

Review

An Alternative to Domain-general or Domain-specific Frameworks for Theorizing about Human Evolution and Ontogenesis

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Abstract: This paper maintains that neither a domain-general nor a domain-specific framework is appropriate for furthering our understanding of human evolution and ontogenesis. Rather, as we learn increasingly more about the dynamics of gene-environment interaction and gene expression, theorists should consider a third alternative: a domain-relevant approach, which argues that the infant brain comes equipped with biases that are relevant to, but not initially specific to, processing different kinds of input. The hypothesis developed here is that domain-specific core knowledge/specialized functions do not constitute the start state; rather, functional specialization emerges progressively through neuronal competition over developmental time. Thus, the existence of category-specific deficits in brain-damaged adults cannot be used to bolster claims that category-specific or domain-specific modules underpin early development, because neural specificity in the adult brain is likely to have been the emergent property over time of a developing, self-structuring system in interaction with the environment.

Keywords: domain-specific; domain-general; domain-relevant; neuroconstructivism; emergent structure; evolution

1. Domain-specific vs domain-general frameworks

The Nature *or* Nurture debate is obviously not new. It already raged in the 4th and 5th centuries amongst the ancient Greeks. The modern version is often, although not always, couched in terms of domain-specific versus domain-general accounts of human cognition, in which some theorists argue that the infant brain comes equipped with primitive conceptual representations or domain-specific core knowledge or domain-specific learning algorithms [1–5], while others invoke more domain-general learning mechanisms without the need for representational content [6–8]. Yet, despite the fact that most scientists now fully recognise that evolution and ontogenetic development must derive from very complex interactions of Nature *and* Nurture, the dichotomy continues in the

21st Century to influence arguments about the structure of the human mind/brain [9], how it evolved [10], and how to interpret data such as category-specific or domain-specific deficits in children and adults [11–13]. Is the infant brain a undifferentiated tabula rasa on which experience imprints itself via (innate) general learning mechanisms, or does the infant brain come with innate, specialized knowledge upon which further domain-specific learning takes place? Caution is required, however, because opting for the innately-specified perspective can, as Bates and colleagues [14,15] pointed out, lead to conflating innateness with: (a) *domain specificity* (outcome X is so singular that it must be innate), (b) *species specificity* (humans are the only species who do X, so X must be specified in the human genome), (c) *localization* (outcome X is mediated by a particular brain region, so X must be innate), and (d) *learnability* (we cannot establish how X could be learned, so X must be innate).

The existence of brain-damaged adult patients presenting with uneven neuropsychological profiles and very specific deficits led many researchers to divide the mind/brain into separate modules for number, face processing, space, semantics, syntax and so forth, each cognitive domain being processed within a specialized region of the brain, characterized as either intact or impaired. This view is of course not one endorsed by all neuropsychologists. However, modular, domain-specific explanations have not been confined to neuropsychological patients. The concept has also been generalised to studies of typically developing infants/children by researchers of a nativist persuasion [1,2,16–19]. Rather than being accounted for by plasticity and an early capacity for learning, competences detected within the first months of life have often been explained in terms of built-in core knowledge/conceptual representational primitives, i.e., infants are born with innately-specified modules or sub-modules for processing specific types of input from each domain: number processing modules [20–22], face-processing modules [23–26], grammatical sub-modules [5], spatial cognition sub-modules [27], and modules containing core knowledge of the constraints governing the physical world [18]. Within this framework, learning was initially relegated to a very secondary role [28]. However, as the importance of learning has increasingly come to the fore, researchers of this persuasion have invoked conceptual reorganization or the role of language in extending domain-specific core knowledge of infant sensitivities to different cognitive domains into more abstract, adult-like knowledge [1,29].

Furthermore, one brand of so-called evolutionary psychology [10,30,31] makes similar domain-specific claims, arguing that the relationship between our ancestral past and the human brain today can be understood in terms of innately-specified, cognitive-level modules passed on through Evolution (see discussion in Bolhuis et al., 2011 [32]), e.g., a cheater detection module, a grammar module, a face-processing module, a number module, and so forth, each coded for by specific sets of genes. Under this view, the brain is conceptualized in terms of the metaphor of a Swiss army knife, each tool (cognitive domain or sub-domain) being exquisitely fashioned and dedicated solely to carrying out a set of very circumscribed tasks, passed on by evolution from our hunter-gatherer ancestors.

Some researchers studying neurodevelopmental disorders in children have also proposed a strictly modular explanation. Among the first to take this position were Baron-Cohen, Leslie, Frith and colleagues who claimed that autism could be accounted for by the lack of or damage to a theory-of-mind module [33,34], due to the mutation of a specific gene or set of genes which interfered with the development of a specific brain region, the orbito-frontal cortex [35]. This region was dedicated, it was argued, to computations dealing with the attribution of intentional states to

others, an ability that is compromised in autism. Rapidly the explanatory framework of damaged versus intact modules or sub-modules was generalised to a vast number of other neurodevelopmental disorders [12,36–41], particularly those which present with uneven cognitive profiles, such as dyslexia [42], Specific Language Impairment [4,5,43], Williams syndrome [12], developmental dyscalculia [21,36], and developmental prosopagnosia [23].

In summary, early competences identified in typically developing infants, deficits found in brain-damaged adults or in children with genetic disorders, as well as arguments from evolutionary psychology, all seemed to corroborate the claim that the domain-general framework could not explain early competences nor atypical uneven cognitive profiles and that, instead, the human mind/brain had to be conceived of as being composed of highly specialized input/output systems, at both the perceptual and cognitive levels. So, why not indeed endorse this domain-specific framework? It would seem to account well for those typically developing children who show strong proficiency in, say, numeracy and relative weakness in literacy, or vice versa. At first blush, it also seems to be the appropriate explanation for adult neuropsychological patients presenting with domain-specific or category-specific deficits. Likewise for atypically developing children: the domain-specific framework would seem to offer a plausible explanation for the existence of children with autism who may present with high abilities in, say, mathematics but are incapable of dealing with the simplest of aspects of social interaction, or those with Williams syndrome who speak fluently but cannot do simple additions. How could a domain-general view explain such specificities? Indeed, the existence of such uneven cognitive profiles is well established. So, is the only alternative to the domain-general approach that of opting for innate, domain specificity as the most appropriate explanatory framework for understanding the roots of human cognition? Not necessarily, because the domain-specific framework is itself questionable. Crucially, it ignores the developmental history of the organism.

2. Neuroconstructivism and the *domain-relevant* framework

So, are there only two alternatives—domain-specific/domain-general—for theorising about human evolution and ontogenesis? I argue that there is a third alternative: a *domain-relevant* framework—one which would likely be endorsed by several theorists working within a more domain-general framework. A domain-relevant approach to understanding specialization in adult brains argues that the brain starts out with a number of basic-level biases each of which is somewhat more relevant to the processing of certain kinds of input over others, but which only *become* domain-specific over developmental time. This occurs, it is argued, through a process of neuronal competition and gradual specialisation, location and modularisation of function—a neuroconstructivist approach [6,44–48]. Neuroconstructivism does not believe that the infant brain is an undifferentiated tabula rasa upon which experience simply imprints itself. Nor does it rule out domain-specificity; it argues that, when one discovers specialised functions in the adult brain, it cannot be taken for granted that the same automatically holds for the start state of the infant mind/brain [45,49,50]. Rather, neuroconstructivism maintains that domain-specificity, i.e., functional specialization, is the emergent outcome of developmental processes rather than the start state.

It is within the neuroconstructivist account of development [6,44–46,48,51–53] that domain-relevant approaches have been put forward, particularly with respect to neurodevelopmental disorders. These latter are deemed to be explicable at a very different level from the intact/impaired

domain-specific cognitive modules often invoked. Rather, atypical phenotypic outcomes are argued to be rooted in perturbations in far more basic processes early in development, such as a lack of/over-exuberant pruning, or differences in synaptogenesis, dendritic growth, in the density/type of neurons, in firing thresholds, in poor signal to noise ratios, or generally in terms of atypical timing across developing systems. In other words, from a neuroconstructivist viewpoint, adult brain specialization *emerges developmentally* [6,44,45,51,54]. In this sense, I would argue that domain-specific outcomes may actually be impossible in the absence of a gradual process of development over time.

But why shouldn't the scientist consider the infant brain as starting out highly specialized with built-in conceptual knowledge, as some leading theorists maintain [2,3]? This is because multiple facets of early human development point instead to a flexible, plastic, self-structuring system, open to extensive environmental influences at the level of gene expression, brain, cognition and behaviour. Indeed, developmental change is the rule at every level, not the exception. Four examples serve to illustrate this: 1) cortical areas are initially much more highly interconnected in the infant brain than in the adult brain [52,55,56], and it is only progressively over time, with the strengthening of some connections and the pruning of others, that localisation and specialisation of brain function occur [51]; 2) the ratio of white matter to gray matter is not static; it changes over developmental time [57]; 3) the thickness of fibre bundles in the corpus callosum between the two hemispheres is different in infancy compared to later brain development [58]; and, 4) studies of neural processing of faces or language, for instance, reveal that early on in development neural activity is widespread in several cortical regions across both hemispheres. It is only later in development that neural activity becomes progressively fine-tuned more predominantly to one hemisphere (RH for face identification; LH for grammar production and phonemic processing) [51,59–65]. Indeed, as Stiles stresses [65] decades of cross-sectional and longitudinal studies of children with peri-natal focal lesions (reviewed in Bates & Roe, 2001[66]; Stiles, 2012 [65]) have shown over developmental time only very transient relationships between specific lesion sites and language deficits. Furthermore, such relationships turn out to be very different from those present in adult aphasic patients. As Stiles points out, it turns out that there is no evidence of long-lasting language delays associated with left versus hemisphere perinatal focal brain injury [65].

These four developmental examples clearly indicate that the microstructure of the brain is neither pre-specified nor static. Indeed, cortical networks are not genetically determined, to be spared or impaired in genetic disorders. Rather, they are the emergent outcome of progressively changing neural function and structure [67–69], which dynamically interact with one another and with environmental input, ultimately giving rise to the structured adult brain. Emergent specialization of function is viewed by Neuroconstructivists as the fine-tuning of initially diffuse, domain-relevant, but coarsely coded systems, which become increasingly domain specific over developmental time.

3. Changing views on modularity

Of course, Neuroconstructivism is not alone in taking a far more dynamic approach to developmental change and to adult patient data. For development, this has been particularly true of those espousing dynamical systems accounts (e.g., Smith & Thelen, 2003 [70]), although they tend to rule out any notion of representation. For healthy ageing in adults, similar arguments have emerged about the dynamic and changing nature of neural processes with respect to grammatical

processing [71]. Modules are now considered far less encapsulated, allowing them to operate both in a relatively independent way in some circumstances, but modifiable by other processes in other circumstances (e.g., Dehaene & Cohen, 2007 [72]). However, while it is unlikely that theorists such as Spelke or Pinker would take a strictly Fodorian view of modules [73], their current theorizing nonetheless continues to invoke innate specification of highly specialized systems [5,18], a very different approach from that of Neuroconstructivists.

Dehaene and collaborators' recent work ([72,74], see, also, [75]) has several features in common with the domain-relevant approach. The neuronal recycling hypothesis argues that cultural inventions like reading and writing, which are too recent to be genetically determined, invade evolutionarily older cortical circuits, initially devoted to different but similar functions, thereby inheriting many of the structural constraints of the original circuits [76]. The notion that these new functions seek out a 'neuronal niche', i.e., a cortical area in the brain *relevant* to the processing required, is clearly along the same lines as the domain-relevant approach outlined above [45]. Dehaene argues that these novel cultural functions must identify a cortical area whose prior function is similar but also sufficiently plastic to accommodate its processing demands. A domain-general account could not accommodate these ideas because the neuronal niche must already be domain-relevant to the invading process. It also would not be accommodated by a strictly domain-specific modular approach, because the 'invasion' requires plasticity and flexibility.

Dehaene and collaborators also address the perennial problem of conscious access to knowledge, offering an impressive mathematical and neural model [74]. Here again, they argue for plasticity and neural cross-talk, relaxing the encapsulated nature of modules in favour of flexible routing and global sharing across brain regions, i.e. the flexible sharing of information throughout cortex. As the authors state: "While non-conscious stimuli are processed in parallel by specialized cortical processors, conscious perception would be needed in order to flexibly route a selected stimulus through a series of non-routine information processing stages". Global information sharing would therefore depend on a set of interconnected high-level cortical regions forming what Dehaene calls a "global workspace", i.e., a distributed network of cortical areas tightly interconnected by long-distance axons, which sends information back to specialized processors.

Dehaene's theory mainly targets the explanation of data from the adult brain. But what about developmental issues, the focus of this paper? Some time ago I proposed the Representational Redescription Hypothesis, arguing that two parallel processes underlie human cognitive development [44]. Based on the notion of domain relevance, the first concerned how the child brain becomes increasingly specialized/automatized in processing of different kinds of stimuli. Instead of modules, I argued for a gradual process of relative modularization over developmental time, i.e., functional specificity was viewed as the outcome of this process. However, to allow for conscious access to the same knowledge, I argued for a second, parallel process: representational redescription, i.e., representations embedded *in* brain processing that are implicit to the organism are redescribed, and thereby become knowledge *to* the brain, with the redescrptions from one domain becoming transportable to other domains (see details and supporting developmental data in Karmiloff-Smith, 1992 [44]). I would now argue that this process is likely to occur via brain activity during sleep. In summary, each of the two parallel processes plays a different role in development: the first increases automaticity, the second makes redescribed representations available to conscious access without interfering with the automaticity of rapid processing.

4. Genetics within the Neuroconstructivist Framework

The human genome contains a large number of transcription factors, whose function it is to turn on and off the functions of other genes. Given the complexity of a fully-formed brain (over 100 billion neurons, each connected to up to 10,000 others), and the dearth of genetic material available to make a brain (some 30,000 genes, at most), DNA clearly does not/cannot provide a total blueprint for the microstructure of the brain. Rather, it provides a sufficient outline to kick-start development with, for instance, higher than necessary connectivity levels within and between cortical regions [56]. Generally, the newborn cortex does not display strict localization or specialization of function [77]; rather, these *emerge* as an activity-dependent function of interactions at the cellular, neural, cortical, and environmental levels [45,46,51,52,78–81,]. Near and distant brain regions not only become progressively *co-active* but also gradually functionally *cooperative*, leading to a dense brain network with functionally connected, multiple brain regions [82]. The shift from distributed to more localized processing is a developmental process that unfolds over many months or even years, well into adolescence [83], with other processes revealing a pattern of local to global change [84], i.e., various forms of neural change are the rule across developmental time. Thus, the adult's relatively specialised cognitive architecture can be thought of in terms of gradual emergence over time, but the starting point is neither predetermined nor fixed [45,85,86].

Indeed, to date there little if any evidence to suggest tight functional specificity of gene expression in the brain, i.e., that genes which are expressed in the brain target from the very start discrete cognitive-level cortical circuits. Rather, gene expression in the brain changes from initial widespread, diffuse, large-scale gradients across cortex [87], only later progressively restricting expression to more specialized cognitive-level circuits, although genetic control of primary areas seems to occur very early in development [88]. Thus, genetic mutations contributing to developmental disorders in infants are likely to affect widespread systems within the brain, some more seriously/more subtly than others, depending on their domain relevance [45]. This does not preclude that the outcome of the dynamic developmental processes could result in some areas being more impaired than others, due to the processing demands of certain types of input to those areas as well as to differences in synaptogenesis across various cerebral regions [56].

It is important to recall that gene expression in the brain (when a gene is expressed during day/night or across development, how much protein product is expressed, and in which brain region[s] it is expressed) is not strictly predetermined, but also a function of environmental experience over developmental time. Studies of rodents illustrate this point, highlighting the potential role of the environment in shaping patterns of gene expression [89]. The researchers tracked brain development in rodent pups and showed that differences in the amount of maternal postnatal pup grooming/stroking actually changed the amount and location of the expression of genes involved in the brain's responses to stress, which had lifelong effects on the animal's behaviour. Such dynamic environment-gene interactions are likely to be pervasive in mammalian brain development, including that of humans. In general, epigenesis is not deterministic under tight genetic control. Rather, epigenesis is probabilistic and only under broad genetic control [90]. So, if the architecture of the adult brain is highly specialized, this is likely to be the end-product of very complex gene-brain-cognition-behaviour-environment dynamics [91], which give rise over developmental time to relatively domain-specific outcomes and, in the case of semantics, to category-specific specialisations. And this flexible, dynamic framework makes evolutionary sense.

5. Evolution and the Neuroconstructivist Framework

In their excellent review of comparative neuroscience, Quartz and Sejnowski (1997) [92] remark on how the degree of genetic pre-specification varies in non-random ways across species. They argue that it is highest in animals the most distant from humans, and lowest in our closest relatives. Recall that many other species have complex special-purpose responses to the environment, e.g., the spider's web, that human infants do not possess. Yet humans develop an exceedingly rich, flexible cognitive system over time. Thus, rather than endow humans with maximal pre-specified core knowledge (hyperspecialization), it seems that evolution leans instead toward increasing plasticity for learning. So, instead of being hyperspecialized, human infants are more likely born with a collection of reflexes together with some domain-general learning algorithms (for attention, discrimination, novelty detection, memory, etc.), but also some domain-relevant biases that provide not only a starting point but also an initial trajectory vector in cognitive space, one that will be affected not only by experience but also by interactions within and between levels of causal change (cellular, neural, body, environment; see Marescal et al., 2007 [46]; Westerman et al. 2007, 2010 [47,48]). It is also worth recalling that, unlike other species, humans remain totally dependent upon their social environment during a lengthy period of early development, allowing the social environment to impact significantly upon brain development.

So, instead of invoking a start state of innate, domain-specific modules handed down by evolution, the neuroconstructivist framework would argue that evolution may be driven predominantly by increased plasticity for learning [93–96], rather than increased genetic complexity, i.e., for a limited number of domain-relevant biases, which *become* domain-specific over developmental time via their competitive interaction with each other when attempting to process different kinds of environmental stimuli [45,51]. In this sense, evolution can be considered a trade-off between hyperspecialization but a lack of flexibility, on the one hand and, on the other hand, maximum (but not unbounded) plasticity with some domain-relevant constraints [44,92,97].

6. Why has the domain-specific view remained so attractive?

Why, then, does the domain-specific framework continue to be so attractive to many of those studying typical and atypical development in children? This may reside in the fact that the explanatory framework is static, thereby leading to simpler types of research strategy focused on a specific domain. Moreover, if the researcher considers the normal brain to be composed of pre-specified modules and the atypical brain to be a juxtaposition of intact and impaired modules, then the brain can theoretically be represented by a number of independently functioning modules, suggesting that impairments in one module will have no developmental effects on other specialised functions. This gives rise to a focus on the impaired domains and a mere cursory investigation of the “intact” ones. Interestingly, once such a strategy forms the basis of research on children with genetic disorders, *relative* differences (domain X is more impaired than domain Y) slip into *absolute* statements: X is impaired, domain Y intact [9,12,27,28,98,99], whereas in fact *both* are impaired, albeit to different degrees, when compared with the levels of healthy controls.

A very different approach is taken if the researcher considers the initial state of the neonate cortex to be composed of many interconnected, interacting parts. The neuroconstructivist framework would consider a genetic mutation to be initially expressed quite widely throughout many brain

regions and to affect many emerging cognitive domains, but to different degrees. What may seem like a domain-specific (or category-specific) impairment in the adult phenotypic outcome may simply be due to the fact that the neuronal and biochemical properties of one brain region are more affected by the mutation than other regions but, crucially, that these other regions may also be affected albeit to a less obvious degree. This means that the neuroconstructivist researcher will not only focus on areas of serious deficit, but pay as much attention to domains where the child displays proficient performance, even those in which the child's scores fall in the "normal range". This often leads to the identification of unusual cognitive and neural processes that underlie "normal" behavioural scores [100]. Furthermore, the neuroconstructivist researcher will systematically try to trace cognitive-level deficits back to their basic-level origins in infancy [45,101].

A lack of specialization and localization of function, also referred to as a lack of progressive modularization [44,102], leads to the speculation that the brains of individuals with developmental disorders may remain more highly interconnected than the brains of healthy controls which undergo progressive pruning over the course of development. The resulting atypical (under-pruned) brain would then be overly active across multiple brain regions resulting, for instance, in problems with multitasking [103]. This certainly holds for individuals with fragile X syndrome, whose brains have been shown to have abnormally high synaptic densities through to adulthood [56,104]. But the converse is also possible. In some developmental disorders, the brain may commit too rapidly to specialization and localization of function [53,105], resulting in less flexibility for processing novel stimuli. Such considerations, in which the time course of ontogenetic change plays a pivotal role, lead us far from the metaphor of static intact and impaired modules.

A crucial error is to conflate the specialised, mature brains of adults, which have developed normally prior to damage in later life, with those of infants and children, which are still in the process of developing [54,94,95,106]. Rather, we need to think in terms of a gradual process of recruitment of particular pathways and structures for specific functions [6], such that brain pathways that were previously partially activated in a wide range of contexts increasingly confine their activation to a narrower range of inputs and situations [107]. Importantly, to fully comprehend typical and atypical developmental pathways, the process of ontogenetic change must always constitute a fundamental part of the explanation of human cognition. The brain is not static; it undergoes extensive developmental changes over ontogenesis [51,57,67,68,].

Finally, it remains an open question as to whether even the mature adult brain is as strictly modular as some theorists would claim, particularly with respect to the cognitive level, since changing interactivity of dynamic cerebral networks seems to be the case throughout life. This is nicely illustrated by neural studies of healthy ageing adults whose behavioural scores on, say, syntactic comprehension tasks are identical to those of younger adults but whose ageing brains approach the language task bilaterally compared to the predominantly left-hemisphere specialisation of the younger adults ([71]; see, also, [76]).

7. Concluding thoughts

So, theorists have several options to explain the start state of the human brain and its subsequent development, amongst which the four frameworks discussed in this paper: (1) the domain-general framework of an initially undifferentiated brain which gains its structure solely from interacting with the environment; (2) the core knowledge or conceptual representations framework where the brain

starts out with very domain-specific knowledge upon which more abstract knowledge is built through, *inter alia*, language or conceptual reorganization; (3) the neuronal recycling, plasticity/flexibility framework, built on domain-specific starting states, but open subsequently to substantial cross-domain interactions; or, (4) the neuroconstructivist framework, together with a process of representational redescription, which invokes domain-relevant—not domain-specific—biases to kick-start development, as well as initial neuronal competition and gradual emergent functional specialization.

Although plasticity is seen by some as a response solely to injury, for many neuroscientists nowadays, plasticity is the rule for development, normal or atypical [32,66,74,108–110]. But plasticity is not, of course, unconstrained. As Hensch and collaborators [111,112] have elegantly demonstrated in a series of molecular studies on the excitatory-inhibitory balance across brain regions and systems that place constraints on plasticity, it would make no sense in evolutionary terms to have unbounded plasticity. A developing organism needs to be both constrained and flexible [44,113], and achieving that dynamic balance is what ontogenetic development is all about.

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Conflict of Interest

The author declares no conflict of interest.

References

1. Baillargeon R, Carey S (2012) Core cognition and beyond: The acquisition of physical and numerical knowledge. In S. Pauen (Ed.) *Early childhood development and later outcome*. Cambridge, England: Cambridge University Press, 33-65.
2. Carey S (2011) The Origin of Concepts: A Precip. *Behav Brain Sci* 34: 113-162
3. Spelke ES, Kinzler KD (2007) Core knowledge. *Dev Sci* 10: 89-96.
4. van der Lely HKJ (2005) Domain-specific cognitive systems: Insight from grammatical specific language impairment. *Trends Cog Sci* 9: 53-59.
5. Van der Lely JKJ, Pinker S (2014) The biological basis of language: insights from developmental grammatical impairments. *Trends Cog Sci* 18: 586-595.
6. Elman JL, Bates E, Johnson MH, et al. (1996) *Rethinking Innateness: A Connectionist Perspective on Development*. Cambridge, Mass: MIT Press.
7. Kirkham NZ, Slemmer JA, Johnson SP (2002) Visual statistic learning in infancy: evidence for a domain-general learning mechanism. *Cognition*, 83: B35-B42.
8. Lany J, Saffran JR (2013) Statistical learning mechanisms in infancy. In: *Comprehensive Developmental Neuroscience: Neural Circuit Development and Function in the Brain, Volume 3* (Rubenstein JLR, Rakic P, eds). Amsterdam, Elsevier: 231-248.
9. Pinker S (2002) *The Blank Slate. The modern denial of human nature*. New York, NY: Penguin

Group.

10. Cosmides L, Barrett HC, Tooby J (2010) Adaptive specializations, social exchange, and the evolution of human intelligence. *Proc Natl Acad Sci* 107: 9007-9014.
11. Agrillo C, Ranpura A, Butterworth B (2010) Time and numerosity estimation are independent: Behavioral evidence for two different systems using a conflict paradigm. *Cog Neurosci* 1: 96-101.
12. Clahsen H, Temple C (2003) Words and rules in children with Williams syndrome. In: *Language competence across populations* (Levy Y, Schaeffer J eds). Erlbaum: Mahwah, NJ, 323-352.
13. Cohen Kadosh R, Bahrami B, Walsh V, et al. (2011) Specialization in the human brain: The case of numbers. *Front Hum Neurosci* 5: 62.
14. Bates E, Elman J, Johnson MH, Karmiloff-Smith A, et al. (1998) Innateness and Emergentism. In *A Companion to Cognitive Science* (Bechtel W, Graham G, eds). Oxford: Basil Blackwell, 590-601.
15. Karmiloff-Smith A, Plunkett K, Johnson M, et al. (1998) What does it mean to claim that something is 'innate'? *Mind Lang* 13: 588-597.
16. Hyde DC, Spelke ES (2011) Neural signatures of number processing in human infants: Evidence for two core systems underlying numerical cognition. *Dev Sci* 14: 360-371.
17. Lee SA, Spelke ES (2010) A modular geometric mechanism for reorientation in children. *Cognitive Psychology* 61: 152-176.
18. Spelke ES, Kinzler KD (2007) Core Knowledge. *Dev Sci* 10: 89-96.
19. Xu F, Spelke ES, Goddard S (2005) Number sense in human infants. *Dev Sci* 8: 88-101.
20. Dehaene S (1997) *The number sense: how the mind creates mathematics*. Oxford: Oxford University Press.
21. Butterworth B (1999) *The mathematical brain*. Macmillan: London.
22. Gelman R, Gallistel R (1986) *The child's understanding of number*. Cambridge, MA: Harvard University Press.
23. Duchaine B (2000) Developmental prosopagnosia with normal configural processing. *NeuroReport* 11: 79-83.
24. Duchaine B, Nakayama K (2005) Dissociations of face and object recognition in developmental prosopagnosia. *J Cog Neurosci* 17: 249-261.
25. Duchaine B, Nakayama K (2006) Developmental prosopagnosia: a window to content-specific face processing. *Curr Op Neurobio* 16: 166-173.
26. Duchaine B, Yovel G, Butterworth E, et al. (2006) Prosopagnosia as an impairment to face-specific mechanisms: elimination of the alternative explanations in a developmental case. *Cognitive Neuropsychology* 23: 714-747.
27. Landau B, Hoffman JE, Kurz N (2005) Object definitions with severe spatial deficits in Williams syndrome: sparing and breakdown. *Cognition* 100: 483-510.
28. Piattelli-Palmarini M (2001) Speaking of learning: How do we acquire our marvellous facility for expressing ourselves in words? *Nature* 411: 887-888.
29. Spelke ES, Kinzler KD (2009) Innateness, learning and rationality. *Child Dev Pers* 3: 96-98.
30. Duchaine B, Cosmides L, Tooby J (2001) Evolutionary psychology and the brain. *Curr Op Neurobio* 11: 225-230.
31. Hauser MD, Spelke ES (2004). Evolutionary and developmental foundations of human knowledge: A case study of mathematics. In M Gazzaniga (Ed.) *The Cognitive Neurosciences*, Vol. 3. Cambridge: MIT Press, 853-864.

32. Bolhuis JJ, Brown GR, Richardson RC, et al. (2011) Darwin in Mind: New Opportunities for Evolutionary Psychology. *PLoS Biology* 9: 1-8.
33. Baron-Cohen S, Leslie AM, Frith U (1985) Does the autistic child have a “theory of mind”? *Cognition* 21: 37-46.
34. Leslie AM (1992) Pretence, autism, and the theory-of-mind-module. *Cur Dir Psych Sci* 1: 18-21.
35. Baron-Cohen S, Ring HA, Wheelwright S, et al. (1999) Social intelligence in the normal and autistic brain: an fMRI study. *Eur J Neurosci* 11: 1891-8.
36. Temple CM (1997) Cognitive neuropsychology and its application to children. *J Child Psychol Psychiatry* 38: 27-52.
37. Butterworth B (2008) State-of-science review: Dyscalculia. In Goswami UC (Ed.) *Foresight Mental Capital and Mental Wellbeing*. Office of Science and Innovation, London.
38. Duchaine B, Nieminen-von Wendt T, New J, et al. (2003) Dissociations of visual recognition in a developmental agnostic: evidence for separate developmental processes. *Neurocase* 9: 380-389.
39. Molko N, Cachia A, Rivière D, et al. (2003) Functional and Structural Alterations of the Intraparietal Sulcus in a Developmental Dyscalculia of Genetic Origin. *Neuron* 40: 847-858.
40. Rice M (1999) Specific grammatical limitations in children with Specific Language Impairment. In: *Neurodevelopmental disorders* (Tager-Flusberg H, ed). Cambridge, Mass: MIT Press, 331-360.
41. Shalev RS, Manor O, Gross-Tsur V (2005) Developmental dyscalculia: a prospective six-year follow-up. *Dev Med Child Neurol* 2: 121-125.
42. Castle A, Coltheart M (1993) Varieties of developmental dyslexia. *Cognition* 47: 149-180.
43. Gopnik M (1990) Genetic basis of grammar defect. *Nature* 347: 26-26.
44. Karmiloff-Smith A (1992) *Beyond Modularity: A Developmental Perspective on Cognitive Science*. Cambridge, Mass: MIT Press/Bradford Books.
45. Karmiloff-Smith A (1998) Development itself is the key to understanding developmental disorders. *Trends Cog Sci* 2: 389-398.
46. Mareschal D, Johnson MH, Sirios S, et al. (2007) *Neuroconstructivism: Vol. I. How the brain constructs cognition*. Oxford, England: Oxford University Press.
47. Westerman G, Mareschal D, Johnson MH, et al. (2007) Neuroconstructivism. *Dev Sci* 10: 75-83.
48. Westermann G, Thomas MSC, Karmiloff-Smith A (2010) Neuroconstructivism. In: *Handbook of Childhood Development* (Goswami U, ed). Oxford: Wiley-Blackwell, 723-748.
49. Karmiloff-Smith A (2013) Challenging the use of adult neuropsychological models for explaining neurodevelopmental disorders: Developed versus developing brains. *Quart J Exp Psych* 66: 1-14.
50. Paterson SJ, Brown JH, Gsödl MK, et al. (1999) Cognitive Modularity and Genetic Disorders, *Science* 286: 2355-2358.
51. Johnson MH (2001) Functional brain development in humans. *Nat Rev Neurosci* 2: 475-483.
52. Stiles J (2009) *The Fundamentals of Brain Development*. Cambridge MA: Harvard University Press.
53. Thomas MSC, Knowland VC, Karmiloff-Smith A (2011) Mechanisms of developmental regression in autism and the broader phenotype: a neural network modeling approach. *Psych Rev* 118: 637-654.
54. Casey BJ, Giedd JN, Thomas KM (2000) Structural and functional brain development and its relation to cognitive development. *Biol Psychol* 54: 241-257.

55. Huttenlocher PR, de Courten C (1987) The development of synapses in striate cortex of man. *Hum Neurobio* 6: 1-9.
56. Huttenlocher PR, Dabholkar AS (1997) Regional Differences in Synaptogenesis in Human. *J Comp Neurol* 387: 167-178.
57. Giedd J, Blumenthal J, Jeffries N, et al. (1999) Brain development during childhood and adolescence: A longitudinal MRI study. *Nat Neurosci* 2: 861-863.
58. Giedd J, Rumsey J, Castellanos F, et al. (1996) A quantitative MRI study of the corpus callosum in children and adolescents. *Dev Brain Res* 91: 274-280.
59. Cohen Kadosh K., Henson RN, Cohen Kadosh R, et al. (2009) Task-dependent activation of face-sensitive cortex: an fMRI adaptation study. *J Cog Neurosci*, 22: 903-917.
60. De Haan M, Humphreys K, Johnson MH (2002) Developing a brain specializes for face processing: A converging methods approach. *Dev Psychobiology* 40: 200-212.
61. Krishnan S, Leech R, Mercure E, et al. (2014) Convergent and divergent fMRI responses in children and adults to increasing language production demands. *Cereb Cortex* pii: bhu120.
62. Mills DL, Coffy-Corins S, Neville H (1997) Language comprehension and cerebral specialisation from 13-20 months. *Dev Psych* 13: 397-445.
63. Minagawa-Kawai Y, Mori K, Naoi N, et al. (2007) Neural attunement processes in infants during the acquisition of language-specific phonemic contrasts. *J Neurosci* 3: 315-321.
64. Neville H, Mills D, Bellugi U (1994) Effects of altered auditory sensitivity and age of language acquisition on the development of language-relevant neural systems: Preliminary studies of William syndrome. In: *Atypical cognitive deficits in developmental disorders: Implications for brain function* (Broman S, Grafman J, eds). Hillsdale, NJ: Erlbaum, 67-83.
65. Stiles J (2012) Neural plasticity and cognitive development: Insights from children with perinatal brain injury. Oxford UK: Oxford University Press.
66. Bates E, Roe K (2001) Language development in children with unilateral brain injury. In: *Handbook of Developmental Cognitive Neuroscience* (Nelson CA, Luciana M, eds). Cambridge, MA: MIT Press, 281-307.
67. Casey BJ, Tottenham N, Liston C, et al. (2005) Imaging the developing brain. What have we learned about cognitive development? *Trends Cog Sci* 9: 104-110.
68. Durston S, Davidson MC, Tottenham N, et al. (2006). A shift from diffuse to focal cortical activity with development. *Dev Sci* 9: 1-8.
69. Paterson SJ, Heim S, Friedman JT, et al. (2006) Development of structure and function in the infant brain: Implications for cognition, language and social behaviour. *Neurosci Biobehav Rev* 30: 1087-1105.
70. Smith LB, Thelen E (2003) Development as a dynamic system. *Trends Cog Sci* 7: 343-348.
71. Tyler LK, Shafto MA, Randall B, et al. (2010) Preserving Syntactic Processing across the Adult Life Span: The Modulation of the Frontotemporal Language System in the Context of Age-Related Atrophy. *Cereb Cortex* 20: 352-364.
72. Dehaene S, Cohen L (2007) Cultural recycling of cortical maps. *Neuron* 56: 384-398.
73. Fodor J (1983) Modularity of Mind. Cambridge, MA: MIT Press.
74. Dehaene S, Charles L, King JR, et al. (2014) Toward a computational theory of conscious processing. *Curr Op Neurobio* 25: 76-84.
75. Anderson ML (2010) Neural reuse: A fundamental organizational principle of the brain. *Behav Brain Sci* 33: 245-313.
76. Dehaene S (2009) Reading in the brain: The science and evolution of a human invention. New York, NY: Penguin Group

77. Goldman-Rakic PS (1987) Circuitry of the pre- frontal cortex and the regulation of behavior by representational knowledge. In: *Handbook of Physiology 5* (Plum F, Mountcastle V, eds). Bethesda, MD: American Physiological Society, 373-417.
78. Johnson MH (2004) Plasticity and functional brain development: The case of face processing. In: *Attention & Performance XX: Functional neuroimaging of visual cognition* (Kanwisher N, Duncan J eds). Oxford: Oxford University Press, 257- 263.
79. Kuhl P (2004) Early language acquisition: cracking the speech code. *Nat Rev Neurosci* 5: 831-843.
80. Majdan M, Schatz CJ (2006) Effects of visual experience on activity-dependent gene regulation in cortex. *Nat Neurosci* 9: 650-659.
81. Meaney MJ, Szyf M (2005) Maternal care as a model for experience-dependent chromatin plasticity? *Trends Neurosci* 28: 456-463.
82. Bhattacharya J, Petsche H (2005) Drawing on mind's canvas: Differences in cortical integration patterns between artists and non-artists. *Hum Brain Mapping* 26: 1-14.
83. Crone EA, Elzinga B (2014) Changing brains: How longitudinal fMRI studies can inform us about cognitive and social-affective growth trajectories. *WIREs Cogn Sci* 6: 53-63.
84. Brown TT, Petersen SE, Schlaggar BL (2006) Does human functional brain organization shift from different to focal with development? *Dev Sci* 9: 9-10.
85. Karmiloff-Smith A (1997) Promissory notes, genetic clocks or epigenetic outcomes? *Behav Brain Sci* 20: 359-377.
86. Karmiloff-Smith A (2007) Atypical epigenesis. *Dev Sci* 10: 84-88.
87. Kingsbury MA, Finlay LF (2001) The cortex in multidimensional space: where do cortical areas come from? *Developmental Science* 4: 125-142.
88. Bishop KM, Goudreau G, O'Leary DDM (2000) Regulation of area identity in the mammalian neocortex by Emx2 and Pax6. *Science* 288: 344-349.
89. Kaffman A, Meaney MJ (2007) Neurodevelopmental sequelae of postnatal maternal care in rodents: clinical and research implications of molecular insights. *J Child Psychol. Psychiatry* 48: 224-244.
90. Gotlieb, G (2007) Probabilistic epigenesis. *Dev Sci* 10: 1-11.
91. Scerif G, Karmiloff-Smith A (2001) Genes and environment: What does interaction really mean? *Trends Gen* 17: 418-419.
92. Quartz S, Sejnowski T (1997) The neural basis of cognitive development: A constructivist manifesto. *Behav Brain Sci* 20: 537-596.
93. Finlay BL (2007) E pluribus unum: Too many unique human capacities and too many theories. In: *The evolution of mind: Fundamental questions and controversies* (Gangestad S, Simpson J, eds). New York: Guilford Press, 294-304.
94. Karmiloff-Smith A (2010) Neuroimaging of the developing brain: Taking “developing” seriously. *Hum Brain Map* 31: 934-941.
95. Karmiloff-Smith A (2012) Brain: The Neuroconstructivist Approach. In: *Neuro- developmental disorders across the lifespan: A neuroconstructivist approach* (Farran EK, Karmiloff-Smith A, eds) Oxford: Oxford University Press, 37-58.
96. Karmiloff-Smith A, Thomas MSC (2004) Can developmental disorders be used to bolster claims from Evolutionary Psychology? A neuroconstructivist approach. In: *Biology and Knowledge Revisited: From Neurogenesis to Psychogenesis* (Langer J, Taylor Parker S, Milbrath C, eds) Mahwah, NJ: Lawrence Erlbaum Associates, 307-322.
97. Karmiloff-Smith A (2009) Nativism versus Neuroconstructivism: Rethinking the Study of

- Developmental Disorders. Interplay of Biology and Environment, *Dev Psych* 45: 56-63.
98. Pinker S (1994) *The language instinct: How the mind creates language*. New York: W.Morrow.
 99. Pinker S (1999) *Words and rules: The ingredients of language*. New York: Basic Books.
 100. Karmiloff-Smith A, Thomas MSC, Annaz D, et al. (2004) Exploring the Williams Syndrome Face Processing Debate: The importance of building developmental trajectories. *J Child Psych Psychiatry* 45: 1258-1274.
 101. Karmiloff-Smith A, D'Souza D, Dekker TM, et al. (2012) Genetic and environmental vulnerabilities in children with neurodevelopmental disorders. *Proc Natl Acad Sci* 109: 17261-5.
 102. D'Souza D, Karmiloff-Smith A (2011) When modularization fails to occur: a developmental perspective. *Cog Neuropsych* 28: 276-287.
 103. Mackinlay R, Charman T, Karmiloff-Smith A (2006) High functioning children with autism spectrum disorder: A novel test of multitasking. *Brain Cog* 61: 14-24.
 104. Comery TA, Harris JB, Willems PJ, et al. (1997) Abnormal dendritic spines in fragile X knockout mice: Maturation and pruning deficits. *Proc Natl Acad Sci* 94: 5401-5404.
 105. Oliver A, Johnson MH, Karmiloff-Smith A, et al. (2000) Deviations in the emergence of representations: A neuroconstructivist framework for analysing developmental disorders. *Dev Sci* 3: 1-23.
 106. Karmiloff-Smith A, Scerif G, Ansari D (2003) Double dissociations in developmental disorders? Theoretically misconceived, empirically dubious. *Cortex* 39: 161-163.
 107. Johnson MH, Halit H, Grice S, et al. (2002) Neuroimaging of typical and atypical development: A perspective from multiple levels of analysis. *Dev Psychopath* 14: 521-536.
 108. Cicchetti D, & Tucker D (1994) Development and self-regulatory structures of the mind. *Dev Psychopath* 6: 533-549.
 109. Sur M, Pallas SL, Roe AW (1990) Crossmodal plasticity in cortical development: Differentiation and specification of sensory neocortex. *TINS* 13: 227-233.
 110. Webster MJ, Bachevalier J, Ungerleider LG (1995) Development and plasticity of visual memory circuits. In: *Maturational windows and adult cortical plasticity in human development: Is there reason for an optimistic view?* (Julesz B, Kovacs I, eds). Reading, MA: Addison-Wesley.
 111. Bavelier D, Levi DM, Li RW, et al. (2010) Removing brakes on adult plasticity: from molecular to behavioural interventions. *J Neurosci* 30: 14964-71.
 112. Hensch TK (2005) Critical period plasticity in local cortical circuits. *Nat. Rev. Neurosci* 6: 877-888.
 113. Karmiloff-Smith A (1994) Transforming a partially structured brain into a creative mind. *Behav Brain Sci* 17: 732-745.

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