CHAPTER - V

HISTOPATHOLOGICAL CHANGES INDUCED BY PESTICIDES ON THE ASSOCIATED GLANDS OF ALIMENTARY CANAL AND SPLEEN OF RATTUS RATTUS ALBINO
NORMAL HISTOLOGY OF PANCREAS

The pancreas is an exocrine and endocrine gland where the exocrine part is formed by numerous irregular lobules having large number of acini. The acini are polyhedral in shape and consists of few pancreatic cells with prominent nucleus and granular cytoplasm. The lobules are separated by intralobular septa formed of connective tissue. The intralobular area contains pancreatic duct and blood vessels. The endocrine part in formed of the islet of Langerhans situated in between certain oobules. There were no structural changes in the pancreas of simultaneous controls (Fig. 81).

EFFECT OF DDT

After four weeks of administration, pancreatic acini showed vacuolation, space formation and fusion. Some of the pancreatic acini were hypertrophied. Space formation was seen in islet of Langerhans (Fig. 82) and dilation and space formation was also seen in pancreatic duct, (Fig. 83).

After five weeks of treatment, damage was more pronounced as indicated by necrotic and vacuolated pancreatic acini. Pancreatic duct and blood vessels were shrunked and formation
of interlobular space was there (Fig. 84).

After six weeks of administration, necrosis, vacuolation, space formation and shrinkage in the pancreatic acini was more prominent. Some of the pancreatic acini lost their normal structure and hypertrophied. Islet of Langerhans became compact and were not normal (Fig. 85).

**EFFECT OF THC**

After four weeks of administration, hypertrophied pancreatic acini showed space formation and vacuolation. Dilation and space formation was also seen in the pancreatic duct (Fig. 86a). At places space formation and fusion was seen in pancreatic acini(Fig. 86b).

After five weeks of treatment, necrosis, splitting and vacuolation was seen in pancreatic acini and islet of Langerhans. There was shrinkage of blood vessels (Fig. 87).

After six weeks of administration, pancreatic acini showed space formation, fusion and vacuolation (Fig. 89). Dilation, space formation and vacuolation was seen in pancreatic duct (Fig. 89).

**EFFECT OF MALATHION**

After 10 days of administration, pancreatic acini showed splitting and fusion. Dilation was seen in pancreatic duct
which was ruptured and the blood vessels were damaged (Fig. 90).

After 20 days of exposure, pancreatic acini were ruptured and shrunk due to which there was space formation. Clumping of zymogen secretory cell was seen and pancreatic duct and blood vessels were shrunked (Fig. 91).

Changes after 30 days of exposure were remarkable showing splitting and space formation in pancreatic acini. Dilation and vacuolation was seen in the pancreatic duct, (Fig. 92).

**EFFECT OF CARBAKYL**

After four weeks of administration, pancreatic acini were ruptured, having vacuolation and degenerated (at some places) and blood vessels were shrunked (Fig. 93). Ruptured pancreatic duct and acini showed splitting and vacuolation (Fig. 94).

After five weeks of administration, splitting and vacuolation was seen in pancreatic acini with space formation. Some of the pancreatic acini showed fusion (Fig. 95).

After six weeks of exposure, pancreatic acini were ruptured, vacuolation and necrosis was also seen. Pancreatic ducts were ruptured and vacuolated and the blood vessels were shrunked (Fig. 96).

**NORMAL HISTOLOGY OF SPLEEN**

Spleen is made up of white pulp, the red pulp and poorly
defined marginal zone in between red pulp and white pulp. The white pulp is scattered throughout the spleen as tiny greyish islands. The red pulp consists primarily of cords separated by sinuses. The marginal zone is formed of recticular mesh work, the trabecular arteries which are the branches of the splenic artery are found in white pulp, where they are known as central arteries. Most of the lymphocytes are also found scattered in the white pulp and a few are found in the red pulp (Fig. 97,98, 99, 100).

**EFFECT OF DYT**

After four weeks of administration, vacuolation were seen in white pulp having space formation and lymphocytes were irregular in shape. The central artery was ruptured (Fig. 101). Space formation and vacuolation was seen in red pulp (Fig.107) and trabecular arteries were shrunked and ruptured (Fig. 103).

After five weeks of administration, image was more pronounced showing space formation and vacuolation, pycnotic lymphocyte and clumping of cell was seen in the white pulp area (Fig. 104). Space formation and rupture was seen in the trabecular arteries and central artery (Fig. 105). Red pulp and marginal zone of white pulp showed vacuolation (Fig. 106).

Structural changes after six weeks of exposure were characterised by fusion of red and white pulp, vacuolation
was also seen (Fig. 107). The central artery and trabecular arteries were ruptured (Fig. 108). Space formation, vacuolation and clumping of lymphocytes were seen in the white pulp (Fig. 109).

EFFECT OF BHC

After four weeks of administration, splitting of red and white pulp was seen due to which there was space formation (Fig. 110, 111). Red pulp became compact (Fig. 112) and the trabecular artery were swollen. There was space formation and the central artery was dilated (Fig. 113).

After five weeks of treatment, splitting was more pronounced in the white and red pulp, and clumping of lymphocytes were observed (Fig. 114, 115). Vacuolation was seen in white pulp and there was clumping of lymphocytes (Fig. 116). In the red pulp, vacuolation and clumping of cells was noticed (Fig. 117).

After six weeks of administration, red and white pulp showed splitting (Fig. 113). The central artery was dilated (Fig. 119). Accumulation of lymphocyte was seen due to which there was space formation. Trabeculae were somewhat dilated and swollen (Fig. 120).
EFFECT OF MALATHION

After 10 days of administration, fusion of red and white pulp was noticed and the central artery was shrunken (Fig. 121). White pulp became vacuolated and there was space formation. Irregular lymphocytes were also observed (Fig. 122).

After 20 days of treatment, red and white pulp became more fused and compact (Fig. 123). White pulp had pycnotic lymphocyte and clumping of cell was observed. Central artery became dilated (Fig. 124).

Changes after 30 days of exposure, were more remarkable resulting in shrinkage of red and white pulp and shrinkage of central artery was also observed (Fig. 125, 126). Vacuolation, space formation was seen in the white pulp and clumping of lymphocytes was also seen (Fig. 127).

EFFECT OF CARBARYL

After four weeks of administration, there was splitting in red and white pulp due to which space formation was seen (Fig. 128). In the white pulp, space formation, vacuolation and clumping of lymphocyte was seen. The trabeculae were shrunken (Fig. 129).

After five weeks of treatment, splitting was more pronounced in the white and red pulp and clumping of lymphocytes
were observed (Fig. 130) vacuolation and space formation was seen in the white pulp (Fig. 131).

After six weeks of exposure, the red and white pulp showed more splitting (Fig. 132). Space formation and vacuolation was seen in the red pulp (Fig. 133). Whereas the white pulp showed splitting, vacuolation and clumping of lymphocytes, (Fig. 134).

NORMAL HISTOLOGY OF LIVER

The liver is composed of hexagonal insular lobules with a central vein in centre and at the corner lie the portal canals. The portal canals consists of connective tissue strand in which the "portal triads" are conspicuous. The triad includes a branch of portal vein, the hepatic artery and the bile duct. The liver cells are arranged as plates usually one cell thick extending radially from the central vein to the periphery of the lobule. Sinusoids run inward concentrically towards the central vein.

Each lobule consists of hepatic cells arranged in radiating manner forming hepatic cords. Smaller branches of blood vessels penetrate between the hepatic lobules, called interlobular vessels. The interlobular vessels give out fine branches which run radially to the centre of lobules. As the lining of these capillaries are deficient, they are called
sinusoids. These converge to the centre of the lobule and form the interlobular vein. The interlobular veins open into the sublobular veins which joins to form the hepatic vein.

Each hepatic cell has a large spherical nucleus and granular cytoplasm. Its protoplasm is provided by a network of canaliculi which would receive blood directly from the sinusoids. In between the hepatic cells lie small channels called bile capillaries or canaliculi, towards the periphery of the lobule. These channels join to form the interlobular bile ducts, these in their turn join and form the hepatic ducts of the lobules, which join to form common bile duct.

Each lobules is composed of small, many sided lobules enclosed in a connective tissue sheath known as Glisson's capsule. In the centre is a intralobular vein and radiating out from this are minute vessels called sinusoids, which lead into interlobular veins in the capsule which originate from the portal vein. There were no structural changes in the liver of simultaneous controls (Fig. 135,136,137.).

EFFECT OF DMT

After four weeks of administration, the hepatic cells were ruptured having vacuolation and degenerated nuclei. In between hepatic cords spaces were formed (Fig. 138). The
Glisson's capsule showed hypertrophy and space formation was seen surrounding it (Fig. 139). The intralobular vein was hypertrophied (Fig. 140).

After five weeks of treatment, damage was more pronounced as indicated by necrotic and vacuolated hepatic cells. The hepatic cords showed splitting whereas their nuclei became pycnotic and somewhat degenerated (Fig. 141). The Glisson's capsule and interlobular vein were ruptured (Fig. 142).

Structural changes after six weeks of exposure, were characterised by necrosis and vacuolation in hepatic cells having pycnotic nuclei. Space formation was seen in hepatic cord's (Fig. 143) and the intralobular vein was hypertrophied (Fig. 144). The Glisson's capsules were hypertrophied and surrounding them space formation was seen, the interlobular vein were ruptured, (Fig. 145).

**EFFECT OF BHC**

After four weeks of administration, hepatic cells were ruptured having pycnotic and degenerated nuclei and space formation was seen in the hepatic cords (Fig. 146).

After five weeks of treatment, hepatic cells were hypertrophied and pycnotic and degenerated nuclei were seen. These cells looked like spongy or foamy in structure (Fig. 147).
The interlobular vein was ruptured at this stage (Fig. 148).

After six weeks of exposure, hepatic cells were ruptured having vacuolation and some of the hepatic cells were binucleated (Fig. 149). The Glisson's capsule and intralobular vein were also hypertrophied and surrounding them space formation was seen (Fig. 150). The interlobular veins were hypertrophied and ruptured (Fig. 151).

**EFFECT OF MALATHION**

After 10 days of administration, hepatic cells were ruptured showing vacuolation and pycnotic nuclei and the hepatic cords showed space formation (Fig. 152). The intralobular vein was also hypertrophied (Fig. 153).

After 20 days of treatment, spaces were formed in between the hepatic cords, whereas hepatic cells showed, vacuolation having pycnotic and degenerated nuclei (Fig. 154). The Glisson's capsule showed hypertrophy and surrounding them space formation was seen (Fig. 155).

Changes after 30 days of exposure were more remarkable which resulted in hypertrophied and vacuolated hepatic cells having pycnotic and degenerated nuclei. Some of the hepatic cells were binucleated and cytoplasm of some of them showed degeneration due to which spaces were formed inbetween the
hepatic cords (Fig. 155,137).

**EFFECT OF CANARYL**

After four weeks of administration, hepatic cells were ruptured showing vacuolation having pyknotic and degenerated nuclei. Space formation was seen in hepatic cords (Fig. 158). Intralobular vein was hypertrophied (Fig. 159) and the interlobular vein was ruptured (Fig. 160).

After five weeks of treatment, the damage was more pronounced as indicated by hypertrophied and vacuolated hepatic cells with pyknotic and degenerated nuclei, some of the hepatic cells were binucleated (Fig. 160).

After six weeks of treatment, hepatic cells were ruptured showing vacuolation and pyknotic nuclei and the liver showed somewhat spongy structure and at places binucleate hepatic cells were also seen, there was space formation in between hepatic cords (Fig. 162a,162b).
Fig. 81  Photomicrograph of section of pancreas of control rat. Delafield's Haematoxylin-Eosin. X 200.

Fig. 82  Photomicrograph of section of pancreas of rat after four weeks of DDT treatment. Delafield's Haematoxylin-Eosin. X 200.
Fig. 83. Photomicrograph of section of pancreas of rat after four weeks of DDT treatment showing pancreatic duct. Delafield's haematoxylin-Eosin. X 200.

Fig. 84. Photomicrograph of section of pancreas of rat after five weeks of DDT treatment. Delafield's Haematoxylin-Eosin. X 200.

Fig. 85. Photomicrograph of section of pancreas of rat after six weeks of DDT treatment. Delafield's Haematoxylin-Eosin. X 200.
Fig. 86A. Photomicrograph of section of pancreas of rat after four weeks of BHC treatment. Delafield's Haematoxylin-Eosin. X 200.

Fig. 86B. Photomicrograph of section of pancreas of rat after four weeks of BHC treatment. Delafield's Haematoxylin-Eosin. X 200.

Fig. 87. Photomicrograph of section of pancreas of rat after five weeks of BHC treatment. Delafield's Haematoxylin-Eosin. X 200.
**Fig. 88.** Photomicrograph of section of pancreas of rat after six weeks of BHC treatment. Delafield's Haematoxylin-Eosin. X 200.

**Fig. 89.** Photomicrograph of section of pancreas of rat after six weeks of BHC treatment showing pancreatic duct. Delafield's Haematoxylin-Eosin. X 200.

**Fig. 90.** Photomicrograph of section of pancreas of rat after 10 days of malathion treatment. Delafield's Haematoxylin-Eosin. X 200.
Fig. 91. Photomicrograph of section of pancreas of rat after 20 days of malathion treatment. Delafield's Haematoxylin-Eosin. X 200.

Fig. 92. Photomicrograph of section of pancreas of rat after 30 days of malathion treatment. Delafield's Haematoxylin-Eosin. X 200.

Fig. 93. Photomicrograph of section of pancreas of rat after four weeks of carbaryl treatment. Delafield's Haematoxylin-Eosin. X 200.
Fig. 94. Photomicrograph of section of pancreas of rat after four weeks of carbaryl treatment showing pancreatic duct and blood vessels. Delafield's Haematoxylin-Eosin. X 200.

Fig. 95. Photomicrograph of section of pancreas of rat after five weeks of carbaryl treatment. Delafield's Haematoxylin-Eosin. X 200.

Fig. 96. Photomicrograph of section of pancreas of rat after six weeks of carbaryl treatment. Delafield's Haematoxylin-Eosin. X 200.
Fig. 97A. Photomicrograph of section of spleen of control rat showing red and white pulp and trabeculae. Delafield's Haematoxylin-Eosin. X 60.

Fig. 97B. Photomicrograph of section of spleen of control rat showing red and white pulp and trabeculae. Delafield's Haematoxylin-Eosin. X 60.

Fig. 98A. Photomicrograph of section of spleen of control rat showing red pulp, trabeculae and marginal zone. Delafield's Haematoxylin-Eosin. X 100.
Fig. 98B. Photomicrograph of section of spleen of control rat showing red and white pulp. Delafield's Haematoxylin-Eosin. X 100.

Fig. 99. Photomicrograph of section of spleen of control rat showing red pulp. Delafield's Haematoxylin-Eosin. X 200.

Fig. 100. Photomicrograph of section of spleen of control rat showing white pulp. Delafield's Haematoxylin-Eosin. X 200.
Fig. 101. Photomicrograph of section of spleen of rat after four weeks of DDT treatment showing white pulp. Delafield's Haematoxylin-Eosin. X 200.

Fig. 102. Photomicrograph of section of spleen of rat after four weeks of DDT treatment. Delafield's Haematoxylin-Eosin. X 200.

Fig. 103. Photomicrograph of section of spleen of rat after four weeks of DDT treatment showing trabecular serrated arteries. Delafield's Haematoxylin-Eosin. X 200.
Fig. 104. Photomicrograph of section of spleen of rat after five weeks of DDT treatment showing white pulp. Delafield's Haematoxylin-Eosin. X 200.

Fig. 105. Photomicrograph of section of spleen of rat after five weeks of DDT treatment showing trabecular arteries. Delafield's Haematoxylin-Eosin. X 200.

Fig. 106. Photomicrograph of section of spleen of rat after five weeks of DDT treatment showing red pulp, white pulp and marginal zone. Delafield's Haematoxylin-Eosin. X 200.
Fig. 107. Photomicrograph of section of spleen of rat after six weeks of DDT treatment showing red and white pulp. Delafield's Haematoxylin-Eosin. X 60.

Fig. 108. Photomicrograph of section of spleen of rat after six weeks of DDT treatment showing trabecular and central arteries. Delafield's Haematoxylin-Eosin. X 60.

Fig. 109. Photomicrograph of section spleen of rat after six weeks of DDT treatment showing white pulp. Delafield's Haematoxylin-Eosin. X 200.
Fig. 110. Photomicrograph of section of spleen of rat after four weeks of BHC treatment showing white pulp.
Delafield's Haematoxylin-Eosin. X 60.

Fig. 111. Photomicrograph of section of spleen of rat after four weeks of BHC treatment showing red pulp.
Delafield's Haematoxylin-Eosin. X 60.

Fig. 112. Photomicrograph of section of spleen of rat after four weeks of BHC treatment showing red pulp.
Delafield's Haematoxylin-Eosin. X 100.
Fig. 113. Photomicrograph of section of spleen of rat after four weeks of BHC treatment showing trabecular arteries. Delafield's Haematoxylin-Eosin. X 200.

Fig. 114. Photomicrograph of section of spleen of rat after five weeks of BHC treatment showing red and white pulp. Delafield's Haematoxylin-Eosin. X 60.

Fig. 115. Photomicrograph of section of spleen of rat after five weeks of BHC treatment. Delafield's Haematoxylin-Eosin. X 100.
**Fig. 116.** Photomicrograph of section of spleen of rat after five weeks of BHC treatment showing white pulp. Delafield's Haematoxylin-Eosin. X 200.

**Fig. 117.** Photomicrograph of section of spleen of rat after five weeks of BHC treatment showing red pulp. Delafield's Haematoxylin-Eosin. X 200.

**Fig. 118.** Photomicrograph of section of spleen of rat after six weeks of BHC treatment showing red and white pulp. Delafield's Haematoxylin-Eosin. X 60.
Fig. 119. Photomicrograph of section of spleen of rat after six weeks of BHC treatment showing central arteries. Delafield's Haematoxylin-Eosin. X 100.

Fig. 120. Photomicrograph of section of spleen of rat after six weeks of BHC treatment showing trapeicular arteries. Delafield's Haematoxylin-Eosin. X 200.

Fig. 121. Photomicrograph of section of spleen of rat after 10 days of malathion treatment showing red and white pulp. Delafield's Haematoxylin-Eosin. X 60.
Fig. 122. Photomicrograph of section of spleen of rat after 10 days of malathion treatment showing white pulp. Delafield's Haematoxylin-Eosin. X 200.

Fig. 123. Photomicrograph of section of spleen of rat after 20 days of malathion treatment showing red and white pulp. Delafield's Haematoxylin-Eosin. X 60.

Fig. 124. Photomicrograph of section of spleen of rat after 20 days of malathion treatment showing white pulp and central artery. Delafield's Haematoxylin-Eosin. X 200.
Fig. 125. Photomicrograph of section of spleen of rat after 30 days of malathion treatment showing red and white pulp. Delafield's Haematoxylin-Eosin. X 60.

Fig. 126. Photomicrograph of section of spleen of rat after 30 days of malathion treatment showing central artery. Delafield's Haematoxylin-Eosin. X 60.

Fig. 127. Photomicrograph of section of spleen of rat after 30 days of malathion treatment showing white pulp. Delafield's Haematoxylin-Eosin. X 200.
Fig. 128. Photomicrograph of section of spleen of rat after four weeks of carbaryl treatment showing red and white pulp. Delafield's Haematoxylin–Eosin. X 60.

Fig. 129. Photomicrograph of section of spleen of rat after four weeks of carbaryl treatment showing white pulp and trabeculae. Delafield's Haematoxylin–Eosin. X 100.

Fig. 130. Photomicrograph of section of spleen of rat after five weeks of carbaryl treatment showing red and white pulp. Delafield's Haematoxylin–Eosin. X 100.
Fig. 131. Photomicrograph of section of spleen of rat after five weeks of carbaryl treatment showing white pulp. Delafield's Haematoxylin-Eosin. X

Fig. 132. Photomicrograph of section of spleen of rat after six weeks of carbaryl treatment showing red and white pulp. Delafield's Haematoxylin-Eosin. X 60.

Fig. 133. Photomicrograph of section of spleen of rat after six weeks of carbaryl treatment showing red pulp. Delafield's Haematoxylin-Eosin. X 200.
Fig. 134. Photomicrograph of section of spleen of rat after six weeks of carbaryl treatment showing white pulp. Delafield's Haematoxylin-Eosin. X 200.
Fig. 135. Photomicrograph of section of control liver of rat. Delafield's Haematoxylin-Eosin. X 300.

Fig. 136. Photomicrograph of section of control liver of rat showing intralobular vein. Delafield's Haematoxylin. X 100.

Fig. 137. Photomicrograph of section of control liver of rat showing interlobular vein. Delafield's Haematoxylin-Eosin. X 300.
Fig. 138. Photomicrograph of section of liver of rat after four weeks of DDT treatment. Delafield's Haematoxylin-Eosin. X 300.

Fig. 139. Photomicrograph of section of liver of rat after four weeks of DDT treatment showing Glisson's capsule. Delafield's Haematoxylin-Eosin. X 100.

Fig. 140. Photomicrograph of section of liver of rat after four weeks of DDT treatment showing intralobular vein. Delafield's Haematoxylin-Eosin. X 100.
Fig. 141. Photomicrograph of section of liver of rat after five weeks of DDT treatment. Delafield's Haematoxylin-Eosin. X 300.

Fig. 142. Photomicrograph of section of liver of rat after five weeks of DDT treatment showing interlobular vein. Delafield's Haematoxylin-Eosin. X 300.

Fig. 143. Photomicrograph of section of liver of rat after six weeks of DDT treatment. Delafield's Haematoxylin-Eosin. X 300.
Fig. 144. Photomicrograph of section of liver of rat after six weeks of DDT treatment showing intralobular vein. Delafield's Haematoxylin-Eosin. X 100.

Fig. 145A. Photomicrograph of section of liver of rat after six weeks of DDT treatment showing Glisson's capsule. Delafield's Haematoxylin-Eosin. X 300.

Fig. 145B. Photomicrograph of section of liver of rat after six weeks of DDT treatment showing interlobular vein. Delafield's Haematoxylin-Eosin. X 300.
Fig. 146. Photomicrograph of section of liver of rat after four weeks of BHC treatment. Delafield's Haematoxylin-Eosin. X 300.

Fig. 147. Photomicrograph of section of liver of rat after five weeks of BHC treatment. Delafield's Haematoxylin-Eosin. X 300.

Fig. 148. Photomicrograph of section of liver of rat after five weeks of BHC treatment showing interlobular vein. Delafield's Haematoxylin-Eosin. X 300.
Fig. 149. Photomicrograph of section of liver of rat after six weeks of BHC treatment. Delafield's Haematoxylin-Eosin. X 300.

Fig. 150. Photomicrograph of section of liver of rat after six weeks of BHC treatment showing Glisson's capsule and intralobular vein. Delafield's Haematoxylin-Eosin. X 300.

Fig. 151. Photomicrograph of section of liver of rat after six weeks of BHC treatment showing interlobular vein. Delafield's Haematoxylin-Eosin. X 300.
Fig. 152. Photomicrograph of section of liver of rat after 10 days of malathion treatment. Delafield's Haematoxylin-Eosin. X 300.

Fig. 153. Photomicrograph of section of liver of rat after 10 days of malathion treatment showing intralobular vein. Delafield's Haematoxylin-Eosin. X 100

Fig. 154. Photomicrograph of section of liver of rat after 20 days of malathion treatment. Delafield's Haematoxylin-Eosin. X 300.
Fig. 155. Photomicrograph of section of liver of rat after 20 days of malathion treatment, showing Glisson's capsule Delafield's Haematoxylin-Eosin. X 100.

Fig. 156. Photomicrograph of section of liver of rat after 20 days of malathion treatment, showing Glisson's capsule Delafield's Haematoxylin-Eosin. X 100.

Fig. 157. Photomicrograph of section of liver of rat after 30 days of malathion treatment. Delafield's Haematoxylin-Eosin. X 300.
Fig. 158. Photomicrograph of section of liver of rat after four weeks of carbaryl treatment. Delafield's Haematoxylin-Eosin. X 300.

Fig. 159. Photomicrograph of section of liver of rat after four weeks of carbaryl treatment showing intra-lobular vein. Delafield's Haematoxylin-Eosin. X 300.

Fig. 160. Photomicrograph of section of liver of rat after four weeks of carbaryl treatment showing inter-lobular vein. Delafield's Haematoxylin-Eosin. X 300.
**Fig. 161.** Photomicrograph of section of liver of rat after five weeks of carbaryl treatment. Delafield's Haematoxylin-Eosin. X 300.

**Fig. 162.A.** Photomicrograph of section of liver after six weeks of carbaryl treatment showing spongy structure. Delafield's Haematoxylin-Eosin. X 300.

**Fig. 162.B.** Photomicrograph of section of liver after six weeks of carbaryl treatment showing spongy structure. Delafield's Haematoxylin-Eosin. X 300.
B. DISCUSSION

By reviewing the literature it seems that very limited work has been done on the associated glands of alimentary canal and spleen of mammals including rats, induced by chemicals especially pesticides. Rich and Duff (1936) reported pathological changes induced by India ink on the duct acini intestinal tissue of dog. They also reported effect of sterile Locke's solution on pancreas of dog such as oedema and scattered leucocytes in the inter-lobular tissue. Injection of trypsin and bile in dog resulted in haemorrhagic pancreatitis and necrosis in pancreatic tissue including fat necrosis as reported by Rich and Duff (1936). Netto and Dreiling (1960) reported edema, haemorrhage and moderate to severe degrees of edematus and haemorrhagic pancreatitis in dogs, receiving G-27202 (Tendearil) including edematus and haemorrhagic pancreatitis and few scattered area of bleeding. The dogs treated with piptal J-3437 and propylthiuracil also showed several pancreatic edema and haemorrhage. Anderson and Schiller (1968) reported in dog, pancreatitis, severe pancreatic edema and haemorrhage or massive haemorrhagic necrosis after exposure to trypsin or trypsin digested blood which contained Indian ink. Lilander and Johansson (1976) described karyopyknosis, karyorrhexis, cytoplasmic vacuolation and scattered exocrine cells in the pancreas of monkey after injection of N-nitrosomethylurea.
Necrosis of beta cells, reduced in number in the pancreas of rats after exposure to alloxan were reported by Chakravarthy et al. (1983). Aihlawat et al. (1985) reported edema, cellular infiltration, lobular necrosis and vasculitis, pancreatic haemorrhage, ductal dilation, ductal lining, intraductal and peri-ductal changes etc. in rats due to duodenal obstruction. Tsukamoto et al. (1988) described hypogranulation and apoptosis of acinar cells, focal lesions, characterized by fat necrosis, mononuclear cell infiltration, fibrosis, acinar atrophy, ductal dilation and intraductal mucious or proteinaceous plugs and acute focal pancreatitis in rat pancreas after treatment with ethanol and high fat diet.

In the present work pathological changes caused by DDT treatment viz. hypertrophied pancreatic acini with space formation and vacuolation, formation of a space in the islets of Langerhans and pancreatic duct, dilation of pancreatic duct, shrunken blood vessels, formation of interlobular space and compact islets of Langerhans in the pancreas of albino rats were seen.

The treatment with HCH resulted in space formation and vacuolation in pancreatic duct and damage to blood vessels. Pancreatic acini showed space formation, fusion and vacuolation in the pancreas of rat after treatment with HCH.

In case of malathion treated rats, pancreatic acini
showed hypertrophy, space formation, vacuolation and dilation and rupture in the pancreas.

The carbaryl treatment caused necrotic changes such as ruptured pancreatic acini showing splitting and vacuolation, shrunken blood vessels, damaged and vacuolated pancreatic duct in the pancreas.

Langevoort (1963) reported histological changes in the spleen of rabbit after different intervals of a single intravenous injection of HOGG (Horse-y-globulin). He described inactive follicular center, plasma blasts, lymphocyte blast cells, follicular intermediate cells, periarteriolar sheath and enlarged and highly active follicular centre. Taub and Lance (1968) described striking depletion of small lymphocytes and a rim of immunoblast surrounding the central arteriole due to the treatment of ARS and NAS enlarged spleen with generalized follicular hyperplasia with immunoblast and plasma cells within and at the edge of follicle erythroid hyperplasia of red pulp in the spleen of mice. Athanassiades and Morse (1973) reported increase in number of neutrophill leukocytes in the periarteriolar lymphatic sheaths and in germinal centres, necrotic cells or tingible bodies, decreased cellularity in the spleen, obliterated follicular pattern of the white pulp, associated blast cells with foci of poiesis appeared in white pulp in the spleen of mice after exposure to different
period of supernatant fluids of *Bordetella pertussis* cultures.

Distinctly narrowed marginal zones, markedly increased germinal centers in size and number, noticeable infiltration of the red pulp, enhanced granulocytes in the germinal center, increase in number of macrophagus in the red pulp and germinal centers after different exposure to endotoxin in rats spleen were reported by Satodate et al. (1976). Rappaport et al. (1979) reported histological changes after different time periods of radiation in mice. They observed cellular depletion in splenic red pulp, lympholysis and occasional plasma cell in white pulp. Repopulated splenic white pulp, large lymphblastoid cells in the red pulp, white cells occupied by large lymphoid cells and vacuolated macrophagus, haemorrhage, necrosis of the red pulp in the spleen of rats exposed to major and minor GVHR are reported by Rappaport et al. (1979).

Whereas in the present work organochloro-pesticides showed vacuolation, space formation, irregular and clumping of lymphocytes in the white pulp, vacuolated central artery and red pulp with space formation, ruptured and shrunken trabeculae, vacuolated marginal zone and fused red and white pulp with DDT treatment.

In the BHC treated rats, pathological changes such as splitting in the white and red pulp, compactness of red pulp,
shrinkage in the trabeculae with space formation, dilated central artery, clumping of lymphocytes and vacuolated white and red pulp were seen in the spleen.

Fusion of red and white pulp, shrinkage as well as dilated central artery, space formation and vacuolation in white pulp and irregular pycnotic lymphocyte were noticed in the spleen of rats treated with malathion.

Carbaryl also caused pathological changes such as splitting and vacuolated red and white pulp, clumping of lymphocytes and shrunked trabeculae in the spleen of rats.

Fitzhugh and Nelson (1947) reported occurrence of hepatic cell tumours and nodular adenomatoid hyperplasia of the liver in rats receiving DDT in the diet at levels of 200-300 ppm. Fitzhugh et al. (1950) described focal necrosis in the liver of rats exposed to BHC and its alpha, beta and gamma isomers. Roe (1954), Berenblum and Haran (1955), Tannenbaum and Silverstone (1958), Klein (1962) have described haemorrhage as a consequence of urethan damage to endothelial lining of blood vessels in the liver. Increase in incidence of liver tumours in treated animals was higher than that reported by Davis and Fitzhugh (1962) treated with dieldrin, Trainin (1963) described tumours in liver of mice by urethan, a carbamate pesticides. No pathological changes were reported by Krishnamurty et al. (1964)
in the liver of rats after administration of 800 ppm malathion. Ortega (1966) observed moderate centrolobular hypertrophy of hepatocytes, increased nuclear size, margination of cytoplasm and basophilic granules and increased cytoplasmic hyalinization in the liver of rats after exposure to lower and higher doses of DDT. In the present study histopathological changes such as rupture and vacuolization in liver cells and pycnotic and degenerated nuclei was seen in hepatic cells. Ruptured intralobular vein and interlobular vein were also noticed, indicating that damage is done due to the treatment of DDT in rats. Tumour formation, dilated sinusoidal spaces, pycnotic and irregular nuclei in mice treated with benzene hexachloride were reported by Nagasaki et al. (1971). In the present study after administration of DHC histopathological changes such as pycnotic nuclei, sinusoidal spaces, hypertrophy of hepatic cells, intralobular vein and Glisson's capsule were seen, but tumour formation could not be observed as reported by Nagasaki et al. (1971). Kimbrough et al. (1971) reported enlargement of liver cells, smooth looking cytoplasmic margination, cytoplasmic inclusion in almost all cells with nuclei of many of the cells appearing to be exceptionally big in the liver of rats after exposure to DDT and dieldrin and as well as with the combination of both. Walker et al. (1972) reported tumour formation, nuclear abnormalities, cytoplasmic changes including hydropic changes, fatty changes and hyaline droplet formation and focal necrosis in mice after administration of dieldrin and DDT. In the present
investigation hypertrophy, vacuolation, space formation, pycnotic nuclei in hepatic cells are seen. Degenerated nuclei and space formation was also seen in the interlobular vein which were ruptured at some places. Formation of liver tumours in mice after prolonged exposure to dieldrin was the only long term adverse effect of the compound and no changes in the incidence of tumours were found in rats and dogs by Walker et al. (1969). Goto et al. (1972) reported, that mice exposed to alpha BHC (660 ppm) for 12 weeks developed liver tumours. Tomatis et al. (1972) also reported liver tumours in male and female mice treated with DDT. Ballooning and nuclear pycnosis and cellular damages in the liver of rats after exposure to methyl parathion plus DDT were reported by Dikshith and Datta (1972). Nagasaki et al. (1972) reported that technical grade of benzene hexachloride (BHC) had a carcinogenic effect on the liver of male mice. Liver cell tumours were not reported in mice given the control diet or lower dietary concentration of DDT by Terracini et al. (1973). But Terracini et al. (1973) observed liver cell tumour after administration of 250 ppm technical grade DDT. Thorpe and Walker (1973) reported tumours in the liver of mice after exposure to various pesticides (dieldrin, DDT, phenobarbitone, beta BHC and gamma BHC). In the present study tumour formation could not be seen.

Risk of liver tumour development can be modified by hormonal status and diet and is certainly related to genetic
background of the mouse strain used according to Heston and Ulahakis (1961, 1966) and Roe et al. (1971). There is some evidence that liver lesions in a strain of mice with high incidence of spontaneous liver tumours progresses with age from nodular hyperplasia to small and large hepatocellular carcinomas. Reuber (1971) and Ito et al. (1973a) reported that in mice who received BHC, the centrolobular liver cells were hypertrophied having nuclear irregularities in the cells of hypertropic areas. However, mitotic figures, fatty changes and necrotic signs in the cells were rarely observed although hypertropic changes of parenchymal cells also occurred but such changes were slight in mice treated with beta and gama BHC respectively. Ito et al. (1973b) also reported hypertropic changes of liver parenchymal cells, nodular hyperplasia and hepatocellular carcinoma etc. in alpha BHC or beta BHC plus PCBS treated mice. The present study also showed hypertrophy of hepatic cells with all the four pesticides. Hypertrophic changes reported by Datta and Dikshith (1973) showed changes like pycnosis of hepatocytes and congestion of blood vessels and parenchyma, treated with ethyl parathion and methyl para- tion plus DDT in rats. Kashyap et al. (1977) reported tumours of lymphoid tissues, lung and liver after oral and subcutane- neous DDT treatment in swiss mice. Slight increase in liver weight and vacuolation of cytoplasm in albino rats after expo- sure to dieldrin were reported by Hurkat (1977). Vacuolation were also seen in liver of albino rats with DDT, BHC, malathion
and carbaryl in the present study.

Dikshith et al. (1978) observed hypertrophy of hepatocytes, highly vacuolated cytoplasm, pycnthetic nuclei and swelling and proliferation of epithelial cells of bile ducts of BHC treated guinea pigs. Author noticed hypertrophy, pycnotic nuclei, vacuolation, space formation, ruptured hepatic cells etc. in rats liver treated with DDT, BHC, malathion and carbaryl. Mansour and Sayouri (1980) described several histopathological changes like congestion of sinusoids, nuclear pycnosis, liver fibrosis and necrosis in albino rats due to tetrodotoxification. Dikshith et al. (1980) reported no degenerative changes in liver of rats after exposure to CCl₄ BHC, but CCl₄ alone causes morphological changes such as swollen hepatocytes, vacuolization in cytoplasm with nuclei pushed to a corner of cell and necrosis of the hepatocytes in some area of the parenchyma. Jonsson et al. (1981) reported vacuolated hepatocytes and cellular necrosis with moderate hepatocyte regeneration after exposure to 75 or 150 ppm DDT for 36 weeks. Shivanandappa and Krishnakumari (1981) reported hypertrophy and hyperplasia, vacuolation and focal necrotic areas in liver of rats with dietary levels of BHC. Bhatt et al. (1981) reported tumours in liver of swiss mice induced by hexachlorocyclo-hexane (BHC). Cabral and Ponomarkov (1982) reported no significant increase in incidence of liver cell tumours in mice after exposure to pesticide maleic hydrazide (MH). In the present study no liver cell tumours in albino
rats after exposure to DDT, BHC, malathion and carbaryl could be seen as has also been reported by Baronia (1989).

Cabral et al. (1982) reported that exposure of DDT resulted in statistically significant incidence of liver cell tumours in female treated rats only. Nigam et al. (1982) described histopathology of liver showing clear oval cells, hypertrophied cells with foci and neoplastic nodules that were apparent during the final 3 to 6 month of exposure of BHC in mice. Several pathological changes in the liver of albino rats after administration of paracetamol were reported by Mehrotra et al. (1982) and Sharma et al. (1983). They also observed necrosis, ballooning degeneration, mononuclear cell infiltration, vacuolation and loss of pyroninophilia. Vacuolation and hypertrophy of hepatic cells of rabbit during malathion toxicity has been reported by Ali and Abdul (1981, 1983). Kandarkar et al. (1983) observed centrilobular necrosis, hypertrophied nuclei, adenomas and hepatocellular carcinomas in BHC exposed mice. Nigam et al. (1984) reported tumours and sequential cellular alterations in the liver of mice after exposure to BHC. Singh et al. (1984) described pathological changes consisting of focal necrosis, mononuclear cell infiltration, more eosinophilic cytoplasm, disorganization of hepatic cords and hypertrophied kuffer cells in the liver of goat after exposure to aldrin. Such results could not be observed in the liver of rat in the present study. Hypertrophy of hepatocytes, nuclear deformation, increased pycnotic
nuclei and cytoplasm containing numerous vacuoles and lipid droplets in the liver of mice after exposure to aminotriazole were reported by Reitze and Seitz (1985). Anthony et al. (1986) reported vacuolation or fatty changes in hepatocytes of male rats after 14 and 23 weeks of administration of Diazinon (organophosphate). Shatnagar and Jain (1986) reported vacuolization, sinusoidal space, midzonal necrosis and enlargement of nuclei of hepatocytes and mild fatty degeneration after 30 and 60 days exposure to phosphamidon. In the present investigation, vacuolation, sinusoidal space formation, hypertrophied and ruptured hepatic cell and interlobular vein in the rat liver after exposure to malathion are reported. Singh et al. (1983) reported no pathological lesions in the liver and other organs of albino rat during metanil yellow and orange II toxicity. Shatia and Sood (1983) observed necrosis and ballooning degeneration in liver of rat induced due to paracetamol.

Baronia and Sahai (1989) reported several pathological changes such as hypertrophied and ruptured hepatic cells, pyeototic nuclei, vacuolation, space formation, hypertrophied focal cell and ruptured intralobular vein in the liver of albino rat after exposure to DDT. In the present investigations hypertrophy and ruptured hepatic cells with pyeototic nuclei, vacuolation, space formation and ruptured intralobular vein were in confirmation with Baronia and Sahai (1989). Other
histopathological changes such as splitting, degenerated nuclei and ruptured interlobular vein in the liver of albino rats after exposure to DDT were also noticed.

Sahai et al. (1990) reported several pathological changes due to malathion treatment, such as necrosis of hepatic cells, vacuolation, space formation, swollen and pycnotic nuclei and binucleated hepatic cells. In the present work changes reported by Sahai et al. (1990) were confirmed and in addition it was seen that intralobular veins were damaged in the liver of albino rats after treatment with malathion. Baronia (1989) reported several pathological changes such as hypertrophied hepatic cells with pycnotic and degenerated nuclei, vacuolation, space formation, blood haemorrhage, spongy or foamy structure in liver of albino rats after exposure to BHC. In the present investigations, hypertrophy of hepatic cell with pycnotic and degenerated nuclei, vacuolation, space formation, spongy or foamy structure in liver were in confirmation with Baronia (1989) and other histopathological changes such as hypertrophy and ruptured intralobular vein and ruptured interlobular vein in the liver of albino rats after exposure to BHC were also noticed. Baronia (1989) reported several pathological changes such as ruptured hepatic cells, pycnotic and degenerated nuclei, vacuolation, space formation, blood haemorrhage, hypertrophy and ruptured intralobular vein in the liver of albino rats after exposure to carbaryl. In the present investigations, blood haemorrhage could not be
seen and other changes reported by Baronia (1989) were in confirmation and in addition it was seen that interlobular vein were ruptured in the liver of albino rat after treatment with carbaryl.

It is concluded that the pancreas, spleen and liver is damaged with all the pesticides studied in the present work.