Chapter 7

Summary & Conclusions

1. Novel profiles that do not conform to classical dose-response effects were observed in the *in vitro* studies. Incorporation of redox additives (esp. type II binders) in heme-enzyme reactions resulted in activations and inhibitions of reactions. The same additive molecules could both inhibit and activate reactions in the presence of the same reaction components. A molecule hitherto recognized only as an inhibitor could also activate reactions and vice versa. This is explained by the occurrence of competing reactions and stability of intermediates in the reaction milieu.

2. Type-II binding, which was originally attributed to be the reason for inhibitions, is over-rated. This work (steady state kinetic studies, spectral binding and docking studies) reveals that type II binding is not the reason for observed inhibitions. The effects of stored Phz/Azd observed in this work have immense implications in physiological realms and in routine analytical procedures like ELISA.

3. Non-active site reactions hold significant sway over reaction outcomes. The newly proposed reaction network better explains the diversity and promiscuity of one-electron reactions in heme-enzyme systems.

4. The modulation of heme-enzyme reactions by redox active molecules *in vivo* adds an additional tier of regulation, apart from transcription and translation; also, loss of metabolic control is more forthrightly explained by the bizarre modulatory effects of the redox active molecules.

5. The findings of this work afford a molecular basis for explaining the long-standing biological conundrum of hormesis.