Translating Available Food Into the Number of Eggs Laid by Drosophila melanogaster

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ABSTRACT

In Drosophila and other insects egg production is related to the nutrients available. Somehow the nutritional status of the environment is translated into hormonal signals that can be “read” by each individual egg chamber, influencing the decision to either develop into an egg or die. We have shown that BR-C is a control gene during oogenesis and that the differential expression of BR-C isoforms plays a key role in controlling whether the fate of the egg chamber is to develop or undergo apoptosis.

One such hormone, 20-hydroxyecdysone (20E), induces resorption of nurse cells that are degraded by apoptosis in early vitellogenic egg chambers (Soller et al. 1999). Normally, apoptosis is not induced in nurse cells until late in oogenesis as part of the program that causes the nurse cells to degenerate once they have fulfilled their function (Foley and Cooley 1998). However, in flies that have 20E at physiological concentrations injected into their abdomen, apoptosis is observed much earlier, at stage 9 of oogenesis (Soller et al. 1999). Simultaneous application of a juvenile hormone analog (JHA) suppresses apoptosis of the stage 9 egg chamber (Soller et al. 1999). Thus as well as nutrition regulating egg production, the balance between ecdysone and juvenile hormone (JH) in the fly also affects this process and is therefore likely to be crucial in controlling the genetic response of the ovarian cells to nutritional signals.

Ecdysone functions through the ecdysone/ultraspiracle nuclear receptor complex. The complex directly regulates early ecdysone response genes, such as the Broad-Complex (BR-C), E74, and E75. The BR-C encodes a family of zinc-finger transcription factors (DiBello et al. 1991). Apoptosis in many tissues and glands in insects is induced by 20E particularly during morphogenesis and is regulated by ecdysone response genes (Buszczak and Segraves 2000). The BR-C isoforms required for transcription of the cell death genes reaper (White et al. 1996) and hid (Abbott and Lengyel 1991) in Drosophila salivary glands (Jiang et al. 2000).

In this article, we investigate the choices between vitellogenesis and apoptosis in relation to the nutritional environment during oogenesis and the role of the BR-C in this decision. Under conditions of nutritional shortage, such as when flies are grown only on sugar, some oocytes are degenerated by premature apoptosis at stage 8 or 9 of oogenesis. We propose that at stage 8/9 there is a selection between the expression of the apoptosis
genes and the yp genes needed for vitellogenesis to proceed, i.e., a choice between the development of an egg and apoptosis of an egg chamber. We show that these developmental choices are controlled by different expression patterns of BR-C isoforms. We demonstrate that the nutritional environment of the female affects BR-C expression. As a result, the cells in each individual egg chamber become committed either to apoptosis or to progress through normal development. By executing this decision, the flies regulate the number of eggs produced in relation to food.

MATERIALS AND METHODS

Drosophila maintenance: Flies were maintained on standard yeast, maize meal, sugar, and agar medium at 25°C. The wild-type strain, Oregon-R, was used throughout. Three-day-old flies were transferred from a standard diet to one with sugar (starved, 1% agar medium, which contains 5% sucrose and 0.005% of a 10% nipagin solution in 95% ethanol) or one with yeast (fed, 2 g baker's yeast on ~50 ml 1% agar medium, which contains 2.5% corn flour, 5% sucrose, 1.75% hypophos- lized yeast, and 0.005% of a 10% nipagin solution in 95% ethanol). After 3 days on sugar or yeast, flies were dissected (starved, S3; fed, F3), transferred to sugar for 1 day after 3 days on yeast (F3S1), or topically treated with JHA methoprene (ZR515, Zocon) and maintained on sugar and water for 1 day (F3F1S1), injected with 20E (Sigma, St. Louis), and maintained on yeast for 1 day (F3F1E1). We used BR-C transgenic flies, which are TNQ^Q-Z1, Z2, Z3, and Z4 (kindly provided by C. Bayer; Bayer et al. 1997). The flies were maintained at 25°C for 3 days with yeast and underwent heat shock at 39°C for 30 min and were maintained at 25°C for 6 hr.

Injection of 20E and application of JHA: 20E was dissolved in insect Ringer's solution (130 mM NaCl, 4.7 mM KCl, 1.9 mM CaCl_2) and 50 nl was injected at a concentration of 2 µg/ml (Soller et al. 1997). The concentration of 20E was determined according to Bownes (1989). With a hemolymph volume of ~1 µl/female (Soller et al. 1997), 100 pg 20E/ female leads to a concentration of 2 × 10^-7 M. Methoprene was applied topically to the ventral abdomen in 100 nl acetone. Methoprene diluted 1:100 in acetone corresponds to a concentration of ~1 µg/100 nl. Controls were undertaken injecting Ringer's only and treating flies with acetone.

Hoechst staining and in situ hybridization: Hoechst staining has been described by Soller et al. (1999) with some modifications. Ovaries were fixed in 4% formaldehyde in PBS. After fixation, the ovaries were stained in 1 µg/ml Hoechst and the samples were observed using fluorescence filters. The hybridization probes, BR-C core, Z1, Z2, Z3, and Z4 were labeled with digoxigenin (DIG) as described by the supplier (Boehringer Mannheim Biochemicals).

The protocol is based on the procedure previously described (Tautz and Pfeifle 1989) and modified as follows. The ovaries were dissected in Ringer's solution and fixed for 20 min in 4% formaldehyde in PBS. After rinsing the tissue in PBT (0.1% Tween-20 in 1× PBS), it was treated for 10 min in methanol/0.5 M EGTA, pH 8 (9:1). The ovaries can then be stored in methanol at ~20°C for several months. The stored ovaries were rehydrated in PBT. The prehybridization was carried out for 1 hr at 45°C in DNA hybrid buffer (50% deionized formamide, 5× SSC, 100 µg/ml sonicated salmon sperm DNA, 50 µg/ml heparin, 0.1% Tween 20). The ovaries were hybrid- ized overnight at 45°C in DNA hybrid containing digoxigenin-labeled probe (DIG-DNA labeling and detection kit, Boehringer Mannheim, Indianapolis). For detection a 1:1000 dilution of anti-DIG-alkaline phosphatase (AP)-conjugated Ab was used. The staining reaction was performed in 100 mM Tris pH 9.5, 50 mM MgCl, 10 mM NaCl, 0.2% Tween 20, 8 mM levamisole, 4.5 µl/ml 4-nitro blue tetrazolium chloride, and 3.5 µl/ml X-phosphate (Boehringer Mannheim). Anti-DIG-AP conjugate was preabsorbed with postfixd milk-type (Oregon-R) ovaries at 4°C overnight. The ovaries were mounted in a mixture of PBS/glycerol (1:4) for microscopy. Egg chambers were staged according to size and morphology (King 1970).

RNA extraction and RT-PCR: The levels of yp's and BR-C transcripts in ovaries were detected by reverse transcriptase (RT)-PCR as described previously (Hodgetts et al. 1995). The details of total RNA extraction, reverse transcription reactions, and the PCR reaction are described in Tzolovsky et al. (1999). The expression levels were quantified by National Institutes of Health image (http://rsb.info.nih.gov/nih-image/download.html).

RESULTS

Egg-chamber progression differs between fed and starved flies: Drosophila ovaries develop differently in the fed flies maintained on yeast and in the starved flies maintained on sugar. The fed (F3) flies had more egg chambers than the starved (S3) flies (Figure 1A). In particular, the number of chambers at stages 8 and 9 were significantly different (Figure 1A). This development was also observed in the ovaries of flies that were transferred to a starvation regime after being fed with yeast for 3 days (Figure 1B). In these flies, the number of egg chambers at stages 8 and 9 decreased as the period of starvation was extended (Figure 1B).

A reduction in the number of egg chambers at stages 8 and 9 is also induced by injection of 20E and this reduction is inhibited by application of a JHA (Soller et al. 1999). As shown in Figure 1C, if flies were fed for 3 days and then starved for 1 day with JHA, the number of stage 8 and stage 9 egg chambers increased. There was no significant difference in the number of stage 8 egg chambers between control and JHA-treated flies. However, at stage 9 there was a significant rescue by JHA treatment (Figure 1C).

There were large differences in the number of egg chambers at stages 8, 9, and 10 when control (F3) and 20E-injected (F3EF1) flies were compared (Figure 1D). The reduction in stage 8 and 9 egg chambers was greatly increased in 20E-injected flies (Figure 1D).

The reduction in egg chambers at stages 8 and 9 is caused by apoptosis: Nuclear condensation and nuclear fragmentation were observed in the nurse cells following Hoechst staining (Figure 2A), consistent with the reduction in the number of stage 8–10 egg chambers in starved and 20E-injected flies being the result of apoptosis. The proportion of egg chambers undergoing apoptosis at stages 8 and 9 was increased in starved and 20E-injected flies (Figure 2B). In contrast, the proportion of egg chambers undergoing apoptosis was decreased in the JHA-treated flies (Figure 2B).

These results indicate that 20E and JHA had opposite
**Figure 1.**—Changes in the distribution and appearance of egg chambers during starvation. (A–D) The number of egg chambers at each stage of oogenesis. The bars represent the mean number ±SD in the flies at each stage under various conditions. n = 12 flies. (A) Comparison between fed flies (F3: with yeast for 3 days) and starved flies (S3: without yeast for 3 days). (B) Comparison of fed flies (F3) and flies with various periods of starvation (starved flies: S3 and F3S1, F3S2, F3S3; starved for 1, 2, and 3 days, respectively, after maintaining with yeast for 3 days). (C) The effect of the application of JHA (1 µg applied). Flies were maintained on yeast for 3 days and treated with JHA and flies were maintained without yeast (starved, F3JHS1). (D) The effect of the injection of 20E (100 pg injected). Flies were maintained on yeast for 3 days, injected with 20E, and retained on yeast (F3EF1). (*) Significant differences within a 5% level.

**BR-C Controls Drosophila Oogenesis**

Effects on the progression of oogenesis in fed (F3) and starved (S3, F3S1~3) flies. Furthermore, the difference in the numbers of normal egg chambers under specific nutritional or hormonal conditions was caused by apoptosis. Apoptosis was induced by 20E and inhibited by JHA.

**BR-C expression is altered in ovaries undergoing apoptosis at stages 8 and 9:** One of the ecdysone-responsive genes, **BR-C**, plays an important role in egg-chamber development (Deng and Bownes 1997). We therefore determined the spatial and temporal expression of **BR-C** to examine if the expression pattern was altered with changes in nutrition or hormone level.

Expression of **BR-C** was first detected in all follicle cells at stage 5 of oogenesis and was stronger than that at stage 6 (Figure 3A, F, and H). Strong **BR-C** expression in the starved flies (S3 and F3S1) was first detected in follicle cells at stage 6 (Figure 3B, C, and I), and **BR-C** expression at stage 5 either was not detected or was very faint (Figure 3G). However, when JHA was applied to the abdomen of starved flies, **BR-C** expression was induced in the follicle cells at stage 5 and to a higher level than in cells at stage 6 (Figure 3E). Thus in starved flies, JHA treatment induced a **BR-C** expression pattern that was similar to that of fed flies. In contrast, when 20E was injected into the abdomen of fed flies, strong **BR-C** expression was induced in follicle cells at stage 6 (Figure 3D), and the **BR-C** expression pattern in 20E-injected flies was similar to that of starved flies. Thus the timing of expression of **BR-C** in follicle cells is altered according to the nutritional conditions and in response to hormones.

**BR-C transcripts encoding different isoforms show different expression patterns:** **BR-C** has four isoforms, Z1, Z2, Z3, and Z4 (DiBello et al. 1991). The overall distribution of **BR-C** mRNA was detected by a core **BR-C** probe (this detects a common region in all splice variants). In addition, specific probes were used to detect the RNA that would encode each isoform separately. Figure 4A shows the proportion of **BR-C** expression in the egg chambers at stages 5 and 6 in flies under various experimental conditions. The proportion of Z1 expression at stage 5 is almost the same as the overall **BR-C** expression (core); however, the proportion of the expression at stage 6 was substantially lower. Z2 and Z3 were expressed at stage 5 in similar proportions to each other in fed flies. Z2 expression at stage 6 was not induced in fed flies and was reduced by JHA treatment. Z3 expression at stage 6 was constant under all experimental conditions. Z4 expression at stages 5 and 6 was very weak; this gene was expressed at lower levels than the other isoforms.
Stage 8 in oogenesis is an important stage in development. YP synthesis starts at this stage in the follicle cells of egg chamber (Wilson 1982; Bownes 1986; Soller et al. 1999), and uptake of yolk from the hemolymph begins. Starvation and 20E injection induces apoptosis at this stage (Figure 2). We therefore assume that at this point there should be a key developmental decision in oogenesis. BR-C and Z1 expression at stage 8 was almost the same under all experimental conditions and Z4 was not detected at this stage (Figure 4B). The proportion of the Z2 and Z3 expression increased in the egg chambers of starved and fed, 20E-treated flies, namely under conditions that induce apoptosis (Figure 4B). These results suggest a key role for Z2 and Z3 in regulating the apoptosis switch.

**BR-C Z3 activates Z2 expression in fed condition**: Previously, it has been shown that BR-C isoforms interact with each other to control gene expression and tissue differentiation in response to ecdysone during Drosophila metamorphosis (Mugat et al. 2000). In third instar larvae, Z3 antagonizes Z2 repression and primes enhancer activation (Mugat et al. 2000). As the hormone titer rises in late third instar, the ligand-bound ecdysone receptor no longer silences gene expression and, together with the Z1, Z3, and Z4 BR-C isoforms, activates transcription (Mugat et al. 2000).

Since it seemed possible that BR-C isoforms suppress or enhance the expression of other isoforms, we investigated the effect of the BR-C isoform overexpression on the expression of other isoforms. In fed flies transgenic for Z3, not only Z3 but also Z2 expression was increased in oogenesis. BR-C Z2 and Z3 expression at stage 8 was activated by heat shock of Z3 transgenic flies under adequate nutritional conditions, but was not activated without heat shock in control flies. The level of Z2 expression in heat-shocked Z3 transgenic flies increased compared with non-heat-shocked Z3 transgenic flies (Figure 5B). These results indicate that Z3 overexpression induces Z2 expression in fed flies but not in starved flies.

**BR-C regulates YP gene expression**: Two key choices are made at stage 8 of oogenesis: the start of YP synthesis or the induction of apoptosis. BR-C Z2 and Z3 had a role in the induction of apoptosis at stages 8 and 9.
(Table 1). So we next examined the role of the $BR-C$ in regulating $yp1$ expression.

$yp1$ gene expression was inhibited by starvation in F3S1 flies, but not in S3 flies (Figure 6A) and JHA treatment rescued $yp1$ expression in starved flies. 20E did not affect $yp1$ expression (Figure 6A). $yp2$ and $yp3$ expression were suppressed in both starved flies, F3S1 and S3, and also rescued by JHA treatment. 20E injection into the fed flies resulted in suppression of $yp2$ and $yp3$ expression.

$BR-C$ Z1 and Z2 overexpression also affect $yp$ expression in fed and starved conditions (Figure 6B). Z1 and Z3 overexpression suppressed $yp1$, $yp2$, and $yp3$ expression in both fed and starved flies, but Z2 and Z4 did not affect $yp$ expression under any conditions.

DISCUSSION

A poor nutritional environment induces apoptosis in the egg chamber at stages 8 and 9: In Drosophila oogenesis, the nutritional status of the female’s environment affects YP synthesis and egg production (Schwartz et al. 1985; Bownes and Blair 1986; Bownes et al. 1988; Bownes 1989; Bownes and Reid 1990; Søndergaard et al. 1995). We have shown that stage 8 and 9 egg chambers in starved flies are degenerated by apoptosis, leading to a reduced number of egg chambers beyond stage 8. Hence fewer eggs are laid (Figures 1 and 2). During nutritional shortage, the nutrients must be used to develop a smaller number of normal eggs. To achieve this, it is essential to select which egg chambers will develop to mature eggs and which will die.

When the ovary accumulates stage 14 oocytes in the sexually mature virgin female, the production of new eggs is prevented by resorption of egg chambers at stage 9, mediated by the high level of 20E in the hemolymph (Bownes 1989; Harshman et al. 1999; Soller et al. 1999). It seems likely that the resorption of stage 9 egg chambers under nutritional stress may well occur by a similar mechanism, since reduced food induces increased ecdysone concentration in the flies (Bownes 1989), and 20E in turn induces apoptosis of the egg chambers at stages 8 and 9. While ecdysone induces a reduction in the number of egg chambers, JH acts to suppress this reduction (Soller et al. 1999 and this article). Females homozygous for $ap^{bw}$ produce low levels of JH but are fertile although vitellogenesis is subject to a delay (Altaratz et al. 1991). The $ap^{bw}$ ovaries are capable of ultimately producing greater than wild-type amounts of ecdysteroids (Richard et al. 1998). It is possible that the ecdysone concentration in the flies under starvation is suppressed when JHA is applied to the abdomen of the flies and thus JHA suppresses the ecdysone-induced apoptosis in egg chambers at stage 8/9. It is likely that both ecdysone and JH levels are affected by the nutritional status of Drosophila. Ecdysone and JH concentration could be modulated by synthesis of hormones or by inducing metabolic pathways.
that degrade the hormones. Using this mechanism a female can relate the number of eggs produced to the food available.

**BR-C is the key gene for selecting between apoptosis and development:** BR-C isoforms and BR-C mRNA are expressed differentially in a number of tissues during metamorphosis (Huet et al. 1993; Emery et al. 1994) and control subsequent cell differentiation. Late in Drosophila oogenesis, BR-C regulates the dorsal-ventral signaling pathway (Deng and Bownes 1997) and leads to prolonged endoreplication of follicle cell DNA and to additional amplification of specific genes (Tzolovsky et al. 1999). The BR-C expression pattern differs between starved and fed flies earlier in oogenesis at stages 5, 6, and 8 (Figures 3 and 4). BR-C expression in oogenesis is first detected in the egg chamber at stage 5 under normal nutritional conditions (Figure 3), but under nutritional shortage it is detected later, at stage 6 (Figure 3). This shows that nutritional shortage is detected in the follicle cells of the egg chamber in previtellogenic stages. During metamorphosis of holometabolous insects, the developmental program is switched from “larval” to “pupal” during the final larval instar, a switch that is referred to as pupal commitment (Riddiford 1985). To execute apoptosis, tissues and glands must first become committed to apoptosis and then execute the decision and die. We propose that the altered timing of BR-C expression affects this commitment stage in the egg chamber. By stage 8, when the egg chamber must develop or die, the different isoforms of BR-C, specifically Z2 and Z3, differ between starved and fed flies. Z2 and Z3 are not expressed in egg chambers at stage

**Figure 4.**—The expression of BR-C isoforms at stage 5, 6, and 8 in various conditions. The graphs show the percentage of egg chambers expressing each isoform of BR-C at stages 5 and 6 (A) and at stage 8 (B). The expression signals were detected by in situ hybridization for using each probe, core (common region of Z1–Z4), Z1, Z2, Z3, and Z4. The flies called F3AS1 were maintained on yeast for 3 days, treated with 100 nl acetone, and maintained 1 day without yeast as a control for F3JHS1. The flies of F3EF1 were injected with 50 nl insect Ringer’s and maintained on yeast for 1 day as a control for F3EF1. The values were calculated as percentages (mean of egg chambers, expressed/mean of egg chambers in each ovary x 100; n = 12 flies). (*) Significant differences compared to the control (F3 vs F3S1, F3 vs F3EF1, and F3S1 vs F3JHS1) within 5% level.

**Figure 5.**—BR-C Z3 overexpression induces BR-C Z2 expression at stage 8. (A) The result of RT-PCR in the ovaries of transgenic flies hsZ2 and hsZ3. rp 49 is control. (B) The percentage of Z2 and Z3 expression at stage 8 in hsZ2 and hsZ3 flies. The expression signals were detected by in situ hybridization. y-Axis indicates the percentage of the egg chambers in which Z2 or Z3 is expressed (n = 12 flies). (*) Significant differences within 5% level.
### TABLE 1
Percentage of egg chambers showing apoptosis at stages 8 and 9 in the ovary of flies misexpressing BR-C isoforms

<table>
<thead>
<tr>
<th></th>
<th>Fed (F3)</th>
<th>Starved (F3S1)</th>
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<tbody>
<tr>
<td></td>
<td>+hs Stage 8</td>
<td>−hs Stage 8</td>
</tr>
<tr>
<td>Control OrR</td>
<td>2.2</td>
<td>1.7</td>
</tr>
<tr>
<td>BR-C Z1</td>
<td>3.8</td>
<td>1.8</td>
</tr>
<tr>
<td>Z2</td>
<td>2.2</td>
<td>2.2</td>
</tr>
<tr>
<td>Z3</td>
<td>4.6</td>
<td>2.3</td>
</tr>
<tr>
<td>Z4</td>
<td>2.8</td>
<td>3.4</td>
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</tbody>
</table>

The percentage of apoptosis at stages 8 and 9 in ovaries of heat-shock transgenic flies for BR-C isoforms with and without heat shock is shown. Heat-shock flies underwent heat shock at 39°C for 30 min and were maintained at 25°C for 6 hr with yeast prior to dissection. Nuclear condensation and fragmentation were detected by Hoechst staining and observed under a fluorescent microscope. The fed flies (F3) were maintained 3 days with yeast, and the starved flies (F3S1) were maintained for 1 day of starvation after maintaining them for 3 days with yeast. We used BR-C transgenic flies that are TN-Q1-Q2-Z1, Z2, Z3, and Z4 (construction is described in Bayer et al. 1997). An asterisk indicates significant differences within 5% (n = 12 flies). −hs, without heat shock; +hs, with heat shock.

8 under normal nutritional conditions, but they are expressed in egg chambers at stage 8 under nutritional shortage or if 20E is injected into fed flies (Figure 4B). This means that Z2 and Z3 are expressed under apoptotic conditions. Furthermore, Z2 and Z3 overexpression induces apoptosis at stages 8 and 9 (Table 1).

In the ovary of starved flies, Z2 and Z3 expression is independent, but in fed flies Z3 overexpression induces Z2 expression (Figure 5). 20E injection overrides the nutritional signals and induces apoptosis in the egg chambers at stages 8 and 9 in fed flies (Figure 2) probably by activating Z3 that in turn induces Z2 expression. Z3 therefore acts as key transcript for inducing apoptosis. On the basis of these results, BR-C acts as a selector gene for progressing oogenesis or inducing apoptosis at stages 8 and 9.

The nutritional environment affects oogenesis by regulating BR-C expression: Figure 7 shows a model for the control of oogenesis in relation to the nutritional status. Each Drosophila egg chamber selects between two fates...

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**Figure 6.—Nutritional conditions and BR-C overexpression affect YP gene expression in the ovary.** (A) Starvation, JHA application, and 20E injection affect YP gene expression in the ovaries. The graph indicates the expression level using relative value with ±SD (measured by National Institutes of Health image). (B) The BR-C isoform overexpression affects YP gene expression in the ovary. An asterisk indicates significant differences within 5%.
during development. These fates are triggered by the hormonal conditions that are in turn regulated by the nutritional environment. The higher ecdysone concentration in starved flies induces BR-C Z2 and Z3 expression at stage 8, and these genes activate the pathway of apoptosis in the egg chambers at stages 8 and 9. In fed flies, the ecdysone concentration is lower and hence Z2 and Z3 are not expressed in the egg chambers at stage 8. The JH analog, methoprene, suppresses apoptosis at stages 8 and 9, suggesting that JH acts as a suppressor of apoptosis and an inducer of normal development. The normal developmental pathway requires that yp genes are expressed by stage 8; however, Z3 suppresses yp expression (Figure 7). Thus yp expression is suppressed by nutritional shortage and by 20E injection in fed flies. The expression of Z3 that is induced by nutritional shortage or 20E injection suppresses yp expression at stage 8 in the follicle cells and hence prevents normal development from continuing.

In summary, we have shown that the environmental status of the fly is sensed by each individual egg chamber and that some develop and some die in proportion to the food available. The nutritional status of the environment controls the developmental fate of the egg chamber in Drosophila by adjusting the hormonal balance in the fly. We have established that the key gene in follicle cells that is controlled by the hormone balance is BR-C and that the BR-C Z3 isoform particularly is important for egg-chamber development in Drosophila in relation to the nutrition available.

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Figure 7.—The role of BR-C in oogenesis progression. Complete nutrition induces normal development of the mature egg during oogenesis. In this case, only BR-C Z1 is expressed in the egg chamber at stage 8. But if there are problems during oogenesis, or if 20E is injected, the apoptosis pathway is activated and apoptosis is executed at stages 8 and 9. Nutritional shortage induces apoptosis commitment and the progression of apoptosis. In this case, not only Z1 but also Z2 and Z3 are expressed in the egg chamber at stage 8. This apoptosis is suppressed by application of JHA onto the abdomen.


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