DISCUSSION
Chlorinated hydrocarbons are often referred to as 'organo-chlorines' and, collectively, they are a broad-spectrum and very persistent group of insecticides. They are being used as very effective insecticides both in the old world and in the Third world countries. Thus widespread use of chlorinated pesticides to control pest species creates ecological disturbances which, in turn, affect the non-target organisms. These hydrocarbons, through recirculation in the environment, reach the human beings, and their chronic usage brings about cellular disorders.

A good amount of information on the toxicities of pesticides on different aquatic insects is available. The histopathological effects of certain insecticides on different insects have been studied by Woke (1940) on the midgut wall of Prodenia eridania, Salkeld (1950) on the ventriculus of honeybee associated with the ingestion of certain insecticides; Chadbourne and Rainwater (1953) on the larval tissues of boll-worm, El-Deeb and Zeid (1961) on the various tissues of pink bollworm; Soliman et al. (1971) on the larvae of Drosophila melanogaster. Toxicity of three chlorinated hydrocarbons, viz., BHC, aldrin and
DDT has been studied in Hemiptera, *Dysdercus similis* Ahi (1984). Out of these three chlorinated hydrocarbons tested, aldrin was found to be the most toxic.

*Teoon* and *Cleveland* (1955), *Davis* and *Fitzhugh* (1962), *Fitzhugh et al*. (1964), *Nagasaki et al*. (1971), *Nagasaki et al*. (1972a and b), *Huff* (1980) and *Farber* (1980a) have studied toxicity as well as carcinogenicity of certain chlorinated hydrocarbons. These authors have suggested that certain chlorinated hydrocarbons are carcinogenic in mammals. They were concerned with tissue stress leading to carcinogenesis. Effects of suspected carcinogens have been studied in rat and mouse.

Here, in the present study, the effect of benzene hexachloride (BHC), aldrin and endosulfan has been noticed on the insect tissues in order to see if these insecticides have any tumorigenic effect, specially when *Davis* and *Fitzhugh* (1962) have reported hepatic tumors in mice by feeding both aldrin and dieldrin, and *Nagasaki et al*. (1971) have reported the development of hepatomas in mice treated with benzene hexachloride.

The insect selected for the present study is *Poekilocerus pictus* (Fabr.), and various tissues taken are
the midgut, the adipose tissue and the gonads. The various chemicals tried are benzene hexachloride, aldrin, endosulfan and benzidine. The first three are chlorinated hydrocarbons, and the last one is aromatic amine. The three were tested for chlorinated type and the fourth one, which is an aromatic amine, is a known established carcinogen, was tried to compare these results. Among the chlorinated hydrocarbons tried, aldrin, BHC and endosulfan were found to be very effective in the order of potency. The carcinogenic effect of benzidine has been shown in rats by Spitz et al. (1950), Vesselinovitch et al. (1975), and Morton et al. (1981). Maltoni and Gnetti (1964) have reported the formation of tumors in the upper urinary tract of workers in the dyestuff factory, exposed to benzidine.

No doubt, some usual secretions of exocrine and holocrine nature were seen in the midgut; still the tissue stress was noticed in the regenerative crypts, which abnormally increased, in size and number, the cellular epithelium, the nuclei, the musculature and the peritoneal membrane. A large number of projections were formed in the midgut lumen, which eventually broke free into the lumen. Similar effects were also noticed by Woke (1940), Chadbourne and Rainwater (1953), Soliman and Soliman (1958), and Soliman et al. (1971). Chadbourne and Rainwater (1953) did not notice very
significant changes in the nervous system of the larvae of bollworm. However, dieldrin caused most marked changes in the midgut. All epithelial cells of the midgut showed marked degeneration. Cell wall became distorted. The striated border was also destroyed. It also affected the peritrophic membrane. The basement membrane and the muscles showed less degeneration.

Here, in the present study, it was found that aldrin was very effective, and the details found for the cells were similar to those found by Chadbourne and Rainwater (1953) on the larvae of boll-worm. However, in the present study, the basement membrane, the circular and the longitudinal muscles and the peritoniun covering the gut, was greatly affected. It may be mentioned that Chadbourne and Rainwater have not commented on the observations on the peritoniun. Acutally, the haemocytes cling to the peritoniun and in many cases the peritoniun becomes thicker. The toxicity of endosulfan was the least; BHC was second, and aldrin like dieldrin was very effective. These effects were similar to those found by Soliman et al. (1971) on the larvae of Drosophila melanogaster.

As regards the effect of certain carcinogens on the midgut, the work of Cantwell et al. (1966), Sutherland (1969), and Baewald and Boush (1969) deserves special consideration.
Cantwell et al. (1966) fed the larvae of housefly with 2-fluorenamine derivatives and found that the area most affected was the midgut; both epithelial lining and the regenerative nidi appeared hypertrophic and hyperplastic. In the treated larvae, the nidi apparently increased, and the cells proliferated rapidly and increased in size. Large segments of cytoplasm were also seen in the lumen, apparently as a result of cytoplasmic shedding.

Sutherland (1969) worked on the nymphs and adults of Periplaneta americana by treating them with MCA, FL, and DPAA. He found that growths of abnormalities were not specific. Such growths were of wound-healing type, and growths of midgut also consisted of exfoliation of the epithelium. Regeneration of the epithelium was controlled by the encapsulating haemocytes, and the compounds were carcinogenic.

Similarly, Baerwald and Boush (1969) examined the gut by giving intracolomic injections of Benzo(a)pyrene to Periplaneta americana. The treated insects revealed abnormal cell responses after 15, 23 and 28 days of treatment. Such tumifications occurred in 6 males and 4 females out of 40 roaches examined. Mitotic figures were revealed in one response, 28 days after treatment. Abnormal cell responses
were associated with the midgut and hindgut. In addition, brown melanin-like material was seen typically encapsulated by haemocytes.

Here, in *P. pictus*, similar observations have been made as mentioned by Cantwell *et al.* (1966), Sutherland (1969) and Baerwald and Boush (1969). The cells of the regenerative nidi proliferated rapidly, and increased in size in insects treated with aldrin, BHC, endosulfan and benzidine. Cell exfoliation was also noticed in the gut lumen in aldrin-treated insects. It was also noticed that the epithelial cells appeared to be continuous with the mass of cells which were formed inside the gut lumen, implying that the abnormal cells so formed were of epithelial origin in aldrin-treated insects, as also shown by Baerwald and Boush (1969). Further, a 'brown coloured material' was noticed at the peritoneal surface of the gut which was encircled by haemocytes, in BHC treated insects. Regeneration of the gut epithelium can also be seen in BHC treated insects. These findings further get support from the work done by Stark (1935) on hereditary tumor of the gut of *Drosophila*. Bird (1949) working with virus infected European spruce sawflies, reported similar findings in the region of the regenerative nidi.

With reference to toxicants, the adipose tissue has
been studied by Chadbourne and Rainwater (1953) who studied the effect of DDT, calcium arsenate and dieldrin on the larval tissues of boll-worm. They showed that nuclei of the adipose tissue were clumped into dense masses. Soliman and Soliman (1958) studied the effect of DDT, parathion, toxaphene on *Prodenia litura* and Soliman *et al.* (1971) studied the effect of DDT, malathion and sevin on the larvae of *Drosophila melanogaster*. They have found shrinkage of the cytoplasm of the fat cells and distortion of the fat body structure.

Here, in the present findings, similar observations were made in the adipose tissue of *P. pictus*, when treated with aldrin, BHC and endosulfan as shown by Chadbourne and Rainwater (1953), Soliman and Soliman (1958), and Soliman *et al.* (1971). However, these authors did not show the formation of 'brown coloured bodies', their encapsulation by the haemocytes and their subsequent melanization as was observed in the present study of insects treated with aldrin and BHC and benzidine. Here, in the adipose tissue of *P. pictus* it was found in the case of aldrin and BHC treated insects that at first the haemocytes, then the peritoneal wall and thereafter changes in the fat body were evident. In later stages in aldrin
and benzidine-treated insects, the haemocytes were infiltra-
ted into the adipose tissue, thus making the fat cells
vacuolated. Melanized fat cells were also seen in BHC,
aldrin and benzidine-treated insects. As far as the
author is aware, such changes in the adipose tissue by
chlorinated hydrocarbons have not been reported by any
worker so far.

As regards the effect of certain carcinogens on the
adipose tissue, the work of Cantwell et al. (1966) deserves
special consideration. They reported that, as melanin
deposition took place, the fat cells became vacuolated,
and groups of fat cells in this condition appeared to
aggregate into a mass, and melanin appeared intercellularly
and concentrated at cell membrane. They also mentioned
that melanized fat cells were surrounded by haemocytes.
They called the melanized cells 'black bodies'.

Here, in P. pictus, similar phenomena have been noticed.
In benzidine treated insects, the adipose tissue is heavily
melanized, and the fat cells show vacuolization in some
cases. The melanized masses are also surrounded by spindle
shaped haemocytes. As the pigmentation continues, the
cellular structure of the adipose tissue becomes obliterated.
The haemocytes are infiltrated into the fat cells in later stages. These findings further get support from the work done by Wilson et al. (1955) who have described a similar condition in the fat body in the tu^W strain of D. melanogaster. They also stated that as the pigment continues to accumulate, the cellular structure becomes obliterated and the pseudotumor appears as an amorphous, friable, black mass. Evidently they found no fat cell multiplication in such melanotic masses. Schlumberger (1952), Shatoury (1955) and Kaplan (1956) described similar conditions in the fat body of other species of insects. Schlumberger placed methylcholanthrene or talc in the body cavity of Periplaneta americana. Both materials caused injury to the fat cells, and resulted in encapsulation of groups of these cells by haemocytes and in the production of large pigmented bodies. Further, Harshbarger and Moore (1966) X-irradiated the larvae of Galleria mellonella, and found similar changes in the fat cells as observed in P. picta. A whorl of spindle-shaped cells was seen to surround a melanized core which was embedded in the larval fat. The fat cells were highly vacuolated with reduced nuclei. Similarly, very recently, Nappi (1984), working on the haemocytic reactions during melanotic tumor formation in Drosophila melanogaster
suggests abnormal development in the adipose cells. They were highly vacuolated and hypolipidic, and the cytoplasmic vacuoles had also increased in number. Some of these adipose cells were dissociated from the adjacent cells and were seen in various stages of disintegration. These findings are similar to those found in the adipose tissue of benzidine treated *P. pictus*.

As regards the study on the testes and ovaries, tissue hyperactivity and cellular deformations were noticed. Although such studies have not been described so far, these observations can be compared with the observations of tissue stress as shown by the effect of chemosterilant on the reproductive organs of *Locusta* as reported by Vishwanath et al. (1976, 1978) and Mittal et al. (1978).

In the present study, the visible damage to the male gonads due to the effect of BHC, aldrin, endosulfan and benzidine appears to be degeneration of the germ cells and differentiating germ cells, loss of sperm motility (hypertrophied sperms) and also degeneration and resorption of spermatozoans. The testis follicles were surrounded by melanized cells in BHC and benzidine treated insects. In benzidine treated insects, the melanization was so severe that the testis follicles were seen depleted of their contents.
There has been some controversy in chemosterilant treated insects about when the development and the maturation of sperms are most severely affected. Some researchers LaBrecque and Fye (1978) opine that the sperms already formed at the time of treatment have the highest incidence of dominant lethal mutations. The gonial cells usually appear less susceptible, and there is eventual recovery of fertility. Others claim that the spermato-gonial zone is the most receptive and the damage is irreversible LaBrecque and Fye (1978). In the present study with chlorinated hydrocarbons and aromatic amine, the author accepts the later view that there is a progressive testicular degradation and re-sorption.

As regards the female gonads, the visible damage to the ovaries due to the effect of BHC, aldrin, endosulfan and benzidine is that vitellogenesis is arrested in all the chemicals tried. The yolk platelets, present prior to the treatment, become disintegrated and destroyed. In aldrin treated insects, abnormal fragmentation of the oocytes is noticed. The oocytes are also seen distorted in shape. However, in BHC treated insects, vacuolization of the ooplasm, division of the ooplasm into outer thin and inner thick ooplasm as well as formation of the multinucleate oocytes are the abnormal features observed. It can be concluded that the multinucleate condition of the oocyte
was due to amitosis. The action of BHC on the amitotic division of the nucleus of the oocyte suggests that BHC not only makes the haemocytes and the adipose tissue hyperactive and causes cellular deformations but also regulates the amitotic division of the oocyte nucleus.

As regards the effect of carcinogens, in the present study in benzidine-treated insects, the 'brown coloured bodies' were evident and also the oocytes were seen surrounded by melanised cells. As far as the author is aware, such studies regarding the effect of carcinogens on the ovary of insects have not been made so far. However, hereditary ovarian tumors in Drosophila have been reported by King and associates (1957, 1961, 1966, 1969, 1981 and 1984).

Tumors are of genetic origin as described in Drosophila. Pseudotumors which are not malignant have also been described in Drosophila by Barigozzi (1954, 1956, 1958, and 1964). These pseudotumors are found in the fat and the pericardial cells. Tumors are a result of abnormal cell proliferation from tissue growth. The cellular behaviour of tumor is inherited. So they were studied in vertebrates but not in detail in invertebrates till the researches of Scharrer and Lochhead (1959). Earlier it was thought that tumors cannot be found in invertebrates. It was for the first time that two French and a Russian
Russian scientist showed the formation of tumors in invertebrates, Thomas (1932), Codreanu (1939) and Finkelstein (1944). However Scharrer has criticized the data, as the work has been done by pathologists alone. Their physiological aspect has remained uncovered.

Scharrer suggested that tumor may be examined with careful methods, and may not be confused with other inflammatory tissue. Usually the cells surround the tissues which degenerate under stress conditions, and a cyst is formed. Sometimes hypertrophy of cells is noticed, and sometimes encapsulation of the parasites by the haemocytes forms tumors.

Stark (1918) was the first to study hereditary tumor in Drosophila. She contributed the finding that genetic factors bring about tumor formation. Stark described malignant tumors but the evidences produced were insufficient. Similarly Wilson (1924) studied Drosophila tumors dependent on multiple hereditary factors as were those studied by Stark.

Tumors were studied in detail by Kaplan (1955, 1956), Friedman and Burton (1956), Burton and Friedman (1956), Burton et al. (1956b), Sang and Burnet (1967) Harker (1958, 1963), Matz (1961, 1967a and b, 1975 and 1979), Harshbarger

Kaplan (1956) has described tumors in insects and suggested that the tumors of insects are similar to those of vertebrates except that they regress during metamorphosis. According to him, the normal biochemical processes are upset by some biochemical change, and the tumor process is initiated. In *D. melanogaster*, the pigmented cell tumors, called melanoma appear during larval period through gene control, and they can be regulated by hormones. The melanization occurs in the spindle cells and in the spherical cells.

Matz et al. extended Scharrer's work and worked on an orthopteran insect *L. migratoria* and showed that RNA factor was responsible for tumor formation (1966).

Harker (1963) has reviewed and described tumors due to different factors. She has also described benign tumors in *Drosophila*. They are genotypes. Malignant tumors or lethal tumors were described by her from the work of Russell (1940). The various factors which regulate tumor formation have been mentioned by her. Factors such as nerve severance, hormonal imbalance and carcinogens, have been mentioned.
The researches done here with aromatic amine can be compared to those described by her. In every case we find that the blood cells are rapidly implicated. In the present study, it has been found that BHC, aldrin, endosulfan and benzidine affect the haemocytes and produce flattened blood cells which can be compared to the lamellocytes of *Drosophila*.

Although the difficulties of classifying conditions of abnormal growth in insects are such as to discourage definitions concerning insect tumors, any concept of tumor implies multiplication of cells. In the case of the so-called benign tumors of *Drosophila*, the question must therefore be raised whether any multiplication of cells is involved. All the evidence suggests that benign or pseudo tumors are due to an aggregation of haemocytes either as a group or around specific tissues and that, in turn, stimulates the haemocytic reaction of melanization.

Many researchers have also not hesitated to describe cell responses as tumors, Harshbarger and Taylor (1968). Little progress has been made in insect oncology, due to various haemocyte reactions. Difficulties in interpreting possible neoplasms have resulted largely from misleading forms of haemocyte encapsulations. However, the studies of Salt (1956), Jones (1962) and Grimstone *et al.* (1967) form a sound basis for an understanding of the role of
haemocytes as a defence mechanism.

The haemocytes possibly encapsulate the phagocitized haemocytes and form small flat cells around them. These are called cell aggregates. This is comparable to the cell reaction to the haemocytes. Possibly the surface of the haemocytes changes. Similar studies have also been done by Salt (1970).

The haemocytic reactions and cellular changes during melanotic tumor formation have been demonstrated by Rizki (1957, 1960, 1962), Rizki and Rizki (1978, 1979) and Nappi (1984).

Although Nappi's work on D. melanogaster is on melanotic hereditable benign growths, still the behaviour of haemocytes in P. pictus shows similar stages. The haemocytes adhere to some phagocytized haemocytes and get flattened and form cellular capsules, and such capsules with aggregates of haemocytes were observed on the surface of adipose tissue and other tissues. In the present observation the haemocytes were also found infiltrated into the adipose tissue at a later stage in aldrin and benzidine treated insects, as found by Nappi (1984).

The encapsulation process as described by Rizki (1979) is similar to that found in P. pictus. The encapsulation
process follows the initial period of phagocytic activity by the haemocytes. Layering of haemocyte upon haemocyte continues until a laminated capsular wall surrounds the entire area of afflicted adipose cells. The fully formed melanized capsule has a covering of flat cells. The disturbances at the adipose cell surfaces may serve as the direct stimulus for the encapsulation response as well as the phagocytic reaction. Rizki and Rizki (1973) suggest that the stimulus for encapsulation process by the lamellocytes is provided by the activity of the phagocytes establishing first contact with the adipose cells, since some of these haemocytes appear to exude material over the substrate.

In the present work, it appears that the haemocytes get affected and form a defensive covering over the tissues. This response is possibly a hormonal response and in this process, although Ashhurst (1968) has suggested that haemocytes play a minor role in the formation of connective tissue. But in the present study of the so-called tumors of *P. pictus*, it has become abundantly clear that haemocytes definitely cling to and cover the connective tissue sheath in the midgut, in the fat body, in the testis and in the ovary. This tissue is protected by some factor secreted by the
haemocytes on one side and by the formation of capsules on the other side. It may be true that connective tissue is not a product of the haemocytes but certainly some biochemical products are definitely secreted by the haemocytes to contribute towards the thickening of the connective tissue layer. As Wigglesworth (1956) has suggested, there may be neutral acid mucopolysaccharide or other substances contributing to the formation of connective tissue in insects.

These studies were undertaken with a view to explore the formation of cellular abnormalities and tumor formation in insects with reference to the action of chlorinated hydrocarbons and aromatic amine taken as a carcinogenic agent. The intention was also to find the role of haemocytes and their regulation in tumor biology, and also possibly the hormonal role in controlling the melanization and tumor formation.

Tumors, as such in insects appear to be the melanotic tumors of *Drosophila* which are hereditable tumors, such as described by Stark (1918), Burdette (1950, 1959) Offedal (1953) Kaplan (1955), Burton (1956), Burton et al. (1956b), Barigozzi (1958, 1964), Barigozzi et al. (1958), Kanehisa and Fujita (1960) Burnet and Sang (1964), Gateff (1978, 1981)
and others. Other tumors which are described by Shortino et al. (1963), Cantwell et al. (1966), Matz (1967), Sutherland (1969b), Harshbarger (1974) and others, can be said to be tumors which regress during different experimental conditions but are not melanogenetic. No doubt, melanotic tumors through nerve severance have been described by Scharrer (1945, 1949, 1953), Harker (1958), Hema (1966, 1967), Sutherland (1967) and Matz (1967, 1979). In tumors which appear due to nerve severance, the cells have some nucleic acid factor or viral factors which are perhaps induced to form tumors in insects. These factors could be injected into other healthy hosts and can induce tumors, Matz et al. (1966) and Matz and Bergoin (1984).

Here, in the present studies, the tumors, the melanin growths appearing during the treatment with different chemicals suggest that there is no mitotic division in the tissue, what-so-ever is there is definite stress on the tissues. The tissues become hypertrophic. Some haemocytes and tissues degenerate and in order to protect them, these haemocytes get phagocitized and form melanin, and form different types of tumors of different origin, shape and constitution in the tissues and over the tissues. The action of chronic, chlorinated hydrocarbons definitely creates stress on the tissues and the tissues, specially the ovary, start amitotic division. Possibly such amitotic division in the
haemocytes as mentioned by Sutherland (1971) takes place and this physiology controls tissue damage. The cell surface is definitely regulated by hormones specially when we find that a large number of tumors are not found in the nymphs which have high titer of ecdysteroids and also in the experimental adults in which a high titer of ecdysone and juvénoid was maintained. This conclusion gets support from the work of Burdette (1954a and b), Rizki (1957) and Burdette (1960).

The role of moulting hormones on insect tissues, has been studied by Gilbert et al. (1980). The dynamics of ecdysone secretion was worked out by Ohtaki et al. (1968), and ecdysone metabolism by King (1972), but the actual effect of ecdysone on tumours was studied by Burdette (1960) and Hirono et al. (1969). The effect of ecdysone on insect tissues and tumors was studied by Burdette (1954a and b, 1960) Gvozdev, et al. (1973) and Courageon (1975).

Burdette (1954) reported that melanotic tumors in Drosophila do not persist in pupal and imaginal stages but regress and remain as inert pigmented masses. Since metamorphosis in Diptera is controlled by hormones from the ring gland, it is possible that the action of these hormones may be responsible for the regression of tumors as well as other larval tissues. These hormones were injected into
mice and tried on mammalian tumors. This did not have effect on the tumors or cause regression in size of the tumors, but gave a longer survival time. However, this research was not confirmed in detail. And it cannot be said that the evidence produced is completely against the possibility that larval hormone may alter mammalian tumors. It needs further confirmation.

Grozdev et al. (1973) studied the effect of B-ecdysone on D. melanogaster. The effect of B-ecdysone was studied at cellular level. B-ecdysone causes efficient inhibition of cell proliferation. The inhibition of cell growth by B-ecdysone may be reversed after 24 hours of treatment but it is irreversible after 48 hours.

Courgeon (1975) tried B-ecdysone on D. melanogaster. She found that the cells responded with characteristic morphological modifications accompanied by an arrest of cell multiplication. Some cells only stopped dividing, others reacted with immediate death of cells, while some cells were found to be resistant to ecdysone.

O'Farrell and Stock (1964) studied the effect of Farnesyl Methyl Ether which possesses juvenile hormone activity on regeneration.
Bryant and Sang (1969) reported that JH and juvenile hormone mimic FME, and their derivatives are effective in inducing tumor expression and many of them reduce tumor frequency.

In the present studies, the author has tried ecdysone and JH analogue on the benzidine treated adult *P. pictus*, and found that although the formation of 'brown coloured bodies' could not be controlled in the testis (while it was controlled in the ovary and adipose tissue), the cellular damage was comparatively less than that in only benzidine treated insects.

One point which was of interest was that, these hormones gave a longer survival time to the experimental insects. In the case of ecdysone treated insects, the survival of females was increased by 15 days while in males the survival was increased by 8 days. In juvenoid treated insects, the survival was increased by 4 days in females while the males died after 16 days.

Although the conclusions here drawn do not directly imply that these hormones control pigmentation in the disorganised cells, they definitely suggest that the vulnerability of the cell is definitely saved and the insects' survival in ecdysone and juvenoid treated milieu provides a longer survival time to the experimental
insects, which was also noticed by Burdette (1954a and b), Rizki (1957) and Burdette (1960).

These findings are also supported by the fact that the nymphs, when treated with similar doses of benzidine, also survived for a longer period (that is, 18 days) than the adult treated insects which survived for 16 days.

In all probability, the ecdysone regulates the cellular disintegration and tissue hypertrophy, and can be effective to the extent that a smaller number of melanized tumors are formed.

The haemolymph proteins of normal insects have been studied by a number of workers. However, the haemolymph proteins of orthopteran insects have been studied by Kulkarni and Mehrotra (1970) in *Schistocerca gregaria* and by Gupta (1975) in *Poekilocerus pictus*. As regards the stress proteins, it has been found in the experiments done that they do not appear de novo in the haemolymph of *P. pictus*, but those proteins which are present get depleted. Very likely they are consumed towards the biochemical process which is involved in the cellular tissue defence.

As regards the factors of tumor formation, no doubt various factors have been suggested by Harker (1963) and
by Nappi (1984). Nappi has suggested that the factor comes out from the adipose tissue which induces capsule formation in the haemocytes. Here in P. pictus this factor appears to be released from the phagocytized haemocytes or from affected endocrines which should be explored.

However, it can be concluded that chlorinated hydrocarbons, no doubt, cause cellular deformations, irritation and stress; and this leads to abnormalities and amitotic division in the insect tissues, but the insect tissues have definitely some physiological mechanism present in the haemocytes which controls the formation of melanotic tumors through other carcinogenic materials. The so-called carcinogenic tumors produced by regulators have to be reconsidered except those which are of genetic origin or which have been induced by nerve severance.