Brain and behavioral development (II): cortical

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Introduction

The changing structure of the cerebral cortex can be put to many uses in understanding behavioral and cognitive development. Developmental psychologists often look to biology for structural confirmation of the theories about the developing organization of behavior, for physical realization of stages, connections, and modules. Alternatively, structure is often invoked to imply constraints on behavior (e.g., perhaps the absence of some essential physical building block, such as a certain transmittter or myelination of a pathway, prevents the emergence of some particular behavior until a certain age). The role of cause and constraint can also be reversed: the onset of a particular sensory experience or maternal interaction often induces structural alterations in the brain greater than the simple registration of the event, changing the character of future information handling. Overall, structure and physiology give an added dimension to the description of behavioral change, and often can supply direct insights into the causes of behavioral and cognitive determinants.

Since cortical cells are electrophysiologically active from the moment they are generated, even in transit from their natal site to their terminal position, it is wrong to say the cortex 'turns on' at any particular developmental point. Rather, the cortex is in a continually changing state of activity, and uses its own activity to construct itself in combination with the instructions of intracellular and extracellular molecules specified by the genome. Later, information from the external environment is added to the mix, as represented by the activity of inputs to the cortex.

Our main goal will be to describe this complex system in the appropriate dynamical terms. We will begin with a description of fundamental cortical structure: how and when the cortex develops in early embryogenesis. The embryology will be considered in light of evidence for and against early cortical specialization and modularity. Then we will consider maturational gradients of various kinds as they have been described in the cortex

and how they might be related to behavioral maturation. These events are primarily prenatal and concern the structure with which the cortex first addresses the world. Finally, we will consider the postnatal development of the brain and how it links to behavioral development.

Structure of the cerebral cortex

Adult organization: cortical layers, columns, and their specializations

The six-layered scheme to describe the cortex laid out by Karl Brodmann (1868–1918) in his publications on cortical architecture in the period 1903–1918 is the one still in use today (Figs. 1 and 2). First, a note on nomenclature. The word 'cortex' when used technically refers to any layered, external structure. In this entry, we are discussing the neocortex or isocortex, the six-layered structure that dominates the surface of the human brain. As commonly, but not technically used, the words 'cortex' and 'cerebral cortex' will refer to the isocortex or neocortex.

The key to understanding cortical structure, in both development and adulthood, is to understand that the cortical column (the fundamental, repeating unit of the cortex, see below) does a stereotyped intake, transformation, and distribution of information within a matrix of local and distant influences from the rest of the cortex, though the functional contents of a column are wildly diverse (Rakic, 1990). The principal input to the cortex comes from the thalamus, a collection of nuclei in the diencephalon that gets its input from: (1) the senses including sight, sound, touch, and kinesthesis, (2) other parts of the brain that give information about the body's state of motion, homeostasis, and arousal state, and (3) cortical areas (Fig. 1). The input from the thalamus goes to the middle layer, Layer IV, and, to a lesser extent, to the upper part of Layer VI (Fig. 2).

This thalamic information in Layer IV is then relayed up and down, to Layers II and III, and to Layers V and
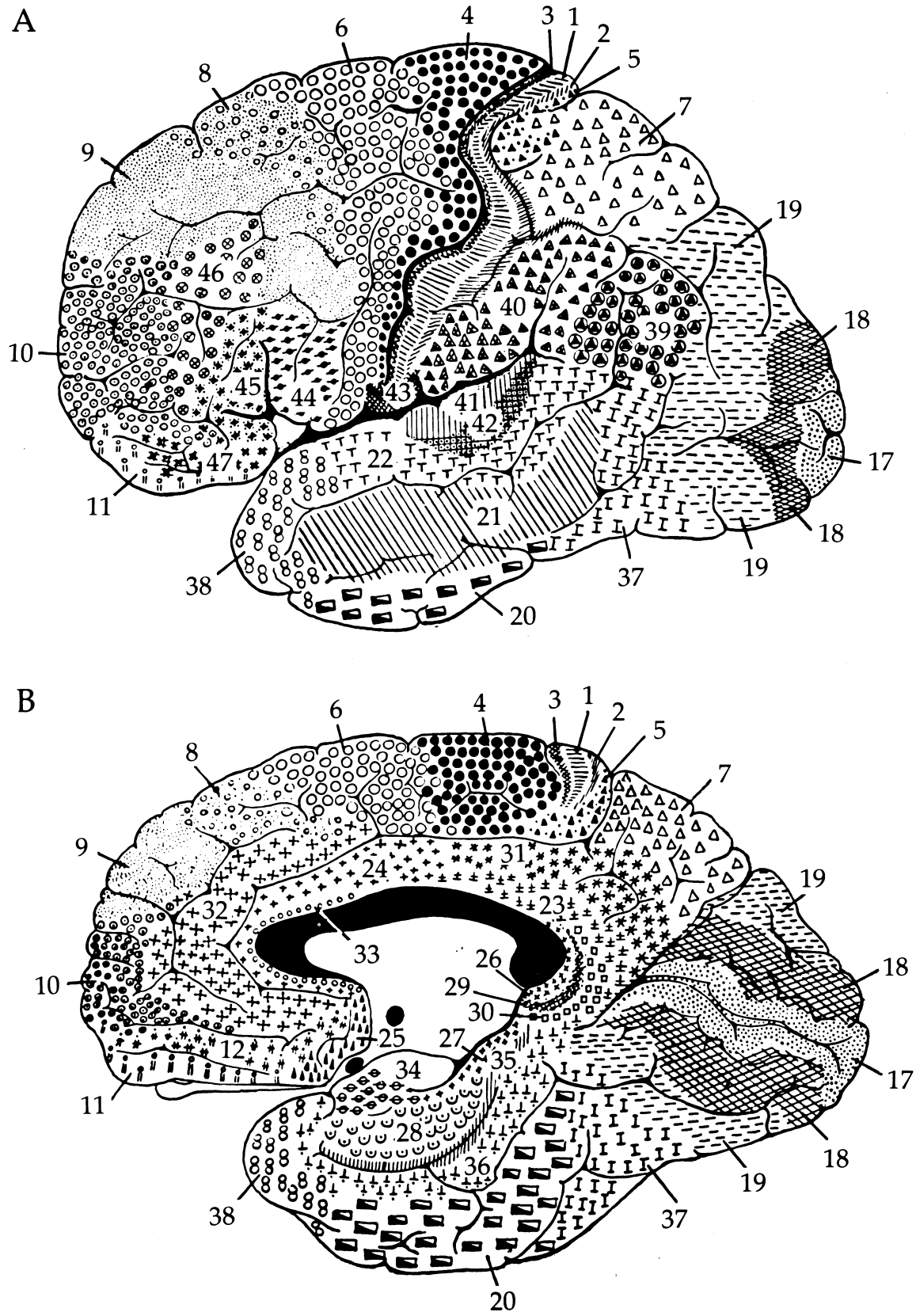
Figure 1. Areas of the cortex as described by Brodmann. Lateral (A) and medial (B) views of the cortex are shown. The numbered divisions are based on the thickness, density, and cell size of the cortical layers.

VI (Fig. 2). The majority of synaptic connections most input cells of the cortex will make are restricted to the column several cells wide that extends perpendicularly from Layer IV to the cortical surface, and down to Layer VI. All the cells in a particular column participate in similar contexts. For a few examples, taken from widely separated parts of the cortex, one column might fire to stimulation of a particular location and type of sensation on the body surface, another before a particular trajectory of arm movement, and another might fire when the individual is anxious in a social context.

The activity in these columns is continually modified by the local and distant context supplied from other cortical areas. Output from Layers II and III is long-range and connects the cortex to itself. Axons from these areas distribute to neighboring cortical areas (e.g., from primary to secondary visual cortex), to distant cortical areas (e.g., from secondary visual cortex to visuomotor fields in frontal cortex), and across the corpus callosum to the cortex on the other side of the brain. The cells of Layer V distribute their axons sub-cortically to every sort of effector center throughout the brain, including the motoneurons of the spinal cord. The principal output connection of Layer VI is a reciprocal connection back to the thalamus, which can be massive.

This same organization of layers and repeating columns is found throughout the cortex. The next layer of organization, the cortical area has inputs, descending connections, and specializations of layers that reflect their relative position in the intake or distribution of information (Fig. 2). The primary visual cortex, which receives a massive thalamic input relayed from the retina, has an unusually large number of cells in Layer IV. In the motor cortex, Layer IV is almost absent, and the cells of Layer V, the output layer connecting to downstream motor centers and the spinal cord, are unusually large and prominent. Each cortical area typically contains a topographic representation of a sensory, motor, or other computed dimension (any derived ordered array, such as location in 3-D auditory space, which has no sensory surface like the retina). Each area has unique input and output and a limited repertoire of physiological transformations of its thalamic input, such as the elongated, orientation-selective visual receptive fields of the primary visual cortex that are constructed from the symmetrical center-surround visual fields of their visual input.

It is critical to understand what is meant by a cortical area because many of the questions that have been raised about the relationship of behavioral development to cortical development assume that particular perceptual and cognitive mechanisms can be mapped directly onto particular cortical areas, using the concept of a cortical module to subsume both structure and function. Such functions can vary from basic perceptual operations to
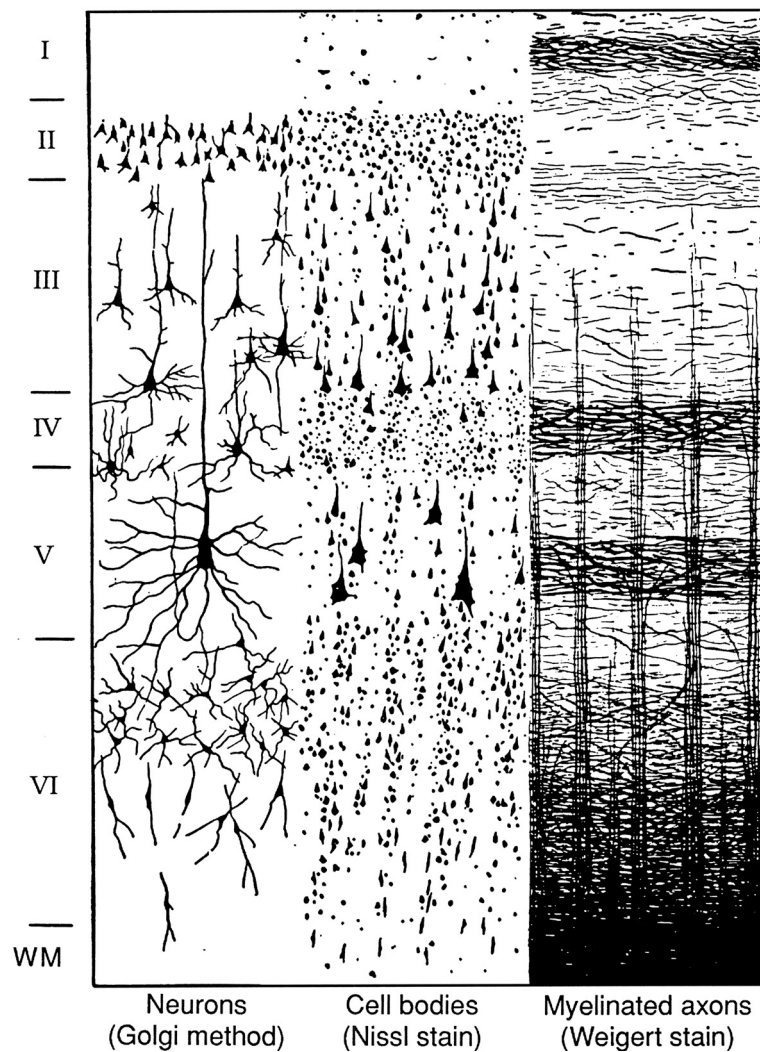

Figure 2. The layers of the cortex. Cortical neurons and axons are arranged in six principal layers designated by Roman numerals; "WM" refers to 'white matter,' the myelinated axons passing underneath the cortex. Three types of stains are represented: the Golgi method, which stains whole cells and all their processes, the Nissl stain, which shows only cell processes, and the Weigert stain, which stains axons.

complex aspects of cognition, like mapping spatial translocations over time ('A-not-B') or syntax in language. For example, if there is a 'face recognition area' located in the mature cortex necessary and sufficient for all face recognition, then to understand the maturation of that area is to understand the maturation of face perception. Many investigators, however, emphatically disagree with the idea of modular structure-function relationships realized in the cortical area, arguing for a more distributed representation, which sets a very different agenda for developmental research. The pieces of developmental information that will bear on this question relate to when each cortical area is specified for its function, how that function is realized, and how plastic the functions of each area are.
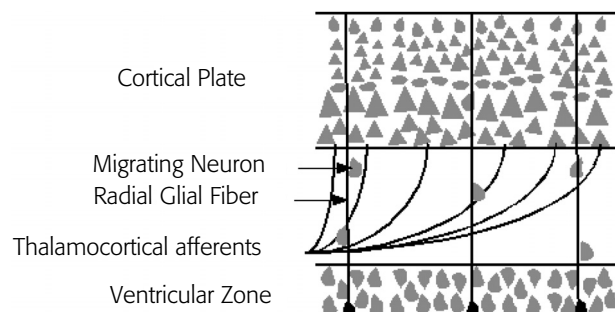
Figure 3. A schematic diagram illustrating the general development of the cortex. Cortical neurons are generated in the ventricular zone and migrate into the cortex along radial glial fibers (black vertical lines) or tangential pathways (not shown). Neurons settle within the developing cortical plate according to an inside-out gradient. Afferents from thalamic nuclei begin to colonize the intermediate zone as cortical neurons migrate through them.

Cortex development

Making layers and columns

The area where cells that will make up the cerebral cortex divide and propagate is called the ventricular zone (Fig. 3). Early in development, before the proliferation of many cells and their connections, the ventricular zone and cortical plate (the primordial cortex) are directly apposed. Later, however, as the region between them fills up with cells from other regions and connecting processes, the cells migrate along progressively stretching glial cells that hang on to their connection to the ventricular zone on the inside and the cortical wall on the outside, letting go only when all cortical cells have been generated. Due to these glial highways, each location in the ventricular zone makes a column (or more properly, a column-shaped zone) extending the depth of the mature cortex (Rakic, 1990). The second dimension of developmental time corresponds directly to the cortical layers, from inside (VI) to outside (II). Recently, a second population of interneurons of the cortex have been discovered that arise from a special generative region in the ventricular zone, separate from the source of radially migrating neurons described here, and migrate tangentially through the cortex to populate it.

Several structural aspects of this pattern of development are significant for behavioral development, particularly the debate about the modular nature of the cortex. Firstly, most of the cellular constituents of a cortical area come from a particular region of the ventricular zone, and thus could capably transmit genetic instructions that are locally specified in the ventricular zone, such as instructions for particular wiring patterns or neurotransmitter expression. Secondly, most of the events just described happen very early in development, before three months
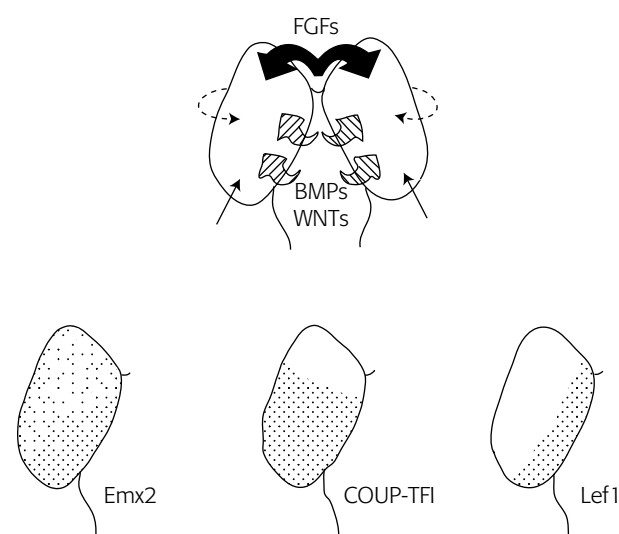

Figure 4. Schematic diagrams of cortical patterning in the embryonic mouse cerebral cortex showing signaling centers that set up patterns of transcription factors that control regional differentiation and differential growth, and the patterns of transcription factor expression. (a) Signaling centers (b-d) Putative polarizing molecules: FGFs, BMPs, WNTs. Transcription factors: Emx2; COUP-TFI; Lef1. Redrawn from C. W. Ragsdale and E. A. Grove, 2001. Patterning the mammalian cerebral cortex. *Current Opinion in Neurobiology*, II, 50–58.

post-conception. Finally, the principal input to the cortex, the thalamus, can influence cortical organization from the very earliest stages, as the axons from the thalamus are in fact a large part of developing processes interposed between the ventricular zone where cortical neurons are generated, and the cortex, where they eventually mature. The cortical neurons migrate directly through their eventual input. The thalamus could potentially transmit information on the fetus's own movement, the body surface, or any structured or unstructured activity arising in any part of the brain.

Making areas

What is the source of the information that gives cortical areas their distinct input and output connections and other specialized local features? Only recently, the cortex has come to be understood in the same genomic terms that structures like the entire vertebrate body plan, or spinal cord segmentation, are understood (Grove & Fukuchi-Shimogori, 2003) (Fig. 4). These schemes are rather counter-intuitive to the adult human architect's first guess about how to build something, where expression of Gene A corresponds to Segment A or Cortical Area A, and Gene B to Segment B, and so on. Rather, adult 'parts,' like a spinal cord segment or cortical area, correspond to regions in overlapping and nested patterns of regulatory genes. These genes will in time control the expression of other regulatory genes and, eventually, particular molecules like structural proteins,

cell-cell recognition molecules, and transmitter production and uptake systems that are the physical components of the cortex. Very rarely would the domain of expression of an early regulatory gene be identical with a recognizable chunk of adult morphology. With the exception of a few genes and other markers that are partially localized to the primary sensory cortices, particularly visual and somatosensory, there is no mosaic organization of gene expression that in any way mirrors the adult mosaic of cortical areas.

A 'polarizer' has been discovered at the front of the growing cortical plate that appears to control the orientation of the cortex. Thus, if this region is transplanted to the back, the topographic organization of cortical areas turns around from the normal arrangement, such that the somatosensory cortex is at the back and the visual cortex in the front (Fig. 4). This polarizer could control the expression of genes in the proliferative zone for the cortical plate, the cortical plate itself, and secondarily the recognition molecules that direct particular regions of the thalamus to particular parts of the cortex. So far, predominating types of gene expression found to be under control of these polarizers are different kinds of cell recognition molecules that control axon pathway selection.

One clear outcome of this specification is that thalamic input to the cortex is very topographically precise in early development. For example, in the adult, the lateral geniculate nucleus in the thalamus gets a point-to-point projection from the retina and confers this representation directly on the visual cortex. Even on first contact, the topology of the projection from the retina to the cortex is nearly as specific as it is in adulthood. The timing and precise placement of thalamic input is well suited to specify further many of the local features of cortical areas. Thalamus-controlled differentiation steps include differences in numbers of cells in the various layers, expression of particular transmitters, and, perhaps most important, the effects of the nature and pattern of activity relayed through the thalamus on how cortical neurons wire up.

In great contrast to the thalamic connections, the early connections of the cortex with itself are made wholesale (Bates *et al.*, 2002). In rodents, the first connections from a very small area of the cortex are in potential reach of a third to half of the entire cortical surface. Intracortical connections are the principal substrate of cortical plasticity of all types that involve reallocation of the cortex to new functions, from gross to fine functional readjustments. Demonstrations of this range from local plasticity that might be caused by producing a gap in the sensory information coming in from the periphery within a particular sensory modality, to experimental 're-wiring' of cortex done by inducing retinal axons to innervate auditory centers, which causes the auditory cortex to take on visual properties (Pallas,

2001). A recent demonstration of multimodal activation of the visual cortex in the early blind, including activation during Braille reading, almost certainly uses intracortical pathways to produce the observed re-organization (Sadato *et al.*, 1996).

The significance of embryology for modularity and plasticity

Understanding the cortex in terms of general vertebrate (and invertebrate!) mechanisms that produce the basic body plan opens a wealth of analogies and models. Segmentation of a uniform field into a number of repeating units, and then subsequent differentiation of each unit is the central strategy for creation of the body in embryos, from the segments of an insect body or a worm, to the segments of the vertebrate spinal cord. This 'theme-and-variation' strategy maps in a fairly direct way onto an 'easy' evolutionary alteration of the genome that preserves function while allowing adaptation: duplications of individual genes or groups of genes, followed by modification of the duplicate gene while conserving the original. A typical modification would allow different genes to come under the control of the regulatory genes in separate segments of a segmented structure. Consider the spinal cord of a fish, which has a repeating segmental structure that maps onto the relatively uniform (front to back) musculature of the fish trunk for the rhythmical motion of swimming. In animals with limbs, those segments that innervate the limbs must have evolved to acquire new instructions, including some very specific new wiring for complicated limb musculature. Yet, if these same segments are transplanted into the body trunk region, they are capable of expressing the old trunk pattern and wire up successfully. By analogy, the question of whether particular cortical areas, like the 'face area' or the 'motion area,' are wired for particular functions or are generic has a very likely answer in this developmental context: both.

Maturational gradients: what is the sequence of maturation of cortical areas, and how does it correspond to behavior?

Neurogenesis and innervation

Anyone hoping for fundamental simplicity in the patterning of the early maturation of the cortex will be disappointed. There is no single dimension of 'maturational state' that any area of the isocortex can be retarded or advanced on (which makes it even less likely there could be a moment when a region 'turns on'). Rather, each isocortical area is best viewed as an
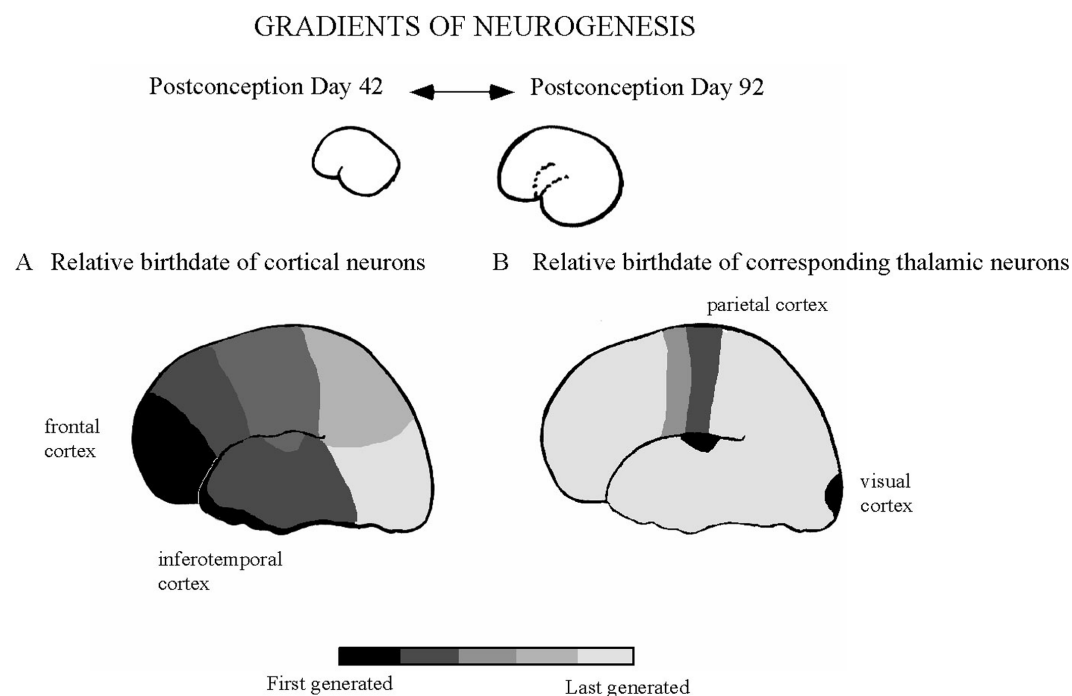
GRADIENTS OF NEUROGENESIS

Postconception Day 42 ⟷ Postconception Day 92

A   Relative birthdate of cortical neurons   B   Relative birthdate of corresponding thalamic neurons

First generated   Last generated

Figure 5. Maturational gradients in the early postnatal development of the human cortex on the day of birth (Postconception day 270). (A) Neurogenesis of cortical neurons begins at the rostrolateral margin of the cortex and proceeds posteriorly through the parietal cortex to the primary visual cortex, framing a period of genesis of about fifty days (Postconception days 42 to 92). (B) Neurogenesis of corresponding thalamic neurons begins with the medial geniculate body (auditory cortex, in black), the lateral geniculate body (on the pathway from the retine to the visual cortex, in black), and the ventrobasal complex (somatosensory cortex, next to the motor cortex) followed by neurons that innervate the motor cortex (in gray). The last thalamic neurons to be produced are located in the nuclei that innervate the frontal, parietal, and inferotemporal cortex.

assembly of different features, including neurogenesis, the maturation of its input, and the maturation of its output, all of those in the context of the maturation of the entire organism (Bates *et al.*, 2002). Because different areas of the brain follow maturational gradients that do not match in order, interesting mismatches occur. Thus, in some areas, intracortical connections will be relatively more mature than thalamic connections (the frontal cortex), and in others, the reverse will hold (primary visual cortex). Figure 5 compares the gradients of original generation for two central aspects of cortical development: the timing of the original generation of the cortex itself compared to the order of innervation of each cortical region by the thalamus.

The cortex has an intrinsic gradient of maturation. Neurogenesis begins at the front edge of the cortex and proceeds back to the primary visual cortex; the limbic cortices on the midline also get an early start. As depicted in the maturational gradients in Figure 5, paradoxically, the frontal cortex, viewed in hierarchical models as the last maturing cortical area, is in fact one of the first to be produced and thus quite 'mature' in some features. The order of thalamic development is quite different. In general, the primary sensory nuclei in the thalamus are generated first and establish their axonal connections to the cortex first. Various other nuclei, motor and cingulate, are intermediate in their timing,

and the last to be produced are the thalamic nuclei that innervate the frontal, parietal, and part of the inferotemporal cortex. The thalamic order of neurogenesis suggested a hierarchical notion of cortical development (primary sensory areas mature early, 'association' areas late), but it is not the whole story.

So what might the dual gradients mean for the frontal cortex, the area so often described as "maturing late?" The fact that the frontal cortex matures early, but receives its input from the thalamus relatively late, could predispose it for intracortical processing. In other words, this difference in developmental gradients might mean that the frontal cortex is primed for higher-order associative function from the start, not by virtue of being 'out of the circuit' early on.

A uniform perinatal burst of synaptogenesis

Synapses begin to be formed in the cortex from the time that the first neurons move into place, and a fair number are in evidence at 6 months post-conception. The first synapses must account for the many demonstrations of early activity-dependent organization in the cortex, and perhaps for several types of in utero learning (e.g., preferences for the language rhythms of the mother). Just before birth, all over the cortex, the density and number of excitatory synapses surge ten- to a hundredfold.

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What causes the dramatic acceleration of synapto-genesis? Using the visual cortex as a test case, the possibility that this marked increase is actually caused by the barrage of experience that occurs around birth was investigated. When monkeys were deprived of visual input, the initial acceleration and peak of synaptogenesis were unchanged. A second complementary experiment showed that when monkeys were delivered three weeks prematurely, so that experience began much sooner than it would normally occur, the peak of synaptogenesis still occurred on the monkey's anticipated birthday, not its premature one. In both experiments, secondary effects on types and distributions of synapses were also seen in this study, so experience does matter, but not to the timing of the synaptic surge (Bates *et al.*, 2002).

Why are the connections between neurons made just around birth, before most experience, rather than as experience occurs? In fact, the number of synapses produced is in excess of the eventual adult number. The immediate postnatal phase of development is distingui-shed by axon retraction and synapse elimination, regressive events, as well as growth and addition. In the mature nervous systems, synapses are both added and subtracted during learning. Perhaps the developing nervous system is both allowing activity (though initially disorganized) to be easily propagated through itself, and also allowing itself the possibility of both addition and subtraction of synapses, rather than simply additive ones, by the instalation of large numbers of synapses just prior to experience. This initial overproduction of synapses may be a way of producing continuity in mechanisms of synaptic stabilization from initial development to adulthood. The impressive statistical learning capabilities of infants soon after birth may require this highly elaborated substrate.

Experience-induced maturation

One of the best-studied features of perinatal develop-ment from both behavioral and neural perspectives is the development of binocular vision, and its relationship to binocular interactions in the visual cortex, both anatomically and physiologically defined (Dannemiller, in Nelson & Luciana, 1999). Several observations of interest about structure-function links arise from this work. Firstly, in normally developing individuals with normal experience and reasonable optics, there is a critical period for the establishment of a balance of influence from the two eyes on perceptual decisions, for sorting information by eye-of-origin, and for the development of stereoscopic depth perception that happens in the first several years of life. Absence of activity in either eye or incoordination of the eyes can permanently derail the development of normal visual function during this period. If all experience is denied,

however, and both eyes are closed (in experimental animals), what occurs is delay of the critical period – the representation of the two eyes does not begin its segre-gation into ocular dominance columns, and the special neurotransmitters and receptors that are responsible for this structural change are held at their initial state. When experience is re-instated, anatomical, pharma-cological, and physiological events then progress, as they would have, independently of the animal's age (to a point).

Presumably this allocation of cortical tissue to particular functions on the basis of activity occurs everywhere in the cortex. Initial function to structure allocation is often called maturation and the property of maturity is ascribed to the tissue, but the example above shows this need not be so. For example, the immaturity of the frontal cortex on which many executive and self-monitoring functions depend could reflect an absence of events likely to activate frontal cortex in early childhood, not a maturational deficit of the tissue itself.

Continuous brain, discontinuous behavior

One instructive structure-function relationship that appeared in the binocular interaction research was a mismatch between the gradual spatial segregation of the neurons responsive to either the right or left eye in the cortex, and a stepwise change in an aspect of visual behavior likely to be dependent on it, the development of binocular rivalry. In early infancy, as demonstrated in the laboratory of Richard Held, infants presented with horizontal stripes to one eye and vertical stripes to the other indicate by their behavior that their experience is a checkerboard, and not the alternating rivalry between the horizontal and vertical stripes that an adult experi-ences. In longitudinal studies, the infants switched in a matter of days from the immature to mature perception at about 3 months of age, while no such instant of sharp segregation has ever been described in the presumably corresponding anatomy. A different discontinuity with a similar lesson was described in the development of infant walking by Esther Thelen. At birth, all infants will show an alternating stepping movement when supp-orted over a surface, which disappears around 2–3 months, with real walking appearing at about 1 year of age. This progression was first described as a spinal reflex becoming supplanted by cortical control as the cortex matured. In fact, the spinal reflex never disappears, is the basis of adult walking, and can be elicited at any time if the infant is appropriately weighted and balanced. In this case, spinal circuitry can produce many different rhythmical patterns at any age, dependent on the particular pattern of peripheral load, and 'maturation' lies in the changing periphery.

Mapping complex changing functions onto complex changing tissue

The point of the prior section on maturation is to discount as much as possible the notion of cortical areas maturing as single functional modules blossoming one at a time, and rather to emphasize the continuous activity of the cortex from the time of its generation, with a single point of punctuation in the surge of synapse production at the time of birth. What then is known about the postnatal maturation of the cortex and its relationship to behavior? Until quite recently, very little. Attempts had been made to locate discontinuities or inflections in graphs of changes in the volume or structure of brain tissue, synapses, and process and correlate them with discontinuities in behavior (e.g., the period of very rapid vocabulary addition in learning), although, as we have discussed, the assumption that anatomical and physiological discontinuities should correspond is questionable (Bates *et al.*, 2002). Myelination, the growth of the insulating glial sheaths that increase the speed of axon conduction of impulses, is something that occurs postnatally, and could be correlated with behavior, but not with any great insight. Measurements of spontaneous electrical activity in the cortex (electroencephalograms, EEGs) and evoked activity (ERPs) could be compared from infant to adolescent to adult, with the typical result revealing that the frontal cortex, and sometimes the parietal cortex, showed the mature pattern later than sensory cortices.

Better imaging techniques of all kinds now allow closer structure-function mappings (M. H. Johnson, 2002). While behavioral information about children with brain damage was always available, now various Magnetic Resonance Imaging techniques give a much better idea about what part of the brain is damaged, which is particularly useful for longitudinal studies. Techniques for employing functional magnetic resonance imaging (fMRI) to image the brain's activity while the individual is employed in some task are being adapted to use with children. Exotic techniques, like diffusion tensor imaging, can look at the development of tracts with different physical properties in fully alive subjects; high-density ERPs can be registered with appropriate computer data-gathering that add much more spatial resolution to the already high temporal resolution of the technique. This list is not comprehensive, and it is growing.

Some striking results have already emerged, consistent across both brain damage and imaging studies (Bates *et al.*, 2002; Nelson & Luciana, 1999). Although the cortical areas involved in early and adult performance of the same tasks are rarely disjunct, they are never identical. The structures important for learning language are quite different to those required for mature language performance, both in laterality and in anterior-posterior position, as determined in longitudinal studies of children with early brain damage. A different constellation of areas is activated for facial and spatial judgments in children, though general adult divisions are employed. Overall, there is an interesting tendency for the right hemisphere to be preferentially involved in early learning (in both children and adults). The frontal cortex was found to be *more* active in children engaged in response inhibition tasks than adults, though its activity was not related to success in the task. Identification and understanding of neural structures that are specialized for the acquisition of new knowledge, rather than the performance of practiced abilities, will probably be one of the first outcomes of this research enterprise.

Conclusions

The field of developmental cognitive neuroscience is itself in early development, such that it is not possible to survey the conclusions reached, and suggest new directions. Rather, it is a time to scrutinize fundamental assumptions that guide research, while gathering basic descriptive information. The grammar, the fundamental classificatory scheme for cortex-behavior relationships, is the cortical area. These areas, however, might be not much more than the visible correlate of a mechanism to precisely fan out thalamic information over the cortical surface, to be integrated wholesale later by more general intracortical connections, and may be an unnatural unit for behaviorally defined functions. Are there any intrinsically determined wiring differences between cortical areas, or induced differences? What is the nature of regions of the cortex that are brought into performance early in learning versus those that are employed for mature abilities? Outside of primary sensory areas, we know virtually nothing about how response properties in cortical regions start out and organize, even in experimental animals. The most important fact to remember in all future investigations, however, is that, no matter how privileged the cortex might seem for the unique aspects of human cognition, it is part of the vertebrate body, evolving in the same context, developing with the same genome, with the same rules.

Questions

1. Do any regions of the cortex contain circuitry specific for their mature task (such as face recognition, or syntactic processing), or is cortical circuitry initially task-independent?

2. How do 'cortical areas,' other than primary sensory areas, develop in the primate cortex? Are they specified from the start, or do they arise epigenetically using mechanisms like activity-dependent segregation?

3. What is the best way to organize and understand the vast amount of data about cortical activity varying in space and time collected with all of the new functional and morphological imaging techniques?

See also:
Understanding ontogenetic development: debates about the nature of the epigenetic process; Magnetic Resonance Imaging; Conceptions and misconceptions about embryonic development; Normal and abnormal prenatal development; The status of the human newborn; Cognitive development in infancy; Perceptual development; Motor development; Speech development; Language development; Development of learning and memory; Attention; Brain and behavioral development (I): sub-cortical; Executive functions; Face recognition; Locomotion; Blindness; Cognitive neuroscience; Developmental genetics; Behavioral embryology; George E. Coghill

Further reading

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