Induction of *indora* expression in pole cells by the mesoderm is required for female germ-line development in *Drosophila melanogaster*

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**SUMMARY**

In many animal groups, the interaction between germ and somatic line is required for germ-line development. In *Drosophila*, the germ-line precursors (pole cells) form at the posterior tip of the embryos migrate toward the mesodermal layer where they adhere to the dorsolateral mesoderm, which ensheaths the pole cells to form the embryonic gonads. These mesodermal cells may control the expression of genes that function in pole cells for their development into germ cells. However, such downstream genes have not been isolated. In this study, we identify a novel transcript, *indora* (*idr*), which is expressed only in pole cells within the gonads. Reduction of *idr* transcripts by an antisense *idr* expression caused the failure of pole cells to produce functional germ cells in females. Furthermore, we demonstrate that *idr* expression depends on the presence of the dorsolateral mesoderm, but it does not necessarily require its specification as the gonadal mesoderm. Our findings suggest that the induction of *idr* in pole cells by the mesodermal cells is required for germ-line development.

Key words: pole cell, gonadal mesodermal cell, somatic gonadal precursors, *indora*, *Drosophila melanogaster*

**INTRODUCTION**

Germ-line development involves the interaction between germ-line cells and adjacent somatic cells. Studies in a number of organisms have shown that mesodermal cells of the adult ovary and testis support the differentiation of germ-line cells into mature gametes (Kimble and White, 1981; Tres and Kierszenbaum, 1983; Abe, 1988; Lin and Spradling, 1993; Henderson et al., 1994; Tax et al., 1994; Miura et al, 1996). Despite the importance of the somatic cells to gametogenesis, the role of these cells in germ-line development during embryogenesis is not well understood. In many animal groups, the germ line is proposed to be specified autonomously by maternal factors localized in the germ plasm, a histologically distinct region of the egg cytoplasm (Kimble and White, 1981; Tres and Kierszenbaum, 1983; Abe, 1988; Lin and Spradling, 1993; Henderson et al., 1994; Tax et al., 1994; Miura et al, 1996). Despite the importance of the somatic cells to gametogenesis, the role of these cells in germ-line development during embryogenesis is not well understood. In many animal groups, the germ line is proposed to be specified autonomously by maternal factors localized in the germ plasm (Kimble and White, 1981; Tres and Kierszenbaum, 1983; Abe, 1988). Experimental studies in *Xenopus* and *Drosophila* have demonstrated that factors capable of directing germ-line development are localized in the germ plasm (Ilmensee and Mahowald, 1974; Eddy, 1975). Experimental studies in *Xenopus* and *Drosophila* have demonstrated that factors capable of directing germ-line development are localized in the germ plasm (Ilmensee and Mahowald, 1974; Eddy, 1975). However, germ plasm-bearing cells do not necessarily differentiate into functional germ cells. Wylie et al. (1985) reported that in *Xenopus* embryos, germ plasm-bearing cells isolated during their migration toward the genital ridge are unable to become germ line when implanted into ectopic sites. Thus, germ-line development is not simply the consequence of autonomous function of germ plasm, but is also influenced by cellular environment (Wylie et al., 1985).

In *Drosophila*, the germ plasm is localized in the posterior pole region of early embryos and partitioned into the germ-line progenitors, or pole cells (Mahowald, 1962, 1968; Underwood et al., 1980; Technau and Campos-Ortega, 1986; Hay et al., 1988). During gastrulation, the pole cells move along the dorsal surface of the embryos, along with the posterior midgut rudiment. They then migrate through the midgut epithelium toward the overlying mesodermal layer, where they associate with the dorsolateral mesoderm in parasegments (PS) 10-12 (Boyle and DiNardo, 1995; Boyle et al., 1997; Moore et al., 1998a, b). The dorsolateral mesoderm in PS 10-12 is specified as somatic gonadal precursors (SGPs), and they coalesce with the associating pole cells to form the embryonic gonads (Boyle and DiNardo, 1995; Boyle et al., 1997; Moore et al., 1998a, b). The physical proximity of pole cells to the mesodermal cells raises the question as to whether germ-line development could be partly controlled by the mesodermal cells. Indeed, several mutations which abolish the gonadal mesoderm affect pole cell migration (Broihier et al., 1998; Moore et al., 1998a, b). For example, in *tinman* (*tin*), *zinc finger homeodomain protein-1* (*zfh-1*) double-mutant embryos where the dorsolateral mesoderm is ablated, pole cells pass through the midgut epithelium, but subsequently they are dispersed around the midgut (Broihier et al., 1998; Moore et al., 1998b). Furthermore, in *abdominal-A* (*abd-A*) mutants which fail to specify the dorsolateral mesoderm into SGPs, pole cells...
associate with the dorsolateral mesoderm, but are later released from the mesoderm and disperse throughout the embryo (Brookman et al., 1992; Cumberledge et al., 1992; Boyle and DiNardo, 1995; Greig and Akam, 1995; Broihier et al., 1998; Moore et al., 1998b). These mesodermal cells may induce gene expression in pole cells resulting in their development into functional germ cells. However, such downstream genes have remained elusive.

Here, we describe the identification of a novel transcript called indora (idr), which is expressed in pole cells within the embryonic gonads. Antisense experiments, which reduce the idr mRNA in embryos, reveal that idr is required for germ-line development in females. Furthermore, we present evidence showing that idr expression in pole cells is induced non-autonomously by the dorsolateral mesoderm, although it does not require the specification of the dorsolateral mesoderm as SGPs. These data strongly suggest that the induction of idr expression in pole cells by the mesodermal cells is essential for proper development of the germ line.

MATERIALS AND METHODS

Fly stock
The wild-type strain used was Oregon R. The tudor (tud), abd-A mutant and snail (sna), twist (twi) double-mutant stocks were kindly provided by the Bloomington Drosophila Stock Center. The waben (wan), infra-abdominal 4 (iab-4) and tin, zfh-1 double-mutant stocks were kindly provided by Drs Ken Howard, Shigeru Sakonju and Ruth Lehmann, respectively. Homozygous mutant embryos were collected from balanced heterozygous stocks: Lehmann, respectively. Homozygous mutant embryos were collected for more than 1000 RNA species, we identified a cDNA clone corresponding to mRNA derived from 1.5 kb full length cDNA. The amplified cDNAs were cloned into pBluescript SK and sequenced.

In situ hybridization and antibody staining
In situ hybridization to whole-mount embryos was carried out according to the method described by Tautz and Pfeifle (1989) with modification as described by Kobayashi et al. (1998). Digoxigenin (DIG)-labeled RNA probes were synthesized with T7 or T3 RNA polymerases in the presence of digoxigenin-UTP (Boehringer-Mannheim), using the 1.5 kb cDNA as a template. Following in situ hybridization, immunoperoxidase staining with an antibody against Vasa (a gift from Drs A. Nakamura and P. F. Lasko) was carried out as described previously (Simpson-Brose et al., 1994). The stained embryos were observed under a compound microscope (DMRB, Leica) equipped with Nomarski optics and were staged according to Campos-Ortega and Hartenstein (1985).

In situ hybridization to polytenic chromosomes was performed as described by Engels et al. (1986). Biotinylated probe was synthesized from the 1.5 kb cDNA using a Random Primed DNA Labeling kit (Boehringer Mannheim Biochemica).

Antisense experiments
The fragment corresponding to nucleotides 974-1507 of the 1.5 kb idr cDNA was inserted between the EcoRI and XbaI sites of pCaSpeR-hs (Thummel and Perrotta, 1992) to generate AS-idr, which expresses an antisense idr RNA under the control of the hsp70 promoter. AS-idr was co-injected into w- embryos with a helper plasmid, pnt25.7 wc. Independent w+ transformants were inbred to establish homzygous stock.

RESULTS

Isolation of idr cDNA
idr was identified as a gene expressed in pole cells. Using mRNA differential display technique (Liang and Pardee, 1992; Nakamura et al., 1996), we screened for RNA species that are present in wild-type embryos but absent or rare in the embryos derived from tud mutant females, which fail to form pole cells (Nakamura et al., 1996). From more than 1000 RNA species, we identified a cDNA clone corresponding to mRNA.
which is detected predominantly in pole cells by whole-mount in situ hybridization. This cDNA hybridized with 1.5 and 0.8 kb mRNAs on northern blots (Fig. 1A). We isolated cDNA clones corresponding to these idr mRNAs from an embryonic cDNA library (Fig. 1B). The nucleotide sequence of the 1.5 kb cDNA predicts a protein of 131 amino acids (Fig. 1B). The amino acid sequence showed no significant homology to any known proteins. The putative Idr protein is highly basic (calculated isoelectric pH is 10.1) with a calculated relative molecular mass of 16x10^3. The nucleotide sequence of the cDNAs corresponding to the 0.8 kb mRNA was identical to the 3'-untranslated region of the longer cDNA (Fig. 1B). Using the RACE technique, we isolated cDNA fragments corresponding to the 5' region of the shorter mRNA and determined their nucleotide sequences. These cDNAs initiated at around nucleotide 958 of the 1.5 kb cDNA, suggesting the possibility that the shorter mRNA was produced by alternative promoter usage. In situ hybridization with a fragment specific to the 1.5 kb transcript (351-759 nt in Fig. 1B) revealed that the transcript was expressed in pole cells within the gonads (data not shown). We mapped the location of the gene encoding the idr transcript to 42A on the right arm of the second chromosome by in situ hybridization to polytene chromosomes. During normal development, the expression of idr transcripts became discernible in pole cells at the embryonic stage 14, when pole cells are incorporated into the gonads (Fig. 2B). Expression persisted in pole cells until the completion of embryonic development (Fig. 2C, D). We also examined the expression of idr in testes and ovaries using whole-mount in situ hybridization, but idr expression was undetectable in the adult germ line (data not shown). However, we cannot exclude the possibility that a trace amount of idr mRNAs is expressed in somatic cells as well as in germ line throughout most of the life cycle, because northern blot analysis revealed that idr transcripts were detectable from late embryogenesis to adulthood (Fig. 1A).

**Effect of antisense idr expression on germ-line development**

In order to know whether idr is required for germ-line development, we attempted to reduce idr function in embryos by antisense idr expression. We constructed a fusion gene (AS-idr) which expresses an antisense idr RNA under the control of the heat shock protein 70 (hsp70) promoter. This fusion gene was introduced into flies by P element-mediated transformation, and two independent homozygous viable lines (AS16 and AS23) were established. We found that varying percentages of females from the two transformant lines showed sterility even in the absence of heat treatment (Table 1). Approximately 2% and 6% of the females were sterile in AS16 and AS23 lines, respectively. These sterile females had bilateral agamic ovaries that lacked differentiated ovarioles and developing egg chambers. Unilateral agamic ovaries were also found in 7% and 18% of AS16 and AS23 females, respectively (Table 1). In contrast, no sterility was observed in control females lacking AS-idr. These results show that AS-idr has a moderate effect on female fertility at 25°C.

If this phenotype is caused by antisense idr expression from AS-idr, it is expected that heat treatment would cause a stronger phenotype. Indeed, the sterility was enhanced by heat treatment at 29°C during a period from embryonic stage 10 to eclosion (Table 1). Of the females from heat-treated AS23 line, 21.8% had agamic ovaries, an increase from 6.8% obtained without heat treatment. However, of the AS23 females heat-treated from stage 15 to eclosion, only 8.1% were sterile (total number of females counted; n=148). Thus, the enhancement of the female sterility was no longer discernible when heat treatment had a moderate effect on female fertility at 25°C.

**Fig. 1.** (A) Northern blot of poly(A)^+ RNA from 12-24 hours embryos (lane 1), second instar larvae (lane 2), female adults (lane 3) and male adults (lane 4) probed with idr cDNA. Two idr transcripts (1.5 and 0.8 kb) were detected. (B) Nucleotide and deduced amino acid sequences of the 1.5 kb transcript. The 1.5 kb transcript includes a long open reading frame that encodes a polypeptide of 131 amino acids. The amino acid showed no significant homology to any other proteins identified so far. A cDNA corresponding to the shorter transcript was matched with the untranslated region of 1.5 kb transcript (indicated by underline). The nucleotide sequence has the GenBank accession number, Y13272.
was initiated after stage 15. Similar result was obtained when AS23 females were heat-treated after embryogenesis until their eclosion; only 5.8% of the resulting females were sterile (n=69). These results strongly suggest that the heat induction of antisense idr expression during embryonic stages 10-15 has a marked effect on female fertility. Northern blot analysis revealed that antisense idr expression by heat treatment during these stages reduced idr mRNA to undetectable levels in embryos (Table 1). In the absence of heat treatment, idr mRNA remained detectable, but at a reduced level, when compared with the control embryos lacking the AS-idr construct. The extent of idr mRNA reduction in these embryos correlates well with the severity of the sterile phenotype (Table 1). Taken together, our results demonstrate that the expression of antisense idr RNA causes a reduction of idr mRNA in embryos, leading to female sterility.

The presence of dorsolateral mesoderm is required to induce idr expression in pole cells

We then focused on the mechanism by which idr expression is regulated in pole cells during embryogenesis. We noted in normal embryos that almost all pole cells incorporated within the gonads expressed the idr transcript. In contrast, a small fraction of pole cells which remained outside the gonads never expressed idr mRNA (Fig. 2E). This suggests that idr expression is intimately linked to gonad formation, which involves the coordinate development of pole cells and the mesodermal cells becoming somatic components of the gonads (Brookman et al., 1992; Warrior, 1994; Boyle and DiNardo, 1995; Moore et al., 1998b). In these embryos, idr expression was discernible in pole cells that successfully assembled within the gonads, but not in those cells that remained outside the gonads (Fig. 3C-E).

The above observation suggests that correct migration of pole cells into the mesodermal layer is necessary for idr expression. We next examined idr expression in sna, twist double-mutant embryos, which completely lack mesoderm (Leptin, 1991; Ray et al., 1991). In the absence of mesoderm, pole cells normally pass through the midgut epithelium but subsequently are dispersed around the midgut due to the failure of gonad formation (Fig. 3A; Jarglarz and Howard, 1994; Warrior, 1994). In these embryos, there was severe disruption of idr expression (Fig. 3A), as none of the pole cells in stage 14-16 embryos expressed idr. Failed expression was not caused by loss of general transcription, as zygotic transcription from the vasa gene is discernible in the dispersed pole cells (Van Doren et al., 1998; our unpublished result).

Clearly, the presence of the mesoderm is needed to induce idr expression in pole cells. In order to determine the source of the mesodermal cue, we next analyzed idr expression in the absence of the mesodermal cells that make up the gonads. The origin and development of the somatic components of the gonads have been described (Brookman et al., 1992; Boyle and DiNardo, 1995; Boyle et al., 1997; Broihier et al., 1998; Moore et al., 1998b; Riechmann et al., 1998). The SGPs are specified from the dorsolateral mesoderm within PS 10-12 at stage 11. It has been reported that in tin, zfh-1 double-mutants no dorsolateral mesoderm is formed, which results in loss of SGPs (Broihier et al., 1998; Moore et al., 1998b). In these embryos, pole cells pass through the midgut epithelium, but subsequently they are dispersed around the midgut (Broihier et al., 1998). We found that idr expression was drastically reduced in tin, zfh-1 double-mutants (Fig. 3F). This result shows the requirement of the dorsolateral mesoderm for idr expression in pole cells.

We next asked whether the specification of the dorsolateral mesoderm as SGPs is needed to induce idr expression in pole cells. To examine this, we used the abd-A and iab-4 mutations. abd-A function is required in the mesodermal cells for the specification of SGPs (Brookman et al., 1992; Cumberledge et al., 1992; Boyle and DiNardo, 1995; Boyle et al., 1997; Broihier et al., 1998). In abd-A mutant embryos, pole cells pass through the midgut wall and are normally associated with the dorsolateral mesoderm (Broihier et al., 1998). However, they do not coalesce with the pole cells to form the gonads due to their failure to be specified as SGPs. Consequently, pole cells are released from the mesoderm and scattered throughout the embryo (Boyle and DiNardo, 1995; Broihier et al., 1998). In these embryos, the dispersed pole cells expressed idr

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<th>Transformant lines</th>
<th>Heat treatment</th>
<th>Percentages of adult females with agametic ovaries*</th>
<th>idr mRNA amounts‡</th>
<th>Percentages of embryos developed to adults (n)§</th>
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*Female, the indicated lines, which developed at 25°C or heat-treated at 29°C during the period from 5±1 hour AEL (stage 10-11) to eclosion were dissected to examine their ovaries. The number of females with unilateral and bilateral agametic ovaries were scored. n, the number of females examined.

‡Poly(A)+ RNA was extracted from embryos of the indicated lines, which were cultured at 25°C or heat-treated at 29°C during the period from 5±1 hour AEL (stage 10-11) to eclosion. The amounts of idr mRNA were normalized to those of rp49 mRNA and are presented relative to the w control embryos cultured at 25°C (see Materials and Methods for details). UD, undetectable; the amount of idr mRNA is less than 5% of that of w control embryos.

§The number of embryos from the indicated lines that developed to adults at 25°C or at 29°C during the period from 5±1 hour AEL (stage 10-11) to eclosion were scored. n, the number of embryos examined.
Induction of indora expression in pole cells during stages 14-16 (Fig. 3G). Furthermore, we found that a regulatory mutation in the abd-A locus, iab-4 also had no deleterious effect on idr expression (data not shown). Thus, the specification of the dorsolateral mesoderm as SGPs is dispensable for idr expression.

**DISCUSSION**

We identified a novel transcript, idr, which is expressed only in pole cells within the gonads. We provide evidence showing that the expression of idr is required for germ-line development. Reduction of idr mRNA by expression of antisense idr resulted in the failure of pole cells to produce functional germ line in females. Moreover, our results show that the heat induction of antisense idr expression during embryonic stage 10-15 enhances the female sterility, suggesting an important role of idr expression during these stages in germ-line development. Lastly, we have found that idr expression in pole cells depends on the presence of the dorsolateral mesoderm. These observations suggest that the mesodermal cells induce idr expression in pole cells, which is required for their development into functional germ cells.

**Fig. 2.** idr mRNA is expressed in pole cells within the embryonic gonads. (A) Expression of idr mRNA in wild-type embryo at stage 16. Arrow indicates the gonad. Signal (purple) was detected in pole cells within the gonad by in situ hybridization probed with idr cDNA at stage 14 (B), stage 16 (C), and stage 17 (D). Arrowheads point to pole cells. Note that pole cells within the gonad express idr mRNA. Occasionally signal was detected in a dotted pattern in the cytoplasm of pole cells. Staining in the tracheal system (indicated by small arrows in D) is nonspecific. (E) idr mRNA is detected in pole cells incorporated within the gonad but not in a pole cell which is outside the gonad. The embryo was double-stained by whole-mount in situ hybridization probed with idr cDNA (purple) and immunoperoxidase method with an antibody against Vasa, a marker protein specific to pole cells (brown). Arrowheads indicate double-stained pole cells in the gonad. An arrow marks a pole cell outside the gonad. Scale bar, 20 μm. In all panels, anterior is to the left.

**Fig. 3.** Expression of idr mRNA in pole cells of a sna, twi double-mutant, wun mutant, tin, zfh-1 double-mutant and abd-A mutant embryos. Embryos were double-stained by whole mount in situ hybridization probed with idr cDNA (purple) and by the immunoperoxidase method with an antibody against Vasa (brown). (A) sna, twi double-mutant embryo at stage 15. Note that pole cells are scattered in the haemocoel. idr mRNA is hardly detectable in pole cells (arrowheads). (B-E) wun mutant embryos at stage 15 (B) and 16 (C-E). idr mRNA is hardly detectable in almost all pole cells dispersed throughout the embryos (arrowheads). In contrast, idr mRNA is detected in a small fraction of pole cells incorporated within the gonads (arrows in D). The gonads were identified by morphological features: dorsolateral distinctive cell clusters located in fifth abdominal segment. White arrowhead in D shows the tracheal branch in the sixth abdominal segment. Arrows in E indicate somatic gonadal cells ensheathing pole cells. (F) tin, zfh-1 double mutant embryo at stage 15. idr expression is drastically reduced in pole cells (arrows) scattered on the surface of the dorsolateral portion of the midgut (m). (G) abd-A mutant embryo at stage 15. idr mRNA is detectable in pole cells scattered in the haemocoel (arrows). 'm' indicates the posterior portion of the midgut. Scale bar, 20 μm. In all panels, anterior is to the left.
We have shown that the reduction of idr mRNA to an undetectable level in embryos leads to female sterility without any deleterious effect on the viability. In these embryos, pole cell formation and their migration to the embryonic gonads appear to be unaffected (data not shown). These observations are compatible with the finding that idr expression is initiated after the completion of pole cell migration, and is detectable predominantly in germ line. A simple explanation for the female sterility is that pole-cell differentiation within the gonads is impaired by the reduction of idr function. In this study, we could not observe any defects in male fertility (data not shown). However, this observation is based on the analysis of embryos with reduced idr function caused by antisense idr expression. Thus, it remains possible that null mutations in idr locus may reveal additional functions in male sterility.

Our results demonstrate that the idr expression in pole cells is induced by the specialized mesoderm. The disruption of the dorsolateral mesoderm by the tin, zfh-1 double mutation drastically reduces idr expression in pole cells. The dorsolateral mesoderm within PS10-12 and their derivatives, SGPs are already associated with pole cells at stage 10-11, and remain in contact with pole cells until they coalesce as the gonads at stage 14 (Boyle et al., 1997; Broihier et al., 1998; Moore et al., 1998b). Therefore, we propose that the dorsolateral mesoderm produces a cue for idr expression and their association with pole cells is needed to induce idr expression in those cells. Alternatively, it is possible that the dorsolateral mesoderm within PS 4-9 and PS 13, which develop into fat body (Moore et al., 1998b; Riechmann et al., 1998), produce a diffusible cue acting on pole cells to induce idr expression. Our observations favor the former model. In wild-type embryos, the idr expression is detected only in pole cells that are associated with SGPs within the gonads, while those cells outside the gonads never express idr. A similar situation is observed in wann mutants. In these embryos, only a small fraction of pole cells which successfully colonized the gonads properly express idr, while the pole cells dispersed throughout embryos are unable to express it, even though they are present in fat body immediately in the vicinity of the gonads.

We have shown that idr expression is independent of abd-A function. This suggests that the specification of the dorsolateral mesoderm as SGPs is dispensable for idr expression, since previous studies suggest that abd-A function is required for SGPs specification (Brookman et al., 1992; Boyle and DiNardo, 1995; Boyle et al., 1997; Moore et al., 1998b). It has been reported that even in the absence of abd-A function, pole cells normally migrate from the mid gut into the dorsolateral mesoderm during stage 11 (Broihier et al., 1998; Moore et al., 1998b). The pole cells maintain this association until mid stage 12, and afterwards they disperse. Thus, initial association of pole cells with the dorsolateral mesoderm occurs in an abd-A-independent manner. This suggests that the association of pole cells with the dorsolateral mesoderm during stage 11-12 is sufficient to induce idr expression in pole cells.

It is unclear what defines the stage at which idr expression is initiated. Pole cells are already in contact with the dorsolateral mesoderm at stage 11 (Brookman et al., 1992; Boyle and DiNardo, 1995), but idr expression is undetectable until gonad coalescence at stage 14. It is unlikely that the coalescence itself defines the onset of idr expression, because pole cells normally initiated idr expression at stage 14 without being ensheathed by SGPs in abd-A mutants (Fig. 3G). Based on the recent finding that gene expression, which normally occurs in pole cells at stage 13-14, is repressed autonomously by Nanos until these stages (Kobayashi et al., 1996; Asaoka et al., 1998), we suggest that there may be a requirement for a similar repression mechanism to enable pole cells to properly express idr at the later stage.

Interactions between germ-line progenitors and the surrounding somatic cells might be a general phenomenon in animal development. For example, in the mouse embryo, the migrating primordial germ cells (PGCs) expressing c-kit receptor move along a track of mesodermal cells expressing a membrane-bound form of the Steel ligand (Matsui et al., 1990). The ligand acts as a growth factor on primordial germ cells and also a mediator of cell-cell adhesion (Matsui et al., 1990; Flanagan et al., 1991; Matsui et al., 1991). In Drosophila, the somatic signals acting on pole cells are not well understood. Recent genetic screens have identified several genes required in somatic tissues, rather than in pole cells, for correct migration of pole cells (Zhang et al., 1996a,b; Broihier et al., 1998; Moore et al., 1998a). These genes may ultimately affect the gene expression in pole cells. Interactions between pole cells and somatic cells can also be seen during sex determination of germ line. Staab et al. (1996) have reported that somatic masculinizing signals act on pole cells to induce male germ-line marker 1 (mgl1) in stage 13 embryos. idr does not act in the pathway regulating sex determination as well as pole cell migration, because idr expression initiates after the completion of pole cell migration and is independent of sex (data not shown). Instead, we favor the idea that idr is required for the differentiation of pole cells within the gonad. Isolation of mutations in the idr locus, as well as the identification of the signaling molecules required for its expression, will be informative in elucidating the role of the soma and germ line interaction in Drosophila.

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Induction of indora expression in pole cells 1029


