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Genetical and Physiological Mechanisms of Adult Diapause in Insects

Yong Gyun Kim

ABSTRACT Adult diapause in insects is characterized by suppression of reproductive development. It is induced by environmental cues such as photoperiod, temperature, food availability, and other conditions. Diapause-inducing environment is recognized and analyzed by the brain of the insects. The interpreted information is conveyed via endocrine system to target tissues such as ovaries, fat body, and other tissues. From this signal hierarchy of a brain-endocrine-target tissue axis, several factors are involved to express a diapause trait in a quantitative mode, even though the insects show a binomial phenotype between being in diapause or not. Recent works estimated that the number of the factors is relatively small by a series of crossing trials between high and low diapause lines. Heritability of the diapause is quite high (ca. 70%) in some species. Epistasis, sex-linkage, pleiotropism, and other nongenetic components also affect diapause inheritance. Most physiological studies have been focused on control mechanisms of the juvenile hormone (JH) synthesis in corpora allata (CA) because JH level in hemolymph of teneral adults is critical to decide a later developmental mode. Allatostatin, an antagonizer of JH synthesis, has been believed to be a potent brain message to CA for adult diapause induction.

KEY WORDS Adult diapause, inheritance, minimum number of genes, heritability, juvenile hormone, ecdysone, corpora allata, vitellogenesis, allatostatin

Predictably unfavorable conditions, such as extreme temperatures and photoperiods, drought, and a short food supply are inevitably encountered by most insects established in temperate climates. To survive such unfavorable periods, insects have developed numerous mechanisms ranging from a simple reduction in metabolic rates with quick recovery to complicated, genetically-programmed diapause development. Diapause is a developmental option in a specific stage according to species.

Adult diapause occurs in some Coleoptera, Lepidoptera, Diptera, Homoptera, Hemiptera, Orthoptera, Neuroptera, Trichoptera, and Acarina (Beck 1980; Danilevsky 1965; Saunders 1982).

The unique feature of adult diapause is suppression of reproduction. In females, the primary oocytes within the germarium are formed, but do not undergo vitellogenesis. In males, mature spermatozoa are already formed at adult emergence but the accessory glands are involuted and do not have secretory activity (de Wilde 1983).

Adult diapause also has several metabolic characteristics: a low rate of oxygen consumption (Denlinger 1985, Rosales *et al.* 1994), synthesis of diapause-associated proteins (de Loof & de Wilde 1970, Dortland 1978; Turunen & Chippendale 1980), and high activity of JH specific esterase (Kramer & de Kort 1976; Kim & Krafsur 1994).

These diapause traits are expressed in response to environmental stimuli during the pre-diapause and diapause induction period. Diapause development is mediated through specific endocrine changes (Denlinger 1985; Tauber *et al.* 1986; Burks *et al.* 1992; Kim & Krafsur 1993). Depending on the amount of hormone produced, the hormonal target tissues undergo reproductive or diapause development.

GENETIC MECHANISMS

Diapause as a Quantitative Trait

Diapause expression and its interaction with envi-

Department of Agricultural Biology, College of Natural Sciences, Andong National University, Andong, Korea

ronment are under genetic control (Danks 1987). Diapause is a threshold trait. The threshold levels of critical photoperiod are normally distributed in a population (Gibbs 1975; Beck 1980). Thus, diapause is a quantitative trait. The quantitative trait is measurable and has a continuous property because of simultaneous segregation of many genes affecting the trait and interactions between genotype and environment. Quantitative traits are also called "polygenic" because they are concerned with many small, additive gene effects. Genetic mechanisms in diapause focus on mode of inheritance by actions of diapause-associated genes. Estimations of heritability and number of effective genes are valuable to understand a mode of diapause inheritance.

Variance Components

In the simplest case with no interaction between genotype and environment, measured phenotypic value (P) results from the effects of genotype (G) and environment (E).

$$P=G+E$$

Genotypic value is composed of additive (A), dominance (D) and interaction (I) terms.

$$G=A+D+I$$

In this way, phenotypic variation (σ_P^2) can be divided into genetic (σ_G^2) and environmental (σ_E^2) components. Genetic variance is further partitioned into additive (σ_A^2), dominance (σ_D^2), and interaction (σ_I^2) components.

$$\sigma_P^2=\sigma_G^2+\sigma_E^2=\sigma_A^2+\sigma_D^2+\sigma_I^2+\sigma_E^2$$

Estimation of Heritability

The relative importance of heredity in determining phenotypic values is called heritability (review by Falconer, 1981). Estimates of the proportion of phenotypic variation that is genetic are called broad-sense heritabilities and are based on estimates of genetic (σ_G^2) and phenotypic (σ_P^2) variances. The general formula for broad-sense heritability (H) is

$$H=\sigma_G^2/\sigma_P^2$$

Broad-sense heritability is a general estimate of degree of genetic determination. It includes each average gene effect (additive effect), intra and interlocus interactions (dominance and epistasis). The contribution of intralocus interaction, as well as some interlocus interactions, is not transmitted from parent to progeny because two alleles, or sets of alleles, are separated at meiosis. Therefore, broad-sense heritability would give the breeder an inaccurate measure as to what extent selection would be effective for a trait of interest.

Narrow-sense heritability limits measurement of genetic variation to that associated with the additive effects of individual alleles influencing a character. It determines the degree of resemblance between relatives and so gives the most important information to breeders The additive variance (σ_A^2) is separated from σ_G^2 and the general formula for narrow-sense heritability (h^2) is

$$h^2=\sigma_A^2/\sigma_P^2$$

Difference between narrow-sense heritability and broad-sense heritability estimates calculated from the same data sets are caused primarily by nonadditive gene effects.

Several techniques to estimate heritability have been proposed (Warner 1952; Falconer 1981):

(1) parent-offspring regressions
(2) variance analysis among sibs
(3) variance partitioning using generation variance

Estimation of Minimum Number of Genes

Estimating the number of the genes contributing to quantitative genetic variance is necessary to understand physiological mechanisms as well as evolutionary mechanisms of a biological phenomenon

The original method of Wright (in Castle 1921) used two inbred lines to estimate the minimum effective gene number. Wright later (1968) included backcrosses. Lande (1981) generalized Wright's formulas to crosses between genetically heterogeneous populations to minimize the inbreeding depression in each parental line and to reduce the experimental time. For a normally distributed trial, the minimum number (NE) of genes contributing to quantitative traits is

$N_E=(P_2-P_1)^2/8\sigma_s^2$,

where P_1 and P_2 are phenotypic values of both parents (Lande 1981) Under the assumptions of no dominance, epistasis, and no environmental interaction, genetic variances (σ_s^2) may be calculated by one of the four equations:

(1) $\sigma_s^2=\sigma_{F2}^2-\sigma_{F1}^2$.
(2) $=\sigma_{F2}^2-(1/2\sigma_{F1}^2+1/4\sigma_{P1}^2+1/4\sigma_{P2}^2)$
(3) $=2\sigma_{F2}^2-\sigma_{B1}^2+\sigma_{B2}^2$.
(4) $=\sigma_{B1}^2+\sigma_{B2}^2-(\sigma_{F1}^2+1/2\sigma_{P1}^2+1/24\sigma_{P2}^2)$,

where σ_{P1}^2, σ_{P2}^2 σ_{F1}^2, σ_{F2}^2, σ_{B1}^2, and σ_{B2}^2 are variances of parents, F_1, F_2, and backcrosses. Lande (1981) also mentioned that the effective number of genes estimated by his method cannot exceed the number of crossing-overs at meiosis ("recombination index" of Darlington 1937), which equals the number of recombination events per gamete.

Cockerham (1986) corrected the squared difference between the means of two parents in Lande's equation by subtracting the sum of their experimental variances:

$(P_2-P_1)^2=(P_2-P_1)^2-(\sigma_{P1}^2-\sigma_{P2}^2)$.

While Lande (1981) gave four measures of genetic vanance, Cockerham (1986) estimated only one genetic variance by a least squares method using information from all crosses:

$\sigma_S^2=0.2(4\sigma_{F2}^2+\sigma_{B1}^2+\sigma_{B2}^2)-0.4(\sigma_{P1}^2+\sigma_{P2}^2+\sigma_{F1}^2)$.

Lande (1981) formulated an approximate variance of NE on the assumption that NE is normally distributed:

$V(N_E)=N_E^2\{4(\sigma_{u1}^2+\sigma_{u2}^2)/(P_2-P_1)^2+(V(\sigma_s^2)/\sigma_s^4\}$,

where $\sigma_{ui}^2=\sigma_{Pi}^2/N_{Pi}$. Cockerham (1986) gave one estimate of $V(\sigma_s^2)$ by an unweighted least square method.

$$V(\sigma_s^2)=0.08(16\sigma_{F2}^4/N_{F2}+\sigma_{B1}^4/N_{B1}+\sigma_{B2}^4/N_{B2}) +0.32(\sigma_{P1}^4/N_{P1}+\sigma_{P2}^4/N_{P2}+\sigma_{F1}^4/N_{F1}).$$

From several examples (Lande 1981) in which the parental mean differences ranged from 6 to 30 phenotypic standard deviations, the effective minimum gene numbers were 5 to 10 with occasional values up to 20. Linkage of loci influencing a trait causes N_E to be underestimated because it increases the genetic variance (σ_s^4)

Modes of Diapause Inheritance

Inheritance patterns of insect diapause vary among species. Diapause inheritance in many species has been explained by a polygenic control because of a continuous variation among populations in quantitative characteristics such as critical photoperiod, duration of diapause, and rate of diapause development (Lees 1968; Masaki 1984; Tauber *et al.* 1986) Geographical variation in diapause response was analyzed through hybridization tests with naturally diversified populations of the knotgrass moth, *Acronycta rumicis* and suggested a variation of gene frequencies according to local populations (Danilevsky 1965; Lees 1968). Estimations of the number of the genes involved in polygenic diapause were conducted in the flesh fly, *Sarcophaga bullata* Parker (Henrich & Denlinger 1983), the fruit fly, *Drosophila littoralis* Meigen (Lankinen 1986), and the bug, *Oncopeltus fasciatus* (Dallas) (Hayes *et al.* 1987). These studies showed that a small number of loci may be involved in polygenic control of diapause.

Some species, in contrast, showed a simple Mendelian inheritance of diapause such as in two sibling species of the antlion, *Chrysopa* (Tauber *et al* 1977) and in the blow fly, *Calliphora vicina* Robineau-Desvoidy (Vinogradova & Tsutskova 1978), where one or two genes control diapause with complete dominance In *D. littoralis*, an autosomal unit of closely linked genes ("supergene") is responsible for diapause inheritance. This supergene is segregated in a Mendelian way, but its phenotypic variance follows a polygenic trait (Lumme & Oikarinen 1977; Lumme 1981)

Sex-linkage of diapause-associated genes was reported in many species. The gene carried by the heterogametic sex is normally fully expressed, but the gene in the homogametic sex is expressed according to a dominance relationship. In most sex-linked species in diapause, the inheritance is chiefly through the female parent. Paternal inheritance, however, was reported in the moths, *Pionea forficalis*

(Linnaeus) (King 1974), *Heliothis* species (Stadelbacher & Martin 1981) and some other Lepidoptera. This may be due to the fact that the heterogametic sex is the female in Lepidoptera (Danks 1987)

Epistasis and pleiotropism are also involved in diapause inheritance (Danks 1987). Epistasis, the interaction of two or more loci, was involved in diapause inheritance of *S. bullata* (Henrich & Denlinger 1983). Pleiotropism is indicated when a single gene has more than one phenotypic effect such as both diapause response and body color in *D. littoralis* (Lumme & Oikorinen 1977)

Genetic analysis of diapause gave high heritability estimates (=high additive genetic variance): ca 0.70 for the ages at first reproduction in *O. fasciatus* (Dingle *et al* 1977), 0.43 to 0.82 for heat requirements for the completion of diapause in the moth, *Hyphantria cunea* (Drury) (Morris & Fulton 1970), 0.77 ± 0.36 for the number of days to emergence in *H. zea* (Holtzer *et al.* 1976), and 0.31 to 1.13 for diapause in the cricket, *Allonemobious fasciatus* DeGeer (Mousseau & Roff 1989).

Diapause was genetically correlated with developmental rates in some species. Selection for fast development in the mosquito, *Wyeomyia smithii* (Coquillett), produced a correlated response of a decrease of diapause induction (Istock *et al.* 1976). Selection for late pupariation in *S. bullata* resulted in a line that showed a higher diapause incidence than the parental line (Henrich & Denlinger 1982).

PHYSIOLOGICAL MECHANISMS

Adult diapause (=imaginal diapause) is maintained by the suppression of reproductive development. Thus, adult diapause mechanisms are very closely related to reproductive pathways.

Spermatogenesis

Spermatogenesis produces haploid spermatozoa from diploid spermatogonia in male reproductive systems. In each follicle of the testis, a range of development is present with the earliest stages distally in the germarium and the oldest in the proximal part of the follicle adjacent to the vas deferens. Typically, three developmental zones are formed below the germarium. *A zone* of growth is located just below the germarium, in which spermatogonia, enclosed in cysts, divide and increase in size to form spermatocytes. In a zone of maturation, each spermatocyte undergoes meiosis to produce spermatids. Spermiogenesis, the process in which the spermatids develop into spermatozoa, then occurs in a zone of transformation.

In most male insects, meiosis is complete before adult emergence. Especially in species which do not feed and have a short life span as adults, spermatogenesis may be complete before adult emergence (Engelmann 1970).

Endocrine studies on spermatogenesis are not as frequent as on vitellogenesis. Several early workers suggested that juvenile hormone (JH) inhibited differentiation of sperm, but ecdysone promoted this process (review by Dumser 1980). This model of spermatogenesis indicates that adults are unfavorable for spermatogenesis because they lack ecdysteroids and have active corpora allata Dumser & Davey (1975) proposed that the hormones affect only the rate of spermatogonial mitosis and not differentiation and that there is a basal rate of mitosis even in the presence of JH. Thus in adults, spermatogenesis continues at the basal rate, despite the presence of juvenile hormone in the hemolymph.

Male accessory glands are fully differentiated at adult emergence. During sexual maturation, they become active and increase in size. In many insects, there is a clear correlation between the corpora allata (CA) activity and secretory activity of accessory glands (Engelman 1970; Gillott & Friedel 1976).

In most species, diapause in males is characterized by involution of the accessory glands and the absence of sexual behavior (Denlinger 1985).

Vitellogenesis

Vitellogenesis is the process of egg maturation in the female reproductive system and is mediated by endocrines under the control of external factors such as photoperiod, temperature, and feeding (Adams 1981; Hagedorn & Kunkel 1979). JH, which is synthesized in the CA, acts as a gonadotropin and activates vitellogenin (Vg) synthesis in the fat body of most insect species. Ecdysteroids are produced by

the ovaries and stimulate the fat body to synthesize Vg in some dipteran species, e.g., the mosquitoes, *Aedes aegypti* (Linnaeus) (Hagedorn & Kunkel 1979), the fruit fly, *Drosophila melanogaster* (Meigen) (Postlethwait *et al.* 1980), and the house fly, Musca domestica Linnaeus (Adams & Filipi 1988). The median neurosecretory cells (mNSCs) produce allatotropin, which then activates JH synthesis in the CA, and may also produce egg development neurosecretory hormone (EDNH) that acts as gonadotropin in some insect species. The insect ovary produces ovarian hormones that inhibit either gonadotropin release or the activation of the receptor on the follicle cells by the gonadotropin.

According to the synchrony of egg maturation in an ovariole, vitellogenesis is divided into asynchronous and synchronous types (Adams 1981). Synchronous vitellogenesis is further subdivided into intraovariole, interovariole, and uniovariole synchronous types

Vitellogenin Synthesis

Vitellogenins are the egg proteins. They are synthesized by the fat body, secreted into hemolymph, and taken up by the growing oocytes, where they are called "vitellins" (Engelmann 1979, Hagedorn & Kunkel 1979). In *D. melanogaster,* both the ovary and the fat body synthesize vitellogenin (Bownes 1980, Postlethwait *et al* 1980). Ovarian synthesis of yolk proteins was also found in *M. domestica* (de Bianchi *et al.* 1985)

Few differences have been found to exist between vitellin and vitellogenin, although a little difference was found in lipid content and protein subunit structure in some species (Hagedorn & Kunkel 1979).

Vitellogenins of many insects, vertebrates, nematodes and sea urchins are very similar in size and amino acid composition (Blumenthal & Zucker-Aprison 1986). It appears that they are encoded by the same family of genes Higher dipteran species, however, have a little different protein and gene structures than the other insects. In contrast to those of other insects, their vitellogenins consist of small peptides (45~50 Kd) and have a single copy gene (Rina & Savakis 1991; Spieth *et al.* 1985).

In *D. melanogaster*, three yolk proteins were found and range 44~47 Kd in molecular weights. They are encoded by single copy genes (Yp1, Yp2, & Yp3) which are localized on the X chromosome. Genes Yp1 and Yp2 are separated by 1.2 Kb and transcribed divergently (Hung & Wensink 1982 1983). Yp3 gene is ca. 1000 Kb from the Yp1 and Yp2 genes (Barnet *et al.* 1980). These genes have their own promoters and are coordinately transcribed in response to ecdysone (Hung & Wensink 1982).

Yolk protein synthesis is under hormonal control. In some insects, such as the grasshopper, *Locusta migratoria* (Linnaeus) (Wyatt 1988) and the cockroach, *Leucophaea maderae* (Fabr.) (Engelman 1979), vitellogenin synthesis is said to be regulated only by juvenile hormone. But, in many species of Diptera, both juvenile hormone and ecdysone are involved in yolk protein synthesis in the fat body and/or the ovary (Hagedorn 1985).

Vitellogenin Uptake

JH is known to act on the cell membrane of the follicular epithelium and activate a specific Na^+, K^+-ATPase (Pratt & Davey 1972; Abu-Hakiwa & Davey 1979). Activated Na^+, K^+-ATPase induces reduction of the follicle cell volume which forms intercellular spaces in the follicular epithelium. These spaces allow the entry of hemolymph containing Vg. Ilenchuk & Davey (1985) suggested that JH specific receptors are located on the cell membrane of the follicular epithelium. Protein kinase C is involved in the linking steps between JH-specific receptor and JH-sensitive Na^+, K^+-ATPase (Sevala & Davey 1989).

Vg is specifically taken up and accumulated in the growing oocyte by receptor-mediated endocytosis (Raikhel & Lea 1986). In Rhodnius prolixus Staal, this process is facilitated by the presence of Ca^{2+} and JH. This facilitation may be explained by the increase of the number of receptors (Wang & Davey 1992)

Roles and Structures of JH

Since Wigglesworth (1936) first demonstrated the effects of JH in morphogenic and reproductive development in *R. prolixus,* many other workers have established the physiological roles of JH in different

insects (Engelman 1970). JH also plays a crucial role in various types of polymorphism (Novak 1975): (a) caste polymorphism in ants and termites (b) phase polymorphism in locusts (c) seasonal polymorphism in aphids, and (d) polymorphism in wing development in many species in Heteroptera.

JHs have a common sesquiterpenoid structure. The first characterized JH was JH I from the silk moth, *Hyalophora cecropia* (Linnaeus) (Röller *et al.* 1967). It was the first known natural product with a dihomosesquiterpenoid skeleton (Schooley & Baker 1985). JH II was identified by Meyer *et al* (1968) from the same species. JH III, JH 0, and 4-methyl JH I were discovered by Judy *et al.* (1973) and Bergot *et al.* (1980) from the sphinx moth, *Manduca sexta* (Linnaeus). JH III is the predominant hormone in most adult insects (Schooley *et al* 1976) while JH I and JH II have been identified mainly in larvae of Lepidoptera and in nymphs of the cockroach, *Nauphoeta cinerea* (Lanzrein *et al.* 1975). Recently, JH III bisepoxide (JH B3) has been found to be the major JH in higher Diptera such as *D. melanogaster, S bullata* (Richard *et al.* 1989) and *Calliphora vomitoria* (Linnaeus) (Cusson *et al.* 1991).

Biosynthesis and Degradation of JH

The JH titer in the hemolymph is a dynamic equilibrium between the rate of JH synthesis and release by the corpora allata (CA) and the rate of uptake and degradation of the hormone by the tissues (Feyereisen 1985). After synthesis, JH is not stored in the gland cells but secreted almost immediately (Tobe & Pratt 1974) Therefore, the synthetic activity gives a direct effect on JH titer in hemolymph. The synthetic activity is dependent on the precursor amount and on the activity of the key enzymes in the synthetic pathway Regulation of the JH synthetic activity by the CA has been considered as the primary cause of the low JH titer during adult diapause (Denlinger 1985).

The biosynthetic pathway (Fig. 1) of JH III is similar to cholesterol biosynthesis in vertebrate liver (Schooley & Baker 1985). Up to the formation of farnesyl pyrophosphate ('early steps'), the two biosynthetic pathways are almost identical. Thereafter, several final enzymatic steps ('late steps') catalyze the formation of JH.

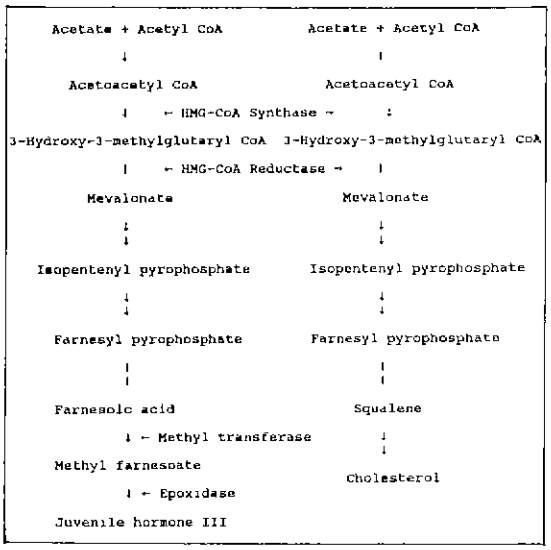

Fig. 1. Biosynthetic pathways of JH III and cholesterol (modified from Khan 1988)

JH II, JH I, JH 0, and 4-methyl JH I do not follow the synthetic pathway of JH III because of their ethyl branches. Two main explanations for the generation of ethyl branches were postulated: methylation after the formation of JH III by a methyl donor such as S-adenosyl-methionine or condensation of a homoisoprenoid synthesized from propionate via homomevalonate Most studies so far support the homomevalonate hypothesis from the degradative analysis of radiolabelled products after *in vitro* culturing in radiolabelled precursors such as acetate, propionate, and mevalonate (Schooley *et al* 1973; Peter & Dham 1975)

The major pathways for JH degradation in insects are hydrolysis of the ester moiety by JH esterase (JHE) and hydration of the epoxide ring by epoxide hydrolase. The primary products of these reactions are JH acid and JH diol (Slade & Zibbit 1972).

Besides JHE and epoxide hydrolase, mixed function oxidases (MFO) are involved in JH catabolism in the house fly, *M. domestica* and some other Diptera (de Kort & Granger 1981). Piperonyl butoxide and carbon monoxide, which are inhibitors of MFO, inhibit JH catabolism in vitro (Yu & Terriere 1978).

Phenobarbital, a potent inducer of MFO activity, blocks metamorphosis and vitellogenesis (Yu & Terriere 1974).

In the hemolymph, JH is degraded only by JHE, which is synthesized in the fat body and released into hemolymph (Whitmore *et al.* 1974) Unlike general esterases, JHE are capable of rapidly hydrolyzing both free JH and JH bound to JH binding protein (Hammock 1985). JHE is insensitive to 10^{-3} M diisopropylfluorophosphate, while general carboxy-esterases are totally inhibited.

The importance of high JHE activity in adult diapause has been shown in *L. decemlineata* (Kramer & de Kort 1976). Theactivity of JHE increases about 10-fold in prediapausing beetles. High JHE activity is not the primary cause for the low JH titer during early diapause. Rather, high JHE activity may make it possible to attain low JH levels rapidly early in diapause induction (de Kort 1990).

Pathways of Stimuli Affecting CA Activity

It has been known from classical endocrinology that insect CA are under the control of the brain (Engelmann 1970) Several factors affect CA activity These factors can be classified as external and internal stimuli (Khan 1988). External stimuli include photoperiod, food availability, and mating. Internal stimuli are further subdivided into neural and humoral factors from the brain and the target organ (e.g. ovaries). The feedback control of JH titer to CA activity is also one of internal stimuli.

Regulation of CA activity in response to external and internal stimuli can be separated into rapid and slow physiological and biochemical responses (Feyereisen 1985) The effects of both stimuli are expressed via controlling the actions of rate-determining enzymes in JH biosynthesis in the CA. Changes in non-rate-determining enzymes, cell volume or number, and cell ultrastructure also affect the total biosynthetic capacity of the CA.

Corpora allata are innervated from both the brain and the suboesophageal ganglion (Tobe & Stay 1985) The connection between the gland and the suboesophageal ganglion does not play a role in controlling CA activity because severance of this nerve did not influence the gland activity in the cockroaches, *Diploptera punctata* (Stay & Tobe 1977) and Periplaneta americana (Linnaeus) (Pipa 1982), and the grasshopper, *L. migratoria* (Couillaud *et al* 1984).

Neural transport of allatostatins from the brain has been reported in many species. Williams (1976) proposed the existence of both an allatotropin and an allatostatin for activating and inactivating JH biosynthesis, respectively. Recently, four related peptides with allatostatic activity in vitro have been isolated and characterized from the brain of *D. punctata* (Woodhead *et al.* 1989). An allatotropin has been also isolated from *M. sexta* (Kataoka *et al.* 1989).

Adult diapause of *L. migratoria* is under dual control by allatotropic and allatostatic factors (Baehr *et al.* 1986). The allatostatic factor is located in the lateral NSCs whose axons reach the CA through NCC II and NCA I (Poras *et al.* 1983). NCC II or NCA I section inhibited diapause development by removal of CA inhibition. The denervated CA which were implanted into diapausing hosts continued to be active until 3 days after the operation. When the denervated CA were cultured *in vitro*. they became active. But, their activity declined rapidly. This difference between *in vivo* and *in vitro* results of the denervated CA can be explained by the presence of a blood-borne allatotropic factor.

Neural and humoral pathways between the brain and the CA are also found in *L. decemlineata* (Khan *et al.* 1983; Khan 1988). The results consistently indicated that any influence by way of the humoral pathway on the CA activity was subordinate to neural inhibition.

The biochemical targets of allatostatin in the CA are as yet unidentified, although they usually appear to be located early in the JH biosynthetic pathway (Feyereisen & Farnsworth 1987a). It is likely that cAMP and protein kinase (e.g., protein kinase C) play some role in the regulation of gland activity (Meller *et al.* 1985; Feyereisen & Farnsworth 1987 b).

The control of JH biosynthesis occurs at the early steps as well as the late steps, and varies among species The control at the early steps is characterized by stimulating the synthetic activity to many-fold its normal value after addition of farnesoic acid to

the incubation medium This control type has been found in *S. gregaria, D. punctata*, and *L. decemlineata* (Feyereisen *et al.* 1981; Khan *et al.* 1982).

3-hydroxy-3-methylglutaryl CoA (HMG-CoA) reductase is a key enzyme in the control of JH biosynthesis. This enzyme catalyzes the reduction of HMG-CoA to mevalonate which is an irreversible and an important regulatory step in cholesterol biosynthesis (Brown & Goldstein 1980). HMG-CoA reductase is a glycoprotein and located in the endoplasmic reticulum (Daughtery-Varsat & Lodish 1983). Several mechanisms are involved in the control of HMG-CoA reductase activity during feedback control of cholesterol biosynthesis: by phosphorylation/dephosphorylation (Brown *et al.* 1979) or by the control at the level of transcription of the enzyme-coding gene (Luskey *et al* 1983)

A good correlation between the activity of HMG-CoA reductase and the rate of JH biosynthesis has been reported in *M. sexta* (Kramer & Law 1980). Its activity seemed to be controlled by phosphorylation/dephosphorylation (Feyereisen *et al.* 1981) Compactin, a substance which inhibits HMG-CoA reductase specifically, inhibited the rate of JH biosynthesis (Edwards & Price 1983).

Diploptera punctata, however, did not show any correlation between the activity of HMG-CoA reductase and the rate of JH biosynthesis (Feyereisen & Farnsworth 1987a). When the rate of JH biosynthesis was inhibited *in vitro* by exposure to high K^+-concentration, the activity of the enzyme was not significantly lowered (Feyereisen & Farnsworth 1987 b).

The control of JH biosynthesis at the late steps was reported in the inactivation of CA at the end of the last larval instar in *M sexta* (Bhaskaran *et al.* 1986). The inactivation was due to the loss of JH-methyl transferase activity but not the HMG-CoA reductase.

Ecdysteroids in Adult Diapause

Ecdysteroids, the molting hormone in insects, are produced in a prothoracic gland during immature stages. This gland is under brain control, via a neuropeptide hormone known as prothoracicotropic hormone. Prothoracic glands degenerate with adult development

The production of ecdysteroids depends on dietary cholesterol because insects cannot synthesize cholesterol. α-Ecdysone is the first characterized ecdysteroid in *Calliphora* (Karson *et al.* 1965). Later, 20-hydroxyecdysone (β-ecdysone) was found in *Bombyx mori* Linnaeus and other insects. 20-Hydroxyecdysone is a more effective form than α-ecdysone in many insects.

In higher diptera, ecdysteroids as well as JH have a role in inducing vitellogenin gene expression in the fat body (Bownes 1980; de Bianchi *et al.* 1985). The major source of ecdysone in adults is the ovaries and the testes even though oenocytes or epidermal cells synthesize a little ecdysone in some species (Hagedorn 1985). In *A. aegypti*, egg developing neurohormone is released from the corpora cardiaca after a blood meal and stimulates the synthesis of ecdysone in the ovaries (Shapiro & Hagedorn 1982). Adams & Filipi (1988) have shown that both JH and 20-hydroxyecdysone are necessary for the production of normal levels of vitellogenin in *M domestica*.

In *L. decemlineata*, ecdysteroid level slowly increased throughout diapause (Briers *et al.* 1982) Lefevere (1989) suggested that ecdysone plays a key role in diapause termination by modifying the regulation of the JH metabolism.

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