

# Reduced Blood Platelet Sensitivity to Aspirin in Coronary Artery Disease: Are Dyslipidaemia and Inflammatory States Possible Factors Predisposing to Sub-optimal Platelet Response to Aspirin?

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**Abstract:** The study was designed to assess blood platelet sensitivity to acetylsalicylic acid and its associations with dyslipidaemia and inflammation in coronary artery disease patients. Platelet non-responsiveness to aspirin is associated with an increased risk of serious cardiovascular events. Several environmental and hereditary factors are reportedly involved in sub-optimal acetylsalicylic acid response. Forty-five coronary artery disease patients and 45 non-coronary artery disease controls received acetylsalicylic acid at a daily dose of 75–150 mg. Controls were examined twice: on the day of entering the study and 10 days later. Urinary 11-dehydrothromboxane B<sub>2</sub> was assessed as the marker of platelet thromboxane generation. Aggregation was studied in platelet-rich plasma using turbidimetric aggregometry with collagen and arachidonic acid. Fifty to seventy percent of coronary artery disease patients showed an extent of collagen-induced aggregation above the upper quartile of the reference range compared with 8–15% in controls ( $P < 0.003$ ). For arachidonic acid-activated aggregation these proportions were 45–50% in coronary artery disease versus 7% in controls ( $P < 0.007$ ). In coronary artery disease patients, the acetylsalicylic acid-mediated platelet inhibition positively correlated with increased triglycerides (in arachidonic acid-stimulated platelets,  $r = 0.30$ ,  $P = 0.0018$ ), total cholesterol ( $r = 0.33$ ,  $P < 0.0001$  in coll and arachidonic acid-activated platelets) and elevated serum C-reactive protein (CRP) ( $r = 0.27$ ,  $P = 0.0024$ ). In coronary artery disease patients urine 11-dehydrothromboxane B<sub>2</sub> concentrations were significantly increased compared to controls after 10 day acetylsalicylic acid intake (563; 313–728 pg/mg creatinine versus 321; 246–488 pg/mg creatinine,  $P = 0.04$ ). The incidence of suboptimal acetylsalicylic acid response incidence was more common in patients with coronary artery disease. Acetylsalicylic acid inhibition of blood platelet reactivity and thromboxane generation was less effective in these patients. Dyslipidaemia and chronic inflammatory states may promote suboptimal acetylsalicylic acid response in coronary artery disease patients.