Biological roles for the Ro and La ribonucleoproteins have yet to be established. Their abundance (10^5 to 10^6 particles with each RNA per cell) and conservation in the mammalian species we have examined (mouse, human, and monkey) suggest an important function. With respect to RNA components of the La family, it has been suggested that the adenoviral-specified VA RNA participates in the splicing of late viral messengers (*18*), whereas other functions have been proposed for the 4.5*S* RNA synthesized by uninfected mouse cells (*13*, *19*). However, our finding that antibody to La recognizes ribonucleoprotein complexes containing VA RNA as well as many cellular RNA's hints that VA RNA may serve adenovirus in a manner similar to that of the cellular RNA's. It is also conceivable that this cross-reactivity is important in the pathogenesis of certain types of lupus erythematosus.

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Nematode Development After Removal of Egg Cytoplasm: Absence of Localized Unbound Determinants

Abstract. *Embryos of* Caenorhabditis elegans *develop into fertile adults after cell fragments, containing presumptive cytoplasm of somatic and germ line precursors, are extruded from uncleaved eggs or early blastomeres through laser-induced holes in the eggshells. This suggests that the determinate development of this worm is not dependent on the prelocalization of determinants in specific regions of the egg cytoplasm.*

The prelocalization of developmental determinants in the cytoplasm or cortex of fertilized eggs or early embryos is often proposed as a mechanism for differential cell determination, especially in organisms that display determinate cleavage patterns (*1*). Nematodes such as *Caenorhabditis elegans*, which is a favorable model for studying animal development (*2*), provide well-known examples of determinate cleavage (*3*). In *C. elegans*, asymmetrical early cleavages, invariant from individual to individual, produce a set of six precursor cells that are the founders of five somatic cell lines and one germ cell line (Fig. 1, A and B) (*4*). The precursor cells are irreversibly determined (*5-7*). Fate maps, describing which cytoplasmic regions of the uncleaved egg develop into the various precursor cells, have been constructed for several nematodes, including *C. elegans* (*6-9*). In each species the egg was found to be partitioned in a simple pattern of six parallel segments, one for each precursor. From these fate maps it has been hypothesized that the region of egg cytoplasm that each precursor receives is critical to its determination (*6*). We have tested this hypothesis in *C. elegans* by removing cell fragments from fertilized uncleaved eggs or from early blastomeres through holes in the eggshell made with a laser microbeam. Our results suggest that factors responsible for determinate development are not prelocalized in specific regions of the cytoplasm.

The embryo of *C. elegans* develops into a larval worm inside an impermeable eggshell. As in other nematodes, the eggshell consists of a rigid outer layer and a distinct lipoprotein inner layer (*10*). The inner layer alone renders the egg impermeable enough for survival of the embryo. To remove cell fragments we place eggs on a microscope slide in an approximately isotonic medium containing a low concentration of the vital dye trypan blue. The eggshell absorbs the blue dye and is thereby sensitized (at 590 nm) to the microbeam emanating from a dye laser containing Rhodamine 6 G; this microbeam is used to puncture the shell. The egg is then compressed briefly by pushing down on the overlying cover slip, and the cytoplasm of the cell adjacent to the hole in the eggshell is partially extruded (Fig. 1, C and D). The hole is sufficiently small to impede extrusion of the nucleus or the spindle, but allows free passage of yolk granules (about 1 μm in diameter). The granules can be seen to stream uniformly from all regions close to the hole. The cell then ligates at the constriction caused by the eggshell. Usually the resultant cell fragment becomes visibly detached from the egg.

Electron micrographs show that the cell fragment is surrounded by a plasma membrane. The extruded cytoplasm looks very similar to that in the corresponding region of an intact egg. It contains the usual organelles, for example, ribosomes, mitochondria, and yolk granules.

The medium used for extruding the fragments does not support the survival of the fragments or the exposed cells. Fragments degenerate soon after extrusion and take up the trypan blue present in the medium. Often an embryo from which a fragment is removed lyses

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immediately after exposure to the laser beam or within a few hours, and takes up trypan blue. Frequently, however, the inner layer of the eggshell reseals, since the embryo does not take up any dye and continues to develop. The hole in the eggshell must be made at a location where the shell is not in contact with the embryo, otherwise the heat generated by the laser beam lyses the embryo. From uncleaved eggs, fragments can only be extruded at the anterior or posterior pole (Fig. 1D). In either case, the eggshell reseals in about 25 percent of the embryos.

In uncleaved eggs the posterior 15 percent of the cytoplasm is normally segregated into the P_2 cell (Fig. 1B) (*11*), which gives rise to two somatic precursor cells (C and D) and the germ line precursor P_4 (Fig. 1A). In contrast, the anterior 60 percent of the egg cytoplasm segregates into only one precursor cell, AB (Fig. 1B).

About 60 percent of the fertilized, uncleaved eggs from which posterior cell fragments were removed at or after fusion of the pronuclei showed no developmental defects and gave rise to normal larvae (Table 1 and Fig. 1, C and D). All those transferred to growth plates formed fertile adults that laid a normal number of eggs (more than 250). We estimate that between 15 and 20 percent of the most posterior cytoplasm was removed from three of the embryos that hatched. The two tested became fertile adults, showing that most or all of the presumptive C, D, and P_4 cytoplasm and a large area of egg membrane can be removed without affecting normal development or fertility.

Although posterior cytoplasm was removed in these experiments, usually the anterior AB cell as well as the posterior P_1 daughter of the first cleavage were smaller than normal. The cells preserved their relative volumes (Fig. 1, C and D), thus displaying an apparent size regulation. In some embryos an incipient first cleavage furrow, already present at the time of extrusion, receded and re-formed more anteriorly. This re-formation was accompanied by an anterior movement of the spindle. If the location of the cytoplasm before cleavage were important, any readjustment of the first cleavage plane should have further altered the distribution of the cytoplasm and the resulting fates of all six precursor cells. Such size regulation is, however, not necessary for normal development, since some embryos in which P_1 remained much too small relative to AB developed into normal larvae.

In the embryos that did not hatch, the pattern of early cleavage was normal at least until the 50-cell stage. These embryos cleaved into several hundred cells, but late morphogenesis was absent or grossly abnormal. The abnormal development could have been due to the compression of the egg or swelling of the embryo. We do not think it was caused by the loss of specifically localized posterior cytoplasmic determinants because similar abnormalities were seen in embryos from which anterior cytoplasm had been removed, and because the frequency of arrested embryos was not correlated with the amount of cytoplasm removed.

Table 1. Proportions of hatched and fertile animals. Data for all embryos not lysing before the 100-cell stage are included. The diameter of the extruded fragment was measured from photomicrographs and the percentage of cytoplasm extruded was estimated to the nearest 5 percent from the known volumes of the precursor cells (*8*).

Region of blastomere extruded (presumptive cytoplasm)	Number of	
	Embryos hatched	Tested animals fertile
P_0 posterior 5 to 15 percent (some P_2)	9 out of 18	3 out of 3
P_0 posterior 15 to 20 percent (all of P_2)	3 out of 3	2 out of 2
P_0 anterior 5 to 15 percent (some AB)	2 out of 5	2 out of 2
P_1 posterior 5 to 15 percent (some P_2)	2 out of 2	2 out of 2
P_2 ventral 5 to 10 percent (some P_4)	2 out of 4	2 out of 2
P_2 ventral 10 to 30 percent (all of P_4)	4 out of 6	4 out of 4
P_2 ventral 30 to 45 percent (all of P_4 and D)	3 out of 6	2 out of 3
P_2 dorsal 5 to 30 percent (some C)	1 out of 6	1 out of 1
P_3 ventral 5 to 20 percent (some P_4)	1 out of 5	1 out of 1
P_3 ventral 60 percent (all of P_4)	1 out of 1	1 out of 1

It is more difficult to remove fragments from later-stage blastomeres because most of the embryos lyse. Nevertheless, 28 embryos continued development after removal of large fractions of the P_2 or P_3 blastomere. Twelve of these hatched (Table 1); those that did not resembled the arrested embryos from which we had extruded fragments at the P_0 stage.

We can estimate from the directions of the P_2 and P_3 divisions (predominantly dorsal-ventral, Fig. 1B) (*4*) and the known volumes (*8*) of P_4 and D that the most ventral 8 percent of the P_2 cytoplasm is eventually segregated into the germ line precursor P_4 and the adjacent 22 percent into D. When all the presumptive P_4 cytoplasm, or all the P_4 and D cytoplasm was extruded from P_2, embryos developed into normal larvae (Fig. 1D, d and e). All except one proved fertile, giving rise to more than 250 progeny each. The single sterile animal had severe abnormalities in the somatic sexual structures, not derived from P_2 (*12*). It is unlikely that a germ cell defect led to the somatic abnormalities, since even when P_4 is completely ablated the resultant animals, though devoid of germ cells, usually have normal somatic sexual structures (*13*).

One embryo, from which 60 percent of P_3 had been extruded (Fig. 1D), developed normally. The extruded fragment included all of the presumptive P_4 cytoplasm. The P_3 cell divided, but the resultant P_4 cell was very small and did not divide again during embryogenesis as it normally would have done to produce two germ line precursor cells at hatching. Nonetheless, P_4 proliferated after hatching. The animal developed apparently normal gonads and laid close to the normal number of eggs (209).

These experiments show that cytoplasmic determinants, including the germ lineage and the somatic C and D lineages, are not localized in the uncleaved egg in an unbound form. The anterior AB extrusions and the readjustment of the first cleavage plane after P_0 extrusions suggest that it holds for the AB, MSt, and E lineages. Putative determinants could still be prelocalized by association with a fixed lattice which must be different from that with which the extruded cell organelles are associated. Such a lattice might include the cytoskeleton and anchored membrane proteins (*14*). Exclusive membrane prepatterning, however, seems to be absent, because large amounts of egg membrane were extruded concomitantly with cytoplasm.

The apparent absence of unbound, prelocalized germ-cell determinants in the uncleaved egg is surprising, because such determinants have been found in other animals (*15*). In *C. elegans*, specific structures have been found apposed to the nuclei of germ line cells and their precursors (*16*). These structures may be equivalent to the "nuages" in the germ cells of mammals and other animals (*15*). They first appear in P_2 and segregate exactly with the germ lineage; first asymmetrically into P_3 and P_4 and not into

their somatic sisters C and D (Fig. 1), then symmetrically into the two daughters of P_4.

An asymmetric segregation of developmental potential must take place in cell divisions generating daughter cells with different fates. Although rigid cytoplasmic localization seems to be excluded, our experiments do not distinguish between irreversible determination in some other (noncytoplasmic) form and a more flexible localization, cytoplasmic or otherwise, coupled with a capacity to regulate. For example, the source of asymmetry could simply be a polarity within the dividing cell (*5*, *6*). Both our results and the abnormal cleavage patterns that follow alteration of the spindle orientation in centrifuged and dispermic *Ascaris* eggs (*5*) are compatible with this notion. The polarity could be a chemical gradient (*17*) affecting the cytoskeleton including the spindle. There is a visible asymmetry of the spindle in the asymmetric divisions of P_0, P_1, P_2, and P_3. The centriolar region at the pole of the incipient somatic cell daughter is spherical, whereas that at the pole of each incipient P daughter is disk-shaped (*8*). The spindle could mediate specific segregation of developmental potential by segregating cytoplasmic determinants attached to the spindle (*18*) or variegated chromosomes [for a discussion of this, see (*19*)] or other nuclear structures, for example, the perinuclear "nuages" in the germ lineage of *C. elegans*.

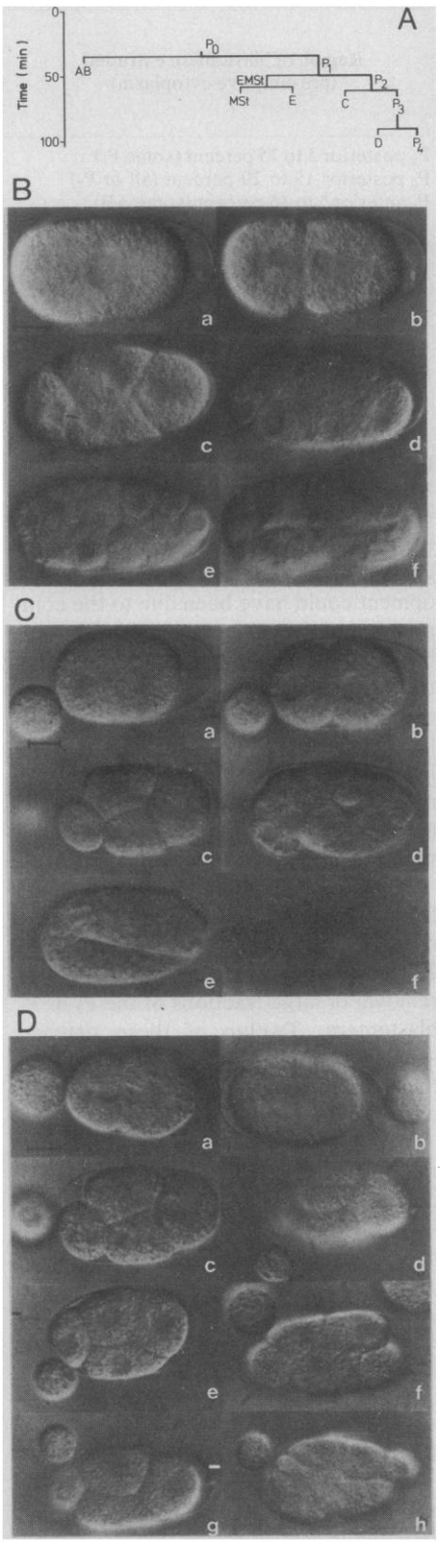

Fig. 1. (A) Generation of the embryonic precursor cells of *Caenorhabditis elegans* (*3*, *4*, *8*). Fertilization occurs at 0 minute. The zygote P_0 divides about 35 minutes later at 21° ± 1°C. Precursor cells arise by invariant asymmetrical divisions from the P line in a typical stem cell pattern. In these divisions the smaller (P_1 to P_4) daughter is the precursor of the germ line. The five somatic precursor cells *AB*, *MSt*, *E*, *C*, and *D* go on to divide (divisions not shown) giving rise to all somatic cells of the animal. P_4 divides symmetrically into the two postembryonic germ line precursors. (B) Development of an intact embryo showing the positions of the P cells and their sisters, and the pretzel stage shortly before hatching [for a detailed description of embryogenesis, see (*3*, *4*, *8*)]. Orientation of all embryos: anterior, right; dorsal, top. (a) Uncleaved egg; (b) two-cell stage, P_1 is posterior, AB is anterior; (c) four-cell stage, line connects P_2 (posterior) and EMSt (AB has also divided); (d) eight-cell stage, line between P_3 (posterior-ventral) and C (slightly below the plane of focus); (e) 24-cell stage, line between P_4 (ventral) and D; (f) pretzel stage shortly before hatching. (C) Normal development of an embryo from which a posterior fragment containing about 10 percent of the total egg cytoplasm was extruded at the one-cell stage (P_0) after the fusion of the pronuclei. (a) Egg during first division showing extruded cell fragment; (b) two-cell stage; (c) four-cell stage; (d) 28-cell stage; (e) plum-stage; (f) empty eggshell after hatching of the larva, which subsequently developed into a fertile adult. *Caenorhabditis elegans*, var. Bristol, strain N2, was grown at 20°C as described in (*2*). Egg culture and Nomarski differential interference-contrast microscopy were as described previously (*4*). Eggs were mounted in a medium containing 120 m*M* NaCl, 10 m*M* Hepes buffer at *p*H 7.2, streptomycin sulfate (100 μg/ml), penicillin (100 U/ml), and trypan blue (0.025 mg/ml). To control swelling we added, per milliliter, 0.045 mg each of 18 of the 20 standard amino acids (omitting asparagine and glutamine) [*Ascaris* coelomic fluid (*7*) did not further improve survival rate]. The laser microbeam system (BTG Biotechnik, München), consisted of a nitrogen laser–pumped dye laser (model K 500-FL105, Lambda Physik, Göttingen) adapted to a modified Leitz Orthoplan microscope fitted with Nomarski optics. The laser spot size was less than 1 μm in diameter when focused through a ×100 objective lens (Leitz, NPL ICT; numerical aperture, 1.30). With Rhodamine 7 G, the laser gave pulses of 4 nsec duration with a maximum energy of about 250 microjoules per pulse at 590 nm. With trypan blue adsorbed to the eggshell, a single pulse of 1/1000 the maximum energy was sufficient to puncture it. (D) Representative embryos with cell fragments extruded at various stages. Embryos are shown shortly after extrusion. In (c) through (h), the fragment has drifted away from the site of extrusion. All eight embryos hatched and developed into adults. All were fertile except (e). (a) Posterior 15 percent of the egg cytoplasm removed from the uncleaved egg (shown at two-cell stage); (b) anterior 10 percent removed from the uncleaved egg; (c) posterior 15 percent of P_1 removed at the two-cell stage (shown at four-cell stage); (d) ventral 30 percent of P_2 removed at the four-cell stage; (e) another embryo as in (d) (ventral 30 percent of P_2 removed) became a sterile adult (see text); (f), ventral 45 percent of P_2 at the six-cell stage; (g) dorsal 30 percent of P_2 removed at the four-cell stage; (h) ventral 60 percent of P_3 removed at the 13-cell stage. Scale bars, 10 μm.

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A Photobiological Evaluation of Tanning Booths

Abstract. *The use of tanning booths as a substitute for natural sunlight is becoming increasingly popular. However, unless careful attention is paid to proper design and maintenance, the radiation field inside a tanning booth can be highly anisotropic. The use of simple, inexpensive ultraviolet radiation meters to measure dosage can lead to serious overexposure. Since the ultraviolet radiation inside a tanning booth has a greater proportion of short wavelengths (< 300 nanometers) than natural sunlight, the amount of skin cancer–inducing radiation received for a tan may be twice that received for a natural suntan.*

Discussion of the potential health hazards of tanning booths has been mainly limited to immediate and obvious hazards such as excessive sunburn, photokeratitis of the eye, and accidents involving broken glass or electrical shock (*1*). A few dermatologists have mentioned possible long-term effects, such as wrinkling and skin cancer, but these have not been quantified. The operators of tanning salons generally claim that their tanning booths simulate sunlight and therefore pose no more long-term risks than does sunlight itself. This report examines the tanning booth from a photobiological point of view.

To provide a basis for a quantitative photobiological assessment, we made measurements in a custom-built tanning booth (*2*) in a local salon. The operator had copied the idea, booth dimensions, and equipment requirements from another salon. Although the booth we examined is not a commercial one and may not be representative of all tanning booths, the results for this booth nonetheless demonstrate many of the aspects that must be considered when evaluating the efficacy and safety of tanning booths in general.

Figure 1 gives a schematic view of the booth we examined. Two 72-inch fluorescent sunlamps in a standard "slimline" fluorescent fixture (without reflector) are mounted vertically in each of the four corners of the booth, the door and walls of which are covered with metallized wallpaper. The ultraviolet (UV) irradiances from the lamps, measured with a commercially available, commonly used UV radiation meter (*3*), are indicated.

The extreme anisotropy of the radiation field is apparently due to lack of sufficient UV reflectivity of the inner wall covering (although it visibly appeared to be a fairly good reflector) and lack of uniformity of the individual lamps (although they did not visibly appear very different). We found that the lamps ranged in age from those that were new (position 1) to those used for 2 hours (positions 5 and 7) or 24 hours (position 3). Subsequent laboratory tests on new sunlamps indicate that the UV irradiance is about 30 percent higher during the first half-hour of use than after 10 hours. After 10 hours the output changes very slowly (a loss of 0.14 percent per hour). In addition, the lamps in position 5 were found to be malfunctioning.

It is clear that unless the user stations himself in the exact center of the booth and rotates uniformly, a highly uneven tan will result—with the possibility of serious sunburn to those areas of skin closest to the lamps. (Note also that it is virtually impossible for a normal adult in this booth to remain at least 1 foot away from each lamp as recommended by the lamp manufacturer.) These problems could be ameliorated by better booth design and by burning the lamps for 10 hours before exposing the patrons to them. However, unless UV irradiance measurements are made, there is no way to ensure that the radiation field is uniform. Most tanning salons are probably not equipped to make such measurements.

We now turn to a quantitative consideration of whether a well-designed tanning booth with a uniform radiation field presents a greater long-term health risk than the sun. To evaluate this we made spectroradiometric measurements (*4*) of pairs of sunlamps under laboratory conditions. We did not attempt to duplicate tanning booth geometry because we were primarily interested in the basic spectral outputs of the lamps, uncomplicated by reflectance and multisource variables. Figure 2A shows the spectral output of two 72-inch, 55-W lamps at 33 cm, the minimum lamp-to-subject distance recommended by the manufacturer. For comparison, Fig. 2A also shows the calculated noontime spectral irradiance (*5*) from the sun at 30°N. At the shorter wavelengths, the lamps' output is significantly higher than the sun's, whereas at wavelengths greater than about 305 nm, the sun's output is higher. Thus the sunlamps do not closely simulate the sun in the UV region.

Figure 2A also shows the spectral irra-

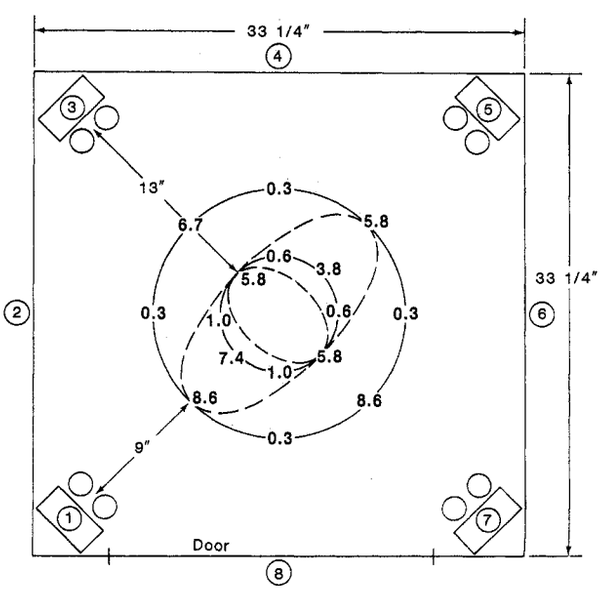

Fig. 1. Schematic view of a tanning booth. Two fluorescent sunlamps are mounted vertically at positions 1, 3, 5, and 7. The dashed ellipses represent rough cross-sectional (transverse) views of head and trunk for a typical person. Numbers indicate readings obtained with a commonly used UV radiation meter (*3*) whose sensor was pointed radially in a horizontal plane 50 inches from the floor at the locations shown. The outer circle corresponds to the position of the arms; the inner circle corresponds to the position of the head or chest.

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