Conclusion
5. CONCLUSION

The altered urinary risk factor profile exhibited by hyperoxaluric individuals and rats were restored by vitamin E therapy and pre-treatment respectively. Of the three major calcium oxalate monohydrate binding proteins isolated in the present study, one was found to be promoting and the other two inhibitory in nature. Interestingly, the excretion of this promoter protein was enhanced and inhibitory protein decreased during stone forming condition. The promoting activity of the promoter protein was aggravated which was responsible for the drive towards crystallization. However, increased excretion along with increased promoting activity of the promoter protein was attenuated by vitamin E supplementation. The efficacy of vitamin E pre-treatment as assessed by the tissue status of the crystallization promoter protein gave satisfactory positive results. Hence, this study brings into light the significance of the protein thiol groups in the lithogenic process and we can conclude that curtailing lipid peroxidation and maintenance of thiol groups of lithogenic proteins essentially reduces the risk of stone formation. Surprisingly, the molecular weight of the protein in both the species vary, the reason behind which will form the basis of the future research work. The effect of proteins in the complicated urinary milieu cannot be predicted in a proper manner with the work done with isolated proteins, as the inhibitor/promoter proteins act in concert and they oppose the activity of one another. Hence future directions of work should be aimed to resolve this. The scientific community is wondered by the fact that rats can stimulate hyperoxaluria, but they cannot renal stones as humans unless
otherwise a foreign body is implanted in the bladder. One of the major observations in the present study is that the molecular weights of the protein in both the species vary, which might be one among the reasons that protect rats from stone formation. Hence, future research should be aimed to resolve the mystery behind the difference in molecular weights.
PROPOSED MECHANISM

Calcium (increased in hyperoxaluria)

Fraction II and III
(COD) in control

Decreased with less inhibitory activity in stone forming condition

No aggregation
No retention

No stone formation

Oxalate (increased in hyperoxaluria)

Free radicals

Damage to cells

Fraction I

Modified (loss of thiol)

COM (Stone formers)

Increased aggregation

Crystal retention

Influences the reaction

Steps inhibited by Vitamin E

Promotes the process

Inhibits the process

and during Vitamin E treatment

Mature stone