cases of replacement of the ISC system are known in unrelated microbial lineages. These are Archamoebae and the breviate amoeba *Pygsuia biforma*, which have instead a bacterial nitrogen-fixation (NIF) system and an archaeal SUF machinery, respectively [12,13]. In both cases, Fe-S requiring enzymes (e.g. [FeFe]-hydrogenase) have been retained along with the MROs where they function, in spite of the absence of the ISC system. Clearly, the situation is different in *Monocercomonoides* sp., which streamlined its mitochondrial function to the extreme — the loss of the organelle. So in such a complex array of organelles and functions, pinpointing an exact set of causes for mitochondrial loss is premature. It is likely that reductive mitochondrial evolution in *Monocercomonoides* sp. and MRO-containing lineages is not just the result of genetic opportunities (e.g. LGTs) and functional redundancy. Other forces are at play, including chance, biological constraints due to specific lifestyles (e.g. energy requirement), as well as varying responses to environmental conditions.

More generally, such comparisons across the eukaryotic diversity vividly remind us, if need be, of the importance of discovery science. Our current understanding of eukaryote diversity and evolution (see [14] for a recent review) compels us to interpret the absence of mitochondrial organelles in *Monocercomonoides* sp. as a derived state. It would have been different 20 years ago, under the so-called Archezoa hypothesis, which postulated that some microbial eukaryote lineages diverged before the mitochondrial endosymbiosis, thus ancestrally lacking mitochondria [15]. If today we are confident in the secondarily amitochondriate nature of *Monocercomonoides* sp., it is because of the continuing discovery and functional characterization of a wide range of MROs in diverse lineages, as well as the improved resolution of the eukaryotic tree. The vast majority of eukaryotic diversity is composed of unicellular microbes — *the protists* — that, much like *Monocercomonoides* sp., are key to understanding the evolutionary paths that gave rise to this biodiversity. As shown here by Karnkowska and co-authors [5], genome sequencing is a powerful tool that can shed light on extraordinary cellular and evolutionary processes in unexplored parts of the biosphere. Current research has barely scratched the surface of protist diversity; it is now time to dig deeper.

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Imitation: Not in Our Genes

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http://dx.doi.org/10.1016/j.cub.2016.03.060

A powerful longitudinal study has failed to find any evidence that newborn babies can imitate facial gestures, hand movements or vocalisations. After 40 years of uncertainty, these findings indicate that humans learn to imitate; this capacity is not inborn.

Humans are hyper-social animals. We depend on cooperation with others — relatives, friends, and strangers — to fulfil our basic needs, and to learn the knowledge and skills that make human lives so very different from those of other animals. Since the 1970s [1], many scientists have been convinced

that, at the psychological and neurological levels, human hyper-sociality depends on an inborn capacity for imitation. A genetically inherited ability to copy body movements is the foundation for development of the other cognitive tools needed for human cooperation, such as empathy, mind reading, and language. Doubts about this view have been raised by small-scale studies that failed to find evidence of neonatal imitation, but neither the positive nor the negative findings were clear-cut. A study by Oostenbroek *et al.* [2] reported in this issue of *Current Biology* is decisive: testing a large sample of infants, longitudinally at four time points and using a wide range of action types, these authors failed to find any evidence of imitation in human newborns. Because of the unprecedented scale and methodological rigour of this study, the negative results indicate that imitation is not 'in our genes'.

Can Newborns Imitate?

In the late 1970s, Meltzoff and Moore [1] reported that human newborns can imitate a range of facial gestures, including tongue protrusion, mouth opening, and lip protrusion. They used a new 'cross-target' procedure in which infants provide evidence of imitation by performing a target action, such as mouth opening, more often when observing an adult performing the target action (mouth opening) than when observing an adult performing one or more alternative actions (for example, tongue protrusion). Meltzoff and Moore's evidence of neonatal imitation was sensational for two reasons. First, it had previously been assumed that infants learn to imitate, and begin to show this capacity at about 9 months of age [3]. Second, an agent can see the facial gestures of others, but can only feel the movements of her own face. Therefore, neonatal imitation of facial gestures suggested that humans have a mysterious, inborn capacity to infer, from the sight of an action, what it would feel like to perform the action.

In the last four decades, more than 80 published experiments have attempted to replicate Meltzoff and Moore's findings using the cross-target procedure [4]. Many failed to find evidence of neonatal imitation. However, because infancy research is very hard to do well, and each experiment involved a modest sample of babies — the average was around 30 — it was impossible to judge with certainty whether the positive or the negative results were reliable. Oostenbroek *et al.* [2] have overcome these problems by testing more than 100 infants, longitudinally at 1, 3, 6 and 9 weeks of age, in a cross-target procedure involving a wide range of targets. They recorded the frequencies of nine target actions — tongue protrusion, mouth opening, happy expressions, sad expressions, index finger protrusion, grasping, MMM sound, EEE sound, tongue click — while infants observed eleven movement stimuli — an adult performing each of the nine actions, and two object movements (spoon protruding through a tube and box opening). The results of this comprehensive study were wholly negative: in no case did the infants consistently perform a target action more often while observing the same action than while observing all of the alternative actions.

Why Did It Take So Long?

In the sea of negative findings reported by Oostenbroek *et al.* [2], one target action stood out. Tongue protrusion out-performed all of the other target actions. Infants protruded their tongues more often when observing tongue protrusion than when observing seven of the ten alternative actions [2]. In combination with the results of a meta-analysis in 1996 [5], a recent review of research on neonatal imitation [4] and studies showing that newborns protrude their tongues in response to a range of arousing stimuli, such as flashing lights and lively music [6], this helps to explain why it has taken so long to establish that human newborns do not imitate. Tongue protrusion, a target action that has been tested more than any other, was giving false hope. It is true that, if you look at a baby and stick out your tongue, the baby is likely to do it back. But the baby is not imitating your action. The baby is just excited by what she is seeing, and when babies are excited, they tend to stick out their tongues.

Figure 1. Tongue protrusion.
Infants tend to stick out their tongues when they are aroused, and the sight of tongue protrusion arouses infants. This non-imitative effect has given the false impression that newborn humans can imitate. (Image courtesy of Jane and Elizabeth Leighton.)

Because of this, the non-imitative tongue protrusion effect has both skewed the scientific data [5] and given many of us the false impression that, when leaning over a cradle, we have experienced neonatal imitation for ourselves (Figure 1).

Factors in the sociology of science may also have delayed a study like that of Oostenbroek *et al.* [2] and thereby extended the period of uncertainty. Infancy research is not only hard but expensive. It requires dedicated testing facilities, large teams of researchers, and long hours of labour to get usable data. Therefore, as theories of cognitive and social development have become increasingly dependent on the assumption that newborns can imitate [7,8], negative results became harder to publish, and believers were more likely than sceptics to get a publication return on their research investment. It may not be incidental that Oostenbroek and colleagues are based in Australia rather than the United States, where commitment to neonatal imitation has been strongest, and that their study is published in *Current Biology* rather than a specialist journal, where stakeholder interests can have a stronger influence.

What Goes and What Stays?

The study by Oostenbroek *et al.* [2] indicates that humans do not genetically inherit a neurocognitive mechanism for imitation. In principle, like secondary sexual characteristics, an imitation mechanism could be genetically inherited without being manifest in early infancy. This is logically possible, but, without evidence of neonatal imitation, it is an hypothesis without empirical support. It would be supported by evidence that imitative ability is highly genetically heritable, but in fact twin studies have shown that individual differences in imitation are associated predominantly with environmental rather than genetic factors [9]. While there is no convergent evidence that the capacity for imitation is inborn, there is a substantial body of evidence indicating that humans learn to imitate through social interaction [4,10]. For example, individual differences in associative learning ability at 1 month predict imitative performance at 9 months [11], and at 7–9 months, neurophysiological responses indicative of imitation can be manipulated by sensorimotor training [12].

The latest findings [2] on neonatal imitation also undermine the view that humans, and monkeys, genetically inherit a predisposition to develop mirror neurons; single cells that fire when a particular action, such as grasping, is observed, and when the same action is executed [13]. The leading evidence that mirror neurons are inborn, rather than products of associative learning, comes from the studies of neonatal imitation that Oostenbroek *et al.* [2] have shown to be unreliable [14,15].

In contrast, by establishing that the capacity to imitate depends on learning through social interaction, rather than an inborn mechanism, the findings of the new study [2] do *not* raise doubts about the importance of imitation in cognitive and social development. There is no reason why a socially constructed capacity for imitation should not play a key role in promoting cooperation [16] and cultural inheritance [17], both directly and as a platform for the development of empathy, mind reading, language and other cognitive skills [18]. The findings reported by Oostenbroek *et al.* [2] call for new research on the origins rather than the consequences of imitation. When there is evidence that a neurocognitive mechanism is inborn, cognitive scientists tend to stop trying to explain how it develops and how it works [19]. Now it is clear that imitation is not 'in our genes', the challenge is to find out what kinds of social interaction are most important for the development of imitation, and exactly how the imitation mechanism operates.

At a broader level, the advance made by Oostenbroek *et al.* [2] encourages us to think more carefully about why human minds and human lives are so different from those of other animals. Many developmental and evolutionary psychologists assume that humans are 'special' because we genetically inherit a set of complex cognitive mechanisms, dedicated to functions such as language, mental time travel, cheater detection, face recognition, and theory of mind. Along with language, imitation was one of the first mechanisms to appear on this list. Now we know that imitation does not belong on the list — it does not depend on an inborn cognitive mechanism — there is renewed impetus for research testing alternative hypotheses. For example, at birth, human minds may be different from those of other animals only in subtle ways, giving us a tendency to stare at faces, an exaggerated sensitivity to social rewards, an extended capacity for learning, and a docile temperament [20]. But these subtle biases connect developing minds to the mature minds around them, and allow children to 'download' more complex cognitive processes through social interaction.

Imitation is a mighty oak of human cognition. Oostenbroek *et al.* [2] have confirmed that it grows from little acorns. Maybe the same is true of other trees in the cognitive forest.

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