# CHAPTER VI

# CONCLUSION

The present study found that 60 mg/d and 300 mg/d aspirin provide similar effect to inhibit platelet aggregation in type 2 diabetic patient. Sixty mg/d aspirin can inhibit approximately 95% of serum thromboxane $B_2$ level in type 2 diabetic patients which met the requirement for inhibit of the platelet aggregation in most patients.

This study investigated aspirin resistance in type 2 diabetic patients received 60-300 mg/d aspirin assessed by optical platelet aggregation and defined aspirin resistance as a mean aggregation $\geq$ 70% with 10 umol/l ADP and a mean aggregation $\geq$ 20% with 0.5 mg/ml arachidonic acid and found that frequency of aspirin resistance was 6.19%, aspirin semi-responder was 25.77% and aspirin sensitive was 68.04%. In this study, aspirin resistance did not associated with sex, age, BMI, fasting plasma glucose, HbA1c, insulin resistance or lipid profile. However HbA1c was associated with platelet aggregation induced by arachidonic acid and hemoglobin and hematocrit were associated with platelet aggregation induced by ADP.

These data suggest that patients with T2DM do not have increased resistance to aspirin when evaluated by the same method and definition. And use of 60 mg/d does not increased resistance to aspirin. Until longitudinal studies verify whether diabetic patients are less responsive to aspirin therapy than other high-risk patients, this study suggest that aspirin is a reasonable first-line antiplatelet agent in patients with diabetes, as in other populations at high risk for cardiovascular disease. This study also suggest that 60 mg/d aspirin is effective as 300 mg/d aspirin to inhibit platelet aggregation in type 2 diabetic patients with good glucose control and have no acute cardiovascular conditions.