that are now restricted to the flowering plants in what appear to be Jurassic deposits. But does this fossil taxon provide insights into angiosperm ancestry based on retained characters shared with nonangiosperms? *Archaefructus* does not share substantial features with modern Gnetales, adding some support to recent nucleic acid sequence–based phylogenies that challenge a close gnetalean-angiosperm relationship (*13*, *14*). Comparisons with bennettitalean reproductive structures also fail to support the possibility of a close relationship. Having found little to link the fossils with the anthophytes, the authors succumb to the temptation to compare the fossils with seed ferns because of some similarities. Yet there is a gap, and the fossils indicate that angiosperms were isolated from other seed plants by the end of the Jurassic, suggesting that it will be difficult to reconstruct ancient angiosperms from comparative studies based only on existing taxa (*16*).

But what are the implications of these fossils for relationships within angiosperms? Do they favor any of the groups that have been hypothesized as primitive? Sun *et al.* note that *Archaefructus* lacks some features of the Chloranthaceae (*1*). It is also substantially different from the flowers or fruits of other putative basal angiosperms. However, *Archaefructus* shares certain characters with Magnoliales and even Magnoliaceae. They may prove even more like the flowers of Magnoliales if stamens are found attached at the base of the floral axis in new specimens, but with a substantial difference. The fossils are a combination of strongly magnolialean characters and a notable nonmagnolialean one: a missing perianth, an unusual condition found only in some species in the families Chloranthaceae and Piperaceae (ironically, families competing with Magnoliales for primitive stature within the angiosperms). This combination of characters does not occur in any extant group of flowering plants, and Sun *et al.* appropriately recognize a new subclass of angiosperms on the basis of *Archaefructus*.

This is potentially a big discovery. Although its age and chimeric nature imply that *Archaefructus* may represent the most "primitive" angiosperm yet discovered, final confirmation of its basal status and its angiospermous nature depend on precise phylogenetic context. Phylogenetic analysis is also necessary to rationalize the conflicting combination of characters now found in opposing models of primitive angiosperms. Can such an analysis be accomplished to everyone's satisfaction with the characters now available in *Archaefructus*? Probably not, because too many important characters are now missing, especially stamen position and structure, pollen morphology, and leaf and seed structure. However, through my experience in fossil collecting, I have learned that the discovery of a few specimens of a new fossil taxon is seldom a unique event—there will be new specimens of *Archaefructus* and the kinds of characters critically needed are also the kinds likely to be preserved. Given the potential informative value of this taxon and the recent pace of innovation in studies of angiosperm systematics and paleobotany today, I predict that the great "abominable mystery," with us for over 100 years, will not last another 10.

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PERSPECTIVES: NEUROSCIENCE

The When and Where of Floor Plate Induction

Jane Dodd, Thomas M. Jessell, Marysia Placzek

The floor plate is a transient embryonic organizing center located at the ventral midline of the neural tube that profoundly influences the development of the vertebrate central nervous system. The specialized histological features of floor plate cells have long been recognized (*1*), but only comparatively recently have the remarkable patterning activities of this ventral midline neural cell group been revealed. Floor plate cells serve as a source of Sonic hedgehog, a cell surface and secreted protein that acts at distinct concentration thresholds to specify the identities of motor neurons and interneurons (*2*). In addition, floor plate cells secrete netrin-1, a chemotropic factor that directs the axonal trajectories of commissural interneurons and certain motor neurons (*3*). Appreciation of the specialized signaling properties of the floor plate has thus brought an enhanced interest in the origins of this neural organizing center.

Many studies have provided evidence that the differentiation of the floor plate requires inductive signals provided by axial mesodermal cells of the notochord that lie under the midline of the neural plate (*4*). Notochord signals can induce floor plate differentiation both in vitro and in vivo. Conversely, selective elimination of the notochord in vivo, without removal of floor plate precursors, results in the failure of floor plate differentiation (*4*). On the basis of these findings, a relatively simple view of floor plate differentiation initially emerged, emphasizing the notochord as a key cellular source of inductive signals. More recent data, however, suggest that there may be more to floor plate differentiation than a single inductive signal provided by the notochord. Indeed, one recent review has questioned the entire concept of induction of the floor plate (*5*).

Here we discuss recent advances in the understanding of the molecular steps of floor plate development, findings that have begun to shed additional light on the timing and position within the embryo at which floor plate differentiation is initiated. We argue that while these findings may indicate new complexities, they nevertheless do not erode the basic case for the operation of an inductive signal that directs floor plate differentiation. The issues at stake can be reduced to three basic questions: Does inductive signaling have a critical role in floor plate differentiation? What are the molecules that control floor plate differentiation? When and where is floor plate differentiation initiated?

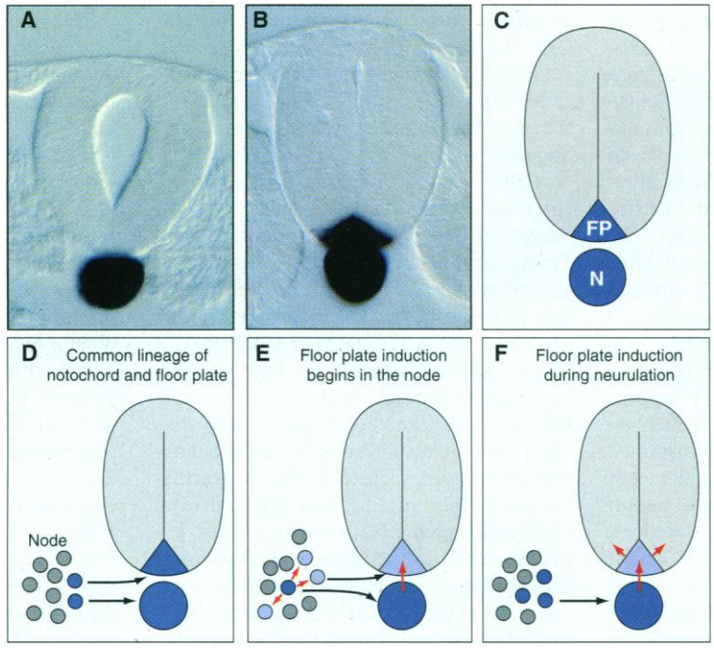
J. Dodd is in the Department of Physiology and Cellular Biophysics and Center for Neurobiology and Behavior, Columbia University, New York, NY 10032, USA. T. M. Jessell is at the Howard Hughes Medical Institute, Department of Biochemistry and Molecular Biophysics, Columbia University, New York, NY 10032, USA. E-mail: tmj1@columbia.edu. M. Placzek is in the Developmental Genetics Programme, Krebs Institute, University of Sheffield, Sheffield S10 2TN, UK.

Induction Between Linearly Related Cells

What is the contribution of inductive signaling to floor plate differentiation? It has been clear for some time that the notochord and floor plate do not fit easily into standard views of inducing and responsive cell groups. In large part this is because many of the key molecules that characterize floor plate cells are expressed at an earlier stage by the notochord (*4*). The discovery of the striking conservation of molecular properties by midline mesodermal and neural cells some time ago (*6*) raised the general issue of whether the notochord and floor plate derive from a common progenitor cell in the gastrula embryo and whether they acquire their characteristic properties simply as a consequence of their shared lineage, independent of any inductive signaling process. If this were the case, the observed dependence of floor plate differentiation on the notochord in vivo could be argued to reflect the incorporation of notochord-like cells into the ventral midline of the neural tube. It is primarily these issues that have resurfaced recently.

Fate mapping studies in vertebrate embryos have shown that precursor cells that give rise to both the floor plate and notochord can indeed be found in the node-organizer region of the embryo (*7–9*). Only those cells in the superficial layer of the node, however, are fated to generate both notochord and floor plate cells, and once cells ingress into the mesodermal layer of the node they contribute only to the notochord (*8*). These findings argue against a late contribution of prospective notochord cells to the floor plate. More generally, such fate mapping studies reveal little about the state of commitment of cells in and around the node. The common lineage of notochord and floor plate cells may be an indication merely of the fact that cells located at the midline of vertebrate embryos fail to disperse laterally (*10*). Thus, fate maps and lineage tracing alone do not provide evidence against inductive signaling.

A key discovery that both supported the involvement of inductive signaling and provided a better molecular understanding of floor plate induction was the identification of Sonic hedgehog (Shh), a member of the *Hedgehog* (*Hh*) gene family. Shh is expressed initially by cells in the node, later by axial mesodermal cells, and finally by floor plate cells themselves (see the figure, panels A to C). Shh can induce the ectopic differentiation of floor plate cells from neural precursors both in vivo and in neural plate tissue in vitro (*4*). Importantly, the concentration of Shh needed to induce floor plate differentiation is higher than that required for the generation of other ventral cell types (*2*, *11*). These ectopic expression studies showed that Shh has all the properties expected of a floor plate–inducing factor. Moreover, Shh activity is required for floor plate differentiation in chick and mouse embryos: Inactivation of Shh signaling through the use of antibodies to Shh or by the targeted inactivation of the *Shh* gene leads to the failure of floor plate differentiation (*12*, *13*). Significantly, the loss of Shh signaling prevents floor plate differentiation without obviously affecting the early development of the notochord, providing genetic evidence that the pathway of floor plate development differs from that of the notochord. Similarly, inactivation of the gene encoding the zinc finger transcription factor Gli2, a component of the downstream Shh-signaling pathway, blocks floor plate differentiation without perturbing the development or apparent signaling properties of the notochord (*14*, *15*). These findings argue against the extreme model (see the figure, panel D) in which floor plate and notochord cells are equivalent, committed descendants of a common node progenitor. Instead they reveal a selective requirement for Shh activity in floor plate differentiation and strongly implicate intercellular signaling in this differentiation process.



Possible mechanisms of floor plate development in the chick embryo. Panels A and B show transverse sections through neural tube–stage chick embryos. *Shh* gene expression is detected by in situ hybridization histochemistry. (**A**) Restriction of *Shh* expression to the notochord at early stages of caudal neural tube development. (**B**) Expression of *Shh* by both the notochord and floor plate at late stages of neural tube development. (**C**) The position of notochord (N) and floor plate (FP) cells. (**D**) A model of floor plate development in which floor plate and notochord cells derive from a common progenitor cell in the node, independent of inductive signaling. (**E**) A model in which the Hedgehog-mediated induction (red arrows) of floor plate cells begin in the node and continues after neural tube closure. (**F**) A model in which the induction of floor plate differentiation occurs primarily after neural tube closure. Floor plate cells may also serve as a source of Hedgehog signals that induce additional neural tube cells to acquire a floor plate fate.

Timing of Floor Plate Induction

Studies of Shh signaling in mouse and chick embryos have not, however, resolved the major question of the time and place at which floor plate differentiation begins. Because notochord and floor plate share many molecular properties, notably expression of the winged helix transcription factor HNF3β and Shh (*4*), individual precursors of these two cell groups cannot be distinguished within the node. Nevertheless, the expression of such genes by node cells leaves open the possibility that the induction of floor plate differentiation begins within the node itself (*16*). For example, it is possible that a subset of cells within the node that expresses Shh induces adjacent cells to embark upon a program of floor plate differentiation (see the figure, panel E). Alternatively, there may not yet be a distinction between notochord and floor plate precursors within the node, in which case Shh signaling could act in a stochastic manner to direct a subset of equivalent node cells to a floor plate fate. The possibility that floor plate differentiation begins in the node is, of course, still compatible with a requirement for Shh-mediated inductive signaling.

Several lines of evidence, however, argue against the idea that the specification

of floor plate cells occurs exclusively within the node, at least in avian embryos. A major fraction of cells destined to populate the floor plate reside in a region anterior to the node in gastrula embryos (*7*, *17*). These more anterior cells do not yet express definitive floor plate markers (*16*), nor do they acquire floor plate properties when grown in isolation (*4*). The differentiation of these anterior cells into floor plate thus appears to take place outside the node and at a later developmental stage (*7*). The importance of a later contribution of signals from the axial mesoderm is also consistent with the finding that at most axial levels, prospective floor plate cells do not express the full complement of floor plate properties, including Shh and netrin-1, at the time that they first occupy the ventral midline of the caudal neural tube, even though such markers are expressed by the notochord at this stage (see the figure, panel A). When taken together with the absence of a floor plate after selective notochord removal, these results support the idea that the progression of floor plate differentiation requires a later or sustained period of signaling from the axial mesoderm as it extends under the caudal neural tube (see the figure, panel F). Moreover, cells in more lateral regions of the neural plate and neural tube can acquire floor plate properties even at much later stages of development if they migrate medially and populate the ventral midline of the neural tube (*18*). Finally, floor plate cells themselves can induce the differentiation of more lateral neural tube cells to acquire a floor plate fate (*19*) (see the figure, panel F), a process of homeogenetic (like-begets-like) induction that may underlie the marked increase in the number of floor plate cells that occurs after neural tube closure (*19*) and may ultimately reinduce floor plate cells after notochord removal. Thus, a substantial proportion of floor plate cells appear to be specified relatively late and to derive from progenitor cells that reside within the neural epithelium itself rather than within the node.

Insights from Zebrafish Mutants

Although the analysis of floor plate differentiation in avian and mammalian embryos presents a reasonably coherent picture of this inductive process, recent genetic analyses of zebrafish development suggest that in this organism, the pathway of floor plate differentiation may be more complex (*5*). Null mutations in the zebrafish *Shh* gene, otherwise called *sonic you*, eliminate a group of lateral floor plate cells but leave intact a more medial strip of floor plate cells (*20*). At first glance, these results might be construed as evidence against an essential role for *Hedgehog* signaling in floor plate induction. However, zebrafish embryos, in contrast to their amniote counterparts, express two other hedgehog genes, *echidna hedgehog* (*ehh*) and *tiggywinkle hedgehog* (*twhh*), in midline mesodermal and neural cells, respectively. The *twhh* gene, like *Shh*, is initially expressed by cells in the embryonic shield (the zebrafish counterpart of the node), and later *ehh* is expressed by axial mesodermal cells (*21*, *22*). It remains possible, therefore, that multiple *hedgehog* genes cooperate in the induction of floor plate cells in zebrafish whereas *Shh* is solely responsible in avian and mammalian embryos.

Other zebrafish mutants, notably *no tail (ntl)* and *cyclops* (*cyc*), also have a profound influence on the differentiation of the midline. Their phenotypes raise the issue of whether floor plate development can proceed in the absence of inductive signaling from the axial mesoderm (*5*). The *ntl* gene encodes a T-box protein closely related to the mammalian brachyury (T) protein, and like mouse *brachyury* mutants, *ntl* mutants also exhibit defective notochord development (*23*, *24*). Nevertheless, the floor plate is present and even overrepresented (*24*). One suggested explanation for the persistence of floor plate differentiation in the absence of the notochord in *ntl* embryos is that axial mesodermal cells are still present at the midline, despite the absence of overt structural features of notochord differentiation. Consistent with this idea, cells underlying the midline of the neural plate still express *Hedgehog* genes (*24*). Alternatively, *ntl* function may normally be required to promote the formation of axial mesoderm, with the consequence that in *ntl* mutants, unspecified progenitor cells within the node are still capable of responding to local Hh signaling but generate only floor plate cells. Thus, the phenotype of the *ntl* mutation does not argue against a requirement for Hedgehog-mediated inductive signaling in floor plate generation, but instead would seem to indicate that a differentiated notochord is not a required source of such signals, at least in zebrafish.

Cyc mutant embryos exhibit a midline phenotype that in some ways is complementary to that of *ntl* embryos, in maintaining a notochord but lacking a floor plate, at least at early developmental stages (*25*). Initially, mosaic analyses suggested that the loss of floor plate cells in *cyc* mutants resulted from a perturbation in the ability of neural cells to respond to axial mesodermal signals (*25*). The *cyc* gene has, however, recently been shown to encode a nodal-related TGFβ superfamily member that is expressed by cells in the embryonic shield but not later by neural tube cells (*26*, *27*). Moreover, the rescue of floor plate differentiation appears to require *cyc* expression in embryonic shield cells rather than in neural cells (*27*). How then may *cyc* act? The finding that *cyc* embryos possess a notochord and express *Hh* genes yet lack a floor plate could reflect the existence of a *Hedgehog*-independent but *cyc*-dependent pathway of floor plate differentiation in zebrafish (*27*). Alternative possibilities are that *cyc* function is required to control the proliferation of axial mesodermal cells or to maintain their inductive signaling properties. In this context, the high Shh concentration threshold requirement for floor plate differentiation (*2*, *11*) could mean that a partial attenuation of Hh signaling from the axial mesoderm in *cyc* embryos would lead to the loss of floor plate cells but the preservation of other ventral cell types: the cellular phenotype of *cyc* mutants. A third possibility is that *cyc* signaling controls the expression of a mesodermal signal that acts in parallel with Hh proteins, perhaps by sensitizing neural cells to Hh signaling. The possibility that *cyc* regulates organizer development is supported by the recent finding of a second nodal-related gene, *squint* (*sqt*), that exhibits partially redundant functions with *cyc* in the formation of the embryonic shield (*28*).

An additional gene required for floor-plate formation is *one-eyed pinhead* (*oep*), which encodes an epidermal growth factor (EGF)–related protein (*29*–*31*). The failure of floor plate differentiation in *oep* mutants may reflect a cell-autonomous role in floor plate precursors themselves (*30*) or a function in the control of axial mesoderm differentiation (*29*), or both. Thus, despite marked defects in floor plate differentiation in many zebrafish mutations, the cellular and molecular analysis of these mutant phenotypes leaves open the possibility that these genes are involved primarily in the regulation of axial mesoderm differentiation, affecting floor plate differentiation only secondarily. Consequently, floor plate differentiation in zebrafish may operate under guidelines more closely related to those in avian and mammalian embryos than has recently been envisaged (*5*, *20*).

Does this mean that the complete picture of floor plate differentiation is now apparent? Almost certainly not. Many aspects of the early cellular interactions that control the decision of axial midline cells to embark upon distinct pathways of notochord and floor plate differentiation need to be defined more clearly. In addition, there may be factors expressed by axial mesodermal cells that regulate the perception of Hh signals by neural cells and, if

so, their characterization could help to clarify many of the unresolved issues discussed above. Zebrafish genetic screens have identified many additional mutations that perturb midline mesodermal and neural differentiation (*32*), and it seems likely that the molecular analysis of some of these mutants will reveal novel components in the pathway of floor plate differentiation. The need to define the relative contributions of Hh-dependent and -independent signaling to floor plate differentiation should maintain this intriguing cell group at the center of developmental studies for some considerable time.

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33. M.-A. Teillet, F. lapointe, and N. Le Douarin [*Proc. Natl. Acad. Sci. U.S.A.* **95**, 11773 (1998)] have recently suggested that the failure of floor plate differentiation after notochord removal results from the coincident elimination of floor plate precursors. However, in many previous studies of the consequences of notochord ablation (*4, 16*) in which notochord cells alone were removed, leaving the node and cordoneural hinge intact, floor plate differentiation still failed to occur. Thus, it is unlikely that the absence of floor plate differentiation in these previous studies is attributable to the removal of floor plate precursors.

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PERSPECTIVES: BOSE-EINSTEIN CONDENSATION

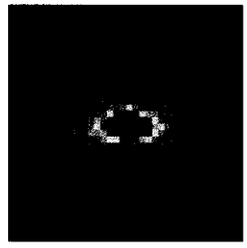
Go Forth and Multiply

Keith Burnett

In Bose-Einstein condensed systems, instead of each atom occupying its own quantum world, they have all entered a single macroscopic quantum state. Over the past several years, exploration of the remarkable properties of these gases has proceeded apace. The first Bose-Einstein condensed atomic gases were produced in 1995 at JILA by the group of Eric Cornell and Carl Wieman (*1*). In these experiments, rubidium atoms were trapped in a magnetic field and cooled to nanokelvin temperatures. Since then other scientists around the world have succeeded in producing condensates in a variety of traps and with several alkali atoms (*2*). At the Massachusetts Institute of Technology (MIT), earlier this year, a condensate has even been produced in atomic hydrogen (*3*). And now, as reported on page 1686 of this issue, Anderson and Kasevich at Yale have demonstrated the Bose-Einstein equivalent of the well-known Josephson effect in superconductivity (*4*).

Greytak and Kleppner's work with hydrogen (*3*) has attained a goal that provided the initial stimulus for the whole search for Bose condensation in the 1970s. These developments herald a new field of coherent matter wave physics that is moving into fresh areas as the range of systems and the degree of control over them steadily expand. In recent studies the macroscopic nature of the wave function of the atoms, or matter wave coherence, has been examined in detail: Why is this such an interesting issue?

Bose-Einstein condensation is one of the most intriguing phenomena one can see in physical systems. When it takes place, the wave-mechanical properties of atoms are amplified to levels at which they can be observed and manipulated directly. We know that in the microscopic world quantum mechanics rules the waves. In most circumstances, however, we can only infer the shape of the waveforms that control the motion of particles in the microscopic world. It is, however, sometimes possible to see these waveforms writ large and accompanied by phenomena such as superfluidity and superconductivity. The macroscopic nature of these quantum mechanical systems is at the heart of the phenomena. It occurs when the individual matter-waves of the constituents of a material start to overlap. If the particles are bosons (more precisely, particles with integral spin that obey Bose-Einstein statistics), they can jump into a shared waveform that we term a condensate: hence, the term Bose-Einstein condensation. In the atomic gases the individual waves get bigger as the gas is cooled and energy is removed from the particles. If the particles are cold enough the waves then overlap and condensation occurs. We can look then directly at the quantum waveform and determine its shape without destroying it. Such direct access is not usually possible because waveforms usually have just the one owner and measuring them destroys them. If, however, we can get many atoms to share the same wave function, observing it just knocks out some of the atoms without destroying the waveform. The wave is then a macroscopic thing: looking at it does not eliminate it.



Cold condensate. Spatial image of a rubidium-87 condensate just below the transition temperature. The condensate contains ~10^4 atoms and has a ~9-μm waist along the horizontal axis. The non-condensed fraction is also visible.

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Bose-Einstein condensed atomic assemblies are particularly interesting and useful for the study of these macroscopic quantum effects. First, the interactions between the particles have only a very small effect on the behavior of the atoms, and nearly all of them share the same waveform. The small effect of interactions also means that it is possible to make first principle calculations of the properties and the behavior of the assembly. (For other systems where condensation occurs, such as liquid helium, the effects of interactions is very large and theory is not so straightforward.) The theory is being subjected to stringent quantitative tests, and for low temperatures the theory has emerged with flying colors. For higher temperatures the atomic Bose-Einstein

The author is in the Department of Physics, University of Oxford, Oxford OX1 3PU, UK. E-mail: k.burnett1@physics.ox.ac.uk