The thromboxane A\textsubscript{2} pathway plays a major role in governing the reactions involved in both the inflammatory and hemostatic biological mechanisms. Cyclooxygenases 1 & 2 act to convert arachidonic acid to thromboxane A\textsubscript{2}.

Scientific studies report cyclooxygenase-2 is up-regulated in many different chronic disease states as well as in chronic inflammation. This explains one major causes of residual thromboxane risk, “aspirin resistance.”

Levels of urinary 11-dehydrothromboxane B\textsubscript{2} reflect activity of components of the thromboxane A\textsubscript{2} pathway that result in thromboxane A\textsubscript{2} generation.

The assay is normalized to the patient’s urine creatinine concentration and is non-invasive and normalized utilizing standard controls.

### Aspirin

1. **Incomplete inhibition of thromboxane biosynthesis by acetylsalicylic acid**
   2008 • Determinants & effects on cardiovascular risk • Clinical Research • 3261 Subjects • Human • Urine • 11-DHTXB2
   
   "In aspirin-treated patients, urinary concentrations of 11-dehydrothromboxane B2 are an externally valid and potentially modifiable determinant of stroke, myocardial infarction, or cardiovascular death in patients at risk for atherothrombotic events."

2. **Aspirin-resistant thromboxane biosynthesis and the risk of myocardial infarction, stoke, or cardiovascular death in patients at high risk for cardiovascular events**
   2002 • Clinical Research • 5529 Subjects • Human • Urine • 11-DHTXB2
   
   "Among aspirin-treated patients at high risk of cardiovascular events, persistent thromboxane generation predicts the risk of the composite outcome of myocardial infarction, stroke, or cardiovascular death, independent of other cardiovascular risk factors."
3. **The Current and Future Landscape of Urinary Thromboxane Testing to Evaluate Atherothrombotic Risk**
   2014 • Review Article • Human • Cell Culture and Urine • 11-DHTXB2
   
   “Aspirin use as been shown to cause a dose-dependent reduction in urinary levels of 11-dehydroTxB2.”

4. **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?**
   2005 • Blood • Human • 45 Patients • Urine • 11-DHTXB2
   
   “Both environmental and genetic factors, including aspirin pharmacokinetics, inflammation, platelet COX-2, use of non-steroid anti-ainflammatory drugs and dyslipidaemia may determine variable platelet response to acetylsalicylic acid.”

   “However, CRP level was significantly associated with the extent of platelet refractoriness to acetylsalicylic acid in these patients, which points out that even subclinical inflammatory states may be considered possible candidates for suboptimal acetylsalicylic acid response.”

5. **The influence of aspirin dose and glycemic control on platelet inhibition in patients with type 2 diabetes mellitus**
   2012 • J Thromb Haemost • Clinical Research • Human • 25 controls/94 patients • 11-DHTXB2
   
   “Our baseline results are in line with earlier studies, which have shown an association between glycemic control and urinary 11-DHTXB2 excretion. In addition, improving glycemic control has been shown to lead to a decreased 11-DHTXB2 excretion.”

   “In the present study, the difference in urinary 11-DHTXB2 excretion was also influenced by C-reactive protein levels, which were different between the study groups and may reflect the influence of the inflammatory state found in diabetes on thromboxane formation.”

6. **Urinary 11-dehydro thromboxane, B2 levels in type 2 diabetic patients before and during aspirin intake**
   2011 • Clin Chim Acta • Clinical Research • Human • 81 patients • 11-DHTXB2
   
   “Urinary 11-DHTXB2 constitutes a readily available marker for in vivo platelet activation and its quantification may be very useful for monitoring type 2 diabetic patients taking low-dose aspirin, considering that an ineffective response may lead to an increased risk of death.”

   “The correct identification of patients who are not adequately protected despite their usual prescribed daily dose of aspirin is warranted.”
7. Antithrombotic properties of aspirin and resistance to aspirin: beyond strictly antiplatelet actions
2007 • Blood • Review Article

“The primary established effect of aspirin on hemostasis is to impair platelet aggregation via inhibition of platelet thromboxane A\textsubscript{2} synthesis, thus reducing thrombus formation on the surface of the damaged arterial wall.”

“We review a number of the reported effects of aspirin on 3 basic elements of hemostasis: platelet activation and aggregation, the formation of the fibrin network, and the fibrinolytic process.”

Statins

1. Statin therapy and inflammation in patients with diabetes treated with high dose aspirin
2016 • Diabetes Complications • Clinical Research • Human • 209 Patients • 11-DHTXB2

“Statins along with aspirin, confers additional anti-inflammatory and antithrombotic effect in diabetics with CAD. Urinary 11-DHTXB2 may be a useful biomarker for personalizing statin therapy.”

2. Inhibition of thromboxane biosynthesis and platelet function by simvastatin in type IIa hypercholesterolemia
1995 • Arterioscler Thromb Vasc Biol • Clinical Research • Human • 24 Patients • 11-DHTXB2

3. Statin therapy and thromboxane generation in patients with coronary artery disease treated with high-dose aspirin
2014 • Thromb Haemost • Clinical Review • [x]

“Elevated 11-DHTXB2 was associated with a prothrombotic state indicated by high TIP-FCS. Our data suggest that measurement of urinary 11-DHTXB2 may be a useful method to optimize statin dosing order to reduce thrombotic risk.”

Thromboxane A\textsubscript{2} pathway schematics