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Neonatal imitation and an epigenetic account of mirror neuron development

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Abstract

Neonatal imitation should not exclusively be considered at the population-level; instead, we propose that inconsistent findings regarding its occurrence result from important individual differences in imitative responses. We also highlight what we consider to be a false dichotomy of genetic versus learning accounts of the development of mirror neurons (MNs) and, instead, suggest a more parsimonious epigenetic perspective.

> A number of lines of evidence support the notion that neonatal imitation is a real phenomenon. Though we realize our commentary is unlikely to settle this debate, we believe that Cook et al. fail to consider the importance of individual differences in neonatal imitation. Neonatal imitation has been demonstrated using more than one gesture (which is critical because it shows specificity in matching) in more than two dozen studies. In fact, recent work – not reported by Cook et al. – refutes the notion that neonatal imitation is simply an arousal effect (Nagy et al. 2012). Similarly, neonatal imitation is not a reflex-like behavior, as newborns appear to remember, after a delay, both the particular gesture (Paukner et al. 2011) and person (Simpson et al., under review) with whom they interacted and initiate interactions. Moreover, nursery infant monkeys, who have no exposure to contingent behaviors from caregivers, and therefore have no opportunities to learn to imitate, still show neonatal imitation (Ferrari et al. 2006). Given that neonatal imitation occurs in a variety of primates, it may be a shared behavioral adaptation (Paukner et al. 2013a).

Critically, neonatal imitation may reflect activity of the nascent mirror neuron system, as it is associated with suppression of specific electroencephalogram (EEG) frequency band activity (Ferrari et al. 2012). This work is consistent with a recent study based on simultaneous EEG and functional magnetic resonance imaging (fMRI) in human adults showing activity of the parietal and premotor/motor cortex (i.e., MN areas) linked to EEG suppression within the alpha band (i.e., mu rhythm) (Arnstein et al. 2011). And, there is

EEG evidence of a functioning mirror neuron system from birth in neonate macaques that lack any early face-to-face contingent experience with social partners (Ferrari et al. 2012).

Inconsistent neonatal imitation findings (e.g., Cook et al.'s Fig. 2) may be the result of variation among infants in imitation, indicating significant individual differences in infants' abilities to learn contingent behavior, upon which critical cognitive and social skills are based (Reeb-Sutherland et al. 2012). In support of this idea, recent findings reveal individual differences in neonatal imitation in monkeys are correlated with visual attention to social partners (Simpson et al., in press; similar findings in humans: Heimann 1989), person recognition (Simpson et al., under review), face viewing patterns (Paukner et al. 2013b; Paukner et al., under review), deferred imitation (Paukner et al. 2011), and goal-directed movement (Ferrari et al. 2009). Therefore, rather than dismissing neonatal imitation – as Cook et al. appear to do – we argue that one should focus on the causes and consequences of individual differences in neonatal imitation through longitudinal (Suddendorf et al. 2012) and comparative (de Waal & Ferrari 2010) studies of newborns. We suggest that it would be insightful to examine neonatal imitation in infants who have siblings with autism spectrum disorder, a high-risk population (e.g., Chawarska et al. 2013), or examine effects of early experiences on neonatal imitation, including behavioral (e.g., Sanefuji & Ohgami 2013) and pharmacological (e.g., Tachibana et al. 2013) interventions.

In addition to questioning their view of neonatal imitation, we, like others (e.g., Casile et al. 2011; Del Giudice et al. 2009), believe that Cook et al. are mistaken in opposing genetic and learning views on mirror neuron system development. Instead, similar to studying any developmental phenomenon, it is important to consider gene expression in different environments, and in different species, in order to understand how evolution produced predictable, functional, and species-specific phenotypes. Using this approach, we can examine how mechanisms of learning evolved to produce adaptive specializations through epigenetic mechanisms (Domjan & Galef 1983). Epigenetics is the study of changes in gene expression as a consequence of an organism's response to different environmental stimuli; genes can be temporally and spatially regulated, and epigenetics is the study of these reactions and the environmental factors – including the prenatal environment – that influence them. Countless examples emerging from the field of epigenetics demonstrate that genetic and epigenetic inheritance is not indicative of innateness, nor are phylogenetically inherited traits insensitive to experience (e.g., Jensen 2013; Roth 2012). Indeed, epigenetic models now focus on the origins of complex behaviors; we propose that such models should be considered along with associate learning mechanisms in predicting developmental trajectories, within and between species. We agree that it is misleading to think that natural selection selects only specific "good" genes. Instead, natural selection acts on phenotypes, which are the result of complex interactions, including environmental effects on gene expression. Therefore, it is more fruitful to identify epigenetic regulatory factors responsible for the emergence of predictable developmental brain/behavior trajectories, than to search for genes that produce specific phenotypes. For example, in macaque infants, we are now beginning to understand the epigenetic mechanisms that can explain how early social adversity increases the risk of disease and disorder (e.g., Provençal et al. 2012).

Behav Brain Sci. Author manuscript; available in PMC 2015 October 28.

We also agree with Cook et al. that learning likely shapes the development of the MN network in the brain, but learning occurs differently as a function of individual characteristics and context. Selection pressures operate not only on the final phenotype, but also on the interactions between genes and the environment and the interactions between molecular factors and the environment (Blekhman et al. 2008). It is possible that MNs evolved to support learning of basic functions of sensorimotor recognition of others' behavior, essential, though not specifically an adaptation for, higher-order cognitive functions, as well as sensorimotor learning (Bonini & Ferrari 2011). The interaction of genes and experience through learning can only occur if the basic neural circuitry is there to support such learning. We contend that MNs may provide the scaffolding for these interactions early in life, having themselves been remodeled by epigenetic processes across evolution.

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Behav Brain Sci. Author manuscript; available in PMC 2015 October 28.

Simpson et al. Page 4

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