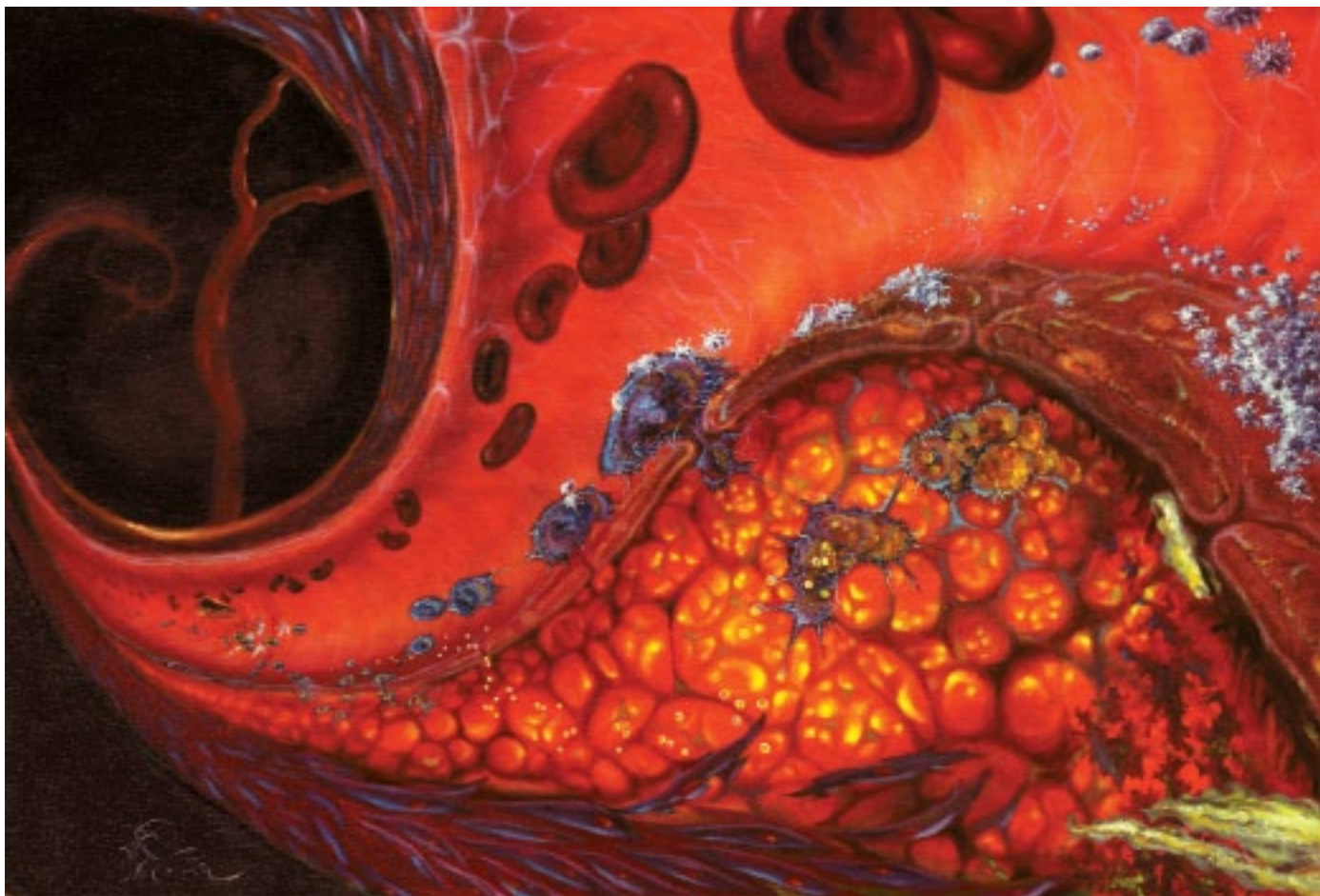


Kirk Maxey, M.D.

Introduction to

Atherosclerosis



Atherosclerosis Cover & Poster Paintings by Veronika Sherwood, Oil on Canvas

Atherosclerosis is responsible for one third of all deaths in North America and for 80% of all deaths among diabetic patients. The health risks of atherosclerosis will become even more dominant in the future as obesity and adult-onset diabetes become more prevalent. Atherosclerosis literally means the turning of the blood vessels to stone, easily seen in the calcified necrotic lesions of chronic disease. This attention to the physical manifestations of the disease continued into the recent past as physicians and the public alike became fixated by the gruel of cholesterol remaining after necrotic disintegration of terminal plaque macrophages called foam cells. More recently, scientists have recognized that foam cells and calcifications are both late manifestations of a chronic inflammatory disease. The molecular details of early atherosclerotic inflammation begin with the activation of vascular endothelial cells, the increased transport of lipoprotein particles into the subendothelial extracellular space, and the oxidative modification of those particles by cellular mechanisms. As basic scientists and clinicians unravel these molecular events, a more rational approach to disease prevention becomes possible. A balance must be reached between suppression of inflammation and increased infection risk. The efficacy and side effects of small molecule intervention can be better managed. Most important, millions of people will be spared the excruciating disabilities associated with atherosclerotic vascular disease.

The Atherosclerosis mini-catalog is the first in a series from Cayman. Our new format seeks to provide focused attention to specific diseases or pathways. In each mini-catalog, brief articles present current information on topics related to the focus. Additional information regarding products listed in these catalogs, as well as additional products from Cayman Chemical, may be found at www.CaymanChem.com.



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warranty and limitation of remedy



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At

ATHEROSCLEROSIS

ordering information

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abbreviations

ACAT	Acyl-Coenzyme A: cholesterol acyltransferase
ADP	Adenosine diphosphate
ATP	Adenosine triphosphate
COX	Cyclooxygenase
CysLT	Cysteinyl Leukotriene
EIA	Enzyme Immunoassay
ELISA	Enzyme-linked Immunosorbent Assay
FABP	Fatty Acid Binding Protein
FW	Formula Weight
GC	Gas Chromatography
HDL	High-Density Lipoprotein
ICC	Immunocytochemistry
IDL	Intermediate-Density Lipoprotein
IHC	Immunohistochemistry
IL	Interleukin
IP	Immunoprecipitation
IP Receptor	Prostaglandin I ₂ Receptor
LC	Liquid Chromatography
LDL	Low-Density Lipoprotein
LDLR	Low-Density Lipoprotein Receptor
LO	Lipoxygenase
LT	Leukotriene
LXR	Liver X Receptor
MF	Molecular Formula
MS	Mass Spectrometry
NOS	Nitric Oxide Synthase
eNOS	Endothelial Nitric Oxide Synthase
iNOS	Inducible Nitric Oxide Synthase
nNOS	Neuronal Nitric Oxide Synthase
oxLDL	Oxidized Low-Density Lipoprotein
PAF	Platelet-activating Factor
PG	Prostaglandin
PLD	Phospholipase D
PMNL	Polymorphonuclear Leukocytes
PPAR	Peroxisome Proliferator-activated Receptor
SREBP-2	Sterol Regulatory Element-binding Protein-2
TG	Triglyceride
TP Receptor	Thromboxane A ₂ Receptor
TX	Thromboxane
VLDL	Very Low-Density Lipoprotein
WB	Western Blot

Olivia May, Ph.D.

Mediating Cholesterol Homeostasis through SREBP-2 / LDLR / PCSK9 Signalling

Lipid homeostasis in vertebrate cells is regulated by sterol regulatory element-binding proteins (SREBPs), unique members of the basic helix-loop-helix leucine zipper family of transcription factors. SREBPs directly activate the expression of over 30 genes involved in both the synthesis and uptake of cholesterol, fatty acids, triglycerides, and phospholipids.^{1,2} They are also involved in activating three genes required to generate NADPH, which is consumed at multiple stages in lipid biosynthesis.

Structural features of SREBP

Three major SREBP isoforms, SREBP-1a, -1c, and -2, have been identified and differ in relative abundance in the liver and other various tissues. SREBP-1c predominates in the liver as it is 10-fold more abundant than SREBP-1a and 2-fold more abundant than SREBP-2. SREBP-1a and -1c are both encoded from a gene on human chromosome 17p11.2, while SREBP-2 is derived from a gene on chromosome 22q13. SREBP proteins are organized into 3 domains—an NH₂-terminal domain that contains the bHLH-Zip region for binding DNA, two hydrophobic transmembrane-spanning segments interrupted by a short loop of about 30 amino acids that project into the lumen of the ER, and a COOH-terminal regulatory domain. SREBP-1a and -2 have relatively long transcriptional activation domains, while the NH₂-terminal acidic domain of SREBP-1c is 18 amino acids shorter. Constitutively expressed at low levels, SREBP-1a is a potent

activator of all SREBP-responsive genes and most likely functions to maintain basal levels of cholesterol and fatty acid synthesis. In contrast, SREBP-1c selectively activates genes involved in fatty acid synthesis, while SREBP-2 preferentially regulates genes important for cholesterol homeostasis by activating the transcription of HMG-CoA synthase, HMG-CoA reductase, LDL receptor (LDLR), and proprotein convertase subtilisin kexin type 9 (PCSK9).

Pathway of SREBP-2 activation and signalling

SREBP-2 activity is tightly regulated by cellular sterol levels. When intracellular levels of cholesterol are high, SREBP-2 is present in the ER as an inactive precursor bound to SREBP cleavage-activating protein (SCAP). Sterols mediate feedback inhibition of SREBP-2 *via* Insig-1 and -2, which bind to SCAP in the ER and prevent movement of the SCAP/SREBP complex to the Golgi. As cholesterol levels decrease, SREBP-2 moves to the Golgi where it is proteolytically cleaved by the protein convertase Subtilisin kexin isozyme/Site-1 protease (SKI-1/S1P) and the intramembranous metalloprotease Site-2 protease (S2P), which act sequentially to release the NH₂-terminal bHLH-Zip domain of SREBP-2 from the membrane. Two N-terminal fragments dimerize, bind importin β , and enter the nucleus to bind to a sterol response element (SRE) in the promoter region of target genes and up-regulate transcription.

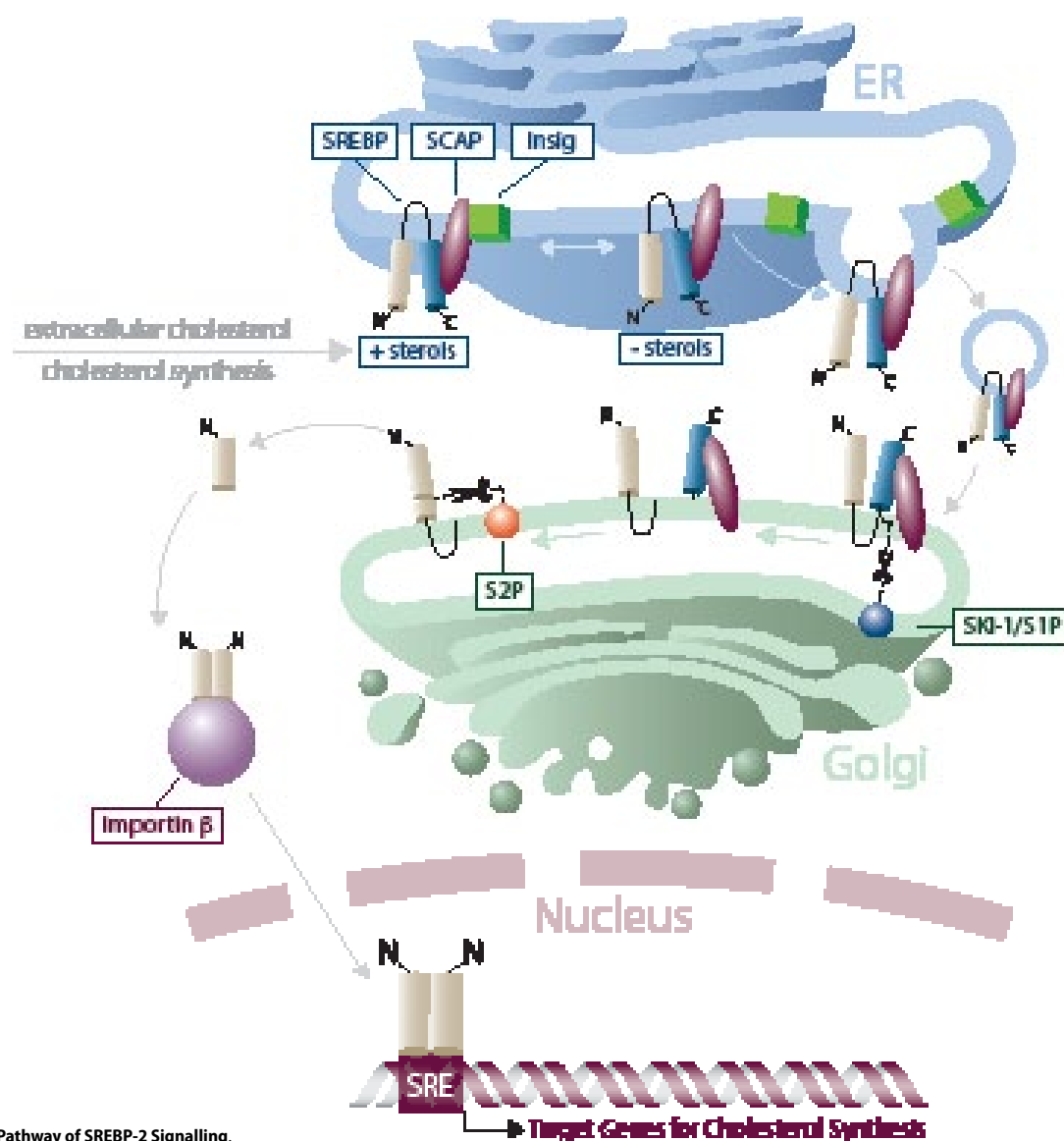


Figure 1. Pathway of SREBP-2 Signalling.

SREBP-2: A paradoxical regulator of plasma LDL

In addition to up-regulation of LDLR transcription, which ultimately increases clearance of LDL from the bloodstream, nuclear SREBP-2 increases the transcription of PCSK9, a sterol-responsive protein that accelerates LDLR turnover in the liver, thereby limiting uptake the lipoprotein. Thus, two opposing effects on plasma cholesterol levels are initiated by the same metabolic signal. As high concentrations of cellular cholesterol suppress SREBP-2 cleavage and release from the ER, PCSK9 transcription is reduced, which subsequently increases LDLR levels helping to maintain cholesterol homeostasis.

Mutations in genes encoding PCSK9

PCSK9 was originally identified as neural apoptosis regulated convertase 1 (NARC-1) because it was upregulated during neuronal apoptosis.³ However, naturally occurring mutations resulting in hypercholesterolemia led to the discovery that circulating levels of PCSK9 were linked with cholesterol metabolism. Three missense, gain-of-function mutations, S127R, F216L, and D374Y, were initially identified in patients in association with increased plasma LDL-cholesterol levels. N425S and R496W were mutations identified later in hypercholesterolemic individuals who also had mutations in the LDLR. When PCSK9 is overexpressed in mice, there is a dramatic decrease in levels of LDLR protein but not mRNA LDLR in the liver. Thus, overexpression of PCSK9 reduces LDLR post-transcriptionally.

Hypocholesterolemia results from many different loss-of-function mutations in PCSK9. The identification of the Y142X, C679X, and R46L mutants broached the potential relationship between plasma levels of LDL and coronary heart disease. A fifteen-year study of patients with nonsense mutations in PCSK9 revealed that naturally-reduced LDL levels (by as much as 28%) decreased the frequency of heart disease by 88%.⁴ Additional in-frame deletions and missense mutations (DR97, G106R, L253F, A443T) have also been identified. In PCSK9 knockout mice, levels of LDLR are elevated and clearance of plasma LDL is accelerated suggesting that PCSK9 normally functions to limit the uptake of LDL by suppressing LDLR levels. In conjunction with reduction of LDLR, PCSK9 can increase the rate of secretion of ApoB-100, the main apolipoprotein of LDL, from the liver, further influencing plasma LDL levels.⁵ Evidence of this can be found in the S127R gain-of-function mutation, which results in a three-fold increase in ApoB compared to controls.

Structural features and site of action of PCSK9

PCSK9 consists of a signal sequence (amino acids 1-30) followed by the prodomain (amino acids 31-152) and catalytic domain (amino acids 153-425). Rather than having a classical P domain required in other proprotein convertases for folding and regulation of protease activity, the tail of the catalytic domain contains a 279-amino acid C-terminal region rich in cysteines and histidines. Synthesized in the ER as a ~72 kDa precursor, the protein undergoes autocatalytic cleavage between the prodomain and catalytic domain. The prodomain (~14 kDa), however, remains associated with the mature protein (63 kDa) as it follows the secretory pathway to the Golgi where both segments undergo tyrosine sulfation before being secreted.

While the exact LDLR binding domain and its inhibitory prosegment are unknown, PCSK9 does not appear to directly cleave LDLR. It is likely that PCSK9 interacts with LDLR protein on the cell surface and functions as a chaperone to interfere with normal LDLR recycling and direct it toward the intracellular degradative pathway.^{6,7} ARH (autosomal recessive hypercholesterolemia), an endocytic adaptor protein necessary for LDLR internalization, must also be present for PCSK9-mediated degradation of LDLR.⁸

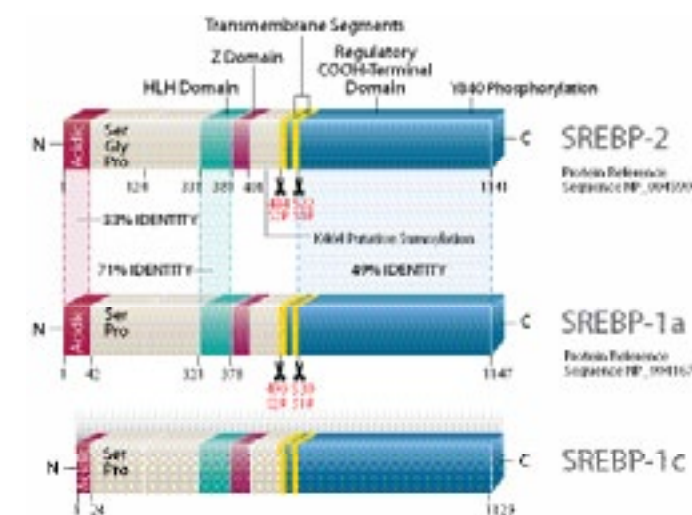


Figure 2. SREBP Isoforms.

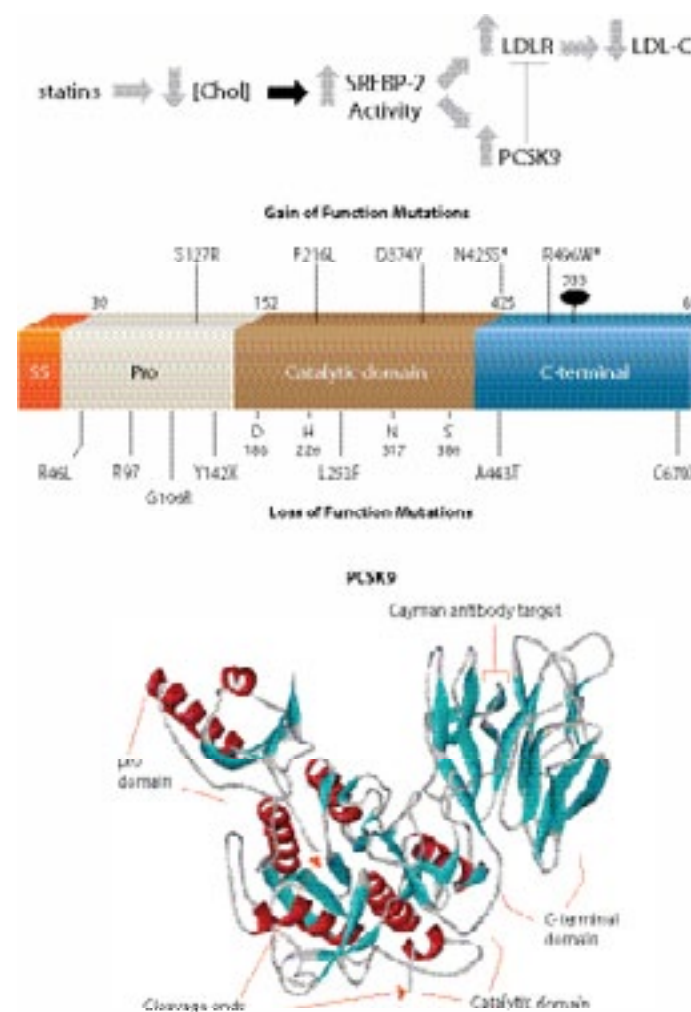


Figure 3. Regulation and structure of PCSK9. Reduction in cholesterol levels increases SREBP-2 activity, leading to increases in both LDLR and PCSK9 transcription. Paradoxically, PCSK9 removes LDLR, reducing LDL-cholesterol removal. Mutations in PCSK9 have been identified that increase ("gain of function") or decrease ("loss of function") LDLR removal.¹⁰ PCSK9 does not remove LDLR through its protease activity. To date, the only known substrate of PCSK9 is itself, with cleavage occurring between residues 152, 153.

PCSK9 potential as a therapeutic target

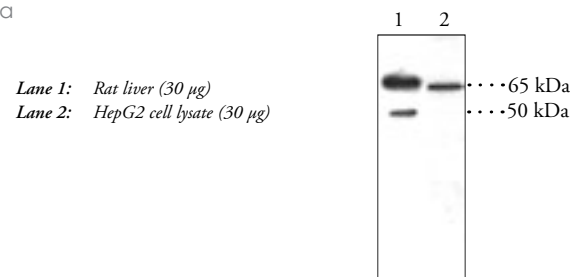
The paradoxical regulation of LDL helps to explain why statins are only marginally effective at regulating LDL plasma levels. That is, a five year treatment with cholesterol-lowering statins reduces the incidence of heart attack by only 40% even when LDL cholesterol is decreased by 80 mg/dl.⁸ Statins inhibit cholesterol synthesis through inhibition of HMG-CoA reductase, which results in an upregulation of both LDLR and PCSK9 mRNA levels *via* sterol-mediated SREBP-2 activation.⁹ Therefore, upregulation of PCSK9 partially negates the LDL-lowering effects of statins. Ideally, a pharmacological inhibitor of PCSK9 could intervene in this atherogenic effect by increasing LDLR levels without affecting other genes regulated by SREBP-2.

References

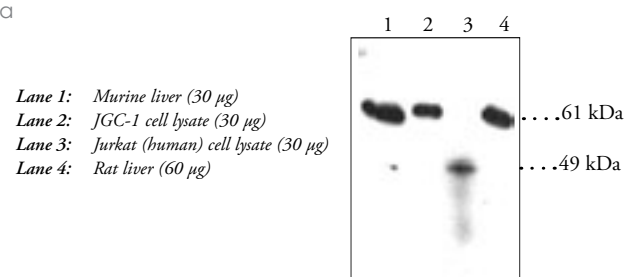
- Osborne, T.F. *J. Biol. Chem.* **275**(42), 32379-32382 (2000).
- Lin, J., Yang, R., Tarr, P.T., et al. *Cell* **120**, 261-273 (2005).
- Naureckiene, S., Ma, L., Sreekumar, K., et al. *Arch. Biochem. Biophys.* **420**, 55-67 (2003).
- Cohen, J.C., Boerwinkle, E., Mosley, T.H., et al. *N. Engl. J. Med.* **354**(12), 1264-1272 (2006).
- Benjannet, S., Rhainds, D., Essalmani, R., et al. *J. Biol. Chem.* **279**(47), 48865-48875 (2004).
- Holla, Ø.L., Cameron, J., Berge, K.E., et al. *BMC Cell Biology* **8**(9), 1-12 (2007).
- Lagace, T.A., Curtis, D.E., Garuti, R., et al. *J. Clin. Invest.* **116**(11), 2995-3005 (2006).
- Baigent, C., Keech, A., Kearney, P.M., et al. *Lancet* **366**, 1267-1278 (2005).
- Rashid, S., Curtis, D.E., Garuti, R., et al. *Proc. Natl. Acad. Sci. USA* **102**(15), 5374-5379 (2005).
- Horton, J.D., Cohen, J.C., and Hobbs, H.H. *Trends Biochem. Sci.* **32**(2), 71-77 (2007).

ACAT-1 Polyclonal Antibody 100028*Sterol O-Acyltransferase 1*Peptide affinity-purified IgG **Stability:** ≥1 year at -20°C**Summary:** Antigen: human ACAT-1 amino acids 6-23 • Host: rabbit • Cross-reactivity: (+) murine, rat, porcine, and human ACAT-1; other species not tested • Applications: WB, IHC, and ICC; other applications not tested • ACAT-1 catalyzes the formation of cholesterol esters from cholesterol and long chain fatty acyl-coenzyme A, and may play a role in the development of atherosclerosis.

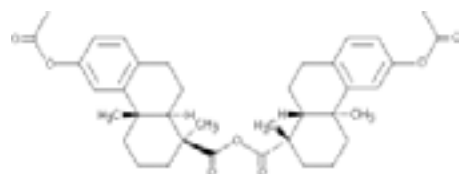
1 ea

**Also Available:** ACAT-1 Blocking Peptide (10005090) 200 µg**ACAT-2 Polyclonal Antibody** 100027*Sterol O-Acyltransferase 2*Peptide affinity-purified IgG **Stability:** ≥1 year at -20°C**Summary:** Antigen: human ACAT-2 amino acids 3-20 • Host: rabbit • Cross-reactivity: (+) human, murine, rat, porcine, and ovine ACAT-2; other species not tested • Applications: WB, ICC, and IHC • ACAT-2 catalyzes the formation of cholesterol esters from cholesterol and long chain fatty acyl-coenzyme A.

1 ea

**Also Available:** ACAT-2 Blocking Peptide (10005091) 200 µg**NEW Acetyl Podocarpic Acid Anhydride** 10007686

[344327-48-6] APD

MF: C₃₆H₄₆O₇ **FW:** 614.8 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C**Summary:** APD is a potent, semi-synthetic LXR agonist that acts through LXR in concert with RXR, its heterodimerization partner, to induce the expression of the ABCA1 reverse cholesterol transporter. This acts to increase the efflux of cholesterol from enterocytes and thus inhibit the overall absorption of cholesterol (ED₅₀ value of 1 nM). APD is approximately 1,000 times more potent and has 8-10 fold greater maximal stimulation of LXR than 22(R)-hydroxy cholesterol. APD can be used as a positive control for the testing of LXR agonists, which have potential as therapeutic agents for the treatment of atherosclerosis.1 mg
5 mg
10 mg
50 mg

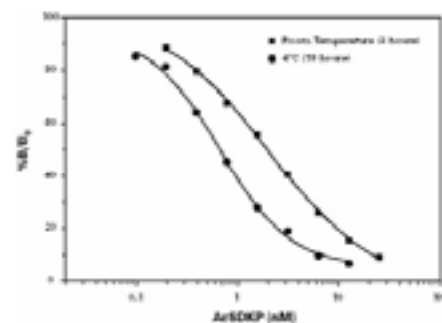
6-(acetyloxy)-1,2,3,4,4a,9,10,10a-octahydro-1,4a-dimethyl-1-phenanthrenecarboxylic acid, anhydride

*SPL-BIO Assays are available through Cayman Chemical only within North & South America and Asia; elsewhere contact SPL-BIO.

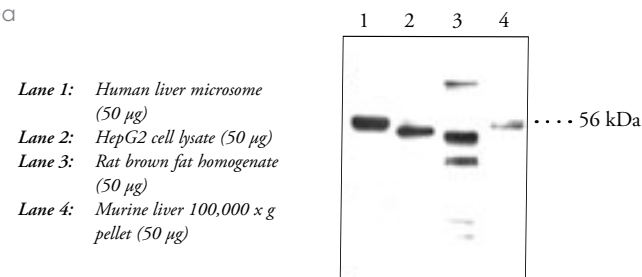
AcSDKP EIA Kit* 589451*N-Acetyl Ser-Asp-Lys-Pro***Stability:** ≥6 months at -20°C**Summary:** AcSDKP is a tetrapeptide growth regulatory hormone which inhibits the proliferation of hematopoietic stem cells. The dipeptidase Angiotensin Converting Enzyme (ACE) actively metabolizes circulating AcSDKP, giving it a brief plasma half-life of 4 to 5 minutes. ACE inhibition is a major therapeutic end point in the treatment of hypertension management. A further consequence of ACE inhibition is the accumulation of AcSDKP in plasma and urine. This accumulation may have physiological effects, which are manifested as the anemia of chronic ACE inhibitor toxicity. More commonly, plasma and urine AcSDKP levels can be used as a biomarker of ACE inhibition and an index of patient compliance with therapy. Measurement of AcSDKP in human urine or plasma can be readily accomplished by EIA.**Sensitivity:** 50% B/B₀: 2.0 nM after 3 hour immunological reaction
0.5 nM after 18 hour immunological reaction
80% B/B₀: 0.2 nM after 18 hour immunological reaction**Specificity:**

AcSDOrnP	500%
AcSDKP	100%
AcSDRP	6%
SDKP	0.5%
Thymosin B ₄	<0.25%
AcSDK	0.03%
AcSDKPDC	<0.01%
AcSDKPY	<0.01%
rTNF	<0.01%

96 wells

**NEW Adipose Triglyceride Lipase Polyclonal Antibody** 10006409*ATGL, Desnutrin, PLA₂ζ*Peptide affinity-purified IgG **Stability:** ≥1 year at 4°C**Summary:** Antigen: human ATGL amino acids 382-400 • Host: rabbit • Cross-reactivity: (+) human, murine, and rat ATGL • Applications: WB and IHC (paraffin-embedded sections)

1 ea

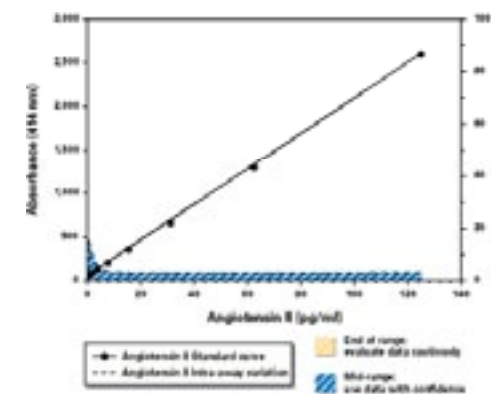
**Also Available:** Adipose Triglyceride Lipase Blocking Peptide (10008492) 200 µg**Angiotensin II EIA Kit*** 589301**Stability:** ≥6 months at -20°C**Summary:** Angiotensin II is a primary reactive vasoconstrictor, the main stimulus for aldosterone release, and one of the causative factors of chronic hypertension. The active angiotensin II octapeptide is released *via* a tightly controlled series of prohormones and proteases. Normal human plasma angiotensin II levels are 10-30 pg/ml when measured at rest in the supine position; they increase on standing, exercise, dehydration, or sodium depletion. The unique, patented 'Immobilized Antigen' technology of this angiotensin II immunometric assay allows reliable detection of 1-2 pg/ml, or as little as 10% of the normal human plasma concentration.**Sensitivity:** Limit of detection: 1.5 pg/ml**Specificity:**

Angiotensin II	100%
Angiotensin III	36%
Angiotensin 3-8	33%
Angiotensin I	4%
Angiotensin 1-7	<0.01%

Homology:

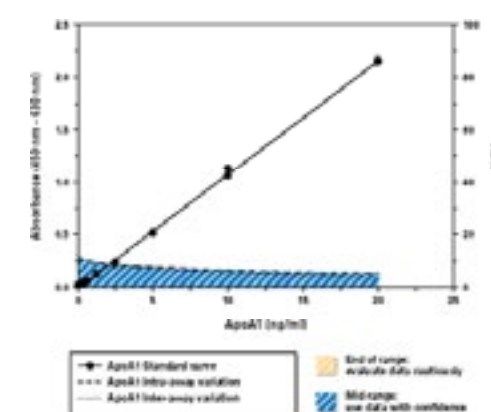
Mammalian Angiotensin II	100%
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96 wells

**NEW ApoA1 EIA Kit** 10010551*Apolipoprotein A1***Stability:** ≥6 months at -20°C**Summary:** ApoA1 is a major protein component of HDL. Clinical studies have demonstrated that lower levels of ApoA1 are associated with an increased risk of myocardial infarction and coronary artery disease. Overexpression of ApoA1 raises HDL cholesterol levels and inhibits the progression of atherosclerosis in mice. For this reason, upregulation of ApoA1 expression is considered to be one of the most promising approaches to the development of new therapies for atherosclerosis targeting HDL. Cayman Chemical's ApoA1 EIA Kit is an immunometric assay which can be used to measure ApoA1 in plasma and serum without prior sample purification. The standard curve spans the range of 0-20,000 pg/ml with a limit of detection of approximately 300 pg/ml.

96 wells

480 wells

**NEW ApoA1 Polyclonal Antibody** 10008463*Apolipoprotein A1*Peptide affinity-purified IgG **Stability:** ≥1 year at -20°C**Summary:** Antigen: human ApoA1 protein amino acids 188-199 • Host: rabbit • Cross-reactivity: (+) human, murine, and rat ApoA1 protein • Applications: WB and ICC

1 ea

NEW ApoA1 Western Ready Control 10009751*Apolipoprotein A1***Purity:** 28 kDa**Stability:** ≥6 months at -20°C**Summary:** Source: human recombinant protein • Application: positive control for WB • ApoA1 is a major protein component of HDL. It acts as an acceptor for sequential transfers of phospholipids and free cholesterol from peripheral tissues and transports cholesterol to the liver and other tissues for excretion and steroidogenesis.

1 ea

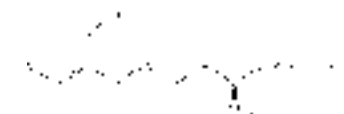
NEW ApoB-100 EIA Kit 10011012*Apolipoprotein B-100***Stability:** ≥6 months at -20°C**Summary:** ApoB-100 is the major protein component of the atherogenic lipoprotein particles VLDL, IDL, and LDL, with each particle possessing one molecule of ApoB-100. ApoB-100 is essential for the binding of LDL particles to the LDLR as the initial step in the cellular uptake and degradation of LDL. Two mutations of ApoB-100 that decrease its ability to bind to the LDLR, and therefore the uptake of LDL into cells, have been linked with familial hypercholesterolemia. Several clinical studies have demonstrated that elevated levels of plasma ApoB-100 are associated with an increased risk of myocardial infarction and coronary artery disease. Some studies suggest that ApoB-100 or the ratio of ApoB-100/ApoA1 is more predictive of the risk of myocardial infarction than is LDL. Cayman's ApoB-100 EIA Kit is an immunometric assay for the measurement of human ApoB-100 in plasma samples.

96 wells

480 wells

N^ε,N^ε-dimethyl-L-Arginine (dihydrochloride) 80230

[220805-22-1] ADMA (dihydrochloride)

MF: C₈H₁₈N₄O₂ • 2HCl **FW:** 275.2 **Purity:** ≥98%A crystalline solid **Stability:** ≥1 year at -20°C**Summary:** ADMA is an endogenous NOS inhibitor. ADMA concentrations increase in several disease states including renal failure, muscular dystrophy, hypercholesterolemia, and pregnancy with preeclampsia, but its precise role in these diseases has not been elucidated.5 mg
10 mg
50 mg
100 mgN^ε-[(dimethylamino)iminomethyl]-L-ornithine, dihydrochloride**Aspirin** 70260

[50-78-2] Acetylsalicylic Acid

MF: C₉H₈O₄ **FW:** 180.2 **Purity:** ≥99%A crystalline solid **Stability:** ≥2 years at room temperature**Summary:** Aspirin is a non-selective, irreversible COX inhibitor. The IC₅₀ values for inhibition of ovine COX-1 and -2 are 0.75 and 1.25 mM, respectively. Aspirin acetylates COX-1 at Ser⁵³⁰ and COX-2 at Ser⁵¹⁶ resulting in irreversible enzyme inhibition.5 g
25 g
50 g
100 g

2-(acetyloxy)-benzoic acid

Athero-PAK 10005292

Stability: ≥6 months at -20°C**Summary:** The Athero-PAK contains our human ACAT-1 polyclonal antibody and blocking peptide. Targeting human amino acids 6-23 of the ACAT-1, these reagents can be used for WB analysis and IHC. Also included are several oxidized bioactive lipid species, including cholesteryl linoleate hydroperoxides, POV-PC, and PGPC.

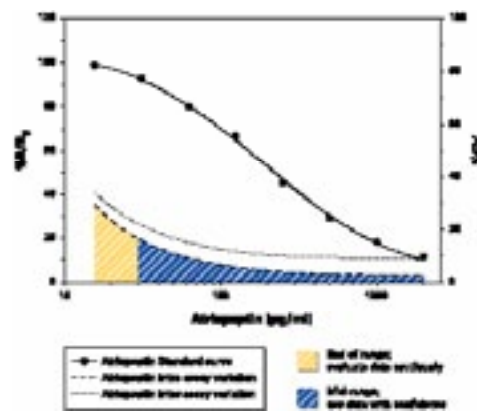
1 ea

Atriopeptin (rat) EIA Kit* 589401

Stability: ≥6 months at -20°C**Summary:** Atriopeptin is a 28 amino acid peptide synthesized primarily in cardiac atria. This peptide hormone acts in opposition to angiotensin II in regulating renal, hemodynamic, and endocrine function. Atriopeptin is released in response to the increased pressure and mechanical stretch of the right atrium due to blood volume overload. Atriopeptin then acts at the nephron to increase salt and water excretion, lowering blood volume and blood pressure. Elevated plasma atriopeptin levels may be produced in experimental models by volume expansion, high salt diets, and in response to vasoconstrictors. Increased plasma concentrations have also been reported in various pathological conditions such as renal disease, congestive heart failure, and paroxysmal atrial tachycardia.**Sensitivity:** 50% B/B₀: 190 pg/ml
80% B/B₀: 60 pg/ml**Specificity:**

Rat Atriopeptin 24	100%
Atrial Natriuretic Peptide (8-33)	100%
Rat Atrial Natriuretic Peptide	100%
Human Atrial Natriuretic Peptide	100%
Atrial Natriuretic Peptide (18-28)	60%
Human β Atrial Natriuretic Peptide	50%
Human 6 Atrial Natriuretic Peptide	40%
Auriculin A	10%
Rat Atriopeptin II	5%
Rat Atrial Natriuretic Peptide (13-28)	1%
Arg ⁸ -Vasopressin	<0.01%
Brain Natriuretic Peptide	<0.01%
Oxytocin	<0.01%
Rat Atriopeptin I	<0.01%
Somastostatin	<0.01%

96 wells



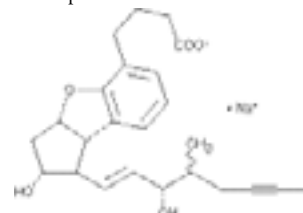
Azelaoyl PAF 60924

MF: C₃₃H₆₆NO₉P **FW:** 651.9 **Purity:** ≥98%A solution in ethanol **Stability:** ≥1 year at -20°C**Summary:** oxLDL particles contain low molecular weight species which promote the differentiation of monocytes *via* the nuclear receptor PPAR γ . One of these substances was recently isolated and purified from oxLDL, and identified as azelaoyl PAF. Azelaoyl PAF is a potent PPAR γ agonist which competes for the thiazolidinedione binding site. Azelaoyl PAF is more potent than 15-deoxy-D^{12,14}-PGJ₂, and equipotent with rosiglitazone as a ligand for this receptor.1 mg
5 mg
10 mg
50 mg

1-O-hexadecyl-2-O-(9-carboxyoctanoyl)-sn-glycerol-3-phosphocholine

Beraprost (sodium salt) 18230

[88475-69-8] ML 1129, Procylin, TRK 100

MF: C₂₄H₂₉O₅ • Na **FW:** 420.5 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C**Summary:** Beraprost is an analog of prostacyclin in which the unstable enol-ether has been replaced by a benzofuran ether function. This modification increases the plasma half-life from 30 seconds to several hours, and permits the compound to be taken orally. Doses of 20-100 μg in humans, given 1 to 3 times per day, have been demonstrated to improve clinical end points in diseases responsive to prostacyclin. Oral beraprost therapy improved the survival and pulmonary hemodynamics of patients with primary pulmonary hypertension. Beraprost inhibits platelet aggregation in healthy subjects and in diabetic patients at similar doses.1 mg
5 mg
10 mg
50 mg

2,3,3a,8b-tetrahydro-2-hydroxy-1-(3-hydroxy-4-methyl-1-octen-6-ynyl)-1H-cyclopenta[b]benzofuran-5-butanoic acid, sodium salt

Berberine 10006427

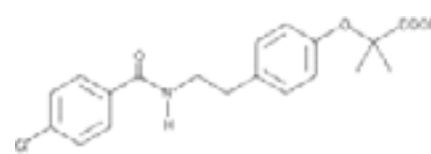
[633-65-8] BBR, Umbellatine

MF: C₂₀H₁₈ClNO₄ **FW:** 371.8 **Purity:** ≥95%A crystalline solid **Stability:** ≥2 years at -20°C**Summary:** Berberine is a widely distributed berberidacean alkaloid that has been employed in traditional medicine as an antiprotozoal and antiarrhythmic agent. Berberine reduces total cholesterol, LDL cholesterol, and TGs in both humans (at 1 g/day) and hamsters fed 50 mg/kg/day along with a high fat diet. Berberine does not act through HMG-CoA reductase inhibition, but instead enhances LDL-receptor protein and mRNA levels in hepatocytes. Berberine is therefore a natural product that may help control serum cholesterol without the side effects typical of the statin family of hypocholesterolemic drugs.1 g
5 g
10 g
25 g

5,6-dihydro-9,10-dimethoxy-benzo[g]-1,3-benzodioxolo[5,6-a]quinolizinium, chloride

NEW Bezafibrate 10009145

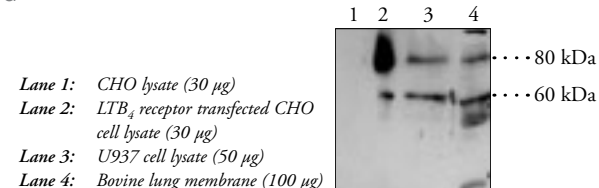
[41859-67-0] Bezofibrate, Bezalip, Bezatrol, BM 15075, Difaterol

MF: C₁₉H₂₀N₄ClNO₄ **FW:** 361.8 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C**Summary:** Bezafibrate is a well established pan-PPAR activator. It activates human PPAR α , PPAR δ , and PPAR γ with EC₅₀ values of 50, 20, and 60 μM, respectively, in a cell-based transcription assay. Bezafibrate helps lower LDL cholesterol and triglycerides while raising HDL cholesterol levels. It also improves insulin sensitivity and reduces blood glucose levels, which in combination with the cholesterol effects significantly lowers the incidence of cardiovascular events and development of diabetes in patients with features of metabolic syndrome.500 mg
1 g
5 g
10 g

2-[4-[2-[(4-chlorobenzoyl)amino]ethyl]phenoxy]-2-methyl-propanoic acid

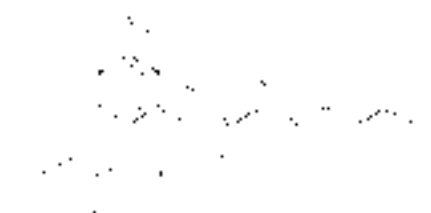
BLT₁ Receptor Polyclonal Antibody 120114BLTR₁, Leukotriene B₄ Receptor 1, LTB₄ Receptor 1Peptide affinity-purified IgG **Stability:** ≥1 year at 4°C**Summary:** Antigen: human BLT₁ receptor amino acids 331-352 • Host: rabbit • Cross-reactivity: (+) human and bovine BLT₁ receptor; (-) murine BLT₁ receptor • Applications: WB, flow cytometry, ICC, and IHC

1 ea

Also Available: BLT₁ Receptor Blocking Peptide (120112) 1 mg

BM 567 10155

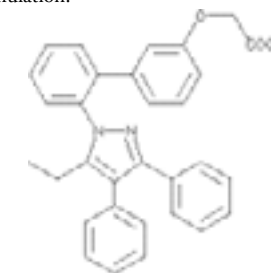
[284464-77-3] Sold and made under non-exclusive license from Université de Liège.

MF: C₁₈H₂₈N₄O₅S **FW:** 412.5 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C**Summary:** BM 567 is a dual acting antithrombogenic agent, acting as an inhibitor of TXA₂ synthase and as an antagonist of the TP receptor, the G protein-coupled receptor mediating TXA₂ activity in platelets and vascular smooth muscle. BM 567 antagonizes the vascular smooth muscle TP receptor with an IC₅₀ value of 1.1 nM. It inhibits platelet TXA₂ synthase with an IC₅₀ value of 12 nM.1 mg
5 mg
10 mg
50 mg

2-(cyclohexylamino)-5-nitro-N-[(pentylamino)carbonyl]-benzenesulfonamide

BMS 309403 10010206

[300657-03-8]

MF: C₃₁H₂₆N₂O₃ **FW:** 474.6 **Purity:** ≥98%A crystalline solid **Stability:** ≥1 year at -20°C**Summary:** A potent and selective FABP4 inhibitor (K_i < 2 nM) that has anti-atherogenic effects including decreasing production of chemoattractant and inflammatory cytokines, reducing transformation of macrophages into foam cells, and diminishing cholesterol ester accumulation.1 mg
5 mg
10 mg
50 mg

[[2'-(5-ethyl-3,4-diphenyl-1H-pyrazol-1-yl)](1,1-biphenyl)]3-yl]oxy]-acetic acid

Butanoyl PAF 60928

MF: C₂₈H₅₈NO₇P **FW:** 551.7 **Purity:** ≥98%A solution in ethanol **Stability:** ≥1 year at -20°C**Summary:** oxLDL particles contain low molecular weight species which promote the differentiation of monocytes and activate PMNL. One of these substances was recently isolated and purified from oxLDL and identified as azelaoyl PC. Butanoyl PAF is a closely related compound which retains at least 10% of the agonist potency of PAF itself. Further, butanoyl PAF is present in oxLDL in amounts more than 100 times greater than enzymatically generated PAF. Butanoyl PAF is therefore one of the important signalling molecules present in oxLDL.1 mg
5 mg
10 mg
50 mg

1-O-hexadecyl-2-O-butanoyl-sn-glycerol-3-phosphocholine

Butenoyl PAF 60929

MF: C₂₈H₅₆NO₇P **FW:** 549.7 **Purity:** ≥98%A solution in ethanol **Stability:** ≥1 year at -20°C**Summary:** Butenoyl is a product of the oxidative decomposition of 2-arachidonoyl-containing phospholipids. Oxygenation of C-5 of the 5,6 double bond followed by cleavage of the hydroperoxide results in a PAF-like compound with a 4-carbon residue esterified in the sn-2 position; similar oxidized lipid products also act as ligands for oxidized lipid receptors and PPAR. Although butenoyl PAF is 10-fold less potent than PAF as a PAF receptor agonist, it is present in amounts 100-fold greater than enzymatically generated PAF.1 mg
5 mg
10 mg
50 mg

1-O-hexadecyl-2-O-butanoyl-sn-glycerol-3-phosphocholine

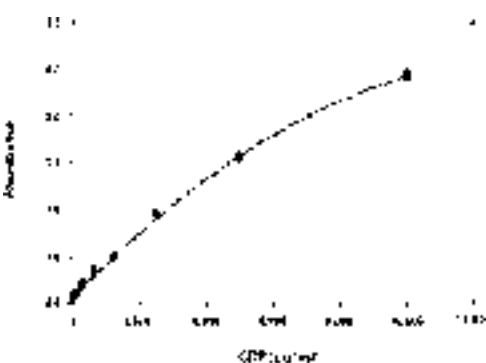
NEW C-reactive Protein EIA Kit

10011236

CRP

CRP is a 224 amino acid protein that is primarily synthesized by hepatocytes, and to some extent adipocytes, and increases ~1,000-fold in response to acute and chronic inflammatory conditions. Because of this rapid and dramatic rise, the plasma concentration of CRP is routinely measured as a gauge of inflammation in a wide range of health and disease conditions. Normal levels of serum CRP (0.64 mg/L) are not different between healthy adult men and women, but tend to increase slightly with age. High plasma CRP concentrations (>3 mg/L) are associated with an increased risk for atherosclerotic vascular disease. While the exact function is unclear, CRP has been implicated as a contributor to atherogenesis by numerous means including modulating endothelial function, stimulating coagulation, marking vascular inflammation by inducing an increase in expression of ICAM-1, VCAM-1, and E-selectin, mediating uptake of LDL into macrophages, and destabilizing plaques.

96 wells
480 wells

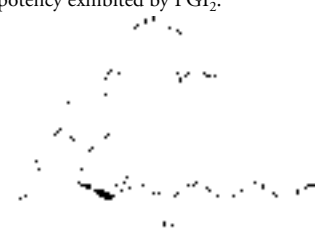
**Carbaprostacyclin**

18210

[69552-46-1] Carbacyclin, cPGI₂**MF:** C₂₁H₃₄O₄ **FW:** 350.5 **Purity:** ≥99%A crystalline solid **Stability:** ≥1 year at -20°C

Summary: Carbaprostacyclin is a stable analog of PGI₂. When infused in rabbits or dogs, it inhibits *ex vivo* platelet aggregation, but the effect persists only 10 minutes after termination of the infusion. This implies rapid metabolic inactivation of carbaprostacyclin. Carbaprostacyclin inhibits platelet aggregation with an ED₅₀ value of 47 nM, which is 10% of the molar potency exhibited by PGI₂.

1 mg
5 mg
10 mg
50 mg



6,9α-methylene-11α,15S-dihydroxy-prosta-5E,13E-dien-1-oic acid

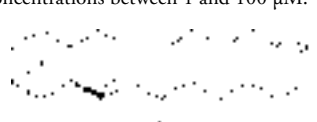
Carbocyclic Thromboxane A₂

19010

[74034-56-3] CTA₂**MF:** C₂₂H₃₆O₃ **FW:** 348.5 **Purity:** ≥98%*A solution in ethanol **Stability:** ≥1 year at -20°C

Summary: CTA₂ is a stable analog of TXA₂. CTA₂ is a potent coronary vasoconstrictor that is effective at concentrations as low as 1 nM in cat coronary arteries. Unlike other vascular TP receptor agonists, CTA₂ is a potent inhibitor of prostanoid-induced platelet aggregation. It inhibits arachidonic acid-induced aggregation with an IC₅₀ value of 4-5 μM. CTA₂ also exhibits selective and dose-dependent inhibition of TXB₂ synthesis in rabbit platelets at concentrations between 1 and 100 μM.

100 μg
500 μg
1 mg
5 mg



9α,11α-methylene-15S-hydroxy-11a-deoxy-11a-methylene-thromba-5Z,13E-dien-1-oic acid

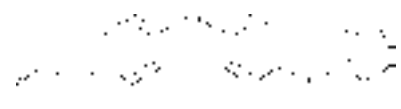
CAY10441

10005186

[221529-58-4]**MF:** C₁₉H₂₃N₃O **FW:** 309.4 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C

Summary: CAY10441 is a high-affinity antagonist for the human IP receptor. It inhibits the binding of tritiated iloprost to rodent neuroblastoma cells with a K_i value of about 1.5 nM. At levels between 2-20 mg/kg in rats, CAY10441 shows significant analgesic activity in standard antinociceptive assays.

1 mg
5 mg
10 mg
50 mg



4,5-dihydro-N-[4-[[4-(1-methylethoxy)phenyl]methyl]phenyl]-1H-imadazol-2-amine

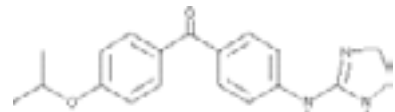
CAY10449

10005913

MF: C₁₉H₂₁N₃O₂ **FW:** 323.4 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C

Summary: CAY10449 is a high-affinity antagonist for the human IP receptor. It inhibits the binding of tritiated iloprost to rodent neuroblastoma cells with a K_i value of about 3 nM.

1 mg
5 mg
10 mg
50 mg



4,5-dihydro-N-[4-[[4-(1-methylethoxy)phenyl]carbonyl]phenyl]-1H-imadazol-2-amine

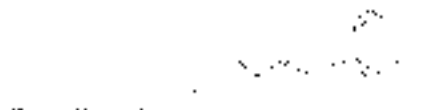
CAY10485

10006482

[615264-62-5] 3,4-dihydroxy Hydrocinnamic acid (L-Aspartic acid dibenzyl ester) amide**MF:** C₂₇H₂₇NO₇ **FW:** 477.1 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C

Summary: ACAT-1 and ACAT-2 catalyze the formation of cholesterol esters from cholesterol and long chain fatty acyl-coenzyme A, and may play a role in the development of atherosclerosis. CAY10485 inhibits human ACAT-1 and ACAT-2 with an IC₅₀ values of 95 and 81 μM, respectively. It also inhibits copper-mediated oxidation of LDLs by 91% at a concentration of 2 μM.

5 mg
10 mg
50 mg
100 mg



N-[3-(3,4-dihydroxyphenyl)-1-oxopropyl]-L-aspartic acid, bis(phenylmethyl) ester

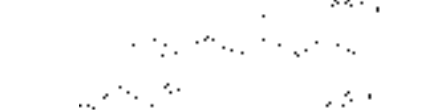
CAY10486

10006452

[615264-52-3] 4-Hydroxycinnamic acid (L-phenylalanine methyl ester) amide**MF:** C₁₉H₁₉NO₄ **FW:** 325.4 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C

Summary: ACAT-1 and ACAT-2 catalyze the formation of cholesterol esters from cholesterol and long chain fatty acyl-coenzyme A, and may play a role in the development of atherosclerosis. CAY10486 inhibits human ACAT-1 and ACAT-2 equally with an IC₅₀ value of approximately 60 μM. It also inhibits copper-mediated oxidation of LDLs by about 28% at a concentration of 3 μM.

5 mg
10 mg
50 mg
500 mg



N-[3-(4-hydroxyphenyl)-1-oxo-2-propenyl]-L-phenylalanine, methyl ester

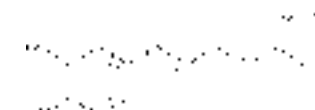
CAY10487

10006480

[778624-05-8] 3,4-Dihydrocinnamic Acid (L-alanine methyl ester) amide**MF:** C₁₃H₁₅NO₃ **FW:** 265.3 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C

Summary: CAY10487 inhibits formation of fatty streak lesions of the thoracic aorta in high cholesterol-fed rabbits without affecting plasma lipid profiles or significantly inhibiting ACAT-1 or ACAT-2 activity. The percent area occupied by the atherosclerotic lesion in rabbits supplemented with 0.05% CAY10487 in the diet was 16.1% compared to 53.5% in control rabbits. CAY10487 also exhibits antioxidant activity, inhibiting copper-mediated oxidation of LDL by about 75% at a concentration of 2 μM.

5 mg
10 mg
50 mg
500 mg



N-[(2E)-3-(3,4-dihydroxyphenyl)-1-oxo-2-propenyl]-L-alanine, methyl ester

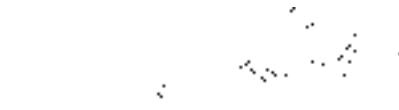
NEW CAY10499

10007875

[359714-55-9]**MF:** C₁₈H₁₇N₃O₅ **FW:** 355.3 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C

Summary: Hormone sensitive lipase (HSL) catalyzes the hydrolysis of tri-, di-, and monoacylglycerols, as well as cholesterol esters, thus mobilizing fatty acids as a primary source of energy in mammals. CAY10499 is a potent inhibitor of human HSL exhibiting an IC₅₀ value of 90 nM for the recombinant enzyme. The *in vivo* pharmacological efficacy of CAY10499 has not been reported.

1 mg
5 mg
50 mg
100 mg



[4-(5-methoxy-2-oxo-1,3,4-oxadiazol-3(2H)-yl)-2-methylphenyl]-carbamic acid, phenylmethyl ester

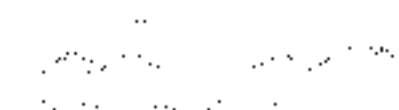
NEW CAY10514

10009017

[868526-38-9] Methyl-8-hydroxy-8-(2-pentyl-oxophenyl)-oct-5-ynoate**MF:** C₂₀H₂₈O₄ **FW:** 332.4 **Purity:** ≥98%A solution in methyl acetate **Stability:** ≥1 year at -20°C

Summary: CAY10514 is an aromatic analog of 8(S)-HETE. It acts as a dual agonist of PPARα and PPARγ with EC₅₀ values of 0.173 and 0.642 μM, respectively.

1 mg
5 mg
10 mg
50 mg



8-hydroxy-8-[2-(pentylloxy)phenyl]-5-octynoic acid, methyl ester

CD36 Monoclonal Antibody

188150

GPIIb, GPIV, Hexarelin Receptor, oxLDL Receptor, Thrombospondin ReceptorPurified mouse anti-CD36 IgA **Stability:** ≥1 year at -20°C

Summary: Antigen: adenovirus expressing full-length murine recombinant CD36 • Host: CD36 null mouse, clone JC63.1 • Cross-reactivity: (+) murine, rat, and human CD36 • Applications: flow cytometry and functional blocking • CD36 is a type-B scavenger receptor that is necessary for the formation of foam cells in atherosclerotic lesions.

100 μg
500 μg

CD36 Polyclonal Antibody

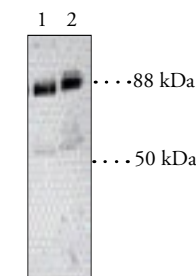
100011

GPIIb, GPIV, Hexarelin Receptor, oxLDL Receptor, Thrombospondin ReceptorPeptide affinity-purified IgG **Stability:** ≥1 year at -20°C

Summary: Antigen: human CD36 amino acids 99-114 • Host: rabbit • Cross-reactivity: (+) human, murine, and rat CD36 • Application: WB • CD36 is a type-B scavenger receptor that is necessary for the formation of foam cells in atherosclerotic lesions.

1 ea

Lane 1: Human platelet lysate (15 μg)
Lane 2: Human platelet lysate (30 μg)



Also Available: CD36 Blocking Peptide (300011)

200 μg

NEW Cetaben

10007171

[55986-43-1] Hexadecylamino-p-amino Benzoic Acid**MF:** C₂₃H₃₉NO₂ **FW:** 361.6 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C

Summary: Cetaben is a unique, PPARα-independent peroxisome proliferator with hypolipidemic activity, characterized by reduction in serum TG and cholesterol concentrations in rats. In male wistar rats, cetaben increased the activity of all peroxisomal enzymes examined in liver and kidney, whereas clofibrate showed a varied regulatory pattern. Cetaben inhibits cholesterol synthesis in the human hepatoma HepG2 cells resulting in reversible changes in Golgi morphology. It also blocked TG synthesis by 99% and reduced cholesterol ester synthesis by >70% at a concentration of 50 μM in these same cells.

5 mg
10 mg
50 mg
100 mg



4-(hexadecylamino)-benzoic acid

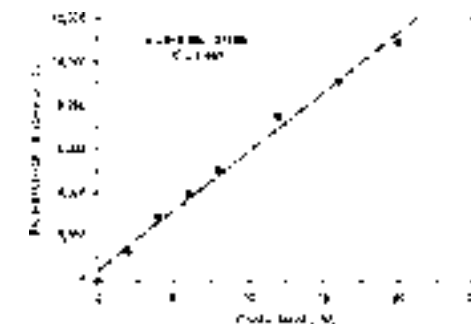
NEW Cholesterol Assay Kit

10007640

Stability: ≥1 year at -20°C

Summary: Cholesterol, particularly in the form of LDLs, is well understood to be associated with increased risk of coronary heart disease. The measurement of cholesterol is one of the most common tests performed in the clinical laboratory setting. However, simple and easy assays for cholesterol in the research lab have not been readily available. Cayman's Cholesterol Assay provides a simple fluorometric method for the sensitive quantitation of total cholesterol in plasma or serum.

96 wells
480 wells



*All 5-cis 2-series PGs (those containing a 5,6-double bond) will contain a small amount of the 5-trans isomer. This isomer is generally undetectable using normal phase silica columns and plates, but may be resolved using RP-HPLC. The purity for all such 2-series PGs excludes the 1-3% trans isomer which will generally be present.

Tom Brock, Ph.D.

Cholesterol Trafficking

Cholesterol is an essential component of cells and is used in the synthesis of steroid hormones. Consistent with its importance, cholesterol synthesis and metabolism are tightly regulated. However, serum cholesterol in particular is thought to be a key factor leading to the development of vascular disease, with the most prevalent form of vascular disease being arteriosclerosis.

Cholesterol from the diet

Surprisingly, the diet is only a minor source of cholesterol. According to the American Heart Association, Americans who eat meat typically take in 200-400 mg cholesterol each day. This amount, from the atherogenic Western diet, is less than half of what the body synthesizes each day. At the intestine, cholesterol is absorbed into enterocytes by a mechanism involving Niemann Pick C1-like protein 1 (NPC1L1). As suggested by its name, this protein is similar to NPC1, which mediates intracellular cholesterol trafficking from the lysosome to the endoplasmic reticulum and the plasma membrane. The NPC1L1 protein is abundant on intestinal brush border membranes. Rare, naturally occurring variants in NPC1L1 are associated with either high or low absorption of dietary cholesterol.¹ The uptake of cholesterol by NPC1L1 can be blocked by the medication ezetimibe. Free cholesterol taken up by the intestines can be moved through hepatic portal vessels to the liver. Alternatively, it may be packaged, with triacylglycerol and apolipoproteins, into lipoprotein particles called chylomicrons and released into the circulatory system. Chylomicrons are the largest and least dense of the lipoproteins. Lipoproteins consist of a lipid core of cholesteryl esters (cholesterol esterified with fatty acid) and triacylglycerol with a shell of free cholesterol, phospholipids and apolipoproteins. Triacylglycerol consists of three fatty acids acylated to a three carbon glycerol backbone. Apolipoproteins are lipid-binding proteins that stabilize the aggregation of many lipid molecules into each lipoprotein particle. The apolipoproteins also can associate with and modulate the activity of enzymes that process lipids. Finally, the apolipoproteins serve as ligands for specific receptors that mediate lipoprotein clearance by endocytosis. The major apolipoproteins in chylomicrons are ApoB-48, ApoC, and ApoE. In fact, chylomicrons are relatively large (100-500 nm diam.) particles composed primarily of triacylglycerol, which affords a very low density (<0.95 g/ml). In the circulatory and lymphatic systems, lipids are released from chylomicrons by lipoprotein lipases, which use ApoC-II in the chylomicron as an activator. The free fatty acids from triacylglycerol degradation are absorbed by muscle and adipose tissue, leaving chylomicron remnants. These remnants are ultimately removed by the liver by the LDL receptor, which recognizes ApoB-48 and ApoE on the remnant surface. In the liver, excess cholesterol may be incorporated into bile acids for secretion, *via* the bile duct, to the intestine. Much of this cholesterol is returned to the liver, again by absorption by enterocytes and migration through hepatic portal vessels.

Distribution of cholesterol

The major source of cholesterol is *de novo* synthesis, within the liver, the intestines and other sites. Cholesterol is synthesized by the mevalonate pathway, with early steps involving the synthesis of hydroxymethylglutaryl-coenzyme A (HMG-CoA) from acetyl-CoA. The rate-limiting step in cholesterol biosynthesis involves the conversion of HMG-CoA to mevalonate by HMG-CoA reductase; this step is inhibited by statins. Free cholesterol in the liver is packaged with triacylglycerol and apolipoproteins (predominantly ApoB-100, ApoC, and ApoE) to produce very low density lipoprotein (VLDL), which is released into circulation. The triacylglycerol within VLDL is progressively hydrolyzed by lipases, which act as glycerol-*sn*-1-fatty acid hydrolases. The best known of these is lipoprotein lipase (LPL), but triacylglycerol within lipoproteins are hydrolyzed by others, including endothelial lipase (EL) and hepatic lipase (HL). LPL is present on capillary endothelial surfaces in mammary, muscle, and adipose tissues and is activated by ApoC-II, which is found on most types of lipoproteins. EL is expressed in endothelium from a wide variety of tissues and its expression is up-regulated by inflammatory cytokines, such as TNF- α and IL-1 β . HL is produced by hepatocytes and remains primarily at the liver and associated endothelium. The liberated fatty acids produced by these enzymes are absorbed by smooth muscle and adipocytes. Hydrolysis of triacylglycerol reduces the triacylglycerol:cholesterol ratio, increasing lipoprotein density, producing intermediate and low density lipoproteins (IDL, LDL). Each LDL particle is very small (18-28 nm diam.) with very little triacylglycerol; a higher protein to cholesterol ratio leads to a higher density (1.019-1.063 g/ml). Each LDL particle contains a single molecule of the ~510 kDa ApoB-100 protein, as well as a variety of the smaller apolipoproteins. LDL serves to transport cholesterol in the blood to peripheral tissues and regulates *de novo* cholesterol synthesis at those sites after being captured by an LDL receptor (LDLR). Ultimately, the LDL remnants are captured at the liver by LDLRs on hepatocytes. The LDLRs recognize ApoB-100 and ApoE, endocytose the remnants and recycle remaining cholesterol to bile or new VLDL.

Lipids in LDL may become oxidized by free radicals in the form of reactive oxygen and nitrogen species, producing oxidized LDL (oxLDL) particles. Oxidization appears to occur both on lipids and apolipoproteins in LDL. These damaged particles are recognized by scavenger receptors (SR-A, CD36) on macrophages, endothelial cells, and dendritic cells. Macrophages are of particular interest in cholesterol trafficking as they consume and accumulate cholesterol and also release cholesterol for transport back to the liver. These macrophages may be found at or below the endothelial surface, interspersed in the smooth muscle layer, as well as throughout the adjacent adipose tissue. Macrophages that consume sufficient oxLDL become foam cells, which accumulate in the fatty streaks that are early

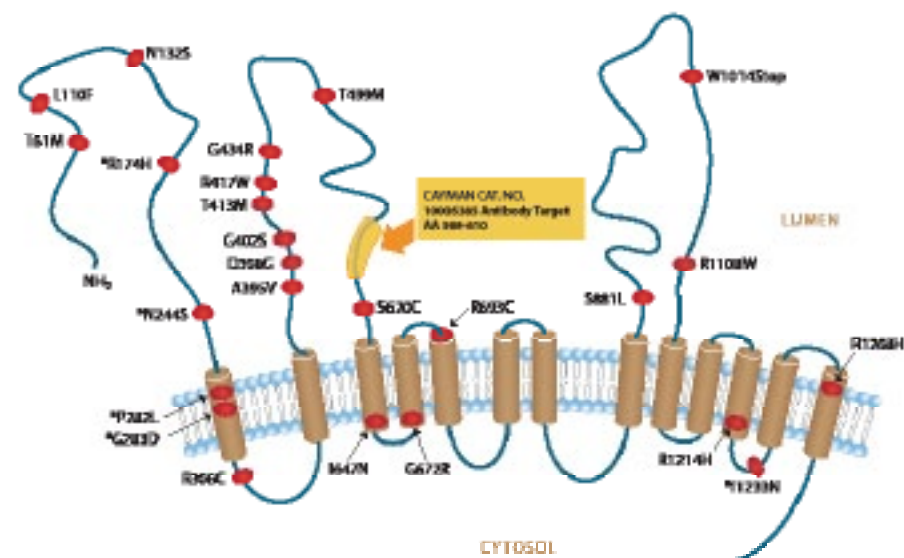


Figure 1. Predicted NPC1L1 Topology and Naturally Occurring Mutations Most mutations are associated with reduced cholesterol absorption, with only four mutations (asterisked) found in patients with high cholesterol absorption.

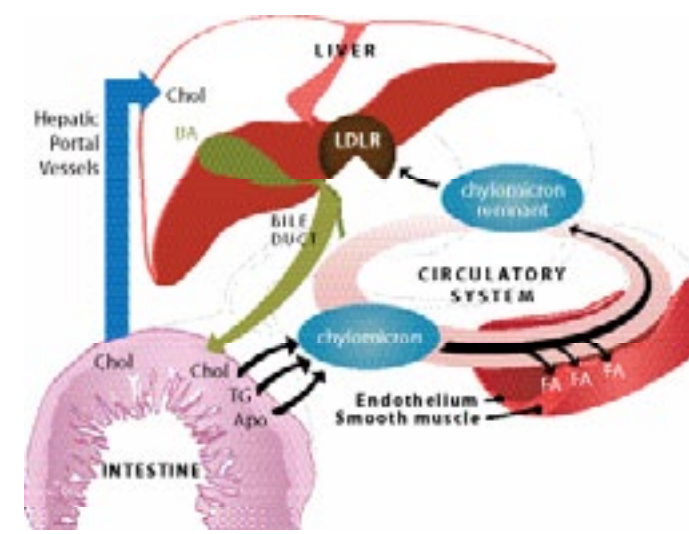


Figure 2. Dietary Cholesterol Trafficking

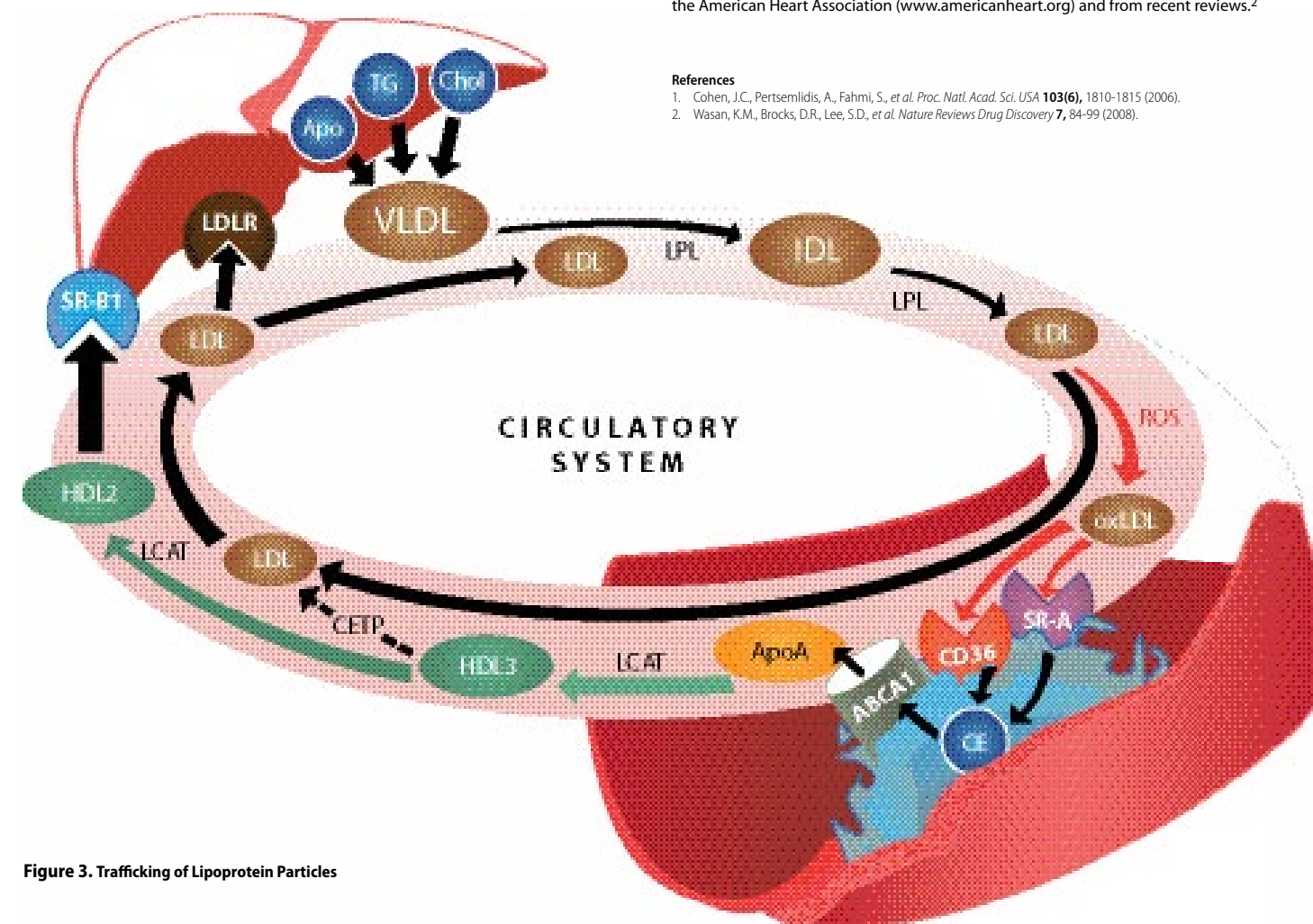


Figure 3. Trafficking of Lipoprotein Particles

indicators of atherosclerosis. OxLDL may be degraded by the cytoplasmic enzyme PAF acetyl hydrolase. Unoxidized LDL is ultimately removed from the blood by the liver *via* LDLRs.

Reverse cholesterol transport

The high density lipoprotein (HDL) serves an important role in removing cholesterol through reverse cholesterol transport. Nascent HDL, consisting predominantly of ApoA1, collects free cholesterol from dying cells and from cell membranes. Importantly, nascent HDL obtains cholesterol from macrophages or foam cells associated with cholesterol plaques. Lipid-free ApoA1 interacts with the macrophage/foam cell surface and receives free cholesterol secreted by the ATP binding cassette transporter 1 (ABCA1), and possibly also ABCG1 and SR-B1. Much of the free cholesterol associated with the nascent HDL is then esterified with fatty acids by lecithin-cholesterol acyltransferase (LCAT) to produce cholesteryl esters. Early HDL particles have been termed HDL3 and mature into late HDL, or HDL2, with continued accumulation of cholesterol into the particle. The very late HDL, HDL1, is much less abundant than HDL2 or HDL3 in blood samples. Several studies have failed to find a major impact of the HDL2:HDL3 ratio, which is typically higher in women than men, on cardiovascular disease. In addition to mediating reverse cholesterol transport, HDL particles have antioxidant effects and have anti-inflammatory effects on the function of endothelial cells and macrophages. Some of the cholesteryl ester is transferred from HDL to IDL and LDL by cholesteryl ester transfer protein (CETP), allowing "forward" transport of cholesterol. HDL is typically cleared from the circulation by binding to the scavenger receptor SR-B1 (= CLA1) on the liver, which recognizes ApoA1 and ApoE.

Additional detail regarding dietary cholesterol and cholesterol trafficking is available from the American Heart Association (www.heart.org) and from recent reviews.²

References

1. Cohen, J.C., Pertsemlidis, A., Fahmi, S., et al. *Proc. Natl. Acad. Sci. USA* **103**(6), 1810-1815 (2006).
2. Wasan, K.M., Brooks, D.R., Lee, S.D., et al. *Nature Reviews Drug Discovery* **7**, 84-99 (2008).

Characteristics of Lipoproteins

Lipoprotein Class	Density (g/ml)	Diameter (nm)	Major Apolipoproteins	Protein (% dry wt)	Phospholipid (%)	Triacylglycerol (% dry wt)	Cholesterol (% dry wt)	Function
HDL	1.063-1.21	5-15	ApoA1, ApoE	33	29	8	12-25	Reverse cholesterol transport
LDL	1.019-1.063	18-28	ApoB-100, ApoE	25	21	4	43-50	Cholesterol transport to all tissues
IDL	1.006-1.019	25-50	ApoB-100, ApoE	18	22	31	25-33	Transport of endogenous triacylglycerols
VLDL	0.95-1.006	30-80	ApoB-100, ApoE	10	18	50	17-27	Transport of endogenous triacylglycerols
chylomicrons	< 0.95	100-500	ApoB-48	1-2	7-9	84	2-4	Transport of exogenous triacylglycerols and cholesterol

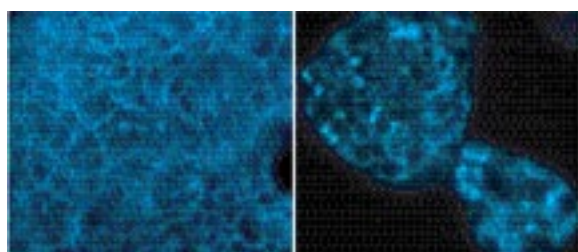
NEW Cholesterol Cell-Based Detection Assay Kit

10009779

Stability: ≥6 months at -20°C

Summary: The mechanism for the movement of cholesterol from intracellular sites to their ultimate cellular destination is an unresolved question of fundamental importance to cell biology and medicine. Thus, defining mechanisms of intracellular cholesterol transport and identifying the cellular factors involved are therefore of great interest. Cayman's Cholesterol Cell-based Detection Assay Kit includes filipin III, fixative, and wash buffer in a ready to use format. It provides a simple fluorometric method to study mechanisms and biological factors that regulate cholesterol metabolism or movement within cells. A cholesterol trafficking inhibitor, U-18666A, is included as a positive control.

192 wells



Accumulation of cholesterol inside HepG2 cells in response to 1.25 μM U-18666A. HepG2 cells were seeded in a 96-well plate at a density of 3×10^4 cells/well and cultured overnight. The next day, cells were treated with DMSO (vehicle) or 1.25 μM U-18666A for 48 hours. **Left panel:** Cells treated with DMSO alone demonstrate that majority of cholesterol is localized on the plasma membrane. **Right panel:** U-18666A treatment for 48 hours induces intracellular accumulation of cholesterol droplets, indicating blockage of intracellular cholesterol transport.

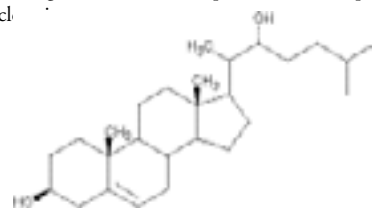
22(R)-hydroxy Cholesterol

89355

[17954-98-2] 22α-hydroxy Cholesterol

MF: C₂₇H₄₆O₂ **FW:** 402.7 **Purity:** ≥98%A crystalline solid **Stability:** ≥1 year at -20°C

Summary: 22(R)-OH-Ch is an endogenous agonist for LXRA and LXRb. The EC₅₀ value for ligand-dependent activation of LXRA by 22(R)-OH-Ch is approximately 325 nM. Acting through LXR in concert with RXR, its heterodimerization partner, 22(R)-OH-Ch induces the expression of the ABCA1 reverse cholesterol transporter. This acts to increase the efflux of cholesterol from enterocytes and thus inhibit the overall absorption of cholesterol. 22(R)-OH-Ch can be used as an endogenous positive control for the testing of LXR agonists, which have potential as therapeutic agents for the treatment of atherosclerosis.

1 mg
5 mg
10 mg
50 mg

cholest-5-ene-3β,22R-diol

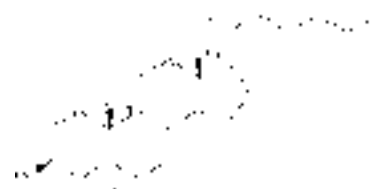
NEW 5α-hydroxy-6-keto Cholesterol

10007601

[13027-33-3] Cholestane-6-oxo-3β,5α-diol

MF: C₂₇H₄₆O₃ **FW:** 418.7 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C

Summary: 5α-hydroxy-6-keto Cholesterol is a major metabolite of cholesterol formed during exposure of lung epithelial cells to ozone, with formation of 5b,6b-epoxycholesterol as a predominant precursor. Exposure of C57BL/6J mice to 0.5-3 ppm ozone produced a dose-dependent formation of 5α-hydroxy-6-keto cholesterol which was detectable in the bronchoalveolar lavage fluid, lavaged cells, and lung homogenates. 5α-hydroxy-6-keto Cholesterol is a potent inhibitor of cholesterol synthesis in human bronchial epithelial cells with an IC₅₀ value of 350 nM and exhibits significant cytotoxicity in the low μM range. Therefore, the toxic effects of ozone may be mediated by formation oxysterols of this type.

1 mg
5 mg
10 mg
50 mg

3β,5α-dihydroxy-cholestan-6-one

Cholesteryl Linoleate Hydroperoxides

48001

MF: C₄₅H₇₆O₄ **FW:** 681.1 **Purity:** ≥98% hydroperoxide contentA solution in ethanol **Stability:** ≥6 months at -80°C

Summary: Cholesteryl linoleate hydroperoxides are derived from the autoxidation of cholesteryl linoleate and contain a mixture of racemic 9- and 13-HpODE cholesteryl esters. (±)9- and (±)13-HODE cholesteryl esters were originally extracted from atherosclerotic lesions and shown to be produced by Cu²⁺-catalyzed oxidation of LDL. 15-LO from rabbit reticulocytes and activated human monocytes oxygenates cholesteryl linoleate to both 9- and 13-hydroperoxy linoleate cholesteryl esters. Cholesteryl ester hydroperoxides may be transferred from LDL to HDL, reduced to the corresponding hydroxides, and cleared *via* the liver.

100 μg
500 μg
1 mg
5 mg(±)-9-hydroperoxy-10E,12Z-octadeca-dienoic acid, cholesteryl ester;
(±)-13-hydroperoxy-9Z,11E-octadeca-dienoic acid, cholesteryl ester

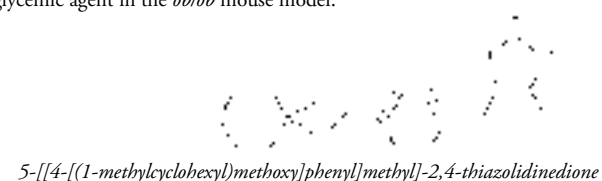
Ciglitazone

71730

[74772-77-3] ADD 3878, U-63287

MF: C₁₈H₂₃NO₃S **FW:** 333.4 **Purity:** ≥98%A crystalline solid **Stability:** ≥1 year at -20°C

Summary: Ciglitazone is an antidiabetic drug of the thiazolidinedione structural class that acts as a potent and selective PPARγ ligand. It binds to the PPARγ ligand-binding domain with an EC₅₀ value of 3.0 μM. Ciglitazone is active *in vivo* as an anti-hyperglycemic agent in the *ob/ob* mouse model.

1 mg
5 mg
10 mg
50 mg

5-[[4-[(1-methylcyclohexyl)methoxy]phenyl]methyl]-2,4-thiazolidinedione

Ciprostene (calcium salt)

18216

[81703-55-1] U-61431F

MF: [C₂₂H₃₆O₄]₂ • Ca **FW:** 384.6 **Purity:** ≥98%A crystalline solid **Stability:** ≥6 months at -20°C

Summary: Ciprostone is the 9b-methyl analog of carbaprostacyclin and a stable analog of PGI₂. Ciprostone exhibits biological activity similar to PGI₂, but is 30-fold less potent. In patas monkeys, ciprostone induces hypotension and causes tachycardia when administered at a dose of 0.16 μg/kg/min. In addition, ciprostone inhibits ADP-induced platelet aggregation *ex vivo* and *in vitro* with ID₅₀ values of 9.1 μg/kg/min and 60 ng/ml, respectively.

1 mg
5 mg
10 mg
50 mg

6,9α-methylene-9β-methyl-11α,15S-dihydroxy-prosta-5Z,13E-dien-1-oic acid, calcium salt

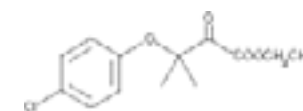
Clofibrate

10005745

[637-07-0]

MF: C₁₂H₁₅ClO₃ **FW:** 242.7 **Purity:** ≥98%A colorless liquid **Stability:** ≥1 year at -20°C

Summary: Clofibrate is PPARα agonist and a member of a class of hypolipidemic drugs that includes fenofibrate and benzafibrate, which have been used clinically to treat dyslipidemia and cardiovascular disease. In a transactivation assay, clofibrate exhibits EC₅₀ values of 50 and 55 μM for murine and human PPARα, respectively. It also binds to PPARγ, but with 10-fold less affinity and is inactive at PPARδ at concentrations up to 100 μM.

500 μl
1 ml
5 ml
10 ml

2-(4-chlorophenoxy)-2-methyl-propanoic acid, ethyl ester

Inhibitors of Lipoprotein Modifying Enzymes

Cat. No.	Catalog Name	Target	IC ₅₀
10006482	CAY10485	ACAT-1 and -2	95 μM (ACAT-1); 81 μM (ACAT-2)
10006452	CAY10486	ACAT-1 and -2	60 μM (both enzymes)
10007875	CAY10499	Hormone Sensitive Lipase	90 nM (human)
10006782	Oleic Acid-2,6-diisopropylanilide	ACAT	7 nM
10006529	Oleyl Anilide	ACAT	26 μM

COX-1 Monoclonal Antibody

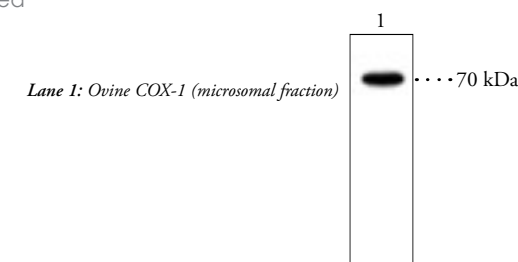
160110

Prostaglandin H Synthase 1

Lyophilized IgG **Stability:** ≥3 years at -20°C

Summary: Antigen: purified ovine COX-1 • Host: mouse, clone CX111 • Cross-reactivity: (+) ovine, bovine, human, murine, rat, and monkey COX-1, ovine COX-2 (50%), and human COX-2 (-5%); (-) murine COX-2 • Applications: WB and IHC • Isotype: IgG_{2b}

1 ea



Lane 1: Ovine COX-1 (microsomal fraction)

Also Available: COX-1 Monoclonal FITC Antibody (160111) 1 ea
COX-1 (murine) Polyclonal Antibody (160109) 1 ea
COX-1 (ovine) Polyclonal Antiserum (160108) 1 ea

CysLT₁ Receptor Polyclonal Antibody

120500

Cysteinyl Leukotriene Receptor 1

Peptide affinity-purified IgG **Stability:** ≥2 years at -20°C

Summary: Antigen: human CysLT₁ receptor C-terminal amino acids 318-337 • Host: rabbit • Cross-reactivity: (+) human and murine CysLT₁ receptor; other species not tested • Applications: WB, ICC, IHC, and flow cytometry • The CysLT₁ receptor is one of two receptor isoforms for LTC₄ and LTD₄.

1 ea

Also Available: CysLT₁ Receptor Blocking Peptide (320500) 200 μg

Cysteinyl Leukotriene EIA Kit

520501

Stability: ≥6 months at -80°C

Summary: The LTs were discovered in 1979 as a group of acute inflammatory mediators derived from arachidonic acid in leukocytes. Their biosynthesis was shown to proceed *via* the 5-LO pathway. LT biosynthesis has subsequently been demonstrated in other bone marrow-derived cells expressing 5-LO including eosinophils, mast cells, and macrophages. 5-LO converts arachidonic acid into LTA₄ with 5(S)-HpETE as an intermediate. The conjugation of glutathione to LTA₄ results in the formation of LTC₄. LTC₄ is rapidly metabolized to LTD₄ and LTE₄. This metabolism is essentially complete within 10 minutes in the human lung. LTC₄, LTD₄, and LTE₄ are collectively referred to as cysteinyl leukotrienes (CysLTs). LTC₄ and LTD₄ are potent mediators of asthma and hypersensitivity. They induce bronchoconstriction, increase microvascular permeability, and are vasoconstrictors of coronary arteries. The biological activity of LTE₄ is much lower in most systems studied, but its presence reflects the prior existence of LTC₄ and LTD₄.

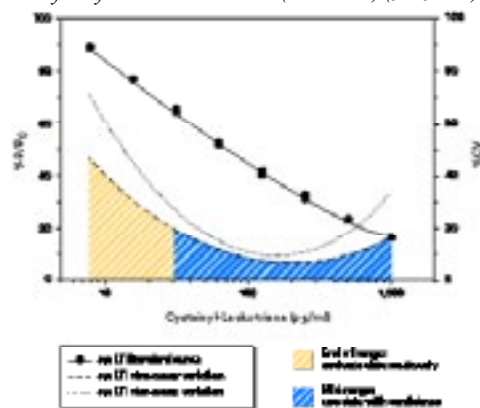
Sensitivity: 50% B/B₀: 57 pg/ml
80% B/B₀: 13 pg/ml

Specificity:

Leukotriene C ₄	100%
Leukotriene D ₄	100%
Leukotriene E ₄	67%
Leukotriene D ₅	61%
Leukotriene C ₅	54%
Leukotriene E ₅	41%
N-acetyl Leukotriene E ₄	10.5%
5-HETE	<0.01%
12-HETE	<0.01%
15-HETE	<0.01%
Leukotriene A ₃	<0.01%
Leukotriene A ₄	<0.01%
Leukotriene B ₃	<0.01%
Leukotriene B ₄	<0.01%
20-hydroxy Leukotriene B ₄	<0.01%
tetranor-PGEM	<0.01%
Prostaglandin D ₂	<0.01%
Prostaglandin E ₂	<0.01%
6-keto Prostaglandin F _{1α}	<0.01%
Prostaglandin F _{2α}	<0.01%
Thromboxane B ₂	<0.01%

96 wells
480 wells

Also Available: Cysteinyl Leukotriene EIA Kit (Solid Plate) (520501.1)



NEW Diphenyl-1-pyrenylphosphine

62237

[110231-30-6] DPPP

MF: C₂₈H₁₉P **FW:** 386.4 **Purity:** ≥98%

A crystalline solid **Stability:** ≥2 years at -20°C

Summary: DPPP is a probe that reacts stoichiometrically with hydroperoxides to yield the fluorescent molecule DPPP oxide. Plasma levels of lipid hydroperoxides of phosphatidylcholine, phosphatidylethanolamine, TGs, and cholesteryl esters have been measured by HPLC with a post column detection system using DPPP. DPPP has also been used as a fluorescent probe for the detection of LDL and cellular oxidation.

5 mg
10 mg
25 mg
50 mg



diphenyl-1-pyrenylphosphine

NEW DP₁ Receptor Polyclonal Antibody

101640

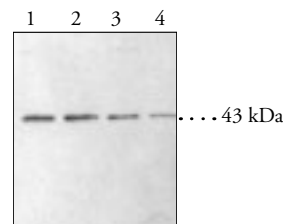
PGD₂ Receptor, Prostaglandin D₂ Receptor 1

Peptide affinity-purified IgG **Stability:** ≥1 year at -20°C

Summary: Antigen: murine DP₁ receptor N-terminal amino acids 2-21 • Host: rabbit • Cross-reactivity: (+) human, murine, and rat DP₁ receptors; other species not tested • Applications: WB and ICC; other applications not tested • The DP₁ receptor is one of two receptor isoforms for PGD₂.

1 ea

Lane 1: HT-29 cell lysate (50 µg)
Lane 2: HT-29 cell lysate (40 µg)
Lane 3: HT-29 cell lysate (30 µg)
Lane 4: HT-29 cell lysate (20 µg)



Also Available: DP₁ Receptor Blocking Peptide (301640) 200 µg

NEW Endothelial Lipase (human) Polyclonal Antibody

100030

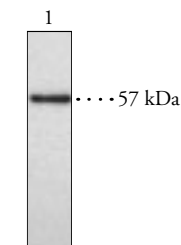
EL, EDL

Peptide affinity-purified IgG **Stability:** ≥1 year at -20°C

Summary: Antigen: human endothelial lipase amino acids 19-32 • Host: rabbit • Cross-reactivity: (+) human, murine, rat, porcine, and ovine endothelial lipase • Applications: WB and IHC • EL is a major genetic determinant for the concentration, structure, and metabolism of HDL, which protects against atherosclerosis.

1 ea

Lane 1: HepG2 cell lysate (30 µg)



Also Available: Endothelial Lipase (human) Blocking Peptide (10004111) 200 µg

Endothelin EIA Kit

583151

ET

Stability: ≥6 months at -20°C

Summary: The endothelin peptide family consists of three isoforms, ET-1 (corresponding to the initially isolated and most predominant isoform), ET-2, and ET-3. ET-1 is a 21 amino acid peptide and is one of the most potent vasoconstrictors currently known. ET-2 displays similar pharmacology to ET-1, whereas ET-3 is a weak vasoconstrictor but more potent inhibitor of platelet aggregation. Cayman's Endothelin Assay is an immunometric (i.e., sandwich) EIA that permits endothelin measurements within the range of 0-250 pg/ml, typically with a limit of detection of 1.5 pg/ml. Inter- and intraassay CV's of less than 10% may be achieved at most concentrations. This assay offers sensitive and specific analysis of endothelin in serum, plasma, urine, or cell culture media.

Sensitivity: Limit of detection: 1.5 pg/ml

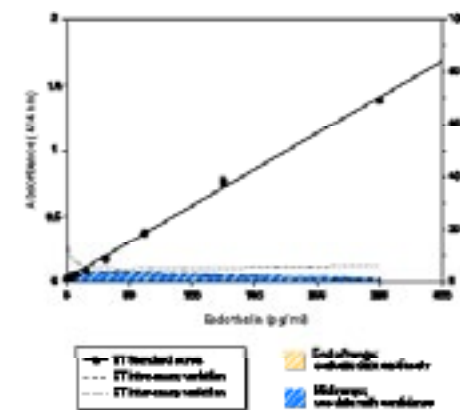
Specificity:

Endothelin-1	100%
Endothelin-2	100%
Endothelin-3	100%
VIC	100%
Big Endothelin	100%

Homology:

Mammalian Endothelin-1	100%
------------------------	------

96 wells
480 wells



NEW FABP1 (human recombinant)

10009547

L-FABP, Liver-FABP

Purity: ≥90% **Stability:** ≥6 months at -80°C

A solution in 50 mM sodium phosphate, pH 7.2, containing 25% glycerol and 100 mM sodium chloride **Stability:** ≥6 months at -80°C

Summary: Source: recombinant N-terminal His-tagged protein expressed in *E. coli* • M_r: 18.3 kDa

25 µg
50 µg
100 µg

NEW FABP1 (rat recombinant)

10005200

L-FABP, Liver-FABP

Purity: ≥95%

A solution in 50 mM sodium phosphate, pH 7.2, containing 20% glycerol and 150 mM sodium chloride **Stability:** ≥6 months at -80°C

Summary: Source: recombinant N-terminal His-tagged protein expressed in *E. coli* • M_r: 18.3 kDa

25 µg
50 µg
100 µg

NEW FABP2 Polyclonal Antibody

10010019

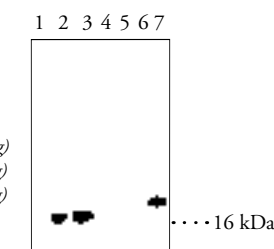
I-FABP, Intestinal-FABP

Peptide affinity-purified IgG **Stability:** ≥1 year at -20°C

Summary: Antigen: human FABP2 amino acids 33-40 • Host: rabbit • Cross-reactivity: (+) human and rat FABP2; but also expected to work with murine and bovine; (-) recombinant FABP1, 3, 4, and 5 • Application: WB

1 ea

Lane 1: Rat Recombinant FABP1 (0.4 µg)
Lane 2: Rat Recombinant FABP2 (0.025 µg)
Lane 3: Rat Recombinant FABP2 (0.050 µg)
Lane 4: Human Recombinant FABP3 (0.4 µg)
Lane 5: Murine Recombinant FABP4 (0.4 µg)
Lane 6: Murine Recombinant FABP5 (0.4 µg)
Lane 7: Human Duodenum Homogenate (30 µg)



Also Available: FABP2 Blocking Peptide (10010020) 200 µg

NEW FABP2 (rat recombinant)

10007938

I-FABP, Intestinal-FABP

Purity: ≥95%

A solution in 50 mM sodium phosphate, pH 7.2, containing 20% glycerol, 100 mM sodium chloride, and 1 mM DTT **Stability:** ≥6 months at -80°C

Summary: Source: recombinant N-terminal His-tagged protein expressed in *E. coli* • M_r: 19.3 kDa

25 µg
50 µg
100 µg

NEW FABP3 (human recombinant)

10007432

H-FABP, Heart-FABP

Purity: ≥95%

A solution in 50 mM sodium phosphate, pH 7.2, containing 20% glycerol and 150 mM sodium chloride **Stability:** ≥6 months at -80°C

Summary: Source: recombinant N-terminal His-tagged protein expressed in *E. coli* • M_r: 19 kDa

25 µg
50 µg
100 µg

FABP4 (human) EIA Kit*

10007614

*A-FABP, Adipocyte-FABP, ALBP, aP2***Stability:** ≥6 months at 4°C

Summary: FABP4 is a 15 kDa member of the intracellular FABP family, which is known for the ability to bind fatty acids and related compounds (bile acids or retinoids). FABP4 is expressed in a differentiation-dependent fashion in adipocytes and is a critical gene in the regulation of the biological function of these cells. In mice, targeted mutations in the FABP4 gene (also called aP2) provides significant protection from hyperinsulinemia and insulin resistance in the context of both dietary and genetic obesity. FABP4 is also expressed in macrophages where it modulates inflammatory responses and cholesterol ester accumulation. Total or macrophage-specific FABP deficiency confers dramatic protection against atherosclerosis in ApoE^{-/-} mice. These results indicate a central role for FABP4 in development of metabolic diseases through its distinct actions in adipocytes and macrophages. This EIA is based on a double-antibody sandwich technique. The wells of the plate supplied with the kit are coated with a goat polyclonal antibody specific of human FABP4. This antibody will bind any human FABP4 introduced in the wells (sample or standard).

Specificity:

FABP4 (chicken)	<0.1%
FABP4 (goat)	<0.1%
FABP4 (hamster)	<0.1%
FABP4 (equine)	<0.1%
FABP4 (murine)	<0.1%
FABP4 (ovine)	<0.1%
FABP4 (porcine)	<0.1%
FABP4 (rabbit)	<0.1%
FABP4 (rat)	<0.1%
Leptin (human)	<0.1%
Leptin Receptor (human)	<0.1%
Resistin (human)	<0.1%

96 wells

NEW FABP4 (human recombinant)

10009549

*A-FABP, Adipocyte-FABP, ALBP, aP2***Purity:** ≥95%

A solution in 50 mM of sodium phosphate, pH 7.2, containing 150 mM sodium chloride, and 20% glycerol **Stability:** ≥6 months at -80°C

Summary: Source: human recombinant N-terminal His-tagged protein expressed in *E. coli* • **M_r:** 18.8 kDa

25 µg
50 µg
100 µg

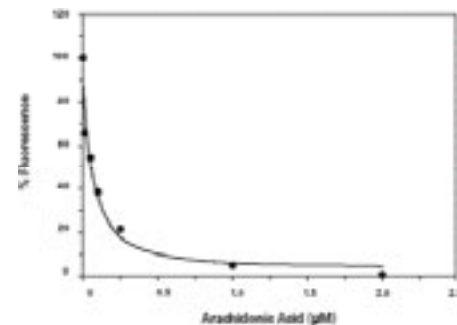
NEW FABP4 Inhibitor/Ligand Screening Assay Kit

10010231

*A-FABP, Adipocyte-FABP, ALBP, aP2***Stability:** ≥6 months at -80°C

Summary: FABP4 is highly expressed in adipocytes and is regulated by PPAR γ agonists, insulin, and fatty acids. Recent studies using FABP4 gene deletion in mice indicate a dominant role for FABP4 in several chronic metabolic diseases. Therefore, inhibiting the function of FABP4 is a potential mechanism for the treatment of metabolic diseases like diabetes and atherosclerosis. Cayman's FABP4 Ligand Binding Assay Kit provides a simple, reproducible, and sensitive tool for the identification of FABP4 ligands. The assay makes use of a Detection Reagent that exhibits increased fluorescence at 500 nm when bound to FABP4. Any strong ligand and/or inhibitor of FABP4 will displace the Detection Reagent thereby reducing the fluorescence. FABP4 is provided in high purity and in sufficient quantity for 100 tests.

96 wells

**NEW FABP4 (murine recombinant)**

10005191

*A-FABP, Adipocyte-FABP, ALBP, aP2***Purity:** ≥95%

A solution in 50 mM sodium phosphate, pH 7.2, containing 20% glycerol and 150 mM sodium chloride **Stability:** ≥6 months at -80°C

Summary: Source: murine recombinant N-terminal His-tagged protein expressed in *E. coli* • **M_r:** 19.5 kDa

25 µg
50 µg
100 µg

NEW FABP4 (murine recombinant) Western Ready Control

10009676

*A-FABP, Adipocyte-FABP, ALBP, aP2***Purity:** 18 kDa (His-tagged), 15 kDa (native)**Stability:** ≥6 months at -20°C

Summary: Source: murine recombinant His-tagged protein expressed in *E. coli* • Application: Positive control for WB

1 ea

NEW FABP4 Polyclonal Antibody

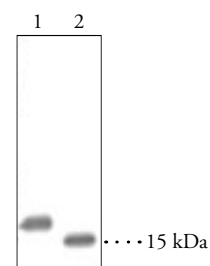
10004944

*A-FABP, Adipocyte-FABP, ALBP, aP2*Peptide affinity-purified IgG **Stability:** ≥1 year at -20°C

Summary: Antigen: human FABP4 amino acids 103-118 • Host: rabbit • Cross-reactivity: (+) human, murine, and rat FABP4; other species not tested • Applications: WB and ICC; other applications not tested

1 ea

Lane 1: His-tagged recombinant rat FABP4 lysate (1 µg)
Lane 2: Rat brown fat (30 µg)



Also Available: FABP4 Blocking Peptide (10006248)

200 µg

NEW FABP4 Western Ready Control

10010463

*A-FABP, Adipocyte-FABP, ALBP, aP2***Purity:** 18.8 kDa (His-tagged), 16 kDa (native)**Stability:** ≥1 year at -20°C

Summary: Source: human recombinant N-terminal His-tagged protein expressed in *E. coli* • Application: Positive control for WB

1 ea

NEW FABP5 (human recombinant)

10010364

*DA11 FABP, E-FABP, Epidermal-FABP, Keratinocyte FABP, Psoriasis-Associated FABP***Purity:** ≥95%

A solution in 50 mM sodium phosphate, pH 7.2, containing 20% glycerol and 100 mM sodium chloride **Stability:** ≥6 months at -80°C

Summary: Source: recombinant N-terminal His-tagged protein expressed in *E. coli* • **M_r:** 18 kDa

25 µg
50 µg
100 µg

NEW FABP5 (murine recombinant)

10007433

*DA11 FABP, E-FABP, Epidermal-FABP, Keratinocyte FABP, Psoriasis-Associated FABP***Purity:** ≥95%

A solution in 50 mM sodium phosphate, pH 7.2, containing 20% glycerol and 150 mM sodium chloride **Stability:** ≥6 months at -80°C

Summary: Source: recombinant N-terminal His-tagged protein expressed in *E. coli* • **M_r:** 19.3 kDa

25 µg
50 µg
100 µg

NEW FABP7 (human recombinant)

10009551

*B-FABP, Brain-FABP***Purity:** ≥90%

A solution in 50 mM sodium phosphate, pH 7.2, containing 20% glycerol and 100 mM sodium chloride **Stability:** ≥6 months at -80°C

Summary: Source: recombinant N-terminal His-tagged protein expressed in *E. coli* • **M_r:** 19 kDa

25 µg
50 µg
100 µg

Fenofibrate

10005368

*[49562-28-9]***MF:** C₂₀H₂₁ClO₄ **FW:** 360.8 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C

Summary: Fenofibrate is a PPAR α agonist and a member of a class of hypolipidemic drugs that includes clofibrate and bezafibrate, which have been used clinically to treat dyslipidemia and cardiovascular disease. In a transactivation assay, fenofibrate exhibits EC₅₀ values of 18 and 30 µM for murine and human PPAR α , respectively. It also binds to PPAR γ , but with at least 10-fold less affinity and is inactive at PPAR δ at concentrations up to 100 µM.

1 g
5 g
10 g
50 g

2-[4-(4-chlorobenzoyl)phenoxy]-2-methyl-propanoic acid, 1-methylethyl ester

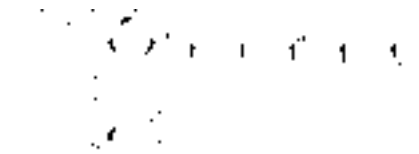
Filipin III

70440

*[480-49-9]***MF:** C₃₅H₅₈O₁₁ **FW:** 654.8 **Purity:** ≥85%A crystalline solid **Stability:** ≥1 year at -20°C

Summary: Filipin is the collective name given to four isomeric polyene macrolides isolated from cultures of *S. filipinensis*; Filipin III is the predominant isomer and the one used in most studies. Filipin binds to cholesterol in membranes, forming ultrastructural aggregates and complexes which can be visualized by freeze-fracture and electron microscopy. The binding of cholesterol decreases the intrinsic fluorescence of Filipin, and this property has been used to detect cholesterol in membrane fractions and whole cells.

1 mg
5 mg
10 mg
50 mg

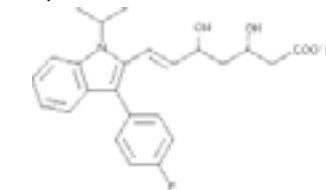
4*S*,6*S*,8*S*,10*R*,12*R*,14*R*,16*S*,27*S*-octahydroxy-3*R*-(1*R*-hydroxy-hexyl)17,28*R*-dimethyl-oxacyclooctacos-17*E*,19*E*,21*E*,23*E*,25*E*-pentaen-2-one**NEW Fluvastatin (sodium salt)**

10010337

*[93957-54-1]***MF:** C₂₄H₂₆FNO₄ • Na **FW:** 433.5 **Purity:** 98%A crystalline solid **Stability:** ≥2 years at -20°C

Summary: Fluvastatin is a competitive inhibitor of HMG-CoA reductase with respect to binding of the substrate HMG-CoA (K_i = 0.3 nM), but not with respect to binding of NADPH. When included in a clinical trial, LDL cholesterol levels were reduced by 27% after six weeks of treatment with a dose of 40 mg/kg twice a day in patients undergoing percutaneous coronary intervention.

10 mg
25 mg
50 mg
100 mg

7-[3-(4-fluorophenyl)-1-(1-methylethyl)-1*H*-indol-2-yl]-3,5-dihydroxy-6-heptenoic acid, sodium salt**FR122047 (hydrate)**

10039

MF: C₂₃H₂₅N₃O₃S • HCl[H₂O] **FW:** 478.01 **Purity:** ≥98%A crystalline solid **Stability:** ≥1 year at -20°C

Summary: FR122047 is a selective inhibitor of COX-1. The IC₅₀ values for inhibition of human COX-1 and COX-2 are 0.028 and 65 µM, respectively. In human platelet-rich plasma, FR122047 inhibits arachidonic acid, collagen, and ADP-induced platelet aggregation with an IC₅₀ value of 180-200 nM, which is nearly 100 times more potent than aspirin.

1 mg
5 mg
10 mg
50 mg

1-[[4,5-bis(4-methoxyphenyl)-2-thiazolyl]carbonyl]-4-methyl-piperazine, monohydrochloride hydrate

Tom Brock, Ph.D.

Inflammation in Atherosclerosis

Oxidants and Oxidized Phospholipids

Nothing has galvanized the health consciousness of the general public quite like the vilification of cholesterol. Despite universal convictions that “cholesterol is bad for you,” the truth is much less clear. External, dietary cholesterol is clearly a minor player. Internal, plasma cholesterol is one of several clear risk factors – but the liver orchestrates this show. There’s “bad” cholesterol, LDL, and then there’s “really bad” cholesterol, oxidized LDL (oxLDL). LDL is taken up by many cell types through the LDL receptor, delivering cholesterol that suppresses the synthesis of additional cholesterol. On the other hand, oxLDL is accumulated without restriction by macrophages, captured by scavenger receptors (e.g., CD36 and SR-A) and promotes differentiation to foam cells. In addition, oxLDL is absorbed by vascular smooth muscle after induction of SR-A. This indicates that the generation of oxidants that oxidize LDL is a critical step in the production of really bad cholesterol.

Curiously, reactive oxygen species (ROS) and nitric oxide (NO) are normal products of a healthy vascular system. ROS are formed as a by-product of the normal metabolism of oxygen and are involved in intracellular signalling and in ATP generation in all cells. NO, produced by endothelial cells, inhibits monocyte adhesion, reduces vascular tone and inhibits platelet aggregation. However, several factors, including inflammation, can dramatically increase the production of ROS. Superoxide (O_2^-), hydrogen peroxide (H_2O_2), and hydroxyl radical (OH^\bullet) are powerful oxidants associated with the phagocytic oxidative burst. These molecules damage lipids, proteins, RNA, and DNA, and transform already dangerous LDL into its most lethal form. Or they can react with NO to produce peroxynitrite ($ONOO^-$) another damaging ROS.

ROS attack polyunsaturated fatty acids (PUFA) in phospholipids on cell membranes and lipoprotein particles. Oxidation of the *sn*-2 PUFA can lead to cleavage of the PUFA to release a short hydrocarbon chain with a highly reactive aldehyde end group. Examples include 4-hydroxy-2-nonenal (HNE) and 4-hydroxy-2-hexenal (HHE). These lipids form adducts with proteins; specific antibodies against protein-bound HNE or HHE have been found in atherosclerotic lesions. These hydroxyl alkenals can also covalently bind to the primary amine moiety of ethanolamine phospholipids (PE), and their carboxylic acid metabolites can be measured in urine as an indicator of oxidative stress.¹

Cleavage of *sn*-2 PUFA leaves an oxidized phospholipid (oxPL) with a truncated *sn*-2 residue, modified with an aldehyde or keto group. Examples include 1-palmitoyl-2-(5-oxovaleroyl)-*sn*-glycero-3-phosphorylcholine (POV-PC), 1-palmitoyl-2-glutaroyl-*sn*-glycero-3-phosphorylcholine (PG-PC), and 1-palmitoyl-2-(5-keto-6-octene-diyl) phosphatidylcholine (KOdiA-PC). The polar aldehyde/keto group induces a conformational change in head group position. Also, the polar group re-orientates the *sn*-2 side chain toward the aqueous phase, producing a “lipid whisker” projecting from the phospholipid bilayer.² Thus displayed, the oxidized lipid presents a pattern recognized by scavenger receptors, such as CD36. The model, then, is that increased ROS will oxidize phospholipids on the surface of

LDL particles which subsequently serve as targets for pattern recognition receptors and, in particular, scavenger receptors. Important questions remain regarding the regulation of these receptors in the context of atherosclerosis.

oxPL initiate and modulate many of the cellular events seen in the developing fatty streak. Complex mixtures of oxPL induce an inflammatory response by the induction of pro-inflammatory genes (MCP-1, IL-8, tissue factor, etc). In endothelial cells, oxPL change the expression of genes related to angiogenesis, atherosclerosis, inflammation, and wound healing. In addition oxPL activate platelets, induce adherence, and differentiation of monocytes and promote the dedifferentiation of smooth muscle cells—processes related to plaque formation. oxPL act *via* transcription factors such as PPAR α , PPAR γ , NFAT, and Egr-1. An example is the potent PPAR γ ligand azelaoyl PAF. They also modulate the fate of an inflammatory response by intervening into such processes as removal of apoptotic cells and by dampening bacterial-induced inflammation.

The recently identified lipoprotein-associated phospholipase A₂ (Lp-PLA₂, same as PAF acetyl hydrolase, PAF-AH) hydrolyzes the *sn*-2 fatty acid of oxLDL in lesion-prone artery walls yielding the pro-inflammatory, atherogenic by-products lysophosphatidylcholine and oxidized nonesterified fatty acids. In turn, these bioactive lipid mediators act as chemoattractants for monocytes, impair endothelial function, disrupt plasma membranes, and induce apoptosis in smooth muscle cells and macrophages. Lp-PLA₂ is produced and secreted by monocytes, macrophages, T-lymphocytes, and liver cells. Expression is dependent on leukocyte activation and is upregulated in macrophages within atherosclerotic plaques. The majority of circulating Lp-PLA₂ is found bound to LDL. As Lp-PLA₂ levels reflect the circulating lipoprotein profile, the state of leukocytes, and the inflammatory composition of the atherosclerotic plaque, it can be used as a prognostic indicator for cardiovascular outcomes.^{3,4} On the other hand, in its role as PAF-AH, this enzyme inactivates the pro-inflammatory mediator PAF. Also, because Lp-PLA₂ removes the “lipid whiskers” that make oxLDL identifiable to scavenger receptors, Lp-PLA₂ may revert oxLDL to normal LDL when oxidative stress is relatively low.⁵ In this scenario, Lp-PLA₂ essentially serves to prevent or delay the conversion of LDL to oxLDL, reducing endocytosis *via* scavenger receptors.

Additional information regarding ROS, oxLDL and oxPL, particularly in the context of atherosclerosis, are available in recent reviews.⁶⁻⁸

References

- Guichardant, M., Bacot, S., Molière, P., et al. *Prostaglandins Leukot. Essent. Fatty Acids* **75**, 179-182 (2006).
- Greenberg, M.E., Li, X.-M., Gugiu, B.G., et al. *J. Biol. Chem.* **283**(4), 2385-2396 (2008).
- Sabatine, M.S., Morrow, D.A., O'Donoghue, M., et al. *Arterioscler. Thromb. Vasc. Biol.* **27**, 2463-2469 (2007).
- Persson, M., Hedblad, B., Nelson, J.J., et al. *Arterioscler. Thromb. Vasc. Biol.* **27**, 1411-1416 (2007).
- Noto, H., Hara, M., Karasawa, K., et al. *Arterioscler. Thromb. Vasc. Biol.* **23**, 829-835 (2003).
- Lambeth, J.D. *Free Radic. Biol. Med.* **43**(3), 332-347 (2007).
- Lindemann, S., Krämer, B., Daub, K., et al. *Curr. Opin. Lipidol.* **18**, 566-573 (2007).
- Zandbergen, F. and Plutzky, J. *Biochim. Biophys. Acta* **1771**(8), 972-982 (2007).

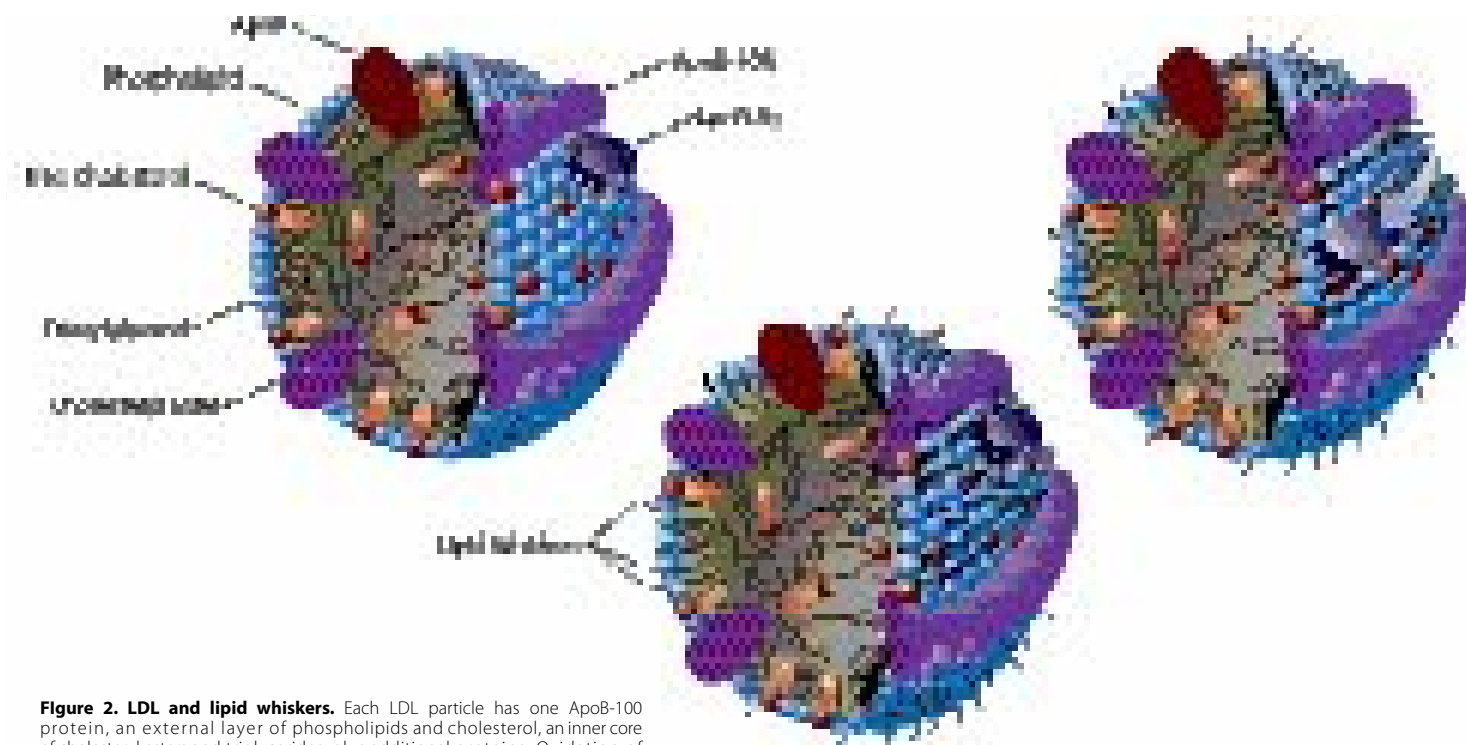


Figure 2. LDL and lipid whiskers. Each LDL particle has one ApoB-100 protein, an external layer of phospholipids and cholesterol, an inner core of cholesteryl esters and triglycerides, plus additional proteins. Oxidation of surface phospholipids involves cleavage of *sn*-2 polyunsaturated fatty acids. The shortened, oxygenated fatty acid, being hydrophilic, moves to the LDL surface, forming a lipid whisker. These whiskers, which are recognized by scavenger receptors and mediate internalization of oxLDL, are removed by Lp-PLA₂, yielding lysophospholipid, released carboxylic acid-aldehyde/ketone whiskers, and a “shaved” LDL particle.

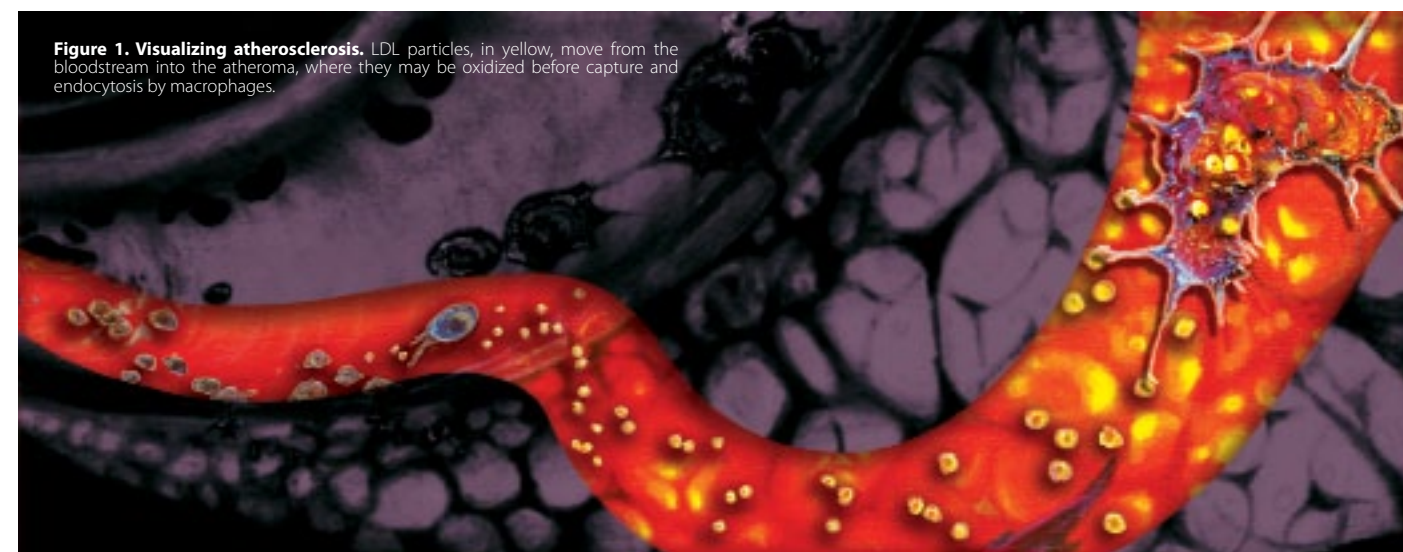


Figure 1. Visualizing atherosclerosis. LDL particles, in yellow, move from the bloodstream into the atheroma, where they may be oxidized before capture and endocytosis by macrophages.

Apolipoproteins

Apolipoprotein	Associates with:	Notes
ApoA1*	HDL, primary apolipoprotein in HDL	Interacts with ABCA1 and stimulates cholesterol efflux from tissues to liver; LCAT co-factor and activator; interacts with ApoA1 binding protein; palmitoylated; deficiency leads to HDL deficiencies (e.g., Tangier disease) and systemic non-neuropathic amyloidosis; 28.3 kDa
ApoA-II	HDL>chylomicron	Homodimer; disulfide-linked; also forms a disulfide-linked heterodimer with ApoD; enhances hepatic lipase activity; deficiency may lead to hypercholesterolemia; 8.7 kDa
ApoA-IV*	HDL>chylomicron	Role unknown, perhaps intestinal lipid absorption; synthesized in the intestine and secreted in plasma; activates LCAT <i>in vitro</i> ; 45.4 kDa (unprocessed precursor)
ApoA-V*	HDL, VLDL	Minor; stimulates ApoC-II-LPL hydrolysis of TG; PPAR α regulated expression; 41.2 kDa (unprocessed precursor)
ApoB-48	chylomicron	Main apolipoprotein of chylomicrons; synthesized in gut; binds LDLR; derived from same gene as ApoB-100 through alternative splicing; 240 kDa (48% of ApoA-100 size)
ApoB-100	VLDL, IDL, LDL, primary apolipoprotein in LDL	Main apolipoprotein of LDL; synthesized in liver; lacks a binding domain for LDLR, so poorly cleared by LDLR, binding better with LDLR-related proteins (LRP); ApoB-100 levels correlate well with risk for coronary heart disease; deficiency associated with familial hypobetalipoproteinemia; palmitoylated; 510 kDa
ApoC-I	VLDL, HDL, chylomicron	Activates LCAT; inhibits CTEP; can inhibit hepatic lipase; 7.0 kDa
ApoC-II	VLDL, HDL, chylomicron	Co-factor for LPL; associates with ApoC-III and ApoE on nascent chylomicron to form mature chylomicron; 8.8 kDa
ApoC-III*	VLDL, IDL, HDL, chylomicron	Inhibits LPL and hepatic lipase <i>in vitro</i> ; may delay catabolism of triglyceride-rich particles; high ApoC-III levels leads to hypertriglyceridemia and increased risk for atherosclerosis; 8.8 kDa
ApoC-IV		Little information available
ApoD	HDL, VLDL	Homologous to plasma retinol-binding protein, lipocalins; homodimer; dimerizes with ApoA-II; associated with LCAT; glycoprotein; 21.3 kDa (unprocessed precursor)
ApoE*	VLDL, IDL, LDL, HDL, chylomicron	Binds to LDLR; essential for normal catabolism of TG-rich lipoprotein constituents; ApoE deficiency leads to impaired clearance of chylomicron, VLDL, and IDL remnants, resulting in increased plasma cholesterol and TG; may also be relevant to Alzheimer's disease, immunoregulation, and cognition; glycosylated; 34.1 kDa
ApoH	chylomicron	Binds negatively charged species, including heparin, phospholipids, and dextran sulfate; may be involved in lipoprotein metabolism, coagulation, and the production of antiphospholipid autoantibodies; also known as β_2 glycoprotein 1; 38.3 kDa (unprocessed precursor)

*A1, C-III, A-IV, and A-V genes are clustered on one chromosome (11 in humans); A1 and A-IV genes are transcribed from the same strand; the A1 and C-III genes are convergently transcribed. Abbreviations: ABCA1, ATP binding cassette transporter 1; CTEP, cholesteryl ester transfer protein; HDL, high density lipoprotein; IDL, intermediate density lipoprotein; LCAT, lecithin:cholesterol acyltransferase; LDL, low density lipoprotein; LDLR, low density lipoprotein receptor; LPL, lipoprotein lipase; TG, triglyceride; VLDL, very low density lipoprotein.

Furegrelate (sodium salt) 70540

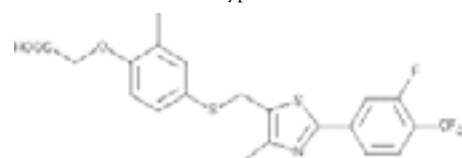
[85666-17-7] U-63557A

MF: C₁₅H₁₀NO₃ • Na **FW:** 275.2 **Purity:** ≥99%A crystalline solid **Stability:** ≥2 years at -20°C**Summary:** Furegrelate is a potent inhibitor of TX synthase with little effect on other enzymes essential for arachidonate metabolism. The IC₅₀ value is 15 nM for human platelet TX synthase.1 mg
5 mg
10 mg
50 mg

5-(3-pyridinylmethyl)-2-benzofuran-2-carboxylic acid, sodium salt

NEW GW 0742 10006798

[317318-84-6]

MF: C₂₁H₁₇F₄NO₃S₂ **FW:** 471.5 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C**Summary:** GW 0742 is a selective PPARδ agonist (EC₅₀ = 1.1 nM) that exhibits 1,000-fold selectivity over the other human PPAR subtypes.5 mg
10 mg
25 mg
50 mg

4-[[[2-[3-fluoro-4-(trifluoromethyl)phenyl]-4-methyl-5-thiazolyl]methyl]thio]-2-methyl phenoxy]-acetic acid

NEW GW 7647 10008613

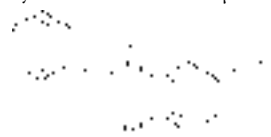
[265129-71-3]

MF: C₂₉H₄₆N₂O₂S **FW:** 502.8 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C**Summary:** GW 7647 is a potent, selective agonist of human and murine PPARα. It activates human PPARα, PPARγ, and PPARδ with EC₅₀ values of 0.006, 1.1, and 6.2 μM, respectively, in a GAL4-PPAR binding assay. GW 7647 lowered TGs 93% and 60% in fat-fed hamsters and rats, respectively, at a dose of 3 mg/kg.1 mg
5 mg
10 mg
25 mg

2-methyl-2-[[4-[2-[[[(cyclohexylamino)carbonyl](4-cyclohexylbutyl)amino]ethyl]phenyl]thio]-propanoic acid

GW 9662 70785

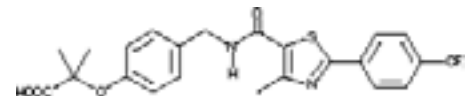
[22978-25-2]

MF: C₁₃H₉ClN₂O₃ **FW:** 276.7 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C**Summary:** GW 9662 is a potent PPARγ antagonist. It blocks the PPARγ-induced differentiation of monocytes to osteoclasts by >90% at a dose of 0.1 μM.1 mg
5 mg
10 mg
50 mg

2-chloro-5-nitrobenzanilide

NEW GW 590735 10009880

[622402-22-6]

MF: C₂₃H₂₁F₃N₃O₄S **FW:** 478.5 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C**Summary:** Activation of PPARα results in increased clearance of TG-rich VLDL via a reduction in plasma levels of ApoC-III and in upregulation of ApoA1. GW 590735 is a potent and selective agonist of PPARα with an EC₅₀ value of 4 nM for the expression of a GAL4-responsive reporter gene and at least 500-fold selectivity versus PPARγ and PPARδ.1 mg
5 mg
10 mg
50 mg

2-methyl-2-(4-((4-methyl-2-(4-(trifluoromethyl)phenyl)thiazole-5-carboxamido)methyl)phenoxy)propanoic acid

Hesperetin 10006084

[520-33-2]

MF: C₁₆H₁₄O₆ **FW:** 302.3 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at room temperature**Summary:** Hesperetin is a citrus flavonoid that has been reported to lower plasma cholesterol. Hesperetin reduces the transcription of ACAT-2 mRNA in HepG2 cells and reduces ApoB protein synthesis in a dose-dependent manner. The EC₅₀ value for these responses is approximately 50 μM. Hesperetin also inhibits histamine release from IgE-challenged RBL-2H3 cells, with a potency comparable to the commercial anti-allergy drug azelastine.25 g
50 g
100 g
500 g

2,3-dihydro-5,7-dihydroxy-2S-(3-hydroxy-4-methoxyphenyl)-4H-1-benzopyran-4-one

12(S)-HHTrE 34590

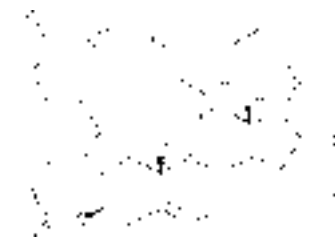
[54397-84-1] 12-HHT

MF: C₁₇H₂₈O₃ **FW:** 280.4 **Purity:** ≥97%A solution in ethanol **Stability:** ≥1 year at -20°C**Summary:** 12(S)-HHTrE is an unusual product of the COX pathway and one of the primary arachidonic acid metabolites of the human platelet. It is biosynthesized by TX synthase from PGH₂ concurrently with TXA₂. The biological role of 12(S)-HHTrE is uncertain. It is avidly oxidized to 12-oxoHHTrE by porcine 15-hydroxy PGDH.25 μg
50 μg
100 μg
250 μg

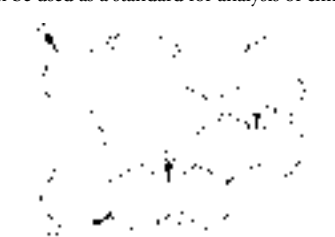
12S-hydroxy-5Z,8E,10E-heptadecatrienoic acid

(±)9-HODE cholesteryl ester 38401

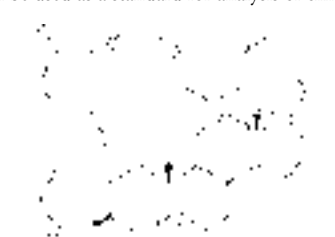
[33783-76-5]

MF: C₄₅H₇₆O₃ **FW:** 665.1 **Purity:** ≥98%A solution in ethanol **Stability:** ≥1 year at -20°C**Summary:** (±)9-HODE cholesteryl ester was originally extracted from atherosclerotic lesions and shown to be produced by Cu²⁺-catalyzed oxidation of LDL. Later studies determined that 15-LO from rabbit reticulocytes and human monocytes were able to metabolize cholesteryl linoleate, a major component of LDL, to 9-HODE cholesteryl ester.25 μg
50 μg
100 μg
250 μg

(±)-9-hydroxy-10E,12Z-octadecadienoic acid, cholesteryl ester

9(R)-HODE cholesteryl ester 38406**MF:** C₄₅H₇₆O₃ **FW:** 665.1 **Purity:** ≥98%A solution in ethanol **Stability:** ≥1 year at -20°C**Summary:** 9(R)-HODE cholesteryl ester was originally extracted from atherosclerotic lesions. It remains uncertain whether the oxidized fatty acid portion of the molecule results from enzymatic lipoxygenation or from random lipid peroxidation. 9(R)-HODE cholesteryl ester can be used as a standard for analysis of chiral HODE cholesteryl esters.25 μg
50 μg
100 μg
250 μg

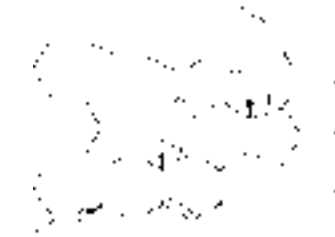
9R-hydroxy-10E,12Z-octadecadienoic acid, cholesteryl ester

9(S)-HODE cholesteryl ester 38411**MF:** C₄₅H₇₆O₃ **FW:** 665.1 **Purity:** ≥98%A solution in ethanol **Stability:** ≥1 year at -20°C**Summary:** 9(S)-HODE cholesteryl ester was originally extracted from atherosclerotic lesions. It remains uncertain whether the oxidized fatty acid portion of the molecule results from enzymatic lipoxygenation or from random lipid peroxidation. 9(S)-HODE cholesteryl ester can be used as a standard for analysis of chiral HODE cholesteryl esters.25 μg
50 μg
100 μg
250 μg

9S-hydroxy-10E,12Z-octadecadienoic acid, cholesteryl ester

(±)13-HODE cholesteryl ester 38601

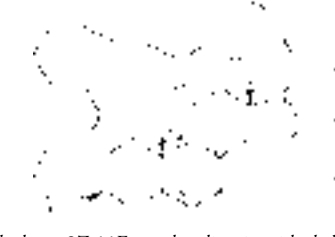
[167354-91-8]

MF: C₄₅H₇₆O₃ **FW:** 665.1 **Purity:** ≥98%A solution in ethanol **Stability:** ≥1 year at -20°C**Summary:** (±)13-HODE cholesteryl ester was originally extracted from atherosclerotic lesions and shown to be produced by Cu²⁺-catalyzed oxidation of LDL. Later studies determined that 15-LO from rabbit reticulocytes and human monocytes were able to metabolize cholesteryl linoleate, a major component of LDL, to 13-HODE cholesteryl ester.25 μg
50 μg
100 μg
500 μg

(±)-13-hydroxy-9Z,11E-octadecadienoic acid, cholesteryl ester

13(R)-HODE cholesteryl ester 38606**MF:** C₄₅H₇₆O₃ **FW:** 665.1 **Purity:** ≥98%A solution in ethanol **Stability:** ≥1 year at -20°C**Summary:** 13(R)-HODE cholesteryl ester was originally extracted from atherosclerotic lesions. It remains uncertain whether the oxidized fatty acid portion of the molecule results from enzymatic lipoxygenation or from random lipid peroxidation. 13(R)-HODE cholesteryl ester can be used as a standard for analysis of chiral HODE cholesteryl esters.25 μg
50 μg
100 μg
250 μg

13R-hydroxy-9Z,11E-octadecadienoic acid, cholesteryl ester

13(S)-HODE cholesteryl ester 38611**MF:** C₄₅H₇₆O₃ **FW:** 665.1 **Purity:** ≥98%A solution in ethanol **Stability:** ≥1 year at -20°C**Summary:** 13(S)-HODE cholesteryl ester was originally extracted from atherosclerotic lesions. It remains uncertain whether the oxidized fatty acid portion of the molecule results from enzymatic lipoxygenation or from random lipid peroxidation. 13(S)-HODE cholesteryl ester can be used as a standard for analysis of chiral HODE cholesteryl esters.25 μg
50 μg
100 μg
250 μg

13S-hydroxy-9Z,11E-octadecadienoic acid, cholesteryl ester

NEW Hormone Sensitive Lipase Polyclonal Antibody 10006371

HSL

Peptide affinity-purified IgG **Stability:** ≥1 year at -20°C**Summary:** Antigen: synthetic peptide from human HSL amino acids 731-741 • Host: rabbit • Cross-reactivity: (+) human, ovine, murine, and rat HSL; other species not tested • Applications: WB and ICC; other applications not tested • HSL catalyzes the hydrolysis of tri-, di-, and monoacylglycerols, as well as cholesterol esters.

1 ea

Also Available: Hormone Sensitive Lipase Blocking Peptide (10006372)

200 μg

I-SAP 19021[133538-58-6] *Iodophenyl sulfonyl amino pinane TXA₂***MF:** C₂₂H₃₀INO₄S **FW:** 531.4 **Purity:** ≥98%A solution in ethanol **Stability:** ≥2 years at -20°C

Summary: I-SAP is a high affinity TP receptor antagonist. At physiologic pH, I-SAP produces platelet shape change, but not aggregation, with an EC₅₀ value of 9.7 nM. I-SAP binds to human platelets with the maximum binding obtained between pH 6.5 and pH 7.4. In washed human platelets, the K_d value for I-SAP is 468 pM at pH 7.4 and 490 pM at pH 6.5.

100 µg
500 µg
1 mg
5 mg

[3S-[1α,2α,3β,5α]]-7-[3-[[[4-iodophenyl]sulfonyl]amino]-6,6-dimethylbicyclo[3.1.1]hept-2-yl]-5Z-heptenoic acid

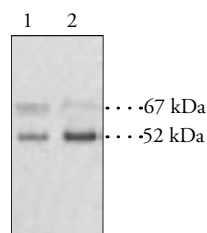
Iloprost 18215[78919-13-8] *Ciloprost***MF:** C₂₂H₃₂O₄ **FW:** 360.5 **Purity:** ≥97%A solution in methyl acetate **Stability:** ≥1 year at -20°C

Summary: Iloprost is a second generation structural analog of prostacyclin (PGI₂) with about ten-fold greater potency than the first generation stable analogs, typified by carbaprostacyclin. Iloprost binds with equal affinity to the human recombinant IP and EP₁ receptors with a K_i value of 11 nM. Iloprost constricts the isolated guinea pig ilium and fundus circular smooth muscle (an EP₁ receptor preparation) as strongly as PGE₂ itself. Iloprost inhibits the ADP, thrombin, and collagen-induced aggregation of human platelets with an ED₅₀ value of about 13 nM. In whole animals, iloprost acts as a vasodilator, hypotensive, antidiuretic, and prolongs bleeding time. It has been evaluated in several human clinical studies as a treatment for idiopathic pulmonary hypertension. In these studies, an aerosolized dose of 30 µg/day was effective, and doses as high as 150 µg/day for up to a year were well tolerated.

500 µg
1 mg
5 mg
10 mg*9S-hydroxy-10E,12Z-octadecadienoic acid, cholesteryl ester***NEW IP Receptor (human) Polyclonal Antibody** 10005518*PGI₂ Receptor, Prostacyclin Receptor*Peptide affinity-purified IgG **Stability:** ≥1 year at -20°C

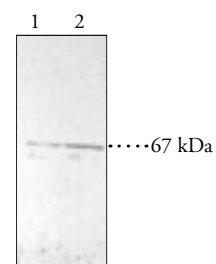
Summary: Antigen: human IP receptor N-terminal amino acids 1-16 • Host: rabbit • Cross-reactivity: (+) human, murine, and rat IP receptors; other species not tested • Application: WB; other applications not tested

1 ea

Lane 1: Human platelet lysate (4 µg)
Lane 2: Human platelet lysate (8 µg)**Also Available:** IP Receptor (human) Blocking Peptide (10005519) 200 µg**NEW IP Receptor (murine) Polyclonal Antibody** 160070*PGI₂ Receptor, Prostacyclin Receptor*Peptide affinity-purified IgG **Stability:** ≥2 years at -20°C

Summary: Antigen: murine IP receptor N-terminal amino acids 3-16 • Host: rabbit • Cross-reactivity: (+) murine and rat IP receptor; (-) human IP receptor; other species not tested • Application: WB; other applications not tested

1 ea

Lane 1: Murine brain homogenate (40 µg)
Lane 2: Murine brain homogenate (80 µg)**Also Available:** IP Receptor (murine) Blocking Peptide (360070) 200 µg**KDdiA-PC** 62935

[439904-34-4]

MF: C₃₆H₆₆NO₁₁P **FW:** 719.9 **Purity:** ≥98%A solution in ethanol **Stability:** ≥1 year at -20°C

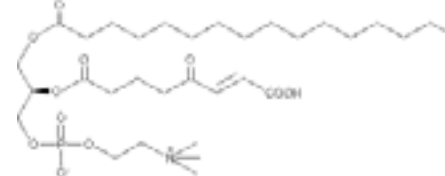
Summary: oxLDL particles contain low molecular weight species which are cytotoxic and pro-atherogenic. Many of these substances have been isolated and purified from oxLDL and identified as phosphatidylcholine species containing a fragmented, oxidized short-chain fatty acid remnant at the sn-2 position. KDdiA-PC is one of the most potent CD36 ligands among the oxLDL species. KDdiA-PC confers CD36 scavenger receptor binding affinity to LDL at a frequency of only two to three KDdiA-PC molecules/LDL particle, and may be one of the more important structural determinants of oxLDL.

1 mg
5 mg
10 mg
50 mg*1-(palmitoyl)-2-(4-keto-dodec-3-ene-diyl)phosphatidylcholine***KOdiA-PC** 62945

[439904-33-3]

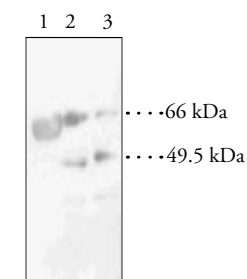
MF: C₃₂H₅₈O₁₁P **FW:** 663.8 **Purity:** ≥98%A solution in ethanol **Stability:** ≥1 year at -20°C

Summary: oxLDL particles contain low molecular weight species which are cytotoxic and pro-atherogenic. Many of these substances have been isolated and purified from oxLDL and identified as phosphatidylcholine species containing a fragmented, oxidized short-chain fatty acid remnant at the sn-2 position. KOdiA-PC, is one of the most potent CD36 ligands among the oxLDL species. KOdiA-PC confers CD36 scavenger receptor binding affinity to LDL at a frequency of only two to three KOdiA-PC molecules/LDL particle, and may be one of the more important structural determinants of oxLDL.

1 mg
5 mg
10 mg
50 mg*1-(palmitoyl)-2-(5-keto-6-octenediyl) phosphatidylcholine***NEW LCAT Polyclonal Antibody** 10009323*Lecithin:Cholesterol Acyltransferase, Phosphatidylcholine-Sterol O-Acyltransferase*Peptide affinity-purified IgG **Stability:** ≥1 year at -20°C

Summary: Antigen: human LCAT protein amino acids 132-143 • Host: rabbit • Cross-reactivity: (+) human, murine, porcine, and bovine LCAT • Application: WB • LCAT catalyzes the fatty acid transfer from the sn-2 position of phosphatidylcholine (lecithin) to cholesterol and to a lesser degree to other acceptor molecules. This enzyme is critical to the process of reverse cholesterol transport or movement of cholesterol esters into HDL particles from cells.

1 ea

Lane 1: Human plasma (20 µg)
Lane 2: Human plasma minus albumin (10 µg)
Lane 3: Human plasma minus albumin (5 µg)**Also Available:** LCAT Blocking Peptide (10009324) 200 µg**NEW LDL-Dylight™ 549** 10011229

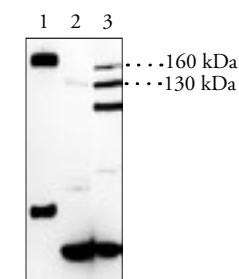
Summary: LDL uptake and its regulation are important therapeutic targets for atherosclerosis and related diseases. This preparation of LDL consists of human LDL conjugated to Dylight™ 549 as a fluorescent probe for detection of LDL uptake into cultured cells.

1 ea

NEW LDL Receptor Polyclonal Antibody 10007665*LDLR*

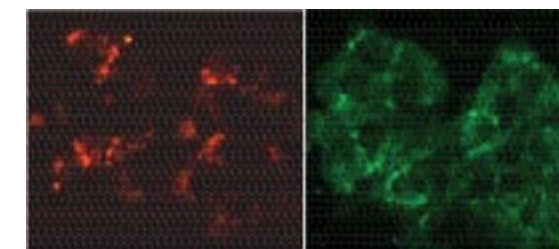
Summary: Antigen: murine LDLR amino acids 499-511 • Host: rabbit • Cross-reactivity: (+) human, murine, and rat LDLRs • Applications: WB and ICC • The LDLRs are cell surface glycoproteins that scavenge LDL from the blood and regulate plasma LDL cholesterol.

1 ea

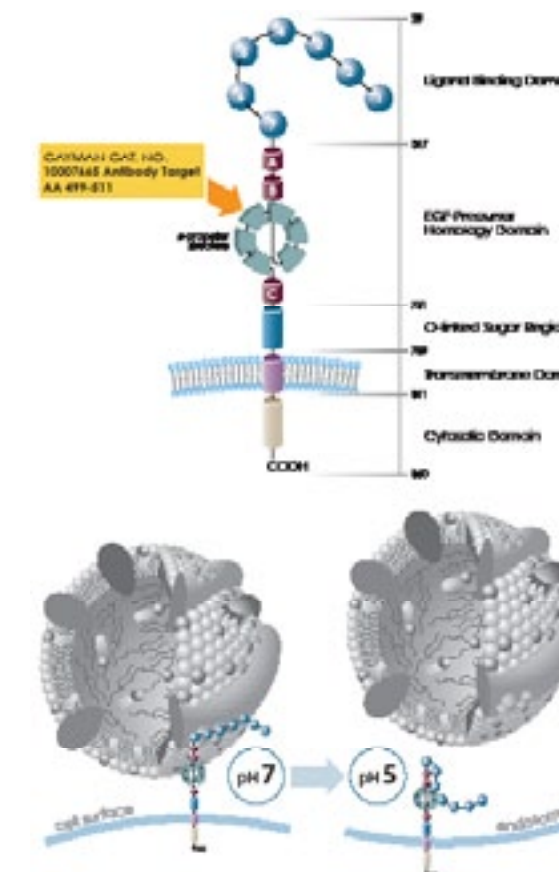
Lane 1: Rat placenta homogenate (60 µg)
Lane 2: Murine liver homogenate (60 µg)
Lane 3: RAW 264.7 cell lysate (60 µg)**Also Available:** LDL Receptor Blocking Peptide (10007672) 200 µg**NEW LDL Uptake Cell-based Assay Kit** 10011125

LDL uptake and its regulation are important therapeutic targets for atherosclerosis and related diseases. Cayman Chemical's LDL Cellular Uptake Assay Kit employs a preparation of human LDL conjugated to Dylight™ 549 as a fluorescent probe for detection of LDL uptake into cultured cells. A LDLR-specific antibody and a Dylight™ 488-conjugated secondary antibody are included in the kit for identifying the distribution of LDLRs. The kit provides a convenient tool for studying LDL uptake and its regulation at the cellular level.

1 ea



LDL Uptake in HepG2 cells: HepG2 cells were cultured at a density of 4 × 10⁵ cells/well in a 96-well plate for two days and then treated with 52 µM EGCG overnight. LDL-Dylight™ 549 (10 µg/ml) was added and incubated for four hours. Cells were fixed and stained for the LDL receptor using a rabbit anti-LDL receptor polyclonal and a goat anti-rabbit IgG Dylight™ 488-conjugated secondary antibody. Left panel: LDL-Dylight™ 549 taken into cells appear in red. Right panel: LDL receptors in green show a distribution pattern that matches cells in the left panel containing LDL Dylight™ 549.

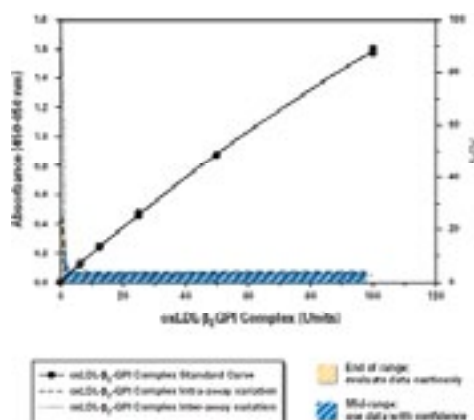
LDLR and Model for Release of LDL in the Endosome

The LDL receptor has five functionally distinct regions. The N-terminal ligand binding repeats 3-7 can bind ApoB on LDL particles at neutral pH. At endosomal pH, LDL dissociates from the receptor as ligand binding repeats 4 and 5 engage in intramolecular contacts with the b propeller domain.

NEW oxLDL-b₂GPI (human) ELISA Kit 10007893Oxidized low-density lipoprotein-b₂ Glycoprotein I (human)**Stability:** ≥6 months at 4°C

Summary: oxLDL is the principal form of cholesterol that accumulates in atherosclerotic lesions or plaques. Unlike native LDL, oxLDL binds to b₂GPI to form oxLDL-b₂GPI complexes. Stable oxLDL-b₂GPI complexes are regarded as pathogenic and appear to be highly clinically relevant. Cayman's oxLDL-b₂GPI (human) ELISA is an immunometric (*i.e.*, sandwich) assay that detects the circulating oxLDL-b₂GPI complex in human serum or plasma. The wells of each 96-well plate are coated with a monoclonal antibody against human b₂GPI. Bound oxLDL-b₂GPI is detected using a horseradish peroxidase (HRP)-labeled monoclonal antibody directed against human ApoB-100. Results are calculated against a standard curve prepared from the Reference Solution provided in the kit.

96 wells

**NEW** Leukotriene A₄ Hydrolase (human recombinant) 10007817LTA₄H**Purity:** ≥90%A solution in 100 mM Tris **Stability:** ≥6 months at -80°C

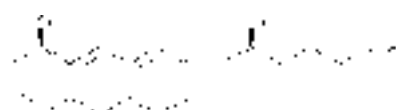
Summary: Source: recombinant C-terminal His-tagged enzyme expressed in *E. coli* • M_r: ~69 kDa

25 µg
50 µg
100 µgLeukotriene B₄ 20110

[71160-24-2]

MF: C₂₀H₃₂O₄ **FW:** 336.5 **Purity:** ≥97%A solution in ethanol **Stability:** ≥1 year at -20°C

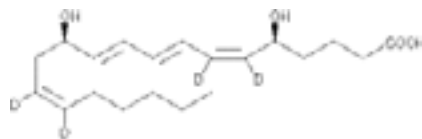
Summary: LTB₄ is a dihydroxy fatty acid derived from arachidonic acid through the 5-LO pathway. It promotes a number of leukocyte functions including aggregation, stimulation of ion fluxes, enhancement of lysosomal enzyme release, superoxide anion production, chemotaxis, and chemokinesis. In subnanomolar ranges (3.9 x 10⁻¹⁰ M), LTB₄ causes chemotaxis and chemokinesis in human PMNL. At higher concentrations, (1.0 x 10⁻⁷ M), LTB₄ leads to neutrophil aggregation and degranulation as well as superoxide anion production.

25 µg
50 µg
100 µg
500 µg

5S,12R-dihydroxy-6Z,8E,10E,14Z-eicosatetraenoic acid

Leukotriene B₄-d₄ 320110**MF:** C₂₀H₂₈D₄O₄ **FW:** 340.5 **Chemical Purity:** ≥97%**Deuterium Incorporation:** ≤1% d₀A solution in acetonitrile **Stability:** ≥1 year at -20°C

Summary: LTB₄-d₄ contains four deuterium atoms at the 6, 7, 14, and 15 positions. It is intended for use as an internal standard for the quantification of LTB₄ by GC- or LC-MS.

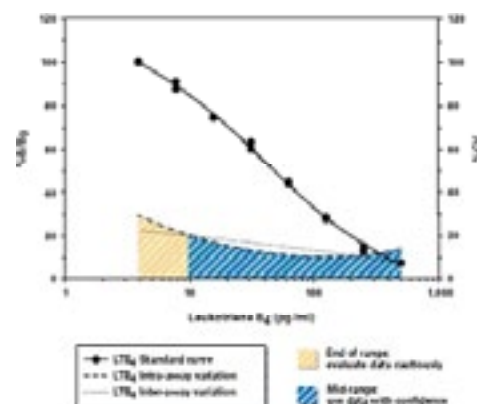
25 µg
50 µg
100 µg
500 µg5S,12R-dihydroxy-6Z,8E,10E,14Z-eicosatetraenoic-6,7,14,15-d₄ acidLeukotriene B₄ EIA Kit 520111**Stability:** ≥6 months at -20°C

Summary: LTB₄ is synthesized from arachidonic acid by the combined action of 5-LO and LTA₄ hydrolase. LTB₄ has long been recognized as a potent mediator of inflammation. It stimulates a number of leukocyte functions, including aggregation, stimulation of ion fluxes, enhancement of lysosomal enzyme release, superoxide anion production, chemotaxis, and chemokinesis. In subnanomolar ranges (3.9 x 10⁻¹⁰ M), LTB₄ causes chemotaxis and chemokinesis in human PMNLs. At higher concentrations (1.0 x 10⁻⁷ M), LTB₄ leads to neutrophil aggregation and degranulation as well as superoxide anion production. Plasma levels of LTB₄ increase from <100 pg/ml to >100 ng/ml following leukocyte stimulation. LTB₄ is metabolized in leukocytes and hepatocytes to less active 20-hydroxy and 20-carboxy LTB₄ by NADPH-dependent cytochrome P450 enzymes followed by β-oxidation at the ω-end to ω-carboxy dinor LTB₄ and ω-carboxy tetranor LTB₄. LTB₄ is not excreted in the urine.

Sensitivity: 50% B/B₀: 50 pg/ml
80% B/B₀: 13 pg/ml

Specificity:

Leukotriene B ₄	100%
5(S)-HETE	6.6%
5(R)-HETE	3.7%
20-hydroxy Leukotriene B ₄	2.7%
15(R)-HETE	0.98%
15(S)-HETE	0.4%
6-trans-12-epi Leukotriene B ₄	0.31%
6-trans Leukotriene B ₄	0.11%
5,6-DiHETE	0.07%
Glutathione	<0.01%
20-carboxy Leukotriene B ₄	<0.01%
Leukotriene C ₄	<0.01%
Leukotriene D ₄	<0.01%
Leukotriene E ₄	<0.01%
19(R)-hydroxy Prostaglandin B ₂	<0.01%

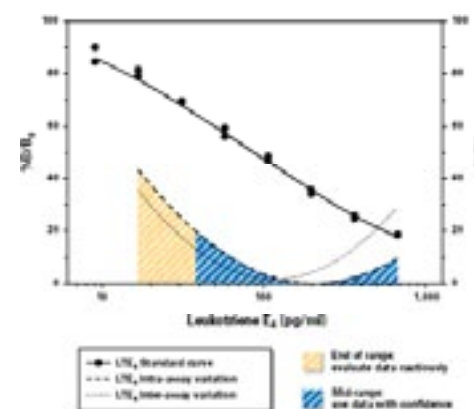
96 wells
480 wellsAlso Available: Leukotriene B₄ EIA Kit (Solid Plate) (520111.1)Leukotriene E₄ EIA Kit 520411**Stability:** ≥6 months at -80°C

Summary: LTE₄ is a product of the 5-LO pathway in activated mast cells, eosinophils, and monocytes. LTA₄, the primary 5-LO metabolite, is converted to LTC₄ and sequentially to LTD₄ and LTE₄ in the host cell, or by transcellular metabolism in erythrocytes, platelets, or neutrophils. This metabolism is rapid and complete, in that plasma levels of LTC₄ are virtually undetectable. Exogenously administered LTC₄ is recovered in the urine as LTE₄ (5-13%) and two prominent oxidized metabolites resulting from several cycles of β-oxidation. Plasma LTE₄ levels are <2 pg/ml as a consequence of the low rate of production and rapid elimination. Normal human urine contains low but detectable amounts of LTE₄, ranging from 10-60 pg/ml. Asthmatic patients in an acute episode of bronchoconstriction may have elevations of urinary LTE₄ to several hundred pg/ml, but their baseline LTE₄ levels are not consistently abnormal.

Sensitivity: 50% B/B₀: 125 pg/ml
80% B/B₀: 30 pg/ml

Specificity:

Leukotriene E ₄	100%
Leukotriene E ₅	100%
N-acetyl Leukotriene E ₄	20%
Leukotriene C ₄	10%
Leukotriene D ₄	7%
11-trans Leukotriene E ₄	6.6%
Leukotriene C ₅	2%
Arachidonic Acid	<0.01%
Leukotriene B ₄	<0.01%
Leukotriene B ₅	<0.01%
Leukotriene D ₅	<0.01%
tetranor-PGEM	<0.01%
tetranor-PGFM	<0.01%

96 wells
480 wellsAlso Available: Leukotriene E₄ EIA Kit (Solid Plate) (520411.1)

Linolein Hydroperoxides 89430

Purity: ≥98% (A mixture of 132 isomers)A solution in ethanol **Stability:** ≥2 years at -80°C

Summary: Linolein hydroperoxides are a mixture of 132 possible isomers of mono-, di-, and tri-hydroperoxides produced from the autoxidation of trilinolein. Autoxidation of linoleic acid-containing TGs (for example, trilinolein) *in vivo* could result in the formation of these hydroperoxides. Unlike the free fatty acid hydroperoxides of linoleic acid (for example, 13-HpODE), linolein hydroperoxides are not readily reduced in human plasma *in vitro*. Circulating linolein hydroperoxides could contribute to the pathophysiology of atherosclerosis.

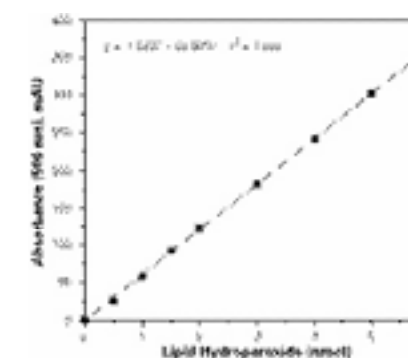
500 µg
1 mg
5 mg
50 mg

Lipid Hydroperoxide (LPO) Assay Kit 705002

Stability: ≥1 year at 4°C

Summary: Quantification of lipid peroxidation is essential to assess the role of oxidative injury in pathophysiological disorders. Lipid peroxidation results in the formation of highly reactive and unstable hydroperoxides of both saturated and unsaturated lipids. Our Lipid Hydroperoxide Assay Kit measures hydroperoxides directly utilizing redox reactions with ferrous ions. An easy to use quantitative extraction method was developed to extract lipid hydroperoxides into chloroform, and the extract is directly used in the assay. This procedure eliminates any interference caused by hydrogen peroxide or endogenous ferric ions in the sample and provides a sensitive and reliable assay for lipid peroxidation. This kit is designed for use with either a single-tube spectrophotometer to read the results or with a 96-well microplate reader. The plate used with the microplate reader is a reusable glass plate which is available with the purchase of Catalog No. 705003. The range of the assay is 0.25-5 nmol hydroperoxide per tube.

100 dtn



Also Available: Lipid Hydroperoxide (LPO) Assay Kit (96 well) (705003)

96 wells

5-Lipoxygenase (human recombinant) 60402

MF: Monomer **FW:** 78 kDa **Purity:** 16,000 x g supernatant

A solution in 100 mM Tris, pH 8.0, containing 2 mM EGTA

Stability: ≥6 months at -80°C

Summary: EC 1.13.11.34 • Recombinant enzyme isolated from a Baculovirus overexpression system in Sf21 cells • Avoid repeated thawing and refreezing • One unit of enzyme consumes one nmol of oxygen per minute at 25°C in 50 mM Tris-HCl buffer, pH 7.5, containing 100 µM arachidonate, 2 mM CaCl₂, and 1 mM ATP

500 units
1 Kunit
2.5 Kunit
5 Kunit**NEW** 5-Lipoxygenase (Phospho-Ser⁵²³) Polyclonal Antibody 10007820Affinity-purified rabbit polyclonal antibody **Stability:** ≥1 year at -20°C

Summary: Antigen: phosphopeptide corresponding to amino acid residues surrounding the phospho-Ser⁵²³ of human 5-LO • Host: rabbit • Cross-reactivity: (+) human, rat, and non-human primate 5-LO • Application: WB

100 µl

FDA Approved - 510(k) CEMark High Risk Atherosclerosis

When Aspirin no longer works



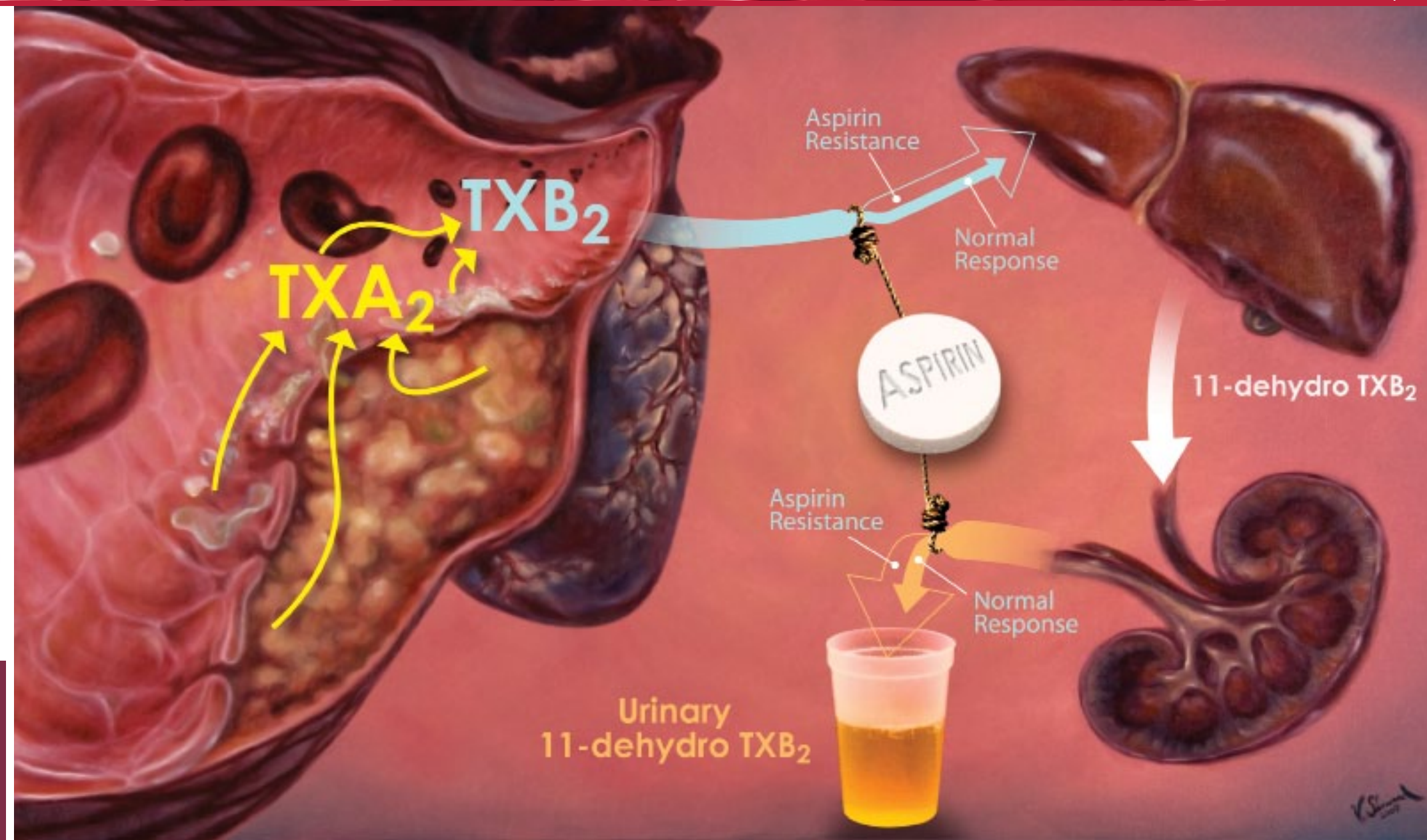
Aspirin Effect-Detection Kit

Millions of Americans ingest a daily 'minidose' (80 mg) of aspirin as a means of selectively inhibiting platelet cyclooxygenase-1 (COX-1) and subsequent production of TXA₂. Recent studies indicate that a subpopulation of these users do not achieve the desired level of inhibition of TXA₂, as determined by its more stable metabolites. The Cayman Aspirin Effect-Detection Kit is a 510(k), clinically-approved diagnostic kit for the measurement of 11-dehydro TXB₂. It is intended to help physicians assess the effectiveness of their patients' aspirin regime, and to help identify the high-risk group who are inadequately controlled on an 80 mg aspirin dose.

Thromboxane A₂ (TXA₂) is produced from arachidonic acid by many cells and causes irreversible platelet aggregation and vascular and bronchial smooth muscle contraction.¹⁻³ TXA₂ is rapidly hydrolyzed non-enzymatically to form TXB₂. Although it is common to estimate TXA₂ levels by measuring TXB₂, most of the TXB₂ measured is due to *ex vivo* platelet activation or intra-renal production.⁴ Measurement errors are compounded by the fact that normal concentrations of circulating TXB₂ are extremely low (1-2 pg/ml), and highly transient (t_{1/2} = 5-7 minutes).^{5,6} To circumvent this problem, it is necessary to measure a metabolite that cannot be formed by platelets or by the kidney. TXB₂ may be metabolized by 11-hydroxy TX dehydrogenase to form 11-dehydro TXB₂, or by

β-oxidation to form 2,3-dinor TXB₂.⁷ Infusion studies using TXB₂ have shown that both metabolites are formed equally, although 11-dehydro TXB₂ has a longer circulating half-life (t_{1/2} = 45 minutes).^{6,8} Therefore, measurement of 11-dehydro TXB₂ in plasma or urine will give a time-integrated indication of TXA₂ production.

Current methods for detecting aspirin resistance include a number of blood-based assays that measure *in vitro* platelet aggregation. However, these methods are not quantitative and can be affected by factors that are unrelated to aspirin sensitivity. In order to more quickly and accurately detect aspirin resistance in at-risk patients, we have developed a rapid, quantitative competitive immunoassay for 11-dehydro TXB₂ using a monoclonal antibody. This new assay can be completed in three hours and utilizes urine as the sample matrix. The assay exhibits intra-assay %CV values of <11% with sensitivity sufficient to detect the lower levels of 11-dehydro TXB₂ in patients that respond well to aspirin. This patented



monoclonal ELISA for 11-dehydro TXB₂ detection combines the speed of the blood-based assays with the sensitivity and quantitation of the ELISA.

References

1. Hamberg, M., et al. *Proc. Natl. Acad. Sci. USA* **72**, 2994-2998 (1975).
2. Ellis, E.F., Oelz, O., Roberts, L.J., et al. *Science* **193**, 1135-1137 (1976).
3. Salzman, P.M., Salmon, J.A., Moncada, S. *J. Pharmacol. Exp. Ther.* **215**, 240-247 (1980).
4. Samuelsson, B., Granström, E., Green, K., et al. *Ann. Rev. Biochem.* **44**, 669-695 (1975).
5. Patrono, C., Ciabattini, G., Pugliese, F., et al. *J. Clin. Invest.* **77**, 590-594 (1986).
6. Lawson, J.A., Patrono, C., Ciabattini, G., et al. *Anal. Biochem.* **155**, 198-205 (1986).
7. Roberts, L.J., II, Sweetman, B.J., and Oates, J.A. *J. Biol. Chem.* **256**, 8384-8393 (1981).
8. Ciabattini, G., Pugliese, F., Davi, G., et al. *Biochim. Biophys. Acta* **992**, 66-70 (1989).

Developed in cooperation by: Cayman Chemical, Corgenix, and Creative Clinical Concepts

NEW Aspirin Effect-Detection Kit - FDA Approved - 510(k) CEMark

10010153

Stability: ≥1 year at 4°C

About the Assay: Cayman's Aspirin-effect Detection Kit provides a highly sensitive urine-based monoclonal ELISA for detection of both 11-dehydro TXB₂ and 11-dehydro-2,3 dinor TXB₂. The detection of multiple TXA₂ metabolites provides additional sensitivity and reproducibility as compared to assays that detect only one metabolite, *i.e.*, 11-dehydro TXB₂.

Limit of Detection: 222 pg/ml

Specificity:

11-dehydro-2,3-dinor Thromboxane B ₂	166%
11-dehydro Thromboxane B ₂	100%
2,3-dinor Thromboxane B ₂	1.90%
Prostaglandin D ₂	0.20%
Thromboxane B ₂	0.05%
PGEM	<0.01%
PGFM	<0.01%
6-keto Prostaglandin _{1α}	<0.01%

1 ea

Tom Brock, Ph.D.

Inflammation in Atherosclerosis Macrophage Functions

Macrophages are highly active and mobile cells that function at multiple levels within the innate immune system. Derived from circulating monocytes, macrophages police the intimal and medial layers below the endothelium of vessels, capturing pathogens, dead cells, and cellular debris. When necessary, they emit an array of chemical messengers to the cells around them to orchestrate changes as part of an immune response. Macrophages are central to vascular inflammation and their role in atherosclerosis was recently reviewed in detail.¹

In healthy individuals, there are few, scattered resident macrophages in all tissues. Part of their function is to maintain sterility in their immediate region by migrating through the tissue and ingesting and killing pathogens. Macrophages are uniquely designed to capture pathogens because their surfaces bristle with receptors that specifically detect, bind, and internalize those targets. Macrophages also are coated with receptors to capture and ingest dead cells and a wide array of cellular debris that they find in their vicinity. Relevant to atherosclerosis, macrophages have specific receptors to identify normal and modified (oxidized, acetylated) lipoprotein particles. These include LDL receptors as well as several scavenger receptors, such as SR-A (or macrophage scavenger receptor 1), CD36 (or fatty acid translocase), and SR-PSOX (or CXCL16).

The two major macrophage classes are the M1 macrophages that drive killer T-cell activation *via* IL-12, and the M2 macrophages that secrete IL-10 and promote a general inflammatory response. Pro-inflammatory M2 macrophages in the vascular smooth muscle bind and transduce signals from oxLDL and thus differ from those in adipose tissue or hepatic tissue. As these macrophages ingest oxLDL to become the lipid-rich foam cells characteristic of atherosclerosis, their molecular signature

changes further. Foam cells differ markedly from normal resident macrophages in healthy vessels in that foam cells are less mobile and secrete more inflammatory mediators than normal macrophages. In addition, a recent study indicates that foam cells may functionally mimic dendritic cells in their ability to present antigens and support an immune response.² Also, macrophages that differentiate into foam cells in the presence of insulin and glucose differ further, suggesting that these cells may contribute to the development of insulin resistance.³

Lipid accumulation, and also macrophage differentiation, is not necessarily unidirectional. Macrophages can export cholesterol, secreting it through ATP-binding cassette transporters such as ABCA1, donating cholesterol to ApoA1 to form HDL. It appears that the rate of cholesterol export is dependent on the amount of ApoA1. As a result, if there is sufficient ApoA1, macrophages can offset the influx of cholesterol from LDL captured by scavenger receptors and have a net loss of cellular cholesterol. This is important because accumulation of cellular cholesterol eventually leads to the secretion of monocyte chemoattractants, like MCP-1, which increases tissue monocyte/macrophage numbers and allows further lipid accumulation. Increasing ApoA1 (and HDL), then, is anti-inflammatory, reducing lipid accumulation as well as keeping the number of lipid-accumulating cells low.

References

1. Yan, Z.-Q. and Hansson, G.K. *Immunol. Rev.* **219**, 187-203 (2007).
2. Cho, H.J., Shashkin, P., Gleissner, C.A., et al. *Physiological Genomics* **29**, 149-160 (2007).
3. Shashkin, P.N., Jain, N., Miller, Y.L., et al. *Cardiovascular Diabetology* **5**(13), 1-11 (2006).

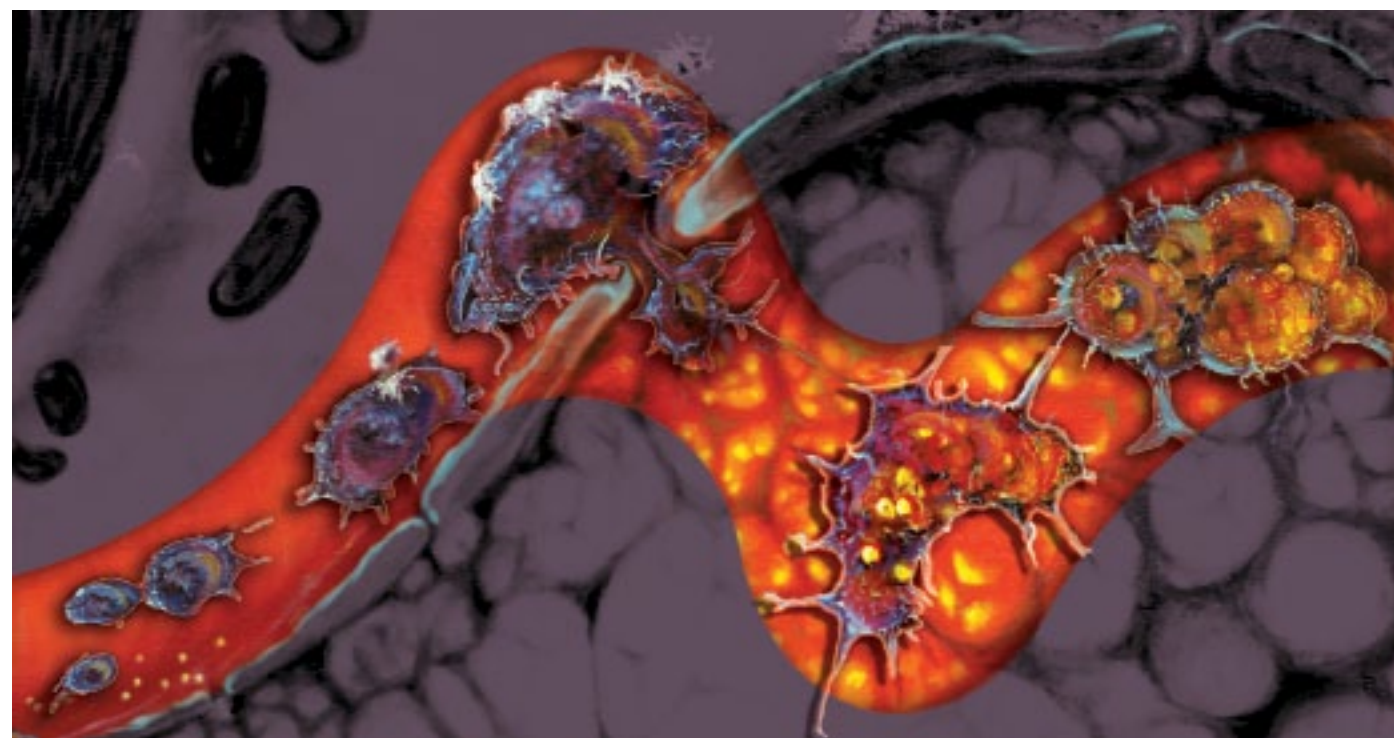


Figure 1. Macrophages as Middlemen in Atherosclerosis. Macrophages absorb LDL and oxLDL, accumulating cholesterol and becoming foam cells. Macrophages also initiate reverse cholesterol transport, donating cholesterol to ApoA1 to form HDL. Also, macrophages secrete numerous mediators, including ROS, leukotrienes, prostaglandins, and cytokines.

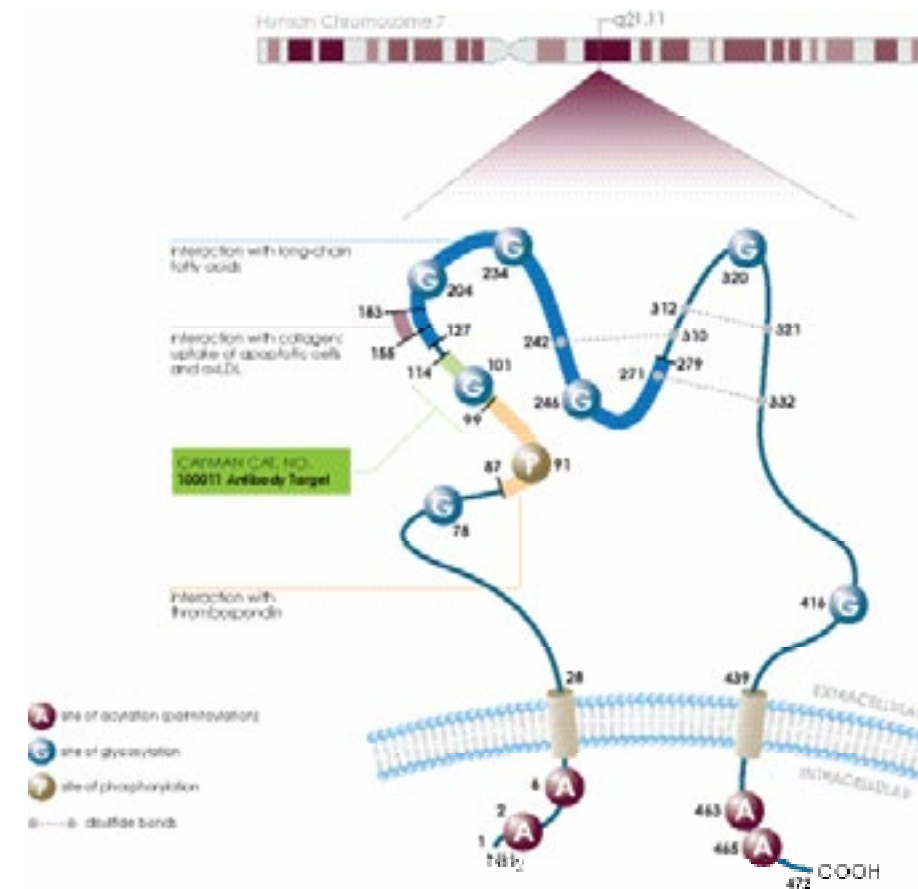


Figure 2. Predicted structure of CD36, receptor for oxLDL, oxidized phospholipids, long chain fatty acids, thrombospondin, and collagen type I.

Scavenger Receptors			
Receptor	Ligands	Function in Atherosclerosis	Notes
SR-A, SCARA1, MSR1	acLDL, oxLDL,	Captures modified LDL and accumulates lipids in macrophages in arterial wall	aka CD204, phSR1, phSR2; three different types (1, 2, 3) generated by alternative splicing; macrophage-specific trimeric integral membrane; induced by high glucose, oxidative stress, IL-6, or PAF
MARCO, SCARA2	Gram negative and gram positive bacteria	unclear	Induced by oxidative stress, depletes reactive oxygen species
SCARA3	unclear	unclear	aka CSR, APC7, CSR1, MSLR1, MSRL1; induced by oxidative stress, depletes reactive oxygen species
SR-BI, CLA1	ApoA1, HDL, normal LDL, oxLDL, serum amyloid A	Capture HDL for cholesterol removal by hepatocytes	aka CD36L1; MGC138242; serum amyloid A decreases HDL cholesterol metabolism by competing for SR-B1; SR-B1 is essential for infection with hepatitis C virus; involved in cholesterol efflux from vessel wall, as well cholesterol uptake by liver
SR-BII	unclear	unclear	aka CD36L2, HLG85, LIMP2; glycoprotein located primarily in limiting membranes of lysosomes and endosomes, involved in cell membrane transport processes
CD36, fatty acid translocase (FAT), thrombospondin receptor	oxLDL, oxidized phospholipids, long chain fatty acids, thrombospondin, collagen type I	Captures modified LDL and accumulates lipids in macrophages in arterial wall	aka GP4, GP3B, GPIV, CHDS7, PASIV, SCARB3; expression up-regulated by oxLDL or resistin, and in type II diabetes mellitus; serves as thrombospondin receptor on platelets; may function as a cell adhesion molecule
SR-PSOX	oxLDL, phosphatidylserine, CXCR6/Bonzo	Captures modified LDL and accumulates lipids in macrophages in arterial wall	aka SCY B16, CXCL16; induces a strong chemotactic response in macrophages; also exists as a soluble form; expressed in spleen, lymph nodes, lung, kidney, small intestine, and thymus
SCARF1	acLDL	Captures modified LDL	aka SREC-I, scavenger receptor expressed by endothelial cells; found on endothelial cells
SCARF2	unclear	Adhesion	aka Scavenger receptor expressed by endothelial cells 2 protein, SREC-II, SRECRP-1; probable adhesion protein; poorly binds acLDL; interacts with SCARF1

Abbreviations: SR, scavenger receptor; MSR1, macrophage scavenger receptor 1; SCARA, scavenger receptor A; acLDL, acetylated LDL; oxLDL, oxidized LDL; PAF, platelet-activating factor; MARCO, macrophage receptor with collagenous structure; CLA1, CD36 antigen-like 1

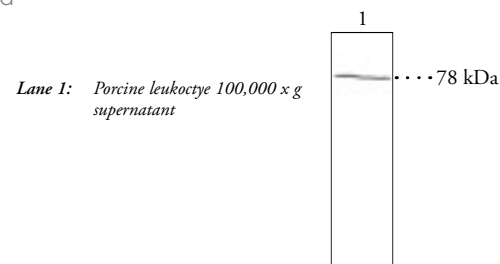
5-Lipoxygenase Polyclonal Antibody 160402

A solution of antiserum containing BSA and 0.02% sodium azide

Stability: ≥2 years at -20°C

Summary: Antigen: human and rat 5-LO amino acids 130-149; The sequence is 95% homologous to 5-LO from mouse and hamster • Host: rabbit • Cross-reactivity: (+) human, rat, murine, hamster, and porcine 5-LO; (-) 12-LO and 15-LO • Applications: WB, ICC, and IHC • 5-LO catalyzes the formation of 5(S)-HpETE from arachidonic acid as well as its subsequent conversion to LTA₄.

1 ea



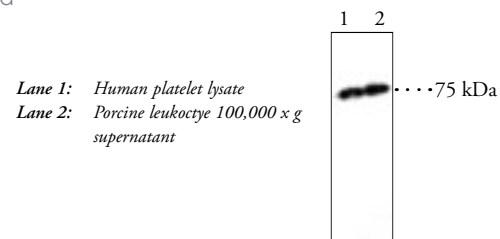
Also Available: 5-Lipoxygenase Blocking Peptide (360402) 200 µg

12-Lipoxygenase (murine leukocyte) Polyclonal Antiserum 160304

100 µl lyophilized antiserum **Stability:** ≥2 years at -20°C

Summary: Antigen: recombinant murine leukocyte 12-LO • Host: rabbit • Cross-reactivity: (+) murine, porcine, and human leukocyte 12-LO and rabbit reticulocyte 15-LO; other species not tested • Application: WB; other applications not tested

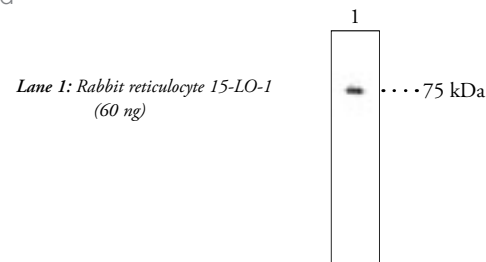
1 ea

**15-Lipoxygenase-1 (rabbit) Polyclonal Antiserum** 160704

50 µl lyophilized antiserum **Stability:** ≥3 years at -20°C

Summary: Antigen: rabbit reticulocyte 15-LO • Host: sheep • Cross-reactivity: (+) rabbit, human, and murine 15-LO-1 and 12-LO (porcine leukocyte) • Application: WB; other applications not tested • 15-LO-1 catalyzes the formation of 15(S)-HETE and 13(S)-HODE from arachidonic acid and linoleic acid, respectively.

1 ea

**Lovastatin** 10010338

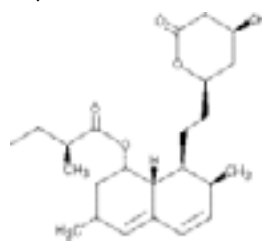
[75330-75-5]

MF: C₂₄H₃₆O₅ **FW:** 404.5 **Purity:** ≥98%

A crystalline solid **Stability:** ≥2 years at -20°C

Summary: Lovastatin is a HMG-CoA reductase inhibitor that was initially isolated from *Aspergillus terreus*. It is a competitive inhibitor of HMG-CoA reductase with a K_i value of 0.6 nM for the open ring, hydroxyacid form of the molecule.

5 mg
10 mg
50 mg
100 mg



Also Available: Lovastatin (sodium salt) (10010339)

1 mg
5 mg
10 mg
50 mg

LXRb Transcription Factor Assay Kit 10011119

Liver X receptor

LXRs are ligand-activated transcription factors that are primarily activated by oxysterols and cholesterol metabolites. As such, LXRs play an important role in the regulation of cholesterol, lipid, and carbohydrate metabolism. There are two known isoforms of LXR: LXRA and LXRb. LXRb is ubiquitously expressed in all tissues while LXRA is primarily expressed in the liver, adipose tissue, small intestine, and macrophages. LXRs are currently being examined as potential therapeutic targets in the treatment of diabetes, cardiovascular disease, Alzheimers disease, obesity, and atherosclerosis. Cayman Chemical's LXRb Transcription Factor Assay is a sensitive colorimetric method for detecting specific transcription factor binding activity from nuclear extracts and whole cell lysates in a 96-well format.

96 wells

MEDICA 16 90290

[87272-20-6]

MF: C₂₀H₃₈O₄ **FW:** 342.5 **Purity:** ≥98%

A crystalline solid **Stability:** ≥1 year at -20°C

Summary: MEDICA 16 is a b,b'-dimethyl hexadecanedioic acid that exhibits hypolipidemic and antidiabetogenic effects in the rat. In animals that were fed a diet which was 0.25% MEDICA 16 by weight, the hypolipidemic effect consisted of a 70-80% decrease in plasma chylomicrons and VLDL-triacylglycerols as well as a 40-60% decrease in plasma VLDL-cholesterol.

1 mg
5 mg
10 mg
50 mg



3,3,14,14-tetramethylhexadecanedioic acid

NEW Mevastatin 10010340

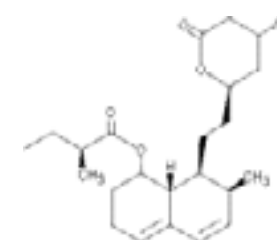
[73573-88-3]

MF: C₂₃H₃₄O₅ **FW:** 390.5 **Purity:** ≥98%

A crystalline solid **Stability:** ≥2 years at -20°C

Summary: Mevastatin is a HMG-CoA reductase inhibitor that was initially isolated from the mold *Pythium ultimum*. It inhibits HMG-CoA reductase in a reversible and competitive manner with a K_i value of 1 nM for the open ring acid form of the molecule. During a 24 week study period, a dose of 30 mg/day mevastatin reduced plasma LDL-cholesterol approximately 29% in patients with familial hypercholesterolemia.

5 mg
10 mg
25 mg
50 mg



Also Available: Mevastatin (sodium salt) (10010341)

5 mg
10 mg
25 mg
50 mg

NEW Myriocin 63150

[35891-70-4] ISP-1, Thermozymocidin

MF: C₂₁H₃₉NO₆ **FW:** 401.5 **Purity:** ≥98%

A crystalline solid **Stability:** ≥2 years at -20°C

Summary: Myriocin is a potent inhibitor of serine palmitoyltransferase (K_i = 0.28 nM), the enzyme that catalyzes the first step of sphingolipid biosynthesis. Myriocin potently suppresses the development of atherosclerosis in apolipoprotein E (ApoE) gene knockout (ApoE^{-/-}) mice fed a high-fat diet.

1 mg
5 mg
10 mg
25 mg



2-amino-3,4-dihydroxy-2-(hydroxymethyl)-14-oxo-6-eicosenoic acid

Nitrotyrosine Monoclonal Antibody 189542

Purified IgG_{2b} in PBS, pH 7.2, containing 50% glycerol, 0.5 mg/ml BSA, and 0.02% sodium azide **Stability:** ≥2 years at -20°C

Summary: Antigen: peroxynitrite-treated KLH • Isotype: IgG_{2b} • Host: mouse • Applications: WB, IHC, IP, and EIA

50 µg
200 µg

Lane 1: Nitrotyrosine BSA (0.05 µg)
Lane 2: Murine macrophage cell lysate treated with 3.1 mM peroxynitrite (0.05 µg)



Also Available: Nitrotyrosine Polyclonal Antibody (189540) 1 ea
Nitrotyrosine Monoclonal Antibody-Biotinylated (10006966) 100 µg
Nitrotyrosine (Peptide) Polyclonal Antibody (10006778) 1 ea

eNOS (bovine recombinant) 60880

ecNOS, NOS III

MF: Homodimer **FW:** 135 kDa/subunit **Purity:** cell lysate 100,000 x g supernatant A solution in 50 mM HEPES, pH 7.4, containing 10% glycerol, 5 mM CHAPS, and 100 µM DTT **Stability:** ≥6 months at -80°C

Summary: EC 1.14.13.39 • Recombinant enzyme isolated from a Baculovirus overexpression system in Sf9 cells • One unit of enzyme produces 1 nmol of NO per minute at 37°C in 50 mM HEPES, pH 7.4, containing 50 µM arginine, 1 mM CaCl₂, 5 µM oxyhemoglobin, 20 µg/ml calmodulin, 0.1 mM NADPH, 12 µM tetrahydrobiopterin, and 170 µM DTT.

10 units

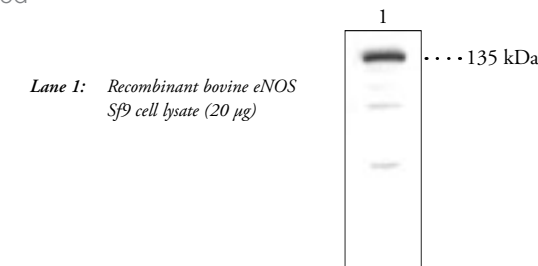
eNOS Polyclonal Antiserum 160880

ecNOS, NOS III

100 µl lyophilized antiserum **Stability:** ≥2 years at -20°C

Summary: Antigen: human eNOS amino acids 1186-1203 • Host: rabbit • Cross-reactivity: (+) bovine and human eNOS; (-) iNOS and nNOS • Applications: WB and IP; other applications not tested • eNOS catalyzes the formation of NO from L-arginine in many cell types including vascular endothelium, bronchiolar epithelium, cardiac myocytes, spleen, and kidney.

1 ea



Also Available: eNOS Blocking Peptide (360881) 200 µg

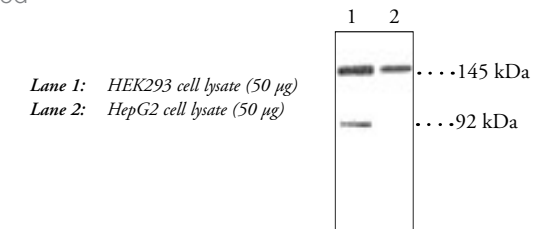
NEW NPC1L1 Polyclonal Antibody 10005385

Niemann-Pick C1 Like 1

Peptide affinity-purified IgG **Stability:** ≥1 year at -20°C

Summary: Antigen: human NPC1L1 amino acids 596-610 • Host: rabbit • Cross-reactivity: (+) human NPC1L1; other species not tested • Applications: WB and ICC; other applications not tested • NPC1L1 is a transmembrane protein expressed in the brush border membrane of small intestine enterocytes. It is required for intestinal uptake of both cholesterol and phytosterols.

1 ea



Also Available: NPC1L1 Blocking Peptide (10006985) 200 µg

Oleic Acid-2,6-diisopropylanilide

10006782

[140112-65-8]

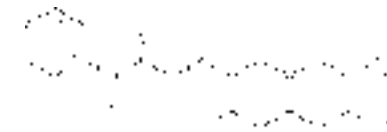
MF: C₃₀H₅₁NO **FW:** 441.7 **Purity:** ≥98%A solution in methyl acetate **Stability:** ≥1 year at -20°C**Summary:** Oleic acid-2,6-diisopropylanilide is an inhibitor of ACAT with an IC₅₀ value of 7 nM. When co-administered to rabbits or rats fed a high fat, high cholesterol diet, oleic acid-2,6-diisopropylanilide decreased LDL and elevated HDL levels when administered at 0.05%.5 mg
10 mg
50 mg
100 mg

N-[2,6-bis(1-methylethyl)phenyl]-9Z-octadecenamide

Oleyl Anilide

10006529

[5429-85-6] OA, Oleic Acid Anilide

MF: C₂₅H₃₉NO **FW:** 357.6 **Purity:** ≥95%A crystalline solid **Stability:** ≥2 years at -20°C**Summary:** OA is a weak inhibitor of ACAT with an IC₅₀ value of 26 μM. OA and the related glyceride dioleoyl phenylamino propane 1,2-diol have been linked to a syndrome of eosinophilia, excessive T-cell activation, and elevated IL-4, soluble IL-2R, and IL-5. The clinical consequences are an acute pulmonary inflammatory reaction followed by chronic neuropathy, myalgia, and autoimmune connective tissue disease, generally referred to as toxic oil syndrome (TOS). Aniline-denatured cooking oil is a source of OA associated with TOS.5 mg
10 mg
50 mg
100 mg

N-phenyl-9Z-octadecenamide

Oxidized Lipid HPLC Mixture

34004

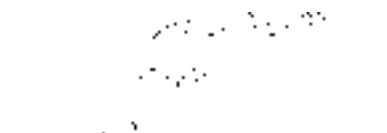
Purity: ≥98% for each compoundA solution in ethanol **Stability:** ≥1 year at -20°C**Summary:** Cholesterol and LDL particles accumulate and become oxidized in the fatty deposits of atherosclerotic plaques. Contained within these lipid deposits are the racemic monohydroxylation products of both linoleic acid and arachidonic acid. This HPLC mixture contains the free acid (non-esterified) forms of racemic 15-HETE, 9-HODE, and 13-HODE. 15-HETE is one of 5 different regioisomers produced by the random oxygenation of arachidonic acid. The 9- and 13- HODEs are the two different monohydroxylated regioisomers of linoleic acid produced during random free radical oxidation. In this mixture, the HODE compounds are provided both in their free acid form, and also esterified to cholesterol.

1 ea

Ozagrel

70515

[82571-53-7] OKY-046

MF: C₁₃H₁₂N₂O₂ **FW:** 228.3 **Purity:** ≥98%A crystalline solid **Stability:** ≥1 year at -20°C**Summary:** Ozagrel is a 1-alkyl imidazole derivative that acts as a selective inhibitor of TXA synthase with an IC₅₀ value of 11 nM. The beneficial effects of TXA synthase inhibition by ozagrel include improved motor coordination after experimentally-induced stroke and antihypertensive effects in spontaneously hypertensive rats.5 mg
10 mg
50 mg
100 mg

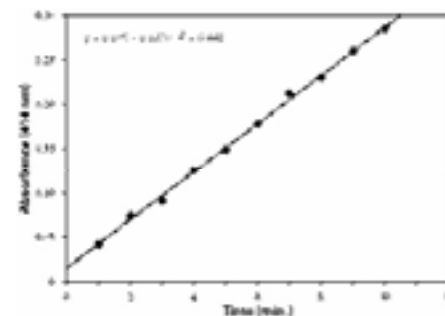
3-[4-(1H-imidazol-1-ylmethyl)phenyl]-2E-propenoic acid

PAF Acetylhydrolase Assay Kit

760901

Lp-PLA₂, PAF-AH**Stability:** ≥1 year at -20°C**Summary:** PAF is a biologically active phospholipid synthesized by a variety of stimulated cells. PAF is converted to the biologically inactive lyso-PAF by the enzyme PAF-AH. PAF-AHs are located intra- and extra-cellularly (e.g., cytosolic and plasma). Recently, plasma PAF-AH has been linked to atherosclerosis and may be a positive risk factor for coronary heart disease in humans. Cayman's PAF-AH assay kit provides an accurate and convenient method for measurement of PAF-AH activity. The assay uses 2-thio PAF which serves as a substrate for PAF-AH. Upon hydrolysis of the acetyl thioester bond at the sn-2 position by PAF-AH, free thiols are detected using 5,5'-dithio-bis-(2-nitrobenzoic acid) (DTNB; Ellman's reagent).

96 wells

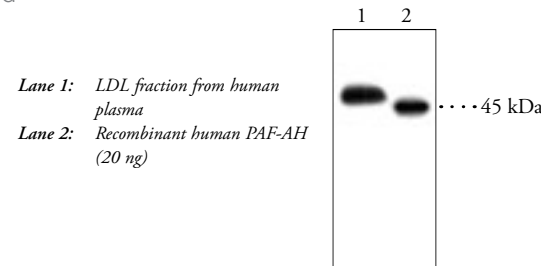


PAF Acetylhydrolase (human) Polyclonal Antibody

160603

LP-PLA₂, PAF-AH100 μl lyophilized antiserum **Stability:** ≥2 years at -20°C**Summary:** Antigen: human PAF-AH C-terminal amino acids 420-441; the peptide sequence used as an antigen is 64% homologous to the corresponding bovine sequence • Host: rabbit • Cross-reactivity: (+) human plasma PAF-AH; (-) murine, guinea pig, canine, and chicken PAF-AH • Application: WB; other applications not tested

1 ea

**Also Available:** PAF Acetylhydrolase (human) Blocking Peptide (360603)

200 μg

NEW PAF Acetylhydrolase (human) Western Ready Control

10010081

Platelet-activating Factor Acetylhydrolase; Lp-PLA₂; PAF-AH**Purity:** 54 kDa**Stability:** ≥6 months at -20°C**Summary:** Source: human recombinant His-tagged protein expressed in *E. coli* • Application: Positive control for WB

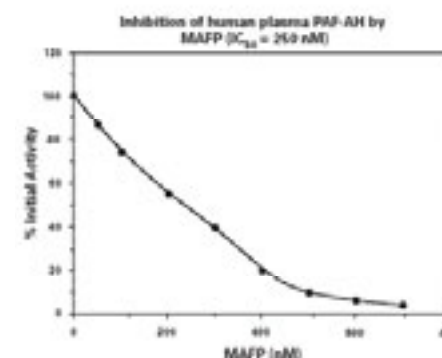
1 ea

NEW PAF Acetylhydrolase Inhibitor Screening Assay Kit

10004380

Lp-PLA₂, PAF-AH**Stability:** ≥6 months at -20°C**Summary:** PAF is a biologically active phospholipid synthesized by a variety of stimulated cells. The biological effects of PAF include activation of platelets, PMNL, monocytes, and macrophages. PAF also increases vascular permeability, decreases cardiac output, induces hypotension, and stimulates uterine contraction. PAF is converted to the biologically inactive lyso-PAF by the enzyme PAF-AH. PAF-AHs are located intra- and extra-cellularly (e.g., cytosolic and plasma). Recently, plasma PAF-AH has been linked to atherosclerosis and may be a positive risk factor for coronary heart disease in humans. Cayman's PAF-AH Inhibitor Screening Assay uses 2-thio PAF as a substrate for PAF-AH. Upon hydrolysis of the acetyl thioester bond at the sn-2 position by PAF-AH, free thiols are detected using 5,5'-dithio-bis-(2-nitrobenzoic acid) (DTNB; Ellman's reagent).

96 wells



PAF Receptor (human) Monoclonal Antibody

160600

100 μg protein-A purified IgG in 200 μl TBS, pH 7.4, containing 0.02% sodium azide **Stability:** ≥6 months at 4°C**Summary:** Antigen: human PAF receptor amino acids 260-269 • Host: mouse • Cross-reactivity: (+) human, bovine, and porcine PAF receptors • Applications: flow cytometry and ICC; does not work for WB • Isotype: IgG_{2a} • Clone designation: 11A4 (clone 21)

1 ea

Also Available: PAF Receptor (human) Blocking Peptide (Monoclonal) (360600)

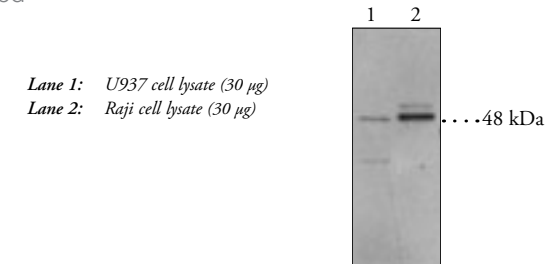
200 μg

PAF Receptor (human) Polyclonal Antibody

160602

Peptide affinity-purified IgG **Stability:** ≥1 year at -20°C**Summary:** Antigen: human PAF receptor amino acids 1-17 • Host: rabbit • Cross-reactivity: (+) human and porcine PAF receptors • Applications: flow cytometry, ICC, and WB

1 ea

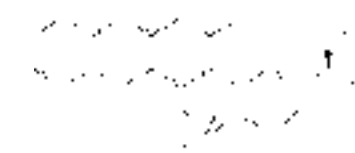
**Also Available:** PAF Receptor (human) Blocking Peptide (Polyclonal) (160604)

200 μg

PAF C-16

60900

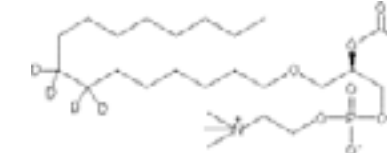
[74389-68-7]

MF: C₂₆H₅₄NO₇P **FW:** 523.7 **Purity:** ≥98%A lyophilized powder **Stability:** ≥1 year at -20°C**Summary:** PAF C-16 is a naturally occurring phospholipid produced upon stimulation through two distinct pathways known as the 'remodeling' and 'de novo' pathways. It is a potent mediator of neutrophil migration and the production of reactive oxygen species and IL-6 in human macrophages. It is a more potent mediator of platelet aggregation than PAF C-18. Pathological processes involving PAF include necrotizing enterocolitis, inflammation, asthma, and allergy.5 mg
10 mg
50 mg
100 mg

1-O-hexadecyl-2-O-acetyl-sn-glycerol-3-phosphorylcholine

PAF C-16-d₄

360900

MF: C₂₆H₅₀D₄NO₇P **FW:** 527.7 **Chemical Purity:** ≥98%**Deuterium Incorporation:** ≤1% d₀A solution in ethanol **Stability:** ≥1 year at -20°C**Summary:** PAF C-18-d₄ contains four deuterium atoms at the 7, 7', 8, and 8' positions. It is intended for use as an internal standard for the quantification of PAF C-16 by GC- or LC-MS.100 μg
500 μg
1 mg
5 mg1-O-hexadecyl-(7,7,8,8-d₄)-2-O-acetyl-sn-glycerol-3-phosphorylcholine

PAF C-18

60910

[74389-69-8]

MF: C₂₈H₅₈NO₇P **FW:** 551.7 **Purity:** ≥97%A solution in ethanol **Stability:** ≥2 years at -20°C**Summary:** PAF C-18 is a naturally occurring phospholipid produced upon stimulation through two distinct pathways known as the 'remodeling' and 'de novo' pathways. It is less potent than PAF C-16 in the induction of platelet aggregation, but equipotent in activation of guinea pig macrophages. PAF C-18 induces the release of PGE₂ and TXB₂ from albumin-elicited guinea pig macrophages and enhances the spreading of plated macrophages. Pathological processes involving PAF include necrotizing enterocolitis, inflammation, asthma, and allergy.5 mg
10 mg
50 mg
100 mg

1-O-octadecyl-2-O-acetyl-sn-glycerol-3-phosphorylcholine

PAz-PC 62924

[117205-52-4] Azelaoyl PC, 1-Palmitoyl-2-azelaoyl PC

MF: C₃₃H₆₄NO₁₀P **FW:** 665.8 **Purity:** ≥98%A solution in ethanol **Stability:** ≥1 year at -20°C**Summary:** PAz-PC is one of the predominant phosphatidylcholine species containing a fragmented, oxidized short-chain fatty acid remnant at the sn-2 position found in oxLDL particles. These low molecular weight species are cytotoxic and pro-atherogenic and may be one of the important structural determinants of oxLDL.1 mg
5 mg
10 mg
50 mg

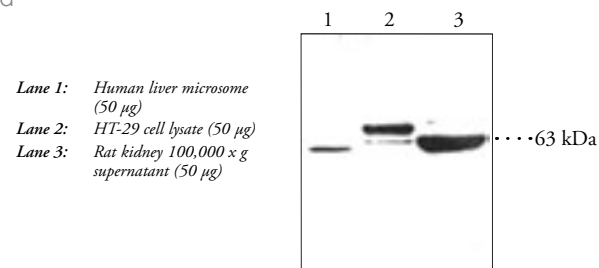
1-O-hexadecanoyl-2-O-(9-carboxyoctanoyl)-sn-glycerol-3-phosphocholine

NEW PCSK9 (human) Polyclonal Antibody 10007185

NARC-1, Proprotein Convertase Subtilisin Kexin 9

Peptide affinity-purified IgG **Stability:** ≥1 year at -20°C**Summary:** Antigen: human PCSK9 amino acids 490-502 • Host: rabbit • Cross-reactivity: (+) human, murine, and rat PCSK9 • Applications: WB and ICC • PCSK9 is a member of the subtilisin serine protease family with an important role in lipoprotein metabolism. Several gain of function mutations in the PCSK9 gene are associated with hypercholesterolemia which is characterized by an increase in LDL cholesterol levels.

1 ea

**Also Available:** PCSK9 (human)

Blocking Peptide (10007186)

200 µg

NEW PCSK9 (murine) Polyclonal Antibody 10008811

NARC-1, Proprotein Convertase Subtilisin Kexin 9

Peptide affinity-purified IgG **Stability:** ≥1 year at -20°C**Summary:** Antigen: mouse PCSK9 amino acids 152-163 • Host: rabbit • Cross-reactivity: (+) human, murine, and rat PCSK9 • Applications: WB and ICC • PCSK9 is a member of the subtilisin serine protease family with an important role in lipoprotein metabolism. Several gain of function mutations in the PCSK9 gene are associated with hypercholesterolemia which is characterized by an increase in LDL cholesterol levels.

1 ea

Also Available: PCSK9 (murine)

Blocking Peptide (10009581)

200 µg

NEW PCSK9 Western Ready Control 10009567

NARC-1, Proprotein Convertase Subtilisin Kexin 9

Purity: 78 kDa tagged; 74 kDa native**Stability:** ≥6 months at -20°C**Summary:** Source: human recombinant C-terminal His-tagged protein expressed in *E. coli* • Application: Positive control for WB

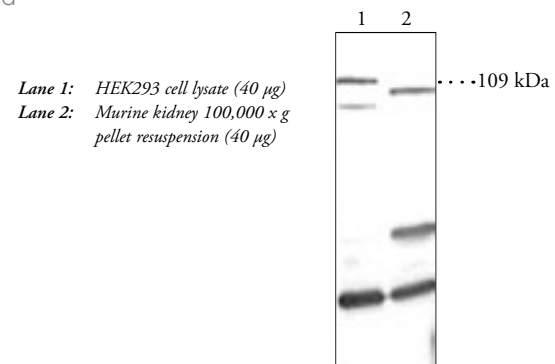
1 ea

NEW PGC-1 Polyclonal Antibody 101707

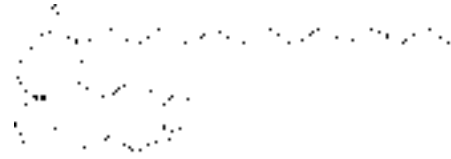
PPARγ Coactivator 1

Peptide affinity-purified IgG **Stability:** ≥1 year at -20°C**Summary:** Antigen: human PGC-1α amino acids 75-90 • Host: rabbit • Cross-reactivity: (+) human, murine, and rat PGC-1α and PGC-1β • Applications: WB and IHC (paraffin-embedded sections) • PPARγ coactivator (PGC-1α) plays a key role in energy metabolism, hepatic gluconeogenesis, and cholesterol homeostasis. PGC-1β is also thought to activate oxidative metabolism in tissues.

1 ea

**Also Available:** PGC-1 Blocking Peptide (301707)

200 µg

PGPC 10044**MF:** C₂₉H₅₆NO₁₀P **FW:** 609.7 **Purity:** ≥98%A solution in ethanol **Stability:** ≥1 year at -20°C**Summary:** PGPC is one of the predominant phosphatidylcholine species containing a fragmented, oxidized short-chain fatty acid remnant at the sn-2 position found in oxLDL particles. PGPC treatment of vascular endothelial cells induces the expression of both E-selectin and VCAM-1, and increases endothelial cell binding by both neutrophils and monocytes.1 mg
5 mg
10 mg
50 mg

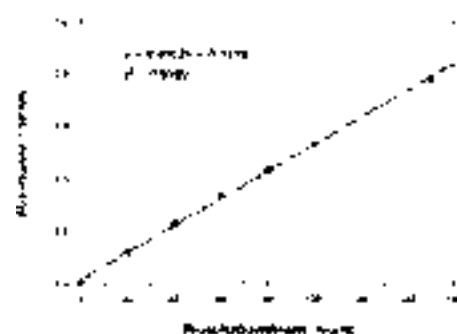
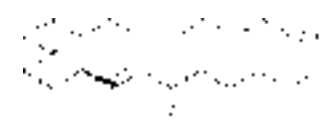
1-palmitoyl-2-(4-carboxybutanoyl)-sn-glycerol-3-phosphatidylcholine

NEW Phosphatidylcholine Assay Kit 10009926

PC

Stability: ≥6 months at -20°C**Summary:** Cayman's PC Assay Kit provides a specific, sensitive, and convenient method for quantifying PC in plasma or serum. In this assay, PC-specific PLD is first used to hydrolyze PC to choline and phosphatidic acid. The newly formed choline is then used to generate hydrogen peroxide in a reaction catalyzed by choline oxidase. Finally, with peroxidase as a catalyst, hydrogen peroxide reacts with DAOS and 4-aminoantipyrine to generate a blue dye with an optimal absorption at 595 nm.

96 wells

**Pinane Thromboxane A₂** 19020[71111-01-8] PTA₂**MF:** C₂₄H₄₀O₃ **FW:** 376.6 **Purity:** ≥98%*A solution in ethanol **Stability:** ≥1 year at -20°C**Summary:** PTA₂ is a stable analog of TXA₂ that acts as a TP receptor antagonist and an inhibitor of TX synthase. PTA₂ inhibits U-46619-induced cat coronary artery constriction (ID₅₀ = 0.1 µM), U-46619-induced aggregation of human platelets (IC₅₀ = 2 µM), and rabbit platelet TX synthase (ID₅₀ = 50 µM). PTA₂ does not affect PGI synthase up to a concentration of 100 µM.500 µg
1 mg
5 mg
10 mg

9α,11α-(dimethyl)methylene-15S-hydroxy-11a-deoxy-11a-methylene-thromba-5Z,13E-dien-1-oic acid

POV-PC 10031

2-(5-oxovaleryl) Phosphatidylcholine

MF: C₂₉H₅₆NO₉P **FW:** 593.7 **Purity:** ≥98%A solution in ethanol **Stability:** ≥1 year at -20°C**Summary:** POV-PC is one of the oxLDL species derived from 2-arachidonoyl or eicosapentanoyl phospholipids. POV-PC confers CD36 scavenger receptor binding affinity more potently than any hydroperoxy PC species, and may be one of the more important structural determinants of oxLDL. Treatment of cultured endothelial cells with POV-PC stimulates monocyte binding, stimulates intracellular cAMP production, and strongly inhibits the LPS-induced binding of neutrophils.1 mg
5 mg
10 mg
50 mg

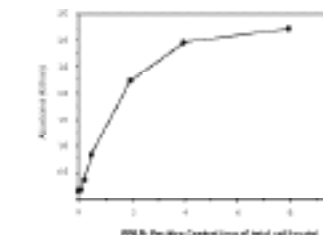
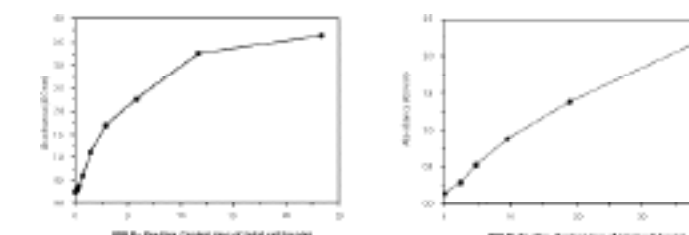
1-palmitoyl-2-(5-oxovaleryl)-sn-glycerol-3-phosphatidylcholine

PPAR Transcription Factor Assay Kits

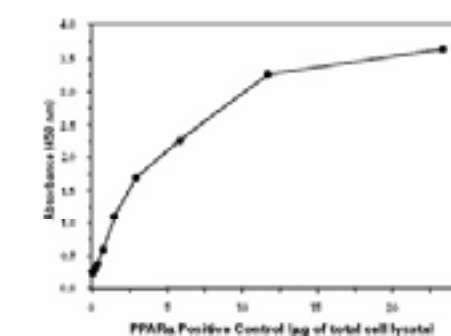
PPARs are ligand-activated transcription factors belonging to the large superfamily of nuclear receptors. PPARα primarily activates genes encoding proteins involved in fatty acid oxidation, while PPARγ primarily activates genes directly involved in lipogenic pathway and insulin signalling. Members of the PPAR family are important direct targets of many antidiabetic and hypolipidemic drugs. Cayman's PPAR Transcription Factor Assays are a non-radioactive, sensitive method for detecting specific transcription factor DNA binding activity in nuclear extracts and whole cell lysates. A 96-well ELISA replaces the cumbersome radioactive electrophoretic mobility shift assay (EMSA). A specific double stranded DNA (dsDNA) sequence containing the PPAR response element is immobilized onto the bottom of wells of a 96-well plate. PPARs contained in a nuclear extract, bind specifically to the PPAR response element. PPARα, δ, or γ are detected by addition of specific primary antibodies directed against the individual PPARs. A secondary antibody conjugated to HRP is added to provide a sensitive colorimetric readout at 450 nm.

NEW PPARα, δ, γ Complete Transcription Factor Assay Kit 10008878**Stability:** ≥6 months at -20°C**Summary:** This kit contains individual primary antibodies for PPARα, δ, and γ to follow detection of each receptor in separate wells of the plate.

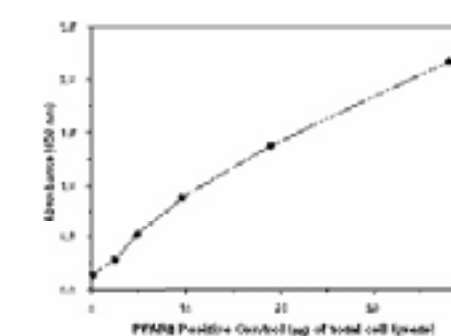
96 wells

**PPARα Transcription Factor Assay Kit** 10006915**Stability:** ≥6 months at -20°C

96 wells

**PPARδ Transcription Factor Assay Kit** 10006914**Stability:** ≥6 months at -20°C

96 wells



*All 5-cis 2-series PGs (those containing a 5,6-double bond) will contain a small amount of the 5-trans isomer. This isomer is generally undetectable using normal phase silica columns and plates, but may be resolved using RP-HPLC. The purity for all such 2-series PGs excludes the 1-3% trans isomer which will generally be present.

Tom Brock, Ph.D.

Inflammation in Atherosclerosis Problems with Platelets & Prostaglandins

Platelets are known for their role in clot formation. However, they also contribute to the formation and extension of atherosclerotic plaques, promote inflammation in vessels, and impact vascular tone. A portion of these effects are through their production of the prostanoid thromboxane (TXA₂) and its interplay with endothelium-derived prostacyclin (prostaglandin I₂, PGI₂). These signalling pathways and their interactions are linked to the benefits of low dose aspirin, as well as the problems of selective cyclooxygenase-2 (COX-2) inhibitors, in the cardiovascular system.¹

Platelets are anucleated cells that can respond to a variety of agonists with the typical cell signalling pathways that do not require transcriptional events. For example, they respond to PGI₂ in a receptor-mediated fashion that results in elevation of intracellular cAMP, which serves to suppress platelet function. On the other hand, platelets respond to thrombin and TXA₂, again through specific receptors, with a transient rise in intracellular calcium. Calcium activates cytosolic PLA₂ (cPLA₂) in platelets, leading to the release of arachidonic acid, which is then metabolized by cyclooxygenase-1 (COX-1) to produce TXA₂. TXA₂ is rapidly secreted and can, in turn, activate neighboring platelets, amplifying TXA₂ production and platelet activation. These activated platelets release a variety of pro-inflammatory mediators that affect the endothelium, smooth muscle and monocytes/macrophages in vessels. For example, activated platelets release IL-1β and CD-40L, which stimulate endothelial cells to synthesize cytokines (e.g., IL-6, IL-8, MCP-1) and tissue factor, produce reactive oxygen species, and increase adhesion of leukocytes. Also, platelets will release P-selectin, which causes monocytes to secrete chemokines and growth factors, as well as increase COX-2 expression, leading to the production of PGI₂ and prostaglandin E₂ (PGE₂). In addition, activated platelets will produce platelet factor 4 to stimulate monocyte differentiation into macrophages and matrix metalloproteases to promote degradation of matrix proteins in the vessel wall. TXA₂, then, is an important early messenger in atherosclerosis; the inhibition of TXA₂ synthesis, as well as the antagonism or knockout of the TXA₂ receptor

(TP), delays atherogenesis in murine models.²⁻⁴ Aspirin acetylates and permanently inhibits both COX-1 and COX-2. Low-dose aspirin effectively inhibits COX in circulating platelets and, as platelets lack nuclei, they are unable to transcribe new COX message. Low-dose aspirin, then, is effective therapy for delaying atherogenesis because it inhibits the generation of TXA₂ by platelets.

Opposing the effects of TXA₂ is another COX product, PGI₂. This prostanoid is manufactured by endothelial cells, monocytes and macrophages, and, in some cases, smooth muscle cells. While the constitutively expressed COX-1 enzyme can produce some PGI₂, induction of COX-2 expression selectively increases the synthesis of PGI₂ and PGE₂ (coupling to PGI synthase and mPGES-1, respectively) over other products, including TXA₂. PGI₂ potently relaxes vascular smooth muscle, is a vasodilator, an inhibitor of platelet aggregation, and PGI₂ analogs are used in the treatment of primary pulmonary hypertension. Through the IP receptor, PGI₂ increases cytoplasmic cAMP, which in turn suppresses activation of endothelial cells and leukocytes. At a more basic level, PGI₂, by elevating cAMP, inhibits cPLA₂ activity and thus prevents the release of arachidonic acid, the precursor for TXA₂ (and other COX products). So, PGI₂ actively reduces vascular tone, counters pro-inflammatory signals by suppressing cell activation, and inhibits the synthesis of pro-inflammatory mediators, like TXA₂. Importantly, selective inhibition of COX-2 may also selectively reduce the synthesis of PGI₂, as compared to TX.⁵ As a result, selective COX-2 inhibitors will diminish the protection provided by PGI₂ against cardiovascular inflammation.

References

1. Davi, G. and Patrono, C. *New England Journal of Medicine* **357**(24), 2482-2494 (2007).
2. Praticò, D., Cyrus, T., Li, H., et al. *Blood* **96**, 3823-3826 (2000).
3. Kobayashi, T., Tahara, Y., Matsumoto, M., et al. *J. Clin. Invest.* **114**(6), 784-794 (2004).
4. Egan, K.M., Wang, M., Lucitt, M.B., et al. *Circulation* **111**, 334-342 (2005).
5. Cohen, J.C., Pertsemlidis, A., Fahmi, S., et al. *Proc. Natl. Acad. Sci. USA* **103**(6), 1810-1815 (2006).

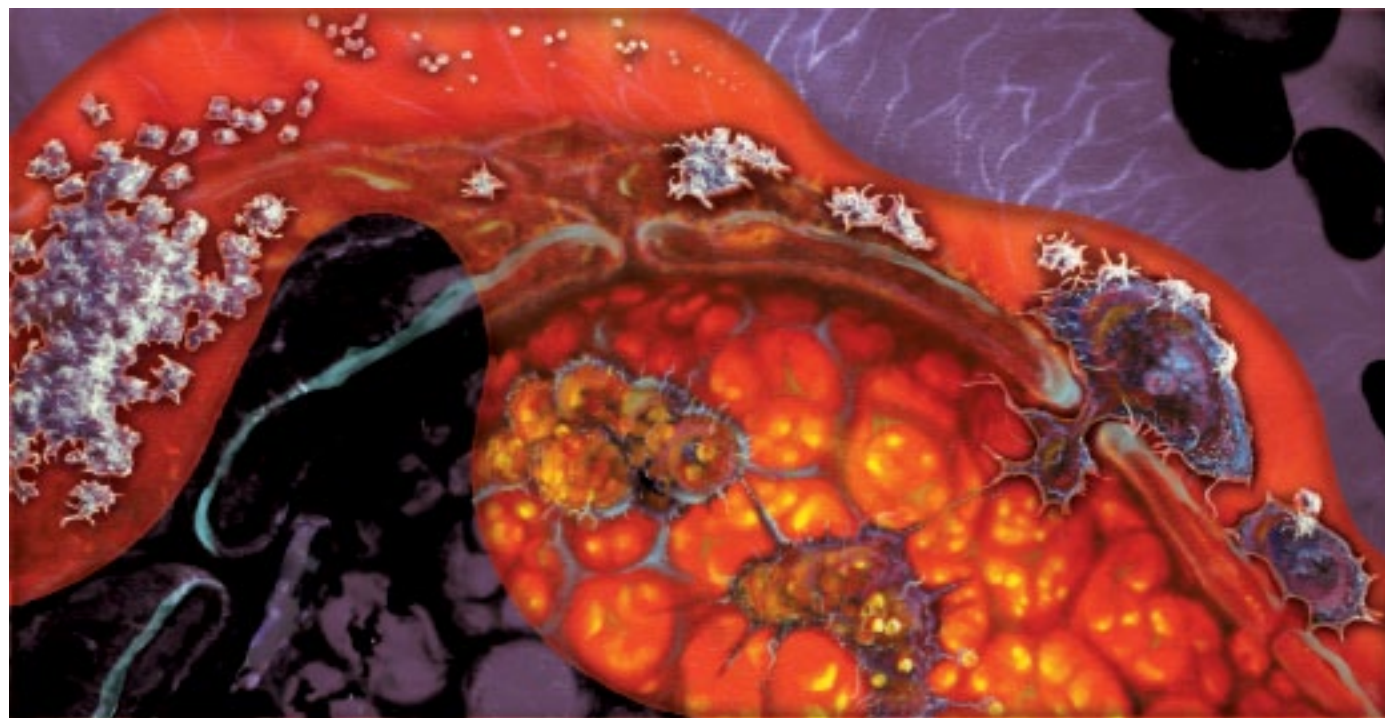


Figure 1. Circulating platelets may be activated by TXA₂ from stimulated monocytes/macrophages. The activated platelets, in turn, release mediators (additional TXA₂, IL-1β, CD-40L, tissue factor, P-selectin, RANTES, MMPs, and platelet factor 4) that drive inflammatory and clotting cascades.

Tom Brock, Ph.D.

Drug Discovery Approaches

A common initial approach to addressing early signs of atherosclerosis involves diet modification, increased exercise, and smoking cessation. If these changes fail, pharmaceutical intervention may be necessary. Much of the current research focuses on developing drugs that alter lipid metabolism. Initial focus centered on lowering cholesterol levels, but with the recognition of “good” and “bad” cholesterol, the emphasis moved to include lowering the “bad” (LDL) cholesterol specifically. While this approach has proven to be quite effective in preventing disease progression, there remains room for improvement, both in preventing the development of advanced symptoms and in driving disease regression. Current efforts focus on increasing “good” (HDL) cholesterol levels, given its ability to actually remove cholesterol from vessels through reverse cholesterol transport. Treatments that increase HDL levels, or alter other aspects of lipid metabolism, are commonly tested in combination with LDL lowering drugs to determine if they provide an added benefit.

Lowering LDL Levels

The statins have been widely prescribed and are effective for the prevention and treatment of atherosclerosis.¹ The statins are inhibitors of HMG-CoA reductase inhibitors, blocking the rate-limiting step in the mevalonate pathway of cholesterol synthesis. Inhibition of this enzyme in the liver stimulates LDL receptor activity, increasing clearance of LDL from the bloodstream and further decreasing blood cholesterol levels. However, the effects of lowering plasma cholesterol are complex, in part because of the multiple genes that are upregulated in response to lowering cholesterol (see Article on SREBP-2, page 4). The statins as a group have relatively few undesirable side-effects and multiple trials have shown strong effects in reducing atherosclerotic disease events. Not all statins are the same, and there may be room to develop a better statin. An effective way to further lower LDL levels involves the use of niacin in combination with statins. Niacin (nicotinic acid, Vitamin B₃) alone lowers LDL levels, but less than statins. However, the combination of niacin and statins is superior to statins alone in decreasing both LDL and triglyceride levels and increasing HDL levels.

Another novel therapy involves blocking the synthesis of ApoB, which is necessary for forming LDL. Such treatments, which might use antisense RNA or siRNA, are currently being investigated.² The serum proprotein convertase subtilisin kexin type 9 (PCSK9) increases the turnover of the LDL receptor, increasing circulating plasma LDL cholesterol levels. Curiously, the expression of both PCSK9 and the LDL receptor are controlled by cholesterol levels through SREBP-2. As PCSK9 levels correlate well with LDL levels,³ lowering PCSK9 expression or protein should be an effective way to reduce LDL.

Increasing HDL Levels

Numerous approaches to increasing HDL levels are currently in development. Niacin, mentioned above, remains the most effective. However, niacin has a ‘nuisance’ side effect, causing noticeable flushing (blushing of the face and neck), which also enhances removal of the niacin. This can be reduced by using extended release niacin. Another approach is to add an inhibitor of the agent that drives the flushing, prostaglandin D₂ (PGD₂). These could include drugs that block PGD₂ synthesis or antagonize the PGD₂ receptor.⁴

Fibrates have been shown to increase HDL, although less effectively than niacin. Fibrates activate peroxisomal proliferator-activated factor α (PPARα), a transcription factor involved in carbohydrate and fat metabolism. *Via* PPARα activation, fibrates increase the synthesis of ApoA1, which increases HDL formation. PPARα activation also increases synthesis of lipoprotein lipase, which hydrolyzes triglycerides in VLDL, IDL, and LDL. Because they modulate inflammatory pathways as well as lipid pathways, the use of PPAR activators represents a promising tool in the treatment of cardiovascular diseases.⁵

There is also interest in therapy with an ApoA1 variant called ApoA1_{Milano}. This naturally-occurring variant, identified by Cesare Sirtori in Milan, has been shown to drive atherosclerosis regression in experiments involving either protein infusion therapy or gene therapy.⁶ In spite of these successes, use has been restricted due to the mode of therapy.

Another promising tactic involves inhibiting the activity of CETP, which transfers cholesteryl esters from HDL to LDL. In theory, this should maximize reverse cholesterol transport. Early trials of CETP inhibitors, either alone or in combination with statins, have shown significant deleterious side effects.⁷ Side effects of the CETP inhibitor torcetrapib have included increased aldosterone, elevated blood pressure, and increased deaths, both cardiovascular and non-cardiovascular related. Importantly, increased aldosterone levels and blood pressure were not observed in rats given anacetrapib, another CETP inhibitor, suggesting that the problems with torcetrapib were unique to that compound and not characteristic of the drug class.

Finally, inhibition of cannabinoid receptors has proven useful. These receptors, which are known to be expressed in the central nervous system as well as in the periphery and regulate the central neural circuits for food uptake and peripheral metabolic circuits, were initially targeted as a way to control body weight. However, tests with rimonabant, a first generation antagonist of the cannabinoid receptor CB₁, have shown that it increases HDL level, decreases serum triglycerides, and improves insulin sensitivity. Additional cannabinoid receptor blockers have been developed and are in testing.

References

1. Davidson, M.H. *American Journal of Managed Care* **13**, S260-S269 (2007).
2. Ito, M.K. *Ann. Pharmacother.* **41**, 1669-1678 (2007).
3. Alborn, W.E., Cao, G., Caresky, H.E., et al. *Clin. Chem.* **53**(10), 1814-1819 (2007).
4. Cheng, K., Wu, T.-J., Wu, K.K., et al. *Proc. Natl. Acad. Sci. USA* **103**(17), 6682-6687 (2006).
5. Bouhrel, M.A., Staels, B., and Chinetti-Gbaguidi, G. *J. Int. Medicine* **263**, 28-42 (2008).
6. Nicholls, S.J., Tuzcu, E.M., Sipahi, I., et al. *J. Am. Coll. Cardiol.* **47**(5), 992-997 (2006).
7. Kastelein, J.J.P. *Am. J. Cardiol.* **100**, 47N-52N (2007).

Cholesterol Synthesis Inhibitors

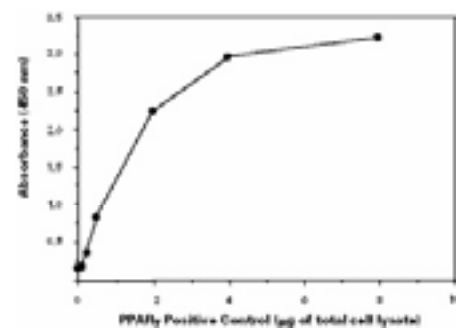
Cat. No. (salt form)	Name	Target Enzyme	K _i
10010334 (10010337)	Fluvastatin	HMG-CoA Reductase	0.3 nM
10010338 (10010339)	Lovastatin	HMG-CoA Reductase	0.6 nM
10010340 (10010341)	Mevastatin	HMG-CoA Reductase	1 nM
10010342 (10010343)	Pravastatin	HMG-CoA Reductase	2.3 nM
10006415	Ro 48-8071	Oxidosqualene Cyclase	1.5-6.5 nM
10010344 (10010345)	Simvastatin	HMG-CoA Reductase	0.12 nM

PPAR γ Transcription Factor Assay Kit

10006855

Stability: ≥ 6 months at -20°C

96 wells

NEW PPAR α LBD (human recombinant)

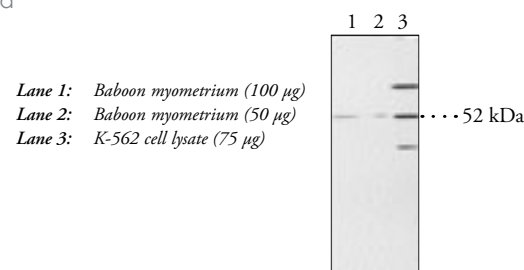
10009088

PPAR α Ligand Binding Domain**Purity:** $\geq 90\%$ A solution in 50 mM sodium phosphate, pH 7.2, containing 20% glycerol, 100 mM sodium chloride, and 1 mM DTT **Stability:** ≥ 6 months at -80°C **Summary:** Source: recombinant His-tagged protein purified from *E. coli* • M_r : 34 kDa25 μg
50 μg
100 μg PPAR α Polyclonal Antibody

101710

Peptide affinity-purified IgG **Stability:** ≥ 1 year at 4°C **Summary:** Antigen: human, murine, and rat PPAR α amino acids 22-36 • Host: rabbit • Cross-reactivity: (+) human, murine, rat, ovine, and porcine PPAR α ; (-) PPAR γ • Application: WB; other applications not tested • PPAR α is a ligand-activated transcription factor involved in the regulation of lipid homeostasis.

1 ea

**Also Available:** PPAR α Blocking Peptide (301710) 200 μg NEW PPAR δ (human recombinant)

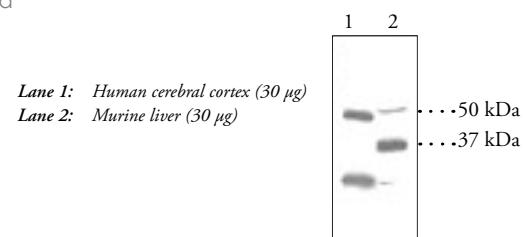
10007451

FAAR, NUC1, Nuclear Hormone Receptor 1, PPAR β **Purity:** $\geq 95\%$ A solution in 50 mM sodium phosphate, pH 7.2, containing 20% glycerol, 150 mM sodium chloride, and 1 mM DTT **Stability:** ≥ 6 months at -80°C **Summary:** Source: recombinant protein isolated from baculovirus overexpression system in Sf21 cells • M_r : 54 kDa10 μg
25 μg
50 μg NEW PPAR δ Polyclonal Antibody

101720

FAAR, NUC1, Nuclear Hormone Receptor 1, PPAR β Peptide affinity-purified IgG **Stability:** ≥ 1 year at -20°C **Summary:** Antigen: human PPAR δ amino acids 39-54 • Host: rabbit • Cross-reactivity: (+) human, murine, ovine, porcine, and rat PPAR δ ; other species not tested • Applications: WB, IHC, and ICC; other applications not tested

1 ea

**Also Available:** PPAR δ Blocking Peptide (10006247) 200 μg NEW PPAR δ Western Ready Control

10009568

FAAR, NUC1, Nuclear Hormone Receptor 1, PPAR β **Purity:** 54 kDa tagged; 51 kDa native**Stability:** ≥ 6 months at -20°C **Summary:** Source: human recombinant N-terminal His-tagged protein expressed in Sf21 cells • Application: Positive control for WB

1 ea

NEW PPAR γ FL (human recombinant from *E. coli*)

61700

PPAR γ Full Length**Purity:** $\geq 90\%$ by SDS-PAGEA solution in 20 mM Tris HCl, pH 8.0, containing 250 mM KCl, 20% glycerol, 5 mM DTT, and 0.5 mM EDTA **Stability:** ≥ 6 months at -80°C **Summary:** Source: human recombinant N-terminal His-tagged protein expressed in *E. coli* • M_r : 60 kDa5 μg
10 μg
25 μg
50 μg NEW PPAR γ FL (human recombinant from Sf21 cells)

10009987

PPAR γ Full Length**Purity:** $\geq 80\%$ by SDS-PAGEA solution in 50 mM sodium phosphate, pH 7.2, containing 100 mM NaCl, 20% glycerol, 1 mM DTT, and 20% mM glycerol **Stability:** ≥ 6 months at -80°C **Summary:** Source: human recombinant N-terminal His-tagged protein expressed in Sf21 cells • M_r : ~60 kDa5 μg
10 μg
25 μg
50 μg NEW PPAR γ LBD (human recombinant)

10007941

PPAR γ Ligand Binding Domain**Purity:** $\geq 90\%$ A solution in 50 mM sodium phosphate, pH 7.2, containing 20% glycerol, 150 mM sodium chloride, and 1 mM DTT **Stability:** ≥ 6 months at -80°C **Summary:** Source: recombinant N-terminal His-tagged protein expressed in *E. coli* • M_r : 34 kDa25 μg
50 μg
100 μg PPAR γ -PAK

71000

Purity: $\geq 98\%$ **Stability:** ≥ 1 year at -20°C **Summary:** The Cayman PPAR γ -PAK contains a combination of frequently used ligands for PPAR γ . Each kit contains ciglitazone, the first characterized member of the thiazolidinedione (TZD) class that binds to the PPAR γ ligand-binding domain with an EC_{50} value of 3.0 μM . Rosiglitazone, a key reference TZD also called BRL 49653, is another PPAR γ agonist provided. Also included is troglitazone (ResulinTM), another TZD; it was withdrawn from human therapeutic use due to hepatotoxicity. Also in this assortment is 15-deoxy-D^{12,14}-PGJ₂, a potent PPAR γ ligand derived from PGD₂. The actions of all of these compounds can be antagonized by the selective PPAR γ antagonist, GW 9662, which is also in the kit.

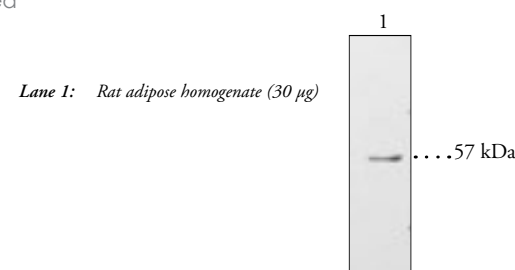
1 ea

PPAR γ Polyclonal Antibody

101700

Peroxisome Proliferator-activated Receptor γ -PAKPeptide affinity-purified IgG **Stability:** ≥ 1 year at -20°C **Summary:** Antigen: human PPAR $\gamma 1$ amino acids 82-101; amino acids 110-129 of PPAR $\gamma 2$ • Host: rabbit • Cross-reactivity: (+) human and murine PPAR $\gamma 1$ and PPAR $\gamma 2$ • Application: WB; other applications not tested • PPAR γ is a ligand-activated transcription factor involved in the regulation of lipid homeostasis and may function as a master regulator of adipogenesis.

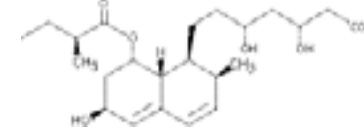
1 ea

**Also Available:** PPAR γ Blocking Peptide (301700) 200 μg

NEW Pravastatin

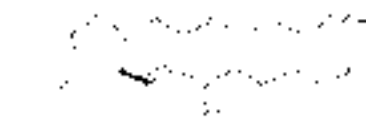
10010342

[81093-37-4]

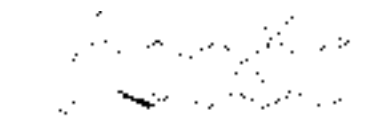
MF: C₂₃H₃₆O₇ **FW:** 424.5 **Purity:** $\geq 98\%$ A crystalline solid **Stability:** ≥ 2 years at -20°C **Summary:** Pravastatin is a HMG-CoA reductase inhibitor that is a ring hydroxylated metabolite of mevastatin. It is a competitive inhibitor of HMG-CoA reductase with a K_i value of 2.3 nM for the active, open ring form of the molecule.10 mg
25 mg
50 mg
100 mg**Also Available:** Pravastatin (sodium salt) (10010343)10 mg
25 mg
50 mg
100 mgProstaglandin E₁

13010

[745-65-3] Alprostadil

MF: C₂₀H₃₄O₅ **FW:** 354.5 **Purity:** $\geq 98\%$ Light yellow to white needles **Stability:** ≥ 2 years at -20°C **Summary:** PGE₁ is the theoretical COX metabolite of dihomo- γ -linolenic acid (DGLA), but it is virtually undetectable in the plasma of normal humans or other animals. Its pharmacology includes vasodilation, hypotension, and anti-platelet activities. The IC_{50} value of PGE₁ for the inhibition of ADP-induced human platelet aggregation is 40 nM. The vasorelaxant and anti-hypertensive effects of PGE₁ are used to treat male erectile dysfunction and to provide emergency vasodilation of the patent ductus arteriosus in infants whose cardiac anomalies require pulmonary shunting for survival.1 mg
5 mg
10 mg
50 mg9-oxo-11 α ,15S-dihydroxy-prost-13E-en-1-oic acidProstaglandin E₁-d₄

313010

Alprostadil-d₄**MF:** C₂₀H₃₀D₄O₅ **FW:** 358.5 **Chemical Purity:** $\geq 99\%$ **Deuterium Incorporation:** $\leq 1\%$ d₀A solution in methyl acetate **Stability:** ≥ 1 year at -20°C **Summary:** PGE₁-d₄ contains four deuterium atoms at the 3, 3', 4, and 4' positions. It is intended for use as an internal standard for the quantification of PGE₁ by GC- or LC-MS.50 μg
100 μg
500 μg
5 mg9-oxo-11 α ,15S-dihydroxy-prost-13E-en-1-oic-3,3,4,4-d₄ acid

2,3-dinor-6-keto Prostaglandin F_{1α} EIA Kit 515121**Stability:** ≥1 year at -20°C

Summary: Prostacyclin (Prostaglandin I₂; PGI₂) is formed from arachidonic acid primarily in the vascular endothelium and renal cortex by sequential activities of COX and PGI₂ synthase. It is a potent vasodilator and inhibitor of platelet aggregation. PGI₂ is non-enzymatically hydrated to 6-keto PGF_{1α}, and then quickly converted to the major urinary metabolite, 2,3-dinor-6-keto PGF_{1α}. Estimates of systemic PGI₂ production have often been assessed by measurement of 6-keto PGF_{1α} alone or in combination with 2,3-dinor-6-keto PGF_{1α}. However, the majority of 6-keto PGF_{1α} in urine is of renal origin with only 14% originating from plasma. Cayman's 2,3-dinor-6-keto PGF_{1α} EIA utilizes a highly selective monoclonal antibody that exhibits no cross reactivity with 6-keto PGF_{1α}, thus providing a method for accurate measurement of systemic PGI₂ production. These measurements are highly relevant to vascular biology research, especially as is relates to the elevated risk of myocardial infarction and stroke associated with the use of COX-2 selective inhibitors.

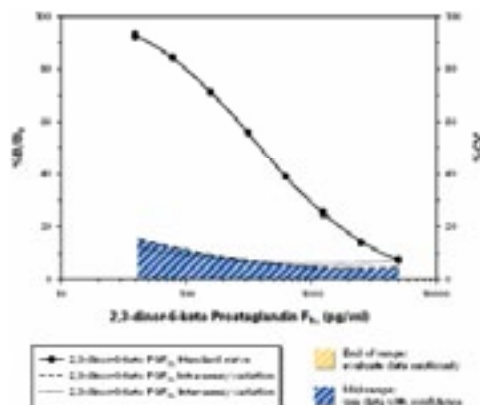
Sensitivity: 50% B/B₀: 400 pg/ml
80% B/B₀: 100 pg/ml

Specificity:

2,3-dinor-6-keto Prostaglandin F _{1α}	100%
tetranor-PGFM	0.07%
Prostaglandin B ₁	<0.01%
Prostaglandin B ₂	<0.01%
Prostaglandin E ₁	<0.01%
Prostaglandin E ₂	<0.01%
Prostaglandin F _{1α}	<0.01%
13,14-dihydro-Prostaglandin F _{1α}	<0.01%
6,15-diketo-13,14-dihydro-Prostaglandin F _{1α}	<0.01%
6-keto Prostaglandin F _{1α}	<0.01%
Prostaglandin F _{2α}	<0.01%
15-keto Prostaglandin F _{2α}	<0.01%
Thromboxane B ₂	<0.01%

96 wells
480 wells

Also Available: 2,3-dinor-6-keto Prostaglandin F_{1α} EIA Kit (Solid Plate) (10008826)

**6-keto Prostaglandin F_{1α}** 15210

[58962-34-8]

MF: C₂₀H₃₄O₆ **FW:** 370.5 **Purity:** ≥99%A crystalline solid **Stability:** ≥2 years at -20°C

Summary: 6-keto PGF_{1α} is the inactive, non-enzymatic hydrolysis product of PGI₂. 6-keto PGF_{1α} serves as a useful marker of PGI₂ biosynthesis *in vivo*. When [³H]-PGI₂ is injected into healthy human males, 6.6% of the radioactivity is recovered from urine as [³H]-6-keto PGF_{1α}.

1 mg
5 mg
10 mg
50 mg



6-oxo-9α,11α,15S-trihydroxy-prost-13E-en-1-oic acid

6-keto Prostaglandin F_{1α}-d₄ 315210**MF:** C₂₀H₃₀D₄O₆ **FW:** 374.5 **Chemical Purity:** ≥98%**Deuterium Incorporation:** ≤1% d₀A solution in methyl acetate **Stability:** ≥1 year at -20°C

Summary: PGF_{1α}-d₄ contains four deuterium atoms at the 3, 3', 4, and 4' positions. It is intended for use as an internal standard for the quantification of 6-keto PGF_{1α} by GC- or LC-MS.

25 μg
50 μg
100 μg
1 mg

6-oxo-9α,11α,15S-trihydroxy-prost-13E-en-1-oic-3,3,4,4-d₄ acid**6-keto Prostaglandin F_{1α} EIA Kit** 515211**Stability:** ≥1 year at -20°C

Summary: Prostacyclin (Prostaglandin I₂; PGI₂) is formed from arachidonic acid primarily by the vascular endothelium and renal cortex. It is a potent vasodilator and inhibitor of platelet aggregation. PGI₂ is non-enzymatically hydrated to 6-keto PGF_{1α} (t_{1/2} = 2-3 minutes), and then quickly converted to the major metabolite, 2,3-dinor-6-keto PGF_{1α} (t_{1/2} = 30 minutes). Prostacyclin was once thought to be a circulating hormone that regulated platelet-vasculature interactions, but the rate of secretion into circulation coupled with the short half-life indicate that prostacyclin functions locally. Although 6-keto PGF_{1α} is commonly measured in plasma and urine as an estimate of prostacyclin synthesis, it should be noted that there may be more than one source of PGI₂ in these samples. For instance, venipuncture may cause the release of prostacyclin which will artifactually increase the 6-keto PGF_{1α} concentration in plasma. Urinary concentrations of 6-keto PGF_{1α} are confounded by the fact that some plasma prostacyclin (~14%) is excreted into urine as 6-keto PGF_{1α} and the remainder is of renal origin. Therefore, it is important to take these factors into account when analyzing data.

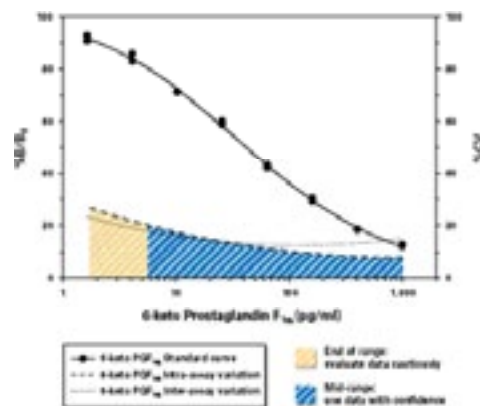
Sensitivity: 50% B/B₀: 43 pg/ml
80% B/B₀: 11 pg/ml

Specificity:

6-keto Prostaglandin F _{1α}	100%
6-keto Prostaglandin E ₁	33.9%
Prostaglandin F _{1α}	28%
Prostaglandin F _{2α}	11%
2,3-dinor-6-keto Prostaglandin F _{1α}	4.9%
Prostaglandin E ₂	1.5%
6,15-diketo-13,14-dihydro Prostaglandin F _{1α}	0.33%
13,14-dihydro-15-keto Prostaglandin F _{1α}	0.05%
Thromboxane B ₂	0.05%
tetranor-PGEM	<0.01%
tetranor-PGFM	<0.01%
Prostaglandin D ₂	<0.01%

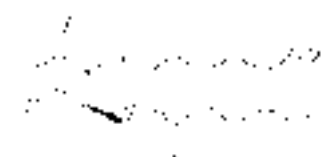
96 wells
480 wells

Also Available: 6-keto Prostaglandin F_{1α} EIA Kit (Solid Plate) (515211.1)

**NEW 9,11-methane-epoxy Prostaglandin F_{1α}** 10007850[72517-81-8] 9,11-epoxymethano PGH₁**MF:** C₂₁H₃₆O₄ **FW:** 352.5 **Purity:** ≥96%A solution in methyl acetate **Stability:** ≥1 year at -20°C

Summary: 9,11-methane-epoxy PGF_{1α} is a stable epoxymethano analog of PGH₁ that produces a strong and dose-related aggregation of washed rabbit platelets (EC₅₀ = 0.88 μM) and contraction of rabbit aortic strips (EC₅₀ = 0.11 μM). 9,11-methane-epoxy PGF_{1α} induces contraction of guinea pig tracheas (EC₅₀ = 3.4 μM) with a maximal contraction of about 60% of that caused by a submaximal dose of histamine.

100 μg
500 mg
1 mg
5 mg



6-(3-hydroxy-1-octenyl)-2-oxabicyclo[2.2.1]heptane-5-heptanoic acid

Prostaglandin H₂ 17020

[42935-17-1]

MF: C₂₀H₃₂O₅ **FW:** 352.5 **Purity:** ≥95%*A solution in acetone **Stability:** ≥6 months at -80°C

Summary: PGH₂ is the product of COX-1 and COX-2 metabolism of arachidonic acid and serves as the precursor for all 2-series PGs and TXs. It is a TP receptor agonist which irreversibly aggregates human platelets at 50-100 ng/ml. PGH₂ is a suicide substrate for platelet TX synthase possessing a K_i value of 18 μM as compared to 28 μM for PGH₁.

25 μg
50 μg
100 μg
500 μg



9α,11α-epidioxy-15S-hydroxy-prosta-5Z,13E-dien-1-oic acid

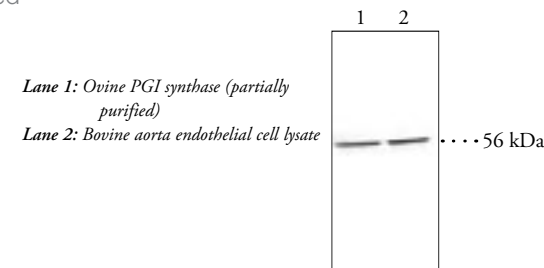
Prostaglandin I Synthase Polyclonal Antibody 160640

PGI Synthase, PGIS, Prostacyclin Synthase

Peptide affinity-purified IgG **Stability:** ≥1 year at -20°C

Summary: Antigen: bovine PGI synthase amino acids 299-329 • Host: rabbit • Cross-reactivity: (+) bovine, ovine, and human PGI synthase; (-) rat PGI synthase • Applications: WB and IP; other applications not tested • PGIS catalyzes the isomerization of PGH₂ to PGI₂, a potent vasodilator and inhibitor of platelet aggregation

1 ea



Also Available: Prostaglandin I Synthase
Blocking Peptide (360640)

200 μg

Prostaglandin I₂ (sodium salt) 18220

[[61849-14-7] Epiprosteno (sodium salt), Prostacyclin (sodium salt)

MF: C₂₀H₃₁O₅ • Na **FW:** 374.5 **Purity:** ≥99%A crystalline solid **Stability:** ≥1 year at -20°C

Summary: PGI₂ is a potent vasodilator and inhibitor of human platelet aggregation with an IC₅₀ value of 5 nM. PGI₂ is rapidly hydrolyzed to 6-keto PGF_{1α} with a half-life ranging from 30 seconds to a few minutes. PGI₂ is administered by continuous infusion in humans for the treatment of idiopathic pulmonary hypertension.

1 mg
5 mg
10 mg
50 mg



6,9α-epoxy-11α,15S-dihydroxy-prosta-5Z,13E-dien-1-oic acid, sodium salt

Prostaglandin I₃ (sodium salt) 18300

[68324-96-9]

MF: C₂₀H₂₉O₅ • Na **FW:** 372.4 **Purity:** ≥99%A crystalline solid **Stability:** ≥6 months at -20°C

Summary: PGI₃ is synthesized from EPA by COX and PGI synthase. PGI₃ has a short *in vivo* half-life and is hydrolyzed to D¹⁷-6-keto PGF_{1α}. The platelet and vascular activity of PGI₃ is equivalent to that of PGI₂.

500 μg
1 mg
5 mg
10 mg



6,9α-epoxy-11α,15S-dihydroxy-prosta-5Z,13E,17Z-trien-1-oic acid, sodium salt

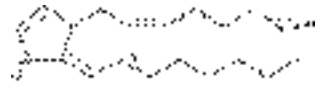
15-deoxy-Δ^{12,14}-Prostaglandin J₂ 18570

[89886-60-2]

MF: C₂₀H₂₈O₃ **FW:** 316.4 **Purity:** ≥97%*A solution in methyl acetate **Stability:** ≥1 year at -20°C

Summary: 15-deoxy-D^{12,14}-PGJ₂ is formed from PGD₂ by the elimination of two molecules of water. It binds selectively to PPARG with an EC₅₀ value of 2 μM in a murine chimera system. 15-deoxy-D^{12,14}-PGJ₂ is more potent than PGD₂, D¹²-PGJ₂, and PGJ₂ in stimulating lipogenesis in C3H10T1/2 cells. The EC₅₀ value for induction of adipocyte differentiation in cultured fibroblasts is 7 μM.

100 μg
500 μg
1 mg
5 mg



11-oxo-prosta-5Z,9,12E,14Z-tetraen-1-oic acid

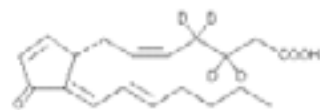
Also Available: 15-deoxy-D^{12,14}-Prostaglandin J₂ (18570.1)

1 mg
5 mg
10 mg
50 mg

*All 5-cis 2-series PGs (those containing a 5,6-double bond) will contain a small amount of the 5-trans isomer. This isomer is generally undetectable using normal phase silica columns and plates, but may be resolved using RP-HPLC. The purity for all such 2-series PGs excludes the 1-3% trans isomer which will generally be present.

15-deoxy- $\Delta^{12,14}$ -Prostaglandin J₂-d₄

318570

MF: C₂₀H₂₄D₄O₃ **FW:** 320.5 **Chemical Purity:** ≥98%***Deuterium Incorporation:** ≤1% d₀A solution in methyl acetate **Stability:** ≥1 year at -20°C**Summary:** 15-deoxy-D^{12,14}-PGJ₂-d₄ contains four deuterium atoms at the 3, 3', 4, and 4' positions. It is intended for use as an internal standard for the quantification of 15-deoxy-D^{12,14}-PGJ₂ by GC- or LC-MS.25 µg
50 µg
100 µg
1 mg11-oxo-prosta-5Z,9,12E,14Z-tetraen-1-oic-3,3,4,4-d₄ acid**Renin (human recombinant)**

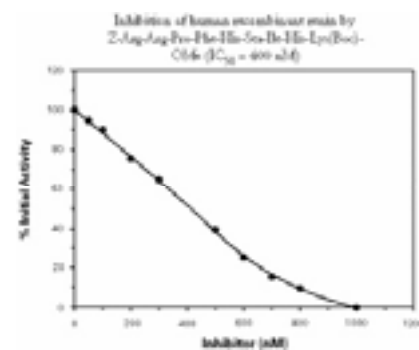
10006217

M_r: 40 kDa **Purity:** ≥99% by SDS-PAGEA solution in sodium acetate buffer **Stability:** ≥1 year at -20°C**Summary:** Source: recombinant enzyme expressed in HEK cells • **M_r:** 40 kDa • Renin is an aspartyl protease that catalyzes the initial and rate limiting step in the renin-angiotensin system (RAS) pathway, converting angiotensinogen into angiotensin I.5 µg
10 µg
25 µg
50 µg**Also Available:** Prorenin (human recombinant) (10007599)25 µg
50 µg
100 µg
500 µg**NEW Renin Inhibitor Screening Assay Kit**

10006270

Stability: ≥6 months at -80°C**Summary:** Renin is an aspartyl protease that catalyzes the initial and rate limiting step in the renin-angiotensin system (RAS) pathway, converting angiotensinogen into angiotensin I. Angiotensin Converting Enzyme (ACE) subsequently converts angiotensin I to angiotensin II, which is a potent vasoconstrictor. The Cayman Chemical Renin Inhibitor Screening Assay Kit provides a convenient assay in a 96-well format for evaluating human renin inhibitors. The assay utilizes a renin-based synthetic peptide substrate which incorporates the fluorophore EDANS at one end and an EDAN-quenching molecule (Dabcyl) at the other end. After cleavage by renin, the peptide-EDANS product is released yielding bright fluorescence that can be easily analyzed using excitation wavelengths of 335-345 nm and emission wavelengths of 485-510 nm. The assay kit includes recombinant human renin (sufficient for 100 reactions), substrate, buffers, and complete instructions.

96 wells

**trans-Resveratrol**

70675

[501-36-0]

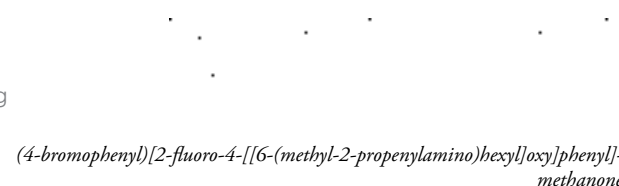
MF: C₁₄H₁₂O₃ **FW:** 228.2 **Purity:** ≥98%A crystalline solid **Stability:** ≥1 year at -20°C**Summary:** trans-Resveratrol is a potent phenolic antioxidant found in grapes and red wine that also has antiproliferative and anti-inflammatory activity.50 mg
100 mg
250 mg
500 mg

5-[(1E)-2-(4-hydroxyphenyl)ethenyl]-1,3-benzenediol

Ro 48-8071

10006415

[161582-11-2]

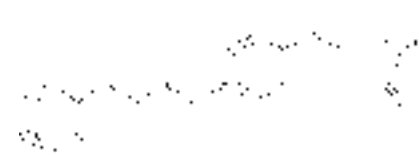
MF: C₂₃H₂₇BrFNO₂ **FW:** 448.4 **Purity:** ≥98%A solution in methyl acetate **Stability:** ≥1 year at -20°C**Summary:** Oxidosqualene cyclase (OSC) is a microsomal enzyme that catalyzes the cyclization of monooxidosqualene to lanosterol in the cholesterol synthetic pathway. Ro 48-8071 is an inhibitor of OSC that has LDL cholesterol lowering activity similar to the HMG-CoA inhibitor simvastatin. It inhibits OSC from human liver microsomes and HepG2 cells with IC₅₀ values of approximately 6.5 nM and 1.5 nM, respectively. Ro 48-8071 lowered LDL cholesterol ~40% in hamsters at a dose of 150 µg/kg without affecting HDL levels and with no sign of liver toxicity.5 mg
10 mg
50 mg
100 mg

(4-bromophenyl)[2-fluoro-4-[[16-(methyl-2-propenylamino)hexyl]oxy]phenyl]methanone

NEW Rosiglitazone

71740

[122320-73-4] BRL 49653

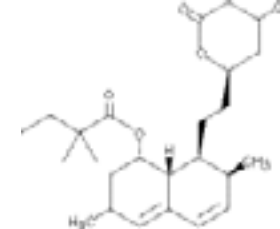
MF: C₁₈H₁₉N₃O₃S **FW:** 357.4 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C**Summary:** Rosiglitazone is a potent and selective PPAR γ ligand. It binds to the PPAR γ ligand-binding domain with a K_d of 43 nM. It activates luciferase-based expression constructs for PPAR γ 1 and PPAR γ 2 with EC₅₀ values of approximately 30 nM and 100 nM, respectively. Rosiglitazone is active *in vivo* as an antidiabetic agent in the *ob/ob* mouse model, and has been used as an oral hypoglycemic agent in the treatment of Type II diabetes in humans for many years.5 mg
10 mg
50 mg
100 mg

5-[[4-[2-(methyl-2-pyridinylamino)ethoxy]phenyl]methyl]-2,4-thiazolidinedione

NEW Simvastatin

10010344

[79902-63-9]

MF: C₂₅H₃₈O₅ **FW:** 357.4 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C**Summary:** Simvastatin is competitive inhibitor of HMG-CoA reductase with a K_i value of 0.12 nM for the hydrolyzed, open ring form of the molecule.5 mg
10 mg
25 mg
50 mg**Also Available:** Simvastatin (sodium salt) (10010345)5 mg
10 mg
25 mg
50 mg**SQ 29,548**

19025

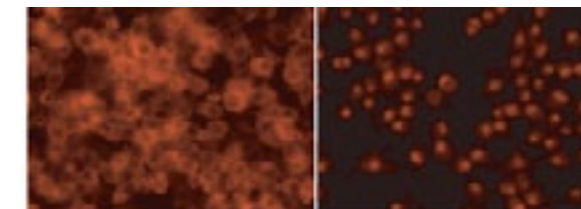
[98672-91-4]

MF: C₂₁H₂₉N₃O₄ **FW:** 387.5 **Purity:** ≥99%*A crystalline solid **Stability:** ≥1 year at -20°C**Summary:** SQ 29,548 is a highly selective TP receptor antagonist which binds to the human recombinant TP receptor with a K_i value of 4.1 nM. It inhibits the aggregation of washed human platelets induced by U-46619 with an IC₅₀ value of 0.06 µM. It antagonizes U-46619 induced contraction of rat and guinea pig tracheal, arterial, and venous smooth muscles with drug/receptor dissociation constants (K_B) in the range of 0.5-1.7 nM.1 mg
5 mg
10 mg
100 mg[1S-[1 α ,2 α (Z),3 α ,4 α]]-7-[3-[[2-[(phenylamino)carbonyl]hydrazino]methyl]-7-oxabicyclo[2.2.1]hept-2-yl]-5-heptenoic acid**NEW SREBP-2 Cell-Based Translocation Assay Kit**

10009239

SREBF-2**Stability:** ≥6 months at -20°C**Summary:** SREBP-2 is a transcription factor that regulates cholesterol synthesis by activating the expression of genes for HMG-CoA reductase and other enzymes of the cholesterol synthetic pathway. Cayman's SREBP-2 Cell-Based Translocation Assay Kit provides the tools needed to study SREBP-2 movement within whole cells. The kit contains a highly specific SREBP-2 primary antibody together with a DyLight™ (trademarked by Pierce Biotechnology Inc.) conjugated secondary antibody in a ready to use format. Also included as a positive control is a cholesterol trafficking inhibitor, U18666A, which has been shown to activate SREBP-2 translocation into nuclei by scientists at Cayman Chemical Company.

96 wells



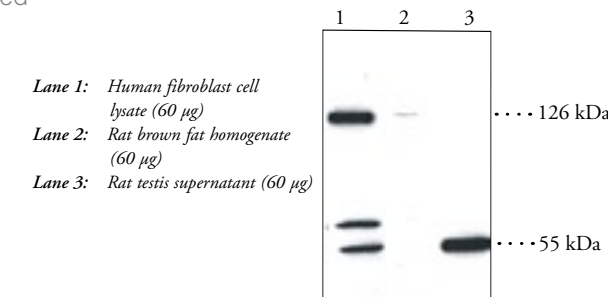
Translocation of SREBP-2 from cytosol to 24 pH6 U-18666A. Raw 264.7 cells were seeded in a 96-well plate at a density of 5x10⁴ cells/well and cultured overnight. The next day, cells were treated with U18666A (vehicle) or 24 pH6 U-18666A for 72 hours. Left panel: Cells treated with DMSO alone demonstrate cytoplasmic localization of SREBP-2, indicating that most of cells have inactive protein. Right panel: U-18666A treatment for three days induced SREBP-2 translocation into the nuclei, indicating that blockade of cholesterol transport in these cells activates the protein.

NEW SREBP-2 Polyclonal Antibody

10007663

SREBF-2Peptide affinity-purified IgG **Stability:** ≥1 year at -20°C**Summary:** Antigen: human SREBP-2 amino acids 455-469 • Host: rabbit • Cross-reactivity: (+) human, murine, and rat SREBP-2 • Applications: WB and ICC • SREBP-2 is a transcription factor that plays a critical role in lipid homeostasis by regulating genes involved in cholesterol and fatty acid metabolism.

1 ea



Lane 1: Human fibroblast lysate (60 µg)

Lane 2: Rat brown fat homogenate (60 µg)

Lane 3: Rat testis supernatant (60 µg)

Lane 3: Rat testis supernatant (60 µg)

Also Available: SREBP-2 Blocking Peptide (10009266) 200 µg

IP & TP Receptor Ligands				
Cat. No.	Catalog Name	Target	Mode of Action	Effective Concentration
18230	Beraprost (sodium salt)	IP	agonist	Doses of 20-100 µg are effective in humans
10155	BM 567	TP	antagonist	IC ₅₀ = 1.1 nM
18210	Carbaprostacyclin	IP	agonist	ED ₅₀ = 47 nM for inhibition of platelet aggregation
19010	Carbocyclic Thromboxane A ₂	TP	agonist	1 nM effectively constricts cat coronary arteries
10005186	CAY10441	IP	antagonist	K _i = 1.5 nM
10005913	CAY10449	IP	antagonist	K _i = 3 nM
18216	Ciprostene (calcium salt)	IP	agonist	ID ₅₀ = 60 ng/ml for inhibition of ADP-induced platelet aggregation <i>in vitro</i>
18215	Iloprost	IP	agonist	K _i = 11 nM (binds with equal affinity to EP ₁ receptor)
19021	I-SAP	TP	antagonist	K _d = 0.5 nM (human platelets)
18220	PGI ₂	IP	agonist	IC ₅₀ = 5 nM for inhibition of human platelet aggregation
19020	Pinane Thromboxane A ₂	TP	antagonist	IC ₅₀ = 2 µM for inhibition of U-46619-induced aggregation of human platelets
19025	SQ 29,548	TP	antagonist	K _i = 4.1 nM
16440	U-44069	TP	agonist	EC ₅₀ = 3 µM for platelet aggregation
16450	U-46619	TP	agonist	EC ₅₀ = 82 nM for human platelet aggregation

Transcription Factor Agonists & Antagonists				
Cat. No.	Catalog Name	Target	Mode of Action	Effective Concentration
10007686	Acetyl Podocarpic Acid Anhydride	LXR	agonist	ED ₅₀ = 1 nM
60924	Azelaoyl PAF	PPAR _γ	agonist	~equal to rosiglitazone
10009145	Bezafibrate	pan PPAR	agonist	EC ₅₀ = 20 - 60 µM
10009017	CAY10514	PPAR _α and PPAR _γ	dual agonist	EC ₅₀ = 0.173 µM (PPAR _α) EC ₅₀ = 0.642 µM (PPAR _γ)
89355	22(R)-hydroxy Cholesterol	LXR	agonist	ED ₅₀ = 325 nM
71730	Ciglitazone	PPAR _γ	agonist	EC ₅₀ = 3 µM
10005745	Clofibrate	PPAR _α	agonist	EC ₅₀ = 55 µM (human)
10005368	Fenofibrate	PPAR _α	agonist	EC ₅₀ = 30 µM (human)
10006798	GW 0742	PPAR _δ	agonist	EC ₅₀ = 1.1 nM
10008613	GW 7647	PPAR _α	agonist	EC ₅₀ = 6 nM (human)
10011211	GW 9578	PPAR _α	agonist	EC ₅₀ = 50 nM (human)
70785	GW 9662	PPAR _γ	antagonist	blocks differentiation of monocytes to osteoclasts by >90% at a dose of 0.1 µM
10009880	GW 590735	PPAR _α	agonist	EC ₅₀ = 4 nM
71740	Rosiglitazone	PPAR _γ	agonist	K _d = 43 nM
10026	T0070907	PPAR _γ	antagonist	IC ₅₀ = 1 nM for inhibition of rosiglitazone binding
71810	T0901317	LXR _α and LXR _β	agonist	EC ₅₀ = 50 nM
71750	Troglitazone	PPAR _γ	agonist	EC ₅₀ = 0.55 µM (human)

Tom Brock, Ph.D.

Inflammation in Atherosclerosis Prostaglandins versus Leukotrienes

Prostaglandins are a diverse group of lipid mediators derived from arachidonic acid by the cyclooxygenase (COX) pathway. Of the two COX isoforms, COX-1 is, in most cases, constitutively expressed, whereas COX-2 is an immediate early gene that is rapidly up-regulated in response to a variety of inflammatory cues. Aspirin and non-steroidal anti-inflammatory drugs (NSAIDs) inhibit both isoforms of COX, with aspirin's effects being irreversible. These inhibitors are effective at inhibiting pain and reducing fever because they inhibit the synthesis of a major COX-2 product, prostaglandin E₂ (PGE₂). However, continued use of these general COX inhibitors has damaging effects on the stomach and kidneys because they inhibit the production of protective PGs by COX-1 at those sites. Selective COX-2 inhibitors, the 'coxibs', were developed to inhibit the inflammatory effects of COX-2 products without affecting the protective effects of the COX-1 PGs.

Both COX-1 and COX-2 convert arachidonic acid to an intermediate, PGH₂, which then must be processed by specific enzymes to produce each type of PG. Surprisingly, activity by the two COX isoforms does not give rise to the same products, as COX-2 tends to generate more prostacyclin (PGI₂) and PGE₂ than COX-1. Perhaps even more surprising are the anti-inflammatory actions shared by these two mediators. PGI₂, through the IP receptor, and PGE₂, through EP₂ and EP₄, activate adenylyl cyclase on leukocytes, elevating cytoplasmic cAMP. The second messenger cAMP activates pathways that inhibit a broad array of leukocyte functions. In platelets, elevated cAMP, as induced by PGI₂, suppresses cell activation and thromboxane synthesis. In monocytes/macrophages, cAMP signalling inhibits cell adherence and migration, scavenger receptor endocytosis, phagocytosis and killing of bacteria, and the synthesis of pro-inflammatory cytokines such as TNF- α and IL-1 β . In the context of atherosclerosis, exposure of platelets, monocytes, and macrophages to the COX-2 products, PGI₂ and PGE₂, should be protective.

Leukotrienes (LTs) are made from arachidonic acid by the 5-lipoxygenase (5-LO) pathway. As 5-LO is expressed primarily by leukocytes, these cells are the major producers of LTs. There are two types of LTs, LTB₄ and the cysteinyl LTs, LTC₄, LTD₄, and LTE₄. LTB₄ is recognized as a pro-inflammatory mediator as it attracts

and activates a broad array of leukocytes, including monocytes, macrophages, and lymphocytes. The cysteinyl LTs are best known for their roles in allergic responses and asthma, where they modulate epithelial, endothelial, and vascular smooth muscle cells. Less well known are the effects of LTs on gene expression. LTB₄ alters gene expression on leukocytes and other cell types bearing LTB₄ receptors and the cysteinyl LTs modulate gene expression on epithelial, endothelial, and vascular smooth muscle cells, as well as other cells. The effects of LTs on gene expression are diverse and cell specific, leading to an array of changes that promote inflammation, alter growth rate, and drive tissue remodeling.¹

Genetic studies pointed to a role for the 5-LO pathway in atherosclerosis when, in 2002, the 5-LO gene was identified as a major contributor to atherosclerosis susceptibility in mice.² Interest in the role of LTs in atherosclerosis rose in 2003 when large numbers of 5-LO positive cells were detected in human atherosclerotic arteries.³ The next year, a genome-wide analysis of two independent populations, divided into those having had a myocardial infarction *versus* healthy controls, identified haplotype variants of the 5-LO activating protein, FLAP, as an indicator of susceptibility to cardiovascular disease.⁴ Haplotype variants in FLAP were then found to associate with incidence of stroke in another population.⁵ Gene expression analysis found that 5-LO expression was higher in "vulnerable", unstable plaques in tissues from patients who had undergone carotid endarterectomy,⁶ whereas increased FLAP expression was associated with obesity and insulin resistance.⁷ These studies link the 5-LO pathway with inflammatory cardiovascular diseases and have led to studies on the role of LTs in the pathogenesis, as well as the benefits of pharmaceutical intervention.

References

1. Flamand, N., Mancuso, P., Serezani, C.H.C., et al. *Cell. Mol. Life Sci.* **64**, 2657-2670 (2007).
2. Mehrabian, M., Allayee, H., Wong, J., et al. *Circ. Res.* **91**, 120-126 (2002).
3. Spanbroek, R., Gräbner, R., Lötzer, K., et al. *Proc. Natl. Acad. Sci. USA* **100**(3), 1238-1243 (2003).
4. Helgadottir, A., Manolescu, A., Thorleifsson, G., et al. *Nature Genet.* **36**(3), 233-239 (2004).
5. Helgadottir, A., Gretarsdottir, D., St. Clair, D., et al. *Am. J. Hum. Genet.* **76**, 505-509 (2005).
6. Cipollone, F., Mezzetti, A., Fazio, M.L., et al. *Arterioscler. Thromb. Vasc. Biol.* **25**, 1665-1670 (2005).
7. Kaaman, M., Rydén, M., Axelsson, T., et al. *Int. J. Obes.* **30**, 447-452 (2006).



Figure 1. Eicosanoids are important in the development of atherosclerosis. Leukotrienes recruit and activate monocytes, mast cells and T-cells, and also alter gene expression in endothelial and smooth muscle cells, promoting inflammatory signalling. Thromboxane activates platelets, initiating inflammatory and clotting cascades. PGE₂ and PGI₂, synthesized by a variety of cell types, suppresses macrophage activation, reduces endocytosis of LDL and oxLDL, and inhibits inflammatory signalling.

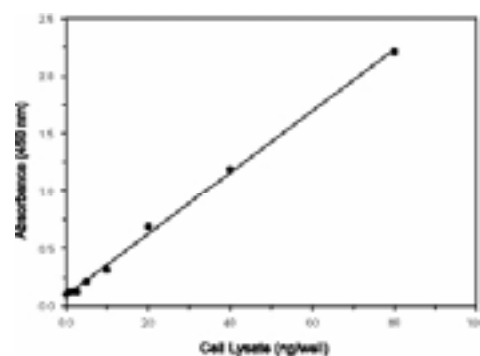
NEW SREBP-2 Transcription Factor Assay Kit 10007819

SREBF-2

Stability: ≥6 months at -20°C

Summary: SREBP-2 is a transcription factor that performs a critical role in the transcriptional regulation of genes involved in cholesterol synthesis and uptake including HMG-CoA synthase, HMG-CoA reductase, and the LDLR. Cayman's SREBP-2 Transcription Factor Assay is a non-radioactive, sensitive method for detecting specific transcription factor DNA binding activity in nuclear extracts and whole cell lysates. A 96-well ELISA replaces the radioactive electrophoretic mobility shift assay (EMSA). A specific double stranded DNA (dsDNA) sequence containing the SREBP response element is immobilized to the wells of a 96-well plate. SREBP contained in a nuclear extract, binds specifically to the SREBP response element. SREBP is detected by addition of specific primary antibody directed against SREBP. A secondary antibody conjugated to HRP is added to provide a sensitive colorimetric readout at 450 nm.

96 wells

**NEW** SREBP-2 Western Ready Control 10009749

SREBF-2

Purity: 94 kDa (GST-tagged); 68 kDa (native)**Stability:** ≥6 months at -20°C

Summary: Source: human recombinant protein expressed in *E. coli* with a N-terminal GST-tag • Application: Positive control for WB • SREBP-2 is transcription factor that plays a critical role in lipid homeostasis by regulating genes involved in cholesterol and fatty acid metabolism.

1 ea

T0070907 10026

[313516-66-4]

MF: C₁₂H₈ClN₃O₃ **FW:** 277.7 **Purity:** ≥98%A crystalline solid **Stability:** ≥1 year at -20°C

Summary: T0070907 is a potent and selective human PPARγ antagonist with an apparent IC₅₀ value of 1 nM for the binding inhibition of rosiglitazone. T0070907 covalently binds to Cys313 of PPARγ, inducing conformational changes that block the recruitment of transcriptional cofactors to the PPARγ/RXR heterodimer.

1 mg
5 mg
10 mg
50 mg

2-chloro-5-nitro-N-(4-pyridinyl)-benzamide

*All 5-cis 2-series PGs (those containing a 5,6-double bond) will contain a small amount of the 5-trans isomer. This isomer is generally undetectable using normal phase silica columns and plates, but may be resolved using RP-HPLC. The purity for all such 2-series PGs excludes the 1-3% trans isomer which will generally be present.

T0901317 71810

[293754-55-9]

MF: C₁₇H₁₂F₉NO₃S **FW:** 481.3 **Purity:** ≥98%A crystalline solid **Stability:** ≥1 year at -20°C

Summary: T0901317 is a potent and selective agonist for both LXRA and LXRb, with an EC₅₀ of about 50 nM. T0901317, acting through LXR and in concert with its RXR heterodimerization partner, induces the expression of the ABCA1 reverse cholesterol transporter. This acts to increase the efflux of cholesterol from enterocytes and thus inhibit the overall absorption of cholesterol.

5 mg
10 mg
50 mg
100 mg

N-(2,2,2-trifluoroethyl)-N-[4-[2,2,2-trifluoro-1-hydroxy-1-(trifluoromethyl)ethyl]phenyl]-benzenesulfonamide

NEW TGX-221 10007349

[663619-89-4]

MF: C₂₁H₂₄N₄O₂ **FW:** 364.4 **Purity:** ≥98%A solution in ethanol **Stability:** ≥1 years at -20°C

Summary: TGX-221 is a potent, selective, ATP-competitive inhibitor of PI3-K p110b. TGX-221 inhibits PtdIns-(3,4)-P₂ production in platelets with an IC₅₀ value of 50 nM. Selective inhibition of PI3K p110b results in defective platelet thrombus formation and defines PI3K as a target for antithrombotic therapy.

100 µg
500 µg
1 mg
5 mg

7-methyl-2-(4-morpholinyl)-9-[1-(phenylamino)ethyl]-4H-pyrido[1,2-a]pyrimidin-4-one

Thromboxane B₂ 19030

[54397-85-2]

MF: C₂₀H₃₄O₆ **FW:** 370.5 **Purity:** ≥99%*A crystalline solid **Stability:** ≥2 years at -20°C

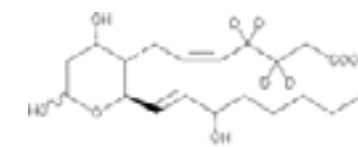
Summary: TXB₂ is a stable, biologically inert metabolite formed from the non-enzymatic hydrolysis of TXA₂, which has a half-life of about 30 seconds. Urinary analysis of TXB₂ accurately reflects intrarenal TXA₂ synthesis, while measurement of 11-dehydro and 2,3-dinor TX metabolites gives the best estimate of systemic TXA₂ secretion.

1 mg
5 mg
10 mg

9α,11,15S-trihydroxythromba-5Z,13E-dien-1-oic acid

Thromboxane B₂-d₄ 319030**MF:** C₂₀H₃₀D₄O₆ **FW:** 374.5 **Chemical Purity:** ≥98%***Deuterium Incorporation:** ≤1% d₀A solution in methyl acetate **Stability:** ≥1 year at -20°C

Summary: TXB₂-d₄ contains four deuterium atoms at the 3, 3', 4, and 4' positions. It is intended for use as an internal standard for the quantification of TXB₂ by GC- or LC-MS.

25 µg
50 µg
100 µg
500 µg9α,11,15S-trihydroxy-thromba-5Z,13E-dien-1-oic-3,3,4,4-d₄ acidThromboxane B₂ 11-dehydrogenase Polyclonal Antiserum 160720100 µl lyophilized antiserum **Stability:** ≥3 years at -20°C

Summary: Antigen: human erythrocyte TXB₂ 11-dehydrogenase • Host: rabbit • Cross-reactivity: (+) human erythrocyte, porcine liver, and ovine liver TXB₂ 11-dehydrogenase • Application: WB; other applications not tested • TXB₂ 11-dehydrogenase catalyzes the conversion of TXB₂ to 11-dehydro TXB₂.

1 ea

Lane 1: Porcine liver homogenate
10,000 × g supernatant (30 µg)
Lane 2: Ovine liver homogenate
10,000 × g supernatant (30 µg)



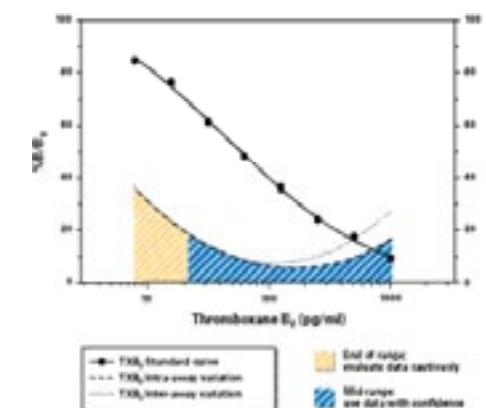
55 kDa

Thromboxane B₂ EIA Kit 519031**Stability:** ≥1 year at -20°C

Summary: TXA₂ is produced from arachidonic acid by many cells and causes irreversible platelet aggregation and vascular and bronchial smooth muscle contraction. TXA₂ is rapidly hydrolyzed non-enzymatically to form TXB₂, which is then quickly metabolized (t_{1/2} = 5-7 minutes) to urinary metabolites for clearance by the kidneys. Because of the transient nature of this compound it is difficult to accurately measure circulating levels in whole-animal experimental models. In fact, it has been shown that plasma and urine levels of TXB₂ are primarily due to *ex vivo* platelet activation and intra-renal production, respectively. Therefore, measurement of TXB₂ metabolites such as 11-dehydro TXB₂ (Catalog No. 519501) and 2,3-dinor TXB₂ (Catalog No. 519051) in urine and plasma may give better estimates of *in vivo* TXA₂ production. TXB₂ measurement is better suited towards samples that are not expected to undergo extensive metabolism such as perfusates, lavage samples, and tissue/cell culture medium or lysates.

Sensitivity: 50% B/B₀: 57 pg/ml
80% B/B₀: 11 pg/ml**Specificity:**

Thromboxane B ₂	100%
Thromboxane B ₃	200%
2,3-dinor Thromboxane B ₂	9.9%
11-dehydro Thromboxane B ₃	0.62%
Prostaglandin D ₂	0.53%
11-dehydro Thromboxane B ₂	0.42%
Prostaglandin F _{2a}	0.25%
Prostaglandin F _{1a}	0.11%
Prostaglandin E ₂	0.09%
6-keto Prostaglandin F _{1a}	0.08%
Leukotriene B ₄	<0.01%
tetranor-PGEM	<0.01%
tetranor-PGFM	<0.01%
13,14-dihydro-15-keto Prostaglandin F _{2a}	<0.01%

96 wells
480 wells**Also Available:** Thromboxane B₂ EIA Kit (Solid Plate) (519031.1)

*All 5-cis 2-series PGs (those containing a 5,6-double bond) will contain a small amount of the 5-trans isomer. This isomer is generally undetectable using normal phase silica columns and plates, but may be resolved using RP-HPLC. The purity for all such 2-series PGs excludes the 1-3% trans isomer which will generally be present.

NEW Thromboxane B₂ Express
EIA Kit - Monoclonal

10004023

Stability: ≥1 year at -20°C

Summary: TXA₂ is produced from arachidonic acid by many cells and causes irreversible platelet aggregation and contraction of vascular and bronchial smooth muscle. TXA₂ is rapidly hydrolyzed non-enzymatically to TXB₂, which is then quickly metabolized to urinary metabolites for clearance by the kidneys. Urinary analysis of TXB₂ accurately reflects intrarenal TXA₂ synthesis, while measurement of 11-dehydro and 2,3-dinor TX metabolites gives the best estimate of systemic TXA₂ secretion. Cayman's TXB₂ Express EIA is a competitive assay that provides accurate measurements of TXB₂ from a variety of sample types. As the name implies, this kit was designed for rapid measurements of TXB₂.

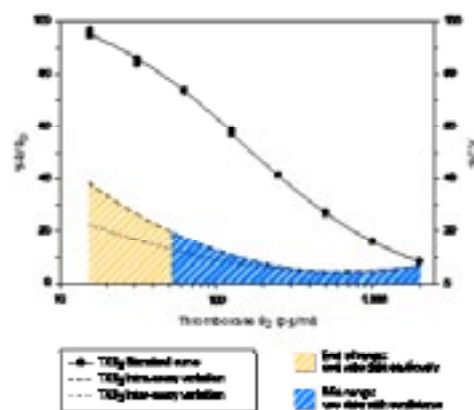
Sensitivity: 50% B/B₀: 176 pg/ml
80% B/B₀: 45 pg/ml

Specificity:

Thromboxane B ₂	100%
Thromboxane B ₃	10.1%
2,3-dinor Thromboxane B ₂	7.9%
Prostaglandin F _{1a}	5.6%
Prostaglandin D ₂	3.1%
Prostaglandin F _{2a}	2.2%
6-keto Prostaglandin F _{1a}	0.74%
Prostaglandin E ₂	0.72%
2,3-dinor-6-keto Prostaglandin F _{1a}	0.04%
11-dehydro Thromboxane B ₂	0.04%
11-dehydro Thromboxane B ₃	0.01%
Leukotriene B ₄	<0.01%
tetranor-PGEM	<0.01%
tetranor-PGFM	<0.01%
13,14-dihydro-15-keto Prostaglandin F _{2a}	<0.01%

96 wells
480 wells

Also Available: Thromboxane B₂ Express EIA Kit (Solid Plate) (10005386)

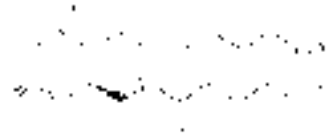
11-dehydro Thromboxane B₂

19500

[67910-12-7]11-keto TXB₂**MF:** C₂₀H₃₂O₆ **FW:** 368.5 **Purity:** ≥98%*A solution in methyl acetate **Stability:** ≥1 year at -20°C

Summary: TXB₂ is released in substantial quantities from aggregating platelets and metabolized during circulation to 11-dehydro TXB₂ and 2,3-dinor TXB₂. 11-dehydro TXB₂ is one of the main plasma metabolites of TXB₂ and can be used as a marker for *in vivo* TXA₂ synthesis. The mean plasma level in human males is 0.9-4.3 pg/ml and the half life is 45-60 minutes. Urinary concentrations of 11-dehydro TXB₂ are approximately 30-70 ng/mmol creatinine.

1 mg
5 mg
10 mg
25 mg



9a,15S-dihydroxy-11-oxothromba-5Z,13E-dien-1-oic acid

*All 5-cis 2-series PGs (those containing a 5,6-double bond) will contain a small amount of the 5-trans isomer. This isomer is generally undetectable using normal phase silica columns and plates, but may be resolved using RP-HPLC. The purity for all such 2-series PGs excludes the 1-3% trans isomer which will generally be present.

11-dehydro Thromboxane B₂-d₄

319500

11-keto TXB₂-d₄**MF:** C₂₀H₂₈D₄O₆ **FW:** 372.5 **Chemical Purity:** ≥99%***Deuterium Incorporation:** ≤1% d₀A solution in methyl acetate **Stability:** ≥1 year at -20°C

Summary: 11-dehydro TXB₂-d₄ contains four deuterium atoms at the 3, 3', 4, and 4' positions. It is intended for use as an internal standard for the quantification of 11-dehydro TXB₂ by GC- or LC-MS.

25 µg
50 µg
100 µg
500 µg

9a,15S-dihydroxy-11-oxothromba-5Z,13E-dien-1-oic-3,3,4,4-d₄ acid11-dehydro Thromboxane B₂ EIA Kit

519501

Stability: ≥1 year at -20°C

Summary: TXA₂ is produced from arachidonic acid by many cells and causes irreversible platelet aggregation and vascular and bronchial smooth muscle contraction. TXA₂ is rapidly hydrolyzed non-enzymatically to form TXB₂. Although it is common to estimate TXA₂ levels by measuring TXB₂, most of the TXB₂ measured in plasma or urine is due to *ex vivo* platelet activation or intra-renal production, respectively. Measurement errors are compounded by the fact that normal concentrations of circulating TXB₂ are extremely low (1-2 pg/ml), and highly transient (t_{1/2} = 5-7 minutes). To circumvent this problem, it is necessary to measure a metabolite that cannot be formed by platelets or by the kidney. TXB₂ can be metabolized by 11-hydroxy TX dehydrogenase to form 11-dehydro TXB₂, or by β-oxidation to form 2,3-dinor TXB₂. Infusion studies using TXB₂ have shown that both metabolites are formed equally, although 11-dehydro TXB₂ has a longer circulating half-life (t_{1/2} = 45 minutes). Therefore, measurement of 11-dehydro TXB₂ in plasma or urine will give a time-integrated indication of TXA₂ production.

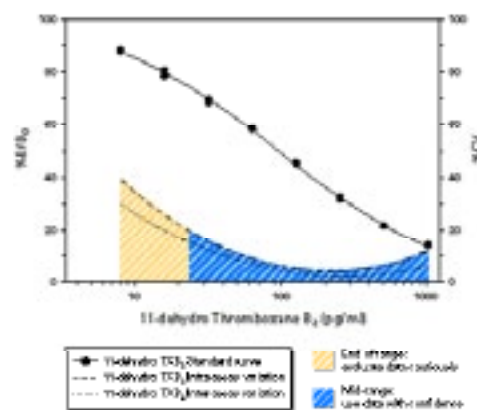
Sensitivity: 50% B/B₀: 93 pg/ml
80% B/B₀: 16 pg/ml

Specificity:

11-dehydro Thromboxane B ₂	100%
11-dehydro-2,3-dinor Thromboxane	11.36%
Prostaglandin D ₂	0.03%
Thromboxane B ₂	<0.01%
Leukotriene B ₄	<0.01%
tetranor-PGEM	<0.01%
tetranor-PGFM	<0.01%
Prostaglandin E ₂	<0.01%
6-keto Prostaglandin F _{1a}	<0.01%
2,3-dinor Thromboxane B ₂	<0.01%

96 wells
480 wells

Also Available: 11-dehydro Thromboxane B₂ EIA Kit (Solid Plate) (519501.1)

2,3-dinor Thromboxane B₂ EIA Kit

519051

Stability: ≥1 year at -20°C

Summary: TXA₂ is produced from arachidonic acid by many cells and causes irreversible platelet aggregation and vascular and bronchial smooth muscle contraction. TXA₂ is rapidly hydrolyzed non-enzymatically to form TXB₂. Although it is common to estimate TXA₂ levels by measuring TXB₂, most of the TXB₂ measured is due to *ex vivo* platelet activation or intra-renal production. Measurement errors are compounded by the fact that normal concentrations of circulating TXB₂ are extremely low (1-2 pg/ml), and highly transient (t_{1/2} = 5-7 minutes). To circumvent this problem, it is necessary to measure a metabolite that cannot be formed by platelets or by the kidney. TXB₂ may be metabolized by 11-hydroxy TX dehydrogenase to form 11-dehydro TXB₂, or by β-oxidation to form 2,3-dinor TXB₂. Infusion studies using TXB₂ have shown that both metabolites are formed equally, but that the circulating half-life of 2,3-dinor TXB₂ is shorter (t_{1/2} = 15 minutes). Therefore, measurement of 2,3-dinor TXB₂ will give a more episodic indication of TXA₂ production.

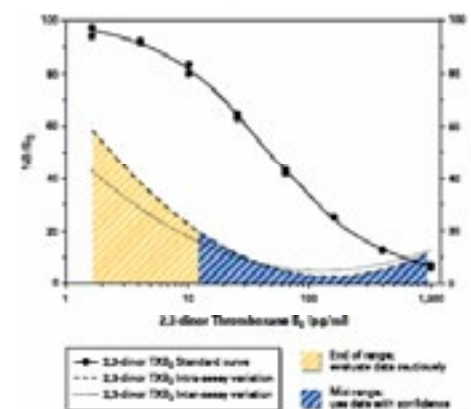
Sensitivity: 50% B/B₀: 45 pg/ml
80% B/B₀: 11 pg/ml

Specificity:

2,3-dinor Thromboxane B ₂	100%
Thromboxane B ₂	100%
2,3-dinor Thromboxane B ₁	3.0%
Prostaglandin D ₂	2.0%
11-dehydro Thromboxane B ₂	1.5%
Prostaglandin F _{2a}	1.0%
Prostaglandin F _{1a}	0.24%
6-keto Prostaglandin F _{1a}	0.06%
2,3-dinor-6-keto Prostaglandin F _{1a}	0.05%
Prostaglandin E ₂	0.04%
13,14-dihydro-15-keto Prostaglandin F _{2a}	<0.01%
tetranor-PGEM	<0.01%
tetranor-PGFM	<0.01%

96 wells
480 wells

Also Available: 2,3-dinor Thromboxane B₂ EIA Kit (Solid Plate) (519051.1)

Thromboxane B₃

19990

[71953-80-5] Δ¹⁷-TXB₂**MF:** C₂₀H₃₂O₆ **FW:** 372.5 **Purity:** ≥98%*A solution in methyl acetate **Stability:** ≥2 years at -20°C

Summary: TXB₃ is the stable hydrolysis product of TXA₃ synthesized from EPA by COX and TX synthase. It is biosynthesized in various tissues such as seminal vesicles, lung, PMNL, and ocular tissues.

50 µg
100 µg
500 µg
1 mg



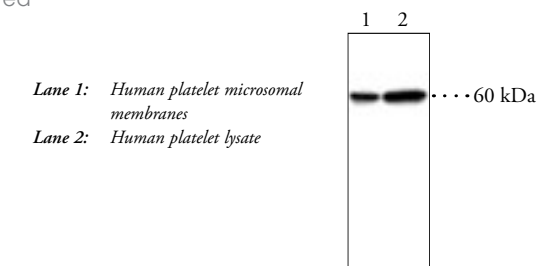
9a,11,15S-trihydroxy-thromba-5Z,13E,17Z-trien-1-oic acid

Thromboxane Synthase Polyclonal Antibody 160715

Peptide affinity-purified IgG **Stability:** ≥1 year at -20°C

Summary: Antigen: human TX synthase amino acids 359-377 • Host: rabbit • Cross-reactivity: (+) human, porcine, murine, and rat TX synthases • Applications: WB and IHC; other applications not tested • TX synthase catalyzes the conversion of PGH₂ to TXA₂, which is a potent vasoconstrictor and inducer of platelet aggregation.

1 ea



Also Available: Thromboxane Synthase
Blocking Peptide (360715) 200 µg

TOFA

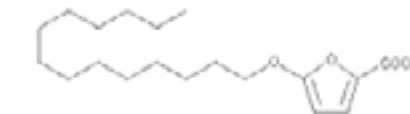
10005263

[54857-86-2] RMI 14514, 5-(Tetradecyloxy)-2-furoic acid

MF: C₁₉H₃₂O₄ **FW:** 324.5 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C

Summary: TOFA is an inhibitor of fatty acid synthesis that blocks the synthesis of malonyl-CoA by acetyl-CoA carboxylase (ACC). TOFA (at about 1 µg/ml) is effective at blocking the incorporation of radiolabeled acetate into palmitate. However, TOFA reduces malonyl-CoA levels rather than elevating them, and TOFA is relatively non-toxic to various cancer cell lines. TOFA also attenuates the inhibition of feeding observed when FAS inhibitors such as cerulenin and C75 are administered to obese *ob/ob* mice.

5 mg
10 mg
50 mg
100 mg



5-(tetradecyloxy)-2-furancarboxylic acid

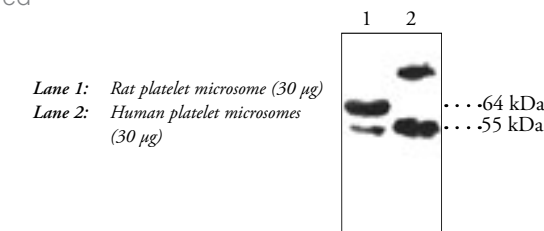
NEW TP Receptor (human)
Polyclonal Antibody

10004452

Peptide affinity-purified IgG **Stability:** ≥1 year at -20°C

Summary: Antigen: human TP receptor C-terminal amino acids 323-343 • Host: rabbit • Cross-reactivity: (+) human, rat, and Cos-7 (African green monkey) TP receptors; other species not tested • Applications: WB and ICC • The TP receptor is a GPCR that mediates the action of TXA₂.

1 ea



Also Available: TP Receptor Blocking Peptide (10009368) 200 µg

*All 5-cis 2-series PGs (those containing a 5,6-double bond) will contain a small amount of the 5-trans isomer. This isomer is generally undetectable using normal phase silica columns and plates, but may be resolved using RP-HPLC. The purity for all such 2-series PGs excludes the 1-3% trans isomer which will generally be present.

NEW Treprostinil 10162

[81846-19-7]

MF: C₂₃H₃₄O₅ **FW:** 390.5 **Purity:** ≥98%A crystalline solid **Stability:** ≥1 year at -20°C

Summary: Treprostinil is a stable analog of prostacyclin that is used clinically for the treatment of primary pulmonary hypertension (PPH) under the trade name Remodulin®. The structural modifications in treprostinil compared to prostacyclin increase the plasma half-life from two minutes to 34 and 85 minutes for intravenous and subcutaneous infusion of the drug, respectively. In addition to treprostinil's direct vasodilatory effects, it also inhibits inflammatory cytokine (TNF-α, IL-1β, IL-6, GM-CSF) production by human alveolar macrophages in the sub-micromolar range by preventing NF-κB translocation to the nucleus.

1 mg
5 mg
10 mg
50 mg

[[[(1R,2R,3aS,9aS)-2,3,4,9,9a-hexahydro-2-hydroxy-1-[(3S)-3-hydroxyoctyl]-1H-benz[f]inden-5-yl]oxy]acetic acid

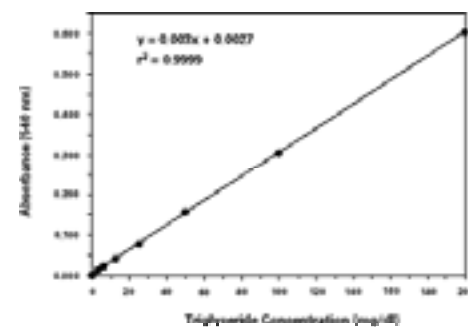
NEW Triglyceride Assay Kit 10010303

TG

Stability: ≥6 months at -20°C

Summary: The measurement of TG levels, in conjunction with other lipid assays, are useful in the diagnosis of primary and secondary hyperlipoproteinemia, dyslipidemia, and triglyceridemia. Cayman's TG Assay Kit provides a simple, reproducible, and sensitive tool for assaying TGs in plasma and serum. The assay is initiated with the enzymatic hydrolysis of the TGs by lipase to produce glycerol and free fatty acids. The glycerol released is subsequently measured by a coupled enzymatic reaction system with a colorimetric readout at 540 nm.

96 wells



Troglitazone 71750

[97322-87-7] Resulin™

MF: C₂₄H₂₇NO₅S **FW:** 441.5 **Purity:** ≥98%A crystalline solid **Stability:** ≥1 year at -20°C

Summary: Troglitazone is a potent and selective PPARγ agonist. The EC₅₀ values for transactivation of human and murine PPARγ in a cell-based assay are 0.55 and 0.78 μM, respectively. In the same assay system, no activation of PPARα and PPARδ was observed at concentrations up to 10 μM.

5 mg
10 mg
50 mg
100 mg

5-[[4-[(3,4-dihydro-6-hydroxy-2,5,7,8-tetramethyl-2H-1-benzopyran-2-yl)methoxy]phenyl]methyl]-2,4-thiazolidinedione

U-44069 16440

[56985-32-1] 9,11-epoxymethano PGH₂, 9,11-dideoxy-9α,11α-epoxymethano PGF_{2α}**MF:** C₂₁H₃₄O₄ **FW:** 350.5 **Purity:** ≥98%*A solution in methyl acetate **Stability:** ≥2 years at -20°C

Summary: U-44069 is a stable analog of the endoperoxide PGH₂, and a TP receptor agonist. It stimulates shape change in human platelets without a measurable increase in [Ca²⁺] with an EC₅₀ value of 1.8 nM. U-44069 has an EC₅₀ value of 3 μM and 54 nM for platelet aggregation and phosphatidate formation in human platelets, respectively.

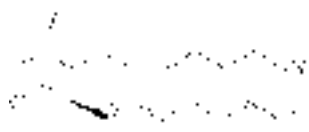
1 mg
5 mg
10 mg
50 mg

9,11-dideoxy-9α,11α-epoxymethano-prosta-5Z,13E-dien-1-oic acid

U-46619 16450

[56985-40-1] 9,11-dideoxy-9α,11α-methanoepoxy PGF_{2α}**MF:** C₂₁H₃₄O₄ **FW:** 350.5 **Purity:** ≥98%*A solution in methyl acetate **Stability:** ≥2 years at -20°C

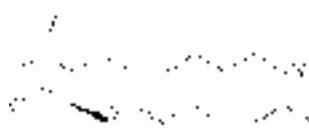
Summary: U-46619 is a stable analog of the endoperoxide PGH₂, and a TP receptor agonist. It exhibits properties similar to TXA₂, causing platelet shape change and aggregation, and contraction of vascular smooth muscle. Mean EC₅₀ values for shape change in human, rat, and rabbit platelets are 4.8, 6.0, and 7.3 nM respectively, and for aggregation are 82, 145, and 65 nM, respectively.

1 mg
5 mg
10 mg
50 mg

9,11-dideoxy-9α,11α-methanoepoxy-prosta-5Z,13E-dien-1-oic acid

Δ¹⁷-U-46619 16460**MF:** C₂₁H₃₂O₄ **FW:** 348.5 **Purity:** ≥98%*A solution in methyl acetate **Stability:** ≥2 years at -20°C

Summary: Δ¹⁷-U-46619 is the first direct, stable analog of TXA₃ ever synthesized. As TXA₃ is a metabolite of EPA, Δ¹⁷-U-46619 can be used to examine the effects ω-3 fatty acids might have at the receptor level, particularly the TP and IP receptors.

50 μg
100 μg
500 μg
1 mg

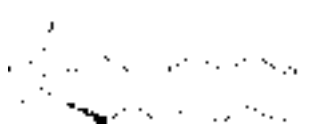
9,11-dideoxy-9α,11α-methanoepoxy-prosta-5Z,13E,17Z-trien-1-oic acid

U-51605 16465

[64192-56-9]

MF: C₂₀H₃₂N₂O₂ **FW:** 332.5 **Purity:** ≥98%*A solution in methyl acetate **Stability:** ≥1 year at -20°C

Summary: U-51605 is a stable analog of the endoperoxide PGH₂. It is an inhibitor of both PGI and TX synthases with more selectivity towards PGI synthase. In human foreskin fibroblasts, U-51605 inhibits PGI synthase at a concentration of 2.8 μM, whereas, human platelet TX synthase is inhibited at a concentration of 5.6 μM. U-51605 (0.1 μg/ml) also inhibits PGH₂-induced human platelet aggregation.

500 μg
1 mg
5 mg
10 mg

9α,11α-azoprosta-5Z,13E-dien-1-oic acid

NEW U-18666A 10009085

[3039-71-2]

MF: C₂₅H₄₁NO₂ • HCl **FW:** 424.1 **Purity:** ≥95%A crystalline solid **Stability:** ≥2 years at -20°C

Summary: U-18666A is a cell permeable drug that inhibits cholesterol trafficking. It inhibits cholesterol transport from late endosomes/lysosomes to the ER, but not cholesterol transport to the plasma membrane as demonstrated in many cell types including macrophages, primary cortical neurons and primary fibroblasts. In macrophages, micromolar concentrations of U-18666A inhibit multiple pathways of cholesterol trafficking from late endosomes, whereas nanomolar concentrations impair cholesterol trafficking to the ER, a response similar to that found in Neimann-Pick disease type C (NPC). U-18666A inhibits oxidosqualene cyclase at high (>0.5 mM) concentrations and oral doses (10 mg/kg) induces cataracts in rats.

5 mg
10 mg
25 mg
50 mg

3β-[2-(diethylamino)ethoxy]-androst-5-en-17-one, monohydrochloride

Vasoactive Eicosanoid HPLC Mixture 10003

Purity: ≥98% for each compoundA solution in methyl acetate **Stability:** ≥6 months at -20°C

Summary: This mixture contains the characteristic metabolites of both PGI₂ and TXA₂. Contents: TXB₂, 11-dehydro TXB₂, 6-keto PGF_{1α}, 2,3-dinor-6-keto PGF_{1α} (100 μg each), and 12(S)-HHTrE (5 μg).

1 ea

Wy 14643 70730

[50892-23-4] Pirinixic acid

MF: C₁₄H₁₄ClN₃O₂S **FW:** 323.8 **Purity:** ≥98%A crystalline solid **Stability:** ≥2 years at -20°C

Summary: Wy 14643 is a PPAR activator. Although this compound is primarily an activator of PPARα, it activates PPARγ as well. The potency of Wy 14643 as an activator of PPARα is species dependent, with receptor activation occurring at concentrations as low as 0.1 μM in the mouse compared to 10 μM in *Xenopus*.

5 mg
10 mg
50 mg
250 mg

[[4-chloro-6-[(2,3-dimethylphenyl)amino]-2-pyrimidinyl]thio]-acetic acid

*All 5-cis 2-series PGs (those containing a 5,6-double bond) will contain a small amount of the 5-trans isomer. This isomer is generally undetectable using normal phase silica columns and plates, but may be resolved using RP-HPLC. The purity for all such 2-series PGs excludes the 1-3% trans isomer which will generally be present.

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Triglyceride Assay Kit	52	320500	15	10007820	27
TRK 100 (Beraprost (sodium salt))	8	360070	24	10007850	43
Troglitazone	52	360402	32	10007875	11
U-44069	52	360600	35	10007893	26
U-46619	52	360603	34	10007938	17
Δ ¹⁷ -U-46619	52	360640	43	10007941	40
U-51605	52	360715	51	10008463	7
U-61431F (Ciprostene (calcium salt))	15	360881	33	10008492	6
U-63287 (Ciglitazone)	15	360900	35	10008613	22
U-63557A (Furegrelate (sodium salt))	19	515121	42	10008811	36
U-18666A	53	515211	42	10008826	42
Umbellatine (Berberine)	8	515211.1	42	10008878	37
Vasoactive Eicosanoid HPLC Mixture	53	519031	49	10009017	11
Wy 14643	53	519031.1	49	10009085	53
		519051	51	10009088	40
		519051.1	51	10009145	9
		519501	50	10009239	45
		519501.1	50	10009266	45
		520111	26	10009323	25
		520111.1	26	10009324	25
		520411	27	10009368	51
		520411.1	27	10009547	17
		520501	16	10009549	18
		520501.1	16	10009551	19
		583151	17	10009567	36
		589301	7	10009568	40
		589401	8	10009581	36
		589451	6	10009676	18
		705002	27	10009749	48
		705003	27	10009751	7
		760901	34	10009779	14
		10004023	50	10009880	22
		10004111	16	10009926	36
		10004380	35	10009987	40
		10004452	51	10010019	17
		10004944	18	10010020	17
		10005090	6	10010081	34
		10005091	6	10010153	29
		10005186	10	10010206	9
		10005191	18	10010231	18
		10005200	17	10010303	52
		10005263	51	10010337	19
		10005292	8	10010338	32
		10005368	19	10010339	32
		10005385	33	10010340	33
		10005386	50	10010341	33
		10005518	24	10010342	41
		10005519	24	10010343	41
		10005745	15	10010344	45
		10005913	10	10010345	45
		10006084	22	10010364	19
		10006217	44	10010463	19
		10006247	40	10010551	7
		10006248	18	10011012	7
		10006270	44	10011119	32
		10006371	23	10011125	25
		10006372	23	10011229	25
		10006409	6	10011236	10
		10006415	44		

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