Patterns of neuronal migration in the embryonic cortex

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Real-time imaging of migrating neurons has changed our understanding of how newborn neurons reach their final positions in the developing cerebral cortex. The migratory routes and modes of migration are more diverse and complex than previously thought. The finding that cortical interneurons migrate to the cortex from origins in the ventral telencephalon has already markedly altered our view of cortical migration. More recent findings have demonstrated additional nuances in the migratory pattern and highlighted differences between subsets of interneurons. Moreover, radial migration of pyramidal neurons does not progress smoothly from ventricle to cortical plate, but is instead characterized by distinct migratory phases in which neurons change shape and direction of movement. Integrating these findings with the molecular machinery underlying migration will provide a more complete picture of how the cerebral cortex is assembled.

A curious feature of brain development is that, although neurons are generated from precursor cells that line the walls of the ventricular system deep within the brain, newborn neurons often migrate long distances from their birthplace to reach their final destinations. This is particularly evident in the process of cortical development, whereby newborn neurons must migrate to the outer surface of the developing cortex. At early stages, when the cortical anlage is small, these distances are short but as development proceeds the distances that neurons must migrate becomes progressively longer, and in the case of the primate brain can reach distances up to 7 mm [1]. Studies of granule cell migration in the developing cerebellum have provided insights into some of the mechanisms of neuronal migration [2–4]. Recently, with the application of molecular and genetic approaches to the study of migration disorders in humans and in mutant mice, many key gene products have been discovered that are involved in neuronal migration [2,5–7]. Given the interactive nature of the process of migration, it is not surprising that these molecules include receptors, transcription factors, adhesion molecules, extracellular matrix proteins, diffusible and intracellular signaling molecules, and components of associated signaling cascades. The focus of the current review, however, is to describe recent advances in understanding the patterns of neuronal migration in the developing cerebral cortex. Studies based largely on real-time imaging of migrating neurons in situ in both rodent and primate cortex have contributed significantly to a more complete description of the routes that neurons take to reach their proper positions and provide a framework into which the molecular steps can be integrated.

Dynamics of cortical assembly
The first postmitotic cortical neurons collect in an outside-in sequence to form a transient layer termed the preplate [8]. Subsequently-born neurons migrate into the preplate to form a new series of layers collectively known as the cortical plate, which thereby splits the preplate into a superficial layer, the marginal zone, and a deeper layer, the subplate. As additional waves of migrating neurons arrive in the cortical plate they bypass earlier-generated neurons to form the cortical layers in an inside-out sequence; deeper layers are the first to form, superficial layers the last [9]. The correlation of laminar fate with birth order holds for all cortical neurons, including pyramidal neurons that constitute the principle projection neurons, and interneurons that are primarily inhibitory local circuit neurons [10–12]. Recent studies probing the sites of origin and mode of migration of cortical neurons have demonstrated that whereas cortical pyramidal neurons are generated in the germinal zones of the dorsal telencephalon [13–15], most, if not all, cortical interneurons are generated in the ventral telencephalon [16–19]. The latter region includes the proliferative zones of the ganglionic eminence. Thus, cortical pyramidal neurons can generally follow a relatively direct radial path to their laminar position in the developing cortex, but interneurons must travel circuitously and traverse a greater distance (Figure 1). The migratory routes of neurons arising in both the dorsal and ventral telencephalon have been the subject of intense study. Recent observations, although still falling short of providing a complete picture, indicate that migration patterns are complex, characterized by spatially and temporally distinct changes in the direction of movement and speed of migration for both pyramidal cells and interneurons.

Non-cortical origin of cortical interneurons
Recent studies have demonstrated that many cortical interneurons originate in the basal ganglia primordia –
the lateral, medial and caudal ganglionic eminences (LGE, MGE and CGE, respectively) — and migrate tangentially into the cerebral cortex. These include studies using dye labeling [18], retroviral lineage analysis or chimeric mice [19–21], as well as analysis of mutant mice lacking the transcription factors Dlx or Mash 1, which are normally expressed in the proliferative zone of the ventral telencephalon. These mice lack most cortical GABAergic interneurons [16, 22–24]. Evidence from cell transplantation and fate-mapping experiments indicates that the MGE is the primary source of cortical interneurons in rodents [19, 25–28], although cortical interneurons might also derive from the LGE [25, 29], CGE [30] and retrobulbar area [31]. Multiple classes of inhibitory neurons are present in the cortex, and it appears that most, with the exception of those that express calretinin, arise in the MGE [32]. In humans, a significant number of cortical GABAergic interneurons also appear to arise from progenitors in the cortical subventricular zone (SVZ) [33, 34]. The exact origin of the SVZ interneuron precursors is still unclear. Many take the most direct route by turning to enter the cortical plate [25, 26, 35–37] (Figure 1). Neurons migrating tangentially to reach their final locations by migrating through specific phases. Most cortical interneurons are generated in the medial ganglionic eminence (MGE) of the ventral telencephalon, and then change direction to enter the cortical plate (CP) by following a radial or an oblique path. The broken line indicates some interneurons have been observed to descend radially into the CP and others to continue radially to deeper laminae. Abbreviations: IZ, intermediate zone; LGE, lateral ganglionic eminence; LV, lateral ventricle; MZ, marginal zone; SVZ subventricular zone; VZ, ventricular zone.

**Tangential migration of interneurons: distinct phases**

Whereas all cortical interneurons arising in the MGE must migrate tangentially to reach the cortex, the migration pathways used by MGE cells change through development [25, 35]. Early in rodent neurogenesis, GABA-expressing cells from the MGE migrate through the intermediate zone (IZ) of the ganglionic eminence and disperse to all cortical layers, whereas later-born cells migrate through the striatal VZ and SVZ to enter the cortex. Within the developing cortex, the initial bands of tangentially migrating interneurons are most prominent in the lower IZ and SVZ and in the marginal zone (MZ) and subplate [19, 25, 27, 29, 35, 36]. Data from rodent brain also suggest that some interneurons could derive from the LGE. Early in neurogenesis, interneurons arising in the LGE appear to populate the cortical preplate [18, 29]. Later in neurogenesis, LGE-derived cells migrate tangentially through the SVZ on their way to the cortical plate [25]. A recent study using real-time imaging of glutamate decarboxylase 67 green fluorescent protein (Gad67-GFP) knock-in mice to examine the migration of GABAergic neurons has provided a potentially more accurate picture of pathways of migration than previous studies based on labeling with 1,1'-dioctadecyl-3,3,3',3'-tetramethylindocarbocyanine perchlorate (DiI) or on transplantation strategies. The data confirm that migration of cortical interneurons appears to occur primarily in two streams, in the cortical MZ and the IZ–SVZ. In the cerebral cortex, interneurons disperse tangentially and then generally enter the cortical plate by turning to migrate radially to their final positions [25, 26, 35–37] (Figure 1). Many take the most direct route by turning to enter the cortical plate. For example, cells migrating tangentially in the IZ–SVZ have been observed to turn and migrate radially or obliquely to enter the cortical plate from below [26, 35, 36], whereas interneurons migrating tangentially in the MZ have been observed to turn and enter the cortical plate from above [35, 36]. Neurons migrating tangentially have been noted to migrate more rapidly (~50 μm h⁻¹) [26, 27, 35, 38, 39] than neurons migrating radially (~10 μm h⁻¹) [39–43]. The radial phase of interneuron migration can be guided by radial glial fibers [33, 35], but although this is an attractive hypothesis it has yet to be proved. The identity of the substrate used by tangentially migrating neurons is also uncertain. Though both radial glia and axons have been suggested to be involved, the evidence is not compelling [33, 35, 37, 44, 45].
Many interneurons appear to take more complex routes to reach the cortical plate. For example, some interneurons migrate tangentially through the IZ–SZV, then turn and migrate radially through the cortical plate to enter the MZ [36]. It has been suggested that they might then disperse in the MZ before turning again to enter the cortical plate to reach their final positions [36]. Interestingly, a proportion of tangentially migrating neurons from all cortical layers were observed to undergo ventricle-directed migration to the VZ in slice-culture preparations [37]. Upon reaching the ventricle, these cells paused briefly and reversed direction to migrate radially into the cortical plate [37]. The majority of these cells were interneurons, and dye labeling of the ganglionic eminence confirmed that they originated in the basal forebrain. Ventricle-directed migration was observed with increasing frequency at later stages of corticogenesis. That some tangentially migrating neurons should contact the ventricle and then ascend into the cortical plate has led to speculation that neurons might obtain laminar address information through epigenetic signals in the cortical VZ [37,46]. Another possibility is that interneuron subtypes might migrate along specific routes. For example, ventricledirected migration could occur for only a particular class of interneuron. This hypothesis should be testable.

A variety of factors have been shown to participate in the guidance of tangentially migrating interneurons into the cerebral cortex. These factors include semaphorin–neuroplins [47,48], cell adhesion molecules such as polysialylated neural cell adhesion molecule (PSA-NCAM), neuregulins [49], and the slit and robo families of attractant and repellent molecules [50,51]. The action of these factors could combine to create permissive corridors to guide interneurons during tangential migration [52].

Modes of radial migration in the developing neocortex
Cortical neurons that migrate radially into the cortical plate are thought to do so through one of two possible modes: translocation or locomotion. Translocating cells possess a relatively long pia-directed process that has a stable attachment with the pial surface or marginal zone [53–56]. The translocating cell moves its nucleus radially within this fixed process to reach an appropriate position in the cortex. By contrast, locomoting cells are freely migrating cells that have a relatively ‘short’ leading process (~100–200 μm) [42,56,57] and that migrate in the radial direction along radial glial guides [3,58,59]. Because cortical neurons derive from radial glial cells [60–64], it has been suggested that a newborn neuron could inherit the radial glial fiber at mitosis and then translocate [62]. Alternatively, a newborn neuron could extend a radial process to the pia before beginning translocation.

Magini made the first observations to link cells with radial processes to neuronal migration. He noted variocities on the radial fibers in embryonic cortex (‘like the grains of a rosary’) and suspected that these were neuronal nuclei migrating along single radial cell processes [65]. Berry and Rogers also studied Golgi-stained tissue and concluded that radial fibers were multinucleated and proposed that neuronal nuclei translocated within the pial fibers [66]. It is now known that radial glia are not multinucleated, but variocities in the radial glial fiber or multiple neurons migrating along a single glial fiber can produce a multinucleated appearance. Most studies have used Golgi-stained developing opossum cortex and suggested that translocation might be the major mode of migration for cortical neurons [53]. However, the Golgi method fails to label freely migrating neurons that are known to be present during embryonic ages (e.g. interneurons), and it is often difficult to phenotype cells based on morphology alone. Brittis and colleagues described cells at early stages of cortical development that were immunopositive for a monoclonal antibody (2G12) that labels phosphorylated growth-associated-protein 43 (GAP-43), which is expressed in neurons [67] and mitotic precursor cells [68,69]. They described cells with nuclei at the ventricular surface and fibers coursing to the pia that were interpreted as translocating neurons. However, these cells resemble radial glial cells in M-phase of the cell cycle that are now known to retain their radial fibers during division [62,70] and to express a variety of phosphorylated proteins, including phosphorylated vimentin [71], phosphorylated mitogen-activated protein kinase (phospho-MAPK), and phosphorylated c-Jun (A.R. Kriegstein and T.A. Weissman, unpublished). These cells were not co-labeled with neuron-specific markers, so it is possible that they were M-phase radial glia rather than translocating neurons. Nadarajah and colleagues performed real-time imaging of migrating cells in slice cultures from mice at embryonic day (E)13 and E14 and distinguished translocating cells from locomoting cells [56]. However, other real-time imaging experiments performed in the E14 mouse have provided contradictory results reporting that the majority of neurons migrate either through translocation [62] or through non-translocation modes [41]. These contrasting findings could be due to differences in cell-labeling techniques, experimental procedures or conditions, as well as in cell-type identification methods.

Translocation has been proposed to be the major mode of migration during the earliest stages of cortical development [46,72]. This model implies that the early-generated preplate neurons or deep layer neurons begin migration from the VZ with a leading process fixed at the pial surface. This might be particularly true during early cortical development when the length of the leading process of a migrating neuron approximates the width of the developing cortical mantle. The early-born neurons would thus be restricted to migrating radially to the cortical plate. In this way the cortical VZ would provide a ‘protomap’ for the location of the early-generated preplate neurons [59,73]. This attractive model could explain why in some neuronal migration disorders the early-forming preplate develops normally but the later-generated cortical plate, when neurons rely more on locomotion, becomes disordered [74]. Real-time imaging experiments performed during the early stages of preplate generation are needed to provide a more complete picture of the roles of locomotion and translocation in cortical development.

Pattern of radial migration in the cortex: distinct phases of locomotion
It has long been suspected that cortical pyramidal neurons arise from the proliferative zones of the dorsal
telencephalon [75]. This has been confirmed in recent experiments including those based on cortical explants isolated from ventral telencephalon [13], fate-mapping studies of transgenic mice [14], and transcription factor expression in pyramidal neurons [15]. Recent studies using time-lapse imaging in slice cultures have demonstrated that neurons generated in the cortical proliferative zones at later stages of development pass through a series of distinct migrational stages characterized by abrupt changes in cell shape, direction of movement, and speed of migration as they move to the cortical plate [41,42] (Figure 2). These observations have led to a scheme that divides radial migration into four distinct phases [42]. In phase one, neurons generated at the ventricular surface move radially away from the ventricle to the SVZ. In phase two, they pause in the IZ–SVZ for as long as 24 h and become multipolar. Previous studies based on birth-dating techniques had also noted that migrating neurons paused or ‘sojourned’ in the lower IZ–SVZ during radial migration [76]. Time-lapse imaging of cells in phase two demonstrates that multipolar cells are highly dynamic, extending and retracting processes and moving within the SVZ. Neurons do not appear to be tightly adhered to radial glial guide fibers at this stage, and are capable of moving tangentially [41]. In some cases cells have been observed to move several cell bodies laterally only to return to their original locations [42]. Many, but not all, neurons pass through a third phase, during which they extend a process towards the ventricle and often also translocate the cell body toward the ventricle [42]. Upon reaching the ventricle, neurons enter phase four of migration. The neurons reverse polarity and extend a pia-directed leading process, take on the bipolar morphology of migrating neurons, and begin radial migration to the cortical plate [42]. Some neurons are observed to begin radial migration following a migratory pause in the SVZ but without retrograde motion toward the ventricle. According to the proposed scheme, these neurons do not pass through phase three, but instead progress directly from phase two to four (Figure 2). This indicates that subsets of excitatory neurons undergo distinct routes of migration to the cortical plate. Phase four migration to the cortical plate appears to be gliophilic and neurons take on the migratory characteristics of radially guided locomoting cells [42].

Transitions between phases of migration
Transitions between phases of migration might involve environmental signals and depend upon specific intracellular events. Moreover, specific neuronal migration disorders might involve failure of neurons to transition from one phase to another [7]. For example, the condition known as doublecortex (subcortical band heterotopia) could involve arrest of neurons in phase two of migration. Recent observations using in utero electroporation of VZ precursor cells with small-interfering RNA (siRNA) of doublecortin protein resulted in premature arrest of migrating multipolar neurons in the IZ–SVZ [77]. This observation is consistent with neurons failing to make the transition from phase two to phase three of migration. Doublecortin protein has a role in microtubule stabilization and has been proposed to have roles in nuclear translocation or process elongation [78–81]. Because dysfunction of doublecortin is thought to produce disruptions in microtubule organization and/or dynamics, these observations suggest that the initial movement of cells from the ventricle to the SVZ and the multipolar ‘sojourn’ phase might be relatively independent of doublecortin function. However, directed radial migration towards the cortical plate or ventricle might depend on doublecortin-dependent microtubule reorganization.

Birthdate-dependent laminar fate for both pyramidal neurons and interneurons
The cortex develops in an inside-out manner, with early-generated neurons occupying deep layers and

![Figure 2](https://www.sciencedirect.com)
late-generated neurons migrating beyond the early-generated neurons to occupy more superficial layers [9]. Birthdate labeling experiments confirm that cortical interneurons and pyramidal cells both follow the inside-out layering sequence [10–12]. An exception appears to be the somatostatin-expressing interneurons that appear to be born early [82]. The realization that GABAergic neurons destined for the cortex arise in the ventral telencephalon and migrate long distances tangentially raises the question of how these cells come to occupy the same cortical layers as radially migrating pyramidal cells born at the same time. The ‘sojourning’ of pyramidal neurons in the SVZ during phase two might delay their migration sufficiently for isochronically-generated GABAergic cells to complete their tangential migration into the dorsal cortex, allowing both cell types to reach the same layer at the same time (Figure 3).

The convergence of the migratory routes of at least some interneurons and pyramidal neurons at the ventricle could provide a mechanism to coordinate their migration. It has been suggested that the ventricle-directed migration of interneurons might serve to direct interneurons to the appropriate cortical layer [37]. Could the pyramidal neurons that undergo similar ventricle-directed migration do so for a similar purpose? Heterochronic transplant experiments involving ferret neocortex suggest that the laminar fate of pyramidal neurons arising in the cortical VZ is indeed determined by local epigenetic factors but, at least for early-generated neurons, commitment to laminar fate is determined before the final cell division [83,84]. If this is so, environmental signals during migration cannot play a role in layer-determination for these cells. More recently, the laminar fate determination of interneurons has been examined. Heterochronic transplant experiments were performed using MGE donor cells transplanted into a cortical VZ host environment [85]. Transplantation of MGE interneuron progenitors indicated that late-born interneurons could change their laminar fate when transplanted to a younger environment, suggesting that extrinsic signals can influence laminar fate decisions for these interneurons. But when early-born interneurons that had undergone S-phase in the young donor environment were transplanted into an older host, they maintained the lower-layer fate of the donor. Therefore, at least for later born interneurons, local epigenetic signals in the MGE might help determine laminar fate. It is thus possible that both interneurons and pyramidal cells undergo ventricle-directed migration to acquire laminar fate determining signals. Alternatively, because late-born cortical progenitors are restricted to populating upper layers only [83,86], whereas late-born interneurons appear uncommitted [85], interneurons could contact specific pyramidal neurons during the ventricular phase of migration, and simply follow them to the appropriate cortical layer. It is also possible that contact of GABAergic and glutamatergic neurons in the VZ could help establish the characteristic local circuit connections [87].

**Tangential dispersion of cortical-derived neurons could occur in phase two**

Cortical neurons arise from radial glia [60–64] and daughter neurons often migrate along the parental fiber [60]. However, this is not the rule, and migrating neurons also travel along parallel adjacent paths, presumably using adjacent radial glial fiber guides [42,88]. In rodents, clonally related neurons become tangentially dispersed, particularly in the upper layers, as reflected by the conical appearance of columns of related cells in chimeric mouse models [20]. Using retroviral lineage analysis with a

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**Figure 3.** Migratory patterns of interneurons and pyramidal neurons converge in the dorsal cortex. This scheme depicts the apparent convergence of the migratory patterns of interneurons (red) and pyramidal cell movements (dark green) in the cortex. Subsets of both cell types display ventricle-directed migration followed by radial movement to the cortical plate (CP). Interneurons might migrate radially along unrelated, adjacent radial glial cells (grey) to reach the cortical plate. Abbreviations: IZ, intermediate zone; MZ, marginal zone; R, radial glial cell; SVZ, subventricular zone; VZ, ventricular zone.
library of genetically tagged retroviruses, Walsh and Cepko demonstrated that clones originating from a single progenitor could disperse progressively over time [89]. Although most remained relatively clustered, others were dispersed by distances of ≥2 mm [89] and could be distributed across widespread functional areas [90]. The recent findings that cortical interneurons from the ganglionic eminence migrate tangentially in the dorsal cortex indicate that these cells could contribute to the tangential dispersion of clonal cells. Even so, the relative contributions of interneurons and pyramidal cells to the patterns of clonal dispersion have not yet been determined, and other mechanisms might also account for dispersion of clonal cells in the dorsal cortex. For example, evidence indicates that tangential dispersion might occur among progenitor cells in the ventricular zone [91,92] and/or postmitotic neurons in the dorsal cortex [89,93].

Time-lapse imaging of migratory behavior in slice cultures has confirmed tangential dispersion of cortical neurons [39]. It has been demonstrated that the majority of newborn neurons move radially to the SVZ (in phase one) [38,42]; thus, it is likely that tangential dispersion of postmitotic neurons occurs during later phases. Neurons could disperse tangentially in phase two of migration, because this is a phase characterized by dynamic ‘exploratory’-type cell movements and a phase where tangential movements have been observed in slice-culture preparations [41,42]. Subsequently, cells migrating tangentially in phase two could account for the migrating postmitotic neurons observed in the VZ, SVZ and lower IZ [38,39]. It is possible that, in addition to postmitotic neurons, some tangentially dispersed cells might also be intermediate precursor cells [42]. In the first case, this would account for single dispersed neurons; in the second, for periodic related cell clusters [89,93]. It is also possible that cells in phase four might be able to follow tangential or oblique migration paths or to jump between radial glial fibers and become dispersed, but it is unlikely that neurons would become widely distributed during this phase of radial-glia-guided migration.

Lower IZ–SVZ might favor tangential migration of both interneurons and pyramidal cells

Tangential migration occurs at all depths of the cortex, but particularly high percentages of tangentially migrating cells have been observed in the SVZ and lower IZ by most observers [19,27,29,36,45,94]. The neocortical SVZ and lower IZ have thus been proposed to serve as a main corridor for the tangential migration of cortical interneurons [27,38]. Interestingly, the lower IZ–SVZ also represents a decision point for radially migrating neurons, which pause in this region and either resume radial or take a tangential path [41,42,76,94]. The IZ–SVZ is also a zone where many neurons become branched and dynamically extend and retract processes. This is the case for pyramidal neurons in the multipolar phase (phase two) of migration [41,42], and also for interneurons migrating into the cortical IZ–SVZ from the striatum [26,72]. Possibly the lower IZ–SVZ is a region rich in morphogenetic signals that influence the behavior of both interneurons and pyramidal cells.

Concluding remarks

Patterns of neuronal migration during cortical development appear more complicated than once thought. Cortical interneurons take a predominantly tangential path to reach the cortex and travel relatively long distances, whereas pyramidal cells take a predominantly radial path and reach the cortex more directly. Detailed studies of the dynamic movements of both cell types have revealed that cells undergo distinct phases in migration. The transitions between these phases might signal changes in the mechanisms of cell guidance or motility. Moreover, it is clear that there are heterogeneous patterns of migration for both interneurons and pyramidal cells. This observation suggests that specific cell types take distinct migration paths. Unraveling the diversity of migrational strategies displayed by cortical neurons could allow a more complete understanding of the mechanisms that contribute to neuronal migration disorders.

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