CHAPTER-11

CONCLUSION

11.0. Conclusion

The metalloid arsenic turns into a new headache for the health of the civilization. It is popular as the slow toxin for the living body. Several paths have been acknowledged for its transmission into the human organisms but drinking water becomes the powerful and easy route for its exposure. Both acute and persistent introduction in human creates sweltering consequences globally. Arsenic extremely caused trouble in metabolic, reproductive and other system in human body. The initiation of oxidative stress and its consequences have often been noticed by arsenic. Ovarian-uterine deformities, abrupted follicular maturation, disproportion of estradiol and infertility all have been well defined with arsenication. Different management plans are being implemented for minimizing arsenic primed deleterious health outcome. Chelating substances have been broadly employed for treating arsenic hazards though these are having numerous side effects with minimum therapeutic worth. However, prolonged curing actions with invasive muscular administration of conventional chelating substance sometimes facilitate re-distribution of this toxic component in brain. Many researchers have considered establishing a proficient and valuable management way against arsenic aggravated repro-malfunctions in female animal model. Therefore, this study was availed to develop as well as expand the therapeutic manifestation of NAC with antioxidant proficiency with least toxic hazards in abolishing arsenic oriented repro-hazards in females. Here, various dosages of NAC have been implemented and the outcome of this bio-substance was documented in protective, preventive plus curative mode along with its direct action against arsenication on ovarian-uterine dysfunctions. The sodium arsenite consumed rat models were challenged with NAC in several diverge

conditions and their output were observed. Here, Wistar strain female animal models were undertaken for experimental purpose. Two separate dosages of NAC i.e. 50 mg per kg body weight plus 100 mg per kg body weight were allowed for oral application initially to oppose arsenicated dysfunctions. But during dietary application this dose was changed to a higher dose of 250 mg per kg body weight, because NAC was administered via diet therefore there might be a possibility to wastage of food by the animals. After standardization of dose 100 mg per kg body weight of NAC orally was regarded for further investigation on rats having 10 mg sodium arsenite per kg body weight. Oxidative stress and its toxic consequences were well managed by NAC. ROS production was perturbed in definite way with concomitant up-gradation of enzymatic antioxidants in ovarian-uterine tissues with the oral delivery of NAC. Also, DNA repairing in uterus, successful folliculogenesis, and usual estradiol surge with steady ER-α expression were re-established with NAC application against arsenite. Estradiol normalization unambiguously assisted for renovation of ovarian-uterine tissues which were drastically affected with arsenite due to poor estradiol level. During histological examination numbers of matured and growing follicles were noted in ovary and renovation of uterine layers with growing secretory glands were found in uterus. This led to the lessening of the vulnerability of abnormal steroidogenesis by rhythmic functioning of steroidogenic enzymes at utmost level. Besides, NAC is able to counteract against inflammatory response and apoptotic sequence caused by arsenite. Arsenic directed adverse condition of pro-inflammatory indicators i.e. TNF-α, NF-κB plus IL-6 could be surprisely amended by NAC introduction. Arsenic encouraged the apoptotic indicator Bax gene appearance and p53 gene expression at supreme level wherein decreased Bcl-2 gene expression and these

circumstances were successively overcome by NAC addition. Moreover, the dietary NAC post-delivery documented the worthful effect about the above mentioned parameters degradation against arsenite in model animals. However, NAC also extended its corrective action against arsenite in liver and kidney where NAC suppressed the over activities of SGOT and SGPT plus creatinine levels in circulation. The *in-vitro* approach explored that NAC probably has direct action to stop arsenite propagated hepatic plus ovarian-uterine malicious outcomes. The preventive plus curative strategy of NAC was expected to be the most significant therapeutic strategy against arsenication. In all experiments NAC at the dosage of 100 mg per kg body weight was more reproducible to eliminate sodium arsenite primed toxic ailments.

Furthermore, this bio-substance, NAC might be accelerated arsenic clearance through enhancing B vitamins. NAC is the source of cysteine residue and thus provided the basic raw materials (cysteine) for proper functioning of enzyme (arsenic methyl transferase) liable for arsenic methylation. The chelating position of NAC provides another gateway for arsenic removal from the system. Together with these features, NAC also keeps the homeostasis of redox condition by triggering GSH creation inside the repro-system and thereby preserves healthy structure of ovarian-uterine tissues. From the preceding consequences of NAC against arsenite on animal model it might be considered for human trial to antagonize arsenic aided female repro-abnormalities via phase wise in depth study. This may be a safe nutraceutical cum medicine to correct arsenic primed reproductive intoxication with precious therapeutic worth.