CHAPTER - VI
SUMMARY AND CONCLUSIONS

The albino rats were administered intramuscularly with active ingredient, azadirachtin for 8, 16 and 24 days.

Azadirachtin administered albino rats exhibited no significant decrease in body weight but slight negligible decrease was found. This non significant decrease in body weight may be due to low intake of food and water due to effect of azadirachtin. It can be also concluded that this negligible loss in weight of body may be resulted due to hypoglycemia caused by the azadirachtin.

The treatment of azadirachtin has no effect on the body size directly but more investigations will be appreciated in this regard.

A progressive decrease in weight of liver of male and female albino rats was observed when albino rats treated with azadirachtin and this loss in weight may be due to the decrease in glycogen content, disintegration of liver cells, hypoglycemia and inactivation of metabolic rate.

The treatment of azadirachtin was resulted into the decrease in kidney weight of male and female rats which may be due to the hepatotoxicity caused by azadirachtin in liver which disturb the balance between the rate of generation of reactive metabolites and their removal. Secondly, this decrease in kidney weight may be resulted due to non accumulation of cellular water in kidney by the action of azadirachtin.

The azadirachtin affects the weight of adrenal gland. The decrease in the weight of adrenal gland which may due to shrinking of cells of zona glomerulosa and degeneration of cells of zona fasciculata.
Intramural administration of azadirachtin resulted in a decrease in the weight of pancreas in albino rats of both sexes which may be due to vacuolisation in acinar cells and degeneration of undifferentiated cells or 'C' cells or 'Y' cells of islet of Langerhaus caused by the effect of azadirachtin.

Administration of azadirachtin in albino rats was resulted into reduction in weight of ovary which may be due to distortion of ovarian follicles, vacuolisation in cytoplasm of peripheral oocytes and nutrient malabsorption caused by the action of azadirachtin.

The azadirachtin treated rats showed progressive decrease in weight of testis and this loss in weight may be due to antispermatogenic effect, antiandrogenic effect and marked decrease in spermatogonia, spermatocytes and spermatids counting caused by the azadirachtin.

The weight of cauda and caput epididymis was significantly decreased when male rats treated with azadirachtin which may be due to the lack of availability of androgen and serum testosterone which may be due to the effect of azadirachtin. It may also be concluded that the decrease in normal sperm counting, increase in abnormal sperm counting, declination in spermatozoa and regression in epididymal epithelium caused by the action of azadirachtin which are responsible for such weight loss.

Administration of azadirachtin in albino rats has resulted into the progressive decrease in blood glucose level which may be due to the hypertrophy in medullary cells of adrenal gland, vacuolisation of zona fasciculata of cortex of adrenal gland which resulted into decrease in epinephrine release which finally inhibited the glycogenolysis in liver cells to develop hypoglycemia.
The treatment of azadirachtin may responsible for the degeneration of undifferentiated cells or 'C' cells or 'Y' cells of islets and vacuolisation in acini of pancreas. Thus, it can be established fact that azadirachtin affect the histology of pancreas.

The azadirachtin treatment resulted into decrease in the diameter of testis which may be due to the shrinking of interstitial cells and degeneration of primary and secondary spermatocytes. The reduction in the counting of spermatogonia, spermatocytes and spermatids was noted which may be exerted due to the degeneration of the germinal cells and arrest of process of spermatogenesis due to the effect of azadirachtin.

Administration of azadirachtin resulted into the reduction in diameter of seminiferous tubule which may be exerted due to the invasion of gonial elements into the lumen of seminiferous tubules. A decrease in sperm motility may be exerted due to changes in microenvironment of seminiferous tubule by the action of azadirachtin.

The decrease in normal sperm counting and increase in abnormal sperm counting may be disarrangement and decrease in size of germinal epithelium. This brought the reduction in number of spermatids which then responsible for decrease in daily sperm production in testis. Such resulted low sperm production then led to decrease sperm release in the epididymis of treated rats which caused a significant sperm abnormality in all treated groups.

The damages in the testicular histology after azadirachtin treatment was significant and greater. The major histological damages were seen in the seminiferous tubule such as reduced number of spermatogonia, spermatocytes and spermatids. Thus, azadirachtin has antifertility effect on testis in our investigation also.
Administration of azadirachtin in albino rats was resulted into the desentigration of corona radiata, distortion of follicles and vacuolisation in the cytoplasm of pheripheral primary oocytes may be due to the diminution of steroidogenesis in ovary which brought about by the action of azadirachtin. These disturbances in ovary caused by azadirachtin inhibit the ovulation. Thus, it can be established that azadirachtin has antifertility effect on ovary also.

The azadirachtin has adverse effect on the histology of adrenal gland such as vacuolisation in the cells of zona glomerulosa, zona fasciculata hypertrophy in medullary cells, loss of orientation in cortical cells and degeneration of cells of zona reticularis of cortex which may decrease the level of epinephrine which affect the reproductive organs.

The azadirachtin treatment brought an increase in the R.B.C. counting which may be due to the hyperstimulation to erythropoetic tissue which may be caused by the stress of azadirachtin. They resulted increase in W.B.C. counting may be due to the change in differential leucocyte counting which may be brought about by the action of azadirachtin.

The azadirachtin treated rats showed increased in E.S.R. which may be due to the increase in R.B.C. counting due to the action of azadirachtin. The increase in P.V.C. of experimental albino rats was noted which may be due to the increase in the haemoglobin percentage due to the effect of azadirachtin but the M.C.V. level of same rat was not affected. Thus, it can be concluded that azadirachtin has effect on E.S.R. and P.C.V. but not on M.C.V.

The dose dependant increase in haemoglobin percentage was noted in experimental rats which may be due to the stress exerted by the
administration of azadirachtin. A fluctuated decrease and increase in clotting and bleeding time of blood was noted which may be exerted due to the stress of azadirachtin or environmental factors or changes in haematological parameters.

Administration of azadirachtin resulted into the fluctuation i.e. decrease or increase in differential leucocyte counting which may be due to the action of azadirachtin. Thus, it can be revealed that azadirachtin affect the differential leucocytes counting.

The azadirachtin treatment resulted into an increase in the protein concentration in kidney of albino rats which may be due to the involved detoxification process in the renal tissue which may be exerted by the action of azadirachtin. The same treatment was responsible to increase the protein concentration in liver of albino rats which may be due to the increase in the process of synthesis of protein due to the influence of azadirachtin.

The treatment of azadirachtin may responsible for the increase in protein concentration in pancreas of the albino rats due to the decreasing influence on pancreatic trypsin and chymotrypsin activities caused by the treatment of azadirachtin.

Thus, it can be concluded that azadirachtin play an important role in the physiological and biochemical process in albino rats but for detail results more investigations will be highly appreciated in this regard.

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