Excess glucose abolished the platelet inhibitory effect of low aspirin concentration but had no effect on inhibition by high aspirin concentration. Similar findings were demonstrated in 23 healthy controls with low aspirin concentration having a non-significant effect on platelet inhibition in the presence of excess glucose (69.2±5.0, 64.3±4.8; for 0 and 1 mg/l aspirin, $p>0.05$ and 37.6±4.7 au for 10 mg/l, $p<0.05$). Aspirin had no effect on ADP-induced platelet stimulation in the diabetes group or controls.
Fibrinolysis in individuals with diabetes showed no difference in the presence of 0, 1 and 10 mg/L aspirin (654±49, 672±43.8 and 657.7±41.5 sec, respectively; $p>0.1$). This was not affected by adding excess glucose to blood samples. In contrast, fibrin clot lysis was enhanced by aspirin treatment in healthy controls (600±63.5, 475±26.6 and 489±29 sec, respectively; $p<0.05$). However, facilitation of fibrinolysis by aspirin in the control group was abolished when excess glucose was added to blood samples.

Summary:
Our data indicate that medium term glycemic control and high glucose concentrations affect platelet response to low aspirin concentrations following AA stimulation. Moreover, the fibrinolytic properties of aspirin are lost in diabetes, which appear to be due to both chronic and acute elevation of blood glucose levels. Future work is warranted to investigate the relationship between clinical aspirin treatment failure and medium/short term glycaemia in aspirin-treated individuals with diabetes.

Keywords: Aspirin, Diabetes, Platelet, Fibrinogen.

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