Opinion

Protracted development of motor cortex constrains rich interpretations of infant cognition

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Cognition in preverbal human infants must be inferred from overt motor behaviors such as gaze shifts, head turns, or reaching for objects. However, infant mammals – including human infants – show protracted postnatal development of cortical motor outflow. Cortical control of eye, face, head, and limb movements is absent at birth and slowly emerges over the first postnatal year and beyond. Accordingly, the neonatal cortex in humans cannot generate the motor behaviors routinely used to support inferences about infants’ cognitive abilities, and thus claims of developmental continuity between infant and adult cognition are suspect. Recognition of the protracted development of motor cortex should temper rich interpretations of infant cognition and motivate more serious consideration of the role of subcortical mechanisms in early cognitive development.

Motor behavior as a window on cognition

It is said that our eyes reveal our thoughts, but the same can be said of any movement. Accordingly, researchers routinely use infant eye and head movements, facial expressions, reaching behaviors, and locomotion to infer what is happening in the infant’s mind – knowledge, emotions, morals, and goals (e.g., [1–4]). When researchers couple inferences about infant cognition with the assumption that the cognitive processes are instantiated in the cerebral cortex, they must also conclude that the infant cortex is the source of motor outflow that crystallizes cognition in movement (Figure 1A).

But what if this conclusion is wrong? Here, we present evidence that the cortical capacity for adult-like motor control is absent in newborns and only begins to emerge around 3 to 6 months of age. Of course, like adults, newborns can move all their body parts, including the eyes, face, head, torso, limbs, and fingers. But in adults, cortical control of movement is fully developed and functionally integrated, whereas in newborns it is not. In newborns, the brainstem produces movements throughout the body, and there is no evidence that the cortex ‘speaks’ to the brainstem so as to influence motor behaviors. The initial absence of cortical motor outflow argues against theories about the developmental and neurobiological continuity of infant cognition, such that infants possess the same ‘core knowledge’ present in adults. Specifically, if the cortex does not organize and execute newborn motor behaviors, and if cortical motor outflow only emerges gradually over the first 6 months and beyond, then either knowledge, emotions, morals, goals, and the like are produced subcortically (and thus are not developmentally continuous with adult cognition) or the behavioral indices of early infant cognition are unreliable. Something’s gotta give.

Note that absence of cortical motor outflow does not mean that the neonatal cortex lies dormant, awaiting the opportunity to ‘turn on’ or ‘come online’. The quantity and patterning of early cortical

Highlights

Researchers routinely use motor behaviors (e.g., eye, face, and limb movements) to index cognition in the human neonate.

When developmental researchers use infant movements to index cognition, they often assume that the cortex is involved in producing the behavior.

However, cortical control of movement is absent at birth, emerging gradually over the first several postnatal months and beyond; before cortical outflow emerges, brainstem networks produce complex motor behavior.

Thus, cortical control of the motor behaviors used to infer cognition in neonates is not neurobiologically plausible.

Researchers should be cautious when making claims about developmental continuity between newborn and adult cognition (i.e., ‘core knowledge’) and its supporting neural architecture.

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Activity are incompatible with such a notion [5–9] (Box 1). Thus, before the emergence of motor outflow, the cortex receives abundant sensory and nonsensory input that lays the foundation for the development of the cortical specializations and functionally integrated cortico-subcortical networks that will support later-developing cognition and behavior.

**Development of cortical control of body movements is protracted**

Primary motor cortex (M1) gets its name from its unambiguous role in adult motor control: M1 neurons fire before self-generated movement, and electrical stimulation of M1 produces both simple and complex movements [10,11]. As shown in Figure 1B, M1 is a major source of the corticospinal tract that projects directly to spinal motor neurons controlling muscles of the
limbs and trunk [12], and the corticobulbar tract that projects to cranial nerves controlling muscles of the face, jaw, and tongue [13] (but not the eyes; see next section). The corticospinal tract also projects to motor structures in the brainstem that, in turn, project to the spinal cord.

In human newborns and the young of other mammalian species, M1 and its descending projections do not exhibit the signature anatomical and functional characteristics with which they are associated in adults [14,15]. Development of these motor systems entails the initial establishment of anatomical connections between cortical axons and brainstem and spinal motor neurons, refinement of established connections, myelination, and formation of topographically precise motor maps [15]. Critically, anatomical evidence of cortical connectivity with downstream targets does not necessarily mean that these connections contribute to behavior. For example, within a few days after birth in rats, M1 has established direct corticospinal connections with spinal motor neurons; nonetheless, M1 does not assume motor functions until at least 3 weeks later [16,17]. In anesthetized rats at postnatal day (P) 25, electrical microstimulation in M1 produces only the simplest forelimb movements (e.g., wrist extension) (Figure 2A); by P30, more complex forelimb movements are produced (e.g., grasping), and movement complexity continues to increase through P60 (i.e., adulthood).

If M1 is only beginning to contribute to behavior at P25 in rats, what does it do before then? Despite its name, M1 initially functions exclusively as a sensory structure (Box 1). Moreover, M1’s early-developing sensory map is somatotopically aligned with its later-developing motor map, which

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**Box 1. The surprising sensory origins of primary motor cortex**

If primary motor cortex (M1) does not contribute significantly to motor control during much of early development, what is it doing? Long before M1 plays any role in motor control, neural recordings in rat pups show that M1 initially functions like a prototypical sensory structure [91,92]. For example, during rapid eye movement (or active) sleep, the brainstem in P6 rats generates hundreds of thousands of brief and discrete limb and whisker twitches daily. Twitches trigger pulses of proprioceptive feedback that, in turn, initiate a cascade of neural activity throughout the sensorimotor system, including primary somatosensory cortex (S1) and M1 [93]. Even in P20 rats, which is near the time of weaning, neurons in the forelimb region of M1 respond exclusively to sensory feedback from sleep- and wake-related movements [9] (Figure I). Such findings suggest that the early-developing and topographically organized sensory map in M1 lays the foundation for its later-developing motor map. The findings also counsel against assuming that the function of a developing cortical structure corresponds in an obvious way with its function in adults.

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**Figure I. Sensory origins of primary motor cortex (M1).** (A) Boundaries of primary cortical areas in rats: primary somatosensory cortex (S1, red) and M1 (blue), and primary auditory (A1) and visual (V1) cortex. (B) Enlargements of red and blue regions in (A) show the somatotopic organization of S1 and M1. Adapted, with permission, from [92]. (C) Perievent histogram showing sensory responsiveness of an individual neuron in the forelimb region of M1 at P20. The neuron’s firing rate is shown in relation to movement onset (vertical broken line) for twitches (red) and wake movements (black). This neuron is representative of all M1 neurons recorded at this age. Neurons fire above baseline (0 on the y-axis) after – not before – movement onset during both sleep and wake, indicative of sensory responding. Adapted, with permission, from [9].
means that the former lays the foundation for the latter. Thus, developmental changes in M1 powerfully illustrate that cortical activity— even task-relevant cortical activity—occurring contemporaneously with a movement does not imply that the cortex plays a causal role in the production of that movement.

The inability of M1 in infant rats to produce movement contrasts with subcortical motor structures. For example, in 1-week-old rats, neurons in the red nucleus—a midbrain motor nucleus—increase their activity before the onset of a forelimb movement, and, moreover, stimulation of the red nucleus evokes movements [18,19]. Thus, through at least the first 3–4 postnatal weeks in rats, brainstem networks are sufficient to support complex postural and locomotor skills [20,21] (Box 2).
Relative to human newborns, rats are extremely immature at birth, but cats are born nearly as mature as humans [22]. Nonetheless, like rat pups, kittens show protracted M1 development [23]. Before P60 in kittens, electrical microstimulation in M1 produces movements at only 5% of electrode sites; adult-like levels (~67% of sites) are not achieved until P81–P90—several weeks after weaning [24]. Along with these changes in stimulation efficacy, the threshold of activation decreases and the representation of the forelimb expands in its motor map (Figure 2B). Finally, as in rat pups, corticospinal connections are established in kittens long before M1 influences movement.

The findings in rats and cats tell a similar story about protracted M1 development. But are the findings relevant for human infants, who, many assume, have more complex brains and developmental timelines? Assumptions about human exceptionalism should be met with skepticism [25]. Indeed, in a detailed analysis of 271 developmental events across 18 mammals—including rats, cats, and humans—a single model is sufficient to predict the timing of all events with great accuracy [22]. The ordering of the 271 events, which includes measures of brain growth, synaptogenesis, myelination, eye opening, and walk onset, was conserved across species, with the relative timing of events showing the most between-species variability. In other words, the order of developmental events is similar in rats, cats, and humans, but the timing varies from days in rats to weeks in cats to months in humans; thus, the first evidence of cortical motor outflow begins around P25 in rats, P81–P90 in cats, and 3–6 months in humans.

In human fetuses, corticospinal axons first reach the cervical spinal cord by 24 weeks postconception, and activation of M1 neurons using transcranial magnetic stimulation elicits movements in preterm and full-term newborns [26–28]. However, as in rats and cats, the onset in humans of anatomical or functional connectivity does not constitute evidence that M1 contributes to motor behavior [15,29].

In humans, compelling evidence of limited cortical motor outflow comes from infants who incurred brain damage from perinatal stroke, the leading cause of cerebral palsy [30–33]. In contrast to the immediate and often devastating paralysis that follows cortical stroke in adults,
similar strokes around birth do not produce immediate paralysis or any other detectable motor
disability. In fact, the disabling effects of cerebral palsy typically do not appear until at least
6 months after birth [34,35].

Microstimulation studies, such as those performed in rat pups and kittens (Figure 2), cannot be
performed in human infants to assess the development of cortical motor maps. Instead, re-
searchers rely on neural imaging, such as functional near-IR spectroscopy (fNIRS). For example,
fNIRS shows diffuse, rather than topographically precise, M1 motor maps as human infants reach
for an object or step on a motorized treadmill [36]. When reaching at 6 months, M1 activation pat-
terns are diffusely organized (i.e., not topographically precise); activation at 12 months is less
diffuse, but still more diffuse than in adults. During stepping, M1 activity at 6 and 12 months is as
diffuse as that of 6-month-olds during reaching. As with rat pups and kittens, the absence in
human infants of topographically precise motor maps provides converging evidence of
protracted development of cortical motor outflow.

In summary, rats, cats, and humans show protracted M1 development. Initially, M1 is incapable of
producing movement. When motor outflow from M1 begins to emerge, activation thresholds are
high and motor maps are not yet organized. In contrast, long before (and after) the emergence of
cortical motor outflow, brainstem networks can organize and implement complex motor behaviors.

**Development of cortical control of eye movements is also protracted**

When human adults respond to visual stimuli, a synergistic network of cortical and subcortical
structures supports saccadic and pursuit eye movements [37–40]. Visual input is conveyed se-
quently from primary visual cortex (V1) to the parietal eye field and then to the frontal eye field,
a region in the frontal cortex that abuts M1; the frontal eye field also receives input from the dorso-
lateral prefrontal cortex (PFC). Accordingly, the frontal eye field integrates visuospatial information,
short-term spatial memory, and decision processes, and it sends projections to the superior
colliculus, whose neurons project to the oculomotor nuclei. Other cortical regions involved in visual
processing, including the parietal eye field and V1, also project to the superior colliculus.

In human newborns, the components required for the cortical control of eye movements are not
yet established. In addition to the substantial postnatal development of V1 itself [41–43], horizont-
al connections from the output layers in V1 to secondary visual cortex do not appear to develop
until after 4 months of age [43], suggesting that V1 cannot yet influence eye movements via the
parietal eye field, the frontal eye field, or the dorsolateral PFC. Furthermore, the white matter tracts
that connect parietal cortex with the frontal eye field and dorsolateral PFC are not evident until 3 or
more months of age [44,45]. In fact, one of these tracts – the superior longitudinal fasciculus – is
among the slowest-developing tracts in the infant brain.

Developmental analyses of the event-related spike potential in parietal cortex provide converging
electrophysiological evidence for the protracted development of V1’s ability to influence the
downstream structures that control eye movements. In adults, this spike potential reliably
precedes the onset of planned saccadic eye movements. But this spike potential is absent in
6-month-olds, and its amplitude is not yet adult-like even in 12-month-olds [46]. Moreover, the
development of covert visual attention further supports the notion that control of eye movements
by cortical structures downstream of V1 emerges between 3 and 6 months of age [47–49].

The process by which infants learn to reach for objects provides additional evidence that the ability
to convey visual information to downstream structures is developmentally protracted. In human
adults, reaches and grasps are mediated by separate cortical pathways, both of which begin in
visual cortex and terminate in M1 [50]. Infants’ first successful reaches (defined as arm extensions that result in contact with the object) appear at approximately 3 months of age under the guidance of proprioceptive (not visual) feedback [51]. Visually guided reaching develops over the next 2 months [52], consistent with the emergence of horizontal connections from V1 to downstream structures [43]. The ability to actually grasp an object develops even later [53].

With minimal or absent cortical contributions to eye movements in the early postnatal period, subcortical structures must be responsible for organizing and implementing functional visuomotor behavior [41,47,54] (Box 2). However, we stress again that the absence of cortical participation in the control of movement does not imply that the cortex does nothing: Even at early ages, the cortex receives and processes modality- and task-specific input. Accordingly, as Johnson [55] put it, ‘[the] activation of visual cortical areas in the first months might have little influence over the visually guided behaviour of the infant’ (p. 770). Why might such visual cortical activity occur when it cannot influence behavior? Likely reasons include the development and maintenance of local neural circuits and the interdigitation of functionally related cortical and subcortical networks. Indeed, Johnson [55] argued that newborn looking preferences in face-detection tasks are initially supported by a subcortical foundation upon which later-developing cortical mechanisms – including those in the specialized fusiform face area – are built.

Implications of protracted cortical motor outflow for claims about cognitive development

Delayed onset of cortical motor outflow sets neurobiological constraints on what can plausibly support cognition in early human infancy. Assessments of plausibility are on a continuum that varies with infant age. Given that cortical motor outflow is absent in newborns, claims about newborn cognition that assume cortical processing should be met with skepticism. But as cortical motor outflow gradually emerges over the first postnatal year, such claims become increasingly plausible. That is, extraordinary claims about cognitive capacities in newborns might be quite ordinary when applied to 1-year-olds. Bottom line: Either the motor behaviors used to index infant cognition are unreliable or the behaviors are produced subcortically (Figure 1). Either way, given the limitations inherent in current methods, the available evidence does not support claims of developmental continuity between early infant and adult cognition.

The examples outlined below illustrate how neurobiological plausibility can inform inferences about infant cognition based on motor behavior.

Newborn orientation discrimination

In the 1980s, some developmental psychologists were convinced that the visual cortex is necessary for newborn visual discrimination. These researchers considered claims to the contrary (i.e., that visual processing in the brainstem is sufficient) as ‘doomed to founder on the rocks of a stubborn neonate who refuses to be relegated to the status of an involuntary, passive reactor’ ([56], p. 13). To demonstrate the necessity of the visual cortex, researchers chose a task – discriminating lines at different orientations – that requires engagement of orientation-selective neurons that, as assumed by some researchers at the time, exist only in the visual cortex [57]. Newborns were visually habituated to bars oriented at 45°, meaning that infants looked repeatedly at the display before turning their eyes and head away due to ‘boredom’. Then, when tested with bars oriented at 135°, newborns dishabituated to the new line orientation (i.e., they showed a recovery in looking time to the display), suggesting that their eye and head movements depended on cortical discrimination.
But if cortical control of eye and head movements is unavailable to newborns, where does that leave the assumption that the cortex is necessary for orientation-specific discrimination? In fact, based on visually evoked potentials, orientation selectivity in visual cortex begins around 6 weeks of age, suggesting that vision relies on subcortical mechanisms at younger ages [58]. Moreover, it turns out that orientation-selective neurons are not exclusive to cortex: Such neurons exist at other locations in the visual system, including the superior colliculus and even the retina [59].

Neonatal imitation
For decades, researchers have argued passionately about whether human neonates can imitate facial expressions – stick out their tongue, for example, after seeing an adult do the same [60–62]. Some labs find behavioral evidence of neonatal imitation, but others do not [63]. Why the controversy? One reason is that imitation is not trivial for a newborn: It requires the conversion of a visual stimulus into a sensory representation in body space followed by the production of a complementary motor response. None of this is easy for newborns, given their poor visual acuity and limited experience linking visual, proprioceptive, and motor systems.

Controversy aside, some researchers propose cortical maps as contributors to neonatal imitation [64]. For cortical maps to support an imitative act such as tongue protrusion – the behavior most reliably associated with neonatal imitation – neurons in V1 must activate neurons in motor cortex, resulting in the transmission of motor signals via the corticobulbar tract to the tongue muscles that produce protrusion. But such long-range horizontal connections from V1 are not available to the newborn [43]. Moreover, some researchers go further and invoke cortical mirror neurons to explain newborns’ purported imitative abilities [65]. This last claim requires functional motor outflow from the neonatal motor cortex and functional outflow from mirror neurons to motor cortex. Evidence for neither exists.

In fact, cortical control of the tongue exhibits protracted development, and tongue protrusion (and retraction) in infants is connected in complex ways with the development of suckling, breathing, swallowing, and eating solid food [66]. Moreover, tongue protrusion is initially produced spontaneously, showing its highest spontaneous rates at birth before declining over the next 3 months [67].

Thus, consideration of cortical motor outflow further supports the notion that neonatal imitation is not the same sort of imitation produced by older children and adults [61,62]. Accordingly, to explain neonatal imitation, researchers should look to subcortical structures, including the superior colliculus [56] (Box 2). As with face processing in newborns [55], an appeal to subcortical mechanisms does not make a phenomenon less interesting. Rather, such an appeal simply points to more plausible neurobiological mechanisms.

Neonatal “crawling” in response to speech
Newborn arm and leg movements are grossly uncoordinated compared with the limb movements required for crawling, cruising, and walking in older infants [68]. The spinal cord, which contains the complex circuitry to enable precisely timed limb alternation in vertebrates [69], has received the bulk of attention from researchers focused on the neural basis of locomotion across human development [70,71]. However, some researchers asked whether supraspinal mechanisms can modulate newborn limb movements by assessing behavioral responses to visual [72] or auditory [73] stimuli. For example, using a mini-skateboard designed to enable newborns to move their arms and legs in a prone posture, researchers found that newborns born to French mothers moved more in response to French speech than to English [73]. Because the researchers discounted the ability of brainstem mechanisms to discriminate French from English,
they concluded that ‘typically developing newborns possess cortical networks ready to recognize their native language’ (p. 4).

However, if cortical motor outflow is not available to support the observed movements, neonates must use subcortical mechanisms to discriminate French from English and convert that discrimination into movement. In fact, speech stimuli, including phonemes, are differentially processed within the brainstem [74]. Thus, it is plausible that neonates can discriminate native and non-native speech using subcortical mechanisms alone. Or, it is possible that the neonatal limb movements were overinterpreted: Indeed, the researchers failed to replicate the effect in newborns born to English-speaking mothers.

**Numerosity in 3-month-olds**

To investigate newborn perception of abstract number, researchers used a cross-modal matching task in which infants were familiarized to auditory stimuli composed of the same syllables repeated four or 12 times [75]. Then infants were tested with visual arrays composed of four or 12 objects. Infants familiarized to four syllables looked longer at the array of four objects, and infants familiarized to 12 syllables looked longer at the array of 12 objects, suggestive of ‘abstract numerical representations at the start of human life’ (p. 10384).

In human children and adults, and in non-human animals, the intraparietal cortex is involved in the perception of numerosity [75]. Because 3-month-olds activate this area of the cortex when detecting a change in the number of objects in a visual array [76], researchers suggested that ‘this parietal sensitivity arises from a predisposition of parietal cortex for spatial and numerical transformations, possibly present since birth’ (p. 275). However, it is neurobiologically implausible that intraparietal cortex contributes to the behavioral expression of numerosity in 3-month-olds, let alone since birth. But if intraparietal cortex is not involved in the looking behavior in this task, how can its early activation be explained when 3-month-olds detect a change in the number of objects in a visual array? One possibility is that this early activation reflects feedforward input from the subcortical structures responsible for the behavior, similar to what was proposed for the early development of face perception [55].

**Social evaluations in 3-month-olds**

Adults routinely evaluate other people’s proclivities to help or to harm. But do babies? For example, after watching a ‘helper’ character (a geometric shape with eyes) ‘assist’ another character up a hill and a ‘hinderer’ character ‘thwart’ the other character’s efforts to go up the hill, 6- and 10-month-old infants reached more frequently for the helper character [77], leading the authors to conclude that this preference ‘may serve as the foundation for moral thought and action’ (p. 557). These findings were subsequently extended to 3-month-olds using preferential looking, in which infants turn their eyes and head to look at a display [78].

Can we conclude that 3-month-olds have an ‘innate moral core’ [3]? The answer to this question depends on whether 3-month-olds can make social evaluations – presumably dependent on cortical structures – and then use those evaluations to execute the required eye movements. Given that cortical mechanisms for controlling motor behavior are unavailable to 3-month-olds, two other possibilities must be considered: either that infants’ social evaluations are enabled initially by subcortical structures (and are thus not continuous with those of adults) or that concerns about whether the findings truly reflect social evaluation – as opposed to low-level perceptual discrimination – should be given more credence [79].
High-level infant cognition
The PFC is a computationally complex network implicated in high-level cognitive processes, such as working memory, decision-making, and motor planning [80]. Given the centrality of the PFC for human cognition, researchers asked when and under what conditions the PFC first develops its functional capabilities [81–83]. For example, using a task that taps into the ability to ‘hold a goal in mind’ when an object is no longer visually accessible, researchers showed that the performance of human infants improves substantially from 7.5 to 12 months of age [84]. Indeed, performance at 12 months of age was similar to that of adult rhesus monkeys, and the performance of monkeys with lesions of the dorsolateral PFC was similar to that of human infants at 7.5 to 9 months of age. Additional support for protracted PFC development comes from consideration of that structure’s functional codependence with the cerebellum, which is also a late-developing structure [85–87].

In contrast, other researchers argue that the PFC contributes to higher-order cognition at birth, enabling newborns to make active choices about where they direct their attention and how they experience the world [88]. Noting the newborn’s limited ability to reach, grasp, and point, the researchers claim that the cognitive contributions of the PFC are evidenced by infants’ ‘control over their gaze and visual attention from the first hours after birth’ (p. 251). To the contrary, there is no evidence that the newborn PFC can influence visuomotor behavior.

Finally, to further support claims about a functional newborn PFC, researchers point to neuroimaging evidence of an adult-like frontoparietal network in human infants before 1 month of age [89]. But again, evidence of early complex organization does not bear on the network’s ability to produce behavior. Indeed, it is more likely that subcortical mechanisms provide structured input to the newborn PFC long before it can actively contribute to the expression of behavior.

Concluding remarks
Large gaps remain in understanding the development of cortical and subcortical motor control, particularly in human infants (see Outstanding questions). For now, we suggest humility when making claims about when and how the cerebral cortex translates cognition into action. Likewise, we suggest caution in asserting that human newborns possess core knowledge, especially if that knowledge depends on a supporting neural architecture that is developmentally continuous with that of adults [1,90]. By carefully considering neurobiological plausibility across age, developmental psychologists will be better positioned to design experiments in which questions, tasks, and interpretations are suitably matched. Perhaps, then, the field can advance on surer footing and avoid needless controversies.

At birth and for several months thereafter, the brainstem is largely responsible for motor behavior in human infants. But the brainstem does not simply hand off responsibility to the cortex and fade away. Instead, development entails the gradual interdigitation of cortical and subcortical networks into a functionally integrated whole. Current imaging methods are not yet able to capture this developmental process and thus cannot address the issue of developmental continuity in infant cognition and behavior.

Imaging studies in infants do reveal cortical activity suggestive of area-specific processing of relevant information. But evidence of cortical processing is not evidence of cortical involvement in motor behavior, even when the cortical processing fits with researchers’ adult-centric expectations. Indeed, in infant rats, individual neurons in M1 increase their activity only after the...
production of movement, indicative of sensory processing [9] (Box 1). Now consider the likely interpretation of this observation had the M1 recordings been performed using fMRI, a method with poor temporal resolution compared with single-cell recordings? Given that M1 is so closely tied to adult motor control, it is likely that researchers would have inferred erroneously that any fMRI-detected increase in M1 activity contemporaneous with movement reflects the role of M1 in the production of that movement. Similar problems confront researchers as they investigate cortical processing and cognition in human infants.

To conclude, researchers should be aware that behavioral indicators of cognitive sophistication may not reconcile with the neurobiological mechanisms available to support the behaviors. Ultimately, a complete and accurate account of the origins of human cognition requires greater understanding of the complementary relations among cortical and subcortical circuits and how those relations emerge across early development to support motor behavior and cognition.

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Declaration of interests

The authors have no interests to declare.

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