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Original Article

Posterior localization of the *Drosophila* Giα protein during early embryogenesis requires a subset of the posterior group genes

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ABSTRACT Shortly after fertilization in *Drosophila* embryos, the G-protein α subunit, Giα, undergoes a dramatic redistribution. Initially granules containing Giα are present throughout the embryonic cortex but during nuclear cleavage they become concentrated at the posterior pole and are lost by the blastoderm stage. Mutations that eliminate anterior structures *bicoid, swallow,* and *exuperantia* did not prevent the posterior accumulation of Giα. Likewise, embryos from mothers with dominant gain of function mutations in the *Bicaudal D* gene show normal polarization of Giα granules. By contrast, a subset of mutations which eliminate posterior structures, *cappuccino, spire, staufen, mago nashi, valois,* and *oskar,* prevented the posterior accumulation of Giα. It is important to note that mutations in posterior genes lower in the putative hierarchy *vasa, tudor nanos,* and *pumilio* did not affect Giα redistribution. From these results we conclude that Giα redistribution to the posterior pole depends on maternal factors involved in the localization of the posterior morphogen *nanos.*

KEY WORDS: *Drosophila, G-protein, localization, embryo, polarized*

Introduction

G protein-coupled signal transduction represents the most diverse and evolutionarily ancient mechanism of transmembrane signaling. This process utilizes a receptor that modulates the activity of intracellular second-messenger systems through the activation of a limited set of intermediary GTP-binding or G proteins. In metazoans, receptors for a wide variety of extracellular signals are all coupled to G proteins and subsequently modulate a variety of effector molecules that synthesize cytoplasmic second messengers or act as ion channels (for review see Neer, 1994, 1995). All G proteins that mediate transmembrane signaling exist as a complex composed of α, β, and γ subunits in their inactive state. In this state, the α subunit contains a bound GDP and the complex associates with a vast array of receptors that have been traditionally characterized as having seven transmembrane domains. When ligand binds to its receptor, the receptor activates one or more G proteins by promoting the exchange of GTP for GDP on the α subunit and the dissociation of α from βγ. Termination of the signal occurs when GTP bound by the α subunit is hydrolyzed to GDP by an enzymatic activity intrinsic to the α subunit (Bourne *et al.*, 1991). Although βγ subunits also have been demonstrated to directly modulate a number of intracellular effector pathways (Clapham and Neer, 1993), the key role of the α subunit of G proteins is to mediate and coordinate signaling from a vast array of receptors to appropriate regulation of effector molecules (Neer, 1995). In addition, since a single receptor activates many G proteins, α subunits are also responsible for amplifying signals from receptors and then directing them to the correct effectors. α subunits can then be thought of as signal integrators and molecular "on/off switches"; central control points regulating and integrating the opening and closing of signaling pathways that result in the modulation of general cellular metabolism, cellular differentiation and cell growth.

In *Drosophila* 6 known G-protein α subunits have been cloned with an eye to using mutational analysis to reveal novel functions in the context of an intact multicellular organisms (reviewed in Forte *et al.*, 1993; Quan *et al.*, 1993). Wolfgang *et al.* (1991) described the embryonic pattern of expression of three α subunits, Gs, Go, and Gi. Each displayed a specific temporal and spatial pattern in the embryo indicating the potential for unique developmental functions for each subunit. Because of the known importance of polarization of macromolecules and cell signal transduction in establishing embryonic polarity and position (St. Johnston and Nüsslein-Volhard, 1992), we found the rapid polar-

Abbreviations used in this paper: bcd, bicoid; BicD, Bicaudal D; cap, cappuccino; exu, exuperantia; mago, mago nashi; nos, nanos; osk, oskar; pum, pumilio; stau, staufen; sww, swallow; tud, tudor; vas, vasa; vls, valois; Giα, G-protein α subunit i; HSP, heat shock protein.

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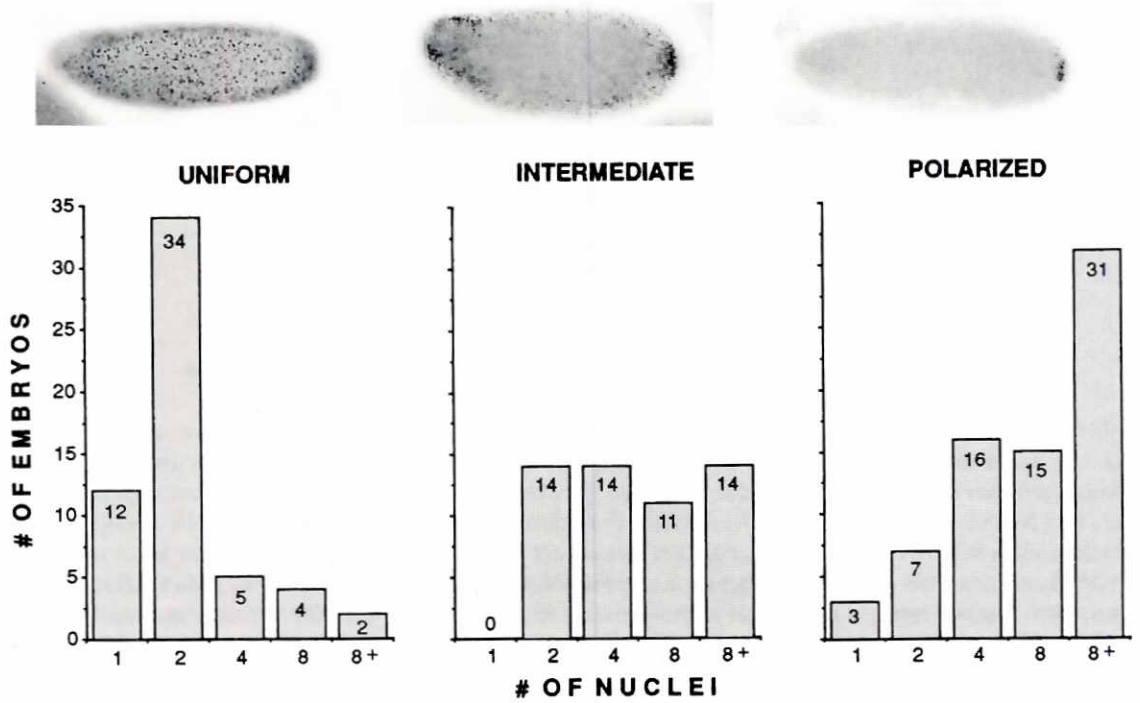

Fig. 1. Giα distribution in cleavage stage embryos. *A 0-3 h collection of embryos were double stained for Giα and nuclei. Each embryo was classified as having a uniform, intermediate or polarized distribution of granules and the number of nuclei noted. The embryo at the top of each histogram illustrates the Giα distribution of the class of embryos counted for the underlying histogram. Among embryos with 1 or 2 nuclei, 66% had uniform, 20% intermediate and 15% polarized granule distribution, whereas, among embryos with 8 or more nuclei, 8% had uniform, 32% intermediate and 60% polarized granule distribution. Thus, initially Giα containing granules are distributed uniformly and then during the early cleavage stage become restricted to the posterior pole. However, this transition is not tightly linked to one nuclear stage and appears to be a gradual process. Polarization is always completed by the syncytial blastoderm stage. Actual number of embryos counted are in the bars.*

ization of granules containing Giα to the posterior pole in early embryos intriguing.

Prior to fertilization, Giα protein is uniformly distributed in the ooplasm but, upon fertilization, becomes concentrated in granules in the cortex of the embryo (Wolfgang *et al.*, 1991). This pattern persists until roughly the 8 nuclei stage when the granules are restricted to the posterior pole. By the syncytial blastoderm stage, the few remaining granules become localized at the boundary between the yolk and the cytoplasm at the posterior pole. After the blastoderm stage the granules are no longer detected. Thus, the restriction of Giα to the posterior pole is rapid, transient and coincides with the time when maternal factors are establishing embryonic position and polarity.

TABLE 1

PUTATIVE HIERARCHY OF MATERNAL GENES OF THE POSTERIOR GROUP AND THEIR EFFECT ON GIα LOCALIZATION

Maternal Mutant*	Giα Localization
cap/spir	-
stau	-
osk	-/+
vas	+
tud	+
nos	+

The localization of gene products lower in the list depends on the normal function of gene products higher in the list (Ephrussi *et al.*, 1991). -, disrupted localization; +, normal localization. * Not all the genes tested in the paper are listed as their position in the hierarchy is unknown or controversial.

In *Drosophila*, the early embryonic pattern is established by the asymmetric distribution of macromolecules in the oocyte and zygote. Mutational analysis (assaying for the loss of anterior or posterior embryonic structures) has identified many of these molecules and to a certain extent the mechanisms by which they become localized and function (St. Johnston and Nüsslein-Volhard, 1992). Thus, the anterior determinant, *bicoid*, will not be properly localized if the mother is mutant for either *staufen*, *exuperantia*, and *swallow* (Frohnhöfer and Nüsslein-Volhard, 1987; Berleth *et al.*, 1988; Stephenson *et al.*, 1988). By contrast, the posterior determinant, *nanos* (Wang and Lehmann, 1991), is mislocalized if the mother carries any one of at least eight different mutations in what are collectively known as posterior group genes *(cappuccino, spire, staufen – stau* is required for both posterior and anterior localization {St. Johnston *et al.*, 1991}, *oskar, vasa, tudor, valois, mago nashi)* (Boswell and Mahowald, 1985; Lehmann and Nüsslein-Volhard, 1986; Schüpach and Wieschaus, 1986; Manseau and Schüpach, 1989; Lasko and Ashburner, 1990; Boswell *et al.*, 1991; St. Johnston *et al.*, 1991; Lehmann and Nüsslein-Volhard, 1991). Furthermore, the posterior localization of HSP 83 protein (Ding *et al.*, 1993), cyclin B mRNA (Raff *et al.*, 1990), and components of the posterior group

themselves *stau,* (St. Johnston *et al.*, 1991) *osk,* (Ephrussi *et al.*, 1991) *tud* (Bardsley *et al.*, 1993) and *vas* (Hay *et al.*, 1990; Lasko and Ashburner, 1990) as well as *nos* (Wang and Lehmann, 1991) have been shown to require the normal function of posterior group genes.

This sort of mutational analysis, observing the consequences of a single mutation on the distribution or function of a particular macromolecule has begun to reveal the hierarchical organization of biochemical elements in early *Drosophila* development (Table 1). For this reason we wished to determine the impact, if any, on Giα distribution of the same constellation of gene products known to be required for the correct localization in the early embryo of other molecules.

In this report, we have tested whether Giα localization depends on the same biochemical mechanisms required for posterior localization of the *nanos* morphogen by examining the Giα distribution in a number of well characterized maternal effect mutants altering the anterior-posterior pattern of the embryo. We find that mutations that eliminate anterior structures *(bicoid, swallow,* and *exuperantia)* have no effect on Giα redistribution. By contrast, a subset of the mutations that block formation of posterior structures also block redistribution of Giα containing granules (Table 1). Some of these mutations, *cappuccino, spire,* and *staufen* are known to be involved in the localization of pole plasm constituents but are not themselves the actual posterior determinants (St. Johnston and Nüsslein-Volhard, 1992). From these results we conclude that the restriction of Giα containing granules to the posterior pole requires a functional system for localizing posterior embryonic determinants.

Results

Giα distribution in wild type embryos

Previous studies showed that shortly after egg laying, uniformly distributed Giα granules become restricted to the posterior pole early during embryonic cleavage stages (Wolfgang *et al.*, 1991). In order to document the time course of this process more accurately, the distribution of Giα containing granules was detected immunohistochemically followed by propidium iodide staining in the same embryos to allow accurate nuclear counts. In Fig. 1, the number of embryos containing 1 through 8 or more nuclei is shown for each of three classes of embryos containing: 1) uniformly distributed granules; 2) partially polarized granules; 3) and fully polarized granules. The results indicate that in a population of embryos the number of embryos containing polarized granules gradually increases during the first four nuclear divisions but clearly is not tightly regulated with respect to during which cleavage stage the polarization occurs. However, after the fourth nuclear division, most embryos (92%) have the polarized or partially polarized appearance. The polarization process is always completed by the syncytial blastoderm stage.

Anterior class mutants

Correct localization of the anterior determinant, *bicoid,* requires the products of at least the *exuperantia*, and *swallow* genes (Frohnhöfer and Nüsslein-Volhard, 1987). To determine if the redistribution of the Giα protein was affected by mutations in these genes, embryos from mothers homozygous for strong mutations in the *bicoid, swallow*, or *exuperantia* genes were collected and stained for Giα containing granules. In each case, the distribution of Giα granules was indistinguishable from wild type mothers or mothers heterozygous for the tested mutation (Fig. 2); i.e. embryos containing both uniform, partially polarized, and polarized distribution of granules were observed and granules were lost by the end of the blastoderm stage. Thus, genes in the pathway responsible for the correct localization and function of the anterior morphogen *bicoid* do not affect the redistribution of the Giα containing granules.

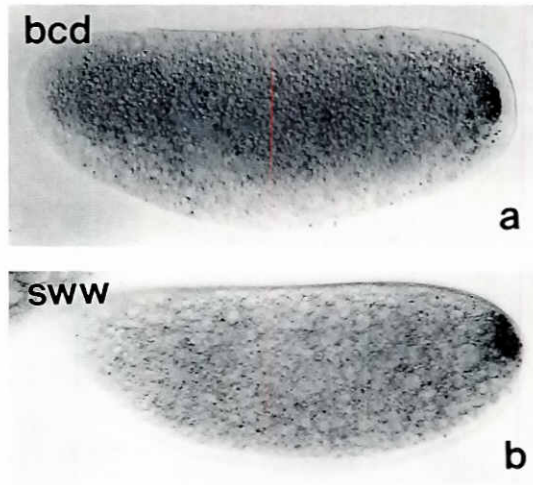

Fig. 2. Giα distribution in anterior class mutants. *Embryos from* **(a)** *bcd,* **(b)** *sww, homozygous mothers show a wild type pattern of Giα redistribution to the posterior pole. Posterior is to the right.*

Posterior class mutants

Localization of *nanos* RNA, the posterior morphogen, requires the function of at least 9 different genes *(cappuccino, spire, staufen, oskar, vasa, tudor, valois, mago nashi, pumilio)* (Boswell and Mahowald, 1985; Lehmann and Nüsslein-Volhard, 1986; Schüpach and Wieschaus, 1986; Manseau and Schüpach, 1989; Lasko and Ashburner, 1990; Boswell *et al.*, 1991; St. Johnston *et al.*, 1991). To test the role of genes in the posterior group in the redistribution of Giα containing granules, embryos collected from females homozygous for strong mutations in each of the genes in this class were examined. Females with mutations in the *cappuccino, spire, staufen, oskar* and *mago nashi* genes produced embryos with uniformly distributed Giα granules which were lost at the blastoderm stage and never became localized to one pole (Fig 3a-d). Heterozygous mothers for these mutations produced embryos with a wild type granule distribution. In syncytial blastoderm stage, embryos from females containing mutations in this group have Giα granules that become concentrated at the boundary of the yolk and cytoplasm around the whole embryo and not just at the posterior pole (particularly evident in *mago* mutations). By contrast, in embryos from mothers homozygous for *vasa, tudor, nanos,* and *pumilio* mutations, the Giα distribution pattern was similar in appearance to embryos from mothers heterozygous or wild type for the tested mutation (Fig. 3e-h). Some $oskar^{54}$ embryos contained weakly polarized Giα granules (Fig. 3i) but in the majority of embryos, the granules were uniformly distributed (Fig. 3d). Since $oskar^{54}$

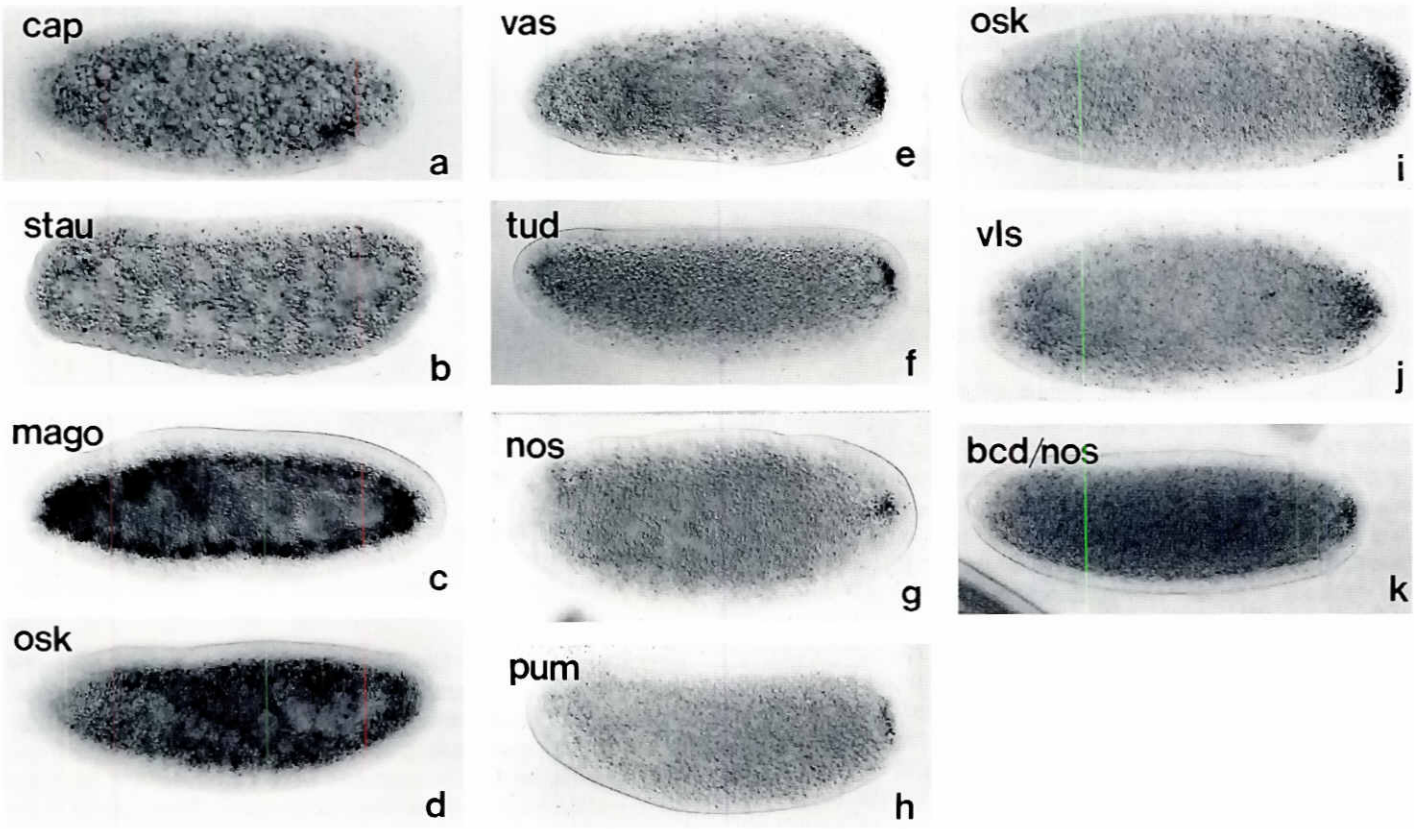

Fig. 3. Giα distribution in posterior class mutants. *Embryos from mothers homozygous for mutations in the posterior group gene indicated* **(a-d).** *In cap, stau, mago, and osk syncytial blastoderm stage embryos Giα staining granules remain uniformly distributed and never become polarized.* **(e-h)** *In contrast, embryos from vas, tud, nos, pum mothers show normal polarization of Giα granules.* **(i and j)** *Some osk and vls embryos show weak polarization of Giα.* **(k)** *bicoid/ nanos double mutant with normal polarized granules. Posterior is to the right.*

disrupts *staufen* protein localization, we examined a second strong *oskar* allele, *oskar*^{166}, which does not disrupt *staufen* localization (St. Johnston *et al.*, 1991). These embryos displayed the same phenotype as *osk*^{54} suggesting that the *oskar* phenotype results from disruption of *osk* and not *stau* mislocalization. *Valois* embryos contained fewer polarized embryos than controls (Fig. 3j) and like *oskar*^{54}, in the few embryos that contained polarized granules, granules were not concentrated at the posterior pole to the same extent as controls. Embryos produced by *bicoid/nanos* double mutants contained fully polarized granules (Fig. 3k).

Bicaudal D embryos

In embryos mutant for the dominant gain of function mutation *Bicaudal D*, a mirror image duplication of the abdomen is formed from the anterior end of the embryo (Mohler and Wieschaus, 1986). Because Giα containing granules become associated with the posterior pole in cleavage stage embryos, we determined whether the polar localization of Giα would also be duplicated in *Bicaudal D* embryos. Compared to wild type controls, *Bicaudal D* embryos showed a wild-type distribution of granules (Fig. 4a). In embryos in which pole cells were present, localization was to the true posterior pole and not the transformed anterior pole.

Recently transformants have been described in which *oskar* gene sequences have been attached to the *bicoid* 3' UTR containing sequences required for the anterior localization of the *bicoid* morphogen (*mob* transformants, Webster *et al.*, 1994; *osk-bcd* 3' *utr,* Ephrussi and Lehmann, 1992). This results in mislocalization of *oskar* to the anterior pole and the production of bicaudal embryos with anterior pole cells. However, as described above for *Bicaudal* D mutations, Giα distribution was normal in *mob* transformant embryos (Fig. 4b).

Discussion

In this study we confirmed and extended our initial observation that Giα granules come to reside at the posterior pole during the first 4 nuclear cleavage cycles and then are lost during the blastoderm stage. During this time, maternally provided information is being interpreted to establish the anterior-posterior and dorsal-ventral embryonic axes (St. Johnston and Nüsslein-Volhard, 1992). In this report, we show that restriction of Giα granules to the posterior pole of the embryo depends on a subset of the maternal genes required for localization of posterior determinants (Table 1). This is significant because it links control of the distribution of Giα, a signal transduction molecule, to the biochemical pathway that establishes the embryonic posterior pole.

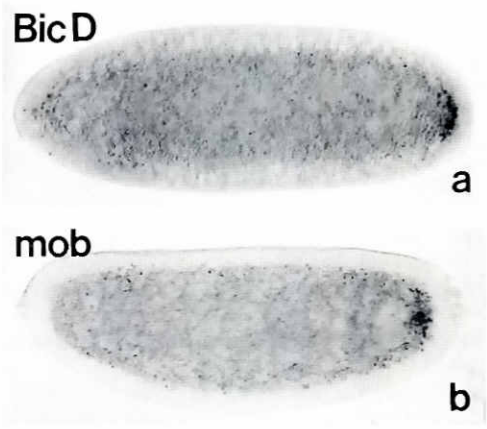

Fig. 4. Giα distribution in bicaudal embryos. *Syncytial blastoderm stage embryos from* **(a)** *$BicD^1/BicD^2$ or* **(b)** *pmob mothers stained for Giα showing the normal redistribution of Giα to the posterior pole. Posterior is to the right.*

The mutations, *bcd, sww,* and *exu* that cause the loss of anterior structures, had no effect on Giα distribution. By contrast, mutations in a subset of the posterior group genes, *(cap, spir, stau, osk, mago,* and *vls)* which eliminate posterior structures, disrupt polarization of Giα. Mutations in other members of this group which eliminate posterior structures *(vas, tud, nos, pum),* had no effect on Giα redistribution as did mutations in the *Bic D* locus or *mob* transformants. No mutations resulted in the absence of Giα containing granules in early cleavage stage embryos or blocked their loss at the blastoderm stage, indicating that synthesis and degradation are regulated by mechanisms other than polarization.

The posterior class mutations have been divided into two groups, based largely on their interaction with the *BicD* locus. When *cap, spir, or stau* are placed in a *BicD* background, an abdomen forms at the anterior end but no abdomen forms at the posterior end. By contrast, when *osk, vas, tud* and *nos* are placed in a *BicD* background neither anterior nor posterior abdomens form. These observations, as well as the results of other experiments in which *osk* or *nos* mRNA is mislocalized (Ephrussi and Lehmann, 1992; Gavis and Lehmann, 1992; Smith *et al.*, 1992; Webster *et al.*, 1994) suggest that *cap, spir,* and *stau* are required for the correct transport and/or anchoring of posterior pole plasm components, whereas, *osk, vas, tud, vls* and *nos* are required for production of the activities that induce the formation of germ cells and the abdomen. In this context, our results show that Giα polarization depends on genes involved in the localization of pole plasm components *(cap, spir, stau)* but not on genes required for the production of an active posterior pole plasm, with the exception of *osk*. This suggests that Giα polarization uses, at least in part, the same machinery as other pole plasm components to become restricted to the posterior pole but does not require an active posterior center. The fact that in *bcd/nos* double mutants Giα polarization is normal (Fig. 3k) demonstrates that neither the anterior nor posterior morphogen are required. The normal distribution of Giα containing granules in *Bic D* and *mob* embryos in which an active posterior center is mislocalized to the anterior pole is consistent with this conclusion.

osk gene products are not believed to participate in global localization processes, yet with two different alleles no or partial polarization of Giα granules is observed. Three plausible explanations are outlined below. 1) The partial localization observed in *osk* may result from some partial function of the mutant gene product. For osk^{166}, which is a single amino acid substitution, this is possible but for osk^{54} this is unlikely as the open reading frame is truncated by a stop codon (Kim-Ha *et al.*, 1991) presumably producing a functionally null gene product. 2) In the absence of *osk* gene function, transport and localization of Giα is largely blocked with a redundant gene product providing the weak and variable polarizing activity observed. 3) *osk* may be required for stabilization of Giα granules to the posterior but not the initial transport and localization. A similar stabilizing role for *osk* has been proposed in the posterior localization of staufen protein (St. Johnston *et al.*, 1991) though the mechanism would be different since osk^{166} but not osk^{54} stabilizes staufen whereas neither allele stabilizes Giα to the posterior.

The intermediate phenotype produced in *valois* embryos (i.e., weak polarization of granules) suggests that polarization may be occurring but cannot be maintained. This is quite similar to the effect of *valois* mutations on *vasa* protein which is initially polarized in *vls* mutants, but the polarization is not maintained (Hay *et al.*, 1990).

Our results demonstrate that the normal restriction of Giα to the posterior pole in cleavage stage embryos depends on a subset of maternal gene products involved in the transport and/or localization of pole plasm components. Furthermore, maintenance of the polarized state may depend on *osk* and *val* whose gene products are required for pole plasm activity. Whether there is a direct dependence of Giα localization on these identified maternal gene products, or whether the mislocalization of Giα occurs because of the absence of some other factor whose function in turn depends on maternal gene products we examined, is not known.

Materials and Methods

Stocks

Stocks were maintained and eggs collected and aged at room temperature or 25°C. Canton S strain were used as wild type controls. Anterior class mutants obtained from the stock center were *bicoid* ^{E1}, *swallow* ^{3}, and *exuperantia*^{PJ}. Posterior group mutants kindly provided by Dr. Ruth Lehmann were *cappuccino*^{RK}, *spire* ^{RP}, *staufen* ^{D3}, *oskar*^{54} (eggs collected from *oskar*^{54}/ *Df (3R)p*^{xt103} females), *vasa*^{D1}, *vasa*^{PD} (eggs collected from *vasa*^{D1}/*vasa*^{PD} females), *tudor* ^{WC8}, *valois*^{PE} (eggs collected from *valois*^{PE}/ *Df (2R) TW2* females), *nanos*^{L7}, *pumilio*^{680}. Dr. Paul MacDonald kindly provided the *oskar*^{166} allele and the *mob* transformants. Dr. Robert Boswell kindly provided the *mago nashi*^{1} flies. Bicaudal embryos were collected from *$BicD^1/BicD^2$* mothers.

Staining

Antibody staining for Giα was as described in Wolfgang *et al.* (1991). For propidium iodide staining of nuclei, after fixation but prior to initiation of antibody staining, embryos were incubated for 2 h at 37°C in 10 ug/ml RNAse. Antibody staining was then completed and embryos mounted in 80% glycerol, 0.02 M Tris (pH 7.7), 1.25 ug/ml propidium iodide. The propidium iodide was viewed with the rhodamine filter set.

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References

BARDSLEY, A., MacDONALD, K. and BOSWELL, R.E. (1993). Distribution of tudor protein in the *Drosophila* embryo suggests separation of functions based on the site of localization. *Development 119:* 207-219.

BERLETH, T., BURRI, M., THOMA, G., BOPP, D., RICHSTEIN, S., FRIGERIO, G., NOLL, M. and NÜSSLEIN-VOLHARD, C. (1988). The role of localization of *bicoid* RNA in organizing the anterior pattern of the *Drosophila* embryo. *EMBO 7:* 1749-1756.

BOSWELL, R.E. and MAHOWALD, A.P. (1985). *tudor*, a gene required for assembly of the germ plasm in *Drosophila melanogaster. Cell 43:* 97-104.

BOSWELL, R.E., PROUT, M.E. and STEICHEN, J.C. (1991). Mutations in a newly identified *Drosophila melanogaster* gene, *mago nashi*, disrupt germ cell formation and result in the formation of mirror-image symmetrical double abdomen embryos. *Development 113:* 373-384.

BOURNE, H., SANDERS, D., and McCORMICK, F. (1991). The GTPase superfamily: conserved structure and molecular mechanism. *Nature 349:* 117-127.

CLAPHAM, D. and NEER, E. (1993). New roles for G protein dimers in transmembrane signaling. *Nature 365:* 403-406.

DING, D., PARKHURST, S.M., HALSELL, S.R. and LIPSHITZ, H.D. (1993). Dynamic *Hsp83* RNA localization during Drosophila oogenesis and embryogenesis. *Mol. Cell. Biol. 13:* 3773-3781.

EPHRUSSI, A. and LEHMANN, R. (1992). Induction of germ cell formation by *oskar. Nature 358:* 387-392.

EPHRUSSI, A., DICKINSON, L.K. and LEHMANN, R. (1991). *oskar* organizes the germ plasm and directs localization of the posterior determinant *nanos. Cell 66:* 37-50.

FORTE, M., QUAN, F., HYDE, D., and WOLFGANG, W. (1993) Gα proteins in *Drosophila:* structure and developmental expression. In *Handbook of Experimental Pharmacology. GTPases in Biology II*, Vol. 108/II (Eds. B.F. Dickey and L. Birnbaumer). Springer-Verlag, Berlin, pp. 319-334

FROHNHÖFER, G.F. and NÜSSLEIN-VOLHARD (1987). Maternal genes required for the anterior localization of bicoid activity in the embryo of *Drosophila. Genes Dev. 1:* 880-890.

GAVIS, E.R. and LEHMANN, R. (1992). Localization of nanos RNA controls embryonic polarity. *Cell 71:* 301-313.

HAY, B., JAN, L.Y. and JAN, Y.N. (1990). Localization of vasa, a component of *Drosophila* polar granules, in maternal-effect mutants that alter embryonic anteroposterior polarity. *Development 109:* 425-433.

KIM-HA, J., SMITH, J.L., and MacDONALD, P.M. (1991) *oskar* mRNA is localized to the posterior pole of the *Drosophila* oocyte. *Cell 66:* 23-35.

LASKO, P.F. and ASHBURNER, M. (1990). Posterior localization of *vasa* protein correlates with, but is not sufficient for, pole cell development. *Genes Dev. 4:* 905-921.

LEHMANN, R. and NÜSSLEIN-VOLHARD, C. (1986). Abdominal segmentation, pole cell formation, and embryonic polarity require the localized activity of *oskar*, a maternal gene in *Drosophila. Cell 47:* 141-152.

LEHMANN, R. and NÜSSLEIN-VOLHARD, C. (1991). The maternal gene *nanos* has a central role in posterior pattern formation of the *Drosophila* embryo. *Development 112:* 679-691.

MANSEAU, L.J. and SCHÜPACH, T. (1989). *cappuccino* and *spire:* two unique maternal effect loci required for both the anteroposterior and dorsoventral patterns of the *Drosophila* embryo. *Genes Dev. 3:* 1437-1452.

MOHLER, J. and WIESCHAUS, E.F. (1986). Dominant maternal-effect mutations of *Drosophila melanogaster* causing the production of double-abdomen embryos. *Genetics 112:* 803-822.

NEER, E. (1994). G proteins; critical control points for transmembrane signals. *Protein Sci. 3:* 3-14.

NEER, E. (1995). Heterotrimeric G proteins: organizers of transmembrane signals. *Cell 60:* 249-257.

QUAN, F., WOLFGANG, W.J. and FORTE, M. (1993) A *Drosophila* G-protein α subunit, Gfα, expressed in a spatially and temporally restricted pattern during *Drosophila* development. *Proc. Natl. Acad. Sci. USA 90: 4236-4240.*

RAFF, W.R., WHITFIELD, W.G.F. and GLOVER, D.M. (1990). Two distinct mechanisms localize cyclin B transcripts in syncytial *Drosophila* embryos. *Development 110:* 1249-1261.

SCHÜPACH, T. and WIESCHAUS, E. (1986). Maternal-effect mutations altering the anterior-posterior pattern of the *Drosophila* embryo. *Roux Arch. Dev. Biol. 195:* 302-317.

SMITH, J.L., WILSON, J.E. and MacDONALD, P.M. (1992). Overexpression of *oskar* directs ectopic activation of *nanos* and presumptive pole cell formation in *Drosophila* embryos. *Cell 70:* 849-859.

St. JOHNSTON, D. and NÜSSLEIN-VOLHARD, C. (1992). The origin of pattern and polarity in the *Drosophila* embryo. *Cell 68:* 201-219.

St. JOHNSTON, D., BEUCHLE, D. and NÜSSLEIN-VOLHARD, C. (1991). *staufen*, a gene required to localize maternal RNAs in the *Drosophila* egg. *Cell 66:* 51-63.

STEPHENSON, E.C., CHAO, Y.C. and FACKENTHAL, J.D. (1988). Molecular analysis of the *swallow* gene of *Drosophila melanogaster. Genes Dev. 2:* 1655-1665.

WANG, C. and LEHMANN, R. (1991). *nanos* is the localized posterior determinant in *Drosophila. Cell 66:* 637-647.

WEBSTER, P.J., SUEN, J. and MacDONALD, P.M. (1994). *Drosophila virilis oskar* transgenes direct body patterning but not pole cell formation or maintenance of mRNA localization in *D. melanogaster. Development 120:* 2027-2037.

WOLFGANG, W.J., QUAN, F., THAMBI, N. and FORTE, M. (1991). Restricted spatial and temporal expression of G-protein α subunits during *Drosophila* embryogenesis. *Development 113:* 527-538.

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