Turning food into eggs: insights from nutritional biology and developmental physiology of *Drosophila*

Christen K Mirth, André Nogueira Alves and Matthew DW Piper

Nutrition plays a central role in fecundity, regulating the onset of reproductive maturity, egg production, and the survival and health of offspring from insects to humans. Although decades of research have worked to uncover how nutrition mediates these effects, it has proven difficult to disentangle the relative role of nutrients as the raw material for egg and offspring development versus their role in stimulating endocrine cascades necessary to drive development. This has been further complicated by the fact that both nutrients and the signalling cascades they regulate interact in complex ways to control fecundity. Separating the two effects becomes important when trying to understand how fecundity is regulated, and in devising strategies to offset the negative effects of nutrition on reproductive health. In this review, we use the extensive literature on egg development in the fruit fly *Drosophila melanogaster* to explore how the nutrients from food provide the building blocks and stimulate signalling cascades necessary for making an egg.

Address
School of Biological Sciences, Monash University, Melbourne, Victoria 3800, Australia

Corresponding author: Mirth, Christen K (christen.mirth@monash.edu)

Current Opinion in Insect Science 2018, 31:49–57
This review comes from a themed issue on Development and regulation
Edited by Elisabet Marchal and Dolores Piulachs

https://doi.org/10.1016/j.cois.2018.08.006
2214-5745/© 2018 Elsevier Inc. All rights reserved.

Introduction
The nutrients obtained from food affect a wide range of life history and health related traits, from regulating the pace of development, to modulating aging, fecundity, and the propensity for disease [1]. The primary goal of nutritional biology is to understand how diet impacts these traits [2,3]. It is apparent that not all nutrients are equal in their effects, and that both their quantities and qualities matter. For example, numerous studies in insects have highlighted that while lifespan is maximized on high carbohydrate, low protein diets, lifetime fecundity requires higher concentrations of protein and lower concentrations of carbohydrates to obtain maximum values [4–8]. Precisely how nutrients are able to exert these effects is a topic of active research.

Perhaps one of the best understood traits relating to how food affects life history is the development of eggs (otherwise known as oogenesis), which relies heavily on the maternal nutritional environment in a diverse range of animals ranging from flies to humans [9]. Restricting animals of dietary protein, carbohydrates, or lipids induces a characteristic, well described range of phenotypes. Despite substantial study, the extent to which these phenotypes result from a lack of raw materials required for egg development or from the nutrients’ effects on the production of developmental hormones necessary to drive reproduction remains poorly understood. Here, we make use of the extensive literature on egg development in the fruit fly *Drosophila melanogaster* in an attempt to disentangle the effects of nutrients themselves from the effects on the signalling pathways they regulate, providing our perspectives on how to best approach this problem in future studies. It is our hope that by separating these effects, we can generate deeper knowledge with regards to how and when nutrients matter for life history traits, and provide the foundation for interventions aiming to offset the effects of poor nutrition.

Effects of nutrition on egg production?
*Drosophila* are generally thought to live on rotting fruit and vegetable matter, where they participate in the decay process along with a succession of invertebrates and microbes [10]. In the laboratory, flies can be reared under a range of nutritional conditions that loosely resemble those in nature, but at a minimum require sugar and yeast for maximal growth and reproduction [11,12]. Yeast provides the majority of essential ingredients, which, depending on the strain and growth conditions, is comprised of carbohydrates (30–50%), protein (30–50%), lipids (5–10%), nucleic acids (5–8%), as well as B-group vitamins, and trace elements (metal ions, phosphorous and sulfur) (5%) [13]. A complete account of the minimal nutritional requirements of *Drosophila* has been developed over ~60 y of work, which has culminated in several completely defined synthetic media for lab culture [14].

Chemically defined diets have been shown to be adequate, but not optimal, to support fly development, and completely sufficient for adults [14–17]. In particular, they have proven to be particularly useful for studying...
adult egg laying and lifespan, offering a high degree of precision with which to dissect the effects of individual nutrients on egg production. Omission of either carbohydrates, protein (essential amino acids), sterols, B-group vitamins, or metal ions results in reduced lifetime egg production, indicating each is required either to stimulate oogenesis and/or provide the biochemical building blocks that are required for eggs to pass quality control [15,16] (Figure 1). Interestingly, the dynamics with which each of these nutrients affects egg laying is different: omission of essential amino acids completely abolishes egg laying within 72 h while omission of each of the other nutrients reduces egg laying over the course of ~1 to 2 weeks [15]. This could indicate a difference in how each nutrient is sourced for egg production (i.e. the proportion available from the diet versus body stores) as well as the extent to which each contributes to the signalling that stimulates and promotes egg laying. Understanding how each

Figure 1

Turning nutrition into eggs. Nutrients serve as both building blocks of egg production and as stimulants of, and interactors with, hormone signalling to regulate egg development. The three macronutrients, carbohydrates, protein, and lipids, and micronutrients, such as vitamins and metal ions, provide the major source of energy and the raw materials for egg production in the *Drosophila melanogaster* ovary. Each ovary is comprised of ~22 ovarioles. The linear progression of egg development within each ovariole is illustrated at the bottom of the figure. Stage 1 occurs in a structure called the gerarium, which contains the germline and somatic stem cells (left tip of ovariole). Stage 14 (rightmost portion of the ovariole) is a mature egg. Each of the three macronutrients have their own effect on egg growth, since they are each required for progression past different stages of development (solid black arrows). Carbohydrates and lipids are mostly recruited at stage 10, whereas protein is principally required for yolk accumulation which starts at stage 8. Metal ions are responsible for the correct development of stage 6–7 egg chambers (grey dashed arrow) and pyridoxine (a vitamin) affects development in the pre-vitellogenic phase (grey dashed arrow) [16]. However, nutrients also act to trigger hormones such as Ecdysone and Insulin (black dashed arrows), which exert their own effects on different stages of egg development (solid black arrows) as well as stimulating the production of other relevant hormones (e.g. Juvenile Hormone).
dietary nutrient affects the processes involved in egg development is key to disentangling these effects.

**How do you make an egg?**

In insects, ovaries are made up of strings of ovarioles, which are essentially assembly lines working in parallel for egg production. In well fed *Drosophila* females, each ovary contains an average of 22 ovarioles [18–21], although this number varies between genotypes [19,22–26]. Ovarioles are composed of germ cells as well as somatic cells necessary to provide structure and to support egg production. The number of ovarioles in the ovary limits the maximum egg production rate of the female [19,21,27].

In the anterior-most tip of the ovariole, termed the germarium, sit 2–3 germ line stem cells [28]. Germ line stem cells are specified during embryogenesis and will give rise to all the eggs the female produces [28]. These stem cells divide to produce cystoblast daughter cells, which will go on to divide 4 more times without completely closing off their cell membranes to result in 16 cells that share cytoplasmic connections [29,30]. While in the germarium, one of these cells becomes destined to be the egg cell, or oocyte, while the remaining 15 cells become nurse cells [29,30]. As the cystoblast moves out of the germarium, it becomes encapsulated in follicle cells produced by the paired follicle stem cells [29–31]. After encapsulation, the combination of somatic and germ cells is called an egg chamber.

Egg chamber development was initially characterised in the 1950s and 60s by King and colleagues, and divided into fourteen stages [31,32] (Figure 1). Stages are defined based on a number of cellular characteristics of the oocyte and nurse cells including the position of the egg chamber along the ovariole, the position and size of the oocyte relative to the nurse cells, the morphology of oocyte and nurse cell nuclei, and the migration of subsets of follicle cells [29–33].

The first six stages of egg chamber development involve the continued differentiation of the oocyte from the nurse cells, the growth of the nurse cells and oocyte, and the division of follicle cells to accommodate the growing egg chamber [29,30]. Follicle cell division ceases at stage 7 [31]. At stage 8, which marks the onset of vitellogenesis, the oocyte begins to take up yolk proteins. As the oocyte continues to take up yolk, it expands compressing the nurse cells [31]. The follicle cells begin to produce the membranes of the egg, the vitelline membrane and chorion, from stage 9 onwards. At stage 9, a specialised set of follicle cells called the border cells migrate through the nurse cells and position themselves above the oocyte nucleus at stage 10 [31]. These cells begin to form the micropyle, the sperm entry point. From stage 11, the nurse cells begin to degenerate and the dorsal appendages of the egg form, reaching their final length at stage 14 [31]. At the final stage, stage 14, when the oocyte has reached its mature size and egg membrane formation is complete, the follicle cells die [31]. Although some egg chambers share characteristics of more than one stage (Cummins and King, 1969), this is rare. These reliably identifiable stages of egg chamber development have provided excellent fodder for decades of work on the genetic mechanisms regulating egg production.

The progression through the 14 stages of egg chamber development requires coordinated interaction between the germline and somatic cells in the ovary, as well as from other organs such as the brain, the corpora allata, and the fat body [34–36]. The nurse cells, as their name implies, provide an important role in loading the developing oocyte with maternally derived mRNA and proteins necessary for the earliest stages of embryogenesis [28]. The follicle cells serve as important signalling centres that define the polarity of the egg, produce its external membranes, and secrete the steroid hormone ecdysone, a hormone necessary for egg development [35,37–39]. Hormones like sex peptide from the seminal fluid, insulin-like peptides from the brain, and juvenile hormone (JH) from the corpora allata also drive the rates of egg chamber development [36,40–44]. Finally, the fat body and follicle cells are responsible for producing the yolk proteins that are loaded into the egg during the vitellogenic stages [34,38]. Thus, egg development involves input from multiple organs throughout the body, and the function of many of these organs depends on the nutritional environment.

**The effects of nutrition on egg development**

Several studies have found that nutrition, specifically yeast starvation, affects many stages of egg chamber development to impact the number of eggs an adult female can lay. Starving female flies of yeast decreases the rate of division in the germline and somatic cells, thereby reducing rates of egg chamber growth [45]. Further, it increases the rate of cell death specifically at the transition between stages 2a and 2b and at stage 8 before vitellogenesis begins [45–49]. Yeast starvation interrupts the transport of ribonucleoprotein complexes, necessary for the early stages of embryonic patterning, into previtellogenic stage oocytes [50,51]. Finally, yeast deprivation also impedes the production of yolk protein by the fat body and follicle cells [46,52–54]. Despite extensive study, whether each of these phenotypes are a result of the direct effects of nutrition on egg development or the effects of nutrition on the synthesis of hormones required for egg development has yet to be clarified.

Many of the effects of yeast starvation on egg development have been ascribed to protein. In the first instance, this could be because protein is required to provide the
raw materials for egg production. In the absence of protein, the amino acids necessary for yolk protein synthesis and the growth of cells in the egg chamber simply are not available.

Alternatively, the effects of protein withdrawal could be due to protein-dependent signalling. Knocking down amino acid transporters specifically in the fat body reduces the rate of egg laying by controlling germline stem cell division and ovulation [55]. Rather than being a result of direct effects, amino acids act through signalling pathways that regulate cell homeostasis. Amino acid insufficiency in the fat body is thought to result in uncharged tRNAs that are sensed by the protein kinase of GCN-2 [55,56]. Activation of GCN-2 in the fat body acts via some unknown signal to affect ovarian germline stem cell division [55]. To alter ovulation, amino acids in the fat body act through the Target of Rapamycin (TOR) pathway [55]. Thus, amino acids induce signalling pathways in the fat body that remotely control processes of egg development unrelated to vitellogenesis.

Furthermore, if amino acids were only required to provide the building blocks for yolk protein, blocking amino acid transport into the fat body cells would be expected to stop yolk protein synthesis and induce apoptosis in previtellogenic egg chambers. Knocking down individual amino acid transporters in the fat body failed to induce apoptosis of egg chambers [55]. This suggests that either multiple amino acid transporters need to be removed to affect yolk protein synthesis in the fat body or that yolk protein synthesis is regulated by nutrition-dependent hormone synthesis.

Although amino acids clearly play an important role, yeast is also an important source of other nutrients like carbohydrates and lipids. Both carbohydrate and lipid concentrations have been shown to be important for regulating egg laying rate [15], however the signalling pathways through which they act are not as well described.

Both lipids and carbohydrates are loaded into the developing oocyte at the end of egg development [57,58*,59**]. These nutrients fuel the early stages of embryogenesis, and depletion of either results in low egg viability [58*,59**60]. Several studies have focussed on lipid and carbohydrate sources as building blocks for developing eggs, however less is known about how variation in these nutrients affects egg production rate.

Lipids begin to accumulate in the oocyte at stage 10 [58*] and are necessary for egg development and to support early embryogenesis. Feeding females on lipid-depleted food activates the transcription factor SREBP in the egg chamber, which in turn increases transcription of the lipid transporter LpR2 [58*]. Reducing SREBP function results in oocytes that fail to accumulate lipids [58*]. Further, animals that are defective in lipid accumulation lay significantly fewer eggs [58*].

The oocyte also begins to accumulate glycogen at stages 10 [57,59**]. Both saccharides, presumably from the diet, and gluconeogenesis from amino acids are important sources for glycogen accumulation in the late stages of egg development [59**]. This increase in glycogen accumulation is driven by downregulation of mitochondrial function [59**,61,62]. If mitochondrial function remains high in the later stages of egg development, glycogen and triglycerides concentrations in the mature egg remain low and the resulting eggs fail to hatch [59**].

The timing and extent of nutrient limitation affects different processes relating to egg development. Oocytes of females fed from eclosion on food containing low quantities of yeast undergo vitellogenesis, but fail to accumulate lipids at stage 10 [58*]—a phenotype not observed in ovaries from females completely deprived of yeast from eclosion [45]. Similarly, females transferred from a yeast rich diet to a yeast deficient diet partially block ovulation, leading to a retention of mature eggs within their ovaries [45]. These examples illustrate how either reducing nutrient concentration or interrupting the supply of essential nutrients at later ages generates differences in egg development and stalling phenotypes.

The differences in the effects of starvation with time occur because immediately after eclosion nutrients are necessary to complete reproductive maturation, which is marked by the initiation of yolk protein synthesis in the fat body and follicle cells of the ovary and the appearance of vitellogenic egg chambers [53,63]. After yolk protein synthesis has been initiated at reproductive maturation, starvation reduces but does not inhibit yolk protein synthesis and affects the rates of egg development [53].

Taken together, nutrients such as protein, carbohydrates, and lipids provide important building blocks for proper egg maturation. Future work exploring the role of other nutrients, including sterols, carbohydrates, minerals, and vitamins, will provide further insights into how the raw materials from the diet contribute to egg production.

How does diet modify physiology to change egg production?

In addition to contributing the building blocks for egg development, nutrition affects the production of at least three hormones important for oogenesis: the insulin-like peptides, ecdysone, and JH. By regulating each other’s activity, these hormones generate a complex network of interactions across organs and cell types to fine tune rates of egg production with the nutritional environment.

The concentration of nutrients circulating in the hemolymph is sensed by the fat body [64–67]. In larvae, at
least four peptide hormones are secreted by the fat body in response to amino acid concentrations [68–70]. It is not yet clear if these same peptides act to signal amino acid availability in adults. Concentrations of dietary lipids and sugars are also sensed by the fat body [66,71,72], and in adults result in the secretion of Unpaired 2 [66]. In all cases, the peptide hormones secreted by the fat body act to regulate the secretion of insulin-like peptides.

D. melanogaster has seven insulin-like peptides (dILP1-7) [73] and a relaxin like peptide (dILP8) [74] that are expressed in different tissues. Nutrition is thought to act primarily through dILP2, 3, and 5, which are expressed in two clusters of 7 cells in the brain in both larvae and adults [73,75]. Under conditions of high protein, these peptides are secreted into the hemolymph and act systemically to control growth, development, and metabolism [1].

dILP signalling has both direct and indirect effects on ovary development [41,42]. Ablating the dILP-producing cells in the brain results in reduced cell division in the germ line and somatic cells of the ovary [41], and arrest of vitellogenic stage egg chambers [40,41]. dILP1-7 are thought to bind to and activate a single Insulin Receptor (InR) in target tissues [73]. Germline cysts that lack InR show significant reductions in the proliferation of the germline stem cells, the rate of egg chamber development, and the egg chambers arrest and degenerate at the vitellogenic stage [41,76], similar to egg chambers from starved females.

The indirect effects of insulin signalling arise from its activity in other organs. Insulin signalling in the corpora allata induces JH synthesis in these glands [44,77**]. JH itself is necessary for initiating yolk protein synthesis by the fat body and ovarian follicle cells [63,78]. Once initiated, the fat body continues to produce yolk protein independent of JH, but JH remains necessary for the uptake of yolk protein in the early stages of oocyte vitellogenesis [43]. In yeast-deprived females, JH levels drop resulting in a reduction of egg production [27,44]. Whether these effects result from reduced vitellogenesis alone or to additional effects of JH on egg development has not been explored.

JH synthesis is further regulated by the steroid hormone ecdysone. In well fed females, ecdysone titres begin to rise in the adult female one to two days after eclosion [79,80]. This rise in ecdysone stimulates the corpora allata to synthesize JH via an additional peptide hormone, eclosion triggering hormone [81*]. Together, the rise in ecdysone, followed by the increase in JH titres in the hemolymph, is responsible for completing reproductive maturation and initiating the synthesis of yolk proteins by the fat body and follicle cells [43,53,81*]. Ecdysone synthesis itself is known to be sensitive to nutrition and insulin signalling [80,82,83,84*,85]. In adult females starved immediately after eclosion, ecdysone titres fail to rise [85] causing delays in the onset of reproductive maturation and yolk protein synthesis. If reproductively mature adult females are starved, ecdysone concentration in the hemolymph plummets [84*,85].

In addition to stimulating JH synthesis, ecdysone exerts its own effects on the fat body and ovary. Perhaps the best studied effect is the role of ecdysone in regulating the production of yolk proteins synthesized by the fat body [43,46,53,63]. Isolated female abdomens produce very little yolk protein [63]. Ecdysone administration can partially restore the rates of yolk protein synthesis [63]. Further, injecting starved females with ecdysone partially rescues yolk protein synthesis [43,46,53].

Ecdysone signalling also regulates pre-vitellogenic stages of egg chamber development. During egg development, ecdysone is required for germline stem cell maintenance [86], germline proliferation [87*], follicle stem cell maintenance [87*], somatic cell proliferation and growth [37,88], cyst encapsulation [89,90], vitellogenesis [37,43,46,53,63], and border cell migration [87*]. Not all functions of ecdysone in egg development overlap with the phenotypes observed during nutritional deprivation, which most likely reflects the fact that while starvation reduces ecdysone synthesis, it does not eliminate it [84*,85]. Thus, ecdysone plays both nutrition-dependent and nutrition-independent roles.

The ability of ecdysone to function in nutrition-dependent and nutrition-independent roles might result from signalling interactions within the ecdysone signalling pathway itself or with other pathways. Several processes during egg development are regulated by specific transcription factors downstream of the ecdysone signalling cascade. For example, ecdysone response gene e75 regulates the progression beyond early stages of vitellogenesis [37], while another ecdysone response gene, e78, mediates germline stem cell niche formation [86]. In addition, the effects of ecdysone depend on the physiological context. In reproductively mature females, injection of ecdysone induces apoptosis of vitellogenic egg chambers [43] whereas treatment with both ecdysone and JH protects the egg chamber from apoptosis and allows yolk protein uptake into the oocyte [43]. Taken together, this demonstrates that the ovary responds to nutrition by integrating a variety of cues including the availability of nutrients and the concentrations of circulating hormones to regulate rates of egg production.

How can we disentangle the effects of nutrients and hormones in building eggs?
Making an egg requires a complex balance of nutritional input and hormone signalling. Nutrients are required
both to provide the raw material for energy storage and support growth and development of the egg chamber and to tune the levels of circulating hormones that drive egg development (Figure 1). Further, withdrawal of nutrients and interfering with these hormone cascades often produce similar types of phenotypes. For example, amino acid starvation or eliminating insulin, JH, or ecdysone signalling all interrupt vitellogenesis leading to apoptosis of previtellogenic egg chambers [35,41,43,45,63]. Understanding to which degree each factor contributes independently remains a complex problem.

A potential approach to solving this problem involves manipulating each of these factors in pairwise combinations, to identify those that contribute the most to each phenotype. This approach has been applied to the developing ovary, which also requires nutrition, insulin, and ecdysone signalling to continue its development [21]. Inducing ecdysone signalling in the ovaries of larvae starved of yeast partially rescues the delays in ovary development induced by starvation, but was not capable of rescuing development to fully fed levels [21]. This suggests that either the nutrients from the food, or insulin signalling, or both, are required for ovary development to be restored to normal levels. Using a similar approach to manipulate each of the nutrients and hormones involved in egg development in combination would provide significant insight into how they interact to modulate rates of egg development. While this approach is promising, simultaneously manipulating multiple nutrients and multiple pathways could prove intractable due to the numbers of genotypes and conditions required.

An alternative method for parsing out the effects of nutrients and hormones on egg development could employ introducing quantitative variation (rather than simply all or none) in nutrient levels and examining the effects on hormone synthesis and egg development. Several frameworks already exist for varying the nutritional components of the diet. Nutritional geometry, which is an analytical framework that captures the effects of a range of varying concentrations of key macronutrients like protein, carbohydrate, and fat, has been employed to study a wide range of life history traits including egg production rate [2–4,91]. In its most precise form, nutritional geometry can be conducted using chemically defined diets allowing multidimensional variation in individual amino acids, carbohydrates, minerals, and vitamins [6,14,15]. Combining quantitative nutritional approaches with measurements of hormone concentrations and their signalling pathways [92,93] and assessments of egg development would provide a powerful set of tools for uncoupling the effects of diet and hormones on egg production rate. Although powerful, these experiments also suffer from the same problem of being large, costly, and time consuming.

A third approach to separating the direct effects of nutrients from the hormones they regulate makes use of naturally-occurring genetic variation in sensitivity to nutrition. Several genome reference panels have been constructed for D. melanogaster [94,95], which are collections of isogenised lines whose genomes have been fully-sequenced. They are useful for exploring genetic variation in traits, and mapping this phenotypic variation to allelic variants. Exploring how egg production responds to specific nutrient dilutions across genotypes has the potential to result in the identification of candidate genes involved in egg production rate. Further, they would allow us to compare how the synthesis of hormones and activity of signalling pathways vary across genotypes in response to nutrition. These initial characterizations would provide a background genetic structure that may further parse out the direct effects of nutrition versus the effects of nutrition via hormone signalling on egg production rate.

**Conclusions**

Decades of research on egg production in *Drosophila* have provided valuable insight into the genetic cascades and nutritional inputs that regulate this process. Despite these insights, we still do not understand to what extent each nutrient is required to function as a building block for egg development versus acting as a regulator of the hormones that control the developmental progression of the egg. This is complicated by the fact that many of the phenotypes induced by starvation seem to be regulated by insulin, JH, and ecdysone. We argue that approaches that integrate the contribution of quantitative variation in individual nutrients with those from hormone signalling will prove vital to disentangle their effects. Because many other life history traits are regulated by complex dynamics between nutrients and the hormone pathways they regulate, we expect that the insights obtained from this work will lead to a deeper understanding of how nutrition modulates other traits in flies and in other organisms.

**Conflict of interest statement**

Nothing declared.

**Acknowledgements**

We thank the editors Dolors Piulachs and Elisabeth Marchal for inviting us to write this review. This work was supported by an Australian Research Council Future Fellowship (FT170100259) to CKM, and an Australian Research Council Future Fellowship (FT150100237) to MDWP.

**References and recommended reading**

Papers of particular interest, published within the period of review, have been highlighted as

- of special interest
- of outstanding interest


59. At the end of egg chamber development, glycogen accumulates in the oocyte. Glycogen accumulation correlates with increased saccharides, glycogenolysis/glucoseogenesis intermediates, and also increased amino acid input into the tricarboxylic acid cycle. Sieberet al. (2016) found that this increase in glycogen accumulation is driven by a reduction in mitochondrial activity. Finally, the authors identified two gene products, FoxO and GSK3, responsible for the reduction in specific subunits of the electron transport chain in late stages of egg chamber development.


70. Koyama T, Mirth CK: Growth blocking peptides as nutrition-sensitive signals for insulin secretion and body size regulation. PLOS Biol 2016, 14:e1002952.


This work explores the regulation of reproductive diapause in Drosophila melanogaster. In this species, reproductive diapause results in arrest of developing egg chambers at the pre-vitellogenic stages. Diapause can
be induced by a combination of short day length, low temperature, and starvation. Ojima et al. (2018) show that diapause results from reduced insulin signalling in the corpora allata, which in turn reduces JH synthesis causing arrest of egg chamber development.


Ecdysis triggering hormone (ETH) has long been known to induce adult eclosion in insects. In this paper, Meiselman et al. uncover a new role for ETH in regulating egg production. ETH acts on the corpora allata to regulate rates of JH synthesis. In turn, the authors find that ETH synthesis is downstream of ecdysone signalling. These results provide new insight into the endocrine regulation of egg production, with ecdysone acting to stimulate ETH production, which in turn controls JH synthesis.


When females are deprived of yeast, they arrest egg development and develop a strong appetite for protein. Because starvation is known to reduce ecdysone production in the follicle cells, Santos and Ribeiro (2017) tested the hypothesis that this reduction in ecdysone instructed the brain to increase protein appetite. They found that although reducing ecdysone titers lowered food intake overall, it did not increase the appetite for yeast.


Ables and coauthors employ a genetic mosaic screen to identify ecdysone response genes that function at various stages of egg development. They identify five classes of phenotypes regulated by ecdysone response genes: germline stem cell maintenance, germline proliferation, growth, and survival, follicle stem cell maintenance, somatic cell proliferation, growth, and survival, and cyst encapsulation. Further, they find that while some ecdysone response genes regulate specific subsets of these processes, others control multiple aspects of egg development.


