8. CONCLUSION

Exploring the nature and degree of HIV-specific functional CD8+ and CD8- T-cell responses between LTNP and progressors reveals no major differences which are contrasting to other major studies yet interesting. It is unclear whether the results derived were because of the selection of peptides spanning the entire length rather than the particular region of the studied epitopes. But LTNP were shown to mount higher responses even to complete epitopes. Evidences also suggest that HIV-specific responses succeed only in a small number of people and fails in most. Taking these factors into consideration, studying and analyzing viral and genetic factors might draw a convincing conclusion for disease progression in LTNP especially in the region were this study is conducted. It can also be hypothesized that ethnic and HIV-1 subtype variations, as evidenced from earlier studies could be the likely factors influencing disease non-progression of LTNP.

Activation rates can be used as an appropriate marker to monitor disease progression in LTNP, since it increases when the PVL levels are >2000 copies/mL and their inverse correlation with T-regs exhibits the equilibrium maintained in LTNP resulting non-progression. Humoral immune activity in terms of memory B-cells phenotyping and bNAb frequencies in LTNP does not provide significant evidences of their roles in disease non-progression. Cytokines and chemokines studied were lower in LTNP than progressors signifies the impact of constant inflammation which might not be seen in
LTNP. Though the HIV-specific responses in this study were contrasting to other studies it is observed that proportion of LTNP that respond to HIV antigens were higher which reveals that there is a sustained protective T-cell activity in LTNP. The quality of such sustained HIV-specific responses should be characterized by studying its virus neutralization abilities along with other T-cell subset dynamics. In addition, tandem observation of viral and host-genetic factors in LTNP might complete the understanding of HIV pathogenesis during non-progression.