CHAPTER - IV

HISTOPATHOLOGICAL CHANGES INDUCED BY
PESTICIDES IN THE ALIMENTARY CANAL OF
RATTUS RATTUS ALBINO
A. OBSERVATIONS

NORMAL HISTOLOGY OF OESOPHAGUS

The histology of oesophagus shows a layer of serosa which is very thick. It is followed by an outer layer of longitudinal muscle and inner layer of circular muscle. This is followed by a submucosal zone which consists of loose connective tissue and blood vessels. Next to this are mucosa, which is the inner most layer and is made up of 4-6 folds. The mucosal folds consists of two layer an outer columnar epithelium layer and an inner stratified epithelial layer. There was no change in the structure of simultaneous control Fig. 1,2,3.

EFFECT OF DDT

After four weeks of administration the serosa was ruptured at some places. The longitudinal and circular muscle layer showed vacuolization and space formation (Fig. 4). The space formation was also seen in the submucosa (Fig. 5). Wall of mucosa of columnar epithelial cell and stratified epithelial layer showed irregular arrangement (Fig. 6).

After five weeks of treatment, serosa was broken at many places. The longitudinal and circular muscles showed necrosis. (Fig. 7). Space formation and vacuolization was also seen
in the submucosa and the walls of mucosa was ruptured at some places (Fig. 8).

After six weeks of administration, the serosa was degenerated. Space formation was more pronounced then the previous stage in submucosa (Fig. 9) and the submucosa was more damaged. It was dilated and filled with blood. The wall of columnar epithelial layer of mucosa showed vacuolization and was ruptured at many places (Fig. 10).

**EFFECT OF BHC**

After four weeks of administration the serosa was ruptured at many places. The longitudinal and circular muscle layer showed fusion and in the mucosal folds, enlargement was seen (Fig. 11). Walls of mucosal stratified epithelial layer were ruptured at some places and its columnar cells also showed irregular arrangement (Fig. 12).

After five weeks of treatment serosa was ruptured at many places. The longitudinal and circular muscle layer showed necrosis (Fig. 13). Submucosa showed damage and space formation and the walls of mucosal folds were ruptured at many places (Fig. 14).

After six weeks of administration, serosa was ruptured at many places and degenerated. Splitting and vacuolization was seen in the longitudinal and circular muscle layers and
only two mucosal folds were seen (Fig. 15). Space formation and ruptured stratified epithelial layer were noticed and the walls of columnar cells showed degeneration (Fig. 16).

**EFFECT OF MALATHION**

After 10 days of administration, space formation was seen in the stratified epithelial layer and the wall of mucosal columnar layer was irregular and ruptured at some places (Fig. 17).

After 20 days of treatment, serosa was ruptured at some places, longitudinal and circular muscle layer was shrunked. Walls of mucosal folds were irregular in their arrangement (Fig. 18).

After 30 days of administration, serosa was ruptured at many places and necrosis was seen in longitudinal as well as circular muscle layer. In mucosal folds structural changes were seen (Fig. 19). Wall of mucosal folds and stratified epithelial layer were ruptured at many places and columnar epithelial layer showed shrinkage. Space formation and vacuolization was seen in the submucosa (Fig. 20).

**EFFECT OF CARBARYL**

After four weeks of administration, serosa was ruptured and longitudinal and circular muscle layer showed necrosis (Fig. 21).
Space formation and vacuolization was seen in the submucosa, and stratified epithelial layer was ruptured at some places. Wall of columnar epithelial layer was irregular in arrangement (Fig. 22).

After five weeks of treatment, serosa was ruptured at some places and the circular muscle layer showed splitting and the mucosal folds showed necrosis (Fig. 23). Space formation and vacuolization was seen in the submucosa. Mucosal stratified epithelial layer was ruptured at many places (Fig. 24).

After six 6 weeks of administration, serosa showed degeneration, space formation and vacuolization was seen in the longitudinal and circular muscle layer (Fig. 25) and the submucosa was damaged. Wall of mucosal columnar layer showed vacuolization and degenerated nuclei were also observed (Fig. 26).

NORMAL HISTOLOGY OF STOMACH

The normal histology of stomach is made up of four coat viz. serosa, muscularis mucosa, sub-mucosa and mucosa, the thickness of these layers varies in different regions. The serosa is the outermost coat in the form of cellular membrane, the muscularis mucosa consists of two layers of smooth muscle fibers an outer longitudinal muscle layer and an inner circular muscle layer. The submucosa consists of loose connective
tissues with blood vessels and the mucosa form internal lining which is thrown into finger like projections, the villi. In the mucosal folds a single layer of columnar epithelial cell forms the lining of the lumen of the stomach. There was no structural changes in the simultaneous controls (Fig. 27, 28, 29).

**EFFECT OF DDT**

After four weeks of administration, submucosa shows damage and space formation was seen in the lamina propria. The mucosal tip was ruptured at some places (Fig. 30). Wall of columnar cell was ruptured and the mucosa showed space formation, vacuolization and pyknotic nuclei (Fig. 31).

After five weeks of treatment, damage was more pronounced as indicated by necrotic and vacuolated mucosa. There was space formation in the submucosa and the lamina propria showed vacuolization, (Fig. 32) mucosa showed vacuolization, space formation and pyknotic nuclei (Fig. 33).

After six weeks of exposure, serosa was ruptured at some places and longitudinal and circular muscle layer showed fusion. Space formation was seen in submucosa and the mucosa was more damaged (Fig. 34). Mucosa showed vacuolization, pyknotic nuclei as well as degenerated nuclei and the wall of mucosal columnar layer was totally ruptured (Fig. 35).
EFFECT OF CHC

After four weeks of administration, serosa was degenerated and the longitudinal and circular muscle layer showed necrosis. Space formation was seen in the submucosa and the mucosal tip was ruptured at some places (Fig. 36).

After five weeks of treatment space formation was seen in circular muscle layer and submucosa and the mucosa showed splitting, (Fig. 37) space formation, pycnotic nuclei, and the wall of columnar layer was ruptured (Fig. 38).

After six weeks of exposure, longitudinal and circular muscle layer showed necrosis, submucosa was damaged and the mucosal tip were ruptured (Fig. 39). The mucosa showed vacuolation, space formation, pycnotic nuclei and it was also degenerated (Fig. 40).

EFFECT OF MALATHION

After 10 days of administration, serosa was ruptured at some places, and longitudinal and circular muscle layer showed vacuolation. Space formation was seen in the submucosa and the mucosa was damaged. (Fig. 41). Space formation and vacuolation was also seen in the submucosa (Fig. 42).

After 20 days of treatment, circular muscle layer was ruptured at many places and space formation was seen in
submucosa (Fig. 43). Mucosa showed vacuolation, space formation and pycnotic nuclei (Fig. 44).

Changes after 30 days of administration were more pronounced resulting in the rupture of longitudinal and circular muscle layer at many places. More damage in submucosa and mucosa were observed (Fig. 45a). Space formation in submucosa and dilated and vacuolated blood vessels were seen at this stage (Fig. 45b). The mucosa showed space formation and was ruptured and pycnotic nuclei were seen (Fig. 46).

**EFFECT OF CARBARYL**

After four weeks of administration, serosa was broken at some places and longitudinal and circular muscle layer showed fusion and became compact. Space formation and vacuolation was seen in the submucosa and the mucosa showed necrosis (Fig. 47). Space formation, vacuolation and pycnotic nuclei were observed in mucosa (Fig. 48).

After five weeks of exposure, serosa was more ruptured and the longitudinal and circular muscle layer showed fusion. Space formation and vacuolation was seen in the submucosa and the mucosa showed ruptured layer with some of the portion as necrotic (Fig. 49). Space formation, vacuolation and pycnotic nuclei were seen in the mucosa (Fig. 50).
After six weeks of administration, space formation was more pronounced and was seen in the longitudinal and circular muscle layer and submucosa (Fig. 51). Mucosa showed vacuolation, space formation, pyknotic and degenerated nuclei (Fig. 52).

NORMAL HISTOLOGY OF SMALL INTESTINE

Histology of the small intestine is made up of an outer most serosa, followed by two layer of longitudinal and circular muscle layer which is followed by a submucosal zone. The submucosa consists of loose connective tissue with interspersed blood vessels and numerous branched Bruner's glands. Next to this are the villi, which are thrown into long heavy folds. The outer borders of the villi, contain mucigen secreting cells and are lined by columnar epithelial cells. The lamina propria forms the center of each villus, between the villi there are present definite crypts of lieberkuhn, or intestinal glands. There were no structural changes in the simultaneous controls (Fig. 53,54).

EFFECT OF DDT

After four weeks of administration, walls of villi were ruptured at some places and damage was seen in the columnar cells lining the walls of villi and villi showed vacuolation. Intestinal gland was damaged and submucosa showed vacuolation.
Bruner's gland showed fusion (Fig. 55).

After five weeks of treatment, walls of villi were more ruptured and showed vacuolation. Submucosa showed vacuolation and Bruner's gland were degenerated and showed vacuolation (Fig. 56).

After six weeks of exposure, wall of villi were ruptured and vacuolated at many places. Vacuolation was also seen in the lamina propria and space formation and vacuolation was seen in the submucosa also. The intestinal gland were more damaged and vacuolated (Fig. 57).

**EFFECT OF BHC**

After four weeks of administration, villi showed flattening and clumping of cells and wall of the villi were vacuolated. Space formation was seen in the lamina propria whereas in submucosa, space formation and vacuolation were seen. Bruner's gland were shrunked and vacuolated (Fig. 58).

After five weeks of treatment, wall of villi were ruptured and vacuolated at some places. Space formation and vacuolation was also seen in lamina propria (Fig. 59).

After six weeks of administration, columnar epithelial cell of villi were ruptured at many places, clumping of cell
was also seen. Villi were more damaged and showed cellular debris. Bruner's gland were shrunk, whereas a space formation and damage was seen in the submucosa (Fig. 60).

EFFECT OF MALATHION

After 10 days of administration, there was space formation, vacuolation and clumping of cells in the villi. Lamina propria showed vacuolization, space formation and vacuolation was seen in submucosa, whereas longitudinal and circular muscle layer showed necrosis (Fig. 61).

After 20 days of treatment, vacuolization was more prominent in the villi and the walls of the villi were ruptured and vacuolated at some places. The lamina propria showed vacuolation (Fig. 62).

After 30 days of administration, walls of villi were ruptured at many places and showed vacuolation and clumping of cells. Vacuolation and shrinkage was seen in lamina propria (Fig. 63).

EFFECT OF CARBAZYL

After four weeks of administration, villi showed vacuolation. Their walls were ruptured at some places. Lamina propria showed vacuolation. Space formation was seen in
submucosa and Bruner's gland, (Fig. 64).

After five weeks of treatment, necrosis of the villi, space formation and vacuolation in the lamina propria and submucosa were seen (Fig. 65).

After six weeks of exposure, walls of villi were ruptured and clumped at many places. Lamina propria was ruptured and vacuolated, space formation and vacuolation was seen in submucosa, whereas the Bruner's gland were vacuolated (Fig. 66).

NORMAL HISTOLOGY OF RECTUM

Rectum is made up of four coats viz. serosa, muscularis mucosa, submucosa and mucosa. Serosa, the outer most layer is followed by a submucosal zone. Next to which is the mucosa, which is thrown into small mucosal folds. The outer most borders of mucosa contains mucus secreting goblet cells and lined by columnar epithelial cells. There were no changes in the structure of simultaneous control (Fig. 67, 68).

EFFECT OF DDT

After four weeks of administration, serosa, longitudinal and circular muscle layer showed necrosis. Space formation was seen in the submucosa, whereas mucosa showed vacuolation and space formation. Wall of mucosal columnar epithelial
layer were ruptured at some places (Fig. 69).

After five weeks of treatment, space formation and vacuolation were seen in submucosa and the mucosa showed vacuolation, wall of mucosa were ruptured at many places. Space formation and vacuolation was seen in the lamina propria (Fig. 70).

After six weeks of exposure, serosa was ruptured at many places. Space formation and vacuolation were seen in submucosa. Mucosa showed vacuolation and its walls were ruptured at many places (Fig. 71).

**EFFECT OF BHC**

After four weeks of administration, space formation was seen in longitudinal and circular muscle layer. Space formation and vacuolation was seen in submucosa and the wall of mucosa were ruptured at some places. Mucosa showed vacuolation and space formation (Fig. 72).

After five weeks of treatment, damage was more pronounced in submucosa and the wall of mucosa were ruptured at many places. Clumping of cells were also observed. There was space formation in the lamina propria (Fig. 73).

After six weeks of exposure, serosa was ruptured at some places, longitudinal and circular muscle layer showed necrosis,
space formation was seen in submucosa. The wall of mucosa was ruptured and vacuolated at some places (Fig. 74).

**EFFECT OF CARBARYL**

After four weeks of administration, serosa was ruptured at some places. The longitudinal and circular muscle layer were hypertrophied. Space formation was seen in submucosa whereas in mucosa acute necrosis was seen and the wall of mucosa were ruptured at some places. Lamina propria was somewhat undamaged (Fig. 75).

After five weeks of treatment, serosa was ruptured at some places and space formation was seen in submucosa. Mucosa was damaged and space formation was seen (Fig. 76).

After six weeks of exposure, serosa was ruptured at many places. Longitudinal and circular muscle layer showed space formation and were ruptured. Vacuolation was seen in submucosa whereas mucosal tips were ruptured. Wall of mucosa was damaged and space formation and vacuolation were seen, (Fig. 77).

**EFFECT OF MALATHION**

After 10 days of administration, there was space formation in submucosa. Mucosa had vacuolation and the wall of mucosal columnar cells were broken at some places. Space formation
and vacuolation was seen in submucosa (Fig. 78).

After 20 days of treatment longitudinal and circular muscle layer showed necrosis, whereas in submucosa vacuolation and space formation was seen. Tip of villi were ruptured at many places and were vacuolated (Fig. 79).

After 30 days of treatment, space formation was seen in submucosa, mucosa showed vacuolation and clumping of cells and the damage was more pronounced. Space formation was seen in lamina propria (Fig. 80).
EXPLANATION TO THE FIGURE

**Fig. 1.** Photomicrograph of T.S. of oesophagus of control rat. Delafield's Haematoxylin-Eosin. X 60.

**Fig. 2.** Photomicrograph of T.S. of oesophagus of control rat showing muscle layers. Delafield's haematoxylin-Eosin. X 300.

**Fig. 3.** Photomicrograph of T.S. of oesophagus of control rat showing mucosa. Delafield's Haematoxylin-Eosin. X 300.
Fig. 4. Photomicrograph of T.S. of oesophagus of rat after four weeks of DDT treatment showing muscle layers. Delafield's Haematoxylin-Eosin. X 300.

Fig. 5. Photomicrograph of T.S. of oesophagus of rat after four weeks of DDT treatment showing submucosa. Delafield's Haematoxylin-Eosin. X 300.

Fig. 6. Photomicrograph of T.S. of oesophagus of rat after four weeks of DDT treatment showing mucosa. Delafield's Haematoxylin-Eosin. X 300.
Fig. 7. Photomicrograph of T.S. of oesophagus of rat after five weeks of DDT treatment. Delafield's Haematoxylin-Eosin. X 60.

Fig. 8. Photomicrograph of T.S. of oesophagus of rat after five weeks of DDT treatment showing mucosa. Delafield's Haematoxylin-Eosin. X 300.

Fig. 9. Photomicrograph of T.S. of oesophagus of rat after six weeks of DDT treatment. Delafield's Haematoxylin-Eosin. X 60.
Fig. 10. Photomicrograph of T.S. of oesophagus of rat after six weeks of DDT treatment showing mucosa. Delafield's Haematoxylin-Eosin. X 300.

Fig. 11. Photomicrograph of T.S. of oesophagus of rat after four weeks of BHC treatment. Delafield's Haematoxylin-Eosin. X 60.

Fig. 12. Photomicrograph of T.S. of oesophagus of rat after four weeks of BHC treatment showing mucosa. Delafield's Haematoxylin-Eosin. X 60.
Fig. 13. Photomicrograph of T.S. of oesophagus of rat after five weeks of BHC treatment. Delafield's Haematoxylin-Eosin. X 60.

Fig. 14. Photomicrograph of T.S. of oesophagus of rat after five weeks of BHC treatment showing mucosa. Delafield's Haematoxylin-Eosin. X 300.

Fig. 15. Photomicrograph of T.S. of oesophagus of rat after six weeks of BHC treatment. Delafield's Haematoxylin-Eosin. X 60.
Fig. 16. Photomicrograph of T.S. of oesophagus of rat after six weeks of BHC treatment showing mucosa. Delafield's Haematoxylin-Eosin. X 300.

Fig. 17. Photomicrograph of T.S. of oesophagus of rat after 10 days of malathion treatment. Delafield's Haematoxylin-Eosin. X 60.

Fig. 18. Photomicrograph of T.S. of oesophagus of rat after 20 days of malathion treatment. Delafield's Haematoxylin-Eosin. X 60.
Fig. 19. Photomicrograph of T.S. of oesophagus of rat after 30 days of malathion treatment. Delafield's Haematoxylin-Eosin. X 60.

Fig. 20. Photomicrograph of T.S. of oesophagus of rat after 30 days of malathion treatment showing mucosa. Delafield's Haematoxylin-Eosin. X 300.

Fig. 21. Photomicrograph of T.S. of oesophagus of rat after four weeks of carbaryl treatment. Delafield's Haematoxylin-Eosin. X 60.
Fig. 22. Photomicrograph of T.S. of oesophagus of rat after four weeks of carbaryl treatment showing mucosa. Delafield's Haematoxylin-Eosin. X 300.

Fig. 23. Photomicrograph of T.S. of oesophagus of rat after five weeks of carbaryl treatment. Delafield's Haematoxylin-Eosin. X 300.

Fig. 24. Photomicrograph of T.S. of oesophagus of rat after five weeks of carbaryl treatment showing mucosa. Delafield's Haematoxylin-Eosin. X 300.
Fig. 25. Photomicrograph of T.S. of oesophagus of rat after six weeks of carbaryl treatment showing muscle layers. Delafield's Haematoxylin-Eosin. X 300.

Fig. 26. Photomicrograph of T.S. of oesophagus of rat after six weeks of carbaryl treatment showing mucosa. Delafield's Haematoxylin-Eosin. X 300.
Fig. 27. Photomicrograph of T.S. of stomach of control rat. Delafield's Haematoxylin-Eosin. X 60.

Fig. 28. Photomicrograph of T.S. of stomach of control rat showing submucosa. Delafield's Haematoxylin-Eosin. X 300.

Fig. 29. Photomicrograph of T.S. of stomach of control rat showing mucosa. Delafield's Haematoxylin-Eosin. X 300.
Fig. 30. Photomicrograph of T.S. of stomach of rat after four weeks of DDT treatment. Delafield's Haematoxylin-Eosin. X 60.

Fig. 31. Photomicrograph of T.S. of stomach of rat after four weeks of DDT treatment showing mucosa. Delafield's Haematoxylin-Eosin. X 300.

Fig. 32. Photomicrograph of T.S. of stomach of rat after five weeks of DDT treatment. Delafield's Haematoxylin-Eosin. X 60.
Fig. 32. Photomicrograph of T.S. of stomach of rat after five weeks of DDT treatment. Delafield's Haematoxylin-Eosin. X 300.

Fig. 34. Photomicrograph of T.S. of stomach of rat after six weeks of DDT treatment. Delafield's Haematoxylin-Eosin. X 60.

Fig. 35. Photomicrograph of T.S. of stomach of rat after six weeks of DDT treatment showing mucosa. Delafield's Haematoxylin-Eosin. X 300.
Fig. 36. Photomicrograph of T.S. of stomach of rat after four weeks of BHC treatment. Delafield's Haematoxylin–Eosin. X 60.

Fig. 37. Photomicrograph of T.S. of stomach of rat after five weeks of BHC treatment. Delafield's Haematoxylin–Eosin. X 60.

Fig. 38. Photomicrograph of T.S. stomach of rat after five weeks of BHC treatment showing mucosa. Delafield's Haematoxylin–Eosin. X 300.
Fig. 39. Photomicrograph of T.S. of stomach of rat after six weeks of BHC treatment. Delafield's Haematoxylin-Eosin. X 60.

Fig. 40. Photomicrograph of T.S. of stomach of rat after six weeks of BHC treatment showing mucosa. Delafield's haematoxylin-Eosin. X 300.

Fig. 41. Photomicrograph of T.S. of stomach of rat after 10 days of malathion treatment. Delafield's Haematoxylin-Eosin. X 60.
Fig. 42. Photomicrograph of T.S. of stomach of rat after 10 days of malathion treatment showing submucosa. Delafield's Haematoxylin-Eosin. X 300.

Fig. 43. Photomicrograph of T.S. of stomach of rat after 20 days of malathion treatment. Delafield's Haematoxylin-Eosin. X 60.

Fig. 44. Photomicrograph of T.S. of stomach of rat after 20 days of malathion treatment showing mucosa. Delafield's Haematoxylin-Eosin. X 300.
Fig. 45A  Photomicrograph of T.S. of stomach of rat after 30 days of malathion treatment. Delafield's Haematoxylin-Eosin. X 60.

Fig. 45B  Photomicrograph of T.S. of stomach of rat after 30 days of malathion treatment showing submucosa. Delafield's Haematoxylin-Eosin. X 300.

Fig. 46.  Photomicrograph of T.S. of stomach of rat after 30 days of malathion treatment showing mucosa. Delafield's Haematoxylin-Eosin. X 300.
Fig. 47.  Photomicrograph of T.S. of stomach of rat after four weeks of carbaryl treatment. Delafield's Haematoxylin-Eosin. X 60.

Fig. 48.  Photomicrograph of T.S. of stomach of rat after four weeks of carbaryl treatment showing mucosa. Delafield's Haematoxylin-Eosin. X 300.

Fig. 49.  Photomicrograph of T.S. of stomach of rat after five weeks of carbaryl treatment. Delafield's Haematoxylin-Eosin. X 60.
Fig. 50. Photomicrograph of T.S. of stomach of rat after five weeks of carbaryl treatment showing mucosa. Delafield's Haematoxylin-Eosin. X 300.

Fig. 51. Photomicrograph of T.S. of stomach of rat after six weeks of carbaryl treatment. Delafield's Haematoxylin-Eosin. X 60.

Fig. 52. Photomicrograph of section of stomach of rat after six weeks of carbaryl treatment showing mucosa. Delafield's Haematoxylin-Eosin. X 300.
Fig. 53. Photomicrograph of T.S. of small intestine of control rat. Delafield's Haematoxylin-Eosin. X 200.

Fig. 54. Photomicrograph of T.S. of small intestine of control rat. Delafield's Haematoxylin-Eosin. X 200.

Fig. 55. Photomicrograph of T.S. of small intestine of rat after four weeks of DDT treatment. Delafield's Haematoxylin-Eosin. X 200.
Fig. 56. Photomicrograph of T.S. of small intestine of rat after five weeks of DDT treatment. Delafield's Haematoxylin-Eosin. X 200.

Fig. 57. Photomicrograph of T.S. of small intestine of rat after six weeks of DDT treatment. Delafield's Haematoxylin-Eosin. X 200.

Fig. 58. Photomicrograph of T.S. of small intestine of rat after four weeks of BHC treatment. Delafield's Haematoxylin-Eosin. X 200.
Fig. 59. Photomicrograph of T.S. of small intestine of rat after five weeks of BHC treatment. Delafield's Haematoxylin-Eosin. X 200.

Fig. 60. Photomicrograph of T.S. of small intestine of rat after six weeks of BHC treatment. Delafield's Haematoxylin-Eosin. X 200.

Fig. 61. Photomicrograph of T.S. of small intestine of rat after 10 days of malathion treatment. Delafield's Haematoxylin-Eosin. X 200.
Fig. 62. Photomicrograph of T.S. of small intestine of rat after 20 days of malathion treatment. Delafield’s Haematoxylin-Eosin. X 200.

Fig. 63. Photomicrograph of T.S. of small intestine of rat after 30 days of malathion treatment. Delafield’s Haematoxylin-Eosin. X 200.

Fig. 64. Photomicrograph of T.S. of small intestine of rat after four weeks of carbaryl treatment. Delafield’s Haematoxylin-Eosin. X 200.
Fig. 65. Photomicrograph of T.S. of small intestine of rat after five weeks of carbaryl treatment. Delafield's, Haematoxylin-Eosin. X 200.

Fig. 66. Photomicrograph of T.S. of small intestine of rat after six weeks of carbaryl treatment. Delafield's Haematoxylin-Eosin. X 200.

Fig. 67. Photomicrograph of T.S. of rectum of control rat Delafield's Haematoxylin-Eosin. X 200.
Fig. 68. Photomicrograph of T.S. of rectum of control rat
Delafield's Haematoxylin-Eosin. X 200.

Fig. 69. Photomicrograph of T.S. of rectum of rat after
four weeks of DDT treatment. Delafield's
Haematoxylin-Eosin. X 200.

Fig. 70. Photomicrograph of T.S. of rectum of rat after
five weeks of DDT treatment. Delafield's
Haematoxylin-Eosin. X 200.
Fig. 71. Photomicrograph of T.S. of rectum of rat after six weeks of DDT treatment. Delafield's Haematoxylin-Eosin. X 200.

Fig. 72. Photomicrograph of T.S. of rectum of rat after four weeks of BHC treatment. Delafield's Haematoxylin-Eosin. X 200.

Fig. 73. Photomicrograph of T.S. of rectum of rat after five weeks of BHC treatment. Delafield's Haematoxylin-Eosin. X 200.
Fig. 74. Photomicrograph of T.S. of rectum of rat after six weeks of BHC treatment. Delafield's Haematoxylin-Eosin. X 200.

Fig. 75. Photomicrograph of T.S. of rectum of rat after four weeks of carbaryl treatment. Delafield's Haematoxylin-Eosin. X 200.

Fig. 76. Photomicrograph of T.S. of rectum of rat. After five weeks of carbaryl treatment. Delafield's Haematoxylin-Eosin. X 200.
Fig. 77. Photomicrograph of T.S. of rectum of rat after six weeks of carbaryl treatment. Delafield's Haematoxylin-Eosin. X 200.

Fig. 78. Photomicrograph of T.S. of rectum of rat after 10 days of malathion treatment. Delafield's Haematoxylin-Eosin. X 200.

Fig. 79. Photomicrograph of T.S. of rectum of rat after 20 days of malathion treatment. Delafield's Haematoxylin-Eosin. X 200.
Fig. 80  Photomicrograph of T.S. of rectum of rat after 30 days of malathion treatment. Delafield's Haematoxylin-Eosin. X 200.
3. DISCUSSION

As far as the author is aware of much work has not been
done on the histopathological changes induced by pesticides
on the alimentary canal of mammals including rats. However,
the changes seen due to pesticides are discussed with the work
available in the related field.

Stinson (1979) reported carcinomas viz. Pedunculated
papillary carcinomas, sessile carcinomas in the oesophagus
of rat after exposure to methylalkylnitrosamine. David et al.
(1986) described oesophageal carcinomas showing basal cell
hyperplasia, atypical basal cells, dyskeratotic cells in the
oesophagus of rats by methylbenzyl-nitrosoamine (MBH). However,
in the present study carcinomas could not be observed, though
some other histopathological changes in the oesophagus of
albino rat such as ruptured serosa, vacuolation and space
formation in longitudinal and circular muscle layers, damaged
mucosal layers, ruptured wall of mucosal stratified epithelial
layer, etc., shrunken columnar layer and necrotic mucosal folds
were noticed in the oesophagus of albino rats with all most all
the pesticides treatment in the present study.

Rasanen (1960) observed that surface epithelium of stomach
mucosa was easily detectable and granules of mucosal mast cells
varied greatly in size during cortisone and ACTH exposed rats. Sugimura and Fujimura (1967) reported tumorous lesions in the glandular stomach especially in the pylorus region of stomach of rats due to N-methyl-N-nitro-N-nitrosoguanidine. Definite serosa involvement with the rupture of mucosal layer was also observed by them. Occurrence of squamas cell carcinoma in the fore stomach of rats after administration of N-methyl-N-nitro-N-nitrosoguanidine was reported by Schoental (1966). Bralow et al. (1970) described carcinoma of glandular stomach and duodenum in rats with N-methyl-N-nitro-N-nitrosoguandin and on long term use the treated rats develop tumours and gastric adenocarcinoma, mostly in the antrum, were also reported by them. Tumour formation in the stomach could not be observed in the present investigation. Fujimura et al. (1970) reported a typical epithelial proliferation in the submucosa invading the muscle layer and partly involving the serosa and adenocarcinomas in the glandular stomach and multiple squamous cell papilloma in the fore stomach of rats induced by N-methyl-N-nitro-N-nitrosoguanidin (NG). Formation of tumour in the glandular stomach and epithelial cell proliferation of rats induced by N-methyl-N-nitro-N-nitrosoguandine was reported by Sugimura et al. (1970). Sato (1971) observed pathological changes such as adenoma like proliferation of glands showing cellular atypism of slight degree adenomatus growths in which more remarkable cellular atypism and malignant lesions were identified adenocarcinomas, sarcomas in the glandular stomach of rats.
after exposure to N-methyl-N-nitro-N-nitrosoguanidine. In present study no such results were found but some other pathological changes viz. ruptured submucosa, mucosal tip and wall of mucosal columnar cells, space formation in lamina propria and mucosa, vacuolation, pychotnic nuclei, fused and necrotic longitudinal and circular muscle layer and mucosa was observed during DDT and BHC treatment in the stomach of rats. McCracken et al. (1974) reported gastric lesions such as catarrhal gastritis characterized by necrosis and desquamation of the superficial lyasers of mucosa and necrotizing celluritis of gastric submucosa in the stomach of rats after exposure to Penicillium viridicatum. Wolfsen et al. (1979) observed several morphological changes in the stomach of rats during different experimental stages of N-methyl-N-nitro-N-nitrosogunidine treatment. They reported that epithelium of the gland became homogeneous, formation of erosions in the superficial layer of membrane propria, disturbances in the arrangement and shape of the glands in some areas of the membrane propria, cystic dilation, signs of atypia and possible malignization appeared in the epithelial proliferates and epithelial cell elements which varied in size and appearance of stomach carcinomas. The hemorrhagic lesions, complete mucosal cell necrosis, extensive hyperemia of mucosal and submucosal vessel, damaged and shrunken chief cells in the fundus of the gastric glands, necrotic and lifting off surface
epithelium from the lamina propria, separated surface epithelium from the mucosa, exfoliated surface intervening layer and early evidence of restitution, space between the dead cell layer and mucosal and whole stomach wall including the muscularis externa appeared damaged in the rats stomach due to ethanol toxicity as reported by Ito and Lacy (1985). In the present study, necrotic mucosa and space formation in mucosa, ruptured mucosa, vacuolization, damaged longitudinal and circular muscle layer in the stomach of rats were noticed. Dupuy and Szabo (1986) described that lesions consisted of sharply demarcated areas of hemorrhagic or anemic necrosis, the sub-mucosa showed massive edema and congested blood vessels in some areas and desquamation of necrotic epithelium in the stomach of rats after treatment to ethanol. Necrotic and detached cell of the surface epithelium and decreased submucosa edema due to low doses and large doses of metals resulted in virtually complete mucosal protection with absence of edema has also been reported by them. Barten (1987) observed different types of tumours in the stomach and upper small intestine of albino rats treated with N-methyl-N-nitro-N-nitrosoguadinine. In present work there was no tumour formation. Yoney and Guth (1983) reported several histologic injury accompanied by necrosis in the stomach of rats after exposure to 75% ethanol. They also observed that surface epithelium cell were completely damaged and partly sloughed and neck mucous cells have lost their outlines. In the present study
necrosis in the stomach (mucosal region) of rats were seen. Terao et al. (1988) observed focal erosions on the glandular mucosa, focal necrosis in the mucosal layer and marked edema in the sub-mucosal layers of stomach of rats after exposure to malitotoxin. Several haemorrhagic necrosis in the gastric mucosa, slight exfoliation in the surface epithelium, completely lifting off of upper part of mucosa and deeper damage into the mucosa of stomach due to different doses of ethanol was reported by Takeuchi et al. (1988). In present study no such results were seen, however, ruptured serosa, vacuolation, space formation, damaged longitudinal and circular muscle layer, pycnotic nuclei in mucosal region of stomach was observed after exposure to malathion and carbaryl. Wallace and John (1988) reported that luminal surface of the mucosa was almost completely denuded and on the luminal surface some cellular debris was evident after administration of 1 ml NaCl to rats. He also observed extensive discontinuities in the surface epithelium, deeper mucosal necrosis and haemorrhage, and mucous and cellular debries on the luminal surface of the gastric cropus of rats during administration of ethanol or pretreatment with 1 ml NaCl plus ethanol and indomethacin plus ethanol treatment. Tatsuta et al. (1988) observed histological changes in stomach of rat treated with NaOH. They reported that fundic mucosa showed slight atrophy with foveolar hyperplasia and in the antral mucosa, moderate or several atrophy with foveolar hyperplasia and atypical glandular hyperplasia was observed.
The significant increase in fundic surface area after administration of tetragastrin plus NaCl treatment was also reported by them. No such results were observed in present work.

Kameda et al. (1968) reported that damage to surface epithelium was mostly marked at the tip of the villi, cell injury and dilation of the lymphatic spaces in the intestine of rats was induced by isotonic and hypertonic solution.

Benitz et al. (1973) studied pathological changes such as mucosal erosions, cellular infiltration, granulation in the lamina propria, formation of multiple crypt abscesses, necrotic epithelium and slight oedema, etc. in colon of monkey after exposure to carrageenan. In the present study such changes could not be seen in the small intestine of rats but other histopathological changes such as ruptured wall of villi, vacuolation, damaged lining of columnar cells having vacuolated submucosa, fused Bruner’s glands having degenerated cell and vacuoles and space formation and vacuolation in submucosa of intestine of rats after exposure to DPT was noticed. Mucosal hyperaemia, necrosis of mononuclear cells of the lamina propria and cellular congestion in the duodenum, Jejunum, ileum and colon of guinea pig due to ochratoxin were reported by Thacker and Carlton (1977). Shira et al. (1979) described that villi of small intestine became irregular and reduced in height. Swelling of the mucosa, dilation of blood vessels, necrotic lesions in the intestinal mucosa and coagulation necrosis and
fragmentation of muscular layers of small intestine after exposure to *Salmonella* infection were reported by them. No such results were seen in the present study. However, after exposure to EMS, ruptured villi with clumped cells and vacuolation, space formation in lamina propria and submucosa, vacuolated submucosa and shrunken Bruner's glands were seen in the small intestine of *Rattus rattus* albino. Nahta and Singh (1979) reported shrinkage in all segment of the wall and mucosal layer, flattening of villi, deeply stained nuclei, pyknotic nuclei, clumping of cells and desquamation of cell at the tip of villi of intestine of rats due to hypertonic solution. In present study clumping of cells was noticed in the villi of small intestine after treatment with pesticides. Kumar et al. (1988) reported cytopathological findings in two cases (men and women) of alpha chain disease. They observed that first biopsy of duodenum showed deformed and shortend villi and a moderate lymphoplasmacytic infiltration in the lamina propria and the second biopsy showed increased lymphoplasmacytic, infiltration in the lamina propria, extending into the surface epithelium and submucosa and markedly deformed villi. In the present work malathion and carbar-1 also causes toxic and necrotic changes in the small intestine of rats such as ruptured villi having space formation, vacuolation and clumping of cells, vacuolated lamina propria, space and vacuolation in submucosa and necrotic longitudinal and circular muscle layers.
Jackson (1974), studied adenomas in the colon and rectum of human patients. Day et al. (1978) reported mucosal oedema, focal inflammation, crypt abscesses and mucous depletion, etc. in the rectum of 22 patients due to Salmonella infection. Chronic inflammation, acute and subacute inflammation, architectural distortion, infiltration with mononuclear cells, epithelial regeneration and infiltration of the lamina propria and epithelium, polymorphonuclear leukocytes and epithelial distroation in colon of monkeys with spontaneous colitis was reported by Allen et al. (1988). In the present study such changes could not be seen. However, some other pathological changes have been seen in the rectum of rat due to pesticides treatment. The changes noticed are necrotic serosa, space formation in longitudinal and circular muscle layers, vacuolated submucosa, mucosa and lamina propria and ruptured columnar epithelial layer and wall of mucosa in case of DDT treatment. Formation of space in longitudinal and circular muscle layer, and lamina propria, ruptured mucosal wall having clumped cells and space formation, vacuolated mucosa and submucosa in case of BHC treatment. Hypertrophied serosa, longitudinal and circular muscle layer, necrotic submucosa with space formation and vacuolation, ruptured mucosal wall and mucosal tip, etc. in case of malathion. Hypertrophied serosa, longitudinal and circular muscle layer, necrotic submucosa with space formation and vacuolation, ruptured mucosal wall and mucosal tip and vacuolation in case of carbaryl.
Thus the present investigation confirms that different pesticides, if enter the body of any mammal including man, will damage the normal histological architecture of entire alimentary canal. Due to this damage this is definite that the normal and natural physiology of the animal will be disrupted and the animal/individual will face lot of digestive problems resulting in the ill health.

As such it is concluded that the pesticides even at sub-lethal dose level are harmful to rat as is evident by the present study.