REVIEW OF LITERATURE

Despite the fact that the nature and properties of water contaminants have been studied for more than a century, not much progress was made till the mid-twentieth century when it became known that toxic effluents and waste waters could create serious pollution problems in industrialized and urbanized areas. Shelford (1917) succeeded in finding suitable methods to minimise the deleterious effects of gas wastes on the fish fauna of the streams. Weibe (1927) made a survey of the Upper Mississippi with reference to pollution and found that oxygen content could be greatly reduced in highly polluted zones of the stream. Carpenter (1924, 1925, 1926) described the role of mining wastes, particularly the heavy metals, in destroying riverine fisheries due to respiratory distress and asphyxiation brought on by heavy metal ions on their reacting with some constituents of mucus secreted by the gills. Ellis (1937) confirmed the findings of Carpenter and observed that acid and other chemicals such as trinitrophenol were fatal to the fish. Dilling and his associates (1926), Jones (1936, 1938, 1940 and 1950) and Doudoroff and Katz (1953) also reported the presence of a large number of inorganic and organic salts of heavy metals in various tissues of fishes while Pearsall and his co-workers (1946) found that copper salts from mine effluents and trade wastes could be greatly hazardous to fish. Doudoroff
and Katz (1953) while reviewing these findings of various workers came to the conclusion that the limiting concentration for different species of fishes given by various authors covered the surprising range of 0.02 to 200 ppm. Copper sulphate concentrations below 0.025 ppm as Cu were not rapidly fatal for most of the common fishes. Katz and Caufin (1952), Alderdice and Brett (1957) studied the effects of sewage and kraft mill wastes on the population dynamics of fishes and found that no fish could be seen in the polluted area where effluents were released but they started appearing as the effluents became diluted downstream.

It has generally been observed that human settlements and industrial establishments along the rivers have adverse effects on fish life. Such activities along the rivers, Mersey and Irwell resulted in virtual elimination of all fish life from English streams (Klein, 1962). Hopkins, Colteoff and McMillin (1931) found that the reproduction rate of oysters declined when a sulphite process pulp and paper mill was installed in the vicinity of the York river.

Our knowledge concerning the environmental pollution of Indian waters is very meagre, and practically nothing has been done to analyse the situation. The first note appeared in 'Current Science' in 1936 on problems of water contamination. Then in 1942, Hora made a brief reference to the contamination of streams in India and its effect on fisheries. Later in 1944, Hora and Nair
pointed out the dangers to fish life from industrial wastes of Mungpoo, Darjeeling. Bose (1944), Bhimachar and David (1946), Bhaskaran (1947), Ganapati and Chacko (1951) and Saxena et al (1966) stressed the need to carry out extensive studies on river pollution due to industrial effluents. Motwani and his associates (1956) made observations on pollution in the river Sone which was caused by toxic effluents arising from Rohtas Industries in Dalmianagar, Bihar, and pointed out that industrial wastes falling in the rivers threatened the very existence of aquatic biota. Their observations revealed that industrial wastes could effect the fish by adversely changing the physical and chemical nature of the river water. The changes caused could be a depletion of oxygen concentration to lethal level, formation of a blanket of suspended organic and inorganic matter on the river bed which could limit the existence of the fish food organisms at the bottom. Qasim and Rehan (1960) and later George, Qasim and Qayyum (1966) determined the extent of pollution in Kalinadi at Bulandshahr and observed a high fish mortality due to effluents from sugar mills. Earlier Banerjee and Motwani (1960) found that wastes from sugar factory could greatly alter the physical, chemical and biological characteristics of stream water so as to make it unfit for the survival of fish. David and Ray (1960) studied tannery and textile waste pollution in the river Ganga at Kanpur and found that an increase in temperature of the water, formation of large amount of foam, gelatinous-like precipitate, opaqueness of water,
production of hydrogen sulphide, high pH and depleted dissolved oxygen could be the factors responsible for mortality of the fishes in the river. Recently Ghosh, Ray and Gopalakrishnan (1973) have studied in details the nature of industrial pollution in the Hooghly estuary where about 95 different types of factories are situated. It was found that effluents discharged by distillery, yeast, cotton, textile, tannery and pulp and paper mills together with highly acidic and toxic wastes from rayon textile adversely affected the fishery resources.

The history of water contamination by pesticides begins with the indiscriminate use of these compounds for the control of insects and other pests of agricultural and public health importance. The insecticides available at the turn of the present century were mostly arsenic compounds, lime-sulphur, petroleum oils and nicotine. Between World War I and World War II fluorine compounds such as sodium fluoride, zinc fluoride, barium fluoride, lead fluoride, calcium and magnesium fluoride, sodium and potassium fluosilicates, pyrethrum and derris were added. Also a number of organic compounds such as thiocyanates and dinitro-compounds and vegetable and animal oil derivatives made their appearance. During this time the possibility that pesticides might be responsible for water contamination seemed rather remote though a few papers concerning the toxicity of the insecticides to fish did appear. Surber (1932) showed that sodium arsenite could be
safely used at a concentration of 1.743 ppm to control vegetation in fish ponds without any apparent harm to the fish. Brown (1951) stated that sodium arsenite was non-toxic to fish at 4.0 ppm, and a study by Alderdice and Brett (1957) gave 48 hour TLm for young chum salmon as 11.0 ppm $\text{As}_2\text{O}_3$. Grindley (1946) showed that limiting concentration of dinitro-phenol for minnow was 30.0 ppm. Of the older insecticides, derris remained the most toxic pesticide to fish and 0.027 ppm of the material could kill gold fish, *Carassius auratus* in 6 hours (Brown, 1951).

During and after the Second World War, DDT was used extensively as an insecticide until it was discovered that it was injurious to fish and other aquatic life. One of the first to note this fact was Pielou (1946) who observed the effects of DDT on *kafue bream*, *Tilapia kafuensis* in Rhodesia. By 1950 other chlorinated hydrocarbons such as toxaphene, aldrin, lindane and BHC (hexachlorocyclohexane) were available for pest control and proved to be equally toxic to the fishes. When their use was added to that of DDT, run off from regular agricultural operations caused fish kills in 14 tributaries of the Tennessee River. Studies conducted by Lawrence (1950) showed that 0.05 ppm toxaphene, 0.15 ppm DDT and 0.2 ppm chlordane caused 100.0% mortality of the bluegills and large mouth bass, but BHC at concentration of 0.01 ppm could kill only 50% of these fishes. It was further observed that in eastern ponds, toxaphene was effective only at a higher concentration of 0.2 ppm and that its effectiveness lasted for more than 10 weeks. According to Surber (1946) the effective
does for the eradication of fish population by toxaphene was 0.25 ppm. He found chlordane to be more toxic to fish. Applications of 0.45 ppm of gamma-BHC did not result in any significant decrease in the population of the fishes. The high toxicity of toxaphene to fish was confirmed by Hampill (1954) who tried it as a fish poison, and found that 0.1 ppm applied to lakes as dust or as solution completely eliminated carps in 72 hours. Stringer and Moomyn (1956) also treated eight lakes in British Columbia with 0.01 to 0.1 ppm of toxaphene and obtained a very high mortality of the fish in 120 hours. They further found that the compound showed an extremely high degree of persistence so much so that even after nine months of treatments there were no survivals in the lakes. Toxaphene has been found to be more toxic than rotenone. Pimentel (1971) recorded that 96 hour LC$_{50}$ for toxaphene was only 3.5 ppb as compared to the 48 hour LC$_{50}$ of 22.0 ppb recorded for rotenone in the case of Leptosia macrochirius. Mehrle and Mayer (1975) found that the fry of fathead minnow and brook trout when exposed to toxaphene developed 'weakness in the backbone' at a concentration of 50.0 ppt. A slight decrease in the growth rate was also noticeable.

Of the organochlorines, endrin seems to be the most toxic insecticide to fish (Henderson, Pickering and Tarawell, 1959, Katz and Chadwick, 1961). Katz and Chadwick (1961) determined the 24, 48, 72 and 96 hour TIm of endrin to Leptosia macrochirius, Salmo gairdneri, Oncorhynchus tohoventcha, O. kesutch and Gambusia affinis at 20.0°C. It was found that O. kesutch was
most sensitive with a 96 hour Tlm of 0.27 ppb endrin. L. macrochirus and C. affinis were more tolerant with a 96 hour Tlm of 0.6 and 0.76 ppb respectively. Iyatomi et al. (1958) while studying the effects of endrin toxicity to Cypinus carpio and Carassius auratus recorded the median tolerance limits as 0.005 and 0.002 ppm respectively at an interval of 48 hour and at a temperature ranging from 27.0° to 28.0°C. These workers further reported that the eggs of Cypinus carpio and Channa argus were far more tolerant to endrin than the larvae while the larvae were more resistant to endrin than the adults. Katz and Chadwick (1961) observed no apparent effect of endrin poisoning on early development of the eggs of Ctenopharyngodon idella. Similarly no pronounced effect could be found on the hatching of the eggs of threespine stickleback and the embryos were more tolerant to endrin than the fully developed fish.

Smith and Cole (1973) observed frequent failure of gastrulation in the embryos and severe vertebral deformities in the larval fry hatched from eggs of winter flounder which had been exposed to 2.0 ppb DDT but they could not find any deformities after the exposure of the fishes to even 2.0 ppm of dieldrin. Earlier, Allison et al. (1963, 1964) observed that continuous exposure of the female trout to high concentration of DDT did not decrease the number of ova nor was the embryonic development affected. The young fry however, showed significant
mortality compared to this. Kacek (1966) found that at sublethal doses DDT when administered through the diet of yearlings of brook trout resulted in their laying fewer eggs. The rate of mortality of the hatched fry was also quite high.

Endrin is a highly toxic organochlorine to marine and estuarine fishes (Korn and Earnest, 1974). Eisler (1970) compared the toxicity of organochlorine and organophosphorus insecticides to *Menidia menidia*, *Thalassoma bifasciatum*, *Mugil cephalus*, *Rundulus majoria*, *Anquilla rostrata*, *Rundulus heteroclitus* and *Sphaeroides maculatus* and recorded 96 hour TLM values. The results obtained showed that *M. menidia* as the most susceptible species while *S. maculatus* was the most tolerant to the toxicant. Korn and Earnest (1974) conducted experiments to determine the toxicity of nine organochlorine insecticides on marine striped bass, *Morone saxatilis* and found endrin to be the most toxic with 96 hour LC50 values as 0.094 ppb followed by DDT and dieldrin.

The 48 hour TLM of endrin for *Clarias batrachus* formulated by Bhattacharya et al. (1975) was 0.005 ppm. It was found that in test solutions the fishes became hypersensitive and showed a rapid rate of opercular movements. They lost their equilibrium after sometime and remained vertically suspended with no movements until death.

Tarnwell and Henderson (1957) determined the median tolerance limit of *Pimephales promelas* at 96 hour time interval as 0.023 to 0.036 ppm of dieldrin. They found dieldrin to be more toxic in hard water than in soft water. Harrington and Bilingmayer (1958)
observed complete kill of the fish and crustacean population by dieldrin at concentrations varying from 0.13 to 0.4 ppm.

Webbe and Shute (1960) observed that even very low concentration of DDT, gamma-BHC, aldrin and dieldrin could cause considerable mortality of Tilapia. Konar (1970) conducted bioassays at laboratory temperature varying from 19.0°C to 24.0°C and determined 168 hour LC30 and LC100 values of heptachlor for Labeo Rohita as 0.0166 and 0.02 ppm respectively.

Arora and his associates (1971) conducted experiments to determine the toxicity of endosulfan to Puntius sophore and recorded TLM values of 0.0023 and 0.0014 mg/l respectively for 24 hours and 48 hours. The same workers in 1972 found that 24 and 48 hour TLM thiodon for Cirrhinus mrigala was 0.00255 and 0.0016 mg/l Myloplus vittatus was, however, more susceptible to endosulfan than Puntius sophore (Reddy and Coworthy, 1977). Sub-lethal concentration of thiodon also depressed the oxygen consumption by about 46.0% while lethal concentration increased the respiratory metabolism and an increase of about 46.0% in oxygen consumption (Arora et al., 1972).

Chandhari (1975) determined the toxicity of the organochlorine insecticides, aldrin, dieldrin and endrin to twelve species of carp, sushels, top minnows and a species of prawn, Palaemon jamaica and found that these pesticides were highly toxic to the fishes as well as the prawns. Among the three insecticides tested, endrin was found to be the most toxic and the aldrin the least
toxic insecticide. Panwar et al. (1976) while testing the toxicity of DDT, BHC and endrin on *Trichogaster fasciatus* found endrin as the most toxic substance, the TLM values for 24 and 48 hour being 0.046 and 0.041 ppm respectively.

Recently Verma et al. (1979) while conducting acute toxicity tests with endosulfan, heptachlor, chloradane, aldrin, lindane and BHC on a fresh water teleost, *Saccobranchus fossilis* observed that endosulfan was most toxic and BHC the least toxic compound to this fish. Ratnakar et al. (1979) conducted experiments on aldrin toxicity to *Cynthus carpio* and found that aldrin was deadly poisonous even at a very low concentration of 0.005 ppm. They also studied the behaviour of the fish in toxic solutions and observed that the fish exhibited an initial period of high excitability followed by muscular spasms causing jerky and violent movements, excessive secretion of mucus by body surface and an increased rate of respiration.

Henderson, Pickering and Tarrwell (1959, 1960) and Pickering et al. (1962) while studying the comparative toxicity of organochlorine and organophosphorus insecticides to fresh water fishes *Notropis macrochirus*, *Carassius auratus*, and *Lebistes reticulatus* showed that organophosphates were less toxic to the fish than the chlorinated hydrocarbon compounds. This could be due to their inferior stability in the aquatic ecosystem.

A detailed study of the toxicity of thirteen organophosphorus insecticides to four species of fishes was conducted by Pickering,
Henderson and Lamale (1962). They observed an extremely wide range in toxicity with 96 hour LC values ranging from 0.005 to 610.0 ppm. *Lepomis macrochirus* were the most sensitive of all the fishes followed by *Lebistes reticulatus*, *Pimelophus promelas* and *Carassius auratus*. They also found that larger *L. macrochirus* were slightly more tolerant than the smaller ones to delnav, parathion and malathion. Green sunfish of similar size and large-mouth bass were almost as susceptible to the pesticides as *L. macrochirus*.

Allabaster (1969) determined the toxicity of some organophosphorus insecticides to the harlequin fish, *Rasbora heteromorpha* and determined 48 hour LC values. He found azinphosmethyl to be the most toxic material followed by parathion, carbophenothon and diazinon. He also found that chlorfenvinphos, formothion, ethion and bromophos were considerably toxic to *R. heteromorpha*. Macalister (1970) studied the comparative susceptibility of fishes belonging to several families to insecticides by plotting 96 hour LC values and found salmonids to be the most susceptible to organophosphate compounds. Ictalurids and cyprinids were the most resistant groups. Contrarchids occupied an intermediate position. Korn and Blanc (1974) studied the toxicity of seven organophosphorus insecticides to *Morone saxatilis* and formulated 96 hour EC values. Tanwar et al. (1976) while testing the toxicity of ethyl parathion and malathion to
**Trichocaster fasciatus** found that malathion was less toxic than ethyl parathion. Verma *et al* (1979) found solone to be highly toxic to *Sacchobranchus fossilis*.

The toxicity of carbaryl to fresh water fish was studied by Haynes (1959) who found the chemical to be considerably toxic to *Carassius auratus*. Henderson, Pickering and Turnbull (1959) reported that *Tm* values of sevin for *Iremia macrochirus* in soft water was 5.3 mg/l active ingredient. Bhatia (1971) while conducting toxicity tests for carbaryl to *Puntius ticto* recorded 24, 48, and 72 hour *Tm* values as 4.2, 3.9 and 3.6 mg/l respectively. Fimenteri (1971) determined the toxicity of carbaryl to *Salmo gairdneri* and recorded 48 hour *IC*\(_{50}\) values as 1,350 ppb. Post and Schroeder (1971) determined 96 hour *IC*\(_{50}\) values of carbaryl to certain salmonoids and three spine sticklebacks and recorded 1,3000, 1,470, and 3,990 ppb respectively for *Uncorhynchus ketaich*, *Salmo gairdneri*, and *Gasterosteus aculeatus*. Kom and Zamest (1974) observed 96 hour *IC*\(_{50}\) values of carbaryl to *Morone saxatilis* as 1,000 ppb while Verma *et al* (1979) found carbofuran to be highly toxic to *Sacchobranchus fossilis*.

Toor and Kaur (1974) studied the toxicity of carbaryl to *Cyprinus carpio* and found much excitation and decreased rate of respiration in the fish when introduced into the toxic solutions. The gills turned light brown and precipitation of mucous on the gill lamellae was quite perceptible.
That the volume of contaminated water available per fish may have a bearing on the mortality of the fish was pointed out by Prevost and his associates (1946) who found that the fish died more rapidly in larger volumes of the toxicant solutions than in smaller volumes. Katz and Chadwick (1961) repeated the experiment with slight modifications. Instead of varying the volume of toxicant solution, the number of fish exposed to the toxicant solution was different. The 72 hour Td for *Lepomis macrochirpus* when 5 and 25 fishes were used in 15 litres of toxicant was 0.95 and 2.7 ppb endrin respectively.

The biological effects of temperature are well known. It also influences the toxicology of pesticides. Johnson (1968) failed to find any definite correlation between temperature and the toxicity of pesticides to fishes. While higher temperatures increased insecticidal toxicity in some cases, the reverse was the case in other experiments. He attributed this difference to the metabolism of the pesticide concerned and the test organism. Katz and Chadwick (1961) and Iyatomi et al. (1968) observed that high temperatures decreased DDT toxicity, but increased the toxicity of endrin. At ambient temperatures between 1.0 to 4.5°C, the estimated 96 hour Td for 0.25 ppb of endrin, whereas at 25.0°C the 96 hour Td was 0.33 ppb of endrin indicating a 25 fold increase in toxicity. They further found that as the experimental temperature increased there was a gradual decrease in 96 hour Td. Cope (1965) claimed that the action of DDT in fishes has a
negative temperature coefficient being more toxic to *Salmo gairdneri* and *Leuciscus idionchirus* at 13.0°C than at 19.0 or 23.0°C. Ogilvie and Anderson (1965) reported that the fishes avoided temperature varying between 1.0° and 5.0°C when they were exposed to sublethal concentrations of DDT.

Eisler (1970) reported an increased DDVP sensitivity in *Fundulus heteroclitus* at higher temperatures. He found interesting relationship of DDVP toxicity to temperature in that the percent mortality in 96 hour, using 3.5 ppb DDVP, was 10.0% at 10.0°C, 35.0% at 15.0°C, 60.0% at 20.0°C and 15.0% at 30.0°C. Macek et al. (1969) found a greater effect of temperature on the toxicity of several organochlorine insecticides tested over a range of 1.0°-12.7°C against *Salmo gairdneri* and suggested that increased toxicity associated with increased temperature was due to an increased rate of take up of the toxicant. They, however, found little effect of temperature on heptachlor toxicity to *Salmo gairdneri*. Bridges (1965) found heptachlor to be 5 times as toxic at 24.0°C as at 7.0°C in rear sunfish. Dieldrin has been reported by Wade (1969) to be twice as toxic to *Percilia lethrinna* at 3.61°C than at 19.4°C, but temperature had only a slight effect on dieldrin toxicity to *Cyprinodon variegatus*. Murphy and Murphy (1971) found a direct relationship between the rate of oxygen consumption and that of DDT uptake from water in *Gambusia affinis*. It was found that the rate of such uptake increased
with an increase in temperature.

Most of the chlorinated hydrocarbons are known to cause nervous system problems, instability, respiratory difficulties and sluggishness in fishes (Johnson, 1966). A variety of histopathological changes have been reported as a result of the bioconcentration of insecticides. Eller (1971) reported that chronic exposure of cutthroat trout to endrin resulted in pathologic lesions in the gills, liver, pancreas, brain and gonads. Endrin has been found to be more toxic at higher temperatures and such a thermal effect might be due to potentiated gill damage resulting from hyperventilation bringing more of the irritant in contact with the sensitive tissue. Walker (1963) confirmed this hypothesis when he found that an elevated temperature could cause more pronounced histopathological lesions in the gills of fishes subjected to endothal pressure.

Temperature has been found to influence organophosphate toxicity also. Sizler (1970) found increased mortality of higher temperatures in Fundulus when it was exposed to methyl parathion. Weins (1959) earlier reported that the time course of thermal acclimation may prove a critical factor with organophosphorus compounds on these chemicals inhibit acetylcholinesterase in nervous tissue and this enzyme undergoes notable changes during thermal acclimation (Balsow and Nigrelli, 1964). In addition to acetylcholinesterase inhibition, histological damage to liver,
gills, muscular tissue and heart have also been reported by Matton and Lattam (1969).

Insecticides reaching the aquatic ecosystem greatly affect the physiology of fishes (Johnson, 1966; Katz et al., 1969; Anderson et al., 1971). Liver and kidney are organs that seem to be most affected by these toxicants (Brown, 1969). King (1962) studied the histopathological changes in the liver, kidney and intestine of \textit{Lepisosteus reticulatus} which had been exposed to sub-lethal concentrations of DDT. Nuclear displacement and vacuolation was apparent in the case of the cells exposed to the chemical and injury to the epithelial cells and mucous membrane was evident. Lillie and his associates (1947), Sarrett and Jandorf (1947) and Durham et al. (1963) found that DDT and related compounds could produce hypertrophy of hepatic cells, liver cord disarray, necrosis of cells, fatty liver and hepatoma in fishes while Mount and Putnicki (1966) could found a large number of vacuolated cells in kidney due to endrin poisoning. Rudd and Genelly (1956) found that DDT produced chronic nephritics in \textit{Carassius auratus}. Tracy et al. (1965) gave an account of the enlargement of liver and kidney and necrosis of convoluted tubules in rats, whereas Holmberg et al. (1972) reported impairment of liver and its functioning on the case of \textit{anguilla anguilla} when exposed to pentachlorophenol. The studies of Ronar (1970) on the liver lesions in \textit{Labeo robins} fingerlings induced by heptachlor revealed
vacuolation in the hepatic cells which became ruptured and swollen and showed surface irregularities. The nuclei were either placed eccentrically or extruded out from the cells.

Mathur (1962, 1965, 1972, 1976) observed histopathological changes in the liver of certain freshwater teleosts even at low dosage of 10.0 ppm of RlC, 15.0 ppm of lindane, 5.0 ppm of DDT and dieldrin. At higher concentrations the effects were more pronounced. The changes occurred predominately in liver where deleterious effects such as nuclear degeneration, cytoplasmic vacuolation and necrosis of hepatic cells were clearly visible. In kidneys a distinct degeneration of the epithelium could be noticed and there was a considerable loss of parenchymatous cells of renal tubules in *Ophioccephalus punctatus*. Mathur and Rana (1979) studied the histopathological changes in the liver of *Rana cyanophlyctis* as a result of endrin poisoning. It was noticed that central lobular hy.ercytosis, necrosis and margination of cells in the liver was common. They also observed displacement of nuclei from their original positions. The blood sinus and bile capillaries were also damaged.

Bhattacharya and his associates (1975) have reported swollen liver cells, liver corn disarray, surface irregularities, rupture and vacuolation in the liver cells of *Clarias batrachus* due to endrin poisoning. Binucleated hepatic cells were produced in abundance in the liver of the fish. The pancreatic acinar
cells lost their usual pyramidal shape and the nucleus remained extruding out of the cells. Recently, Ratnakar and his associates (1979) noticed that aldrin could induce histological changes in the liver, stomach and gills of *Cyprinus carpio*. In the liver they observed vacuolation in hepatic cells with small and large vacuoles, changed positions of nuclei and atrophy of cells. At higher concentration degeneration of the liver cells and displacement of nucleus was much more pronounced. There was degeneration of epithelial lining of the glandular epithelial cells of the stomach. Ruptured mucus membrane, vacuoles in longitudinal and circular muscles could also be seen. At higher concentrations there was disappearance of the glandular epithelium. Similar histopathological changes have been reported in the liver and gills of *Cyprinus carpio* by Grischenko in 1972. Skidmore and Tovell (1972) and Kendall (1968) have also observed epithelial sloughing of the gill lamellae of fishes as a result of poisoning by heavy metals.

The persistence and low water solubility of the pesticides contribute to their greater concentrations in fish tissue and in their food. Johnson and law (1970) pointed out that much of the intraspecific variation in accumulation and elimination of a pesticide may be related to the lipid contents of the fish. They pointed out that organochlorine residues accumulate in aquatic biota as a result of their low solubility in water and
high solubility in fats. Murphy (1971) experimenting with *C. affinis* found that its could remove four times more 14-C labelled DDT from water than a large fish and this showed circumstantially the importance that branchial route could assume. Holden (1962) and Ferguson *et al.* (1966) pointed out that the route of entry of a pesticide in fish was through the gills into the blood vascular system, while Verma *et al.* (1974) reported that gill and blood vessels in the mucous lining of the mouth were the main points of entry of a pesticide into the body of the fish. Earnest and Benville (1971) found that there was great correlation between lipid content of the fish and the accumulation of DDT residues. Johnson and Law (1970) reported greater accumulation of DDT residue in liver than in the kidney, but recorded maximum accumulation in the muscles of *Cyprinus carpio*. Verma *et al.* (1976) observed maximum accumulation of aldrin and ethyl parathion residues in the liver of *Colisa fasciatus* and *Notopterus notopterus*. These workers also found that aldrin accumulation was comparatively greater than that of ethyl parathion. Mathur (1964) determined DDT and BHC residues in the liver and intestine of *Barbus stigma* and observed a greater accumulation of these pesticides in the intestine than in the liver.