Conclusions
CONCLUSIONS

The results of the studies done exclusively on *Nostoc muscorum* and described in this thesis led to the following conclusions:

1) A class of tungsten resistant (W-R) mutants of the cyanobacterium were found severely defective in Mo-transport activity and required vanadium (V) for growth on N₂ or NO as nitrogen source. The requirement of V for nitrate nutrition is the first novel finding reported for any microbial system.

2) *N. muscorum* can make or regulate V-dependent nitrogenase in the absence of active V-dependent nitrate reductase (NR) and vice versa as it makes or regulates its Mo-nitrogenase or Mo-NR independently.

3) The physiology of salinity stress on Mo- or V-dependent diazotrophic growth appears to be similar.

4) Cyanobacterial sensitivity to salinity and osmotic stresses is primarily because of its greater sensitivity to its N₂-fixation process than to its photosynthetic activity.

5) Mutation to salinity or osmotic stress resistance in the cyanobacterium results in severe curtailment in Na influx.

6) NH₃-nitrogen does not offer any protection to the cyanobacterium against salinity or osmotic stress induced lethality and plays no role in regulating Na transport.

7) Exogenous proline is used as a nitrogen source under unstressed conditions while it serves as an osmoprotectant under salinity or osmotic stressed conditions in the cyanobacterium.

8) Physiological responses of the cyanobacterium to osmotic and salinity stresses appear to be similar.
Conclusions

9) The cyanobacterial l-azetidine-2-carboxylate resistant (Ac-R) mutant is a proline overaccumulating strain and shows tolerance to salinity and as well as osmotic stresses.

10) Mutation to the Ac-R phenotype is accompanied by a loss in proline oxidase activity associated with inability to assimilate proline as a nitrogen source. A definite role of proline oxidase in regulating nitrogen nutrition or osmoprotective function of proline is evidenced in the cyanobacterium.

11) Mutational loss in proline oxidase activity seems to be the reason for overaccumulation of proline (leading to high intracellular level of proline) in the Ac-R mutant strain.

12) Proline per se is not the repressor of heterocyst formation and nitrogenase activity in the cyanobacterium.

13) Exogenous betaine functions as a nitrogen source in the cyanobacterium and fails to function as an osmo(salinity)-protectant in it.

14) Betaine does not share a common transport with proline in the cyanobacterium.


16) Spontaneous cyanobacterial mutants resistant to growth toxic effects of alkali metals (Li, Na & Rb) and alkaline pH (pH 11.0) show an enhanced H-gradient dependent multiple alkali cation efflux system and are found sensitive to sucrose-induced osmotic stress.

17) The cyanobacterium N. muscorum shows a definite requirement for Na and K/Rb for optimal growth under diazotrophic growth conditions.
18) Cs uptake and toxicity is diazotrophy-specific and NH₄-repressible.

19) Mutation to caesium resistance phenotype (Cs -R) results in physiological pleiotropy manifest in the form of impaired diazotrophic growth, oxygenic photosynthesis, chlorophyll a content, nitrogenase activity and osmotolerance.

20) Cs /Rb alone is found capable of restoring fully the physiological pleiotropy of the Cs -F mutant strain to its normal level.