7. SUMMARY AND CONCLUSIONS

The conclusions drawn from the current study are:

1. The ability of proteins to remain functional depends on their conformational stability. Further, considering the applications of jacalin in diverse fields, the ability of additives to increase the stability and activity of jacalin was analyzed. Sub-micellar concentration of SDS was demonstrated to confer protection to jacalin, against thermal-induced aggregation. Most importantly, the biological activity of the native protein was found to be retained when the protein was subjected to thermal denaturation, in the presence of SDS.

2. There are various studies hailing the anti-cancer activities of jacalin on cancer cells of different lineages. However, in this study, the mitogenic facet of jacalin was investigated upon, thus, bringing into focus as to whether regular intake of specific food by cancer patients can have detrimental effects. Jacalin treatment resulted in sustained mitogenic effect on the proliferation of K562 cells. It is noteworthy that jacalin was also capable of inducing homotypic aggregation of cells, thereby conferring resistance. Further, jacalin-induced mitogenic effect was perceived to occur as a consequence of activation/deregulation of certain signaling pathway as a result of secretion of certain soluble mediators. Thus, the current *in vitro* study suggests that the consumption of jacalin may be harmful to susceptible individuals; however, this observation needs to be validated by *in vivo* studies.

3. When the possible mechanism underlying the mitogenic effects of jacalin was assessed, Cav-1 was speculated to play the intermediate role. An increase in expression of Cav-1 and increased ERK and AKT (Thr 308) phosphorylation were observed in jacalin-treated K562 cells. Cav-1 is known to interact with PP2A through the scaffold binding domain and hinder the normal catalytic functions of the enzyme. It is possible that, Cav-1, the expression of which was found to be increased in jacalin-treated K562 cells interacts with and disturbs the catalytic activity of PP2A. Since the activity of PP2A may be inhibited, the possibility of incessant phosphorylation of its target proteins such as MAPK,

MEK and AKT, cannot be ruled out. Thus, the sustained phosphorylation of AKT and ERK may probably be the reason behind the observed increase in K562 cell proliferation.

4. Several levels of crosstalk occurs between the tumor cells and surrounding immunological microenvironment. Besides, the effects of lectins on cancerous cells has been the focus of numerous studies. Thus, in order to decipher the effects of jacalin on the modulation of immune responses, the effects of jacalin on the PBMCs were assessed. When the PBMCs were treated with jacalin for a shorter time point, increased mRNA expression of pro-inflammatory cytokine IFN-γ was observed. However, prolonged stimulation of PBMCs resulted in increased expression of anti-inflammatory cytokine, mainly TGF-β. Immune cells are known to modulate tumor-specific immune responses through secretion of cytokines. Also, as cytokines, regardless of their source can stimulate or inhibit tumor growth, the effects of jacalin-prestimulated PBMCs on the HeLa cell proliferation was assessed. While a significant decrease in cell growth was detected in HeLa cells that were directly cultured with the 6 h jacalin-stimulated PBMCs, an increase in cell growth was detected when the HeLa cells were directly cultured with the 24 h jacalin-stimulated PBMCs. Besides, CM obtained from the jacalin-treated PBMCs had no substantial effects on the viability of cancer cells. As lectins are an inevitable part of our daily diet, these observations can have substantial importance under *in vivo* conditions.