Trends in Cognitive Sciences

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Letter

Infant action and cognition: what's at stake?

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In two independent responses to our opinion article [1], Liu *et al*. [2] and Aslin *et al.* [3] (henceforth Liu or Aslin) critiqued our argument that the protracted development of motor cortex in mammals constrains rich interpretations of infant cognition. We welcome this opportunity to clarify what's at stake. One issue concerns the neural basis of cognition across early development and the evidence researchers rely on to reveal it. Another issue concerns 'core knowledge' and whether representations of numerosity, moral reasoning, and the like are developmentally continuous between infant and adult minds. In addition to addressing these issues, we chart a path forward by placing the dispute surrounding our piece into a developmental-comparative framework.

Regarding the neural basis of cognition, the argument in our opinion article was built on a straightforward observation. Primary motor cortex (M1) functions initially as a sensory structure and only begins to transition to its identity as a motor structure relatively late in postnatal development – around 25 days in rats, 2 months in cats, and ≥6 months in humans. The surprising sensory origin of M1 provides a cautionary tale for understanding cortical development in general: even M1, with its seemingly transparent function, does not conform to adult-centric assumptions.

Current recording and imaging methods in human infants are limited in their ability to reveal causal links between brain activity and behavior across age. These limitations are reflected in Aslin's repeated use of noncausal language ('mirrored by', 'are consistent with') to describe the link between, for example, looking time and EEG activity. In non-human animals, the standard approach for determining the onset of cortically mediated movement is to stimulate M1 at different ages and observe whether movement is produced [4–6]; researchers can also infer causation by determining whether M1 activity reliably occurs before (i.e., motor) or after (i.e., sensory) a movement. Based on such evidence, M1 does not begin to produce movement until around 25 days of age in rats. Aslin objected that our evidence for the late onset of cortical motor outflow rested on 'rodents rather than primates', but we presented converging evidence from cats and humans. Here, we provide additional converging evidence from macaques [6] (Box 1).

In our opinion article, we described how subcortical input develops and refines basic features of cortical organization. For example, sensory input shapes the development of the earlier-emerging sensory map in M1, laying the foundation for the later-emerging motor map [7]. In this way, sensory input aligns somatotopic maps across brainstem, thalamic, and cortical structures. Nonetheless, our description of early cortical activity was mischaracterized as 'epiphenomenal' (Liu) and our description of cortical and subcortical activity was mischaracterized as 'independent' and emerging 'in parallel' (Aslin). Such terms are antithetical to our emphasis on the profound developmental influence of ascending input on cortical activity and development.

Perhaps most objectionable to Liu and Aslin, our opinion article leveraged insights about the protracted development of motor cortex to encourage caution when considering the neural substrates of infant cognition. Aslin stated that we 'cite[d] only one study from humans relevant to [our] thesis', but this is incorrect. In fact, we discussed several lines of converging evidence from studies with human infants. Moreover, although we considered movements across the body – eyes, face, head, limbs, and fingers – Liu and Aslin focused almost exclusively on the eyes, reflecting the centrality of looking-time measures to research on infant cognition. However, Liu and Aslin did not rebut our evidence that, as a general matter, the development of cortical motor outflow is protracted. Further, Liu asserted that the available data 'are consistent with gradual, continuous change', and their Figure 1B represents motor–cortical outflow as beginning during the prenatal period.

Aslin encouraged us to provide a productive path forward. We believe such a path requires ground rules for assessing the plausibility of claims about human development. When placed in a developmental–comparative context (Box 1), humans fit within the mammalian pattern [8]. Moreover, within this context, evolution constrains what is developmentally plausible. Acknowledging that humans are not rats, cats, or macaques does not give researchers free rein to draw broad conclusions about the timing of events in human development.

Clearly, cortical motor outflow from M1 starts at some point along the developmental timeline. In rats, cats, and macaques, for which clear data exist [4–6], M1 stimulation fails to produce a movement at one age, after which stimulation is increasingly effective. Moreover, the age at which stimulation begins to elicit movement occurs at a similarly late developmental stage in these three species (Box 1).

In humans, there is no *a priori* reason to expect accelerated development of cortical motor outflow relative to these other species. In fact, studies in human infants using transcranial magnetic stimulation (TMS) converge on a conclusion of

Box 1. Assessing human M1 development through a comparative lens

Much is known about the developmental sequence and timing of neural and behavioral events across species [8]. For four species, Figure I shows estimated postconceptional age in days plotted against an 'event scale' comprising 271 developmental events. The upward accelerating plot for humans reflects the longer time required to complete all 271 events compared to the other species. The semi-log replotting of the data shows that the developmental trajectories for all four species are exponential.

Three additional events are shown. Birth occurs at similar points along the event scale in humans, cats, and rats but is relatively delayed in macaques. Onset of cortical delta, a slow rhythm whose emergence is a milestone in cortical development, is closely aligned on the event scale for humans, cats, and rats [11–13]; because delta emerges prenatally in macaques [14], it is not represented.

Similarly, based on stimulation studies [4–6], the onset of M1 motor outflow is closely aligned on the event scale in rats, cats, and macaques. Given these data, where on the event scale would one predict the onset of M1 motor outflow in humans? We indicate five possible onset times – from arrow a that corresponds with the non-human species to arrow e that corresponds with prenatal onset. From arrows a to e, it is increasingly unlikely that M1 onset in humans occurs at that age. Based on this analysis, it is unlikely that M1's ability to influence movement begins before 6 months of age, which is consistent with TMS studies in human infants [9].

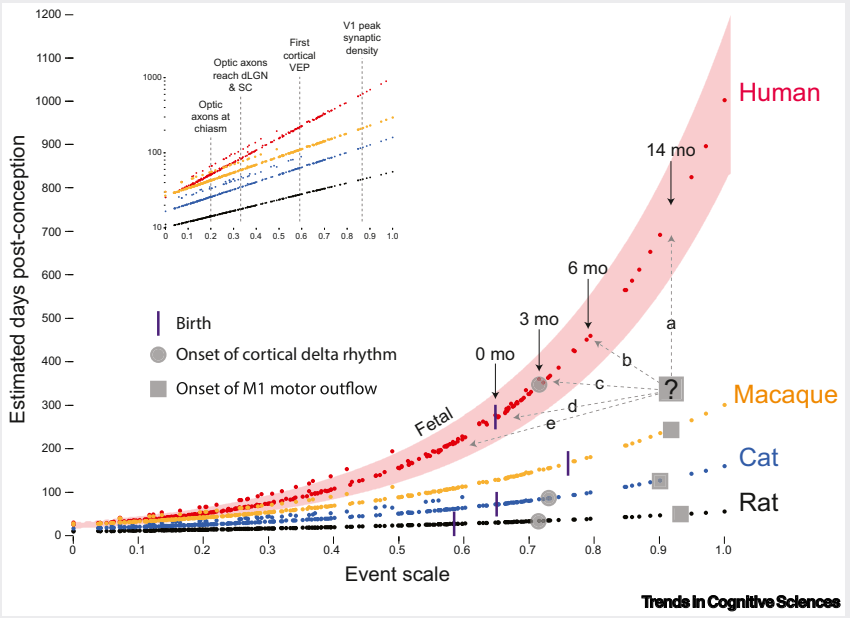

Figure I. Estimating the onset of M1 motor outflow in early human development. Estimated days postconception plotted against event scale for humans, macaques, cats, and rats (data from translatingtime.org). Red-shaded region denotes 95% confidence intervals for human data. Three additional events are shown: birth (vertical bars), estimated onset of cortical delta rhythm (gray circles), and estimated onset of M1 motor outflow based on cortical stimulation studies (gray squares). Based on M1 onset in the non-human species, five possible onsets in humans are represented (arrows a–e). Inset: semi-log replotting of the data shown with four events from the visual system. Abbreviations: dLGN, dorsal lateral geniculate nucleus; SC, superior colliculus; V1, primary visual cortex; VEP, visual evoked potential.

protracted M1 development [9]. In newborns, although TMS evokes action potentials in the arm muscles – indicative of functional corticospinal innervation – it does not produce movement. Also in newborns, TMS activation thresholds are high; conduction times from cortex to muscle are long, decreasing substantially between 6 and 18 months. Nonetheless, such evidence does not reveal when functional cortical control begins for the limbs, let alone the eyes. In our opinion article, we reviewed indirect evidence that eye-movement control emerges between 3 and 6 months. Unfortunately, we found no cortical stimulation studies (in any species) that address eye movements, and so we cannot know definitively if the development of eye-movement control is accelerated relative to the limbs; such studies are needed. In the meantime, the available stimulation data and comparative data do not support Liu's assertions that cortical influence over behavior in humans begins prenatally and develops continuously thereafter.

With regard to the developmental continuity of core knowledge, Pinker famously argued that '…the null hypothesis in developmental psychology is that the cognitive mechanisms of children and adults are identical; hence it is a hypothesis that should not be rejected until the data leave us no other choice' [10]. In their defense of developmental continuity, Liu and Aslin appear to align themselves with this philosophical stance. Regardless, given developmental discontinuities in the brain mechanisms upon which the expression of infant cognition relies, why assume developmental continuity between infant and adult minds? The available evidence leaves us no choice but to reject Pinker's null hypothesis.

Finally, Aslin expressed an ancillary concern that our critique about overly rich inferences drawn from looking-time experiments in infants younger than 4 months of age might be inappropriately generalized to older ages, thus sending 'a chilling message' to a field that relies largely on this method. We believe our title's invocation of 'rich interpretation' points the reader to the true target of our concern. Nonetheless, to drive his point home, Aslin notes that 'only a single study on moral development' was conducted with infants under 4 months of age. However, to be fair, that single study

laid the foundation for the claim that moral reasoning is innate and was cited over 1900 times between its publication in *Nature* in 2007 and the writing of this response. Whereas Aslin is concerned about the chilling effect our paper may have on the perceived value of looking-time studies in older infants, we are concerned about the chilling effect that rich interpretation has already had on developmental science.

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