Hypertension/Stroke ELISA (20-HETE) kit
Cat # 20H 1: ELISA kit for measuring 20-HETE in biological samples: $272

This competitive ELISA kit is for determination of 20-HETE (also known as 20-OH-AA) levels in biological samples. The specificity of the 20-HETE ELISA was investigated using authentic 20-HETE and a panel of fatty acids which, based on their structure, might be anticipated to compete with 20-HETE for binding to antibodies for 20-HETE. Anti-20-HETE did not cross-react with 14,15- and 11,12-DHETs or PGE2 and showed almost no cross-reactivity even with structurally extremely similar arachidonic acid (AA), linoleic acid and linolenic acid (see plot below).

Hypertension was caused by AA ω-hydroxylase (20-HETE synthesis) activity of cytochrome P450 (CYP) 4A, 4F4, 1B17,8 or kidney androgen-regulated protein (KAP)2,16. CYP4F2 genetic variants, which increased urinary 20-HETE secretion, were correlated with the risk for hypertension in a Chinese population1,13. Urinary 20-HETE levels of two-kidney, one-clip (2K1C) rats were higher than control rats12. Co-inhibition of 20-HETE and DHET formation abolished angiotensin II hypertension in mice31, suggesting new generation hypertension drug development opportunity. 20-HETE was a clinical marker of post-transplant allograft function3 and increased after cerebral ischemia, which induced brain injury due to its vasoconstrictive activity18. Recent studies reported that increased 20-HETE synthesis reduced cerebral blood flow20 and vasodilatory effect of eNOS is a result of suppressed 20-HETE synthesis in brain slices21. The interplay of circadian clock, 20-HETE pathway and renal sodium handling was studied in mice17. Blood pressure, 20-HETE formation and CYP23 expression were all decreased by N-palmitylthanolamide treatment in SHR rats25. Thus, 20-HETE is a new biomarker and therapeutic target for hypertension19, stroke and even cancer24.

A sharp decrease in 20-HETE levels in blood, urine and tissue is a clinical marker of septic shock patients. Endotoxin (ET) decreased blood pressure in rats and treatment of the rats with COX and NOS inhibitors or 5,14-HEDGE (20-HETE analogue) reversed the ET-induced hypotension and thus treatment with COX and NOS inhibitors or 20-HETE analogues may lower mortality rate of septic shock patients admitted to intensive care units5,9,14,22,23. High glucose fed rat proximal tubular cells elevated CYP4A expression and 20-HETE formation and activated the mTOR/p7056Kinase pathway which plays a major role in diabetic nephropathy.26

Each kit for triplicate analyses of up to 24 samples contains a 96 well plate, 20-HETE standard, 20-HETE-conjugated horseradish peroxidase (HRP), and buffers for sample and HRP dilutions, and plate washing.

Buy in Quantity and Save! 2-3 kits $258 each; 5-9 kits $245 each; 10 or more kits $231 each

Related Products

Hypertension/Stroke ELISA kits:
- 20-HETE B-glucuronide ELISA
- 14,15-DHET Hypertension/Stroke ELISA
- 11,12-DHET Hypertension/Stroke ELISA
- 12(S)-HETE Hypertension/Stroke ELISA
- 15(S)-HETE Hypertension/Stroke ELISA

Oxidative Stress ELISA Kit:
- 8-isoprostan EIA

Hypertension/Stroke Antibodies:
- Rat: CYP2C23, CYP2C11, CYP2C, CYP4A, sEH
- Human: CYP1B1, CYP2C8/9, sEH, CYP4A

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References


10. Imazumi et al. L-4F differentially alters plasma levels of oxidized fatty acids resulting in more anti-inflammatory HDL in mice. Drug Metab. Letters, 4, 139-148, 2010.


<table>
<thead>
<tr>
<th>Eicosanoids</th>
<th>% Binding of control</th>
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<tbody>
<tr>
<td>20-HETE</td>
<td>100.00</td>
</tr>
<tr>
<td>Arachidonic Acid</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>Linoleic Acid</td>
<td>&lt;0.02</td>
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<tr>
<td>Linolenic Acid</td>
<td>&lt;0.02</td>
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<tr>
<td>15-HETE</td>
<td>&lt;0.02</td>
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<tr>
<td>14,15-DHET</td>
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<tr>
<td>11,12-DHET</td>
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<tr>
<td>PGE₂</td>
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