# On the Origin of Space

## Part 3C: Quantum Spatial Development - The Drosophila Oocyte

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**Abstract**

*Quantum dynamical space reveals itself in various forms within the initial phases of living organisms development. The chemical approach followed by the literature to identify the basic principles of morphogenesis appears then to be fundamentally incomplete by missing the holistic aspect of such developments, where the various cells interact on a spatial level with the principles of the interactions having nothing to do with chemistry. The oocyte generation of drosophila melanogaster gives key examples of such holistic physical interactions.*

**Keywords:** drosophila, oocyte, fusome, follicle cells, stem cells, bicoid, leptonic space manifold

### Introduction

Can we apply the understanding of quantum dynamical spaces, i.e. space being built by its contents, as developed in [**1**] through [**4**], to the set of evolving biological cells called eggs, oocytes, zygotes and embryos, i.e. to the beginnings of living organisms? If we can do that in a meaningful way, we would obtain a strong confirmation about the existence of physical phenomena in Life not found anywhere else, as well as additional insight on how such processes run and are applied by Life. To this effect, we take a number of pictures of developing systems drawn in the literature, and add a description of what is spatially going on in them. We look primarily for logical physical explanations that complement existing chemical descriptions, especially in areas where the research is presently stumped on the nature of the happenings. This article covers the oocyte development of *drosophila melanogaster* as this system is a microcosm of key physical happenings that need description and evaluation in order to understand a number of physical facets fundamental for the existence of Life. The early embryo and other developmental systems will be evaluated separately.

## a. Polarization of the cystoblast – nuclear power

Drosophila egg chambers develop in assembly lines within ovarioles. (Fig. 1a)

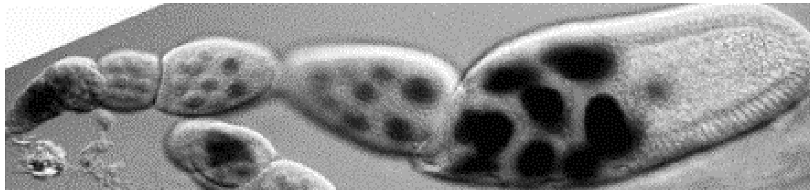


Fig. 1a ([5])

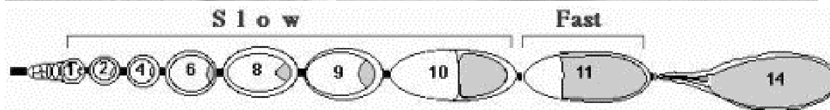



Fig. 1b ([5])

Intercellular cytoplasm transport during oogenesis is required to produce mature eggs that contain maternal components sufficient to support and guide early later embryogenesis. This transport occurs in two phases (Fig. 1b): slow (several days – stages 1 to 10) during which the oocyte grows at about the same rate as the nurse cells, and fast (~30 min. – stages 11 through 14) during which all remaining cytoplasm is squeezed into the oocyte by nurse cell contraction through cytoplasmic bridges called "ring canals." These canals are actin-rich, about 10 microns in diameter, structures established ***as a result of the nuclear system not fully separating*** **between daughter cells,** and thus not allowing the physical completion of MT cytokinetic cell division. Vitellogenesis begins in stage 7 when the oocyte begins to take up large quantities of yolk.

In the *germarium*, (Fig. 1c), a stem cell first produces a "cystoblast" and a

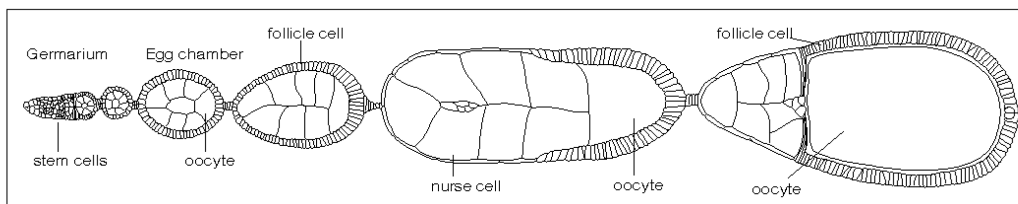



Fig. 1c ([6])

new stem cell (for the next cystoblast production) via an *asymmetrical* cell division. The cystoblast becomes then 16 cells by four *incomplete* cell divisions going through mitosis or meiosis arrested in the prophase I stage, building a "synaptonemal complex." The 15 nurse cells and the future oocyte are connected by ring canals. (Fig. 2) A sheath of somatic "follicle" cells develops from a different stem cell and surrounds the cystoblast.

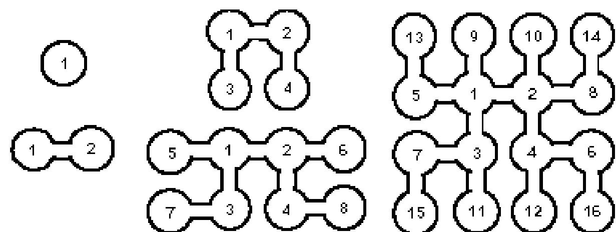



Fig. 2 ([7], Fig. 1)
**Cystoblast cell divisions**

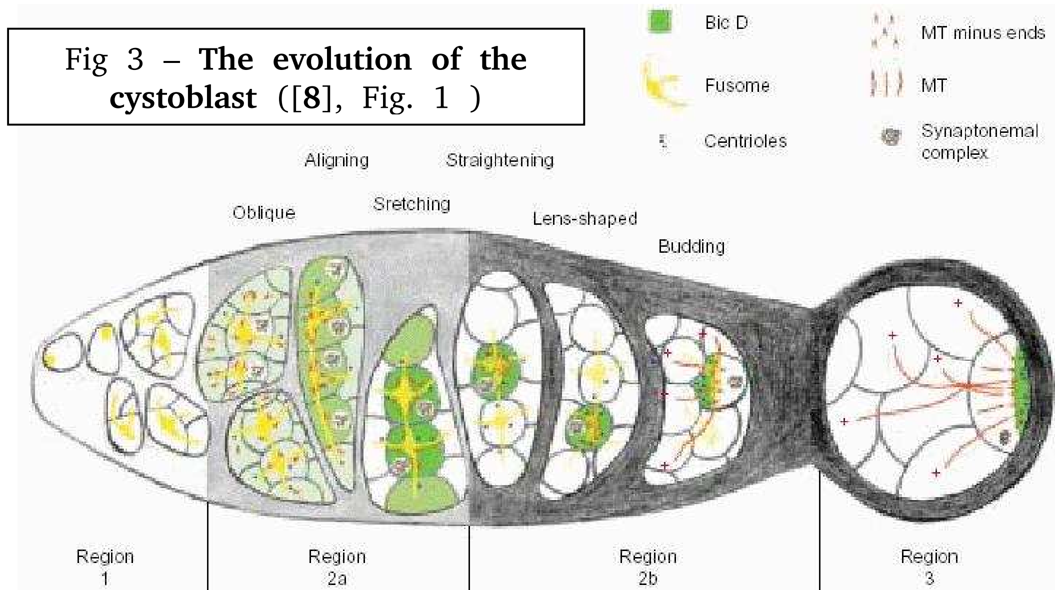



Fig 3 – **The evolution of the cystoblast** ([**8**], Fig. 1 )

The final oocyte is identified as the posterior cell via a chemical and spatial differentiation process bought out first by the inter-cell nuclear system, then by the MT system, and finally by the follicle cells. [**7**, **9**]

## The initial stem cell differentiation – a pure genetic play?

The earliest-acting gene in the cystoblast formation, *bag-of-marbles* (*bam*), is necessary for the asymmetry in the cell division to occur. ***bam is both necessary and sufficient for starting the cystoblast in the germarium stem cells***. Recent data shows that a transcriptional silencer specifically keeps *bam* off in the original stem cell. [**10**]

*Bam* is an essential component of the fusome as described later, where it is required for proper function. The *bgcn* gene is also required specifically for *bam* protein ***localization and function***. *Bam* and *bgcn* proteins interact to promote the initial cystoblast cell differentiation from a stem cell (and thus may tell us *how specialized derivatives become different from their stem cells*). On the face of it, there may be many kinds of stem cells since that particular kind leads to cystoblasts. ***Another stem cell nearby leads to the follicle cells*** for example. So this question has still no answer, and maybe a ***physical factor*** is involved after all.

## The fusome [**10**, **11**, **12**]

We have a programmed series of 4 cell semi-divisions (and 4 exactly) where the nuclei retain a ***specialized endoplasmic reticulum*** (ER) between each other during the divisions. Fig. 3 identifies this connection between nuclei as a "***fusome***." This was the name given (decades ago) to the common ER before it was found that it was an ER. By its mere presence this fusome forces all division MT cleavage furrows to remain open (Figs. 3). It is ***polarized*** from the first cystoblast division, as one daughter cell inherits more fusome material than the

other. This unequal distribution is maintained until the 16-cell cyst has formed, and thus the asymmetry defining the oocyte originates from the nuclear system, not from the MT system. [**9**]

The fusome grows from a prominent spheroidal organelle (called a *spectrosome*) to an elongated and branched structure that connects all mitotic sisters. The organelle is assembled from proteins normally found in the ***nuclear membrane skeleton***. It grows along the remnants of the mitotic spindles after each round of division, with the ***spindles orientating with one pole adjacent to it***. (Fig. 4)

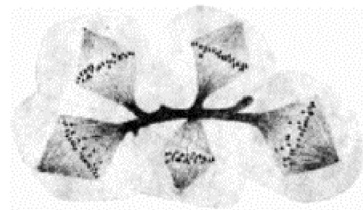


Fig. 4 – **The fusome** ([**11**], Fig. 1a)

The initial spectrosome elongates and migrates toward the daughter cystocyte, and becomes unequally partitioned, 2/3 remaining in the original cell, apparently ***due to manifold geometric constraints*** (being at the opposite side of the splitting cell next to the spindle pole there), thereby defining the cell that will become the oocyte. ***There is then no randomness in this process***.

From this structural outcome, we conclude that (1) the fusome generates its own ***leptonic space manifold*** with the dimensions of the original stem cell centriole manifold, and (2) the generation of more fusomal material by the chromatin at the spindle poles allows this manifold to expand. In effect ***the final oocyte manifold may very well have the dimensions of the original stem cell manifold through the evolution of the fusome***. This would be then also why the physical (spatial) control of the development can be smoothly switched to the oocyte centriole when the fusome has done its job coordinating the building of the 16 cells and directing cell differentiation.

Besides differentiating the oocyte, the fusome is also required to maintain ***mitotic synchrony*** between cystocytes. This is accomplished through ***directed transport of vesicles***, most probably using its leptonic space manifold as spatial guidance (as for MT transport when centriole manifolds are around), and with chemicals: The cell cycle regulator *Cyclin A* transiently associates with the fusome, and since its *overexpression* results in an extra round of cell division giving 32 cells, its presence most likely drives the interconnected cells synchronously into cell division by keeping track of the number of performed cycles. [**13**] This is thus a case of chemical regulation supporting a physical system.

The fusome also helps moving mRNA when MTs can't. Finally, it may associate only with **mitochondria with functional genomes** for their motion into the oocyte. Damaged mitochondrial genomes might be then weeded out when they still represent a small fraction of the total. Such a system would be far more efficient than eliminating defective genomes by inducing the apoptosis of entire

germ cells. Such a purifying mechanism might be particularly important in organisms that need to produce eggs with a high average viability, or that must support long intergenerational life spans. However, for ***such an association to exist, nuclear membranes such as the one in the fusome must sense when DNA is "functional."*** This would require a study of the nuclear membrane and DNA to see how leptonic space manifolds could be generated by them, so their functionality can be ***physically*** (instead of chemically) sensed, and thus would be a much more precise process.

The MT system follows the polarization of the fusome, with the centrioles having only a minor role in maintaining the order of the MTs. In fact, the centrosomes/centrioles are guided by the fusome toward the cell chosen to be the oocyte, very much as centrosomes in normal cell interphase remain close to their nucleus as they have a common space manifold (*a spatial arrangement still with no explanation in the literature*).

***The elongated cystoblast shape in region 2 (Fig. 3) is also indicative of the physical presence of a leptonic space manifold sustained by the fusome, with an intersection common to all the cells***.

## Cystoblast cell differentiation – genetic or physical determination? [14]

Although all 16 cells undergo pre-cell division replication of DNA, only the oocyte remains in the meiotic cycle. 12 cells enter an "*endocycle*" (i.e. alternating mitotic S and G phases without intervening splitting of the nucleus), and thus develop as ***polyploid*** nurse cells. The four cells with 3 or 4 ring canals (1 through 4 in Fig. 2) progress to the meiotic prophase I *zygotene* stage, displaying a ***synaptonemal complex*** (SC) as well as *bicoid* mRNA while in the middle of region 2a. (Fig. 3) However, by the end of that region, the SC disappears from the cells with 3 ring canals, thus becoming nurse cells too, and the two cells with 4 ring canals have their SC reaching its maximum length, i.e. the meiotic prophase I *pachytene* stage. The designated oocyte remains in meiosis with the SC compacting to form a *karyosome*, which disappears by the cell going to meiosis metaphase I (forming a spindle) soon after the cyst leaves the germarium for fertilization. The "losing pro-oocyte" eliminates its SC and reverts to the endocycle of a nurse cell.

What wins the competition between these two pro-oocytes? Is there a competition in the first place? No-one really knows. The literature such as [**14**] advances that the selection may occur by a mechanism similar to other asymmetries found in development based on the ***concentration of gene products***. *bicoid* does indeed become limited to the pro-oocytes, but this concentration looks more like a consequence of the fusome selectively moving mRNA due to its polarization dating from stage 1. As we have seen for mitochondria, the fusome may have the opportunity here to weed out "bad" DNA cells. If the two candidates

are ok, the selection is then from the fact one of the two pro-oocytes has a leptonic space manifold with the same dimensions as the fusome, so it receives more *bicoid* than the other, and *bicoid* is known to promote meiosis. The fact the centriole of that cell will be the one directing the show from there on, and no other, and this from this very choice of oocyte, favors a physical choice here, not a fuzzy chemical one as suggested by the literature, that would potentially lead to errors. We will note that the analysis by [**14**] dismisses the fusome role.

### b. Transport toward the oocyte – centriolar power [15, 16]

By the end of region 2b the fusome has a *crescent* shape and starts disappearing, through the influence of the remaining centriole taking over its manifold, and polarizing the system through its own organization of MTs. In region 3 the centriole and its large MTOC move the oocyte completely to the forefront of the cystoblast by inflating its space manifold via the generation of MTs, as they reach within all the cells of the cystoblast, taking over the fusome manifold through the ring canals, thereby maintaining them open using actin remaining at the intersections with the nuclear manifolds. Since all the other centrioles are now out of the nurse cells, the cystoblast ends up with a single manifold across the 16 cells, except for nuclear manifolds. (Fig. 5)

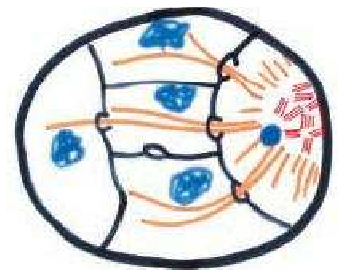


Fig. 5 ([**15**], Fig. 2)

The nuclear ER and the MT systems are thus the physical way to move and accumulate *bicoid* and other mRNA produced in the nurse cells into the oocyte. ***The only function of the genetic system is then to produce material****,* such as *bicoid*, ***as tools for use by physical systems***. Indeed*,* (1) the fusome triggers its own demise via selecting the oocyte through transport of *bicoid*, thereby effecting the transfer of physical spatial power to the centriolar system, (2) the last centriole is in turn eliminated upon accumulation of *bicoid* (and other mRNA) through MT transport to this remaining active centrosome, thereby shutting it down so follicle cells can take over the physical arrangement as the third director of the process, (as we shall see later).

Large molecules such as mRNA cannot move on their own due to their size within the cytoplasm, and thus no stochastic diffusion is "naturally" possible (as Turing and others envisioned decades ago, thinking of Life as a mere classical happening, a view still supported against overwhelming evidence by present biology textbooks). What really happens is that small MT pieces constantly move around through their internal quantum dynamics (not via Brownian motion!) per Fig. 6, allowing the large molecules to be entrained by them and thus ***purposely* quasi-randomized**. Fig. 7 shows five types of directed motions:

(1) Quasi-random movements of MTs to keep mRNA moving (blue arrows),

(2) Particle-gathering material moving into the nuclear periphery to pickup *bicoid* and other mRNA coming out of the nucleus (orange arrows),
(3) Moves toward the rig canal microtubules (green arrows),
(4) Moves through the ring canals (black arrows),
(5) Accumulation at the anterior membrane of the oocyte (red arrows).

Fig. 6 ([**16**], Fig. 9) **Different color MTs = different times**

We shall note here that the conclusion of [**16**], about item 4 in the list above being independent of MTs and actin, was reached through administering a MT depolymerizer, with subsequent repolymerization. *Such a drastic action will put any subsequent proper reconstruction of the cystoblast-wide MT layout in doubt, including the key quantum function of the oocyte centriole originating that MT system in the first place.* Also, this jeopardizes the subsequent switchover to a different spatial manifold mode, as we see below.

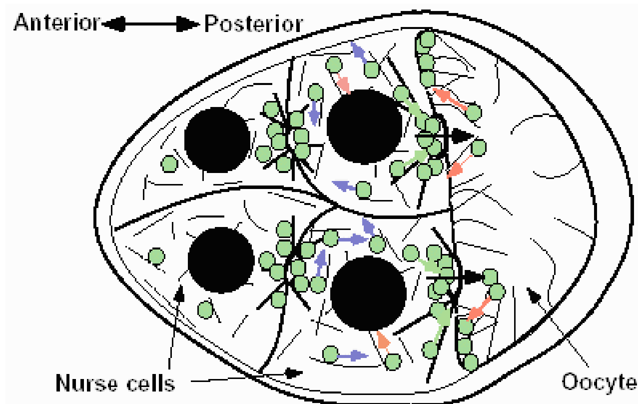



Fig. 7 ([**16**], Fig. 10) **The MT system motions**

## c. Polarization of the oocyte – follicle cells power [17]

The oocyte grows and gets filled from the nurse cells. Its internal yolk is brought in, together with the *mitochondria* that followed the fusome earlier, and now the MTs. The ***follicle cells*** are becoming the third director of the cystoblast development process, following the fusome (nuclear system) and then the oocyte centriole (MT system). Anterior/posterior and dorsal/ventral axes of the oocyte for future use in embryogenesis after fertilization are now being specified through the physical interaction between the oocyte and the follicle cells. Follicle cells adopt a posterior fate ***as a result of their centrioles sensing the internal manifold of the cystoblast receding to the oocyte***. They start then secreting the *vitelline* membrane and *chorion* shell over the entire oocyte.

Internally to the oocyte, the gene products *bicoid* and *oskar* define the A/P axis while *gurken* takes care of the D/V axis. But how do they physically move in position? First, let's look at how textbooks describe this: [**6**]

[The posterior signal from the oocyte to the follicle cells is the *gurken* protein, as it was brought in through the earlier cystoblast MT system phase. *gurken* binds to *torpedo*, a receptor tyrosine kinase. (Fig. 8) The D/V axis is also set up by *gurken*, which signals

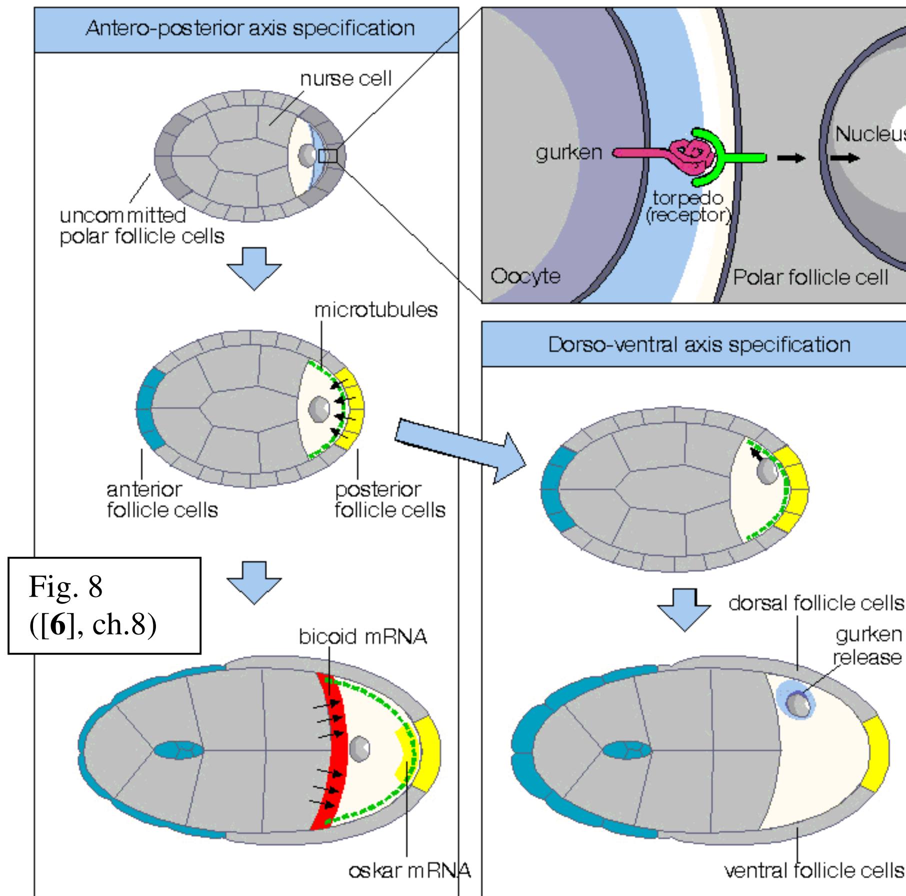



Fig. 8
([**6**], ch.8)

to establish dorsal follicle cells (which do not produce the ventral follicle cell proteins needed for establishing ventral embryo fates). The mRNA for the *gurken* protein is localized between the oocyte nucleus and the dorsal follicle cells. (Fig. 9a)

The *gurken* protein is similarly located, shown in Fig. 9b is at a younger stage. Fig. 9c is a cross section of the egg through the region of *gurken* protein expression.]

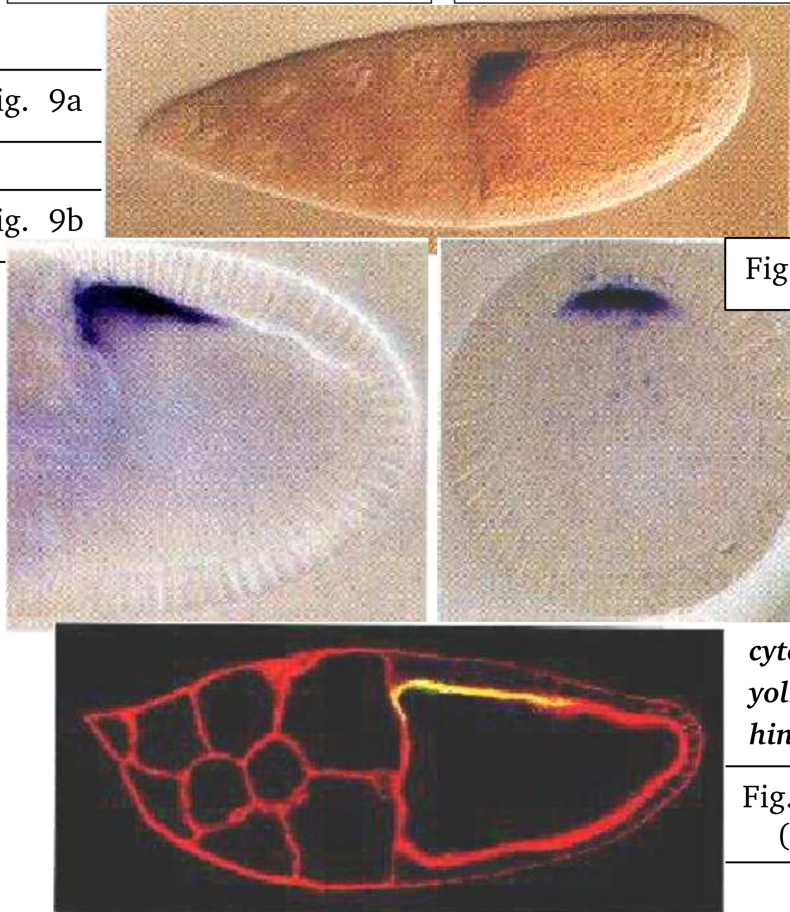


Fig. 9a

Fig. 9b

Fig. 9c

Fig. 10
([**6**])

A **more mature oocyte** in Fig. 10 shows the *gurken* protein (yellow) across the dorsal region. (Actin has been stained red, showing cell boundaries.) It is important to note here ***how narrow the cytoplasm of the oocyte is at that stage vs. the yolk part, which may be a hint about the physics at play***:

As the literature says, [**17**] follicle cells "signal back" to reorganize the oocyte's cytoskeleton, which in turn directs *bicoid* mRNA to the anterior, with the posterior receiving *oskar* mRNA (specifying germ plasm) and *nanos* mRNA. A simple hypothesis [**17**] explaining the above classically is that the "signal" (understood as a chemical agent) from the follicle cells is necessary to polarize the microtubule cytoskeleton of the oocyte, which in turn directs the localization of *bicoid, oskar* etc. mRNAs there:

"While polarization of the oocyte microtubules is thought to be an output of the signal from the follicle cells, ***the nature of this polarity has been questioned***. According to this model, plus ends of microtubules should point toward the posterior of stage 9 oocytes. However, both the heavy chain of cytoplasmic dynein, a minus-end-directed motor, and the *glued* protein, a component of the dynein regulatory complex dynactin, are also posteriorly localized at stage 9. This paradox creates some uncertainty about the polarity of the oocyte microtubule cytoskeleton and suggests that either *Kin:βgal* or dynein is not

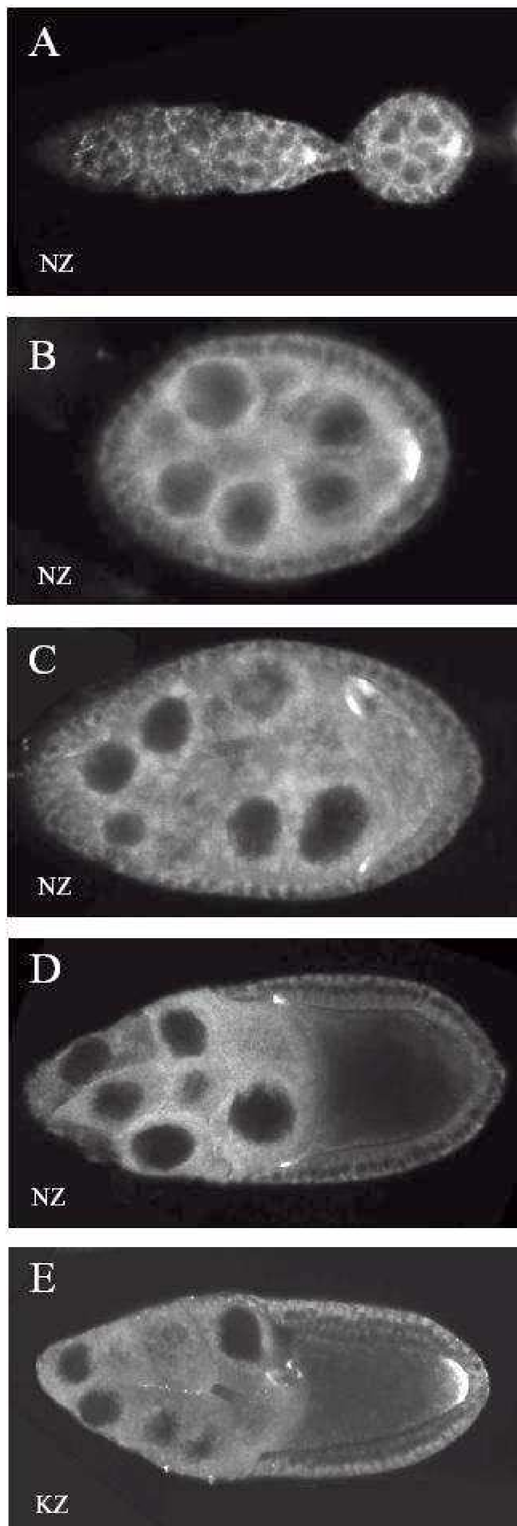



Fig. 11 - ***Nod:βgalactosidase* localization in germline during oogenesis.**
([**17**], Fig. 4 + text)
(A) Germarium and stage 2 egg chamber. *Nod: βgal* localizes to the oocyte.
(B) Stage 6 egg chamber. *Nod: βgal* is concentrated against the posterior cortex of the oocyte.
(C) Stage 8 egg chamber. *Nod: βgal* is seen in cross section at the anterior corners of the oocyte and flanks both sides of the oocyte nucleus.
(D) Late stage 9 egg chamber. *Nod: βgal* is tightly localized to the anterior corners of the oocyte. Some of these proteins are also present in the nurse cells.
(E) For comparison, a late stage 9 egg chamber expressing *Kin:βgal* is shown here. *Kin: βgal* localizes efficiently to the posterior of the oocyte. Note that it is expressed also in *border cells,* a set of follicle cells that migrate to the anterior margin of the oocyte by stage 9.

an accurate reporter of microtubule polarity of the oocyte. The localization of an independent molecule, in particular to the anterior, might help to resolve this dilemma."

**Such a molecule has never been found.** Tests have been made by [**17**] that show the irrefutable fact MTs accumulate strongly at both anterior angles of the oocyte. Fig. 11 provides the proof of this, *Nod:βgal* being a tracer of MTs minus centrosome end, ***very much as if centrosomes were there***, with an accumulation of ***opposite polarity by Kin:βgal at the posterior of the oocyte***.

We will advance from such facts that we are here in a situation similar to the one between glial cells and neurons: Neurons lose their centrosome, but their MTs still maintain a definite pattern and set of motions. Here ***the follicle cells play the role of glial cells***.

We have already seen that these cells are relocating themselves to the posterior of the cystoblast (oocyte), so they had to sense the leptonic space manifold from the oocyte centriole when it was there, and followed this manifold receding with that centriole shutting down until they reached the corners where the oocyte starts. (There is no chemical signal from the oocyte saying "Move here!") This cell motion is reminiscent of neuron-glial cells motion. Fig. 12 (left) shows the location of the centrioles in these cells next to the oocyte at its stage 10b, with Fig. 12 (right) displaying their MT system at the opposite end of the cells: Their manifolds must be connected to the oocyte manifold. This is to be compared with what is happening in the late stage 9 oocyte in Figs. 11d and 11e: Now the oocyte cytoplasm is limited to its surface per Fig. 8, as the oocyte is filling up with yolk, and the internal oocyte centriole can no longer fulfill its directing function (anyway, its job is done since bicoid mRNA and other gene products have been brought in, shutting the centriole down by their presence).

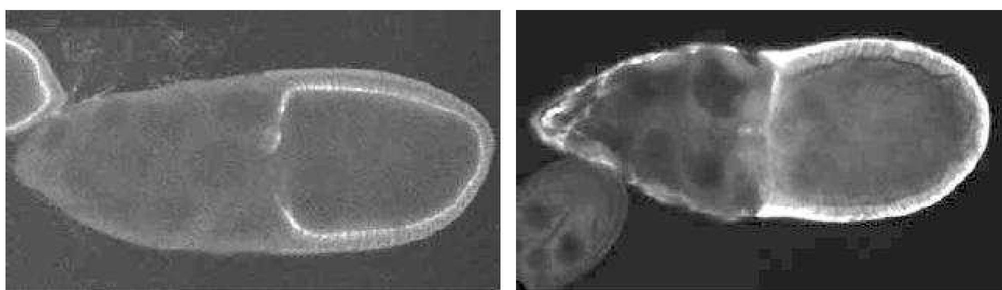


Fig. 12 ([**17**], Figs. 2cd)

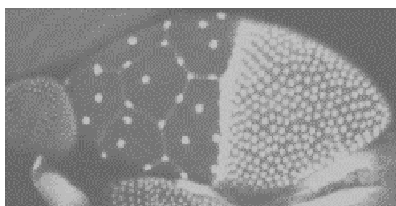


Fig. 13 ([**5**]) **Follicle cell nuclei**

So follicle cells are taking over the oocyte MTs: (Fig. 13)

At that point, ***as for neurons*** (Fig. 14), the oocyte manifold becomes two manifolds sustained by centrioles in the follicle cells at both anterior corners, with one manifold along the anterior cytoplasm and the other along the posterior, and the center of the oocyte MT system becomes the center axis of that new manifold. When combined with the central

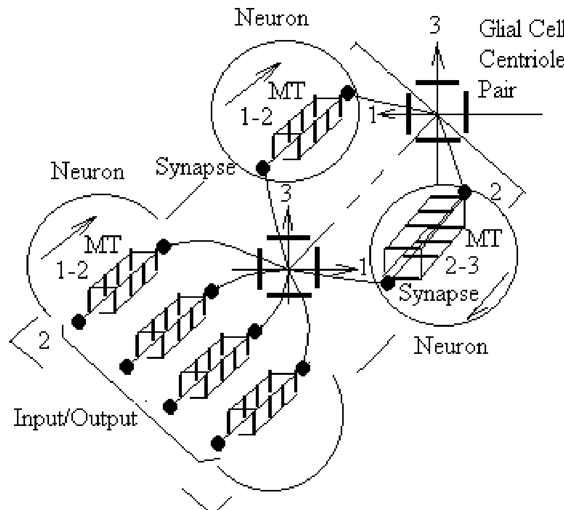



Fig. 14 – **Relationships between neurons and glial cells** (from [**4**])

location of the yolk, pushing the cytoplasm against the follicle cells (through the force of nurse cells deflating), the two different leptonic dimensional arrangements 1-2 and 2-3 explains the selective (MT-helped) motion of molecules, which were already in the oocyte through the earlier oocyte centriole action: (1) *bicoid* at the anterior - plus end of the first manifold, (2) *oskar* at the posterior – plus end of the second manifold, and (3) *gurken* moving to the dorsal part since now this part is the minus end of the second (posterior) manifold where gurken initially was. This respectively defines the anterior/ posterior and dorsal/ventral orientations of the oocyte as described by the literature without the physics. *The posterior and anterior axial parts did become a plus end area for the MTs* ***as in neuronal axons***. When it will be the turn of the follicle cells to disappear, as we shall see below, the anterior manifold will recede toward its dorsal part, and the pronucleus located earlier at the anterior will be moved there also. (This will be a crucial location for the later embryo development.)

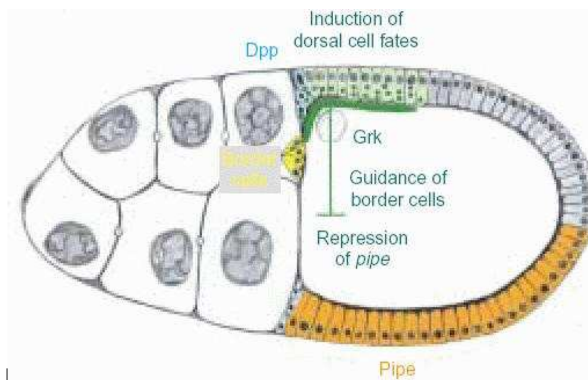



Fig. 15 - **Stage 10 egg chamber.** Note here that this figure from [**8**] is misleading as it implies follicle cells spread gene products (*gurken* and *pipe* ventralizing signals), while *the oocyte shell cytoplasm is the spreading medium*.

This behavior of the follicle cells participating in the finalization of the oocyte has to be put in context with what they are doing outside the oocyte: (Fig. 15) These same cells end up forming in the anterior dorsal area (1) a "*micropyle,*" the egg's terminal structure through which the sperm will pass into the egg, and (2) large anterior appendages for later embryo respiration. They also secrete the three layers of the eggshell: the *vitelline* envelope, the *endochorion*, and the *exochorion*. These facts are evidence that follicle cells are in charge of the oocyte development at that point: They must be themselves taking their cue on position from the gene product *gurken* (relocated via their own earlier action on the oocyte MTs), so they (1) know where to form the dorsal appendages, and (2) in-

stigate *border cells* differentiation ***induced by dpp expression*** for the later formation of a properly located and functional micropyle.

**d. Final fast cytoplasm transport – nurse cells apoptosis [5]**

The preparations and execution of fast transport are part of a programmed cell death (*apoptosis*) ***triggered by the deflation of the centriole leptonic manifold*** seen earlier, leaving the nuclei in charge of their cytoplasm and the elements in it. First, arrays of actin filament bundles are formed, extending ***straight from the cell membranes to the nuclear envelopes*** (that will stop the large nuclei from blocking ring canals during apoptotic contraction). (Fig. 16)

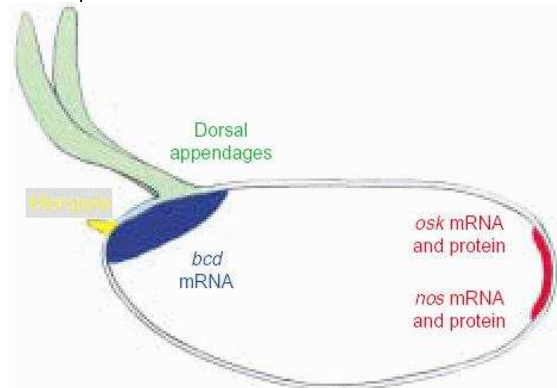
Fig. 16 ([**5**])

The nuclear envelopes then become permeable, releasing nuclear contents into the cytoplasm, including ***free calcium***, which in turn triggers the final nurse cells contraction (effected by cortical actin and cytoplasmic myosin II).

**e. Development of the dorsal appendages [8]**

Dorsal appendage morphogenesis in Drosophila oogenesis is a model system for studying the ***relationship between gene products action vs. physical morphogenesis***. (Fig. 17) Each of the two dorsal appendages of the egg chamber (facilitating gas exchange in the developing embryo) is formed by follicle secretion of eggshell proteins into a tube of cells. (Fig. 17) This tube is generated by cell shape changes and rearrangements within an epithelial sheet. Epithelial morphogenesis is essentially directed by the genetic ***induction signals*** that determine two populations of dorsal follicle cells from the location identified by *gurken*, itself, as we have seen, obtained earlier through physical action by the follicle cells onto the oocyte. This understanding then allows uncoupling *patterning* generated by the presence of gene products, from physical morphological processes not coming from the genes.

Fig. 17 - **Stage 14 - dorsal appendages and micropyle.** *bicoid*, *oskar* and *nanos* remain anchored and translationally dormant in the thin cytoplasm of the egg for use after fertilization. [**8**]



## The End - arrested oocyte meiosis waiting for fertilization

As late as the egg moving through the oviduct and as early as stage 13, i.e. while nurse cells are still degenerating, the oocyte nuclear material goes in a stable arrested ***meiosis metaphase I*** state, with its spindle positioned close and ***parallel to the cortex*** in the dorsal anterior part of the egg. The spindle shows slight movements or changes in position over periods of 15–30 min observation, undergoing dynamic changes such as slight extensions/contractions in length and alternating clockwise- /counter-clockwise rotational movements, probably showing fluctuations in regional energetic supply to the kinetochores maintaining the spindle (no centrosome is present to stabilize it). [**18**]

***The poles of the metaphase I spindle will naturally align parallel to the cortex*** in the dorsal part of the egg's *anterior manifold* as the follicle cells epidermis that formed the dorsal appendages remains there, and so does the dorso-ventral polarization of the oocyte thin cytoplasm at that location.

The weakness of the manifold connection appears to be mainly tied to the chorion envelopes integrity, as any action on the chorion affecting the manifold (natural via sperm entry, or artificial) triggers spindle reorientation, as we shall see in a later embryo study.

## Final remarks

We have here a neat example of a complete animal organism development standing by itself with a single purpose: producing a polarized egg. The evolution of the germarium is not solely a series of chemical happenings, by far. The director of the show is not the part the literature concentrates on, i.e. the chemistry of the nurse cells. For example, it could be verified that, most of the time, the chosen oocyte is the original cell that gave the cystoblast as a result of the fusome physically tracing its fate.

Concerning the polarization of the oocyte, we show via pictures that the follicle cells are very vibrant indeed when it comes to their MT system throughout the process, while the nurse cells are just chemically reacting to orders. After the 16 cells have been produced by programmed division directed by the fusome produced by the nuclear system, there is a long series of happenings that differentiate the oocyte in which genetic material gets produced and the centriolar system changes. Who or what is directing that show? It cannot be the centrioles of the nurse cells as they disappear, and the genes have no means on their own to tell at what stage the system is at when the fusome is gone. The follicle cell centrioles on the other hand do, so they have to be the collective director of the show then, very much like glial cells. The experimenter needs to see what happens if the follicle cell centrioles at the oocyte corners are destroyed.

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