

CHAPTER VI

CONCLUSION

In the present study, the effects of normal HDL and AP-HDL on the growth of *E. coli* and *S. epidermidis* were examined *in vitro*. We found that both normal HDL and AP-HDL, up to the physiological concentrations, could not significantly suppress the growth of *E. coli* or *S. epidermidis*.

In addition, the effects of normal HDL and AP-HDL on LPS-induced leukocyte adhesion on endothelial cells *in vivo* were also studied. We found that both normal HDL and AP-HDL were able to significantly inhibit LPS-induced leukocyte adhesion on endothelial cells in a dose-dependent manner, although AP-HDL appeared to be more effective than normal HDL. The inhibitory effect of HDL on LPS-induced leukocyte adhesion was due to the action of HDL on LPS, rather than on endothelial cells, because neutralization of LPS by HDL required incubation of LPS with HDL and HDL itself did not have any effect on endothelial cells.

Furthermore, we found that the protein component of HDL was responsible for the inhibitory effect of HDL on LPS-induced leukocyte adhesion on endothelial cells, whereas, the lipid component had no effect. Similarly, apo A-I, the major protein of HDL, could also significantly attenuate LPS-induced leukocyte adhesion on endothelial cells.