Pattern formation during gastrulation in the sea urchin embryo

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Summary

The sea urchin embryo follows a relatively simple cell behavioral sequence in its gastrulation movements. To form the mesoderm, primary mesenchyme cells ingress from the vegetal plate and then migrate along the basal lamina lining the blastocoel. The presumptive secondary mesenchyme and endoderm then invaginate from the vegetal pole of the embryo. The archenteron elongates and extends across the blastocoel until the tip of the archenteron touches and attaches to the opposite side of the blastocoel. Secondary mesenchyme cells, originally at the tip of the archenteron, differentiate to form a variety of structures including coelomic pouches, esophageal muscles, pigment cells and other cell types. After migration of the secondary mesenchyme cells from their original position at the tip of the archenteron, the endoderm fuses with an invagination of the ventral ectoderm (the stomodaem), to form the mouth and complete the process of gastrulation. A larval skeleton is made by primary mesenchyme cells during the time of archenteron and mouth formation.

A number of experiments have established that these morphogenetic movements involve a number of cell autonomous behaviors plus a series of cell interactions that provide spatial, temporal and scalar information to cells of the mesoderm and endoderm. The cell autonomous behaviors can be demonstrated by the ability of micromeres or endoderm to perform their morphogenetic functions if either is isolated and grown in culture. The requirement for cell interactions has been demonstrated by manipulative experiments where it has been shown that axial information, temporal information, spatial information and scalar information is obtained by mesoderm and endoderm from other embryonic cells. This information governs the cell autonomous behavior and places the cells in the correct embryonic context.

Key words: sea urchin, pattern formation, gastrulation, endoderm, mesoderm.

Introduction

Each of the deceptively simple morphogenetic events of gastrulation is driven by a number of interactions that are complex at both the molecular and cellular levels. For example, the mesodermal and endodermal lineages are partitioned early by a precise spatiotemporal sequence of cleavage divisions that divide the egg along the animal-vegetal axis. At the 16-cell stage, micromeres at the vegetal pole contain information that allows them to form the skeleton, even if these micromeres are isolated and grown in culture (Ökazaki, 1975). In a beautiful series of manipulations, Horstadius (1939) showed that cell interactions are crucial for maintaining the correct distribution of cell fates along the animal-vegetal axis. If cells along that axis are removed, cells toward the animal pole alter their normal cell fates and replace structures formed by the missing vegetal cells. Horstadius even found that if he placed micromeres at the animal pole they would induce a secondary axis (Horstadius, 1939).

Recently, cell marking experiments have shown that the dorsal-ventral axis is also specified very early during cleavage (Cameron et al., 1989), although, like the animal-vegetal axis, the dorsal-ventral axis can be modified experimentally until some time during gastrulation (see below). To the embryo these axial asymmetries are important because they provide blastomeres with spatial information necessary for organized cellular rearrangements. This information is somehow supplied to each cell and then used to perform a function appropriate to that cell’s particular position in the embryo.

Recent progress, especially in the Drosophila embryo (Ferguson and Anderson, 1991; St Johnston and Nusslein-Volhard, 1992; Wharton and Struhl, 1991; Wang and Lehmann, 1991; Nusslein-Volhard, 1991) and in the frog embryo (Cooke, 1991; Cho et al., 1991; Green and Smith, 1991) have begun to identify genes and growth factors that appear to be involved in specification of the embryonic axes. Presumably then, in those embryos as well as in the sea urchin, the cells, upon receiving the appropriate axial information, go on to perform the embryonic function appropriate to that position. If specification were that
Fig. 1. Gastrulation in the sea urchin embryo. Primary mesenchyme cells have ingressed from the vegetal plate, through the basal lamina lining the blastocoel, and begin migrating along the blastocoelar wall (a). The thickened vegetal plate then bends inward to begin the process of invagination (b). During invagination endoderm cells converge and extend to elongate the archenteron. The filopodial extensions of secondary mesenchyme cells are not necessary for this phase of elongation (c). The archenteron reaches the wall of the blastocoel using filopodial contraction to complete the last one third of the elongation (d) (Hardin, 1988). In the final stage of gastrulation, the tip of the archenteron makes contact with a stomodal invagination and will soon form the mouth (e).

simple, it should be possible to isolate a cell after it has been specified and (assuming the cell is healthy), it should carry out its eventual fate. This rarely happens, however, probably because, although axial information is very important, many other interactions influence the developmental repertoire of a given cell. Here we examine primary mesenchyme cells, secondary mesenchyme cells and endoderm of the sea urchin embryo to ask what combination of cell autonomous behaviors and cell interactions are necessary to form and position the archenteron, and to form the embryonic skeleton. How simple or how complicated is the information necessary for morphogenesis? As our experimental story has developed it has become increasingly clear that these cells utilize a mixture of cell autonomous activities with simultaneous input through a number of cell interactions to regulate pattern.

Invagination of the archenteron

Invagination of the archenteron occurs in three recognizable stages (Fig. 1). First, a thickened plate of the cells at the vegetal pole of the embryo bends inward (Burke et al., 1991). Recent experiments have suggested that movements lateral and more animal to the site of inward bending may provide the driving force for this initial shape change in the vegetal plate. Burke et al. (1991) have shown that, just prior to the first signs of invagination, cells along the sides of the embryo move toward the vegetal plate suggesting that forces from all directions bearing down on the vegetal plate could provide the force that initiates inbending of the vegetal plate. It is hypothesized that somehow the forces generated by the movements of the blastular cells are focused on the vegetal plate so that inward bending is initiated at the center of the plate. Other experiments indicate that there are no additional movements of cells from lateral to the vegetal plate once the initial inbending is started. Marking experiments originally performed by Horstadius (reviewed by Horstadius, 1973) and repeated by Ettensohn (1984) have shown that only the vegetal plate contributes cells to the archenteron. It has also been shown that isolated vegetal plates support invagination (Moore and Burt, 1939; Ettensohn, 1984). Thus forces in the immediate vicinity of the invagination are all that are necessary to drive further invagination (Moore and Burt, 1939; Ettensohn, 1984). Because these movements, once started, are confined to the vegetal plate, they can be said to be cell autonomous (Ettensohn, 1985b).

After the initial inward bending of the vegetal plate, the archenteron begins to elongate. During the first stage of elongation, endoderm cells rearrange by convergent-extension movements in which the cells of the endodermal sheet change location in an organized fashion that elongates the primitive gut tube. This conclusion is based on three sorts of studies. First, an ultrastructural study deduced that cell rearrangements must occur (Ettensohn, 1985a). Second, it was shown that the cell rearrangements in the archenteron do not require participation of secondary mesenchyme cells (Harkin and Cheng, 1986; Hardin, 1988). Finally, labeling studies directly showed the localized shifts in position of endodermal cells in a convergent-extension pattern of movement that elongates the archenteron (Fig. 2) (Hardin, 1989). Although these experiments include time-lapse films that directly visualize the cellular rearrangements, several

Fig. 2. The archenteron elongates by convergence-extension movements of the endoderm (from Hardin, 1989). Single labeled macromere derivatives were incorporated into embryos at the morula stage, and these cells divided to form a patch of labeled cells in the blastula (a). When the patch was located at the vegetal plate, it was included in the indentation movements of the archenteron (b), and by convergent-extension rearrangements the cells changed neighbors to form the elongated archenteron (c).
crucial properties of invagination are not known. These properties include the molecular basis of the motility involved, the importance of possible cell adhesion changes as guidance cues, and the mechanism that imparts directionality to the movements.

The second stage of elongation of the archenteron utilizes secondary mesenchyme cells located at the tip of the archenteron. These cells extend filopodia throughout gastrulation (Gustafson and Kinnander, 1956; Gustafson and Wolpert, 1967) and, though it was once thought that the filopodial extension behavior was important for all of the archenteron elongation movements, it is now appreciated that the filopodial movements are necessary only for about the last third of the elongation, at least in the species that have been examined experimentally (Hardin and Cheng, 1986; Hardin, 1988). Several experimental approaches support this conclusion, including a laser ablation study in which the filopodial extensions were destroyed without affecting archenteron elongation until it reached about two thirds of its final length (Hardin, 1988). In other species, it remains possible that filopodial extension contributes to the mechanics of early archenteron elongation. For example, in species where the archenteron invaginates close to the dorsal or to the ventral wall of the blastocoel (Hardin and McClay, 1990), filopodia may be used. In *Lytechinus*, the genus examined in our studies, the archenteron invaginates into the center of the blastocoel and, while filopodial extension could be used, experiments show filopodia are not necessary mechanistically to provide an elongation force until the final third of archenteron extension.

The archenteron tip ultimately makes contact with a specific region of the blastocoel wall. Experiments have shown that this region is a specific target that is recognized, selectively, by the filopodial attachments of the secondary mesenchyme cells (Hardin and McClay, 1990). Throughout gastrulation, filopodia are extended from the secondary mesenchyme cells in all directions. When the filopodia make contact with the wall of the blastocoel, they adhere for up to ten minutes. When they finally reach the target region, the filopodia adhere, are not released, and the behavior of the secondary mesenchyme cells changes. Only contact with the specific target region will cause the cell behavior change. This change can be brought about precociously if this region is pushed into close proximity to the archenteron tip, or the behavior change can be greatly delayed by contorting the embryo to prevent the filopodia from reaching the target region, or by removing the target region from the embryo (Fig. 3) (Hardin and McClay, 1990). These

![Fig. 3. Secondary mesenchyme cells attach to a specific target region of the blastocoel wall during gastrulation (Hardin and McClay, 1990). Lateral indentations show that although contact can be made to any part of the blastocoelar wall, only contact with a target region ends filopodial extension behavior and halts the movement of the archenteron. By indenting the animal pole one can establish contact with the target region and change the filopodial behavior precociously. By elongating the embryo one can delay contact with this region for several hours, and the filopodial behavior change is delayed as well. If released from the elongation, attachment occurs and the embryo develops normally. If the embryos remain elongated, the secondary mesenchyme cells eventually migrate away from the archenteron and collect at the target region at the animal pole.](image-url)
experiments show the existence of an anatomical target for the extension and attachment of the archenteron to the blastocoel roof. Contact with the target promotes adhesion and a change in behavior of the secondary mesenchyme cells (Hardin and McClay, 1990). Coincident with this contact-stimulated behavioral change, secondary mesenchyme cells lose their ability to change fate as shown experimentally by a loss of an ability to become skeleton-producing cells (Ettensohn and McClay, 1988; Ettensohn, 1990a). The secondary mesenchyme cells then enter a complicated migratory and differentiation pathway to give rise to the coelomic pouches, esophageal muscle, pigment cells and blastocoelic cells of unknown fate (Tamboline and Burke, 1989, 1992). It is not known whether contact with the target region provides information for these differentiation events but it is of interest to know whether these programs of differentiation can be stimulated to occur early or be delayed due to time of contact with the target.

Control over the invagination process appears to be exercised at the beginning of invagination and at the completion of the target contact period. Several inhibitors block gastrulation with the most sensitive window being just prior to the beginning of invagination (Wessel and McClay, 1987; Schneider et al., 1978). Once invagination begins both the presumptive endoderm and the secondary mesenchyme cells appear to act cell autonomously. The presumptive endoderm cells aggregate and sort out to the interior of an aggregate, if mixed with ectoderm (Bemacki and McClay, 1989). The endoderm further differentiates to form an epithelial layer that looks like gut tissue. From the aggregation and sorting data, it would appear that endoderm cells recognize one another (McCoy et al., 1977), and will form an epithelial layer either by invagination or by cavitation. Antigen expression data suggest that archenteron morphogenesis is more complicated than a relatively simple cell autonomous series of behaviors. As the gut forms, regional differences in antigen expression become apparent (McCoy et al., 1983). Monoclonal antibodies that recognize different regions of the gut appear in localized regions before invagination is completed and before there are anatomical landmarks to delineate regions of the gut (McCoy et al., 1983). Thus, while cells of the gut behave cell autonomously when the vegetal plate is isolated, cell interactions between the cells of the gut must convey directional information regarding position. In response to that information, the cells produce molecules appropriate to their location in the gut.

**Pattern formation of the skeleton**

In most species, there is an invariant number of primary mesenchyme cells that do not divide as the skeleton forms. In *Lytechinus*, 64 primary mesenchyme cells ingress from the vegetal pole, migrate within the blastocoel for a period of time, then associate with one another to form a syncytial ring around the base of the invaginating archenteron. In two ventrolateral areas of this ring, clusters of primary mesenchyme cells synthesize triradiate CaCO₃ crystals that initiate spiculogenesis. The arms of these spicules elongate, branch, and eventually form a skeleton that has a morphology with species-specific characteristics (Fig. 4). This section examines the interactions required for skeletal pattern formation.

Two kinds of studies demonstrate that skeletogenesis is largely a cell autonomous property of the primary mesenchyme cells. First, Okazaki's studies (Okazaki, 1975) demonstrated that micromeres could be isolated at the 16-cell stage, and grown in culture (with only the addition of horse serum), where they grew spicules. The spicule rods were not precisely of the pattern produced in vivo, but they had some of the pattern properties that are normally displayed in vivo.

Cell autonomy has been demonstrated recently in a different way. Primary mesenchyme cells from an embryo of one species were transplanted to the blastocoel of a primary mesenchyme-depleted embryo of a second species (Armstrong and McClay, in preparation). The skeleton produced was of the donor phenotype, again demonstrating cell autonomy of pattern (Fig. 4). Examination of single, or small numbers of cells, transplanted into a second species revealed that cell autonomy was present. In some cases, single donor primary mesenchyme cell made a portion of the skeleton appropriate to the region of the host but of the donor pattern. Thus, while cell autonomy exists in the expression of skeletal morphology, the inserted cell is provided with information regarding its position within the embryo so that the appropriate piece of skeleton is made.

What kind of information is given to the primary mesenchyme cells that allows them to make a skeleton in the correct pattern? The experiments below show that primary mesenchyme cells make the piece of skeleton appropriate to their position in the embryo, at the appropriate time, of the appropriate size, and in coordination with other primary mesenchyme cells (the skeleton is a single crystal of CaCO₃). Earlier studies suggested that mesenchyme cells do receive external information: in Okazaki's experiments (Okazaki, 1975), for example, although skeletons were made in culture, they did not fully resemble the final patterns that are seen in vivo, but those experiments did not reveal what types of cues were being received by the primary mesenchyme cells. The following series of experiments show the diversity of cues affecting skeletal pattern.

**Timing of skeletogenesis**

Micromeres, if grown in vitro, divide, form small clusters, migrate, and finally associate with one another and begin to form spicules, a behavior that parallels the behavior of these cells in vivo, but the timing is somewhat different relative to the behavior in vivo. (Fink and McClay, 1985). If primary mesenchyme cells are transferred from one embryo to another, one can ask whether the timing of skeletogenesis is intrinsic to the primary mesenchyme cells, or whether there are interactions with the surrounding embryo that somehow coordinate the timing of skeletogenesis. If older donor primary mesenchyme cells were transplanted into the blastocoel of younger host embryos, the skeleton was produced on the host's timetable. When the older cells were transplanted into blastulae prior to primary mesenchyme cell ingress, the donor cells did not begin to
produce a skeleton until the host primary mesenchyme cells had ingressed and started skeletogenesis (Ettensohn and McClay, 1986). Thus the timing of skeletogenesis is controlled, at least in this experiment, by the host environment. (Ettensohn and McClay, 1986). In experiments where cells of one species are transplanted into the blastocoel of a second species, this observation is reinforced. The two species used, *Lytechinus* and *Tripneustes*, make spicules at different rates, yet, when together in the same embryo, the primary mesenchyme cells are able to coordinate (on the timetable of the host) to make a skeleton that appears normal, albeit with a modified pattern that fits the pattern input of each species (Armstrong and McClay, in preparation). In each of these transplantation experiments, the skeleton grows at a rate appropriate to the host embryo. The timetable used by micromeres grown in vitro may therefore be some sort of default timing pathway. Indeed, in vitro spiculogenesis follows in same behavior sequence but with a timing delay relative to the in vivo pattern (Fink and McClay, 1985).

**The size and scale of the skeleton**

Experiments by Horstadius (Horstadius and Wolsky, 1936), and by others since, have demonstrated that each of the cells from the 4-cell stage, when isolated, will make a whole embryo, though the embryo is one quarter the size of the normal embryo. Similarly, blastomeres separated at the 2-cell stage give rise to perfect half-sized embryos (Fig. 5). In each case, the organs of the embryo are scaled appropriately. The scale regulation could be an intrinsic property of the cells of each tissue, or there could be a regulation imposed by one or more tissues to coordinate the size of the others. A hint that the skeleton’s size is regulated by other tissues was seen in experiments by Ettensohn (1990b). When extra primary mesenchyme cells were transplanted into the blastocoel of host embryos, the skeleton that resulted was no bigger than that seen in control embryos. Even when more than twice the normal number of primary mesenchyme cells were added to the blastocoel, the skeleton that grew was of a normal size and mass. Examination of the cells and skeletons indicated that the donor cells joined into the syncitium and appeared to participate in skeletogenesis but the increase in number of cells had no effect on size.

Components of skeletal size regulation can be seen in experiments contrasting growth in vitro with the size of skeletons in vivo, and in transplantation experiments where primary mesenchyme cells are inserted into embryos of different size (Fig. 5). If primary mesenchyme cells are cultured they have the ability to produce skeletons that are more than five times as long as those produced in vivo (Armstrong and McClay, in preparation). Thus, in vitro there appears to be a lack of constraint on the size of the spicules that can grow. In half- and quarter-sized embryos there normally are 32 or 16 primary mesenchyme cells respectively. These produce half- or quarter-sized skeletons. If one experimentally supplements the number of primary mesenchyme cells in the half- or quarter-sized embryos, the half- and quarter-sized embryos with extra primary mes-
enchyme cells still produce half- or quarter-sized skeletons (Fig. 5) (Armstrong and McClay, in preparation). Scale, therefore, is somehow imposed on the skeleton-producing machinery. The timing of the regulation must occur at the time of skeletogenesis because cells transplanted from normal-sized embryos (and therefore in a full-sized embryo from fertilization until the time of transplantation), produce skeletons appropriate to the size of the host. Thus somehow information on scale is delivered to primary mesenchyme cells as they produce a skeleton of size appropriate to the host. Experiments to be described below will show how the primary mesenchyme cells receive spatial information telling them where to make particular skeletal structures. It is possible, indeed likely, that 'scale' may simply be a function of the dorsal-ventral and animal-vegetal coordinate information received by primary mesenchyme cells. That is, somehow the cells receive the correct positional information and it is the positional information that is scaled appropriately. What is unusual is that not only is the pattern of the skeleton positionally correct but also the mass of the skeleton is appropriate to the size of the embryo. Thus primary mesenchyme cells receive positional information that is scaled correctly, and they also receive information that somehow governs the amount of spicule that can be produced at any given spot.

**Spatial information for pattern formation**

Skeleton formation begins with two triradiate spicules that grow bilaterally from ventrolateral clusters of primary mesenchyme cells. The skeletal rods elongate from each triradiate spicule and primary mesenchyme cells join the syncitium along the way to make two half-skeletons that are the mirror image of one another (Fig. 6b).

The next series of experiments show the kinds of information received by the primary mesenchyme cells. The ability to produce a skeleton may be cell autonomous, but the spatial organization of a correct pattern requires several sorts of cues from the surrounding embryo.

If primary mesenchyme cells are inserted into the blastocoel, the cells move toward the vegetal plate (Ettensohn and McClay, 1986). Only primary mesenchyme cells respond in this way. When other cell types are injected into the blastocoel, they either do not move or they wander about without direction (Ettensohn and McClay, 1986). Therefore, the first cue that ingressed primary mesenchyme cells apparently recognize is a unique site near the vegetal plate. Although each of the primary mesenchyme cells is equivalent in its pattern-forming potential (Ettensohn, 1990b), the two triradiate spicule primordia arise in the embryo from a ventrolateral cluster of cells. The normal initiation of only two spicule primordia appears to be a restriction of the full potential of the primary mesenchyme cells because when these cells are cultured in vitro they will initiate a number of spicules, often with a spicule arising from a cluster of only two cells (Armstrong and McClay, in preparation). If embryos are grown in NiCl₂ from midblastula until mesenchyme blastula stage, many more than two spicule primordia arise (Figs. 6, 7) (Hardin et al., 1992; Armstrong et al., in preparation). Nickel has been found to disrupt the dorsal-ventral axis if administered during the late blastula stage (Hardin et al., in preparation). As a result, ventral marker proteins are expressed all around the embryo and dorsal markers are severely underexpressed. In nickel-treated embryos, there are an average of six spicule primordia. Do the additional primordia arise from a loss of embryonic control over the primary mesenchyme cells (so they could make more spicules as was seen in vitro)? Or, did the additional spicules arise because ventrolateral-like information was now present all around the floor of the blastocoel in the radialized embryos? To distinguish between these possibilities, extra primary mesenchyme cells were inserted into the blastocoel (Fig. 8). If there were a
Fig. 6. The skeletal pattern requires cues from the ectoderm along the animal-vegetal axis and along the dorsal-ventral axis. The normal skeleton is shown in b, with the oral hood (O) out of focus and the anus in the center of the aboral region (A). If the embryo is cut at the equator (see Fig. 9), the two halves round up. If primary mesenchyme cells are inserted into the animal half embryos, a skeleton of the morphology of the oral hood results (a) (note the mouth in the center of this half embryo). If one cultures the vegetal half embryo, a skeleton appropriate to the vegetal portion of the embryo results (c). In experiments examining the dorso-ventral axis contribution, two spicule primordia arise from ventro-lateral clusters of primary mesenchyme cells (d). (This view looks up from the vegetal plate). If one alters the dorsal-ventral axis and ventralizes the embryos, more spicule primordia arise (e).
Fig. 7. Experiments with nickel-treated embryos. Treatment with nickel causes the embryo to synthesize supernumerary spicules. Other experiments suggest the extra spicules result from an alteration of the dorso-ventral axis. Addition of extra primary mesenchyme cells to the nickel-treated embryos has no augmenting effect on the nickel-induced pattern, just as additional primary mesenchyme cells has no effect on the two primordia produced in normal embryos.

Fig. 8. The number of spicule primordia is, at least in part, a function of the ectoderm. If ectoderm is treated with nickel and control primary mesenchyme cells added, extra primordia are observed. If one adds nickel-treated primary mesenchyme cells to untreated ectoderm, the normal number of primordia results.
vegetal halves synthesized aboral elements and lacked oral structures. These results show that primary mesenchyme cells synthesize the portion of the skeleton appropriate to the ectodermal region surrounding these spicule-producing cells. Apparently, therefore, the ectoderm contains all, or much of the axial information necessary for spicule pattern formation and transfers the correct information to the primary mesenchyme cells. The degree to which primary mesenchyme cells might also influence the pattern of their immediate neighbors is not known.

The interactions described above represent only a few of the signals that an embryo must use during embryogenesis. Elsewhere in this volume (Ettensohn, 1992), primary mesenchyme cell interactions with secondary mesenchyme cells are shown to regulate cell lineage decisions. Here it is shown that the primary mesenchyme cells receive spatial, temporal and scalar information necessary for pattern formation. Secondary mesenchyme cells interact with a specific region to regulate the correct positioning of the archenteron. In each of these cases, information is transferred from one cell to another. This information can be fairly complex because, for example, individual primary mesenchyme cells will produce a skeletal element that is spatially correct. These cells must receive the correct three-dimensional coordinates for that to happen. In other cases, primary mesenchyme cells actively prevent secondary mesenchyme cells from expressing primary mesenchyme cell fates, an interaction that is sensitive to the number of primary mesenchyme cells (Ettensohn and McClay, 1988). For each of these cases, there appears to be a default mode. For example, if one removes the animal pole recognition region altogether, the secondary mesenchyme cells eventually stop displaying filopodial behavior and make many of their normal derivatives. Primary mesenchyme cells, in the absence of ectodermal cues, make skeletons, though the spicules that form are made on a different schedule, without the correct pattern, and without the correct scale. Our impression of embryogenesis, given these results, is that cells require large numbers of interactions for the correct pattern to form. There are many cell autonomous morphogenetic properties in the embryos but those autonomous activities appear to receive frequent inputs of information from other cells for correct patterns to emerge.

In each case that we have examined, there appears to be a complex mixture of cues. Endodermal cells appear to be programmed to rearrange. In order to coordinate that movement they require directional information. Secondary mesenchyme cells display stereotypical behavior by extending filopodia on a certain schedule. This movement is terminated following an interaction with a specific region of the ectoderm. Primary mesenchyme cells appear to be programmed (are cell autonomous) in their ability to make spicules. In order to make the correct pattern they require input of several kinds of information from the surrounding ectoderm. The signals are highly varied. Each activity must have a beginning and an ending and all the activities are coordinated with developmental events elsewhere in the embryo. The mixture of cues described here appears to be part of a large network that coordinates morphogenesis.

Given these data, it is important that we now learn how the cells of the embryo store structural information and how the cues are transferred between cells. In other systems analyses of this sort are already underway, though mostly at the genetic level. In the formation of the vulva of C. elegans, for example, genetic analyses suggest that a whole sequence of cell interactions regulates a cellular hierarchy, and that, in turn, regulates vulval pattern formation (Sternberg and Horvitz, 1989; Horvitz and Sternberg, 1991). A number of mutants disrupt the normal pattern-forming capacity during neurogenesis in Drosophila (Camposortega and Jan, 1991). Many of these genes appear to be involved in adhesive functions, or in information transfer between cells, or as DNA-binding proteins. During amphibian gastrulation and neurulation, there appear to be a number of genes that provide spatial cues for the correct pattern to form (Cooke, 1991). In these, and in other examples, the emerging picture is one of a hierarchy of regulatory cues governing pattern formation. Many of the cues are sent and
received via cell interactions. Presumably, those cues are transduced intracellularly and the cell responds by making a portion of a structure appropriate to the mixture of cues that it receives. At the same time the cell provides information to other cells. Thus pattern formation is a highly interactive property of embryonic cells. Both the embryos that were traditionally thought of as ‘mosaic’ as well as the more regulative embryos appear to utilize these interactions. In the end we will begin to understand how the organism encodes the information necessary for assembling a three-dimensional structure. Clearly genes for the “bricks and mortar” of that structure are going to be few in number relative to the many interactions required to fit the structure into the correct context.

References


