechanisms exist in species that do not have a MNS, some alternative mechanism *must* interact with associative learning in order to produce the evolutionary pressure required for the origin and maintenance of the MNS in humans. To avoid directly addressing the evolutionary advantages the social-cognitive functions of the MNS confer, Cook et al. use a "straw man" argument. They attack the most extreme proposal of the role of social-cognitive functions in the evolution of the MNS – evolutionary selection via action understanding. The "associative learning in vivo" and "evolutionary selection based upon action understanding" accounts represent polar extremes, both of which are unlikely to reflect reality. Simultaneously, however, the adaptive advantages of the social-cognitive capacities (e.g., action perception, processing, and prediction) ascribed to the MNS enhance individuals' reproductive fitness, creating precisely the evolutionary pressure that the authors propose has not, and does not, exist.

Cook et al.'s depiction of the role of developmental research in elucidating biological/genetic versus environmental/learning influences on the MNS is concerning. We agree that evidence for neonatal imitation is limited and, even if it is present, is unlikely to be driven by MNS mechanisms since cortical regions that contain MNs are not fully developed at birth. However, the postnatal developmental timeline of the MNS neither rules out genetic/biological and evolutionary processes nor demonstrates the role of associative learning. It is well known that frontal and association cortices that house MNs undergo striking synaptic development and myelination between 8-months and 3-years of age (Huttenlocher 2002; Imada et al. 2006; Locke et al. 1995). Developmental EEG evidence similarly indicates protracted cortical development in these regions (Hagne 1968; Southgate et al. 2009), with continuing maturation until late childhood or adolescence (Martineau & Cochin 2003). Therefore, biological factors may explain protracted MNS development.

Cook et al. also dismiss EEG mu suppression as an index of MNS functioning too quickly. The strong relationship between the mu rhythm and action observation/execution can be traced back to 1954, when Gastaut and Bert reported that the mu rhythm was consistently reduced when stationary subjects "identified themselves with an active person represented on a screen" (see also Pineda 2005). We also recently published a re-analysis of pooled data from four published studies, including a total of 66 individuals with autism spectrum disorders (ASD), demonstrating that, across the age-span from 6–17 years, there was significantly less mu suppression in individuals with ASD compared with matched controls during action observation, but not during self-movement (Oberman et al. 2013). Although source estimation indicates that the generator of the mu rhythm is in the postcentral gyrus rather than premotor or primary motor cortex (Hari & Salmelin 1997), the possible downstream modulation of motor cortex by the MNS is tangential to their mirror properties. Cook et al. also ignore recent studies showing that the same stimuli that elicit mu suppression also activate MN regions (as indicated by BOLD response; Perry & Bentin 2009) and modulate a TMS-induced motor evoked potential (Lepage et al. 2008), suggesting that all three indices are likely capturing the same underlying cortical mechanism.

In summary, we argue, contrary to Cook et al., that the origins and evolution of mirror neurons are unlikely to be driven by associative learning alone, but, rather, to be a consequence of a combination of evolutionary, biological, developmental, social-cognitive, and experience-based influences. Indeed, we speculate that the MNS is not functionally fixed, but rather a currently evolving, flexible, semi-modular neural network that interacts with multiple other neural systems, including the motor and social-motivation systems (Oberman et al. 2008). The functioning of such a system at any point in development should be viewed as a snapshot of a dynamic system that is constantly modulated by these influences and interactions with other systems (Johnson 2011; Johnson et al. 2002). Environmental and biological influences unfold simultaneously and interactively, not separately and sequentially, and their relative roles can only be disentangled with careful measurement and calculation (Dobkins et al. 2009; Smit et al. 2012). Cook and colleagues attack theories that argue for the evolution of the MNS based upon its proposed role in action understanding (Rizzolatti & Fadiga 1998; Rizzolatti et al. 1996), but we believe that the theory proposed by Cook et al. arguing that associative learning mechanisms alone can account for the origins and development of the MNS is equally as unlikely. Both models ignore the reciprocal relationships between evolutionarily adaptive psychological mechanisms and the lower-level biological mechanisms that are required for their existence.

Testing key predictions of the associative account of mirror neurons in humans using multivariate pattern analysis

doi:10.1017/S0140525X13002434

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Abstract: Cook et al. overstate the evidence supporting their associative account of mirror neurons in humans: most studies do not address a key property, action-specificity that generalizes across the visual and motor domains. Multivariate pattern analysis (MVPA) of neuroimaging data can address this concern, and we illustrate how MVPA can be used to test key predictions of their account.