Hormonal Control of Diapause

Suspend development

The period of suspended development at any stage of the life cycle, under adverse conditions accompanied by greatly decreased metabolism is called diapause. This phenomenon is governed by a variety of external and internal factors. This is an adaptation to tide over unfavourable environmental condition like cold, dessication, draught, extreme temperature, humidity, photoperiod, nutritional, vitamin and other deficiencies. The other factors which determine the diapause and are equally responsible are the endocrine glands.

External / internal factors

Overcome the unfavourable condition

The hormonal regulation of diapause was earlier proposed by Wigglesworth 1934 and was confirmed by the experiments of Williams (1946) on Hyalophora which normally enters diapause just after pupation and may be induced to resume development by several months of chilling at 3–5°C. In saturniids the pupal diapause is caused by the absence of ecdysone, which results from inactivity of neurosecretory cells of the pars intercerebralis. Diapause has been broken in a number of species by the injection of α or β-ecdysone with the onset of adult development. A permanent diapause may be produced even in non-diapausing species by (removing) brain removal. This responses will vary from species to species.

Pupal — ecdysone

α-β-ecdysone → injection

Brain removed → diapause

non diapause

Thus the neurosecretory cells of the brain are the controlling elements in initiation and termination of hormonally regulated diapause. However, developmental arrest is not necessarily the result of a lack of ecdysone in all cases. Adult diapause may be brought on by corpora allata inactivity under the control of neurosecretory cells of the protocerebrum. In this case also as in the case of ecdysone regulated diapause, Again reactivation of the neurosecretory cells in the brain is required to terminate the conditions and restore reproductive activities.

There are 3 ~~principles~~ principal types of diapause can be distinguished on the basis of their hormonal mechanism.

A) Diapause cause by a lack of brain hormone and moulting hormone. This category belong to all cases of larval and pupal diapause (~~prothoracic diapause~~)

B) Diapause control by lack of brain hormone and Juvenile hormone. This mechanism is concerned with imaginal diapause. (Adult)

C) Diapause caused by the action of neurosecretory factor produced by the suboesophageal ganglion of the female and affecting the development of egg. This category comprises early embryonic diapause.

Adult diapause:

The adult diapause is the result of a deficiency in the brain hormone and JH. i.e. inactivity of the neurosecretory cells of the pars intercerebralis and CA. Morphologically, the adult diapause is only seen in the interruption of the growth of the ovaries and in the suppression of the functions of the accessary glands of both the male and female individuals. In calliphara, the implantation of CA can induce the ripening of some of the eggs but complete termination of diapause is possible only by the simultaneous action of JH and brain hormone. Apart from this, the presence of Brain hormone seems indispensible for the activity of CA. Evidences for CA (JH) regulation of adult diapause comes for a number of other species like - Aedes aegypti, Culex pipiens, Gryllodes etc. The experimental evidences clearly suggest that the basic intrinsic factor of imaginal diapause lies in the neurosecretory cells of the brain which appears to be part of the mechanism of external stimuli.

Pupal Diapause:

The principal cause of pupal diapause is the failure of endocrine activity i.e activity of the neurosecretory cells of the brain and the ecdysial gland. Thus to break the pupal diapause, presence of active ecdysial glands is necessary in addition to brain hormone. Implantation of active Ecdysial gland also breaks the pupal diapause. It is important to note that- implantation of one or more active brain into ~~an~~ an isolated abdomen in Hyalophora has no effect whereas the implantation of active ecdysial glands breaks the diapause and development continues. Injection of ecdysone has the same effect as the implantation of Ecdysial gland. Pupal diapause can takes place at ~~any~~ any point during the pupal stage. Its occurance in a given species is bound to be in the same phase of the pupal period and in most species it is genetically fixed. The common examples of pupal diapausing insects are Anisaeta moorei, Anisaeta collaris etc.

Larval Diapause -

It has been found that in the diapausing penultimate instar nymph of Gryllus ecdysial gland show reduced secretory activities and the brain appears to be responsible for this. Both the brain and ecdysial glands have also been shown to control diapause in larvae of Bombyx mori. It has been remarked that- the brain controls the activity of ecdysial gland and the CA and the principal control centre of diapausing larvae. It is estimated that- required photoperiod and temp which stimulate the nervous system to activate the MNSC of the brain to to release PTTH which thereafter activate the ecdysial gland to produce ecdysone or moulting hormone.

Early Embryonic Diapause:

The early embryonic diapause is associated with the diapausing hormone secreted by the suboesophageal ganglion. Removal of the suboesophageal ganglion from diapause producing pupae shortly after pupation completely eliminates diapause from the eggs eventually produced. When the operation is delayed a progressively larger proportion of eggs enters diapause.

The incidence of the diapause in the eggs is conditioned by endocrine activity of the neurosecretory cells of the suboesophageal ganglion of females.

→

The extirpation of SOG at the beginning of the pupal stage causes eggs which were determined for diapause through their mother. In the silk moth eggs development of one generation undergoes diapause while that of other generation is free from it. The diapausing eggs are laid in autumn. The cause seems to be a complex hormonal influence. The SOG contains some neurosecretory cells which secrete the diapause hormone or hibernation factor which acts on the ovaries. The secretion of diapause hormone lies under the control of the brain which is of nervous kind acting via circumo-oesophageal connectives. The brain stimulates or inhibits the release of Diapause hormone in the pupa and as a result emerging female lays the diapausing eggs in one generation and non-diapausing eggs in the other.

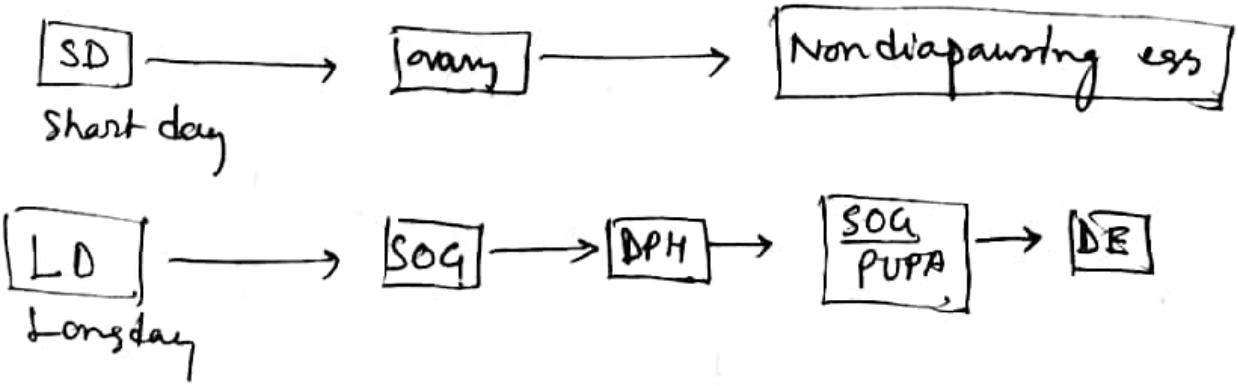

Fig - Hormonal Regulations of diapause in bivoltine B. mori.

In B. mori DNA and RNA content increases logarithmically in non-diapausing eggs but it remains a low level in diapausing eggs. Silkworm eggs under diapause are black in colour due to accumulation of pigment- ommochrome (in serosa cell) while in non-diapausing egg are light- yellow and devoid of the pigment. The pigment precourse B-hydroxykynurenine (B-OHKyn) is accumulated into the follicular cells during pupal development. The B-OH kyn synthesize in developing ovaries from tryptophane which is taken up from haemolymph. The diapause hormone elevates B-OH kyn absorption in the follicles from haemolymph.

Glycogen content- is initially higher in diapause eggs but it is later on, converted into polyols, sorbitol and glycerol. ovarian glycogen is derived from the haemolymph trehalase and it is synthesize as follows —

Trehalose
↓
Glucose
↓
Glucose – 6- phosphate
↓
Glucose-1- phosphate
↓
UDPG

Ovarian glycogen.
↑

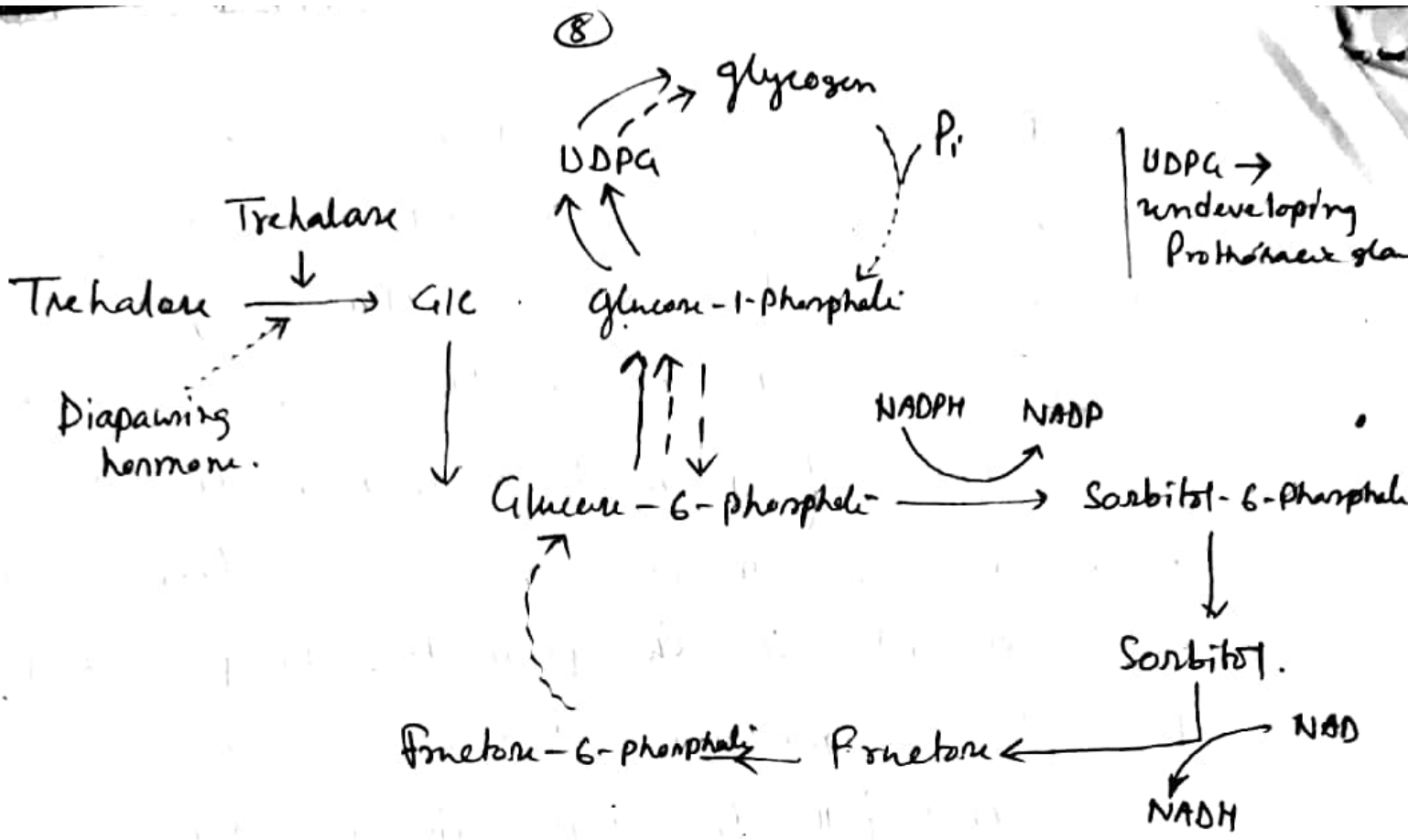

Fig- Metabolism of Carbohydrates in Diapausing egg; B. mor

1. HORMONAL CONTROL OF METAMORPHOSIS

Insect metamorphosis is controlled by a variety of hormones of which the most important are (1) brain hormone, (2) ecdysone and (3) juvenile hormone. Most of the work with insect hormones has been carried out by legaturing the larvae or pupae at various levels between anterior and posterior ends, by making grafts or by the injection of extracts of endocrine glands or synthetic hormones.

(1) BRAIN HORMONE

This is secreted by the neurosecretory cells of the brain which are usually 4-clusters a pair of medial and a pair of lateral cluster. The hormone secreted by these cells pass down the nerve axons of these cells and accumulates in the corpus cardiacum. Release of the hormone into the blood occurs at the corpus cardiacum level through several ways i.e. either by disintegration of granules and their diffusion into the blood or by means of exocytosis.

Experimental investigations in *Cecropia* silkworm show that both the median and lateral neurosecretory cells are necessary for the formation of this hormone. In fact the brain hormone is a mixture of several hormones secreted by different types of neurosecretory cells present in the brain.

Role of brain hormone in metamorphosis : This hormone has an indirect effect on metamorphosis. The chief function of this hormone is to control the secretion of the prothoracic glands. Consequently, the removal of the brain has the same result as the

removal of the prothoracic glands. In both cases the secretion of ecdysone is stopped and the insect enters into a state of diapause. Thus, diapause is due to the failure of the endocrine activity.

(2) ECDYSONE

This is also known as moulting hormone. Fraenkel (1935) found that when mature fly larvae were legatured, only the front end was able to form a puparium while the hinder part remained unchanged. Blood transfusion from the front to the hind end even in the legatured fly larvae immediately caused a change resulting in the pupation of the posterior part. From these observations, Fraenkel suggested that the growth of the larva into the pupa was dependent on a hormone secreted somewhere in the anterior end. Fukuda (1940) clearly demonstrated that it is secreted by the prothoracic glands or ecdysial glands. Finally it was Karlson (1954) who isolated the hormone in a highly purified stage and named it as "Ecdysone". Chemical study of ecdysone has clearly revealed that it is composed of two closely related molecules viz. α-ecdysone and β-ecdysone and it is steroidal in nature.

Effect of ecdysone on metamorphosis

Ecdysone is the only hormone which has a central control on the developmental events at the cellular level in insects. When the prothoracic glands stop the secretion of ecdysone the result is developmental stand still. This is what happens during diapause. It must be mentioned here however that the development of muscles during metamorphosis is not governed by ecdysone alone. There is another factor called neurosecretory factor or a neurosecretory control (hormone) which when produced during the same period when ecdysone is released in the blood causes muscle development. This observation clearly points that there is a close integration of the prothoracic secretory process with the activity of the neurosecretory system. Further integration is through the neurosecretory control of the brain over the prothoracic gland.

(3) JUVENILE HORMONE

This is secreted by corpora allata, a paired or unpaired small cephalic endocrine glands. The hormone is known as juvenile hormone (JH), corpus allatum hormone or neotenin which prevents

the real change of stage in the developing insect. Wigglesworth (1934) was the first to demonstrate this function of the juvenile hormone in his experiments with *Rhodnius prolixus*. However other workers such as Bounhiol (1938) and Fukuda (1944) and several others have confirmed the observations of Wigglesworth by their experiments on caterpillars. The JH is terpenoid in chemical nature.

Role of Juvenile hormone in metamorphosis

Its chief function is to maintain the immature stages i.e. larvae or nymphs of insects without causing pupation or the development of adult stage i.e. the actual metamorphosis is blocked. In a normal metamorphosing insect the corpora allata decline its secretory activity and thus a sudden change from an immature young stage to adult happens. Removal of corpora allata in the juvenile period of growth in insects have clearly shown a precocious metamorphosis in the life history.

In short the process of metamorphosis takes place as given below:

The brain hormone secreted by the neurosecretory cells stimulates the prothoracic glands to secrete ecdysone or moulting hormone, but moulting occurs only when the juvenile hormone is either absent or present in very small amounts. In the last larval instar of Holometabola the corpora allata becomes almost inactive, hence a low concentration of JH is present causing larva to moult into a pupa. Similarly during later part of the pupa or late pupa, again the corpora allata become inactive and in total absence of JH the pupa moults into an adult insect. In Hemimetabolous insects also the same phenomena is repeated i.e. the corpora allata become inactive in the last instar nymphs and in the complete absence of JH imaginal ecdysis takes place. Thus metamorphosis in insects is controlled by a delicate balance in the timing of secretion and concentration of two hormones viz. JH and MH secreted by the corpora allata and ecdysial or prothoracic glands. The total absence of JH is the most significant factor for imaginal ecdysis in both, Hemimetabolous and Holometabolous insects.

If much JH is present then larva will moult into a larva, if small amount of JH is present then pupation takes place and in the total absence of JH adult emergence occurs. And if the balance of hormones is disturbed then monsters, intermediate between larva and pupa or