Annual Review of Entomology

Adaptive Plasticity of Insect Eggs in Response to Environmental Challenges

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Annu. Rev. Entomol. 2023. 68:451–69

First published as a Review in Advance on October 20, 2022

The *Annual Review of Entomology* is online at ento.annualreviews.org

https://doi.org/10.1146/annurev-ento-120120-100746

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Keywords

phenotypic plasticity, development, microbe, defense, plant, environment

Abstract

Insect eggs are exposed to a plethora of abiotic and biotic threats. Their survival depends on both an innate developmental program and genetically determined protective traits provided by the parents. In addition, there is increasing evidence that (*a*) parents adjust the egg phenotype to the actual needs, (*b*) eggs themselves respond to environmental challenges, and (*c*) egg-associated microbes actively shape the egg phenotype. This review focuses on the phenotypic plasticity of insect eggs and their capability to adjust themselves to their environment. We outline the ways in which the interaction between egg and environment is two-way, with the environment shaping the egg phenotype but also with insect eggs affecting their environment. Specifically, insect eggs affect plant defenses, host biology (in the case of parasitoid eggs), and insect oviposition behavior. We aim to emphasize that the insect egg, although it is a sessile life stage, actively responds to and interacts with its environment.

1. INTRODUCTION

Most insect species are oviparous and expose their eggs to many environmental threats (142). Despite numerous abiotic and biotic environmental factors that endanger eggs, oviparity is obviously a successful strategy. This success is due to numerous egg-protective measures provided by the insect parents. While parental investment in insect egg protection is well known, little attention has been paid to the protective activities of the developing zygote itself.

In this article, we provide an overview of parental and zygotic measures that protect eggs from extreme abiotic conditions, predation, and parasitization. Furthermore, we highlight studies showing that oviposition-associated symbiotic microorganisms not only serve later developmental stages but also contribute to egg protection. In addition to egg-protective traits, which are fixed in a developmental program, we also consider trait changes in response to current challenges. Insect eggs not only respond to their environment, but also influence it. Oviposition by herbivorous insects on plants induces changes in the plant transcriptome and metabolome, which in turn modulate herbivory. Egg laying by parasitoids in herbivorous larvae alters host behavior and physiology and may even affect hyperparasitoids. In this article, we draw attention to a tiny insect developmental stage that both responds to its environment and exerts significant effects of its own on different trophic levels.

2. INSECT EGGS AND THEIR ADAPTIVE PLASTICITY IN RESPONSE TO ABIOTIC CONDITIONS

2.1. Thermal Plasticity

Insects have evolved a wide range of molecular and physiological traits to survive extreme temperatures and to adjust themselves to changing thermal conditions (15, 79). In addition, mobile insect stages can escape from detrimental temperatures through behavioral adaptations, e.g., hiding from sunlight, searching for diapause sites with suitable temperatures, or warming up by exercising their flight muscles (28, 83). In contrast, eggs as immobile stages cannot leave sites with unfavorable temperatures. However, they are well protected from extreme temperatures by their parents and by their own thermoprotective activity (**Figure 1*a***).

Insect mothers contribute to thermoprotection of their eggs by several means. The maternal timing of egg deposition and the choice of oviposition sites significantly contribute to egg survival (e.g., 105). Furthermore, covering eggs with plant tissue, feces, or maternal secretions can protect overwintering offspring from detrimental temperatures (e.g., 49, 53). In addition to such protective measures, insect mothers endow the inside of their eggs with maternal messenger RNA, proteins, lipids, and carbohydrates, which provide the initial developmental basis for the embryo (38, 126, 150). In the very early embryonic stage, this maternal provisioning of the eggs contributes to thermoprotection via compounds such as heat shock proteins (75) or cryoprotective lipids (e.g., 150).

The temperatures experienced by the parents may significantly affect the egg phenotype and its survival. Phenotypic plasticity of egg size in response to temperature has long been known (e.g., 37). However, few studies have tested the adaptive significance of egg phenotypes, which are determined by temperatures experienced by the parents shortly before oviposition. Eggs have been found to survive best at the temperature experienced by their mother during the oviposition period (33). Additionally, the father's thermal experience also may influence which temperatures are optimal for the eggs' survival (138). The mechanisms of such intergenerational thermopriming are not yet fully understood, but epigenetic transcriptional regulation of thermosensitive genes has been predicted. Indeed, heat stress–induced epigenetic changes, which depend on the transcription

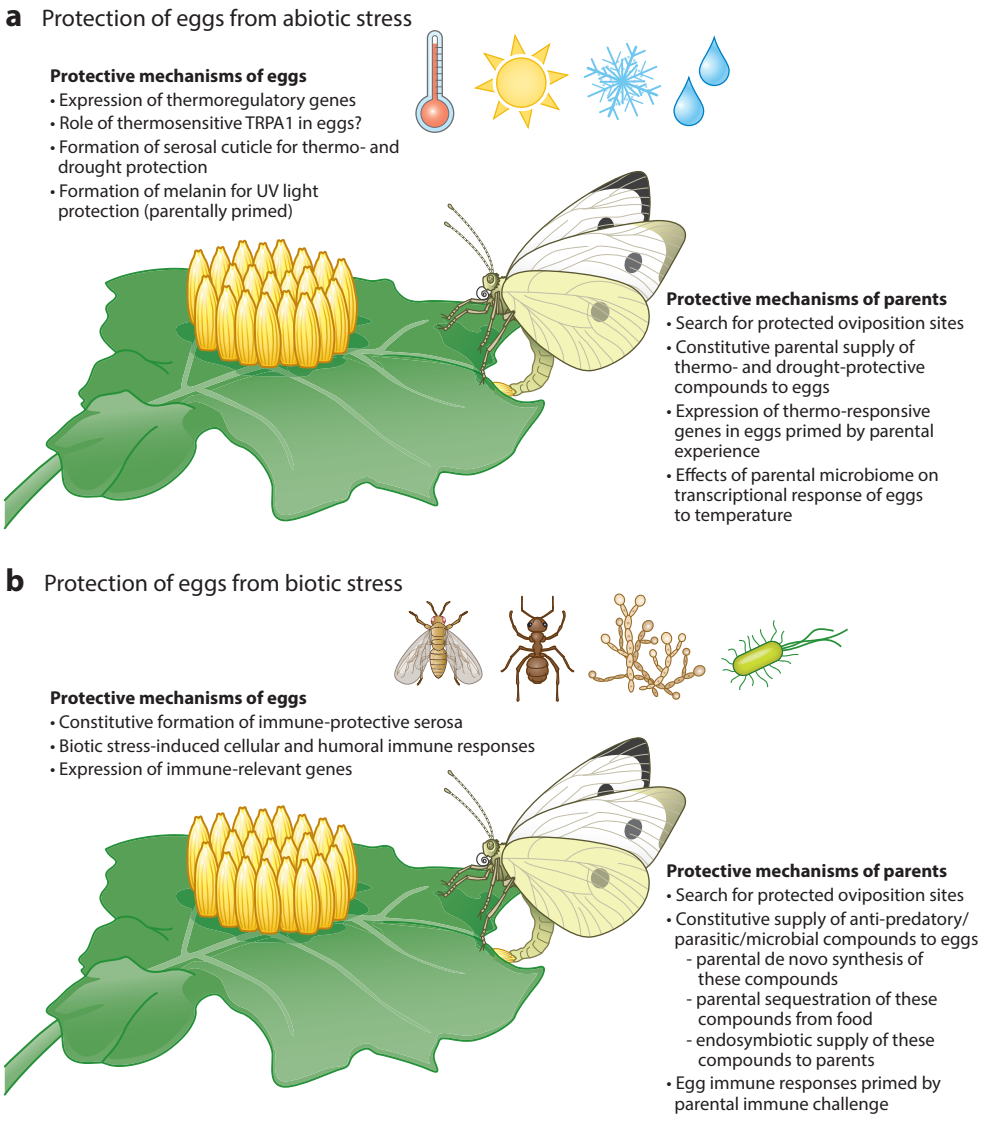

Figure 1

Insect eggs and how they cope with stress. (*a*) Constitutive and induced responses of eggs to abiotic stress and the effects of parental inputs. (*b*) Constitutive and induced responses of eggs to biotic stress and the effects of parental inputs. For references, see Sections 2 and 3.

factor ATF-2, have been shown to be transmittable from one *Drosophila melanogaster* generation to the next (119).

After oviposition, eggs initially make use of their energy-rich maternal provisions while they undergo the maternal-to-zygotic transition (137), optimizing their survival chances. An effective innate developmental program contributes to buffering thermal variation, as has been shown for *Drosophila* embryonic development (85). Insect eggs show higher survival rates at extreme

Maternal-to-zygotic transition: the time during which a zygote metabolizes maternally provided resources (e.g., mRNA, proteins) and the zygotic genome takes over the developmental control

cold (frost) if they themselves (not their parents) had previously been exposed to sublethal cold temperatures (e.g., 89, 96). To achieve such primed frost tolerance, the developing zygote needs to adjust its internal compounds or its egg shell to the anticipated imminent temperatures. Indeed, insect eggs express genes involved in the biosynthesis of cryoprotectants such as glycerol and sorbitol (e.g., 96). Furthermore, they can respond to cold by increasing the thickness of the middle serosa cuticle, which has been suggested to protect against inoculating ice formation (70). The serosa and its cuticle are formed by developing zygotes of almost all winged insect species; the exception is a single dipteran taxon, the Schizophora (57, 95). The developing embryo inside the egg is surrounded by yolk, which is enclosed by the membranous serosa. To the outside, the serosa is covered by the maternally provided vitelline membrane and the chorion, which forms the outermost layer of the egg.

Serosa: envelops the egg yolk and lines the inside of the egg shell

Transient receptor potential (TRP) channels: thermosensitive ion channels that can process thermal information via the nervous systems of insect larvae and adults

In addition to the parental and zygotic molecular and physiological activities, there is another player involved in thermotolerance of insect eggs: the parental microbiome. The composition of the microbiome deposited by *Drosophila* flies at oviposition sites depends on temperature. Interestingly, the transcriptional response of the offspring to temperature is dependent not only on the temperatures experienced by the parents, but also on the temperature-dependent parental microbiome composition (66). Thus, the parental microbiome affects the transcriptional response of the offspring to temperature.

To date, studies on the ability of insect eggs to adjust their phenotype to ambient temperatures have not really examined the associated question of how insect eggs sense temperature. While an increase in temperature of 10°C usually enhances the reaction speed of enzymes by a factor of two to three, thermosensitive proteins such as transient receptor potential (TRP) channels are expected to show an increase in activity by more than a factor of three when exposed to 10°C-higher temperatures (41, 82, 146). The parents can sense temperature via these proteins and use the information for intergenerational thermopriming of eggs. Several studies also have shown that thermosensitive *TRPA1* is expressed in eggs of moths (117), beetles (60), and flies (to a very small extent) (125). Induction of *TRPA1* in eggs may even be important for diapause induction in eggs of the next generation. Specifically, when *Bombyx mori* eggs are exposed to warmer temperatures, this exposure induces the release of diapause hormone (DH) later during the pupal stage, and the resulting females thus lay eggs that diapause (117). Regulation of egg diapause by maternal factors is well known to prepare the progeny for anticipated environmental conditions (e.g., 21). The peculiarity of the regulation of egg diapause in *B. mori* is that induction of *TRPA1* already occurs in the eggs, which then develop into females that lay diapausing eggs. To date, it is not known which signaling pathway is involved in this intergenerational regulation of egg diapause (117).

2.2. Egg Responses to Drought and UV Radiation

As with thermal protection of eggs, protection from desiccation may be significantly affected by the mother's oviposition behavior, both in the choice of oviposition sites and in egg clustering (e.g., 44). In addition, the mother may protect her eggs from desiccation by providing them with sufficient internal water, with high amounts of hydrocarbons in the egg shell (135), or by coating the eggs with materials such as hydrogels (e.g., 11, 32) (**Figure 1*a***).

While genotypic local adaptation to drought risks is known for mosquito populations, which coat their eggs with high amounts of hydrocarbons in dry regions (135), no such local adaptation to dry conditions was found in *Manduca sexta*. Instead, this moth oviposits on the lower side of leaves, where leaf transpiration may have a cooling effect and compensate for low air humidity (106).

To the best of our knowledge, evidence for phenotypic priming of eggs for improved drought resistance by maternal experience of low humidity is only available in arachnids, i.e., mites (72).

Nevertheless, we also expect priming of eggs by maternal drought experience to exist in insects because there is ample evidence that insect mothers can adjust the phenotype of their eggs to various environmental factors (e.g., 86) (**Figure 1**).

The deposited egg also can protect itself from desiccation (**Figure 1*a***); the serosa and its cuticle contribute significantly to egg drought resistance (e.g., 56, 136). The serosal contribution to drought resistance may even be enhanced by formation of dark eumelanin in the endochorion (29). Natural eumelanin is a very robust biopolymer, which is insoluble in water and even in organic solvents; its biosynthesis starts with the hydroxylation of the aromatic amino acid tyrosine, thereby forming dopa. Oxidative polymerization of dihydroxyindoles, which are derived from dopa and its derivatives, leads to eumelanin (140). The formation of serosal layers is part of the developmental program in many insect species, and eggs exposed to low humidity might benefit from accelerating this developmental process. However, whether the developmental kinetics are adjusted to the needs of the egg remains unknown. Furthermore, the question arises of whether insect eggs can adjust their physiological activity to their need for water, apart from developmentally programmed traits. Future studies need to unravel how an egg's internal water state is regulated. Active regulation may be achieved by, e.g., adjustment of egg shell conductance (145) or active uptake of water vapor (147). Passive regulation may be provided by an effective developmental program, by water provisioning by the mother, or by passive take-up of water via osmotic forces (47).

Exposure to UV light results in generation of reactive oxygen species (ROS), which have the potential to cause cellular damage (20). Pigmentation by melanin can protect the egg from damage by UV light. Melanin is highly efficient in adsorbing especially the short wavelengths of solar light via its numerous cyclic structures with conjugated electron pairs (29). While melanization of the egg shell is part of the innate developmental program in some insects, females of the stink bug *Podisus maculiventris* can adjust coloration of their eggs by melanin to the expected exposure to UV light (2). Even in the absence of melanin, the egg shell can provide protection from damage by UV light, as has been found for *M. sexta* eggs (107), which are colored greenish by yellow carotenoids and a blue biliprotein, insecticyanin (65). Carotenoids are very common egg pigments, and they also are expected to contribute to protection of eggs from UV radiation damage due to their ROS scavenging properties (120). A recent review by Guerra-Grenier (45) addresses further ecological functions of egg coloration in addition to protection from UV radiation, among them especially camouflage, mimicry, and various warning functions.

3. INSECT EGG DEFENSES AGAINST BIOTIC THREATS

3.1. Constitutive Defenses Provided by the Developing Egg or Its Parents

As sessile life stages, insect eggs are exposed to a high risk of parasitization and predation. They are a protein-rich food source not only for parasitoids and many predatory insects, but also for other arthropods like spiders and even for birds (81, 91, 98). Infection of eggs by pathogenic bacteria is uncommon but may occur, for example, via maternal transfer of bacteria to the oocyte or via attack by parasitoids. Infection of eggs by fungi, which can penetrate the egg shell with their hyphae, is more widespread than bacterial disease of eggs (42). Insect eggs are defended against these biotic risks by constitutive mechanisms, as well as by attack-induced defensive responses (**Figure 1*b***). In this section, we first address the constitutively available defenses.

Constitutive egg defenses are provided by parents in numerous ways. A recent review (30) describes the different mechanisms used by insect parents to protect their eggs from attack by egg parasitoids. Females of many insect species incorporate defensive compounds into their oocytes or into secretions deposited with the eggs (for an overview of low-molecular-weight organic compounds, see 13; for proteinaceous compounds, see, e.g., 23). These compounds are either produced

de novo or sequestered from food. In addition, symbiotic microbes harbored by the mother are known to produce defensive compounds contributing to egg protection (see Section 3.2). Insect fathers can contribute to protection of eggs from biotic threats by transferring defensive chemicals to females during mating. The females incorporate these compounds into the eggs or into secretions coating the eggs (26). Alternatively, some insects protect their eggs with sticky fecal covers or hairy tufts that impair access to the eggs or provide parental brood care by guarding, carrying, or grooming eggs (53).

To date, there is no clear evidence that insect parents adjust the provisioning of their eggs with defensive compounds to the actual risk of predation (99, 100). However, they may adjust their oviposition behavior to ambient egg predation risks and reduce egg predation by selection of oviposition sites with low predator abundance (109). The lack of evidence of phenotypic plasticity in parentally provided physical and chemical egg defenses against predators is contrasted by studies showing that immune-challenged insect parents can prime egg immune responses to pathogens and parasitoids (see Section 3.4).

Constitutive egg defenses against microbial infection are also produced by the eggs themselves even in the absence of biotic attack. These defenses include immune-relevant proteins and antimicrobial peptides. For example, freshly laid, nonattacked eggs of *M. sexta* constitutively express immune-related genes encoding a prophenoloxidase activating protease (*PAP I*), prophenoloxidase (*ProPO*), and several antimicrobial peptides (1, 42). Constitutive expression of immune-related genes encoding receptor proteins recognizing microbial attack, proteins of the Toll and IMD signaling pathways, and class B scavenger receptors in the serosa was demonstrated in eggs of *Tribolium confusum* beetles (57). These findings corroborate the idea that the serosa functions as an immune-competent barrier protecting the developing embryo (42, 57).

Constitutive production of defensive compounds of low molecular weight by the eggs is almost unknown. A notable exception is the release of nitric oxide radicals from eggs of the European beewolf *Philanthus triangulum* (124). Eggs of this species are laid into subterranean brood cells where the threat of microbial infection is high. Nitric oxide radicals emitted from beewolf eggs react with oxygen to form nitrogen dioxide radicals, which have antifungal activity when released in high amounts. The beewolf eggs are thought to be able to release relatively large amounts of nitric oxide radicals because of a specific modification of their nitric oxide synthase, an otherwise common enzyme regulating biochemical processes in almost all organisms. Thus, a modification of this enzyme might have led to this key evolutionary innovation in the European beewolf.

3.2. Constitutive Chemical Egg Defenses Provided by Microbial Symbionts

Symbiont-mediated protection is increasingly being recognized as a common source of constitutive egg defenses, especially among insect taxa that oviposit in environments where the eggs have a high risk of being exposed to pathogenic threats. Fungal and bacterial microbes extracellularly transmitted to the oviposition site or directly onto the eggs have been shown to protect eggs from infection by pathogens or predation (34, 103, 141). Symbiont-bearing secretions deposited on the eggs are especially known to provide subsequent developmental stages with a range of services (115). Beyond ensuring the faithful transmission of obligate symbionts (54, 68, 114), these secretions may nourish early instar larvae and nymphs after hatching (64). In this section, we focus on the egg-protective function of microbes released during oviposition.

Many ant, termite, and beetle species transport fungal spores to their oviposition sites. This transport serves to grow fungal gardens, which provide a nutritional resource for progeny (87). The fungal mycelia in termite nests can produce a variety of secondary compounds, including those exhibiting antimicrobial activities (e.g., 118). Provisioning and protection of offspring by

fungiculture is particularly well known for social insects such as termites but has also been found in some nonsocial insect clades. For example, the weevil *Eups chinensis* creates plant cradles for its offspring by leaf rolling. The cradles are inoculated with spores of *Penicillium herquei*. The female weevils release these spores from their mycangia, which are special structures adapted for transport of symbiotic fungi (74, 113, 141). The fungal mycelium encases the eggs within a few days, and hatching larvae feed on both the leaf roll and the fungal mycelium. In addition to the protective function of the cradle, *P. herquei* suppresses the growth of plant-associated bacteria and fungi, as well as pathogenic microbes (141). Biochemical elucidation of the fungus's inhibitory effects revealed that *P. herquei* produces (+)-scleroderolide, a broad-spectrum antimicrobial agent, which underpins the defensive role of the fungus (141).

Transmission of egg-protective bacterial symbionts directly onto the eggs is most common in species that oviposit in sites with high microbial abundance. For example, females of the house fly, *Musca domestica*, typically oviposit in animal manure, exposing their eggs to a rich nutritional resource but one that is also prone to microbial overgrowth (71, 151). Flies overcome this challenge by coating their eggs with a conserved bacterial consortium that suppresses growth of pathogenic fungi (71). The inhibitory effects of the members of this consortium are synergistic, conferring egg protection collectively, likely through a combination of competitive exclusion and chemical defense (71).

Oviposition in soil also exposes insect eggs to a high risk of microbial infection. Tenebrionid species of the taxon Lagriinae, which typically oviposit in soil, rely on *Burkholderia* symbionts to suppress egg infection by entomopathogenic fungi (35). *Lagria villosa* has been shown to harbor multiple strains of *Burkholderia* and transmit them through egg smearing (34). Several bioactive secondary metabolites have been identified from different symbiotic *Burkholderia* strains that could contribute to antimicrobial defense (24, 36, 90). These include the antifungal polyketide lagriamide, which is structurally similar to defensive polyketides characterized in marine tunicates (36).

Horizontal gene transfer is increasingly being recognized as a common mechanism by which microbes acquire and integrate genes coding for secondary metabolites and toxins, as demonstrated in microbial symbionts of aphids (92), psyllids (88), beetles (103), and sponges (143). Horizontal gene transfer to symbiotic *Pseudomonas* bacteria harbored by rove beetles (Staphylinidae) likely enables this symbiont to produce a toxic polyketide, pederin. This compound has only been detected in rove beetles of the genus *Paederus* and in marine sponges. Eggs and larvae are chemically defended by this *Pseudomonas*-supplemented toxin against predation (67, 103, 104). Both immature stages exhibit higher concentrations of pederin (67) than do other life stages, likely reflecting their vulnerability to predation. The biosynthetic gene cluster underlying toxin production in *Pseudomonas* appears to have been acquired following horizontal gene transfer (104). This finding is consistent with the sequence-based homology shared between pederin and diaphorin (88, 103), a toxin produced by distantly related symbionts in psyllids.

3.3. Induced Defensive Responses of Eggs to Pathogens and Parasitoids

During infections by pathogens and attack by parasitoids, eggs do not remain passive but express immune responses (42, 93), which are similar to those in larvae, pupae, and adults (e.g., 73). Infection of eggs by pathogens elicits immune responses in the extraembryonic tissue—especially the serosa—as well as in the embryo. Eggs respond to invading bodies such as pathogens with cellular responses resulting in phagocytosis and/or encapsulation of the intruder, as well as with humoral responses that fend off the invaders with antimicrobial peptides and/or by encasing them in a layer of melanin. Cellular immune responses are provided by embryonic hemocytes (139, 144).

Teratocytes: cells originating from the parasitoid egg serosa; they disperse inside a host when the parasitoid larva hatches from the parasitoid egg

The serosa plays a significant role in egg immune responses to pathogens in some species but not in all. The serosal membrane of *Tribolium castaneum* responds to immune challenges by expressing numerous immune-related genes (57, 58); however, the presence of a serosal membrane is no guarantee of infection-inducible production of antimicrobial agents by eggs. Eggs of the carrion beetle *Nicrophorus vespilloides*, which—like *Tribolium*—develop a serosa, do not produce antimicrobial peptides in response to bacterial infection (59). Freshly laid eggs of *Drosophila*, which do not form an extraembryonic serosal layer, respond only very moderately to bacterial infections (58, 129). However, at later stages, embryonic hemocytes in the *Drosophila* eggs were shown to successfully phagocytose *Escherichia coli*, whereas they were inactivated by a toxin released by the entomopathogenic bacterium *Photorhabdus* (139).

The battle between a host embryo and egg parasitoids is also mediated by toxins released by the parasitoid. The venom released by egg parasitoids such as *Trichogramma* spp. consists of proteases and phosphatases, which enhance the degradation of the host embryo and the digestion of the yolk (101). Egg-larval parasitoids, which complete their development in the host larva, must ensure that the host embryo remains alive. Thus, when they inject their egg into the host egg, parasitoids of this type release viruses in addition to venom. These viruses manipulate the host larval development (61). Several reviews have comprehensively addressed how parasitoids of insect eggs and other developmental stages affect the immune system, development, and even behavior of their hosts. To successfully attack a host and suppress its immune system, parasitoids use a sophisticated and varied arsenal of weapons, consisting of venom, viruses, virus-like particles, and compounds on their egg surface, as well as substances produced by teratocytes (e.g., 9, 22, 101, 130).

To counteract the weapons used by egg parasitoids, host eggs can strike back via immune responses. For example, eggs of the cerambycid beetle *Phoracantha recurva* respond to attack by the encyrtid wasp *Avetianella longoi* by cellular encapsulation and melanization of the parasitoid egg and larva (108), thereby significantly increasing their survival rates (77). In contrast, *M. sexta* eggs do not survive egg parasitism, although they show increased expression of several immune-relevant genes in response to parasitism. However, these responses result in significant reduction of parasitoid survival (1).

A lack of immune responses does not mean that the eggs have no chance to survive. Insects may compensate for the lack of efficient egg immune responses to parasitoids and pathogens with a short developmental time (59), oviposition at protected sites, constitutively expressed chemical defenses, microbial symbionts provided by the parents, or parental care (see Sections 3.1 and 3.2).

3.4. Priming Egg Defenses Against Pathogens and Parasitoids by a Parental Legacy

Adult insects that have experienced an immune challenge can prime the immune state of their offspring. Both females and males are known to contribute to such intergenerational immune priming. The primed offspring shows more efficient immune responses (25, 69, 111, 112, 132, 148), which are already detectable in the egg stage. The eggs laid by immune-challenged females show higher antimicrobial activity than do eggs from naive females. Such intergenerational immune priming of the egg stage against entomopathogens is known for several insect taxa, including social Hymenoptera (112), Coleoptera (69, 148), and Lepidoptera (132). Furthermore, when eggs resulting from mating between immune-challenged females and males are attacked by egg parasitoids, they respond by boosting transcription of several immune-relevant genes to a much greater extent than do eggs from unchallenged parents (133).

Several mechanisms are involved in generating a primed egg immune state. There is evidence that increased antimicrobial activity of primed eggs is due to transfer of high concentrations of

antimicrobial peptides from the immune-challenged mother into the eggs (e.g., 131). The finding that eggs resulting from mating between immune-challenged males and females show higher transcript levels of immune-related genes than do eggs resulting from unchallenged parents (e.g., 133) raises the question of how this effect is mediated. Given that fluorescence-labeled bacterial material orally taken up by the females in the larval stage was found in the ovaries and eggs, one possible explanation of this effect is that bacterial components induce enhanced expression of genes encoding antimicrobial peptides in the eggs (39, 69). Microbe-associated molecular patterns (MAMPs) on the surface of bacteria and even surface patterns of viruses have been found to bind in the hemolymph to vitellogenin, the precursor of egg yolk protein (134). Thus, vitellogenin can transfer MAMPs present in infected parents into the eggs (116). Genes encoding vitellogenin and thus providing yolk protein have formerly been thought to be predominantly expressed in females (17), but increasing evidence suggests that yolk proteins are also expressed in reproductive tissues of male insects and coat sperm (7, 80). However, it is not yet known whether yolk proteins detected in males contribute to the known paternally mediated intergenerational immune priming of the offspring (25, 111). Expression of immune-relevant genes in eggs resulting from immune-challenged females and/or males might also be determined by epigenetic marks set by the challenge. Indeed, Gegner et al. (40) found differential expression of genes involved in DNA methylation and histone (de)acetylation when comparing (*a*) microbially primed and nonprimed males and females, as well as (*b*) unchallenged offspring of primed and nonprimed parents. While maternal microRNAs are known to be transferred into insect eggs, where they take over regulatory functions (48, 121), their role in priming egg immunity has not yet been studied.

4. INSECT EGGS IN DYNAMIC INTERACTION WITH THEIR ENVIRONMENT

Above, we focus on how insect eggs adjust themselves to their environment. We present multiple types of evidence that eggs are capable of responding to abiotic and biotic threats (see Sections 1–3), but in turn, the eggs themselves also affect different trophic levels, as we discuss in this section (**Figure 2**).

4.1. Oviposition by Herbivores Affects the Plant

Eggs laid by herbivorous insects can elicit defensive plant responses directed against the eggs, such as elicitation of hypersensitive response (HR)-like leaf necrosis or neoplasm formation, which may cause egg desiccation or egg detachment from leaves. Furthermore, plants may respond to insect eggs by producing ovicidal compounds or by emitting plant volatiles that recruit egg parasitoids to find and kill the eggs (for reviews, see 10, 50, 51).

Recent studies on interactions between plants and insect eggs have focused on molecular and physiological mechanisms (e.g., 76, 122). Direct plant responses induced by eggs show conserved, common patterns in the different plant species tested and striking similarities to plant responses to phytopathogens. These responses include formation of HR-like traits; accumulation of ROS; increase in salicylic acid (SA) concentrations; enhanced transcription of SA-responsive genes, including pathogenesis-related (*PR*) defense genes; and accumulation of phenylpropanoid derivatives. Thus, egg-laden plants may defend themselves not only against the initial step of insect infestation, the egg deposition itself, but also against phytopathogens (e.g., 4), which might be transmitted during oviposition.

HR-like leaf traits induced by eggs of pierid butterflies laid onto brassicaceous plant species have been intensively studied (e.g., 43, 44, 50). If pierid egg depositions elicit a strong HR-like leaf necrosis in these plants, then this egg-induced response causes desiccation or detachment

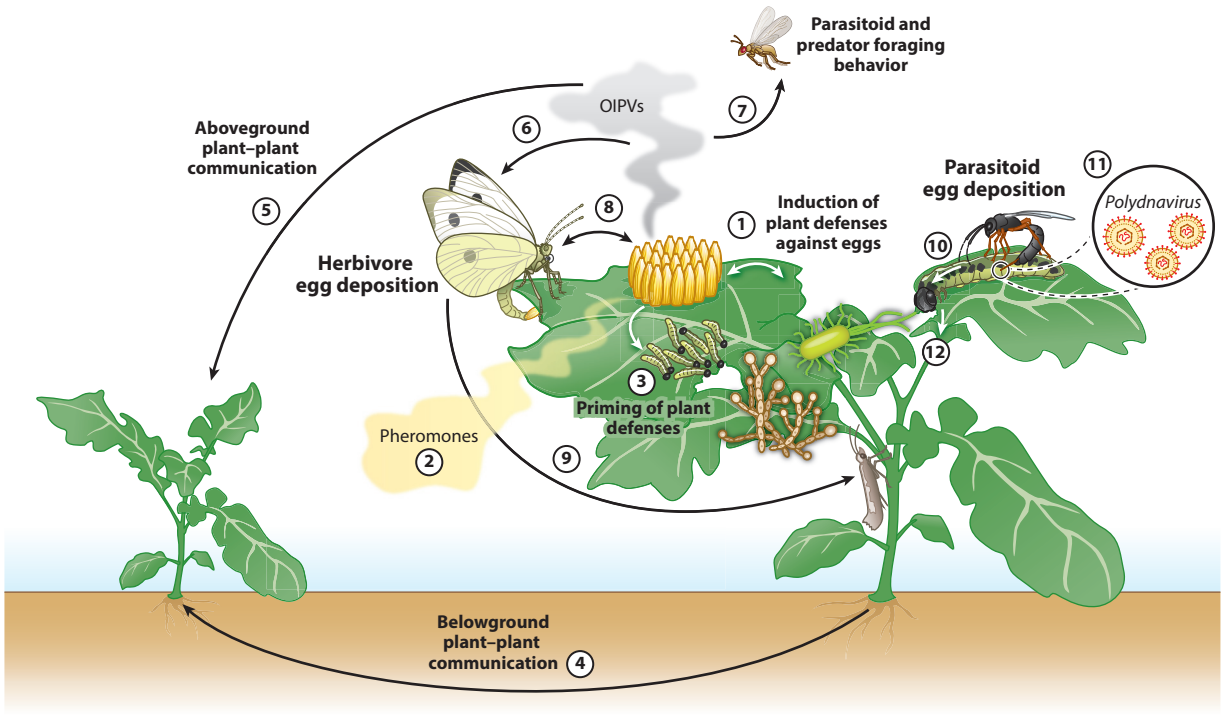

Figure 2

Interactions of insect eggs with their environment. Egg deposition by herbivorous insects affects the first trophic level by inducing plant defenses targeting the eggs (1). These egg-induced defenses can be primed by prior exposure of the plant to insect sex pheromones (2). Eggs on a plant can also prime defenses against hatching larvae and/or phytopathogens (3). Egg-induced plant cues may affect the resistance of neighboring plants against phytopathogens via belowground signaling (4). Furthermore, oviposition-induced plant volatiles (OIPVs) may elicit responses in neighboring plants and affect the neighboring plant's defenses against larval herbivory and susceptibility to oviposition by herbivores (5). OIPVs emitted from an egg-laden plant may deter further oviposition by herbivores (6), but they can also attract egg parasitoids and predators to attack the eggs (indirect plant defense) (7). Eggs already laid on a plant may be marked by chemicals deterring further oviposition by conspecific (8) and heterospecific (9) herbivores. Parasitoids also may mark their oviposition sites with pheromones, which deter further attack by conspecific parasitoids on the same host individual (10). Polydnaviruses, which are injected into host larvae during parasitoid oviposition (11), can change the composition of the oral secretions of the host larvae, and this in turn affects plant defense responses (12). For references, see Section 4.

of pierid butterfly eggs from leaves (43). In *Brassica nigra*, eggs of different Pieridae showed high species specificity in their induction of necrosis. Only Pieridae specialized on Brassicaceae induced an HR-like leaf necrosis, which was linked with enhanced *PR1* expression and ROS accumulation. In contrast, pierid species adapted to other plant families induced neither HR-like necrosis nor mild chlorosis (43). To better understand the responses of different plant species to egg deposition by different insect species, further identification of egg-associated elicitors of plant responses is needed (110).

Most of the known egg-associated elicitors of plant defenses are low-molecular-weight organic compounds (62). The first proteinaceous plant defense elicitor associated with insect eggs is an annexin-like protein that has recently been identified in secretion attached to the eggs of pine sawflies. It induces the production of a pine volatile compound [(*E*)-β-farnesene, a sesquiterpene] that attracts egg parasitoids (55). The effects of egg-associated elicitors on plant defense can be highly context and species specific if the triggered defense response involves the attraction of

egg parasitoids. The effects of an elicitor associated with *Pieris brassicae* eggs are specific to the plant species and even to the strain of the responding parasitoids (e.g., 3). We need to gather further data not only on the context dependency and species specificity of egg-associated plant defense elicitors, but also on the plant receptors involved in egg detection. Moreover, we need more detailed knowledge as to how a plant processes the information that insect eggs are being deposited on leaves (110).

Pipecolic acid: accumulates in plants after bacterial infection; treatment with pipecolic acid increases plant resistance to phytopathogens by inducing defense gene expression

Plant defensive responses to insect eggs can even be improved by prior exposure of a plant to insect sex pheromones. A recent study showed that defenses of *Pinus sylvestris* against eggs of the herbivorous sawfly *Diprion pini* can be primed by prior exposure of pine to the sawfly sex pheromones. Survival of eggs on pheromone-exposed pine was significantly reduced. This priming effect was linked with enhanced hydrogen peroxide accumulation and differential gene expression in pheromone-exposed, egg-laden pine (12). The species specificity of this priming effect remains to be studied.

Besides inducing plant defenses targeting the eggs, insect eggs may indirectly warn a plant of impending biotic threats. Egg-induced plant signals involving the pipecolic acid pathway were shown to be transmitted belowground via the roots to neighboring plants, thus improving the neighbors' resistance to phytopathogens (94). Furthermore, eggs may be taken by a plant as warning of impending larval herbivory, allowing the plant to upregulate its defenses against hatching larvae. This priming of defenses against larvae by insect eggs seems to be widespread across plants, with several recent studies providing new insights into the molecular and transcriptional fine-tuning of plant defenses against larvae by prior egg deposition or egg-induced plant volatiles (e.g., 14, 76, 97).

4.2. Oviposition Affects Further Oviposition by Second and Third Trophic Level Members

Female herbivores and parasitoids searching for a suitable oviposition site can be either deterred by or attracted to the presence of con- or heterospecifics' eggs (e.g., 10, 18). Their choice of an oviposition site may be manipulated by egg-induced plant cues or cues emanating from previously laid eggs.

The induction of plant volatiles by insect egg deposition has been addressed in several reviews (e.g., 10, 16). Oviposition by herbivorous insects onto leaves induces plant volatiles [oviposition-induced plant volatiles (OIPVs)], which affect the behavior of adult female herbivores and their choices for further oviposition (50). OIPVs might signal impending competition among the hatching herbivorous larvae and thus repel further oviposition (31). In contrast, host-searching egg parasitoids are known to be attracted to OIPVs or to intensify their search in response to egg-induced changes of the leaf surface chemistry (6, 30, 50, 102). The effect of OIPVs on the oviposition behavior of herbivores is not limited to the egg-laden plant itself. Eggs of the moth *Micromelalopha sieversi* laid on poplar leaves stimulated the emission of specific monoterpenes (3-carene and β-pinene) not only from the egg-laden focal plants, but also from egg-free neighboring plants (46). The apparent plant–plant interaction via leaf volatiles resulted in significantly fewer eggs on the neighboring plants when these were also offered to the moths for oviposition.

In addition to OIPVs, insect oviposition-deterring pheromones (ODPs) may significantly affect the choice of an oviposition site. ODPs, which are deposited by the egg-laying female either directly onto the eggs or adjacent to them at the oviposition site, can warn of impending larval competition at a site with limited resources for the offspring. Despite our understanding of the ecological effects of ODPs, only a few egg- or oviposition-associated ODPs of herbivores have been identified (5). Similarly, our knowledge of the chemistry of marking pheromones used by egg-laying parasitoids is very limited (18). Detection of host-marking pheromones

allows a parasitoid female to avoid hosts in which her offspring would be subject to intraspecific competition with the offspring of the conspecific marking female (e.g., 102). Since a marked host is unlikely to receive further oviposition, the host-marking parasitoid itself benefits as well from host marking if host resources are limited. Conversely, laying more parasitoid eggs into one host individual may also help to overwhelm the host's immune responses and encapsulation (78).

Polydnaviruses (PDVs): viruses that replicate in the ovaries of certain parasitoids without harming the parasitoids and suppress the immunity of the parasitoid's host

In contrast to ODPs, aggregation pheromones are well known to stimulate oviposition (63, 84). Recent studies have addressed the effects of chemicals released by females of *Drosophila suzukii*, which oviposit into ripening fruits and mark the site with an anal fluid. This marking fluid contains several compounds, which have been identified in the aggregation pheromone of *D. melanogaster* and also elicited clear electroantennographic responses in *D. suzukii* (127). However, the function of the *D. suzukii* marking fluid in determining the oviposition choices of this species seems to be highly context dependent (27, 127).

4.3. Oviposition by Parasitoids Affects Second and First Trophic Level Members

Oviposition into hosts is associated with an intriguing type of mutualism between the parasitoid host egg and certain types of viruses, so-called polydnaviruses (PDVs) (8, 123). The PDVs injected into host larvae by an egg-laying parasitoid not only inhibit encapsulation of parasitoid eggs and/or parasitoid larvae by the host's immune system, but also lead to physiological changes in herbivorous host larvae. The viral infection reaches the salivary glands of the herbivorous host larvae, where viral-encoded proteins secreted with the host saliva into feeding sites may either directly change the host plant's phenotype or manipulate the biosynthesis of plant defense elicitors in the host's saliva (128, 149). As a consequence, the plant's defense against host larvae is attenuated, which may benefit the development of the parasitoid inside the host. However, hyperparasitoids are attracted by the odor of plants that are infested by parasitized, PDV-infected host larvae. Thus, parasitoid egg deposition associated with PDVs affects not only the second and first trophic levels, but also the fourth level (19, 22).

5. CONCLUSIONS

While insect eggs have been intensively studied with respect to their innate developmental program (95, 126, 134, 137), much less attention has been paid to their adaptive plasticity in response to environmental conditions. In addition to a plethora of parentally provided egg-protective measures, the adaptive abilities of the insect eggs themselves must certainly have contributed to the evolutionary success of egg laying as a reproductive strategy in insects. However, we still do not really know how insect eggs sense their environment and how they sense temperature, relative humidity, and other crucial environmental information. Furthermore, future studies need to address how events experienced in the egg stage affect later developmental stages. To date, egg-associated microbes have been shown to contribute to protection of eggs from biotic threats, but it remains to be explored whether egg-associated symbionts also contribute to protection from unfavorable abiotic conditions such as cold or drought. Moreover, it is now abundantly clear that insect eggs exert significant effects on various trophic levels, and these effects may cascade up and down in food webs. There is still a tremendous amount to be learned about the dynamic multipartite systems composed of host plants; parental insects and their eggs; and the plethora of parasitoids, predators, and pathogens that attack eggs.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

ACKNOWLEDGMENTS

This work was supported by grants from the German Research Foundation (DFG-SFB-973) to M.H. and the Max Planck Society to H.S.

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Annual Review
of Entomology

Volume 68, 2023

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Errata

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