Compound Summary for CID 2244

Aspirin

Cite this Record

STRUCTURE VENDORS DRUG INFO PHARMACOLOGY LITERATURE PATENTS BIOACTIVITIES

PubChem CID:	2244
Chemical Names:	Aspirin; ACETYLSALICYLIC ACID; 50-78-2; 2-Acetoxybenzoic acid; 2-(Acetyloxy)benzoic acid; O-Acetoxybenzoic acid More...
Molecular Formula:	$C_9H_8O_4$ or $CH_3COOC_6H_4COOH$ or $HC_9H_7O_4$
Molecular Weight:	180.159 g/mol
InChI Key:	BSYNRYMUTXBXSQ-UHFFFAOYSA-N
Drug Information:	Drug Indication Therapeutic Uses Clinical Trials FDA Orange Book FDA UNII
Safety Summary:	Laboratory Chemical Safety Summary (LCSS)

Aspirin is the prototypical analgesic used in the treatment of mild to moderate pain. It has anti-inflammatory and antipyretic properties and acts as an inhibitor of cyclooxygenase which results in the inhibition of the biosynthesis of prostaglandins. Aspirin also inhibits platelet aggregation and is used in the prevention of arterial and venous thrombosis. (From Martindale, The Extra Pharmacopoeia, 30th ed, p5)

from MeSH

Aspirin is a Nonsteroidal Anti-inflammatory Drug and Platelet Aggregation Inhibitor. The mechanism of action of aspirin is as a Cyclooxygenase Inhibitor. The physiologic effect of aspirin is by means of Decreased Prostaglandin Production and Decreased Platelet Aggregation. The chemical classification of aspirin is Nonsteroidal Anti-inflammatory Compounds.

FDA Pharmacology Summary from FDA Pharm Classes

Aspirin is an orally administered non-steroidal antiinflammatory agent. Acetylsalicylic acid binds to and acetylates serine residues in cyclooxygenases, resulting in decreased synthesis of prostaglandin, platelet aggregation, and inflammation. This agent exhibits analgesic, antipyretic, and anticoagulant properties.

Pharmacology from NCIt

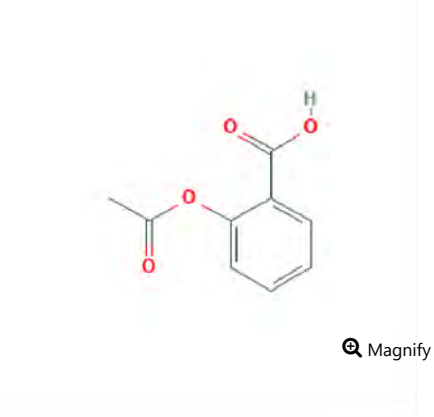
PUBCHEM > COMPOUND > ASPIRIN

Modify Date: 2019-01-12; Create Date: 2004-09-16

Contents

1 2D Structure

Search Download Get Image



Magnify

from PubChem

2 3D Conformer

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Show Hydrogens Show Atoms Animate

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3 Names and Identifiers

3.1 Computed Descriptors

3.1.1 IUPAC Name

2-acetyloxybenzoic acid

from PubChem

3.1.2 InChI

InChI=1S/C9H8O4/c1-6(10)13-8-5-3-2-4-7(8)9(11)12/h2-5H,1H3,(H,11,12)

from PubChem

3.1.3 InChI Key

BSYNRYMUTXBXSQ-UHFFFAOYSA-N

from PubChem

3.1.4 Canonical SMILES

CC(=O)OC1=CC=CC=C1C(=O)O

from PubChem

3.2 Molecular Formula

$C_9H_8O_4$
$CH_3COOC_6H_4COOH$

from ILO-ICSC

$HC_9H_7O_4$

from Wikipedia

$C_9H_8O_4$

from Wikipedia, PubChem

3.3 Other Identifiers

3.3.1 CAS

50-78-2

from CAMEO Chemicals, ChemIDplus, DTP/NCI, DrugBank, EPA Chemicals under the TSCA, EPA DSStox, European Chemicals Agency (ECHA), Human Metabolome Datab...

3.3.2 EC Number

200-064-1

from European Chemicals Agency (ECHA)

3.3.3 ICSC Number

0822

from ILO-ICSC

3.3.4 NSC Number

406186

from DTP/NCI

755899

from DTP/NCI

27223

from DTP/NCI

3.3.5 RTECS Number

VO0700000

from The National Institute for Occupational Safety and Health (NIOSH)

3.3.6 UN Number

2811

from CAMEO Chemicals

3.3.7 UNII

R16CO5Y76E

from FDA/SPL Indexing Data

3.3.8 Wikipedia

Title	acetylsalicylic acid
Title	aspirin
Title	acetylsalicylate
Description	chemical compound
Title	Bufferin
Description	chemical compound

from Wikipedia

3.4 Synonyms

3.4.1 MeSH Entry Terms

1. 2-(Acetyloxy)benzoic Acid
2. Acetylsalicylic Acid
3. Acetysal
4. Acid, Acetylsalicylic
5. Acylpyrin
6. Aloxiprimum
7. Aspirin
8. Colfarit
9. Dispril
10. Easprin
11. Ecotrin
12. Endosprin
13. Magnecyl
14. Micristin
15. Polopirin
16. Polopiryna
17. Solprin
18. Solupsan
19. Zorprin

from MeSH

3.4.2 Depositor-Supplied Synonyms

1. aspirin
2. ACETYLSALICYLIC ACID
3. 50-78-2
4. 2-Acetoxybenzoic acid
5. 2-(Acetyloxy)benzoic acid
6. o-Acetoxybenzoic acid
7. Acylpyrin
8. O-Acetylsalicylic acid
9. Polopiryna
10. Easprin
11. Ecotrin
12. Acetylsalicylate
13. Acenterine
14. Acetophen
15. Acetosal
16. Colfarit
17. Salicylic acid acetate
18. o-Carboxyphenyl acetate
19. Enterosarein
20. Aceticyl
21. Acetonyl
22. Acetosalin
23. Acetylin
24. Aspergum
25. Aspirdrops
26. Benaspir
27. Micristin
28. Pharmacin
29. Premaspin
30. Salcetogen
31. Temperal
32. Ecolen
33. Empirin
34. Endydol
35. Rhodine
36. Saletin
37. Rheumintabletten
38. Solprin acid
39. Acidum acetylsalicylicum
40. Acetisal
41. Acetylsal
42. Aspirine
43. Bialpirina
44. Bialpirinia
45. Bufferin
46. Claradin
47. Clariprin
48. Entericin
49. Enterophen
50. Enterosarine
51. Globentyl
52. Measurin
53. Neuronika
54. Salacetin
55. Solpyron
56. Acesal
57. Acisal
58. Asagran
59. Asteric
60. Cemirit
61. Decaten
62. Duramax
63. Extren
64. Globoid
65. Helicon
66. Idragin
67. Levius
68. Pirseal
69. Rhonal
70. Solfrin

from PubChem

4 Chemical and Physical Properties

4.1 Computed Properties

Property Name	Property Value
Molecular Weight	180.159 g/mol
Hydrogen Bond Donor Count	1
Hydrogen Bond Acceptor Count	4
Rotatable Bond Count	3
Complexity	212
Topological Polar Surface Area	63.6 A^2
Monoisotopic Mass	180.042 g/mol
Exact Mass	180.042 g/mol
XLogP3	1.2
Compound Is Canonicalized	true
Formal Charge	0
Heavy Atom Count	13
Defined Atom Stereocenter Count	0
Undefined Atom Stereocenter Count	0
Defined Bond Stereocenter Count	0
Undefined Bond Stereocenter Count	0
Isotope Atom Count	0
Covalently-Bonded Unit Count	1

from PubChem

4.2 Experimental Properties

4.2.1 Physical Description

PHYSICAL DESCRIPTION: Odorless white crystals or crystalline powder with a slightly bitter taste. (NTP, 1992)

from CAMEO Chemicals

Withheld

from EPA Chemicals under the TSCA

Solid

from Human Metabolome Database (HMDB)

COLOURLESS-TO-WHITE CRYSTALS OR WHITE CRYSTALLINE POWDER WITH CHARACTERISTIC ODOUR.

from ILO-ICSC

Odorless, colorless to white, crystal-line powder.

from OSHA Occupational Chemical DB

Odorless, colorless to white, crystal-line powder. [aspirin] [Note: Develops the vinegar-like odor of acetic acid on contact with moisture.]

from The National Institute for Occupational Safety and Health (NIOSH)

4.2.2 Color

Monoclinic tablets or needle-like crystals

O'Neil, M.J. (ed.). The Merck Index - An Encyclopedia of Chemicals, Drugs, and Biologicals. Whitehouse Station, NJ: Merck and Co., Inc., 2006., p. 140

from HSDB

Colorless to white, crystalline powder.

NIOSH. NIOSH Pocket Guide to Chemical Hazards & Other Databases CD-ROM. Department of Health & Human Services, Centers for Disease Prevention & Control. National Institute for Occupational Safety & Health. DHHS (NIOSH) Publication No. 2005-151 (2005)

from HSDB

4.2.3 Odor

Odorless, but in moist air it is gradually hydrolyzed and acquires odor of acetic acid

O'Neil, M.J. (ed.). The Merck Index - An Encyclopedia of Chemicals, Drugs, and Biologicals. Whitehouse Station, NJ: Merck and Co., Inc., 2006., p. 140

from HSDB

Odorless [Note: Develops the vinegar-like odor of acetic acid on contact with moisture].

NIOSH. NIOSH Pocket Guide to Chemical Hazards & Other Databases CD-ROM. Department of Health & Human Services, Centers for Disease Prevention & Control. National Institute for Occupational Safety & Health. DHHS (NIOSH) Publication No. 2005-151 (2005)

from HSDB

4.2.4 Boiling Point

284° F at 760 mm Hg (decomposes) (NTP, 1992)

from CAMEO Chemicals

140 °C

from DrugBank

284°F (decomposes)

from OSHA Occupational Chemical DB

284°F (Decomposes)

from The National Institute for Occupational Safety and Health (NIOSH)

4.2.5 Melting Point

275° F (NTP, 1992)

from CAMEO Chemicals

135 °C

PhysProp

from DrugBank

135 deg C (rapid heating)

O'Neil, M.J. (ed.). The Merck Index - An Encyclopedia of Chemicals, Drugs, and Biologicals. Whitehouse Station, NJ: Merck and Co., Inc., 2006., p. 140

from HSDB

135 °C

from Human Metabolome Database (HMDB)

135°C

from ILO-ICSC

275°F

from OSHA Occupational Chemical DB, The National Institute for Occupational Safety and Health (NIOSH)

4.2.6 Flash Point

482° F (NTP, 1992)

from CAMEO Chemicals

4.2.7 Solubility

less than 1 mg/mL at 73° F (NTP, 1992)

from CAMEO Chemicals

Water Solubility

4600 mg/L (at 25 °C)

YALKOWSKY,SH & DANNENFELSER,RM (1992)

from DrugBank

1 g sol in: 300 mL water at 25 deg C, 100 mL water at 37 deg C, 5 mL alcohol, 17 mL chloroform, 10-15 mL ether; less sol in anhydrous ether

O'Neil, M.J. (ed.). The Merck Index - An Encyclopedia of Chemicals, Drugs, and Biologicals. Whitehouse Station, NJ: Merck and Co., Inc., 2006., p. 140

from HSDB

In water, 4,600 mg/L at 25 deg C

Yalkowsky SH, Dannenfelser RM; Aquasol Database of Aqueous Solubility. Version 5. College of Pharmacy, University of Arizona - Tucson, AZ (1992)

▸ *from HSDB*

Solubility in water, g/100ml at 15°C: 0.25 (poor)

▸ *from ILO-ICSC*

(77°F): 0.3%

▸ *from The National Institute for Occupational Safety and Health (NIOSH)*

4.2.8 Density

1.4 (NTP, 1992)

▸ *from CAMEO Chemicals*

1.40

O'Neil, M.J. (ed.). The Merck Index - An Encyclopedia of Chemicals, Drugs, and Biologicals. Whitehouse Station, NJ: Merck and Co., Inc., 2006., p. 140

▸ *from HSDB*

1.4 g/cm^3

▸ *from ILO-ICSC*

1.35

▸ *from OSHA Occupational Chemical DB, The National Institute for Occupational Safety and Health (NIOSH)*

4.2.9 Vapor Pressure

0 mm Hg (approx) (NIOSH, 2016)

▸ *from CAMEO Chemicals*

2.52X10-5 mm Hg at 25 deg C (calc)

Eisenreich SJ et al; Environ Sci Technol 15: 30-8 (1981)

▸ *from HSDB*

Vapour Pressure

Vapour pressure, Pa at 25°C: ~ 0.004

▸ *from ILO-ICSC*

0 mmHg (approx)

▸ *from OSHA Occupational Chemical DB, The National Institute for Occupational Safety and Health (NIOSH)*

4.2.10 LogP

1.19

HANSCH,C ET AL. (1995)

▸ *from DrugBank, Human Metabolome Database (HMDB)*

log Kow = 1.19

Hansch, C., Leo, A., D. Hoekman. Exploring QSAR - Hydrophobic, Electronic, and Steric Constants. Washington, DC: American Chemical Society., 1995., p. 54

▸ *from HSDB*

1.19

▸ *from ILO-ICSC*

4.2.11 Stability

STABLE IN DRY AIR; IN MOIST AIR IT IS GRADUALLY HYDROLYZED INTO SALICYLIC AND ACETIC ACIDS

Budavari, S. (ed.). The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals. Rahway, NJ: Merck and Co., Inc., 1989., p. 134

▸ *from HSDB*

In aqueous solutions, aspirin is most stable at a pH of 2-3, less stable at a pH of 4-8, and least stable at a pH less than 2 or greater than 8. In a saturated aqueous solution at a pH of 5-7, aspirin is almost completely hydrolyzed within 1 week at 25 deg C.

McEvoy, G.K. (ed.). American Hospital Formulary Service. AHFS Drug Information. American Society of Health-System Pharmacists, Bethesda, MD. 2007., p. 2037

▸ *from HSDB*

4.2.12 Decomposition

When heated to decomposition it emits acrid smoke and fumes.

Lewis, R.J. Sr. (ed) Sax's Dangerous Properties of Industrial Materials. 11th Edition. Wiley-Interscience, Wiley & Sons, Inc. Hoboken, NJ. 2004., p. 57

from HSDB

140°C

from ILO-ICSC

4.2.13 Caco2 Permeability

-5.06

ADME Research, USCD

from DrugBank

4.2.14 pKa

3.49 (at 25 °C)

MERCK INDEX (1983)

from DrugBank

4.2.15 Dissociation Constants

pKa = 3.49 at 25 deg C

O'Neil, M.J. (ed.). The Merck Index - An Encyclopedia of Chemicals, Drugs, and Biologicals. Whitehouse Station, NJ: Merck and Co., Inc., 2006., p. 140

from HSDB

4.2.16 Kovats Retention Index

Standard non-polar	1270, 1315, 1309, 1309

from NIST

4.3 Crystal Structures

Crystal Structures: 1 of 18 (CCDC Number)	
CCDC Number	617840
Crystal Structure Data	DOI:10.5517/ccnqxbl
Thumbnail (Generated by CCDC using JSmol)	CLICK TO LOAD...
Associated Article	DOI:10.1002/anie.200603373

from The Cambridge Structural Database

Crystal Structures: 2 of 18 (CCDC Number)	
CCDC Number	820697
Crystal Structure Data	DOI:10.5517/ccwk03j

Crystal Structures: 2 of 18 (CCDC Number)	
Thumbnail (Generated by CCDC using JSmol)	CLICK TO LOAD...
Associated Article	DOI:10.1039/c1sc00430a

from The Cambridge Structural Database

Crystal Structures: 3 of 18 (CCDC Number)	
CCDC Number	904406
Crystal Structure Data	DOI:10.5517/cczc3dt
Thumbnail (Generated by CCDC using JSmol)	CLICK TO LOAD...
Associated Article	DOI:10.1021/cg300269n

from The Cambridge Structural Database

View All 18 Crystal Structures

4.4 Spectral Properties

Intense mass spectral peaks: 92 m/z, 120 m/z, 138 m/z, 180 m/z

Pfleger, K., H. Maurer and A. Weber. Mass Spectral and GC Data of Drugs, Poisons and their Metabolites. Parts I and II. Mass Spectra Indexes. Weinheim, Federal Republic of Germany. 1985., p. 256

from HSDB

MAX ABSORPTION (ETHER): 275 NM (LOG E= 3.11); SADTLER REF NUMBER: 51 (IR, PRISM)

Weast, R.C. (ed.). Handbook of Chemistry and Physics. 57th ed. Cleveland: CRC Press Inc., 1976., p. C-191

from HSDB

UV max: (0.1 N H2SO4): 229 (E(1%)(cm) 484); (CHCl3): 277 (E(1%)(cm) 68)

O'Neil, M.J. (ed.). The Merck Index - An Encyclopedia of Chemicals, Drugs, and Biologicals. Whitehouse Station, NJ: Merck and Co., Inc., 2006., p. 140

from HSDB

IR: 5063 (Coblentz Society Spectral Collection)

Lide, D.R., G.W.A. Milne (eds.). Handbook of Data on Organic Compounds. Volume I. 3rd ed. CRC Press, Inc. Boca Raton ,FL. 1994., p. V2: 1347

from HSDB

UV: 25 (Sadtler Research Laboratories Spectral Collection)

Lide, D.R., G.W.A. Milne (eds.). Handbook of Data on Organic Compounds. Volume I. 3rd ed. CRC Press, Inc. Boca Raton ,FL. 1994., p. V2: 1347

from HSDB

NMR: 18001 (Sadtler Research Laboratories Spectral Collection)

Lide, D.R., G.W.A. Milne (eds.). Handbook of Data on Organic Compounds. Volume I. 3rd ed. CRC Press, Inc. Boca Raton ,FL. 1994., p. V2: 1347

from HSDB

Raman: 417 (Sadtler Research Laboratories Spectral Collection)

Lide, D.R., G.W.A. Milne (eds.). Handbook of Data on Organic Compounds. Volume I. 3rd ed. CRC Press, Inc. Boca Raton ,FL. 1994., p. V2: 1347

from HSDB

MASS: 23813 (NIST/EPA/MSDC Mass Spectral database, 1990 version)

Lide, D.R., G.W.A. Milne (eds.). Handbook of Data on Organic Compounds. Volume I. 3rd ed. CRC Press, Inc. Boca Raton ,FL. 1994., p. V2: 1347

from HSDB

4.4.1 Infrared Spectra

Infrared Spectra: 1 of 2 (FTIR Spectra)	
Technique	KBr WAFER
Source of Sample	MCB MANUFACTURING CHEMISTS, NORWOOD, OHIO
Copyright	Copyright © 1980, 1981-2018 Bio-Rad Laboratories, Inc. All Rights Reserved.
Thumbnail	CLICK TO LOAD...

from SpectraBase

Infrared Spectra: 2 of 2 (ATR-IR Spectra)	
Instrument Name	Bio-Rad FTS
Technique	ATR-Neat (DuraSamplIR II)
Source of Spectrum	Forensic Spectral Research
Source of Sample	Sigma-Aldrich Inc.
Catalog Number	A3160-1VL
Lot Number	108K8711(traceable to USP 32 Lot H)
Copyright	Copyright © 2012-2018 Bio-Rad Laboratories, Inc. All Rights Reserved.
Thumbnail	CLICK TO LOAD...

from SpectraBase

4.4.2 1D NMR Spectra

1D NMR Spectra: 1 of 8 (1H NMR Spectra)	
1H NMR Spectra	1. 1D NMR Spectrum 1776 - 1H NMR Spectrum (HMDB0001879) 2. 1D NMR Spectrum 2407 - 1H NMR Spectrum (HMDB0001879)
13C NMR Spectra	1D NMR Spectrum 3097 - 13C NMR Spectrum (HMDB0001879)

from Human Metabolome Database (HMDB)

1D NMR Spectra: 2 of 8 (13C NMR Spectra)	
Copyright	Copyright © 2016 W. Robien, Inst. of Org. Chem., Univ. of Vienna. All Rights Reserved.
Thumbnail	CLICK TO LOAD...

from SpectraBase

1D NMR Spectra: 3 of 8 (13C NMR Spectra)	
Copyright	Copyright © 2016 W. Robien, Inst. of Org. Chem., Univ. of Vienna. All Rights Reserved.
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from SpectraBase

View All 8 1D NMR Spectra

4.4.3 2D NMR Spectra

2D NMR Spectra: 1 of 1 (1H-13C NMR Spectra)	
1H-13C NMR Spectra	2D NMR Spectrum 1716 - [1H,13C] 2D NMR Spectrum (HMDB0001879)

from Human Metabolome Database (HMDB)

4.4.4 Mass Spectrometry

4.4.4.1 General MS

General MS: 1 of 10 (MS)	
MoNA ID	FiehnHILIC002889
MS Category	Experimental
MS Type	Chromatography identified as LC-MS
MS Level	MS2
Precursor Type	[M-H]-
precursor m/z	179.0338
Instrument	SCIEX TripleTOF 6600
Instrument Type	LC-ESI-QTOF
Ionization Mode	negative
Collision Energy	35 eV
Splash	splash10-000f-9500000000-f60e6b8b20c1b43831b1
Thumbnail	CLICK TO LOAD...
Submitter	Megan Showalter, University of California, Davis

from MassBank of North America (MoNA)

General MS: 2 of 10 (MS)	
MoNA ID	JP003149
MS Category	Experimental
MS Type	Chromatography identified as GC-MS
MS Level	MS1

General MS: 2 of 10 (MS)	
Instrument	Unknown
Instrument Type	CI-B
Ionization Mode	positive
Splash	splash10-00di-0900000000-113943b65024522c1712
Thumbnail	CLICK TO LOAD...
Submitter	Kimito Funatsu, Graduate School of Engineering, The University of Tokyo

from MassBank of North America (MoNA)

General MS: 3 of 10 (MS)	
MoNA ID	FiehnHILIC001404
MS Category	Experimental
MS Type	Chromatography identified as LC-MS
MS Level	MS2
Precursor Type	[M-H]-
precursor m/z	179.0342
Instrument	Thermo Q Exactive HF
Instrument Type	LC-ESI-QFT
Ionization Mode	negative
Collision Energy	HCD (NCE 20-30-40%)
Splash	splash10-000i-4900000000-b39247c3e51c069673e8
Thumbnail	CLICK TO LOAD...
Submitter	Megan Showalter, University of California, Davis

from MassBank of North America (MoNA)

View All 10 General MS

4.4.4.2 GC-MS

1. GC-MS Spectrum 786
2. GC-MS Spectrum 1003
3. GC-MS Spectrum 7757
4. GC-MS Spectrum 27401
5. GC-MS Spectrum 27421
6. GC-MS Spectrum 30117
7. GC-MS Spectrum 30469
8. GC-MS Spectrum 31361
9. GC-MS Spectrum 32298
10. GC-MS Spectrum 38166

from Human Metabolome Database (HMDB)

1 of 5	
NIST Number	250572
Library	Main library
Total Peaks	74
m/z Top Peak	120
m/z 2nd Highest	138
m/z 3rd Highest	43
Thumbnail	CLICK TO LOAD...

from NIST

4.4.4.3 MS-MS

1. MS-MS Spectrum 1777
2. MS-MS Spectrum 1778
3. MS-MS Spectrum 1779
4. MS-MS Spectrum 5543
5. MS-MS Spectrum 5544
6. MS-MS Spectrum 255762
7. MS-MS Spectrum 255763
8. MS-MS Spectrum 255764
9. MS-MS Spectrum 275703
10. MS-MS Spectrum 275704
11. MS-MS Spectrum 275705
12. MS-MS Spectrum 436679
13. MS-MS Spectrum 436680
14. MS-MS Spectrum 436681
15. MS-MS Spectrum 436682
16. MS-MS Spectrum 436683
17. MS-MS Spectrum 436684
18. MS-MS Spectrum 436685
19. MS-MS Spectrum 436686
20. MS-MS Spectrum 436687
21. MS-MS Spectrum 444374
22. MS-MS Spectrum 444375
23. MS-MS Spectrum 444376
24. MS-MS Spectrum 444377
25. MS-MS Spectrum 444378

from Human Metabolome Database (HMDB)

MS-MS: 1 of 1 (MS-MS Fields)	
NIST Number	1184852
Instrument Type	IT/ion trap
Collision Energy	0
Spectrum Type	MS2
Precursor Type	[M-H]-
Precursor m/z	179.035
Total Peaks	20
m/z Top Peak	134.9
m/z 2nd Highest	134
m/z 3rd Highest	151
Thumbnail	CLICK TO LOAD...

from NIST

4.4.4.4 EI-MS

EI-MS Spectrum 628

from Human Metabolome Database (HMDB)

4.4.5 Other Spectra

Other Spectra: 1 of 1 (Raman Spectra)	
Technique	FT-Raman
Source of Spectrum	Forensic Spectral Research
Source of Sample	Sigma-Aldrich Inc.
Catalog Number	A3160
Lot Number	108K8711(traceable to USP 32 Lot H)
Copyright	Copyright © 2012-2018 Bio-Rad Laboratories, Inc. All Rights Reserved.
Thumbnail	CLICK TO LOAD...

from SpectraBase

5 Related Records

CLICK TO LOAD...

from NCBI

5.1 Related Compounds with Annotation

CLICK TO LOAD...

from PubChem

5.2 Related Compounds

Same Connectivity	13 records
Same Parent, Connectivity	126 records
Same Parent, Exact	114 records
Mixtures, Components, and Neutralized Forms	644 records
Similar Compounds	5167 records
Similar Conformers	11509 records

from PubChem

5.3 Substances

5.3.1 Related Substances

All	1864 records
Same	306 records
Mixture	1558 records

from PubChem

5.3.2 Substances by Category

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from PubChem

5.4 Entrez Crosslinks

PubMed	26424 records
Protein Structures	6 records
Taxonomy	9 records
OMIM	83 records
Gene	768 records

from PubChem

6 Chemical Vendors

CLICK TO LOAD...

from PubChem

7 Drug and Medication Information

7.1 Drug Indication

For use in the temporary relief of various forms of pain, inflammation associated with various conditions (including rheumatoid arthritis, juvenile rheumatoid arthritis, systemic lupus erythematosus, osteoarthritis, and ankylosing spondylitis), and is also used to reduce the risk of death and/or nonfatal myocardial infarction in patients with a previous infarction or unstable angina pectoris.

from DrugBank

FDA Label

from DrugBank

Treatment of polycythemia vera

from EU Community Register of Medicinal Products

7.2 Drug Classes

Antithrombotic Agents, Antiinflammatory Agents, Salicylates

from LiverTox

7.3 FDA Orange Book

7.3.1 Prescription Drug Products

Prescription Drug Products: 1 of 15 (RX Drug Ingredient)	
Drug Ingredient	ASPIRIN; CAFFEINE; ORPHENADRINE CITRATE
Proprietary Name	ORPHENADRINE CITRATE, ASPIRIN, AND CAFFEINE
Applicant	SANDOZ (Application Number: A074654)

from FDA Orange Book

Prescription Drug Products: 2 of 15 (RX Drug Ingredient)	
Drug Ingredient	ASPIRIN; CARISOPRODOL; CODEINE PHOSPHATE
Proprietary Name	CARISOPRODOL, ASPIRIN AND CODEINE PHOSPHATE
Applicant	1. INGENUS PHARMS NJ (Application Number: A040860) 2. SANDOZ (Application Number: A040118)

from FDA Orange Book

Prescription Drug Products: 3 of 15 (RX Drug Ingredient)	
Drug Ingredient	ASPIRIN; BUTALBITAL; CAFFEINE; CODEINE PHOSPHATE
Proprietary Name	BUTALBITAL, ASPIRIN, CAFFEINE, AND CODEINE PHOSPHATE
Applicant	1. MAYNE PHARMA INC (Application Number: A203335) 2. NEXGEN PHARMA INC (Application Number: A075231) 3. STEVENS J (Application Number: A074951)

from FDA Orange Book

View All 15 Prescription Drug Products

7.3.2 Over-the-Counter Drug Products

Over-the-Counter Drug Products: 1 of 3 (OTC Drug Ingredient)	
Drug Ingredient	ASPIRIN
Proprietary Name	VAZALORE
Applicant	PLX PHARMA (Application Number: N203697. Patents: 8865187, 9101637, 9216150, 9226892, 9351984)

from FDA Orange Book

Over-the-Counter Drug Products: 2 of 3 (OTC Drug Ingredient)

Over-the-Counter Drug Products: 2 of 3 (OTC Drug Ingredient)	
Drug Ingredient	ACETAMINOPHEN; ASPIRIN; CAFFEINE
Proprietary Name	EXCEDRIN (MIGRAINE)
Applicant	GLAXOSMITHKLINE CONS (Application Number: N020802)

from FDA Orange Book

Over-the-Counter Drug Products: 3 of 3 (OTC Drug Ingredient)	
Drug Ingredient	ACETAMINOPHEN; ASPIRIN; CAFFEINE
Proprietary Name	ACETAMINOPHEN, ASPIRIN AND CAFFEINE
Applicant	PERRIGO (Application Number: A075794)

from FDA Orange Book

7.3.3 Discontinued Drug Products

Discontinued Drug Products: 1 of 50 (DISCN Drug Ingredient)	
Drug Ingredient	ASPIRIN; BUTALBITAL; CAFFEINE
Proprietary Name	BUTALBITAL, ASPIRIN AND CAFFEINE
Applicant	1. ACTAVIS ELIZABETH (Application Number: A086710) 2. FOSUN PHARMA (Application Number: A086398) 3. HALSEY (Application Number: A089448) 4. IVAX PHARMS (Application Number: A085441) 5. NOSTRUM LABS INC (Application Number: A078149) 6. PURACAP PHARM (Application Number: A087048) 7. QUANTUM PHARMICS (Application Number: A088972) 8. WATSON LABS (Application Number: A086231) 9. WATSON LABS (Application Number: A086237)

from FDA Orange Book

Discontinued Drug Products: 2 of 50 (DISCN Drug Ingredient)	
Drug Ingredient	ASPIRIN; BUTALBITAL; CAFFEINE
Proprietary Name	FIORINAL
Applicant	ALLERGAN SALES LLC (Application Number: N017534)

from FDA Orange Book

Discontinued Drug Products: 3 of 50 (DISCN Drug Ingredient)	
Drug Ingredient	ASPIRIN; BUTALBITAL; CAFFEINE
Proprietary Name	LANORINAL
Applicant	LANNETT (Application Number: A086986)

from FDA Orange Book

View All 50 Discontinued Drug Products

7.4 Drug Labels for Ingredients

Drug Labels for Ingredients: 1 of 11 (Label Title)	
Label Information	Total 97 labels
Drug Ingredient	ACETAMINOPHEN; ASPIRIN; CAFFEINE
NDC Code(s)	0113-0374-62, 0113-0374-71, 0113-0374-78, 0363-0159-12, 0363-0159-15, 0363-0159-29, 0363-0247-25, 0363-0374-62, 0363-0374-78, 0363-0374-82 ... total 184.
Packagers	7-Eleven; ARMY AND AIR FORCE EXCHANGE SERVICE; Acme United Corporation; Afassco Inc.; Alva-Amco Pharmacal Companies, Inc.; American Sales Company; Amerisource Bergen; AmerisourceBergen (Good Neighbor Pharmacy) 46122; BJWC; Better Living Brands, LLC ... total 60.

from DailyMed

Drug Labels for Ingredients: 2 of 11 (Label Title)	
Label Information	Total 4 labels
Drug Ingredient	ASPIRIN; CARISOPRODOL
NDC Code(s)	0185-0724-01, 0185-0724-05, 0185-0724-10, 21695-570-30, 21695-570-60, 23155-145-01, 64980-175-01, 64980-175-05
Packagers	Eon Labs, Inc.; Heritage Pharmaceuticals Inc.; Rebel Distributors Corp; Rising Pharmaceuticals, Inc.

from DailyMed

Drug Labels for Ingredients: 3 of 11 (Label Title)	
Label Information	Total 2 labels
Drug Ingredient	ASPIRIN; DIPYRIDAMOLE
NDC Code(s)	68382-618-14, 70771-1172-6
Packagers	Cadila Healthcare Limited; Zydus Pharmaceuticals (USA) Inc.

from DailyMed

View All 11 Drug Labels for Ingredients

7.5 Clinical Trials

Download

1 to 5 of 609 View More

Record ID	Title	Status	Phase
NCT00000151	Early Treatment Diabetic Retinopathy Study (ETDRS)	Completed	3
NCT00000152	Randomized Trial of Beta-Carotene and Macular Degeneration	Unknown status	3
NCT00000157	Randomized Trial of Aspirin and Cataracts in U.S. Physicians	Terminated	3
NCT00000161	Randomized Trials of Vitamin Supplements and Eye Disease	Unknown status	3
NCT00000469	Asymptomatic Carotid Artery Plaque Study (ACAPS)	Completed	2

from ClinicalTrials.gov

7.6 Therapeutic Uses

Anti-Inflammatory Agents, Non-Steroidal; Cyclooxygenase Inhibitors; Fibrinolytic Agents; Platelet Aggregation Inhibitors

National Library of Medicine's Medical Subject Headings online file (MeSH, 1999)

from HSDB

Salicylates are indicated to relieve myalgia, musculoskeletal pain, and other symptoms of nonrheumatic inflammatory conditions such as athletic injuries, bursitis, capsulitis, tendinitis, and nonspecific acute tenosynovitis. /Included in US product labeling/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2574

from HSDB

Salicylates are indicated for the symptomatic relief of acute and chronic rheumatoid arthritis, juvenile arthritis, osteoarthritis, and related rheumatic diseases. Aspirin is usually the first agent to be used and may be the drug of choice in patients able to tolerate prolonged therapy with high doses. These agents do not affect the progressive course of rheumatoid arthritis. Concurrent treatment with a glucocorticoid or a disease-modifying antirheumatic agent may be needed, depending on the condition being treated and patient response. /Included in US product labeling/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2574

from HSDB

Salicylates are also used to reduce arthritic complications associated with systemic lupus erythematosus. /Salicylates; NOT included in US product labeling/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2574

from HSDB

Salicylates are indicated to reduce fever and inflammation in rheumatic fever. However, they do not prevent cardiac or other complications associated with this condition. Sodium salicylate should be avoided in rheumatic fever if congestive cardiac complications are present because of its sodium content. Also, large doses of any salicylate should be avoided in rheumatic fever if severe carditis is present because of possible adverse cardiovascular effects. /Salicylates; Included in US product labeling/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2574

from HSDB

Aspirin is used in low doses to decrease the risk of thromboembolism following orthopedic (hip) surgery (especially total hip replacement) and in patients with arteriovenous shunts. Platelet aggregation inhibitors, although not as consistently effective as an anticoagulant or an anticoagulant plus dipyridamole, may provide some protection against the development of thromboembolic complications in patients with mechanical prosthetic heart valves. Therefore, administration of aspirin, alone or in combination with dipyridamole, may be considered if anticoagulation therapy is contraindicated for these patients. Patients with bioprosthetic cardiac valves who are in normal sinus rhythm generally do not require prolonged antithrombotic therapy, but long-term aspirin administration may be considered on an individual basis. Aspirin is also indicated, alone or in

combination with dipyridamole, to reduce the risk of thrombosis and/or reocclusion of saphenous vein aortocoronary bypass grafts following coronary bypass surgery. Aspirin is also indicated, alone or in combination with dipyridamole, to reduce the risk of thrombosis and/or reocclusion of prosthetic or saphenous vein femoral popliteal bypass grafts. Because the patient may be at risk for thromboembolic complications, including myocardial infarction and stroke, long-term aspirin therapy may also be indicated for maintaining patency following coronary or peripheral vascular angioplasty and for treating patients with peripheral vascular insufficiency caused by arteriosclerosis. Prolonged antithrombotic therapy is generally not needed to maintain vessel patency following vascular reconstruction procedures in high-flow, low-resistance arteries larger than 6 mm in diameter. However, long-term aspirin therapy may be indicated, because patients requiring such procedures may be at risk for other thrombotic complications. /NOT included in US product labeling/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2575

from HSDB

Aspirin is indicated in the treatment of men who have had transient brain ischemia due to fibrin platelet emboli to reduce the recurrence of transient ischemic attacks and the risk of stroke and death. /Included in US product labeling/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2575

from HSDB

Aspirin is also used in the treatment of women with transient brain ischemia due to fibrin platelet emboli. However, its efficacy in preventing stroke and death in female patients has not been established. Aspirin is also indicated in the treatment of patients with documented, unexplained transient ischemic attacks associated with mitral valve prolapse. However, if transient ischemic attacks continue to occur after an adequate trial of aspirin therapy, aspirin should be discontinued and an oral anticoagulant administered instead. Aspirin is also indicated to prevent initial or recurrent cerebrovascular embolism, transient ischemic attacks, and stroke following carotid endarterectomy. Aspirin is indicated in the treatment of patients who have had a completed thrombotic stroke, to prevent a recurrence. /NOT included in US product labeling/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2575

from HSDB

Aspirin is indicated for its anti-inflammatory, antipyretic, and antithrombotic effects in the treatment of Kawasaki disease (Kawasaki syndrome, mucocutaneous lymph node syndrome) in children. It reduces fever, relieves inflammation (e.g., lymphadenitis, mucositis, conjunctivitis, serositis), and may reduce the occurrence of cardiovascular complications. However, the combination of high-dose intravenous gamma globulin and aspirin has been shown to be more effective than aspirin alone in reducing the formation of coronary artery abnormalities. /NOT included in US product labeling/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2575

from HSDB

Thrombotic thrombocytopenic purpura is a severe multisystemic disorder of unknown origin. The association of relapsing thrombotic thrombocytopenic purpura with pregnancy is rare but well documented and high mortality rates of mothers and fetuses have been reported so far. Since the introduction of plasma therapy for treating the acute exacerbations of the disease, overall mortality rates have decreased significantly. It is now evident that the manifestations of the disease may reappear even after long disease free intervals and as many as a third of the recovering patients may develop a relapse. Presented are two thrombotic thrombocytopenic purpura patients with relapsing thrombotic thrombocytopenic purpura complicating their pregnancies. Prophylactic treatment with aspirin and dipyridamole during their last three successful pregnancies prevented or minimized the severity of thrombotic thrombocytopenic purpura relapses.
Abstract: PubMed

Ezra Y et al; Int J Gynaecol Obstet 29 (4): 359-63 (1989)

from HSDB

Low doses of aspirin (<100 mg daily) are used widely for their cardioprotective effects.

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 688

from HSDB

VET: In veterinary medicine, aspirin is used primarily for the relief of mild to moderate pain associated with musculoskeletal inflammation or osteoarthritis. ... Aspirin has been used in the treatment of laminitis in horses ... . In cats, aspirin may be used for its anti-platelet effects in thromboembolic disease ... .

Kahn, C.M. (Ed.); The Merck Veterinary Manual 9th ed. Merck & Co. Whitehouse Station, NJ. 2005, p. 2133

from HSDB

7.7 Drug Warning

Aspirin use may be associated with the development of Reye's syndrome in children and teenagers with acute febrile illnesses, especially influenza and varicella. It is recommended that salicylate therapy not be initiated in febrile pediatric or adolescent patients until after the presence of such an illness has been ruled out. Also, it is recommended that chronic salicylate therapy in these patients be discontinued if a fever occurs, and not resumed until it has been determined that an illness that may predispose to Reye's syndrome is not present or has run its course. Other forms of salicylate toxicity may also be more prevalent in pediatric patients, especially children who have a fever or are dehydrated.

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2577

from HSDB

Especially careful monitoring of the serum salicylate concentration is recommended in pediatric patients with Kawasaki disease. Absorption of aspirin is impaired during the early febrile stage of the disease; therapeutic anti-inflammatory plasma salicylate concentrations may be extremely difficult to achieve. Also, as the febrile stage passes, absorption is improved; salicylate toxicity may occur if dosage is not readjusted.

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2577

from HSDB

Requirements of Vitamin K may be increased in patients receiving high doses of salicylate. /Salicylate/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2578

from HSDB

IF RENAL FUNCTION IS COMPROMISED IN SALICYLATE INTOXICATION, POTASSIUM LOST FROM CELLS ACCUMULATES IN EXTRACELLULAR FLUID & POTASSIUM INTOXICATION MAY OCCUR.

Gilman, A.G., T.W. Rall, A.S. Nies and P. Taylor (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 8th ed. New York, NY. Pergamon Press, 1990., p. 650

from HSDB

Aspirin-induced gastric bleeding sometimes is painless, and if unrecognized may lead to iron-deficiency anemia. The daily ingestion of antiinflammatory doses of aspirin (4 or 5 g) results in an average fecal blood loss of between 3 and 8 mL per day, as compared with approximately 0.6 mL per day in untreated subjects. Gastroscopic examination of aspirin-treated subjects often reveals discrete ulcerative and hemorrhagic lesions of the gastric mucosa; in many cases, multiple hemorrhagic lesions with sharply demarcated areas of focal necrosis are observed.

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 688

▸ *from HSDB*

Salicylates can cause hepatic injury, usually in patients treated with high doses of salicylates that result in plasma concentrations of more than 150 g/ml. The injury is not an acute effect; rather, the onset characteristically occurs after several months of treatment. The majority of cases occur in patients with connective tissue disorders. There usually are no symptoms, simply an increase in serum levels of hepatic transaminases, but some patients note right upper quadrant abdominal discomfort and tenderness. Overt jaundice is uncommon. The injury usually is reversible upon discontinuation of salicylates. However, the use of salicylates is contraindicated in patients with chronic liver disease. /Salicylates/

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 688

▸ *from HSDB*

HYPOGLYCEMIA MAY BE ... CONSEQUENCE OF SALICYLATE TOXICITY IN YOUNG CHILDREN. /SALICYLATES/

Gilman, A.G., T.W. Rall, A.S. Nies and P. Taylor (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 8th ed. New York, NY. Pergamon Press, 1990., p. 651

▸ *from HSDB*

High therapeutic doses /of asprin/ (>3 g daily), as might be given for acute rheumatic fever, salt and water retention can lead to an increase (up to 20%) in circulating plasma volume and decreased hematocrit (via a dilutional effect). There is a tendency for the peripheral vessels to dilate because of a direct effect on vascular smooth muscle. Cardiac output and work are increased. Those with carditis or compromised cardiac function may not have sufficient cardiac reserve to meet the increased demands, and congestive cardiac failure and pulmonary edema can occur. High doses of salicylates can produce noncardiogenic pulmonary edema, particularly in older patients who ingest salicylates regularly over a prolonged period.

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 688

▸ *from HSDB*

/SALICYLISM/ ... OCCURS ONLY AFTER REPEATED ADMIN OF LARGE DOSES. /SALICYLATES/

Goodman, L.S., and A. Gilman. (eds.) The Pharmacological Basis of Therapeutics. 5th ed. New York: Macmillan Publishing Co., Inc., 1975., p. 336

▸ *from HSDB*

There is no evidence that moderate therapeutic doses of salicylates are teratogenic in human beings; however, babies born to women who ingest salicylates for long periods may have significantly reduced birth weights. When administered during the third trimester there also is an increase in perinatal mortality, anemia, antepartum and postpartum hemorrhage, prolonged gestation, and complicated deliveries; thus, its use during this period should be avoided. /Salicylates/

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 689

▸ *from HSDB*

Aspirin can cause a mild degree of hemolysis in individuals with a deficiency of glucose-6-phosphate dehydrogenase.

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 688

▸ *from HSDB*

Due to the association with Reye's syndrome, aspirin and other salicylates are contraindicated in children and young adults less than 20 years old with fever associated with viral illness. Reye's syndrome is characterized by the acute onset of encephalopathy, liver dysfunction, and fatty infiltration of the liver and other viscera. The etiology and pathophysiology are not clear. However, the epidemiologic evidence for an association between aspirin use in children and Reye's syndrome was sufficiently compelling that labeling of aspirin and aspirin-containing medications to indicate Reye's syndrome as a risk in children was mandated in 1986. Since then, the use of aspirin in children has declined dramatically, and Reye's syndrome has almost disappeared.

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 682

▸ *from HSDB*

The ingestion of salicylates may result in epigastric distress, nausea, and vomiting. Salicylates also may cause gastric ulceration, exacerbation of peptic ulcer symptoms (heartburn, dyspepsia), gastrointestinal hemorrhage, and erosive gastritis. These effects occur primarily with acetylated salicylates (i.e., aspirin).

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 688

▸ *from HSDB*

VET: Adverse effects are common following aspirin administration and appear to be dosage dependent. Even at therapeutic dosages ... plain aspirin may induce mucosal erosion and ulceration in dogs. Vomiting and melena may be seen at higher doses.

Kahn, C.M. (Ed.); The Merck Veterinary Manual 9th ed. Merck & Co. Whitehouse Station, NJ. 2005, p. 2133

▸ *from HSDB*

Large doses of salicylates may cause hyperglycemia and glycosuria and deplete liver and muscle glycogen. /Salicylates/

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 689

▸ *from HSDB*

Ingestion of aspirin by healthy individuals prolongs the bleeding time. For example, a single 325-mg dose of aspirin approximately doubles the mean bleeding time of normal persons for a period of 4 to 7 days. ... Patients with severe hepatic damage, hypoprothrombinemia, vitamin K deficiency, or hemophilia should avoid aspirin because the inhibition of platelet hemostasis can result in hemorrhage. If possible, aspirin therapy should be stopped at least 1 week before surgery

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 688

▸ *from HSDB*

Drugs that have been associated with Significant Effects on some Nursing Infants and should be given to Nursing Mothers with Caution: Aspirin (salicylates): Metabolic acidosis (1 case). /From Table 5/

Report of the American Academy of Pediatrics Committee on Drugs in Pediatrics 93 (1): 139 (1994)

▸ *from HSDB*

POTENTIAL ADVERSE EFFECTS ON FETUS: In animal studies, use in early pregnancy causes various malformations, including facial clefts, CNS and eye defects, and visceral and skeletal malformations. Controlled human studies have not demonstrated teratogenicity. During last weeks of gestation, long-term high-dose salicylate therapy may cause prolonged gestation, increased risk of postmaturity and fetal neonatal hemorrhage. Theoretically, regular use of aspirin in late pregnancy could cause premature closure or constriction of fetal ductus arteriosus. Decreased birth weight and increased risk of stillbirth have not been found with therapeutic doses of aspirin. POTENTIAL SIDE EFFECTS ON BREAST-FED INFANT: Excreted in low concentrations. Potential risk of adverse effects on infant platelet function if taken in high doses. FDA Category: C (C = Studies in laboratory animals have revealed adverse effects on the fetus (teratogenic, embryocidal, etc.) but there are no controlled studies in pregnant women. The benefits from use of the drug in pregnant women may be acceptable despite its potential risks, or there are no laboratory animal studies or adequate studies in pregnant women.) /from table II/

Stockton DL, Paller AS J Am Acad Dermatol 23 (1):87-103 (1990)

▸ *from HSDB*

In high doses, salicylates have toxic effects on the CNS, consisting of stimulation (including convulsions) followed by depression. Confusion, dizziness, tinnitus, high-tone deafness, delirium, psychosis, stupor, and coma may occur. The tinnitus and hearing loss of salicylate poisoning are caused by increased labyrinthine pressure or an effect on the hair cells of the cochlea, perhaps secondary to vasoconstriction in the auditory microvasculature. Tinnitus typically is observed at plasma salicylate concentrations of 200 to 450 g/ml, and there is a close relationship between the extent of hearing loss and plasma salicylate concentration. An occasional patient may note tinnitus at lower plasma concentrations of salicylate. Tinnitus generally resolves within 2 or 3 days after withdrawal of the drug. /Salicylates/

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 691

▸ *from HSDB*

VET: Dogs tolerate aspirin better than cats; however, prolonged use can lead to the development of gastric ulcers. Dosages of 25 mg/kg, tid, of regular aspirin have caused mucosal erosions in 50% of dogs after 2 days. Gastric ulcers were seen by day 30 in 66% of dogs given aspirin at 35 mg/kg, PO, tid. Similarly, 43% of dogs given aspirin at 50 mg/kg, PO, bid, showed gastric ulcers after 5-6 wk of dosing. Acute ingestion of 450-500 mg/kg can cause GI disturbances, hyperthermia, panting, seizures, or coma. Alkalosis due to stimulation of the respiratory center can occur early in the course of intoxication. Metabolic acidosis with an elevated anion gap usually develops later.

Kahn, C.M. (Ed.); The Merck Veterinary Manual 9th ed. Merck & Co. Whitehouse Station, NJ. 2005, p. 2528

▸ *from HSDB*

Acetylsalicylic acid suppositories can cause rectal irritation; absorption is slow and unpredictable.

International Programme on Chemical Safety; Poisons Information Monograph: Acetylsalicylic Acid (PIM 006) (1991) Available from, as of March 10, 2008: http://www.inchem.org/pages/pims.html

▸ *from HSDB*

7.8 Drug Idiosyncracies

... SOME PEOPLE MANIFEST IDIOSYNCRASY IN FORM OF ALLERGIC SENSITIVITY TO SALICYLATES, ESP ASPIRIN, & MAY SUFFER FROM SERIOUS IF NOT FATAL ASTHMA AFTER INGESTION OF SINGLE 0.3 G DOSE.

Osol, A. and J.E. Hoover, et al. (eds.). Remington's Pharmaceutical Sciences. 15th ed. Easton, Pennsylvania: Mack Publishing Co., 1975., p. 1049

▸ *from HSDB*

Aspirin is a known respiratory and systemic allergen and can produce anaphylactic phenomena even after small doses.

American Conference of Governmental Industrial Hygienists. Documentation of the TLV's and BEI's with Other World Wide Occupational Exposure Values. CD-ROM Cincinnati, OH 45240-1634 2007.

▸ *from HSDB*

An acute irritant to the gastric mucosa ... Ingestion of aspirin produce an increased tendency to bleed (increased clotting time) due to its interference with platelet aggregation. A normal, therapeutic dose ... 600 mg can produce these abnormalities for five days or longer. However, these toxic effects have also been demonstrated by ingestion of 150 mg, the smallest dose reported to have a pharmacologic effect.

American Conference of Governmental Industrial Hygienists. Documentation of the TLV's and BEI's with Other World Wide Occupational Exposure Values. CD-ROM Cincinnati, OH 45240-1634 2007.

▸ *from HSDB*

7.9 Minimum/Potential Fatal Human Dose

The lethal dose of aspirin for an adult is probably in the region of 25 to 30 g but recovery has been achieved by appropriate treatment after the ingestion of twice or thrice this amount.

Reynolds, J.E.F., Prasad, A.B. (eds.) Martindale-The Extra Pharmacopoeia. 28th ed. London: The Pharmaceutical Press, 1982., p. 236

▸ *from HSDB*

7.10 Maximum Drug Dose

The usual dose of aspirin as an analgesic and antipyretic is 0.3 to 1 g, which may be repeated every 4 hours according to clinical needs, up to a maximum of 4 g daily.

Reynolds, J.E.F., Prasad, A.B. (eds.) Martindale-The Extra Pharmacopoeia. 28th ed. London: The Pharmaceutical Press, 1982., p. 240

▸ *from HSDB*

8 Pharmacology and Biochemistry

8.1 Pharmacology

Acetylsalicylic acid is an analgesic, antipyretic, antirheumatic, and anti-inflammatory agent. Acetylsalicylic acid's mode of action as an antiinflammatory and antirheumatic agent may be due to inhibition of synthesis and release of prostaglandins. Acetylsalicylic acid appears to produce analgesia by virtue of both a peripheral and CNS effect. Peripherally, acetylsalicylic acid acts by inhibiting the synthesis and release of prostaglandins. Acting centrally, it would appear to produce analgesia at a hypothalamic site in the brain, although the mode of action is not known. Acetylsalicylic acid also acts on the hypothalamus to produce antipyresis; heat dissipation is increased as a result of vasodilation and increased peripheral blood flow. Acetylsalicylic acid's antipyretic activity may also be related to inhibition of synthesis and release of prostaglandins.

from DrugBank

Aspirin is an orally administered non-steroidal antiinflammatory agent. Acetylsalicylic acid binds to and acetylates serine residues in cyclooxygenases, resulting in decreased synthesis of prostaglandin, platelet aggregation, and inflammation. This agent exhibits analgesic, antipyretic, and anticoagulant properties.

from NCIt

8.2 MeSH Pharmacological Classification

Antipyretics

Drugs that are used to reduce body temperature in fever.
See a list of PubChem compounds matching this category.

from MeSH

Anti-Inflammatory Agents, Non-Steroidal

Anti-inflammatory agents that are non-steroidal in nature. In addition to anti-inflammatory actions, they have analgesic, antipyretic, and platelet-inhibitory actions.They act by blocking the synthesis of prostaglandins by inhibiting cyclooxygenase, which converts arachidonic acid to cyclic endoperoxides, precursors of prostaglandins. Inhibition of prostaglandin synthesis accounts for their analgesic, antipyretic, and platelet-inhibitory actions; other mechanisms may contribute to their anti-inflammatory effects.
See a list of PubChem compounds matching this category.

from MeSH

Fibrinolytic Agents

Fibrinolysin or agents that convert plasminogen to FIBRINOLYSIN.
See a list of PubChem compounds matching this category.

from MeSH

Cyclooxygenase Inhibitors

Compounds or agents that combine with cyclooxygenase (PROSTAGLANDIN-ENDOPEROXIDE SYNTHASES) and thereby prevent its substrate-enzyme combination with arachidonic acid and the formation of eicosanoids, prostaglandins, and thromboxanes.
See a list of PubChem compounds matching this category.

from MeSH

Platelet Aggregation Inhibitors

Drugs or agents which antagonize or impair any mechanism leading to blood platelet aggregation, whether during the phases of activation and shape change or following the dense-granule release reaction and stimulation of the prostaglandin-thromboxane system.
See a list of PubChem compounds matching this category.

from MeSH

8.3 FDA Pharmacological Classification

8.3.1 Active Moiety

ASPIRIN

from FDA Pharm Classes

8.3.2 FDA UNII

R16CO5Y76E

from FDA Pharm Classes

8.3.3 Pharmacological Classes

Mechanisms of Action [MoA]	Cyclooxygenase Inhibitors
Physiologic Effects [PE]	Decreased Prostaglandin Production
Chemical/Ingredient structural concept [Chemical/Ingredient]	Nonsteroidal Anti-inflammatory Compounds

Established Pharmacologic Class [EPC]	Nonsteroidal Anti-inflammatory Drug
Established Pharmacologic Class [EPC]	Platelet Aggregation Inhibitor
Physiologic Effects [PE]	Decreased Platelet Aggregation
Mechanisms of Action [MoA]	Cyclooxygenase Inhibitors
Physiologic Effects [PE]	Decreased Prostaglandin Production
Chemical/Ingredient structural concept [Chemical/Ingredient]	Nonsteroidal Anti-inflammatory Compounds
Established Pharmacologic Class [EPC]	Nonsteroidal Anti-inflammatory Drug

from FDA Pharm Classes

8.4 ATC Code

A - Alimentary tract and metabolism
A01 - Stomatological preparations
A01A - Stomatological preparations
A01AD - Other agents for local oral treatment
A01AD05 - Acetylsalicylic acid
More information...

from WHO ATC

B - Blood and blood forming organs
B01 - Antithrombotic agents
B01A - Antithrombotic agents
B01AC - Platelet aggregation inhibitors excl. heparin
B01AC06 - Acetylsalicylic acid
More information...

from WHO ATC

N - Nervous system
N02 - Analgesics
N02B - Other analgesics and antipyretics
N02BA - Salicylic acid and derivatives
N02BA01 - Acetylsalicylic acid
More information...

from WHO ATC

8.5 Absorption, Distribution and Excretion

Absorption

Absorption is generally rapid and complete following oral administration but may vary according to specific salicylate used, dosage form, and other factors such as tablet dissolution rate and gastric or intraluminal pH.

from DrugBank

The materno-fetal transfer of salicylic acid and its distribution in the fetal organism was investigated in women of early pregnancy. Acetylsalicylic acid was administered orally in a single dose or in repeated doses at different times before legal interruption. The mean passage rates were about 6-15%. They were independent of the maternal serum concentrations of salicylic acid. The distribution of salicylic acid on the fetal liver, intestine, kidneys, lungs and brain was different. All fetal organs (9th to 15th week of gestation) studied exhibit an acetylsalicylic acid-splitting esterase activity. The esterase activity of the fetal liver was about 30% of the hydrolytic activity of the adult liver. The esterase activity was mainly located in the 105 000 X g-supernatant of cell homogenates.
Abstract: PubMed

Amon I et al; Biomed Biochim Acta 42 (7-8): 997-1004 (1983)

from HSDB

Approximately 80-100% of an oral dose of aspirin is absorbed from the GI tract. However, the actual bioavailability of the drug as unhydrolyzed aspirin is lower since aspirin is partially hydrolyzed to salicylate in the GI mucosa during absorption and on first pass through the liver. There are relatively few studies of the bioavailability of unhydrolyzed aspirin. In one study in which aspirin was administered IV and as an oral aqueous solution, it was shown that the solution was completely absorbed but only about 70% reached the systemic circulation as unhydrolyzed aspirin. In another study in which aspirin was administered IV and orally as capsules, only about 50% of the oral dose reached the systemic circulation as unhydrolyzed aspirin. There is some evidence that the bioavailability of unhydrolyzed aspirin from slowly absorbed dosage forms (e.g., enteric-coated tablets) may be substantially decreased. Food does not appear to decrease the bioavailability of unhydrolyzed aspirin or salicylate; however, absorption is delayed and peak serum aspirin or salicylate concentration may be decreased. There is some evidence that absorption of salicylate following oral administration may be substantially impaired or is highly variable during the febrile phase of Kawasaki disease.

McEvoy, G.K. (ed.). American Hospital Formulary Service. AHFS Drug Information. American Society of Health-System Pharmacists, Bethesda, MD. 2007., p. 2036

from HSDB

A 52 year-old woman ingested approximately 300 tablets (325 mg) of aspirin in a suicide attempt. ... The concentrations of salicylic acid in heart and femoral blood were 1.1 mg/mL and 1.3 mg/mL, respectively; the results were far higher than the lethal level. The concentration of salicylic acid was 0.3-0.4 mg/g in brain, 0.9-1.4 mg/g in lung, 0.6-0.8 mg/g in liver and 0.9 mg/mL in kidney.
Abstract: PubMed

Ihama Y t al; Chudoku Kenkyu 20 (4): 375-80 (2007)

from HSDB

The study was undertaken to determine the distribution of aspirin and its metabolites in the semen of humans after an oral dose of aspirin. Each of seven healthy male volunteers was given a single oral dose of 975 mg of aspirin on an empty stomach together with 200 mL of water. Timed samples of blood and semen were obtained from each subject, and the concentrations of aspirin, salicylic acid, and salicyluric acid determined by a specific high-performance liquid chromatographic assay. The mean peak concentration of aspirin was 6.5 micrograms/mL in plasma (range, 4.9-8.9 micrograms/mL), reached in 26 minutes (range, 13-33 minutes). The half-life of aspirin was 31 minutes. The concentration ratio of aspirin (semen/plasma) was 0.12 (except for one subject in whom it was 0.025). The mean peak concentration of salicylate in plasma was 49 micrograms/mL (range, 42-62 micrograms/mL), reached in 2.5 hours (range, 2.0-2.8 hours). Salicylate distributed rapidly into semen and maintained a concentration ratio (semen/plasma) of 0.15. Salicyluric acid (the glycine conjugate of salicylic acid) was found in the semen. Its high concentration in some subjects' semen (four times the concurrent plasma concentration) was attributed to contamination of semen sample with residual urine, containing salicylurate, in the urethra of those who urinated after the dose of aspirin. Possible side effects of aspirin and salicylate in semen include adverse effects on fertility, male-medicated teratogenesis, dominant lethal mutations, and hypersensitivity reactions in the recipients. Abstract: PubMed

Kershaw RA et al; J Clin Pharmacol 27 (4): 304-9 (1987)

from HSDB

The volume of distribution of usual doses of aspirin ... in normal subjects average about 170 mL/kg of body weight; at high therapeutic doses, this volume increases to about 500 mL/kg because of saturation of binding sites on plasma proteins. Ingested aspirin mainly is absorbed as such, but some enters the systemic circulation as salicylic acid after hydrolysis by esterases in the gastrointestinal mucosa and liver. Aspirin can be detected in the plasma only for a short time as a result of hydrolysis in plasma, liver, and erythrocytes; for example, 30 minutes after a dose of 0.65 g, only 27% of the total plasma salicylate is in the acetylated form.

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 689

from HSDB

Cats are deficient in glucuronyl transferase and have a prolonged excretion of aspirin (the half-life in cats is 37.5 hr).

Kahn, C.M. (Ed.); The Merck Veterinary Manual 9th ed. Merck & Co. Whitehouse Station, NJ. 2005, p. 2528

from HSDB

Effervescent or noneffervescent oral aqueous solutions of aspirin appear to be completely absorbed. Oral buffered aspirin tablets, uncoated plain aspirin tablets, and methylcellulose film-coated (non-enteric) plain aspirin tablets are approximately 80-100% absorbed. Erratic and incomplete absorption of some enteric-coated aspirin tablets (particularly those with shellac coatings) has been reported, but recent studies indicate that the extent of absorption of currently available enteric-coated aspirin tablets is similar to that of buffered, uncoated plain, and film-coated plain aspirin tablets. Although well-designed studies are lacking, the extent of absorption of extended-release aspirin tablets appears to be similar to that of uncoated plain aspirin tablets. There are apparently no published studies on the bioavailability of aspirin capsules. Following rectal administration as a suppository, aspirin is slowly and variably absorbed; the extent of absorption increases with increasing rectal retention time. In general, 20-60% of the dose is absorbed if the suppository is retained for 2-4 hours and 70-100% is absorbed if the suppository is retained for at least 10 hours.

McEvoy, G.K. (ed.). American Hospital Formulary Service. AHFS Drug Information. American Society of Health-System Pharmacists, Bethesda, MD. 2007., p. 2036

from HSDB

Peak salicylate concentrations of 173 to 483 ug/mL have been measured in /breast milk/ 5 to 8 hours after maternal ingestion of a single 650 mg dose /of aspirin/.

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2576

from HSDB

In one study in patients with rheumatic disease who received a single 650-mg oral dose of buffered aspirin, aspirin was detected in synovial fluid within 10-30 minutes and salicylate was detected in synovial fluid within 15-35 minutes. In this study, peak aspirin concentrations in synovial fluid occurred after an average of 1.3 hours and were about 75% of peak blood concentrations; peak salicylate concentrations in synovial fluid occurred after an average of 2.2 hours and were about 60% of peak blood concentrations.

McEvoy, G.K. (ed.). American Hospital Formulary Service. AHFS Drug Information. American Society of Health-System Pharmacists, Bethesda, MD. 2007., p. 2036

from HSDB

Aspirin is rapidly and widely distributed, apparently into most body tissues and fluids. The volume of distribution of aspirin is approximately the same as that of salicylate and is generally 0.15-0.2 L/kg.

McEvoy, G.K. (ed.). American Hospital Formulary Service. AHFS Drug Information. American Society of Health-System Pharmacists, Bethesda, MD. 2007., p. 2036

from HSDB

In one study in healthy fasting adults given a single 975-mg oral dose of aspirin (as three 325-mg uncoated plain tablets), peak serum salicylate concentrations averaged 60-75 ug/mL and occurred within 2 hours. In another study in fasting rheumatoid arthritis patients given a single 1.95-g oral dose of aspirin (as six 325-mg uncoated plain tablets), peak plasma aspirin concentrations of about 12-16 ug/mL occurred within 1 hour and peak plasma salicylate concentrations of about 110-160 ug/mL occurred within 4 hours. When these patients were given the same dose of buffered aspirin (as 6 tablets, each containing 325 mg of aspirin), peak plasma aspirin concentrations of about 14-18 ug/mL occurred within 1-2 hours and peak plasma salicylate concentrations of about 140-160 ug/mL occurred within 1-2 hours.

McEvoy, G.K. (ed.). American Hospital Formulary Service. AHFS Drug Information. American Society of Health-System Pharmacists, Bethesda, MD. 2007., p. 2036

from HSDB

Following oral administration of a single 650-mg dose of aspirin as an effervescent or noneffervescent aqueous solution in healthy adults, average peak plasma aspirin concentrations of about 13 ug/mL are attained within 15-40 minutes and average peak plasma salicylate concentrations of about 40-55 ug/mL are attained within 30-60 minutes. After a single 650-mg oral dose of aspirin (as two 325-mg uncoated plain tablets) in fasting healthy adults, average peak plasma aspirin concentrations of about 7-9 ug/mL occur within 25-40 minutes and average peak plasma salicylate concentrations of about 35-50 ug/mL occur within 1.5-2 hours. Following oral administration of a single 650-mg dose of buffered aspirin (as 2 tablets, each containing 325 mg of aspirin), average peak plasma salicylate concentrations of about 40-60 mcg/mL are attained within 45-60 minutes.

McEvoy, G.K. (ed.). American Hospital Formulary Service. AHFS Drug Information. American Society of Health-System Pharmacists, Bethesda, MD. 2007., p. 2037

from HSDB

8.6 Metabolism/Metabolites

Metabolism

Acetylsalicylic acid is rapidly hydrolyzed primarily in the liver to salicylic acid, which is conjugated with glycine (forming salicyluric acid) and glucuronic acid and excreted largely in the urine.

▸ *from DrugBank*

Acetylsalicylic acid is hydrolyzed in the stomach and in blood to salicylic acid and acetic acid; ... .

International Programme on Chemical Safety; Poisons Information Monograph: Acetylsalicylic Acid (PIM 006) (1991) Available from, as of March 10, 2008: http://www.inchem.org/pages/pims.html

▸ *from HSDB*

MAJOR URINARY METABOLITES OF ASPIRIN INCL SALICYLURONIC ACID ... SALICYL-O-GLUCURONIDE ... & SALICYL ESTER GLUCURONIDE ... & FREE SALICYLIC ACID ... .

The Chemical Society. Foreign Compound Metabolism in Mammals. Volume 2: A Review of the Literature Published Between 1970 and 1971. London: The Chemical Society, 1972., p. 183

▸ *from HSDB*

A 52 year-old woman ingested approximately 300 tablets (325 mg) of aspirin in a suicide attempt. /Investigators/ analyzed the concentrations of salicylic acid (SA) and salicyluric acid (SUA) in body fluids and organs using a modified previous high-performance liquid chromatographic method. The concentrations of SA in heart and femoral blood were 1.1 mg/mL and 1.3 mg/mL, respectively; the results were far higher than the lethal level. The concentration of SA was 0.3-0.4 mg/g in brain, 0.9-1.4 mg/g in lung, 0.6-0.8 mg/g in liver and 0.9 mg/mL in kidney.
Abstract: PubMed

Ihama Y et al; Chudoku Kenkyu 20 (4): 375-80 (2007)

▸ *from HSDB*

8.7 Biological Half-Life

The plasma half-life is approximately 15 minutes; that for salicylate lengthens as the dose increases: doses of 300 to 650 mg have a half-life of 3.1 to 3.2 hours; with doses of 1 gram, the half-life is increased to 5 hours and with 2 grams it is increased to about 9 hours.

▸ *from DrugBank*

15 to 20 minutes (for intact molecule); rapidly hydrolyzed to salicylate. In breast milk (as salicylate): 3.8 to 12.5 hours (average 7.1 hours) following a single 650 mg dose of aspirin.

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2576

▸ *from HSDB*

Cats are deficient in glucuronyl transferase and have a prolonged excretion of aspirin (the half-life in cats is 37.5 hr).

Kahn, C.M. (Ed.); The Merck Veterinary Manual 9th ed. Merck & Co. Whitehouse Station, NJ. 2005, p. 2528

▸ *from HSDB*

8.8 Mechanism of Action

The analgesic, antipyretic, and anti-inflammatory effects of acetylsalicylic acid are due to actions by both the acetyl and the salicylate portions of the intact molecule as well as by the active salicylate metabolite. Acetylsalicylic acid directly and irreversibly inhibits the activity of both types of cyclooxygenase (COX-1 and COX-2) to decrease the formation of precursors of prostaglandins and thromboxanes from arachidonic acid. This makes acetylsalicylic acid different from other NSAIDS (such as diclofenac and ibuprofen) which are reversible inhibitors. Salicylate may competitively inhibit prostaglandin formation. Acetylsalicylic acid's antirheumatic (nonsteroidal anti-inflammatory) actions are a result of its analgesic and anti-inflammatory mechanisms; the therapeutic effects are not due to pituitary-adrenal stimulation. The platelet aggregation-inhibiting effect of acetylsalicylic acid specifically involves the compound's ability to act as an acetyl donor to cyclooxygenase; the nonacetylated salicylates have no clinically significant effect on platelet aggregation. Irreversible acetylation renders cyclooxygenase inactive, thereby preventing the formation of the aggregating agent thromboxane A2 in platelets. Since platelets lack the ability to synthesize new proteins, the effects persist for the life of the exposed platelets (7-10 days). Acetylsalicylic acid may also inhibit production of the platelet aggregation inhibitor, prostacyclin (prostaglandin I2), by blood vessel endothelial cells; however, inhibition prostacyclin production is not permanent as endothelial cells can produce more cyclooxygenase to replace the non-functional enzyme.

▸ *from DrugBank*

Produce analgesia through a peripheral action by blocking pain impulse generation and via a central action, possibly in the hypothalamus. The peripheral action may predominate and probably involves inhibition of the synthesis or prostaglandins, and possibly inhibition of the synthesis and/or actions of other substances, which sensitize pain receptors to mechanical or chemical stimulation. /Salicylates/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2575

▸ *from HSDB*

May produce antipyresis by acting centrally on the hypothalamic heat-regulating center to produce peripheral vasodilation resulting in increased cutaneous blood flow, sweating, and heat loss. The central action may involve inhibition of prostaglandin synthesis in the hypothalamus; however, there is some evidence that fevers caused by endogenous pyrogens that do not act via a prostaglandin mechanism may also respond to salicylate therapy. /Salicylates/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2575

▸ *from HSDB*

CNS ... ESP NUCLEI LOCATED IN HYPOTHALAMUS PLAYS MAJOR ROLE IN REGULATION OF PERIPHERAL MECHANISMS CONCERNED WITH BODY HEAT PRODN & LOSS. WITH SALICYLATES, HEAT PRODN IS NOT INHIBITED, BUT HEAT LOSS IS INCR BY INCR PERIPHERAL BLOOD FLOW & PERSPIRATION. /SALICYLATES/

Evaluations of Drug Interactions. 2nd ed. and supplements. Washington, DC: American Pharmaceutical Assn., 1976, 1978., p. 341

▸ *from HSDB*

Aspirin acetylates prostaglandin endoperoxide synthase (prostaglandin G/H-synthase) and irreversibly inhibits its cyclooxygenase (COX) activity. The enzyme catalyzes the conversion of arachidonic acid to PGH2, the first committed step in prostanoid biosynthesis. Two isoforms of prostaglandin endoperoxide synthase exist, PGHS-1 and PGHS-2 (also referred to as COX-1 and COX-2, respectively). PGHS-1 (COX-1) is expressed constitutively in most cell types, including platelets. PGHS-2 (COX-2) is undetectable in most mammalian cells, but its expression can be induced rapidly in response to mitogenic and inflammatory stimuli. Aspirin is a relatively selective inhibitor of platelet PGHS-1 (cyclooxygenase-1, COX-1). The existence of 2 isoenzymes with different aspirin sensitivities, coupled with extremely different recovery rates of their cyclooxygenase (COX) activity following inactivation by aspirin, at least partially explains the different dosage requirements and durations of aspirin effects on platelet function versus the drug's analgesic and anti-inflammatory effects. Human platelets and vascular endothelial cells process PGH2 to produce thromboxane A2 and prostacyclin (epoprostenol, PGI2), respectively. Thromboxane A2 induces platelet aggregation and vasoconstriction, while prostacyclin inhibits platelet aggregation and induces vasodilation. Aspirin is antithrombotic in a wide range of doses inhibiting thromboxane A2 and prostacyclin.

McEvoy, G.K. (ed.). American Hospital Formulary Service. AHFS Drug Information. American Society of Health-System Pharmacists, Bethesda, MD. 2007., p. 2035

from HSDB

Aspirin (but not other salicylates) inhibits platelet aggregation induced by epinephrine or low concentrations of collagen but not that induced by thrombin or high concentrations of collagen. Aspirin inhibits the second phase of platelet aggregation by preventing release of adenosine diphosphate (ADP) from platelets. The drug also prevents release of platelet factor 4 from platelets. Mean bleeding time may be prolonged by several minutes (approximately doubled) in healthy individuals and longer in children or in patients with bleeding disorders (e.g., hemophilia). In healthy individuals receiving a single 325-mg oral dose of aspirin, bleeding time may increase to a maximum within 12 hours and generally return to normal within 24 hours; any increase is usually of little clinical significance. Some clinicians have reported that mean bleeding time is progressively prolonged with increasing single doses of up to 1 g, but may be only slightly prolonged or unaffected by higher single doses; however, this has not been consistently found. The effect on bleeding time depends on the measurement method (e.g., Duke, Ivy, Mielke) used and technical variables (e.g., venostasis), and this may partially account for conflicting reports. Like the analgesic and anti-inflammatory effects, the effects of aspirin on platelets appear to be mainly associated with inhibition of prostaglandin synthesis. Aspirin irreversibly acetylates and inactivates cyclooxygenase in circulating platelets and possibly in megakaryocytes. A single 325-mg oral dose of the drug results in about 90% inhibition of the enzyme in circulating platelets. This inactivation prevents platelet synthesis of prostaglandin endoperoxides and thromboxane A2, compounds which induce platelet aggregation and constrict arterial smooth muscle. Since cyclooxygenase in platelets is not resynthesized, this effect of aspirin on platelet function persists for the life span of platelets (4-7 days). When approximately 20% of circulating platelets have not been exposed to aspirin (about 36 hours after the last dose), the hemostatic function of the platelet pool generally returns to normal; however, altered hemostasis has been reported to persist longer in some patients receiving long-term therapy.

McEvoy, G.K. (ed.). American Hospital Formulary Service. AHFS Drug Information. American Society of Health-System Pharmacists, Bethesda, MD. 2007., p. 2035

from HSDB

inducer of platelet aggregation and a potent vasoconstrictor. Aspirin blocks production of thromboxane A2 by acetylating a serine residue near the active site of platelet cyclooxygenase (COX-1), the enzyme that produces the cyclic endoperoxide precursor of thromboxane A2. Since platelets do not synthesize new proteins, the action of aspirin on platelet cyclooxygenase is permanent, lasting for the life of the platelet (7 to 10 days). Thus, repeated doses of aspirin produce a cumulative effect on platelet function. Complete inactivation of platelet COX-1 is achieved when 160 mg of aspirin is taken daily. Therefore, aspirin is maximally effective as an antithrombotic agent at doses much lower than those required for other actions of the drug.

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 1482

from HSDB

CHRONIC ADMIN ... DECR PLASMA PROTEIN-BOUND IODINE & THYROIDAL UPTAKE & CLEARANCE OF IODINE ... EFFECTS ... PROBABLY DUE TO ... DISPLACEMENT BY SALICYLATE OF THYROXINE & TRIIODOTHYRONINE FROM PREALBUMIN & THYROXINE-BINDING GLOBULIN IN PLASMA ... . /SALICYLATES/

Gilman, A.G., T.W. Rall, A.S. Nies and P. Taylor (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 8th ed. New York, NY. Pergamon Press, 1990., p. 647

from HSDB

VERY LARGE DOSES ... STIMULATE STEROID SECRETION BY ADRENAL CORTEX THROUGH EFFECT ON HYPOTHALAMUS & INCR ... PLASMA CONCN OF FREE ADRENOCORTICOSTEROIDS BY DISPLACEMENT FROM PLASMA PROTEINS. /SALICYLATES/

Gilman, A.G., T.W. Rall, A.S. Nies and P. Taylor (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 8th ed. New York, NY. Pergamon Press, 1990., p. 648

from HSDB

SALICYLATES REDUCE LIPOGENESIS BY PARTIALLY BLOCKING INCORPORATION OF ACETATE INTO FATTY ACIDS ... HIGH DOSES ... ACTIVATE CENTRAL SYMPATHETIC CENTERS & THEREBY CAUSE RELEASE OF EPINEPHRINE FROM ADRENAL MEDULLA. /SALICYLATES/

Gilman, A.G., T.W. Rall, A.S. Nies and P. Taylor (eds.). Goodman and Gilman's The Pharmacological Basis of Therapeutics. 8th ed. New York, NY. Pergamon Press, 1990., p. 648

from HSDB

... Cyclooxygenase-2 (COX-2) and intercellular adhesion molecule-1 (ICAM-1) expression, IkappaB and p38 mitogen-activated protein kinase (MAPK) phosphorylation were determined in endothelial cells exposed to oxidized low-density lipoprotein (ox-LDL) in the presence of aspirin. The results showed that aspirin significantly suppressed COX-2 and ICAM-1 expression induced by ox-LDL and also inhibited IkappaB phosphorylation in human umbilical vein endothelial cells (HUVECs). Moreover, aspirin reduced the level of p38 MAPK phosphorylation. Our findings suggest that aspirin can decrease inflammatory responses induced by ox-LDL, and the mechanism might be associated with NF-kappaB activation pathway and inhibition of p38 MAPK phosphorylation.
Abstract: PubMed

Zhao J et al; J Cardiovasc Pharmacol 51 (1): 32-7 (2008)

from HSDB

Aspirin at low concentrations from 1X10(-10) mol/L to 1X10(-8) mol/L decreased the apoptosis and Phospho-p38 mitogen-activated protein kinase (p38 MAPK) phosphorylation induced by H2O2 in BAEC, while high doses of aspirin (1X10(-7)-1X10(-4) mol/L) induced typical apoptotic changes in BAEC and stimulated the expression of phospho-p38 MAPK in a concentration-dependent manner. SB203580, a specific p38 MAPK inhibitor, blocked such effects. Aspirin exhibits a biphasic effect on the apoptosis in BAEC, reducing apoptosis at low concentration and inducing apoptosis at high concentration.
Abstract: PubMed

Chen QQ et al; Acta Pharmacol Sin 28 (3): 353-8 (2007)

from HSDB

Aspirin exerts its unique pharmacological effects by irreversibly acetylating a serine residue in the cyclooxygenase site of prostaglandin-H(2)-synthases (PGHSs). Despite the irreversibility of the inhibition, the potency of aspirin varies remarkably between cell types, suggesting that molecular determinants could contribute to cellular selectivity. Using purified enzymes, /investigators/ found no evidence that aspirin is selective for either of the two PGHS isoforms, and we showed that hydroperoxide substrates of the PGHS peroxidase inhibited the rate of acetylation of PGHS-1 by 68%. Using PGHS-1 reconstituted with cobalt protoporphyrin, a heme devoid of peroxidase activity, we demonstrated that reversal by hydroperoxides of the aspirin-mediated acetylation depends upon the catalytic activity of the PGHS peroxidase. ... Inhibition of PGHS-2 by aspirin in cells in culture is reversed by 12-hydroperoxyeicosatetraenoic acid dose-dependently (ED(50)=0.58+/-0.15muM) and that in cells with high levels of hydroperoxy-fatty acids (RAW264.7) the efficacy of aspirin is markedly decreased as compared to cells with low levels of hydroperoxides (A549; IC(50)s=256+/-22muM and 11.0+/-0.9muM, respectively). Together, these findings indicate that acetylation of the PGHSs by aspirin is regulated by the catalytic activity of the peroxidase, which yields a higher oxidative state of the enzyme.[Bala M et al; Biochem Pharmacol 75 (7): 1472-81 (2008)] Full text: PMC2693035
Abstract: PubMed

from HSDB

8.9 Human Metabolite Information

8.9.1 Metabolite Description

Description

Aspirin is only found in individuals who have consumed this drug. Aspirin or acetylsalicylic acid (acetosal) is a drug in the family of salicylates, often used as an analgesic (against minor pains and aches), antipyretic (against fever), and anti-inflammatory. It has also an anticoagulant effect and is used in long-term low-doses to prevent heart attacks and cancer. It was isolated from meadowsweet (Filipendula ulmaria, formerly classified as Spiraea ulmaria) by German researchers in 1839. While their extract was somewhat effective, it also caused digestive problems such as irritated stomach and diarrhoea, and even death when consumed in high doses. In 1853, a French chemist named Charles Frederic Gerhardt neutralized salicylic acid by buffering it with sodium (sodium salicylate) and acetyl chloride, creating acetosalicylic anhydride. Gerhardt's product worked, but he had no desire to market it and abandoned his discovery. In 1897, researcher Arthur Eichengrun and Felix Hoffmann, a research assistant at Friedrich Bayer & Co. in Germany, derivatized one of the hydroxyl functional groups in salicylic acid with an acetyl group (forming the acetyl ester), which greatly reduced the negative effects. This was the first synthetic drug, not a copy of something that existed in nature, and the start of the pharmaceuticals industry. The name 'aspirin' is composed of a- (from the acetyl group) -spir- (from the plant genus Spiraea) and -in (a common ending for drugs at the time). It has also been stated that the name originated by another means. As referring to AcetylSalicylic and 'pir' in reference to one of the scientists who was able to isolate it in crystalline form, Raffaele Piria. Finally 'in' due to the same reasons as stated above. Salicylic acid (which is a naturally occurring substance found in many plants) can be acetylated using acetic anhydride, yielding aspirin and acetic acid as a byproduct. It is a common experiment performed in organic chemistry labs, and generally tends to produce low yields due to the relative difficulty of its extraction from an aqueous state. The trick to getting the reaction to work is to acidify with phosphoric acid and heat the reagents under reflux with a boiling water bath for between 40 minutes and an hour. Aspirin acts as an inhibitor of cyclooxygenase which results in the inhibition of the biosynthesis of prostaglandins. Aspirin also inhibits platelet aggregation and is used in the prevention of arterial and venous thrombosis. (From Martindale, The Extra Pharmacopoeia, 30th ed, p5).

from Human Metabolome Database (HMDB)

8.9.2 Tissue Locations

Platelet

from Human Metabolome Database (HMDB)

8.9.3 Cellular Locations

Cytoplasm

from Human Metabolome Database (HMDB)

8.9.4 Metabolite Pathways

Acetylsalicylic Acid Action Pathway

from Human Metabolome Database (HMDB)

9 Use and Manufacturing

9.1 Uses

EU Pharmaceutical Product Classes

Human drug

from EU Community Register of Medicinal Products

9.1.1 Industry Uses

Paint additives and coating additives not described by other categories

from EPA Chemicals under the TSCA

9.1.2 Consumer Uses

Paper products

from EPA Chemicals under the TSCA

9.2 Methods of Manufacturing

Acetylsalicylic acid is prepared by reacting acetic anhydride with salicylic acid at a temperature of <90 deg C either in a solvent (e.g., acetic acid or aromatic, acyclic, or chlorinated hydrocarbons) or by the addition of catalysts such as acids or tertiary amines.

Ullmann's Encyclopedia of Industrial Chemistry. 6th ed.Vol 1: Federal Republic of Germany: Wiley-VCH Verlag GmbH & Co. 2003 to Present, p. V31 725 (2003)

from HSDB

Manufacture from salicylic acid and acetic anhydride. ... Crystallization from acetone..

O'Neil, M.J. (ed.). The Merck Index - An Encyclopedia of Chemicals, Drugs, and Biologicals. Whitehouse Station, NJ: Merck and Co., Inc., 2006., p. 140

from HSDB

9.3 Impurities

4-hydroxybenzoic acid; 4-hydroxybenzene-1,3-dicarboxylic acid (4-hydroxyisophthalic acid); salicylic acid; 2-[[2-(acetyloxy)benzoyl]oxy]benzoic acid (acetylsalicylsalicylic acid); 2-[(2-hydroxybenzoyl)oxy]benzoic acid (salicylsalicylic acid); 2-(acetyloxy)benzoic anhydride (acetylsalicylic anhydride)

Council of Europe, European Directorate for the Quality of Medicines. European Pharmacopoeia, 5th Ed., Volume 2; Strasbourg, France, p.917 (2004)

from HSDB

9.4 Formulations/Preparations

Aspirin Formulations:

Preparations	Dose (mg)	Product	Manufacturer
Pieces, chewing gum	227	Aspergum	Heritage, Schering Plough
Tablets	81	Aspirin Adult Low Strength	Geri-Care,Mango-Humphries
	81	Aspirin Low Dose	Basic Viatamins
	325	Norwich Aspirin	Chattem
	325 with buffers	Magnaprin, Improved	Rugby
	325 with buffers	Magnaprin Arthritis Strength	Rugby
	500	Norwich Aspirin Maximum Strength	Chattem
	650	Norwich Aspirin Maximum Strength	Chattem
Tablets, chewable	81	Bayer Children's Chewable aspirin	Bayer
	81	Aspirin Childrens	AmerisourceBergen, Cardinal Health, Chain Drug Marketing, Eckerd, Ivax, Major, PDK, Prime Marketing, Qualitest, Rugby, URL
	81	Aspirin for Children	Geri-Care
	81	St. Joseph Aspirin Low Strength Chewable	McNeil
Tablets, delayed-	81	Aspirin Adult Low Strength	AmerisourceBergen

release (enteric-coated)			
	81	Aspirin Adult Low Strength	Ivax
	81	Aspirin Enteric Coated	Advance
	81	Aspirin Low Dose	Qualitest
	81	Aspirin Regimen	PDK
	81	Aspirin Regimen Low Strength	Cardinal Health
	81	Bayer Aspirin Regimen Adult Low Strength	Bayer
	81	Ecotrin Adult Low Strength (with propylene glycol)	GlaxoSmithKline
	81	Ecotrin Adult Low Strength (with propylene glycol)	GlaxoSmithKline
	81	St. Joseph Adult Low Strength Enteric Coated Tablets	McNeil
	81 with buffers	Ascriptin Enteric Regular Strength	Novartis
	162	Halfprin	Kramer
	325	Aspirin for Arthritis	Cardinal Health
	325	Bayer Aspirin Regimen Regular Strength Caplets (with propylene glycol	Bayer
	325	Ecotrin Regular Strength (with propylene glycol)	GlaxoSmithKline
	325	Genacote	Ivax
	325 with buffers	Ascriptin Enteric Regular Strength	Novartis
	500	Aspirin Extra Strength	Medicine Shoppe
	500	Aspirin Maximum Strength	Chain Drug Marketing
	500	Ecotrin Maximum Strength (with propylene glycol)	GlaxoSmithKline
	650	Aspirin Delayed Release Tablets	Time-Cap, United Research
	975	Easprin	Harvest
Tablets, extended-release	800	ZORprin	Par
Tablets, film-coated	81 with buffers	Women's Aspirin Plus Calcium Caplets (with calcium carbonate and crospovidone)	Bayer
	325	Aspirin Lite Coated	AmerisourceBergen
	325	Aspirin Microthin Coating	Cardinal Health
	325	Bayer Aspirin Caplets	Bayer
	325	Genuine Bayer Aspirin Tablets	Bayer
	325 with buffers	Ascriptin Regular Strength	Novartis
	325 with buffers	Ascriptin Arthritis Pain Caplets	Novartis
	325 with buffers	Bufferin Tablets (with povidone and propylene glycol),	Novartis Consumer Health
	500	Bayer Aspirin Extra Strength Caplets (with propylene glycol)	Bayer
	500	Bayer Aspirin Extra Strength Gelcaplets (with parabens)	Bayer
	500	Bayer Aspirin Extra Strength Tablets	Bayer
	500 with buffers	Ascriptin Maximum Strength Caplets	Novartis
	500 with buffers	Bayer Aspirin Plus Buffered Extra Strength Caplets (with calcium carbonate and propylene glycol)	Bayer
	500 with buffers	Bufferin Arthritis Strength Caplets (with povidone and propylene glycol)	Bristol-Myers
	500 with buffers	Bufferin Extra Strength (with povidone and propylene glycol)	Novartis Consumer Health

Tablets, for solution	325	Alka-Seltzer Effervescent Pain Reliever and Antacid (with citric acid 1 g and sodium bicarbonate 1.916 g)	Bayer
	325	Alka-Seltzer Lemon-Lime Effervescent Pain Reliever and Antacid (with aspartame citric acid 1 g and sodium bicarbonate 1.7 g)	Bayer
	500	Alka-Seltzer Extra Strength Effervescent Pain Reliever and Antacid (with citric acid 1 g and sodium bicarbonate 1.985 g)	Bayer
Rectal Suppositories	60	Aspirin Suppositories	Consolidated Midland
	120	Aspirin Suppositories	Consolidated Midland
	200	Aspirin Suppositories	Consolidated Midland
	300	Aspirin Suppositories	Consolidated Midland, Paddock
	300	Aspirin Suppositories	Consolidated Midland, Paddock

McEvoy, G.K. (ed.). American Hospital Formulary Service. AHFS Drug Information. American Society of Health-System Pharmacists, Bethesda, MD. 2007., p. 2037

▸ *from HSDB*

9.5 Consumption

54% AS AN ANALGESIC IN COMBINATION WITH OTHER ACTIVE OR INERT INGREDIENTS; 46% AS AN ANALGESIC IN SINGLE INGREDIENT ASPIRIN TABLETS (1973)

SRI

▸ *from HSDB*

CHEMICAL PROFILE: Aspirin. Almost all aspirin manufactured in the US is used in aspirin tablets, pharmaceutical products or in conjunction with other ingredients for its analgesic and antipyretic properties. Approx 10% of US production is exported in bulk.

Kavaler AR; Chemical Marketing Reporter 231 (8): 54 (1987)

▸ *from HSDB*

CHEMICAL PROFILE: Aspirin. Demand: 1986: 29.5 million lb; 1987: 30.0 million lb; 1991 /projected/: 31.8 million lb.

Kavaler AR; Chemical Marketing Reporter 231 (8): 54 (1987)

▸ *from HSDB*

CHEMICAL PROFILE: Aspirin. Almost all aspirin manufactured in the US is used in aspirin tablets, pharmaceutical products or in conjunction with other ingredients for its analgesic and antipyretic properties. Approximately 5% of US production is exported in bulk.

Kavaler AR; Chemical Marketing Reporter 237 (10): 50 (1990)

▸ *from HSDB*

CHEMICAL PROFILE: Aspirin. Demand: 1989: 23 million lb; 1990 /projected/: 23 million lb; 1994 /projected/: 23 million lb. (Production last year /1989/ was 19.2 million lb; significant inventory carryovers from the previous year supplied a few million lb of demand. Imports were 2.6 million lb last year /1989/, and exports were 1.3 million lb.)

Kavaler AR; Chemical Marketing Reporter 237 (10): 50 (1990)

▸ *from HSDB*

CHEMICAL PROFILE: Acetylsalicylic acid. US Demand: 1995, 24 million pounds; 1996, 23.6 million pounds; 2000, 22 milliuon pounds /projected/

Anon. (1996) Chemical Marketing Reporter, January 8, 1996

▸ *from HSDB*

CHEMICAL PROFILE: Acetylsalicylic acid. US Demand: 1998, 23.5 million pounds; 1999, 23.7 million pounds; 2003, 24/7 million pounds /projected/

Anon. (2000) Chemical Marketing Reporter, 258(6): 45, August 7, 2000

▸ *from HSDB*

CHEMICAL PROFILE: Acetylsalicylic acid. US Demand: 1997, 23.4 million pounds; 1998, 23.5 million pounds; 1999, 23.7 million pounds; 2000, 23.9 million pounds; 2001: 23.8 million pounds; 2002: 23.9 million pounds; 2006: 24.6 million pounds, projected

Kirschner M (2003) Chemical Market Reporter, August 18/25 (2003)

▸ *from HSDB*

9.6 U.S. Production

(1972) 1.59X10+10 GRAMS

SRI

▸ *from HSDB*

(1975) 1.16X10+10 GRAMS

SRI

▸ *from HSDB*

(1984) 1.54X10+10 g

USITC. SYN ORG CHEM-U.S. PROD/SALES 1985 p.97

▸ *from HSDB*

Production volumes for non-confidential chemicals reported under the Inventory Update Rule.

Year	Production Range (pounds)
1986	>1 million - 10 million
1990	No Reports
1994	10 thousand - 500 thousand
1998	10 thousand - 500 thousand
2002	No Reports

US EPA; Non-confidential Production Volume Information Submitted by Companies for Chemicals Under the 1986-2002 Inventory Update Rule (IUR). Benzoic acid, 2-(acetyloxy)- (50-78-2). Available from, as of March 20, 2008: http://www.epa.gov/oppt/iur/tools/data/2002-vol.html

▸ *from HSDB*

CHEMICAL PROFILE: Production capacity in 1996: 36 million pounds

Anon. (1996) Chemical Marketing Reporter, January 8, 1996

▸ *from HSDB*

CHEMICAL PROFILE: Production capacity in 2000: 24 million pounds

Anon. (2000) Chemical Marketing Reporter, 258(6): 45, August 7, 2000

▸ *from HSDB*

CHEMICAL PROFILE: As of February 2003, there are no US producers of acetylsalicylic acid. Rhodia, the leading global aspirin producer, closed the last remaining acetylsalicylic acid plant in the US in February 2003, eliminating 20 million pounds of annual capacity. The company maintains aspirin production units at Saint Fons, France and Bangpoo, Thailand.

Kirschner M (2003) Chemical Market Reporter, August 18/25. 2003

▸ *from HSDB*

9.7 U.S. Imports

(1972) 2.04X10+8 GRAMS

SRI

▸ *from HSDB*

(1975) 1.42X10+8 GRAMS

SRI

▸ *from HSDB*

(1984) 1.65x10+9 g

BUREAU OF THE CENSUS. U.S. IMPORTS FOR CONSUMPTION AND GENERAL IMPORTS 1984 p.1-340

▸ *from HSDB*

CHEMICAL PROFILE: Aspirin imports were 2.6 million lb last year /1989/.

Kavaler AR; Chemical Marketing Reporter 237 (10): 50 (1990)

▸ *from HSDB*

CHEMICAL PROFILE: 1994, 2.5 million pounds

Anon. (1996) Chemical Marketing Reporter, January 8, 1996

▸ *from HSDB*

CHEMICAL PROFILE: 1998, 4.5 million pounds; 1999, 6.9 million pounds

Anon. (2000) Chemical Marketing Reporter, 258(6): 45, August 7, 2000

▸ *from HSDB*

9.8 U.S. Exports

(1972) 8.74X10+8 GRAMS (BULK)

SRI

▸ *from HSDB*

(1975) 1.06X10+8 GRAMS

SRI

▸ *from HSDB*

(1984) 1.26X10+9 g (bulk)

BUREAU OF THE CENSUS. U.S. EXPORTS, SCHEDULE E, 1984 p.2-83

▸ *from HSDB*

CHEMICAL PROFILE: Aspirin exports were 1.3 million lb /in 1989/.

Kavaler AR; Chemical Marketing Reporter 237 (10): 50 (1990)

from HSDB

CHEMICAL PROFILE: 1994, 2.5 million pounds

Anon. (1996) Chemical Marketing Reporter, January 8, 1996

from HSDB

CHEMICAL PROFILE: 1998, 2.2 million pounds; 1999, 900,000 pounds

Anon. (2000) Chemical Marketing Reporter, 258(6): 45, August 7, 2000

from HSDB

CHEMICAL PROFILE: 2001, 0.9 million pounds; 2002, 0.9 million pounds

Kirschner M (2003) Chemical Market Reporter, August 18/25 (2003)

from HSDB

10 Identification

10.1 Analytic Laboratory Methods

THIN LAYER CHROMATOGRAPHY. GAS LIQUID CHROMATOGRAPHY.

Sunshine, I. (ed.). CRC Handbook of Analytical Toxicology. Cleveland: The Chemical Rubber Co., 1969., p. 3

▸ *from HSDB*

GENERAL SAMPLE, SPECTROPHOTOMETRY.

Sunshine, I. (ed.). CRC Handbook of Analytical Toxicology. Cleveland: The Chemical Rubber Co., 1969., p. 3

▸ *from HSDB*

Analyte: aspirin; matrix: chemical identification; procedure: infrared absorption spectrophotometry with comparison to standards

U.S. Pharmacopeia. The United States Pharmacopeia, USP 30/The National Formulary, NF 25; Rockville, MD: U.S. Pharmacopeial Convention, Inc., p.1443 (2007)

▸ *from HSDB*

Analyte: aspirin; matrix: chemical identification; procedure: dissolution in water; reaction with ferric chloride to produce a violet-red color

U.S. Pharmacopeia. The United States Pharmacopeia, USP 30/The National Formulary, NF 25; Rockville, MD: U.S. Pharmacopeial Convention, Inc., p.1443 (2007)

▸ *from HSDB*

Analyte: aspirin; matrix: chemical purity; procedure: dissolution in sodium hydroxide; addition of phenolphthalein indicator; titration of excess sodium hydroxide with sulfuric acid

U.S. Pharmacopeia. The United States Pharmacopeia, USP 30/The National Formulary, NF 25; Rockville, MD: U.S. Pharmacopeial Convention, Inc., p.1443 (2007)

▸ *from HSDB*

Analyte: aspirin; matrix: pharmaceutical preparation (bolus; buffered tablet; capsule; delayed-release capsule; delayed-release tablet; extended-release tablet; suppository; tablet); procedure: dissolution in water; reaction with ferric chloride to produce a violet-red color (chemical identification)

U.S. Pharmacopeia. The United States Pharmacopeia, USP 30/The National Formulary, NF 25; Rockville, MD: U.S. Pharmacopeial Convention, Inc., p.1443 (2007)

▸ *from HSDB*

Analyte: aspirin; matrix: pharmaceutical preparation (bolus); procedure: retention time of the aspirin peak of the liquid chromatogram with comparison to standards (chemical identification)

U.S. Pharmacopeia. The United States Pharmacopeia, USP 30/The National Formulary, NF 25; Rockville, MD: U.S. Pharmacopeial Convention, Inc., p.1443 (2007)

▸ *from HSDB*

Analyte: aspirin; matrix: pharmaceutical preparation (bolus); procedure: liquid chromatography with detection at 254 nm and comparison to standards (chemical purity)

U.S. Pharmacopeia. The United States Pharmacopeia, USP 30/The National Formulary, NF 25; Rockville, MD: U.S. Pharmacopeial Convention, Inc., p.1443 (2007)

▸ *from HSDB*

Analyte: aspirin; matrix: pharmaceutical preparation (buffered tablet; capsule; delayed-release capsule; delayed-release tablet; extended-release tablet; suppository; tablet); procedure: infrared absorption spectrophotometry with comparison to standards (chemical identification)

U.S. Pharmacopeia. The United States Pharmacopeia, USP 30/The National Formulary, NF 25; Rockville, MD: U.S. Pharmacopeial Convention, Inc., p.1444 (2007)

▸ *from HSDB*

Analyte: aspirin; matrix: pharmaceutical preparation (capsule; suppository); procedure: ultraviolet absorption spectrophotometry at 280 nm with comparison to standards (chemical purity)

U.S. Pharmacopeia. The United States Pharmacopeia, USP 30/The National Formulary, NF 25; Rockville, MD: U.S. Pharmacopeial Convention, Inc., p.1444 (2007)

▸ *from HSDB*

Analyte: aspirin; matrix: pharmaceutical preparation (buffered tablet; delayed-release capsule; delayed-release tablet; effervescent tablet for oral solution; extended-release tablet; tablet); procedure: liquid chromatography with detection at 280 nm and comparison to standards (chemical purity)

U.S. Pharmacopeia. The United States Pharmacopeia, USP 30/The National Formulary, NF 25; Rockville, MD: U.S. Pharmacopeial Convention, Inc., p.1445 (2007)

▸ *from HSDB*

Analyte: aspirin; matrix: pharmaceutical preparation (effervescent tablet for oral solution); procedure: dissolution in hydrochloric acid; reaction with ferric chloride to produce a violet-red color (chemical identification)

U.S. Pharmacopeia. The United States Pharmacopeia, USP 30/The National Formulary, NF 25; Rockville, MD: U.S. Pharmacopeial Convention, Inc., p.1448 (2007)

▸ *from HSDB*

Analyte: aspirin; matrix: pharmaceutical preparation (effervescent tablet for oral solution); procedure: dissolution in water; reaction with calcium hydroxide to form a white precipitate (chemical identification)

U.S. Pharmacopeia. The United States Pharmacopeia, USP 30/The National Formulary, NF 25; Rockville, MD: U.S. Pharmacopeial Convention, Inc., p.1448 (2007)

▸ *from HSDB*

Analyte: acetylsalicylic acid; matrix: chemical identification; procedure: infrared absorption spectrophotometry with comparison to standards

Council of Europe, European Directorate for the Quality of Medicines. European Pharmacopoeia, 5th Ed., Volume 2; Strasbourg, France, p.917 (2004)

▸ *from HSDB*

Analyte: acetylsalicylic acid; matrix: chemical identification; procedure: reaction with dilute sodium hydroxide and dilute sulfuric acid produces a crystalline precipitate that melts between 156 deg C and 161 deg C

Council of Europe, European Directorate for the Quality of Medicines. European Pharmacopoeia, 5th Ed., Volume 2; Strasbourg, France, p.917 (2004)

▸ from HSDB

Analyte: acetylsalicylic acid; matrix: chemical purity; procedure: dissolution in alcohol; addition of sodium hydroxide and phenolphthalein indicator; titration with sulfuric acid

Council of Europe, European Directorate for the Quality of Medicines. European Pharmacopoeia, 5th Ed., Volume 2; Strasbourg, France, p.917 (2004)

▸ from HSDB

Analyte: acetylsalicylic acid; matrix: bulk material; procedure: micellar electrokinetic capillary electrophoresis with ultraviolet detection at 214 nm

Walker JA et al; J Forensic Sci 40: 6-9 (1995). As cited in: Lunn G; HPLC and CE Methods for Pharmaceutical Analysis. CD-ROM. New York, NY: John Wiley & Sons (2000)

▸ from HSDB

Analyte: acetylsalicylic acid; matrix: pharmaceutical preparation (tablet); procedure: micellar electrokinetic capillary electrophoresis with ultraviolet detection at 214 nm

Fujiwara S, Honda S; Anal Chem 59: 2773-2776 (1987). As cited in: Lunn G; HPLC and CE Methods for Pharmaceutical Analysis. CD-ROM. New York, NY: John Wiley & Sons (2000)

▸ from HSDB

Analyte: acetylsalicylic acid; matrix: pharmaceutical preparation (tablet); procedure: micellar electrokinetic capillary electrophoresis with ultraviolet detection at 214 nm

Boonkerd S et al; J Chromatogr A 695: 97-102 (1995). As cited in: Lunn G; HPLC and CE Methods for Pharmaceutical Analysis. CD-ROM. New York, NY: John Wiley & Sons (2000)

▸ from HSDB

Analyte: acetylsalicylic acid; matrix: pharmaceutical preparation (solution); procedure: capillary electrophoresis with ultraviolet detection at 214 nm

Swartz ME; J Liq Chromatogr 14: 923-938 (1991). As cited in: Lunn G; HPLC and CE Methods for Pharmaceutical Analysis. CD-ROM. New York, NY: John Wiley & Sons (2000)

▸ from HSDB

Analyte: acetylsalicylic acid; matrix: pharmaceutical preparation (solution); procedure: capillary electrophoresis with ultraviolet detection at 214 nm

McLaughlin GM et al; J Liq Chromatogr 15: 961-1021 (1992). As cited in: Lunn G; HPLC and CE Methods for Pharmaceutical Analysis. CD-ROM. New York, NY: John Wiley & Sons (2000)

▸ from HSDB

Analyte: acetylsalicylic acid; matrix: pharmaceutical preparation (solution); procedure: capillary electrophoresis with ultraviolet detection at 200 nm

Altria KD et al; J Pharm Biomed Anal 15: 1091-1101 (1997). As cited in: Lunn G; HPLC and CE Methods for Pharmaceutical Analysis. CD-ROM. New York, NY: John Wiley & Sons (2000)

▸ from HSDB

Analyte: acetylsalicylic acid; matrix: pharmaceutical preparation (bulk, tablet); procedure: high-performance liquid chromatography with ultraviolet detection at 254 nm

Pfeiffer CD, Pankey JW; J Pharm Sci 71: 511-514 (1982). As cited in: Lunn G; HPLC and CE Methods for Pharmaceutical Analysis. CD-ROM. New York, NY: John Wiley & Sons (2000)

▸ from HSDB

Analyte: acetylsalicylic acid; matrix: pharmaceutical preparation (tablet); procedure: high-performance liquid chromatography with ultraviolet detection at 295 nm

Fogel J et al; J Chromatogr 317: 507-511 (1984). As cited in: Lunn G; HPLC and CE Methods for Pharmaceutical Analysis. CD-ROM. New York, NY: John Wiley & Sons (2000)

▸ from HSDB

Analyte: acetylsalicylic acid; matrix: pharmaceutical preparation (tablet); procedure: high-performance liquid chromatography with ultraviolet detection at 300 nm

Galante RN et al; J Pharm Sci 73: 195-197 (1984). As cited in: Lunn G; HPLC and CE Methods for Pharmaceutical Analysis. CD-ROM. New York, NY: John Wiley & Sons (2000)

▸ from HSDB

Analyte: acetylsalicylic acid; matrix: pharmaceutical preparation (tablet); procedure: high-performance liquid chromatography with ultraviolet detection at 254 nm

Thomis R et al; J Pharm Sci 73: 1830-1833 (1984). As cited in: Lunn G; HPLC and CE Methods for Pharmaceutical Analysis. CD-ROM. New York, NY: John Wiley & Sons (2000)

▸ from HSDB

Analyte: acetylsalicylic acid; matrix: pharmaceutical preparation (aqueous suspension, oil, tablet); procedure: high-performance liquid chromatography with ultraviolet detection at 240 nm; limit of detection: 5 ug/mL

Walters MJ et al; J Assoc Off Anal Chem 73: 904-926 (1990). As cited in: Lunn G; HPLC and CE Methods for Pharmaceutical Analysis. CD-ROM. New York, NY: John Wiley & Sons (2000)

▸ from HSDB

Analyte: acetylsalicylic acid; matrix: pharmaceutical preparation (tablet); procedure: high-performance liquid chromatography with ultraviolet detection at 254 nm; limit of quantitation: 6 ug/mL

Sane RT et al; Indian Drugs 29: 240-244 (1992). As cited in: Lunn G; HPLC and CE Methods for Pharmaceutical Analysis. CD-ROM. New York, NY: John Wiley & Sons (2000)

▸ from HSDB

Analyte: acetylsalicylic acid; matrix: pharmaceutical preparation (powder); procedure: high-performance liquid chromatography with ultraviolet detection at 254 nm

Ferguson GK; J Chem Educ 75: 467-469 (1998). As cited in: Lunn G; HPLC and CE Methods for Pharmaceutical Analysis. CD-ROM. New York, NY: John Wiley & Sons (2000)

▸ from HSDB

10.2 Clinical Laboratory Methods

BLOOD, FLUOROMETRY; BLOOD, SPECTROPHOTOMETRY; URINE, SPECTROPHOTOMETRY.

Sunshine, I. (ed.). CRC Handbook of Analytical Toxicology. Cleveland: The Chemical Rubber Co., 1969., p. 3

▸ from HSDB

Analyte: acetylsalicylic acid; matrix: blood (serum); procedure: capillary electrophoresis with ultraviolet detection at 210 nm

Goto Y et al; J Chromatogr B 706: 329-335 (1998). As cited in: Lunn G; HPLC and CE Methods for Pharmaceutical Analysis. CD-ROM. New York, NY: John Wiley & Sons (2000)

▸ from HSDB

Analyte: acetylsalicylic acid; matrix: blood (plasma); procedure: high-performance liquid chromatography with ultraviolet detection at 237 nm; limit of quantitation: 20 ng/mL

Benedek IH et al; J Clin Pharmacol 35: 1181-1186 (1995). As cited in: Lunn G; HPLC and CE Methods for Pharmaceutical Analysis. CD-ROM. New York, NY: John Wiley & Sons (2000)

▸ *from HSDB*

Analyte: acetylsalicylic acid; matrix: blood (serum); procedure: high-performance liquid chromatography with ultraviolet detection at 254 nm

Nimura N et al; J Chromatogr A 689: 203-210 (1995). As cited in: Lunn G; HPLC and CE Methods for Pharmaceutical Analysis. CD-ROM. New York, NY: John Wiley & Sons (2000)

▸ *from HSDB*

Analyte: acetylsalicylic acid; matrix: blood (plasma, whole); procedure: high-performance liquid chromatography with ultraviolet detection at 233 nm

Tracqui A et al; J Forensic Sci 40: 254-262 (1995). As cited in: Lunn G; HPLC and CE Methods for Pharmaceutical Analysis. CD-ROM. New York, NY: John Wiley & Sons (2000)

▸ *from HSDB*

Analyte: acetylsalicylic acid; matrix: blood (plasma); procedure: high-performance liquid chromatography with ultraviolet detection at 225 nm

McMahon GP, Kelly MT; Anal Chem 70: 409-414 (1998). As cited in: Lunn G; HPLC and CE Methods for Pharmaceutical Analysis. CD-ROM. New York, NY: John Wiley & Sons (2000)

▸ *from HSDB*

Analyte: acetylsalicylic acid; matrix: blood (plasma), urine; procedure: high-performance liquid chromatography with ultraviolet detection at 280 nm; limit of detection: 2 ug/mL

Harrison LI et al; J Pharm Sci 69: 1268-1271 (1980). As cited in: Lunn G; HPLC and CE Methods for Pharmaceutical Analysis. CD-ROM. New York, NY: John Wiley & Sons (2000)

▸ *from HSDB*

Analyte: acetylsalicylic acid; matrix: urine; procedure: high-performance liquid chromatography with ultraviolet detection at 237 nm; limit of detection: 500 ng/mL

Beaumier PM et al; Equine Vet J 19: 207-213 (1987). As cited in: Lunn G; HPLC and CE Methods for Pharmaceutical Analysis. CD-ROM. New York, NY: John Wiley & Sons (2000)

▸ *from HSDB*

11 Safety and Hazards

11.1 Hazards Identification

11.1.1 GHS Classification

Signal: Warning
GHS Hazard Statements
Aggregated GHS information provided by 132 companies from 19 notifications to the ECHA C&L Inventory. Each notification may be associated with multiple companies.

Reported as not meeting GHS hazard criteria by 1 of 132 companies. For more detailed information, please visit ECHA C&L website

Of the 18 notification(s) provided by 131 of 132 companies with hazard statement code(s):

H302 (91.6%): Harmful if swallowed [Warning Acute toxicity, oral]
H315 (33.59%): Causes skin irritation [Warning Skin corrosion/irritation]
H319 (37.4%): Causes serious eye irritation [Warning Serious eye damage/eye irritation]
H335 (32.06%): May cause respiratory irritation [Warning Specific target organ toxicity, single exposure; Respiratory tract irritation]

Information may vary between notifications depending on impurities, additives, and other factors. The percentage value in parenthesis indicates the notified classification ratio from companies that provide hazard codes. Only hazard codes with percentage values above 10% are shown.

Precautionary Statement Codes
P261, P264, P270, P271, P280, P301+P312, P302+P352, P304+P340, P305+P351+P338, P312, P321, P330, P332+P313, P337+P313, P362, P403+P233, P405, and P501
(The corresponding statement to each P-code can be found here.)

from European Chemicals Agency (ECHA)

View all (2) GHS Classification entries

11.1.2 Health Hazard

Exposure Routes: inhalation, ingestion, skin and/or eye contact Symptoms: Irritation eyes, skin, upper respiratory system; increased blood clotting time; nausea, vomiting; liver, kidney injury Target Organs: (NIOSH, 2016)

from CAMEO Chemicals

11.1.3 Fire Hazard

This chemical is combustible. (NTP, 1992)

from CAMEO Chemicals

Combustible. Finely dispersed particles form explosive mixtures in air.

from ILO-ICSC

11.1.4 Fire Potential

SLIGHT WHEN EXPOSED TO HEAT OR FLAME

Sax, N.I. Dangerous Properties of Industrial Materials. 6th ed. New York, NY: Van Nostrand Reinhold, 1984., p. 88

from HSDB

11.1.5 Skin, Eye, and Respiratory Irritations

Aspirin is an acute irritant to ... the skin and eyes. Direct contact with the eye is painful ...

American Conference of Governmental Industrial Hygienists. Documentation of the TLV's and BEI's with Other World Wide Occupational Exposure Values. CD-ROM Cincinnati, OH 45240-1634 2007.

from HSDB

Aspirin is a known respiratory ... allergen.

American Conference of Governmental Industrial Hygienists. Documentation of the TLV's and BEI's with Other World Wide Occupational Exposure Values. CD-ROM Cincinnati, OH 45240-1634 2007.

from HSDB

11.2 Safety and Hazard Properties

11.2.1 Flammability

Combustible Powder; explosion hazard if dispersed in air.

from The National Institute for Occupational Safety and Health (NIOSH)

11.2.2 Physical Dangers

Dust explosion possible if in powder or granular form, mixed with air.

from ILO-ICSC

11.2.3 Chemical Dangers

The solution in water is a weak acid.

from ILO-ICSC

11.2.4 OSHA Standards

Vacated 1989 OSHA PEL TWA 5 mg/cu m is still enforced in some states.

NIOSH. NIOSH Pocket Guide to Chemical Hazards. DHHS (NIOSH) Publication No. 97-140. Washington, D.C. U.S. Government Printing Office, 1997., p. 359

from HSDB

11.2.5 NIOSH Recommendations

Recommended Exposure Limit: 10 Hr Time-Weighted Avg: 5 mg/cu m.

NIOSH. NIOSH Pocket Guide to Chemical Hazards & Other Databases CD-ROM. Department of Health & Human Services, Centers for Disease Prevention & Control. National Institute for Occupational Safety & Health. DHHS (NIOSH) Publication No. 2005-151 (2005)

from HSDB

11.3 First Aid Measures

11.3.1 First Aid

EYES: First check the victim for contact lenses and remove if present. Flush victim's eyes with water or normal saline solution for 20 to 30 minutes while simultaneously calling a hospital or poison control center. Do not put any ointments, oils, or medication in the victim's eyes without specific instructions from a physician. IMMEDIATELY transport the victim after flushing eyes to a hospital even if no symptoms (such as redness or irritation) develop. SKIN: IMMEDIATELY flood affected skin with water while removing and isolating all contaminated clothing. Gently wash all affected skin areas thoroughly with soap and water. If symptoms such as redness or irritation develop, IMMEDIATELY call a physician and be prepared to transport the victim to a hospital for treatment. INHALATION: IMMEDIATELY leave the contaminated area; take deep breaths of fresh air. If symptoms (such as wheezing, coughing, shortness of breath, or burning in the mouth, throat, or chest) develop, call a physician and be prepared to transport the victim to a hospital. Provide proper respiratory protection to rescuers entering an unknown atmosphere. Whenever possible, Self-Contained Breathing Apparatus (SCBA) should be used; if not available, use a level of protection greater than or equal to that advised under Protective Clothing. INGESTION: DO NOT INDUCE VOMITING. If the victim is conscious and not convulsing, give 1 or 2 glasses of water to dilute the chemical and IMMEDIATELY call a hospital or poison control center. Be prepared to transport the victim to a hospital if advised by a physician. If the victim is convulsing or unconscious, do not give anything by mouth, ensure that the victim's airway is open and lay the victim on his/her side with the head lower than the body. DO NOT INDUCE VOMITING. IMMEDIATELY transport the victim to a hospital. (NTP, 1992)

from CAMEO Chemicals

(See procedures)
Eye: Irrigate immediately
Skin: Soap wash
Breathing: Respiratory support
Swallow: Medical attention immediately

from The National Institute for Occupational Safety and Health (NIOSH)

11.3.2 Inhalation First Aid

Fresh air, rest. Refer for medical attention.

from ILO-ICSC

11.3.3 Skin First Aid

Rinse skin with plenty of water or shower. Refer for medical attention .

from ILO-ICSC

11.3.4 Eye First Aid

First rinse with plenty of water for several minutes (remove contact lenses if easily possible), then refer for medical attention.

from ILO-ICSC

11.3.5 Ingestion First Aid

Rinse mouth. Refer for medical attention .

from ILO-ICSC

11.4 Fire Fighting Measures

11.4.1 Fire Fighting

Fires involving this material can be controlled with a dry chemical, carbon dioxide or Halon extinguisher. A water spray may also be used. (NTP, 1992)

from CAMEO Chemicals

Use water spray, powder, foam, carbon dioxide.

from ILO-ICSC

11.5 Accidental Release Measures

11.5.1 Isolation and Evacuation

Excerpt from ERG Guide 154 [Substances - Toxic and/or Corrosive (Non-Combustible)]: As an immediate precautionary measure, isolate spill or leak area in all directions for at least 50 meters (150 feet) for liquids and at least 25 meters (75 feet) for solids. SPILL: Increase, in the downwind direction, as necessary, the isolation distance shown above. FIRE: If tank, rail car or tank truck is involved in a fire, ISOLATE for 800 meters (1/2 mile) in all directions; also, consider initial evacuation for 800 meters (1/2 mile) in all directions. (ERG, 2016)

from CAMEO Chemicals

11.5.2 Spillage Disposal

Personal protection: particulate filter respirator adapted to the airborne concentration of the substance. Sweep spilled substance into covered containers. If appropriate, moisten first to prevent dusting. Carefully collect remainder. Then store and dispose of according to local regulations.

from ILO-ICSC

11.5.3 Disposal Methods

SRP: At the time of review, criteria for land treatment or burial (sanitary landfill) disposal practices are subject to significant revision. Prior to implementing land disposal of waste residue (including waste sludge), consult with environmental regulatory agencies for guidance on acceptable disposal practices.

from HSDB

11.5.4 Other Preventative Measures

Contact lenses should not be worn when working with this chemical.

NIOSH. NIOSH Pocket Guide to Chemical Hazards & Other Databases CD-ROM. Department of Health & Human Services, Centers for Disease Prevention & Control. National Institute for Occupational Safety & Health. DHHS (NIOSH) Publication No. 2005-151 (2005)

from HSDB

The worker should immediately wash the skin when it becomes contaminated.

NIOSH. NIOSH Pocket Guide to Chemical Hazards & Other Databases CD-ROM. Department of Health & Human Services, Centers for Disease Prevention & Control. National Institute for Occupational Safety & Health. DHHS (NIOSH) Publication No. 2005-151 (2005)

from HSDB

Workers whose clothing may have become contaminated should change into uncontaminated clothing before leaving the work premises.

NIOSH. NIOSH Pocket Guide to Chemical Hazards & Other Databases CD-ROM. Department of Health & Human Services, Centers for Disease Prevention & Control. National Institute for Occupational Safety & Health. DHHS (NIOSH) Publication No. 2005-151 (2005)

from HSDB

SRP: The scientific literature for the use of contact lenses in industry is conflicting. The benefit or detrimental effects of wearing contact lenses depend not only upon the substance, but also on factors including the form of the substance, characteristics and duration of the exposure, the uses of other eye protection equipment, and the hygiene of the lenses. However, there may be individual substances whose irritating or corrosive properties are such that the wearing of contact lenses would be harmful to the eye. In those specific cases, contact lenses should not be worn. In any event, the usual eye protection equipment should be worn even when contact lenses are in place.

from HSDB

SRP: Contaminated protective clothing should be segregated in such a manner so that there is no direct personal contact by personnel who handle, dispose, or clean the clothing. Quality assurance to ascertain the completeness of the cleaning procedures should be implemented before the decontaminated protective clothing is returned for reuse by the workers. Contaminated clothing should not be taken home at end of shift, but should remain at employee's place of work for cleaning.

from HSDB

11.6 Handling and Storage

11.6.1 Nonfire Spill Response

SMALL SPILLS AND LEAKAGE: Should a spill occur while you are handling this chemical, FIRST REMOVE ALL SOURCES OF IGNITION, then you should dampen the solid spill material with 60-70% ethanol and transfer the dampened material to a suitable container. Use absorbent paper dampened with 60-70% ethanol to pick up any remaining material. Seal the absorbent paper, and any of your clothes, which may be contaminated, in a vapor-tight plastic bag for eventual disposal. Solvent wash all contaminated surfaces with 60-70% ethanol followed by washing with a soap and water solution. Do not reenter the contaminated area until the Safety Officer (or other responsible person) has verified that the area has been properly cleaned. STORAGE PRECAUTIONS: You should store this material under ambient temperatures and protect it from moisture. (NTP, 1992)

from CAMEO Chemicals

11.6.2 Safe Storage

Well closed.

from ILO-ICSC

11.6.3 Storage Conditions

Chewable aspirin tablets containing 81 mg of the drug should be stored in child-resistant containers holding not more than 36 tablets each in order to limit the potential toxicity associated with accidental ingestion in children. Aspirin suppositories should be stored at 2-15 deg C.

McEvoy, G.K. (ed.). AHFS Drug Information 90. Bethesda, MD: American Society of Hospital Pharmacists, Inc., 1990 (Plus Supplements 1990)., p. 998

from HSDB

11.7 Exposure Control and Personal Protection

11.7.1 REL

TWA 5 mg/m^3

from The National Institute for Occupational Safety and Health (NIOSH)

11.7.2 PEL

none See Appendix G

from The National Institute for Occupational Safety and Health (NIOSH)

11.7.3 REL-TWA

5 mg/m³

from OSHA Occupational Chemical DB

11.7.4 IDLH

N.D.
See: IDLH INDEX

from The National Institute for Occupational Safety and Health (NIOSH)

11.7.5 Threshold Limit Values

8 hr Time Weighted Avg (TWA): 5 mg/cu m.

American Conference of Governmental Industrial Hygienists TLVs and BEIs. Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Cincinnati, OH, 2007, p. 10

from HSDB

Excursion Limit Recommendation: Excursions in worker exposure levels may exceed three times the TLV-TWA for no more than a total of 30 min during a work day, and under no circumstances should they exceed five times the TLV-TWA, provided that the TLV-TWA is not exceeded.

American Conference of Governmental Industrial Hygienists TLVs and BEIs. Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Cincinnati, OH, 2007, p. 5

from HSDB

5 mg/m3, as TWA

from ILO-ICSC

11.7.6 Inhalation Risk

Evaporation at 20°C is negligible; a harmful concentration of airborne particles can, however, be reached quickly when dispersed, especially if powdered.

from ILO-ICSC

11.7.7 Effects of Short Term Exposure

The substance is irritating to the eyes, skin and respiratory tract. Ingestion of large amounts could cause effects on the blood and central nervous system.

from ILO-ICSC

11.7.8 Effects of Long Term Exposure

Animal tests show that this substance possibly causes toxic effects upon human reproduction.

from ILO-ICSC

11.7.9 Personal Protection

(See protection codes)
Skin: Prevent skin contact
Eyes: Prevent eye contact
Wash skin: When contaminated
Remove: No recommendation
Change: Daily
Provide: Eyewash, Quick drench

from The National Institute for Occupational Safety and Health (NIOSH)

11.7.10 Respirator Recommendations

Important additional information about respirator selection

from The National Institute for Occupational Safety and Health (NIOSH)

11.7.11 Fire Prevention

NO open flames. Closed system, dust explosion-proof electrical equipment and lighting. Prevent deposition of dust.

from ILO-ICSC

11.7.12 Exposure Prevention

PREVENT DISPERSION OF DUST!

from ILO-ICSC

11.7.13 Inhalation Prevention

Use ventilation (not if powder).

from ILO-ICSC

11.7.14 Skin Prevention

Protective gloves.

from ILO-ICSC

11.7.15 Eye Prevention

Wear safety goggles.

from ILO-ICSC

11.7.16 Ingestion Prevention

Do not eat, drink, or smoke during work.

from ILO-ICSC

11.7.17 Protective Equipment and Clothing

Skin: Wear appropriate personal protective clothing to prevent skin contact. Eyes: Wear appropriate eye protection to prevent eye contact. Wash skin: The worker should immediately wash the skin when it becomes contaminated. Remove: No recommendation is made specifying the need for removing clothing that becomes wet or contaminated. Change: Workers whose clothing may have become contaminated should change into uncontaminated clothing before leaving the work premise. Provide: Eyewash fountains should be provided in areas where there is any possibility that workers could be exposed to the substance; this is irrespective of the recommendation involving the wearing of eye protection. Facilities for quickly drenching the body should be provided within the immediate work area for emergency use where there is a possibility of exposure. [Note: It is intended that these facilities provide a sufficient quantity or flow of water to quickly remove the substance from any body areas likely to be exposed. The actual determination of what constitutes an adequate quick drench facility depends on the specific circumstances. In certain instances, a deluge shower should be readily available, whereas in others, the availability of water from a sink or hose could be considered adequate.] (NIOSH, 2016)

from CAMEO Chemicals

Wear appropriate personal protective clothing to prevent skin contact.

NIOSH. NIOSH Pocket Guide to Chemical Hazards & Other Databases CD-ROM. Department of Health & Human Services, Centers for Disease Prevention & Control. National Institute for Occupational Safety & Health. DHHS (NIOSH) Publication No. 2005-151 (2005)

from HSDB

Wear appropriate eye protection to prevent eye contact.

NIOSH. NIOSH Pocket Guide to Chemical Hazards & Other Databases CD-ROM. Department of Health & Human Services, Centers for Disease Prevention & Control. National Institute for Occupational Safety & Health. DHHS (NIOSH) Publication No. 2005-151 (2005)

from HSDB

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NIOSH. NIOSH Pocket Guide to Chemical Hazards & Other Databases CD-ROM. Department of Health & Human Services, Centers for Disease Prevention & Control. National Institute for Occupational Safety & Health. DHHS (NIOSH) Publication No. 2005-151 (2005)

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NIOSH. NIOSH Pocket Guide to Chemical Hazards & Other Databases CD-ROM. Department of Health & Human Services, Centers for Disease Prevention & Control. National Institute for Occupational Safety & Health. DHHS (NIOSH) Publication No. 2005-151 (2005)

from HSDB

11.8 Stability and Reactivity

11.8.1 Air and Water Reactions

Slowly hydrolyzes in moist air. Has been involved in dust cloud explosions. Water insoluble. Solution in water is acid to methyl red indicator.

from CAMEO Chemicals

11.8.2 Reactive Group

Acids, Carboxylic
Esters, Sulfate Esters, Phosphate Esters, Thiophosphate Esters, and Borate Esters

from CAMEO Chemicals

11.8.3 Reactivity Profile

The active ingredient in common aspirin. Incompatible with oxidizers and strong acids. Also incompatible with strong bases. May react with water or nucleophiles (e.g. amines and hydroxy groups). May also react with acetanilide, amidopyrine, phenazone, hexamine, iron salts, phenobarbitone sodium, quinine salts, potassium and sodium iodides, alkali hydroxides, carbonates, stearates and paracetanol. (NTP, 1992)

from CAMEO Chemicals

11.8.4 Reactivities and Incompatibilities

Solutions of alkali hydroxides or carbonates, strong oxidizers, moisture [Note: Slowly hydrolyzes in moist air to salicyclic & acetic acids].

NIOSH. NIOSH Pocket Guide to Chemical Hazards & Other Databases CD-ROM. Department of Health & Human Services, Centers for Disease Prevention & Control. National Institute for Occupational Safety & Health. DHHS (NIOSH) Publication No. 2005-151 (2005)

from HSDB

Solutions of alkali hydroxides or carbonates, strong oxidizers, moisture [Note: Slowly hydrolyzes in moist air to salicyclic & acetic acids.]

from The National Institute for Occupational Safety and Health (NIOSH)

11.9 Transport Information

11.9.1 DOT Label

Poison

from CAMEO Chemicals

12 Toxicity

12.1 Toxicological Information

12.1.1 Heptatoxicity

Patients on long term, moderate-to-high dose aspirin therapy frequently have elevations in serum ALT levels. With high doses, ALT elevations are common and can be marked and associated with mild increases in alkaline phosphatase and bilirubin. The more dramatic examples of aspirin hepatotoxicity usually occur with doses of 1,800 to 3,200 mg daily (>100 mg/kg) and with salicylate levels of greater than 25 mg/dL, but mild-to-moderate ALT elevations occur with even lower doses and lower serum levels. These abnormalities resolve rapidly with discontinuation of aspirin, but instances of resolution despite continuation of aspirin in the same or lower doses (adaptation) have also been described. The hepatotoxicity of aspirin is usually mild and asymptomatic, although with higher doses symptoms of nausea, anorexia and abdominal pain and even encephalopathy with signs of hepatic dysfunction (hyperammonemia and coagulopathy) can occur. Bilirubin elevations are usually mild or absent. Mild eosinophilia may accompany the enzyme elevations, but rash, fever and other allergic manifestations are rare. Liver biopsy histology generally shows minimal injury despite the height of the enzyme elevations; electron microscopy may reveal fat and mitochondrial abnormalities. Aspirin can often be continued in lower doses safely.

from LiverTox

12.1.2 NIOSH Toxicity Data

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1 to 5 of 289 View More

Measurement	System	Route/Organism	Dose	Effect	Date
Skin and Eye Irritation		skin /human	10%		October 2017
Mutation Data	Cytogenetic Analysis	fibroblast/human	100 mg/L		October 2017
Mutation Data	Cytogenetic Analysis	leukocyte/human	100 µg/L		October 2017
Mutation Data	Cytogenetic Analysis	lymphocyte/human	10 mg/L		October 2017
Mutation Data	Cytogenetic Analysis	lung/hamster	1660 mg/L		October 2017

from The National Institute for Occupational Safety and Health (NIOSH)

12.1.3 Carcinogen

Not listed

from OSHA Occupational Chemical DB

12.1.4 Exposure Routes

The substance can be absorbed into the body by inhalation and by ingestion.

from ILO-ICSC

inhalation, ingestion, skin and/or eye contact

from The National Institute for Occupational Safety and Health (NIOSH)

12.1.5 Symptoms

irritation eyes, skin, upper respiratory system; increased blood clotting time; nausea, vomiting; liver, kidney injury

from The National Institute for Occupational Safety and Health (NIOSH)

12.1.6 Inhalation Symptoms

Cough. Sore throat.

from ILO-ICSC

12.1.7 Skin Symptoms

Redness.

from ILO-ICSC

12.1.8 Eye Symptoms

Redness. Pain.

from ILO-ICSC

12.1.9 Ingestion Symptoms

Nausea. Vomiting.

from ILO-ICSC

12.1.10 Target Organs

Eyes, skin, respiratory system, blood, liver, kidneys

from The National Institute for Occupational Safety and Health (NIOSH)

12.1.11 Interactions

Prolonged concurrent use of acetaminophen with a salicylate is not recommended because chronic, high-dose administration of the combined analgesics (1.35 g daily, or cumulative ingestion of 1 kg annually, for 3 years or longer) significantly increases the risk of analgesic nephropathy, renal papillary necrosis, end-stage renal disease, and cancer of the kidney or urinary bladder; also, recommended that for short-term use the combined dose of acetaminophen plus a salicylate not exceed that recommended for acetaminophen or a salicylate given individually. /Salicylates/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2577

from HSDB

The possibility should be considered that additive or multiple effects leading to impaired blood clotting and/or increased risk of bleeding may occur if a salicylate, especially aspirin, is used concurrently with any medication having a significant potential for causing hypoprothrombinemia, thrombocytopenia, or gastrointestinal ulceration or hemorrhage.

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2577

from HSDB

Aspirin may decrease the bioavailability of many nonsteroidal anti-inflammatory drugs (NSAIDs), including diflunisal, fenoprofen, indomethacin, meclofenamate, piroxicam (up to 80% of the usual plasma concentration), and the active sulfide metabolite of sulindac; aspirin has also been shown to decrease the protein binding and increase the plasma clearance of ketoprofen, and to decrease the formation and excretion of ketoprofen conjugates. Concurrent use of other NSAIDs with aspirin may also increase the risk of bleeding at sites other than the gastrointestinal tract because of additive inhibition of platelet aggregation.

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2577

from HSDB

Concurrent use of these medications /alcohol or other nonsteroidal anti-inflammatory drugs (NSAIDs)/ with a salicylate may increase the risk of gastrointestinal side effects, including ulceration and gastrointestinal blood loss; also, concurrent use of a salicylate with an NSAID may increase the risk of severe gastrointestinal side effects without providing additional symptomatic relief and is therefore not recommended. /Salicylate/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2577

from HSDB

Aspirin may increase urinary excretion of ascorbic acid; clinical significance is unclear, but some clinicians recommend ascorbic acid supplementation in patients receiving prolonged high-dose aspirin therapy.

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2577

from HSDB

Salicylate may displace a coumarin- or indanedione-derivative anticoagulant from its protein-binding sites, and , in high doses, may cause hypoprothrombinemia, leading to increased anticoagulation and risk of bleeding. /Salicylates/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2577

from HSDB

Metabolic acidosis induced by carbonic anhydrase inhibitors may increase penetration of salicylate into the brain and increase the risk of salicylate toxicity in patients taking large (antirheumatic) doses of salicylate; if acetazolamide is used to produce forced alkaline diuresis in the treatment of salicylate poisoning, the increased risk of severe metabolic acidosis and increased salicylate toxicity must be considered and an alkaline intravenous solution given concurrently. /Salicylates/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2577

from HSDB

Alkalinization of the urine by these medications /urinary alkalizers such as carbonic anhydrase inhibitors; citrates; sodium bicarbonate; or chronic high-dose use of calcium- and/or magnesium-containing antacids/ increase salicylate excretion, leading to decreased salicylate plasma concentrations, reduced effectiveness, and shortened duration of action; also, withdrawal of a urinary alkalizer from a patient stabilized on a salicylate may increase the plasma salicylate concentration to a toxic level; however, the antacids present in buffered aspirin formulations may not be present in sufficient quantity to alkalinize the urine. /Salicylates/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2577

from HSDB

Acidification of the urine by these medications /urinary acidifiers such as ammonium chloride, ascorbic acid (Vitamin C), potassium or sodium phosphates/ decreases salicylate excretion, leading to increased salicylate plasma concentrations; initiation of therapy with these medications in patients stabilized on a salicylate may lead to toxic salicylate concentrations. /Salicylates/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2577

from HSDB

Concurrent use of /topical salicylic acid or other salicylates/ with systemic salicylates may increase the risk of salicylate toxicity if significant quantities are absorbed. /Salicylates/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2578

▸ from HSDB

In theory, aspirin may competitively inhibit the hepatic glucuronidation and decrease the clearance of zidovudine, leading to potentiation of zidovudine toxicity; the possibility must be considered that aspirin toxicity may also be increased.

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2578

▸ from HSDB

In addition to increasing the risk of ototoxicity, concurrent use of furosemide with high doses of salicylate may lead to salicylate toxicity because of competition for renal excretory sites. /Salicylates/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2578

▸ from HSDB

Concurrent use /with cellulose-containing laxatives/ may reduce the salicylate effect because of physical binding or other absorptive hindrance; medications should be administered 2 hours apart. /Salicylates/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2578

▸ from HSDB

Salicylates may decrease hydantoin metabolism, leading to increases in hydantoin plasma concentrations, efficacy, and/or toxicity; adjustment of hydantoin dosage may be required when chronic salicylate therapy is initiated or discontinued. /Salicylates/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2578

▸ from HSDB

Effects of these medications /oral antidiabetic agents or insulin/ may be increased by large doses of salicylates; dosage adjustments may be necessary; potentiation of oral antidiabetic agents may be caused partially by displacement from serum proteins; glipizide and glyburide, because of their nonionic binding characteristics, may not be affected as much as the other oral agents; however, caution in concurrent use is recommended. /Salicylates/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2578

▸ from HSDB

Ingestion of large repeated doses /of bismuth subsalicylate/ as for traveler's diarrhea may produce substantial plasma salicylate concentrations; concurrent use with large doses of analgesic salicylates may increase the risk of salicylate toxicity. /Salicylates/

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2578

▸ from HSDB

Two elderly patients, who were chronically receiving aspirin, developed lethargy, incontinence, and confusion after dosing with acetazolamide. Unbound plasma acetazolamide concentrations were elevated and plasma protein binding was reduced, suggesting an interaction with aspirin. In vitro studies demonstrated a concentration-dependent effect of salicylate on acetazolamide binding to serum proteins. At a therapeutic serum acetazolamide level of 8.0 micrograms/ml, the unbound percentage of acetazolamide in serum was 3.3% and increased to 11.0% and 30.0%, with serum salicylate levels of 200 and 386 micrograms/ml, respectively. Furthermore, the apparent association constant of acetazolamide for binding to serum proteins was decreased by 58% and 86% of its control value at these respective salicylate concentrations. The maximal binding capacity of serum for acetazolamide was not affected by salicylate. Pharmacokinetic studies in four volunteers showed that the plasma protein binding and renal clearance of acetazolamide were significantly reduced during chronic salicylate dosing. Salicylate appears to competitively inhibit the plasma protein binding of acetazolamide and simultaneously to inhibit acetazolamide renal tubular secretion. Caution is advised when acetazolamide and salicylate are used concurrently.
Abstract: PubMed

Sweeney KR et al; Clin Pharmacol Ther 40 (5): 518-24 (1986)

▸ from HSDB

Selective serotonin reuptake inhibitors (SSRIs) have been suspected of increasing the risk of bleeding. ... All users of antidepressants in the county of North Jutland, Denmark, from January 1, 1991, to December 31, 1995, were identified in the Pharmaco-Epidemiologic Prescription Database of North Jutland. In the Hospital Discharge Register, hospitalizations for upper GI bleeding were searched among the 26 005 users of antidepressant medications and compared with the number of hospitalizations in the population of North Jutland who did not receive prescriptions for antidepressants. During periods of SSRI use without use of other drugs associated with upper GI bleeding, /investigators/ observed 55 upper GI bleeding episodes, which was 3.6 times more than expected (95% confidence interval, 2.7-4.7), corresponding to a rate difference of 3.1 per 1000 treatment years. Combined use of an SSRI and nonsteroidal anti-inflammatory drugs or low-dose aspirin increased the risk to 12.2 (95% confidence interval, 7.1-19.5) and 5.2 (95% confidence interval, 3.2-8.0), respectively. Non-SSRIs increased the risk of upper GI bleeding to 2.3 (95% confidence interval, 1.5-3.4), while antidepressants without action on the serotonin receptor had no significant effect on the risk of upper GI bleeding. The risk with SSRI use returned to unity after termination of SSRI use, while the risks were similarly increased during periods of use and nonuse of non-SSRIs.
Abstract: PubMed

Dalton SO et al; Arch Intern Med 163 (1): 59-64 (2003)

▸ from HSDB

... The effects of bacterial endotoxin (LPS) on the toxicities of salicylic acid, the main metabolite of aspirin, were investigated in rats. The following results were obtained: 1) The acute toxicity of salicylic acid was significantly potentiated by LPS in male rats. The LD50 of salicylic acid with LPS was about one third of that of salicylic acid alone. 2) The increase of maternal body weight was inhibited significantly after administration of salicylic acid (383 mg/kg, p.o.) with LPS (20 ug/kg, i.v.), but not after administration of salicylic acid alone. 3) The fetal toxicity of salicylic acid including fetal death, resorption, growth retardation and skeletal variations was slightly observed in the dam receiving a single dose of salicylic acid on the 15th day of pregnancy, but it was markedly increased by LPS (20 ug/kg, i.v.). 4) The half-life period of salicylic acid in plasma was increased significantly by the co-administration of LPS in male rats after administration of aspirin or salicylic acid. All of these phenomena in the rats given salicylic acid closely resembled the phenomena previously reported in the rats given aspirin. These results suggest that salicylic acid might play a main role in the acute and fetal toxicities of aspirin, and one of the mechanism of the enhancement effect by LPS on aspirin-induced fetal toxicity might be related to the increase of salicylic acid concentration in the fetus. /Salicylic acid/
Abstract: PubMed

Itami T, Kanoh S; Nippon Yakurigaku Zasshi 84 (5): 411-6 (1984)

▸ from HSDB

The Clopidogrel in Unstable Angina to Prevent Recurrent Events (CURE) study showed that patients with unstable angina pectoris (UAP) and non-ST-elevation myocardial infarction (NSTEMI) benefit from combined therapy with acetylsalicylic acid (ASA) and clopidogrel. However, only patients entering clinical randomized trials were studied. We sought to assess whether the risk of bleeding increased after the introduction of the CURE criteria in an unselected population of Danish patients with NSTEMI or UAP. ... The CURE criteria were implemented in the Department of Cardiology, Odense University Hospital, in December 2001. Two consecutive one-year periods were studied: period 1, December 2000-November 2001, and period 2, December 2001-November 2002. Patient charts were reviewed, and major bleeding complications and the primary clinical end point (non-fatal myocardial infarction, stroke or death) was registered. Follow-up took place one year later. RESULTS: In all, 290 patients were included in period 1 and 189 in

period 2. During period 1, there were 12 (4.1%) and during period 2, 21 (11.1%) major bleeding events (odds ratio 3.07; 95% CI 1.42-6.65; p = 0.005). Compared with the patients treated with clopidogrel and ASA in the CURE study, we also found a three times greater risk of major bleeding in period 2. In particular, patients over 70 years of age and patients undergoing bypass surgery were at heightened risk. The incidence of the primary clinical end point was higher in both period 1 and period 2 than in the CURE study. ... /This/ study demonstrates an increased risk of major bleeding in unselected patients receiving combination therapy with ASA and clopidogrel after UAP or NSTEMI. Major bleeding complications most frequently occur in patients above 70 years of age and following bypass surgery.
Abstract: PubMed

Kjaer J et al; Ugeskr Laeger 168 (38): 3209-14 (2006)

from HSDB

The serum levels of free fatty acids were determined before and after an oral dose of 1000 mg acetylsalicylic acid during the 7th-11th wk of pregnancy in 11 women undergoing abortion & in 8 women undergoing hysterectomy because of fibroids. Thirteen healthy women were controls. Fatty acid levels, including myristic acid were significantly lower in pregnant women than in the hysterectomized group of patients. Treatment with acetylsalicylic acid tended to increase the level of many fatty acids in serum.

HAATAJA M ET AL; PROSTAGLANDINS, LEUKOTRIENES MED 9 (1): 61 (1982)

from HSDB

12.1.12 Toxicity Summary

Toxicity

Oral, mouse: LD_{50} = 250 mg/kg; Oral, rabbit: LD_{50} = 1010 mg/kg; Oral, rat: LD_{50} = 200 mg/kg. Effects of overdose include: tinnitus, abdominal pain, hypokalemia, hypoglycemia, pyrexia, hyperventilation, dysrhythmia, hypotension, hallucination, renal failure, confusion, seizure, coma, and death.

from DrugBank

IDENTIFICATION: Acetylsalicylic acid is colorless or white crystals or white crystalline powder or granules; odorless or almost odorless with a slight acid taste. It is soluble in water. Indications: It is used as an analgesic for the treatment of mild to moderate pain, as an anti-inflammatory agent for the treatment of soft tissue and joint inflammation, and as an antipyretic drug. In low doses salicylate is used for the prevention of thrombosis. HUMAN EXPOSURE: The toxic effects of salicylate are complex. The following appear to be the principal primary effects of salicylate in overdose: Stimulation of the respiratory center; inhibition of citric acid cycle (carbohydrate metabolism); stimulation of lipid metabolism; inhibition of amino acid metabolism; and uncoupling of oxidative phosphorylation. Respiratory alkalosis, metabolic acidosis, water and electrolyte loss occur as the principal secondary consequences of salicylate intoxication. Central nervous system toxicity (including tinnitus, hearing-loss, convulsions and coma), hypoprothrombinemia and non-cardiogenic pulmonary edema may also occur, though for some the mechanism remains uncertain. Target organs: The target organs are: all tissues (whose cellular metabolism is affected), but in particular the liver, kidneys, lungs and the VIIIth cranial nerve. Summary of clinical effects: the following are symptoms of intoxication: Nausea, vomiting, epigastric discomfort, gastrointestinal bleeding (typically with chronic and rarely with acute intoxication); tachypnea and hyperpnea; tinnitus, deafness, sweating, vasodilatation, hyperpyrexia (rare), dehydration; irritability, tremor, blurring of vision, subconjunctival haemorrhages. The following are the effects on blood glucose: hyper- or hypoglycemia; effects on blood: hypoprothrombinemia; effects on liver: increased serum aminotransferase activities (SGOT and SGPT). Non-cardiogenic pulmonary edema; confusion, delirium, stupor, asterixis, coma, cerebral edema (with severe intoxication only); acute renal failure; cardio-respiratory arrest (with severe intoxication only). Absorption by route of exposure: After oral administration, 80 - 100% will be absorbed in the stomach and in the small intestine. However, bioavailability is lower because partial hydrolysis occurs during absorption and there is a "first-pass" effect in the liver. The non-protein bound fraction of salicylate increases with the total plasma concentration, and the binding capacity of albumin is partially saturated at therapeutic concentrations of salicylate. The greater proportion of unbound drug found at high concentrations will mean that greater toxicity will result than would be expected from the total salicylate concentration. Absorption after rectal administration is slow and unpredictable. Timed-release preparations are therapeutically of limited value because of the prolonged half-life of elimination of salicylate. Contraindications: Acetylsalicylic acid is contraindicated for the following: Absorption of enteric-coated tablets is sometimes incomplete. Active peptic ulcer, febrile/post-febrile illness in children, hemostatic disorders, including anticoagulant and thrombolytic treatment, hypoproteinemia; hypersensitivity; and asthma induced by acetylsalicylic acid or other non-steroidal anti-inflammatory drugs. Caution is indicated in patients with: a history of peptic ulceration or gastro-intestinal hemorrhage, hepatic or renal insufficiency, asthma, children < 2 years, especially in those who are dehydrated Routes of entry: The route of entry is oral. Distribution by route of exposure: Salicylic acid is a weak acid; following oral administration, almost all salicylate is found in the unionized form in the stomach. About 50 - 80% of salicylate in the blood is bound by protein while the rest remain in the active, ionized state; protein binding is concentration-dependent. Saturation of binding sites leads to more free salicylate and increased toxicity. Metabolism: approximately 80% of small doses of salicylic acid is metabolised in the liver. Conjugation with glycine forms salicyluric acid and with glucuronic acid forms salicyl acyl and phenolic glucuronide. These metabolic pathways have only a limited capacity. Small amounts of salicylic acid are also hydroxylated to gentisic acid. With large salicylate doses the kinetics switch from first order to zero order. Elimination by route of exposure: salicylates are excreted mainly by the kidney as salicyluric acid, free salicylic acid, salicylic phenol and acyl glucuronides, and gentisic acid.

International Programme on Chemical Safety; Poisons Information Monograph: Acetylsalicylic Acid (PIM 006) (1991) Available from, as of May 3, 2005: http://www.inchem.org/pages/pims.html

from HSDB

12.1.13 Antidote and Emergency Treatment

The use of paralytic agents and difficulty in achieving the very high minute volumes needed tend to induce respiratory acidosis in the patient. Aspirin (pKa = 3.5) becomes non-ionized at an acidic pH and crosses the blood-brain barrier more readily, increasing its toxic central effects. It is the tissue rather than plasma levels that are dangerous to the patient. Noncardiogenic pulmonary edema interferes with oxygenation of the patient and high concentrations of inspired oxygen may be required.

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 692

from HSDB

Aspirin poisoning leads to inappropriate vasodilation compounded by volume depletion and acidosis, which worsens vasodilation. Aggressive volume repletion with intravenous fluids should be instituted. The aim is to achieve large-volume diuresis to optimize salicylate elimination. If necessary, vasopressors (e.g., norepinephrine, phenylephrine) are added.

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 692

from HSDB

Because of the need for respiratory alkalosis to compensate for the metabolic acidosis of salicylate toxicity, intubation should be avoided unless the patient demonstrates hypoventilation or obtundation. /Salicylate/

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 692

from HSDB

Activated charcoal is used to prevent further absorption of aspirin from the GI tract. This is particularly important when enteric-coated aspirin, which has delayed absorption, has been ingested. Sodium bicarbonate should be administered to maintain the pH between 7.5 and 7.55, and if possible, the pH of the urine greater than 8. Forced alkaline diuresis maximizes salicylate elimination. Hemodialysis may be required if the above measures are inadequate, there is clinical deterioration despite therapy, or if plasma salicylate levels

are greater than 1000 mg/mL. Plasma salicylate, glucose, pH, and potassium should be monitored frequently and therapy modified accordingly. Decreased CNS glucose levels may occur despite normal plasma glucose levels, and supplemental glucose should be given in cases of altered mental status, regardless of the plasma glucose levels.

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 692

from HSDB

Emergency and supportive measures: Maintain an open airway and assist ventilation if necessary. Warning: Ensure adequate ventilation to prevent respiratory acidosis and do not allow controlled mechanical ventilation to interfere with the patient's need for compensatory efforts to maintain the serum pH. Administer supplemental oxygen. Obtain serial arterial blood gases and chest x-rays to observe for pulmonary edema (more common with chronic or severe intoxication). Treat coma, seizures, pulmonary edema, and hyperthermia if they occur. Treat metbolic acidosis with intravenous sodium bicarbonate. Do not allow the serum pH to fall below 7.4. Replace fluid and electrolyte deficits caused by vomiting and hyperventilation with intravenous crystalloid solutions. Be cautious with fluid therapy, because excessive fluid administration may contribute to pulmonary edema. Administer supplemental glucose, and treat hypoglycemia if it occurs. Monitor asymptomatic patients for a minimum of 6 hours (longer if an enteric-coated preparation or a massive overdose has been ingested and there is suspicion of a tablet bezoar). Admit symptomatic patients to an intensive care unit. /Salicylates/

Olson, K.R. (Ed.); Poisoning & Drug Overdose. 5th ed. Lange Medical Books/McGraw-Hill. New York, N.Y. 2007., p. 334

from HSDB

There is no specific antidote for salicylate intoxication. Sodium bicarbonate is given frequently both to prevent acidemia and to promote salicylate elimination by the kidneys. /Salicylates/

Olson, K.R. (Ed.); Poisoning & Drug Overdose. 5th ed. Lange Medical Books/McGraw-Hill. New York, N.Y. 2007., p. 334

from HSDB

Urinary alkalinization is effective in enhancing urinary excretion of salicylates, although it is often difficult to achieve in dehydrated or critically ill patients. The goal is to maintain urine pH > or = 7.5. Add ... sodium bicarbonate to ... dextrose in quarter normal saline and infuse intravenously ... . Fluid and bicarbonate administration is potentially dangerous in patients at high risk for pulmonary edema (eg, chronic intoxication). Unless renal failure is present, also add potassium ... to each liter of intravenous fluids (potassium depletion inhibits alkalinization). Alkalemia is not a contraindication to bicarbonate therapy in light of the fact that patients often have a significant base deficit in spite of the elevated serum pH. /Salicylates/

Olson, K.R. (Ed.); Poisoning & Drug Overdose. 5th ed. Lange Medical Books/McGraw-Hill. New York, N.Y. 2007., p. 335

from HSDB

Decontamination is not necessary for patients with chronic intoxication. Administer activated charcoal orally if conditions are appropriate. Gastric lavage is not necessary after small to moderate ingestions if activated charcoal can be given promptly. Note: With large ingestions of salicylate (eg, 30-60 g), very large doses of activated charcoal are theoretically necessary to absorb all the salicylate. In such cases, the charcoal can be given in several .. doses at 3- to 5- hour intervals. Whole-bowel irrigation is recommended to help move the pills and charcoal through the intestinal tract. /Salicylates/

Olson, K.R. (Ed.); Poisoning & Drug Overdose. 5th ed. Lange Medical Books/McGraw-Hill. New York, N.Y. 2007., p. 334

from HSDB

Repeat-dose activated charcoal therapy effectively reduces the serum salicylate half-life, but it is not as rapidly effective as dialysis, and frequent stooling may contribute to dehydration and electrolyte disturbances. /Salicylates/

Olson, K.R. (Ed.); Poisoning & Drug Overdose. 5th ed. Lange Medical Books/McGraw-Hill. New York, N.Y. 2007., p. 335

from HSDB

Hemoperfusion is also very effective but does not correct acid-base or fluid disturbances. /Salicylates/

Olson, K.R. (Ed.); Poisoning & Drug Overdose. 5th ed. Lange Medical Books/McGraw-Hill. New York, N.Y. 2007., p. 335

from HSDB

Hemodialysis is very effective in rapidly removing salicylate and correcting acid-base and fluid abnormalities. Indications for urgent hemodialysis are as follows: Patients with acute ingestions and serum levels higher than 1000 mg/L (100 mg/dL) with severe acidosis and other manifestations of intoxication. Patients with chronic intoxication with serum levels higher than 600 mg/L (60 mg/dL) accompanied by acidosis, confusion, or lethargy, especially if the patients is elderly or debilitated. Any patient with severe manifestations of intoxication. /Salicylates/

Olson, K.R. (Ed.); Poisoning & Drug Overdose. 5th ed. Lange Medical Books/McGraw-Hill. New York, N.Y. 2007., p. 335

from HSDB

12.1.14 Human Toxicity Excerpts

/HUMAN EXPOSURE STUDIES/ ... The objectives in this study were 1) to compare the pharmacokinetic and pharmacodynamic effects of cutaneous and oral aspirin in healthy volunteers and 2) to compare the effects of cutaneous aspirin on gastroduodenal mucosal prostaglandin E2 and F2 alpha content and on mucosal damage, using endoscopy. The bioavailability of cutaneous aspirin was 4%-8% that of oral aspirin. Cutaneous aspirin (750 mg/day for 10 days) significantly lowered serum thromboxane (by 85%) and gastric and duodenal prostaglandins (by 49%-71%); placebo had no effect. Moreover, cutaneous aspirin, but not placebo, resulted in significant gastric mucosal injury. These findings demonstrate that even tiny amounts of aspirin in the blood (2 uM) have inhibitory effects on prostaglandin production in the human stomach and duodenum that result in gastric mucosal damage, even without direct exposure of the stomach to aspirin.
Abstract: PubMed

Cryer B et al; Proc Assoc Am Physicians 111 (5): 448-56 (1999)

from HSDB

/SIGNS AND SYMPTOMS/ Acetylic acid, such as aspirin, is one of the most commonly used medication in Western societies. Aspirin overdosage causes ototoxic side effects in some patients, such as bilateral mild to moderate sensorineural hearing loss and tinnitus.
Abstract: PubMed

Wecker H, Laubert A; HNO 52 (4): 347-5 (2004)

from HSDB

/SIGNS AND SYMPTOMS/ Mild chronic salicylate intoxication is called salicylism. When fully developed, the syndrome includes headache, dizziness, tinnitus, difficulty hearing, dimness of vision, mental confusion, lassitude, drowsiness, sweating, thirst, hyperventilation, nausea, vomiting, and occasionally diarrhea. /Salicylates/

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 691

from HSDB

/SIGNS AND SYMPTOMS/ Acute ingestion. Vomiting occurs shortly after ingestion, followed by hyperpnea, tinnitus, and lethargy. Mixed respiratory alkalemia and metabolic acidosis are apparent when arterial blood gases are determined. With severe intoxication, coma, seizures, hypoglycemia, hyperthermia, and pulmonary edema may occur. Death is caused by CNS failure and cardiovascular collapse. /Salicylates/

Olson, K.R. (Ed.); Poisoning & Drug Overdose. 5th ed. Lange Medical Books/McGraw-Hill. New York, N.Y. 2007., p. 333

from HSDB

/SIGNS AND SYMPTOMS/ Chronic intoxication: Victims are usually confused elderly persons who are taking salicylates therapeutically. The diagnosis is often overlooked because the presentation is nonspecific; confusion, dehydration, and metabolic acidosis are often attributed to sepsis, pneumonia, or gastroenteritis. However, morbidity and mortality rates are much higher than they are after acute overdose. Cerebral and pulmonary edema is more common than with acute intoxication, and severe poisoning occurs at lower salicylate levels. /Salicylates/

Olson, K.R. (Ed.); Poisoning & Drug Overdose. 5th ed. Lange Medical Books/McGraw-Hill. New York, N.Y. 2007., p. 333

from HSDB

/SIGNS AND SYMPTOMS/ Certain individuals display hypersensitivity to aspirin and NSAIDs, as manifested by symptoms that range from vasomotor rhinitis with profuse watery secretions, angioedema, generalized urticaria, and bronchial asthma to laryngeal edema, bronchoconstriction, flushing, hypotension, and shock. Aspirin intolerance is a contraindication to therapy with any other NSAID because cross-sensitivity can provoke a life-threatening reaction reminiscent of anaphylactic shock. Despite the resemblance to anaphylaxis, this reaction does not appear to be immunological in nature. Although less common in children, this syndrome may occur in 10% to 25% of patients with asthma, nasal polyps, or chronic urticaria, and in 1% of apparently healthy individuals. It is provoked by even low doses (<80 mg) of aspirin and apparently involves COX inhibition.

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 685

from HSDB

/CASE REPORTS/ Asthma, aspirin intolerance and nasal polyps form a triad of aspirin-induced asthma (AIA). Eighteen cases, 6 males and 12 females, who complained of asthma attacks and/or rhinorrhea after ingestion of non-steroidal anti-inflammatory drugs were encountered over several years. The mean age of onset was 32.1 for asthma and 25.4 for rhinitis. Asthma was found in all of the 18 cases and nasal polyps in 13 cases (72.2%). The polyps were recurrent and 7 patients had undergone polypectomies. Urticaria was seen in 44.4% and sinusitis diagnosed by X-ray in 81.8%. Sensitivity to at least one allergen was found in 7 out of 9 cases (77.8%) and 6 out of 11 cases (54.5%) gave results positive for RAST. Eosinophilia was seen in 14 out of 16 patients (87.5%). The pathogenesis of AIA is obscure but is probably related to inhibition of prostaglandin biosynthesis.
Abstract: PubMed

Ogino S et al; Acta Otolaryngol Suppl 430: 21-7 (1986)

from HSDB

/CASE REPORTS/ A newborn infant had metabolic acidosis, tachypnea, and hypoglycemia. After the initial diagnosis of neonatal sepsis, she was given antibiotics but failed to respond. Further investigation revealed that her mother had taken aspirin throughout pregnancy. This case illustrates the similarities between symptoms of neonatal sepsis and those of a toxic reaction to salicylate.
Abstract: PubMed

Buck ML et al; J Pediatr 122 (6): 955-8 (1993)

from HSDB

/CASE REPORTS/ ... The case of a man who took two overdoses of aspirin, on each occasion suffering a grand mal fit with blood levels of salicylate of over 5 mmol/L /is described/. The first event was treated with hemodialysis but without effective alkalinization, and the second with alkalinization but without hemodialysis. The rate of decline in salicylate concentration was faster with alkalinization in the first 4 hours. Similar salicylate levels were achieved with both techniques by 24 hours post-overdose. If a case of salicylate poisoning is to be treated with hemodialysis, treatment with alkalinization should still be given without delay, in order to prevent acidemia and to promote elimination of as much salicylate as possible via the kidneys.
Abstract: PubMed

Higgins RM et al; Clin Nephrol 50 (3): 178-83 (1998)

from HSDB

/CASE REPORTS/ A 64-year-old woman, who was taking long-term enteric-coated aspirin therapy for rheumatoid arthritis, was prescribed approximately twice her normal dosage (7.1 g daily) during a ten-day convalescence following surgery. Although she presented with features mimicking sepsis, biochemical analysis, ie, a spuriously high carbon dioxide content, suggested salicylate intoxication (admission salicylate concentration, 5.13 mmol/L). She died on the third day after admission. Autopsy showed no major source of infection except for bronchopneumonia. Long-term users of a high-dose aspirin are at risk for potential salicylate intoxication. The metabolism of salicylate, particularly its excretion kinetics, can make small upward dosage adjustments hazardous. Salicylate has widespread metabolic effects that can mimic other medical conditions, leading to delayed diagnosis of salicylate intoxication. Increased mortality and morbidity may result.
Abstract: PubMed

Shkrum MJ et al; Arch Pathol Lab Med 113 (1): 89-90 (1989)

from HSDB

/CASE REPORTS/ Aspirin overdosage causes ototoxic side effects in some patients, such as bilateral mild to moderate sensorineural hearing loss and tinnitus. .... A young man with an acute moderate aspirin intoxication resulting in asymmetric hearing loss of 50 dB HL and tinnitus for five days /is described/. Otoacoustic emissions were absent on the first day of intoxication but could be measured again on the fifth day after the intoxication. As the ototoxic side effects resolve with in two or three days, no specific treatment is necessary for ototoxicity. ...
Abstract: PubMed

Wecker H, Laubert A; HNO 52 (4): 347-51(2004)

from HSDB

/CASE REPORTS/ Aspirin overdose may result in acid-base disturbances, electrolyte abnormalities, pulmonary edema, chemical hepatitis, seizures, and mental status alteration, but myocardial depression has not been reported following aspirin overdose in children. In addition to these more typical features, the 13-month-old boy reported here developed clinical, radiographic, and echocardiographic evidence of myocardial impairment with pulmonary edema and moderately severe global left ventricular dysfunction (estimated shortening fraction of 23%). Complete resolution of the myocardial dysfunction was demonstrated on follow-up echocardiography as the child recovered from the aspirin intoxication. This case suggests that myocardial dysfunction can occur as a result of toxic aspirin ingestion, and that it may contribute to salicylate-induced pulmonary edema.
Abstract: PubMed

Ralston ME et al; J Emerg Med 13 (5): 657-9 (1995)

from HSDB

/CASE REPORTS/ A 14-year-old white female was evaluated after a single ingestion of 120 tablets of aspirin 81 mg/tablet hours before arrival to the emergency department. She denied nausea, abdominal pain, tinnitus, or shortness of breath. She received one dose of activated charcoal. The first salicylate concentration (4 hr after ingestion) was 1 mg/dL. At 35 hours, the patient became symptomatic (dizziness, tinnitus, epigastric discomfort). Her salicylate concentration at that time was 46 mg/dL. A second dose of activated

charcoal was administered, and intravenous bicarbonate with potassium was started as a continuous infusion for 30 hours. ... In cases with known salicylate ingestion, it is important to follow salicylate concentrations every 4 hours until they are steadily decreasing according to a 4-hour half-life and the patient shows no symptoms of salicylate intoxication.
Abstract: PubMed

Rivera W et al; Ann Pharmacother 38 (7-8): 1186-8 (2004)

from HSDB

/CASE REPORTS/ A 17-year-old, 37-week pregnant woman presented to the hospital stating that she had ingested 50 aspirin tablets per day for 1 month in an attempt to harm her baby and herself. Ultrasound showed fetal demise. Serum salicylate was 620 mg/L with an anion gap of 22.6 and the following blood gases: pO2 108 mm Hg, pCO2 15mm Hg, pH 7.34, and HCO3 8.8 mmol/L. She was successfully treated with alkaline diuresis followed by hemodialysis. She spontaneously delivered a macerated stillborn 2380-g fetus. Autopsy revealed diffuse petechiae in the lungs, heart, thymus, and kidneys. Salicylic acid was found in the cord blood, but quantification was not possible due to the small volume of the blood sample. Our patient supports the hypothesis that the fetus is at greater risk than the mother in salicylate poisoning during pregnancy. Consideration should be given to emergent delivery of term or near-term, aspirin-poisoned fetuses.
Abstract: PubMed

Palatnick W et al; Am J Perinatol 15 (1): 39-41 (1998)

from HSDB

/CASE REPORTS/ ... A 19-year-old woman in her 38th week of pregnancy ingested a total of 16.25 g of aspirin in a suicide attempt. On arrival to the emergency department, the maternal salicylate level was 31.7 mg/dL. Physical examination revealed stable vital signs with mild tachypnea. The patient, however, denied tinnitus, gastrointestinal, or neurological symptoms. Fetal monitoring applied in labor and delivery revealed fetal distress with bradycardia (HR-60) and late decelerations. For this reason an emergency Cesarean section was performed and APGAR scores were noted to be 5 and 7 at 1 and 5 minutes respectively. Bag-valve-mask ventilations were required for a brief period after delivery. Maternal salicylate level drawn just prior to Cesarean section was 14 mg/dL. The baby's salicylate level, drawn immediately after delivery, was 35.2 mg/dL. Newborn vital signs soon after delivery were BP 65/46, HR 142, RR 58, pulse oximetry 100% on room air. Laboratories showed a pH of 7.49 with a pCO2 of 27 mmHg; electrolytes were normal except for a bicarbonate of 18 mEq/L. Serial aspirin levels showed a value of 26.4 mg/dL at 28 hours, and 8.1 mg/dL at 101 hours post delivery. The baby was discharged without any obvious problems. /This is/ a case of perinatal aspirin poisoning, where the baby had higher levels than those of the mother. The baby had significant fetal distress with only minor symptoms reported in the mother. ...

Velez LI et al; J Toxicol Clin Toxicol 39 (5): 483-4 (2001)

from HSDB

/CASE REPORTS/ A patient presented to the emergency room after self-administering, in enema form, approximately 700 aspirin tablets dissolved in water. Over the next 12 hours the patient became progressively acidemic with eventual cardiac arrest and subsequent chronic hypoxic encephalopathy. ... This patient's poor outcome was the result of retained aspirin products in the rectal vault combined with the failure to recognize the delayed absorption properties of rectally administered aspirin. ... In rectal aspirin overdoses, aspirin absorption from the rectum may occur over a long period of time. It is important to remove as much aspirin from the rectum as possible and to closely monitor these patients so that appropriate therapy may be started quickly. Activated charcoal given both in enema and oral form may help decrease aspirin absorption. Hemodialysis should be available and performed without delay should the patient require it.
Abstract: PubMed

Watson JE, Tagupa ET; Ann Pharmacother 28 (4): 467-9 (1994)

from HSDB

/CASE REPORTS/ ... Here an uncommon case of fluid retention simulating acute congestive heart failure, secondary to aspirin consumption, promptly reversible after discontinuation of therapy, and triggered again by pharmacological challenge test.
Abstract: PubMed

Manfredini R et al; Am J Med Sci 320 (1): 72-4 (2000)

from HSDB

/CASE REPORTS/ Cardiac side effects from aspirin are uncommon; however, severe acid-base imbalance, pulmonary edema, ventricular ectopic activity and cardiopulmonary arrest have been reported in patients with toxic serum salicylate concentrations. We saw a patient with salicylate toxicity who developed a variety of sinus and atrioventricular nodal conduction disturbances and atrial arrhythmias with a relatively low toxic serum salicylate concentration. The cardiac rhythm returned to normal as the serum salicylate concentration decreased, and results of subsequent electrophysiologic testing and Holter monitoring were normal. A low serum albumin level may have resulted in altered salicylate binding in this patient, thereby increasing the availability of unbound (active) drug for toxic effects.
Abstract: PubMed

Mukerji V et al; Pharmacotherapy 6 (1): 41-3 (1986)

from HSDB

/EPIDEMIOLOGY STUDIES/ ... This population based case control study, conducted in a California county, investigated potential relations between a variety of maternal exposures during pregnancy and congenital cardiac anomalies in offspring during 1981-83. Data were obtained from telephone interviews with 141 mothers of children with a severe cardiac anomaly and 176 mothers of children born without such an anomaly. As in previous investigations, this study found a positive relation between cardiac anomalies and maternal: epilepsy (odds ratio= 9.0), diabetes (odds ratio= 2.1), insulin use (odds ratio= 11.6), and anticonvulsant use (odds ratio= 6.3) Elevated risks were also found for maternal use of: sulfa drugs (odds ratio= 1.8), larger amounts of alcohol (odds ratio=1.7), cocaine (odds ratio= 2.8), oral contraceptives (odds ratio=2.0), and intrauterine devices (odds ratio= 3.0). No association was observed for maternal use of Bendectin, codeine, aspirin, Tylenol, and cigarettes of 0.5 to 1 pack/day. These results remained when analyses were limited to the 45 cases with lesions of the conotruncus.

Shaw GM et al; Teratology 41 (5): 590 (1990)

from HSDB

/GENOTOXICITY/ CHROMOSOMAL CHANGES WERE INDUCED IN FIBROBLAST CULTURES BY ASPIRIN (104-250 MG/ML). @ CELLULAR LEVEL ASPIRIN APPEARS TO HAVE MUTAGENIC EFFECT WHICH WOULD NOT BE EVIDENT IN 3 DAY LEUKOCYTE CULTURES.
Abstract: PubMed

MEISNER LF, INLORN SL; ACTA CYTOL 16 (1): 41-7 (1972)

from HSDB

/OTHER TOXICITY INFORMATION/ Aspirin use may be associated with the development of Reye's syndrome in children and teenagers with acute febrile illnesses, especially influenza and varicella. It is recommended that salicylate therapy not be initiated in febrile pediatric or adolescent patients until after the presence of such an illness has been ruled out. Also, it is recommended that chronic salicylate therapy in these patients be discontinued if a fever occurs, and not resumed until it has been determined that an illness that may predispose to Reye's syndrome is not present or has run its course. Other forms of salicylate toxicity may also be more prevalent in pediatric patients, especially children who have a fever or are dehydrated.

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2577

▸ *from HSDB*

/OTHER TOXICITY INFORMATION/ Human and animal studies have shown that salicylates alone do not generate a permanent threshold shift of auditory sensitivity. Salicylate inducing hearing loss and deterioration of psychoacoustic abilities are temporary. However, temporary aspirin induced auditory effects should not be ignored. Indeed, these effects might induce accidents, especially in industrial areas where communication in noise and good auditory sensitivity are required. Furthermore, a high serum salicylate level (> 40 mg/100 ml) may exacerbate the effects of noise and increase temporary hearing loss. However, there is no experimental proof that salicylates facilitate the permanent threshold shift of auditory sensitivity caused by noise.

Campo P, Boettcher FA; Cahiers de notes documentaires - S'ecurite' et hygiene du travail 1st Quarter 1991, No 142, Note No. 1871-142-91, p 79-86

▸ *from HSDB*

/OTHER TOXICITY INFORMATION/ ... Human bronchial epithelial cells (HBEC) /were isolated/ from bronchial brushing preparations taken during bronchoscopy of 10 non-asthmatics (NA), 8 aspirin-tolerant asthmatics (ATA) and 9 aspirin-intolerant asthmatics (AIA). HBEC were cultured in serum free medium until 80% confluent. Total cellular RNA was isolated and reversed transcribed using oligo(dT)(15) primers. Real time PCR was performed with primers to COX-1, COX-2, GAPDH and beta-actin in the presence of SYBR green dye. The cycle threshold (C(T)) for COX-1 or COX-2 was normalized using beta-actin and GAPDH as the internal standards.Not only COX-1 but also COX-2 mRNA were expressed by HBEC without any proinflammatory stimulation. ... The smallest amount of COX-1 mRNA in the AIA group /was detected/. The same trend was observed for COX-2 mRNA, though it didn't reach the statistical significance. ... The relationship between DeltaC(TCOX-1) to DeltaC(TCOX-2) /was analyzed/ by calculating the difference DeltaDeltaC(TCOX-1-COX-2). This analysis revealed that AIA group can be characterized by relatively smallest COX-1 mRNA expression in comparison to COX-2. There is a strong positive correlation between C(TCOX1) and C(TCOX2) in NA group (r=0.85; p< 0.001). In both groups of asthmatics this correlation is absent (ATA - r=0.5, p>0.1; AIA - r=0.43, p>0.1). Cyclooxygeneases transcripts expression is altered in HBEC derived from the asthmatic patients, and this phenomenon is pronounced in case of aspirin hypersensitivity.
Abstract: PubMed

Pierzchalska M et al; J Physiol Pharmacol 58 (2): 207-18 (2007)

▸ *from HSDB*

12.1.15 Non-Human Toxicity Excerpts

/LABORATORY ANIMALS: Acute Exposure/ HOMOZYGOUS GUNN RATS, MUTANT WISTARS GENETICALLY LACKING GLUCURONYL TRANSFERASE, DEVELOPED RENAL PAPILLARY NECROSIS AFTER 1 ORAL DOSE OF ASPIRIN & PHENACETIN. LESION APPEARED MORE FREQUENTLY WITH ASPIRIN THAN PHENACETIN, AND AT LOWER DOSES.
Abstract: PubMed

AXELSEN RA; J PATHOL 120 (3): 145-50 (1976)

▸ *from HSDB*

/LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity/ TWENTY-NINE 5-WK-OLD MALE F344/CRL RATS WERE FED A DIET CONTAINING 0 OR 0.5% ASPIRIN CONTINUOUSLY FOR 68 WEEKS. ALL SURVIVING RATS AT WEEK 68 WERE NECROPSIED; ORGANS IN THE THORACIC AND ABDOMINAL CAVITIES, AS WELL AS THE SKIN AND SUBCUTIS, WERE EXAMINED MACROSCOPICALLY. THE BLADDER, STOMACH, AND LIVER WERE PREPARED FOR HISTOPATHOLOGY. BODY WEIGHT GAIN WAS LOWER IN THE ASPIRIN-TREATED GROUP THAN IN THE CONTROL GROUP. NO BLADDER TUMORS WERE REPORTED IN EITHER GROUP, EVEN THOUGH RENAL PAPILLARY NECROSES OF INTERSTITIAL TISSUE, CAPILLARIES, AND LOOPS OF HENLE WERE FOUND IN 13 OF 15 LESIONS EXAMINED IN THE TREATED GROUP. THERE WAS A SIGNIFICANT DECREASE IN THE SEVERITY OF NEPHROPATHY IN AGED RATS IN THE ASPIRIN-TREATED GROUP COMPARED WITH CONTROLS.
Abstract: PubMed

JOHANSSON SL ET AL; TOXICOL APPL PHARMACOL 86: 80-92 (1986)

▸ *from HSDB*

/LABORATORY ANIMALS: Developmental or Reproductive Toxicity/ ... Female /wistar rats/ were confirmed to have mated by observations of sperm in a vaginal smear. The day on which spermatozoa were found in the vaginal smear was considered as day 1 of gestation (GD1). After randomization, mated females were assigned to experimental groups and individually caged, were given 50 mg/kg/day of acetylsalicylic acid (ASA), by needle gavage once daily, during two different periods of pregnancy. One group of dams (n=11) received aspirin from day 1 to 4 of pregnancy (before embryonic implantation) for evaluation of the blastocysts, and another group received aspirin from day 6 to 15 of pregnancy (organogenic period) for fetal evaluation. Control groups (n=12) received distilled water in same volume and during same periods as their respective experimental groups. The treatment of the dams with ASA, according to minimal therapeutic dose used for humans, did not cause embryotoxic or major malformations on experimental animal but was responsible for rate increased of fetuses presenting ureteric dilatation. After analysis of the data, it appears that, although direct conclusive evidence of adverse effects in humans is lacking, a potential hazard dose exists and thus the indiscriminate use of acetylsalicylic acid (aspirin) is contraindicated.
Abstract: PubMed

Damasceno DC et al; Rev Assoc Med Bras 48 (4): 312-6 (2002)

▸ *from HSDB*

/LABORATORY ANIMALS: Developmental or Reproductive Toxicity/ Rat embryos were exposed to aspirin or its metabolite, salicylic acid in culture. In these embryos acute reduction of heart beat was observed during 4 hours of administration compared to that in non-treated one. Protein contents and crown-rump length of cultured embryos were significantly decreased in aspirin-treated group, but were not so decreased in salicylic acid-treated one. The predominant defects of the embryos exposed to aspirin were edematous facial malformations and abnormality of tail. On the other hand, facial anomalies such as cleft lip and curly tail were observed in the embryos cultured with salicylic acid. Anomalies induced by aspirin were systemic, while salicylic acid induced localized malformations. These results might be due to the differences between aspirin and its metabolite, salicylic acid in their teratogenicity.
Abstract: PubMed

Yokoyama A, et al; Res Commun Chem Pathol Pharmacol 46 (1): 77-91(1984)

▸ *from HSDB*

/LABORATORY ANIMALS: Developmental or Reproductive Toxicity/ Three growth models were used to examine the effects of prenatal exposure to aspirin on the postnatal development of brain parts. A total of 60 pregnant rats which were divided into three experimental groups and a control group were exposed to aspirin doses of 12.5, 25, 37.5 mg/kg, and distilled water, respectively. The brain parts of 200 rat pups starting from the first week after birth until the fifth week were weighted and the length and width of the cerebrum and cerebellum were measured to determine the parameters of the growth models. The results indicated that the three models successfully predicted the growth of the different brain parts and that aspirin decreased the total brain weight, cerebrum length and width, and decreased the cerebellum length and width at aspirin dose of 37.5 mg/kg. Further analysis is needed to investigate if aspirin effects were carried out through its role in inhibiting prostaglandin production and consequently affecting the activity of the hypothalamus-pituitary axis.
Abstract: PubMed

Elkarmi A et al; Growth Dev Aging 70 (1): 13-24 (2007)

▸ *from HSDB*

/LABORATORY ANIMALS: Developmental or Reproductive Toxicity/ Pregnant rats were treated orally with aspirin 0.5 or 1.0 g.kg-1 on d 3 (positive vaginal smear was considered as d 0) and were sacrificed on d 4. Some blastocysts collected on d 4 were evaluated for gross morphology and cell number, and the remainings were transfered into

pseudopregnant rats. Results showed that the rate of blastocysts with abnormal morphology were 23.8% and 40.8%, respectively, in 0.5 and 1.0 g.kg-1 of aspirin. These were significantly higher than 6.8% of the control group. The cell number of blastocysts also decreased in the aspirin groups. The rate of implantation and live fetuses in the case of blastocysts with normal morphology were related negatively with the aspirin doses, espesially in the group of 1.0 g.kg-1 of aspirin, the implantation rate was significantly lower (38.6%). However, the implantation rate of blastocysts with abnormal morphology in both groups of aspirin were much less than that of the control group, and all embryos after implantation were resorbed. No significant malformations were observed in the live fetuses. These results suggests that the effects of blastocyst deficiencies induced by aspirin on development of embryos transfered into pseudopregnant rats mainly caused death of embryos, but not malformation of fetuses.
Abstract: PubMed

Ying Y et al; Yao Xue Xue Bao 31 (6): 416-9 (1996)

from HSDB

/LABORATORY ANIMALS: Developmental or Reproductive Toxicity/ ... The effects of bacterial endotoxin (LPS) on the toxicities of salicylic acid, the main metabolite of aspirin, were investigated in rats. The following results were obtained: 1) The acute toxicity of salicylic acid was significantly potentiated by LPS in male rats. The LD50 of salicylic acid with LPS was about one third of that of salicylic acid alone. 2) The increase of maternal body weight was inhibited significantly after administration of salicylic acid (383 mg/kg, p.o.) with LPS (20 ug/kg, i.v.), but not after administration of salicylic acid alone. 3) The fetal toxicity of salicylic acid including fetal death, resorption, growth retardation and skeletal variations was slightly observed in the dam receiving a single dose of salicylic acid on the 15th day of pregnancy, but it was markedly increased by LPS (20 ug/kg, i.v.). 4) The half-life period of salicylic acid in plasma was increased significantly by the co-administration of LPS in male rats after administration of aspirin or salicylic acid. All of these phenomena in the rats given salicylic acid closely resembled the phenomena previously reported in the rats given aspirin. These results suggest that salicylic acid might play a main role in the acute and fetal toxicities of aspirin, and one of the mechanism of the enhancement effect by LPS on aspirin-induced fetal toxicity might be related to the increase of salicylic acid concentration in the fetus. /Salicylic acid/
Abstract: PubMed

Itami T, Kanoh S; Nippon Yakurigaku Zasshi 84 (5): 411-6 (1984)

from HSDB

/LABORATORY ANIMALS: Developmental or Reproductive Toxicity/ Salicylates are among the oldest and most widely used drugs and are known to lead to fetal death, growth retardation and congenital abnormalities in experimental animals. In this study, the effects of acetyl salicylic acid (ASA), salicylic acid (SAL) and sodium salicylate (NaSAL) on early organogenesis and the interaction of these molecules with free radicals has been investigated. Postimplantation rat embryos were cultured in vitro from day 9.5 of gestation for 48 hr. ASA, SAL and NaSAL were added to whole rat serum at concentrations between 0.1 and 0.6 mg/mL. Also, the lowest effective concentration of ASA for all parameters (0.3 mg/mL) and the same concentration of NaSAL and SAL was added to the culture media in the presence of superoxide dismutase (SOD) (30 U/mL) or glutathione (0.5 micromol/mL). The growth and development of embryos was compared and each embryo was evaluated for the presence of any malformations. When compared to growth of control embryos, the salicylates decreased all growth and developmental parameters in a concentration-responsive manner. There was also a concentration-related increase in overall dysmorphology, including the incidence of haematoma in the yolk sac and neural system, open neural tube, abnormal tail torsion and the absence of fore limb bud. When SOD was added in the presence of ASA, growth and developmental parameters were improved and there was a significant decrease in the incidence of malformations. Addition of SOD also decreased the incidence of malformations in the presence of SAL, but did not effect the growth and developmental parameters of SAL and NaSAL. There was no significant difference between the embryos grown in the presence of these three molecules on the addition of glutathione. The effects of salicylates might involve free oxygen radicals by the non-enzymatic production of the highly teratogenic metabolites 2,3- and 2,5-dihydroxybenzoic acid. An enhanced production of these metabolites in embryonic tissues may be directly related to the increased risk of congenital malformations.
Abstract: PubMed

Karabulut AK et al; Toxicol In Vitro 14 (4): 297-307 (2000)

from HSDB

/LABORATORY ANIMALS: Developmental or Reproductive Toxicity/ OFFSPRING OF FOOD-RESTRICTED RATS RECEIVING 250 MG/KG/DAY OF ASPIRIN FROM DAYS 7-10 AFTER MATING HAD TWICE INCIDENCE OF MALFORMATIONS AS THOSE FROM RATS ON UNRESTRICTED DIET PLUS ASPIRIN.
Abstract: PubMed

BEALL JA, KLEIN MF; TOXICOL APPL PHARMACOL 39 (3): 489-95 (1977)

from HSDB

/LABORATORY ANIMALS: Developmental or Reproductive Toxicity/ DAILY ORAL ADMIN OF 600 MG/KG OF ASPIRIN TO PREGNANT RATS CAUSED DEVELOPMENT OF HYDROPS IN FETUSES. 14TH-17TH DAY OF GESTATION MOST SUSCEPTIBLE PERIOD.

MIYAMOTO T; OSAKA SHIRITSU DAIGAKU IGAKU ZASSHI 21 (4-6): 123-39 (1972)

from HSDB

/LABORATORY ANIMALS: Developmental or Reproductive Toxicity/ ORAL ADMIN OF 10 MG/KG/DAY FOR 2-3 DAYS TO RATS ON DAYS 19TH-21ST OF GESTATION CAUSED DELAY IN ONSET OF PARTURITION & SLIGHT INCR IN BLEEDING. FETAL DEATHS INCR.
Abstract: PubMed

WALTMAN ET AL; PROSTAGLANDINS 4 (1): 93-106 (1973)

from HSDB

/LABORATORY ANIMALS: Developmental or Reproductive Toxicity/ MAZE PERFORMANCE OF /RAT/ OFFSPRINGS OF DAMS WHICH HAD RECEIVED 250 MG/KG ON DAYS 8, 9 & 10 OF GESTATION POORER THAN CONTROLS. FETUSES SHOWED WT DECR, MINOR MALFORMATIONS SKELETON & VISCERA.

BUTCHER ET AL; NATURE (LONDON), NEW BIOL; 236 (68): 11-12 (1972)

from HSDB

/GENOTOXICITY/ ASPIRIN WAS TESTED FOR MUTAGENICITY IN THE SALMONELLA/MAMMALIAN MICROSOME ASSAY. SIX TESTOR STRAINS WERE USED (TA1535, TA1537, TA1538, TA100, TA97, AND TA98) AND EXPERIMENTS WERE CONDUCTED IN THE PRESENCE AND ABSENCE OF A RAT-LIVER MICROSOME ACTIVATION SYSTEM. ASPIRIN DID NOT SHOW ANY EVIDENCE OF MUTAGENIC ACTIVITY AT CONCENTRATIONS RANGING FROM 0.01 TO 50 MG PER PLATE.
Abstract: PubMed

JASIEWICZ ML ET AL; MUTAT RES 190: 95-100 (1987)

from HSDB

/OTHER TOXICITY INFORMATION/ VET: Aspirin overdose in any species can result in salicylate poisoning, characterized by severe acid-base abnormalities, hemorrhage, seizures, coma, and death.

Kahn, C.M. (Ed.); The Merck Veterinary Manual 9th ed. Merck & Co. Whitehouse Station, NJ. 2005, p. 2134

from HSDB

/OTHER TOXICITY INFORMATION/ ... /Investigators/ hypothesized that infinitesimal concentrations of aspirin could persist in plasma after its discontinuation, thereby inducing a prothrombotic effect that could be due to a modification in the mechanism of action of aspirin via the cyclooxygenase 1 (COX-1) and COX-2 pathways. ... The effects of ultra-low-dose aspirin (ULDA) as well as those of sc-560 and ns-398, specific COX-1 and COX-2 inhibitors, on induced hemorrhagic time and in a model of laser-induced thrombosis in rats

/were studied/. In the laser-induced thrombosis model, ULDA treatment increased the number of emboli and the duration of embolization, thereby confirming its prothrombotic effect described in previous publications. This effect was also observed in rats pretreated with sc-560 but not in those pretreated with ns-398. ... ULDA induced a prothrombotic effect in the rats studied.
Abstract: PubMed

Doutremepuich C et al; Pathophysiol Haemost Thromb 36 (1): 40-4 (2007)

▸ *from HSDB*

12.1.16 Human Toxicity Values

Potentially lethal intoxication develops /in children/ after ingestions of 6 tablets/kg (480 mg/kg).

Ellenhorn, M.J. and D.G. Barceloux. Medical Toxicology - Diagnosis and Treatment of Human Poisoning. New York, NY: Elsevier Science Publishing Co., Inc. 1988., p. 562

▸ *from HSDB*

12.1.17 Non-Human Toxicity Values

LD50 RABBIT ORAL 1800 MG/KG

Sunshine, I. (ed.). CRC Handbook of Analytical Toxicology. Cleveland: The Chemical Rubber Co., 1969., p. 3

▸ *from HSDB*

LD50 RABBIT INTRAPERITONEAL 500 MG/KG

Sunshine, I. (ed.). CRC Handbook of Analytical Toxicology. Cleveland: The Chemical Rubber Co., 1969., p. 3

▸ *from HSDB*

LD50 Rat oral 1500 mg/kg

American Conference of Governmental Industrial Hygienists. Documentation of the TLV's and BEI's with Other World Wide Occupational Exposure Values. CD-ROM Cincinnati, OH 45240-1634 2007.

▸ *from HSDB*

LD50 Rat oral 200 mg/kg

Lewis, R.J. Sr. (ed) Sax's Dangerous Properties of Industrial Materials. 11th Edition. Wiley-Interscience, Wiley & Sons, Inc. Hoboken, NJ. 2004., p. 57

▸ *from HSDB*

LD50 Rat ip 340 mg/kg

Lewis, R.J. Sr. (ed) Sax's Dangerous Properties of Industrial Materials. 11th Edition. Wiley-Interscience, Wiley & Sons, Inc. Hoboken, NJ. 2004., p. 57

▸ *from HSDB*

LD50 Mouse oral 1100 mg/kg

American Conference of Governmental Industrial Hygienists. Documentation of the TLV's and BEI's with Other World Wide Occupational Exposure Values. CD-ROM Cincinnati, OH 45240-1634 2007.

▸ *from HSDB*

LD50 Mouse oral 250 mg/kg

Lewis, R.J. Sr. (ed) Sax's Dangerous Properties of Industrial Materials. 11th Edition. Wiley-Interscience, Wiley & Sons, Inc. Hoboken, NJ. 2004., p. 57

▸ *from HSDB*

LD50 Mouse ip 280 mg/kg

Lewis, R.J. Sr. (ed) Sax's Dangerous Properties of Industrial Materials. 11th Edition. Wiley-Interscience, Wiley & Sons, Inc. Hoboken, NJ. 2004., p. 57

▸ *from HSDB*

LD50 Mouse sc 1020 mg/kg

Lewis, R.J. Sr. (ed) Sax's Dangerous Properties of Industrial Materials. 11th Edition. Wiley-Interscience, Wiley & Sons, Inc. Hoboken, NJ. 2004., p. 57

▸ *from HSDB*

LD50 Dog oral 700 mg/kg

Lewis, R.J. Sr. (ed) Sax's Dangerous Properties of Industrial Materials. 11th Edition. Wiley-Interscience, Wiley & Sons, Inc. Hoboken, NJ. 2004., p. 57

▸ *from HSDB*

12.1.18 Ecotoxicity Values

LC50; Species: Daphnia magna (Water flea, <24 hr neonate); Conditions: freshwater, static, 21 deg C, pH 7.6; Concentration: 8.15 mM for 24 hr

Lilius H et al; Aquat Toxicol 30: 47-60 (1994) Available from, as of February 18, 2008: http://cfpub.epa.gov/ecotox/quick_query.htm

▸ *from HSDB*

LC50; Species: Daphnia pulex (Water flea, <24 hr); Conditions: freshwater, static, 20 deg C, pH 7.6; Concentration: 2.00 mM for 24 hr

Lilius H et al; Environ Toxicol Chem 14 (12): 2085-8 (1995) Available from, as of February 18, 2008: http://cfpub.epa.gov/ecotox/quick_query.htm

▸ *from HSDB*

LC50; Species: Artemia salina (Brine shrimp, age 2-3 instar larva); Conditions: saltwater, static, salinity 35 ppt; Concentration: 2120 umol/L for 24 hr

Calleja MC, Persoone G; Atla 20: 396-405 (1992) Available from, as of February 18, 2008: http://cfpub.epa.gov/ecotox/quick_query.htm

▸ *from HSDB*

LC50; Species: Streptocephalus proboscideus (Fairy shrimp, age 2-3 instar larva); Conditions: saltwater, static, 25 deg C, salinity 15 ppt; Concentration: 988 umol/L for 24 hr

Calleja MC, Persoone G; Atla 20: 396-405 (1992) Available from, as of February 18, 2008: http://cfpub.epa.gov/ecotox/quick_query.htm

from HSDB

LC50; Species: Brachionus calyciflorus (Rotifer, post-hatch); Conditions: saltwater, static, 25 deg C, salinity 15 ppt; Concentration: 785 umol/L for 24 hr

Calleja MC, Persoone G; Atla 20: 396-405 (1992) Available from, as of February 18, 2008: http://cfpub.epa.gov/ecotox/quick_query.htm

from HSDB

LC50; Species: Brachionus plicatilis (Rotifer, post-hatch); Conditions: saltwater, static, 25 deg C, salinity 15 ppt; Concentration: 745 umol/L for 24 hr

Calleja MC, Persoone G; Atla 20: 396-405 (1992) Available from, as of February 18, 2008: http://cfpub.epa.gov/ecotox/quick_query.htm

from HSDB

12.1.19 Populations at Special Risk

Aspirin use may be associated with the development of Reye's syndrome in children and teenagers with acute febrile illnesses, especially influenza and varicella. It is recommended that salicylate therapy not be initiated in febrile pediatric or adolescent patients until after the presence of such an illness has been ruled out. Also, it is recommended that chronic salicylate therapy in these patients be discontinued if a fever occurs, and not resumed until it has been determined that an illness that may predispose to Reye's syndrome is not present or has run its course. Other forms of salicylate toxicity may also be more prevalent in pediatric patients, especially children who have a fever or are dehydrated.

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2577

from HSDB

Especially careful monitoring of the serum salicylate concentration is recommended in pediatric patients with Kawasaki disease. Absorption of aspirin is impaired during the early febrile stage of the disease; therapeutic anti-inflammatory plasma salicylate concentrations may be extremely difficult to achieve. Also, as the febrile stage passes, absorption is improved; salicylate toxicity may occur if dosage is not readjusted.

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2577

from HSDB

Patients with severe hepatic damage, hypoprothrombinemia, vitamin K deficiency, or hemophilia should avoid aspirin because the inhibition of platelet hemostasis can result in hemorrhage.

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 688

from HSDB

Some persons, particularly asthmatics, exhibit marked sensitivity to acetylsalicylic acid which provokes various reactions including urticaria and other skin eruptions, angioneurotic edema, rhinitis and severe, even fatal, paroxysmal bronchospasm and dyspnea, hypotension, shock and syncope.

International Programme on Chemical Safety; Poisons Information Monograph: Acetylsalicylic Acid (PIM 006) (1991) Available from, as of March 10, 2008: http://www.inchem.org/pages/pims.html

from HSDB

Aspirin can cause a mild degree of hemolysis in individuals with a deficiency of glucose-6-phosphate dehydrogenase.

Hardman, J.G., L.E. Limbird, P.B., A.G. Gilman. Goodman and Gilman's The Pharmacological Basis of Therapeutics. 11th ed. New York, NY: McGraw-Hill, 2006., p. 688

from HSDB

12.1.20 Protein Binding

High (99.5%) to albumin. Decreases as plasma salicylate concentration increases, with reduced plasma albumin concentration or renal dysfunction, and during pregnancy.

from DrugBank

12.2 Ecological Information

12.2.1 Environmental Fate/Exposure Summary

Acetylsalicylic acid 's production and use as a common over-the-counter analgesic, anti-pyretic, anti-inflammatory and anti-thrombetic may result in its release to the environment through various waste streams. If released to air, a vapor pressure of 2.5X10-5 mm Hg at 25 deg C indicates that acetylsalicylic acid is expected to exist in both the vapor and particulate phases in the ambient atmosphere. Vapor-phase acetylsalicylic acid is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 12 days. Particulate-phase acetylsalicylic acid may be removed from the air by wet or dry deposition. Acetylsalicylic acid contains chromophores that absorb at wavelengths >290 nm and therefore may be susceptible to direct photolysis by sunlight. If released to soil, acetylsalicylic acid is expected to have high mobility based upon an estimated Koc of 100. The pKa of acetylsalicylic acid is 3.49, indicating that this compound will exist almost entirely in the anion form in the environment and anions generally do not adsorb more strongly to soils containing organic carbon and clay than their neutral counterparts. Volatilization from moist soil surfaces is not expected to be an important fate process since anions do not volatilize. Acetylsalicylic acid is not expected to volatilize from dry soil surfaces based upon its vapor pressure. No biodegradation studies were located for acetylsalicylic acid in soil or water; however, acetylsalicylic acid was classified as readily biodegradable in screening tests. An aqueous hydrolysis half-life of 6.2 days at pH 7.4 and 17 deg C, suggests hydrolysis may occur in moist soils. If released into water, acetylsalicylic acid is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. Volatilization from water surfaces is not an important fate process since anions do not volatilize. An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low. Aqueous hydrolysis half-lives ranging from 1.2 hours to 12.5 days at pH 3.5 to 11.3 and 17 deg C, indicates hydrolysis will be an important aquatic fate process. Occupational exposure to acetylsalicylic acid may occur through inhalation of dust and dermal contact with this compound at workplaces where acetylsalicylic acid is produced or used. The general population is exposed to acetylsalicylic acid through its ingestion as an over-the-counter medication. (SRC)

from HSDB

12.2.2 Artificial Sources

Acetylsalicylic acid 's production and use as a common over-the-counter analgesic, anti-pyretic, anti-inflammatory and anti-thrombetic(1) may result in its release to the environment through various waste streams(SRC).

(1) O'Neil MJ, ed; The Merck Index. 14th ed Whitehouse Station, NJ: Merck and Co Inc pp. 140 (2006)

from HSDB

12.2.3 Environmental Fate

TERRESTRIAL FATE: Based on a classification scheme(1), an estimated Koc value of 100(SRC), determined from a log Kow of 1.19(2) and a regression-derived equation(3), indicates that acetylsalicylic acid is expected to have high mobility in soil(SRC). The pKa of acetylsalicylic acid is 3.49(4), indicating that this compound will almost entirely exist in anion form in the environment and anions generally do not adsorb as strongly to soils containing organic carbon and clay than their neutral counterparts(4). Volatilization of acetylsalicylic acid from moist soil surfaces will not be an important fate process(SRC) since anions do not volatilize. Acetylsalicylic acid is not expected to volatilize from dry soil surfaces(SRC) based upon a vapor pressure of 2.5X10-5 mm Hg(6). No biodegradation studies were located for acetylsalicylic acid in soil(SRC, 2008); however, acetylsalicylic acid was classified as readily biodegradable in screening tests(7,8). An aqueous hydrolysis half-life of 6.3 days at pH 7.4 and 17 deg C(9), suggests hydrolysis may occur in moist soils(SRC).

(1) Swann RL et al; Res Rev 85: 17-28 (1983) (2) Hansch C et al; Exploring QSAR. Hydrophobic, Electronic, and Steric Constants. ACS Prof Ref Book. Heller SR, consult. ed., Washington, DC: Amer Chem Soc p. 54 (1995)(3) Lyman WJ et al; Handbook of Chemical Property Estimation Methods. Washington, DC: Amer Chem Soc pp. 4-9 (1990) (4) O'Neil MJ, ed; The Merck Index. 14th ed Whitehouse Station, NJ: Merck and Co Inc pp. 140 (2006) (5) Doucette WJ; pp. 141-188 in Handbook of Property Estimation Methods for Chemicals. Boethling RS, Mackay D, eds. Boca Raton, FL: Lewis Publ (2000) (6) Eisenreich SJ et al; Environ Sci Technol 15: 30-8 (1981) (7) Rogers HR; Sci Total Environ 185: 3-26 (1996) (8) Halling-Sorensen B et al; Chemosphere 36: 357-393 (1998) (9) Organ Econ Coop Develop; OECD Guidelines for Testing of Chemicals. OECD. Berlin: Umweltbundesant pp. 842 (1981)

from HSDB

AQUATIC FATE: Based on a classification scheme(1), an estimated Koc value of 100(SRC), determined from a log Kow of 1.19(2) and a regression-derived equation(3), indicates that acetylsalicylic acid is not expected to adsorb to suspended solids and sediment(SRC). A pKa of 3.49(4) indicates acetylsalicylic acid will exist almost entirely in the anion form at pH values of 5 to 9 and therefore volatilization from water surfaces is not expected to be an important fate process. According to a classification scheme(5), an estimated BCF of 3(SRC), from its log Kow(2) and a regression-derived equation(6), suggests the potential for bioconcentration in aquatic organisms is low(SRC). A hydrolysis half-life of 6.3 days at pH 7.4 and 17 deg C was calculated for acetylsalicylic acid(7). No biodegradation studies were located for acetylsalicylic acid in water(SRC, 2008); however, acetylsalicylic acid was classified as readily biodegradable in screening tests(8,9).

(1) Swann RL et al; Res Rev 85: 17-28 (1983) (2) Hansch C et al; Exploring QSAR. Hydrophobic, Electronic, and Steric Constants. ACS Prof Ref Book. Heller SR, consult. ed., Washington, DC: Amer Chem Soc p. 54 (1995)(3) Lyman WJ et al; Handbook of Chemical Property Estimation Methods. Washington, DC: Amer Chem Soc pp. 4-9, 15-1 to 15-29 (1990) (4) O'Neil MJ, ed; The Merck Index. 14th ed Whitehouse Station, NJ: Merck and Co Inc pp. 140 (2006) (5) Franke C et al; Chemosphere 29: 1501-14 (1994) (6) Meylan WM et al; Environ Toxicol Chem 18: 664-72 (1999) (7) Organ Econ Coop Develop; OECD Guidelines for Testing of Chemicals. OECD. Berlin: Umweltbundesant pp. 842 (1981) (8) Rogers HR; Sci Total Environ 185: 3-26 (1996) (9) Halling-Sorensen B et al; Chemosphere 36: 357-393 (1998)

from HSDB

ATMOSPHERIC FATE: According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere(1), acetylsalicylic acid, which has a vapor pressure of 2.5X10-5 mm Hg at 25 deg C(2) is expected to exist in both the vapor and particulate phases in the ambient atmosphere. Vapor-phase acetylsalicylic acid is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals(SRC); the half-life for this reaction in air is estimated to be 12 days(SRC), calculated from its rate constant of 1.3X10-12 cu cm/molecule-sec at 25 deg C(SRC) that was derived using a structure estimation method(3). Particulate-phase acetylsalicylic acid may be removed from the air by wet or dry deposition(SRC). Acetylsalicylic acid contains chromophores that absorb at wavelengths >290 nm(4) and therefore may be susceptible to direct photolysis by sunlight(SRC).

(1) Bidleman TF; Environ Sci Technol 22: 361-367 (1988) (2) Eisenreich SJ et al; Environ Sci Technol 15: 30-8 (1981) (3) Meylan WM, Howard PH; Chemosphere 26: 2293-99 (1993) (4) Lyman WJ et al; Handbook of Chemical Property Estimation Methods. Washington, DC: Amer Chem Soc pp. 8-12 (1990)

from HSDB

12.2.4 Biodegredation

AEROBIC: No biodegradation studies were located for acetylsalicylic acid in soil or natural water(SRC, 2008); however, acetylsalicylic acid was classified as readily biodegradable in screening tests using sewage sludge inoculum(1,2). Conversely, only a 0.09% total biodegradation was predicted in a study of a UK sewage treatment plant; the compound is detected in the UK environment(3).

(1) Rogers HR; Sci Total Environ 185: 3-26 (1996) (2) Halling-Sorensen B et al; Chemosphere 36: 357-393 (1998) (3) Jones OAH et al; Water Res 36: 5013-22 (2002)

from HSDB

AEROBIC: No biodegradation studies were located for acetylsalicylic acid in soil or natural water(SRC, 2008); however, acetylsalicylic acid was classified as readily biodegradable in screening tests using sewage sludge inoculum(1,2). Conversely, only a 0.09% total biodegradation was predicted in a study of a UK sewage treatment plant; the compound is detected in the UK environment(3).

(1) Shelton DR, Tiedje JM; Development of Tests for Determining Anaerobic Biodegradation Potential USEPA 560/5-81-013, NTIS PB84-166495 (1981) (2) Shelton DR, Tiedje JM; App Env Microbiol 47: 850-7 (1984)

from HSDB

12.2.5 Abiotic Degredation

The rate constant for the vapor-phase reaction of acetylsalicylic acid with photochemically-produced hydroxyl radicals has been estimated as 1.3X10-12 cu cm/molecule-sec at 25 deg C(SRC) using a structure estimation method(1). This corresponds to an atmospheric half-life of about 12 days at an atmospheric concentration of 5X10+5 hydroxyl radicals per cu cm(1). Hydrolysis rate constants for acetylsalicylic acid in water at 17 deg C and pHs 3.5, 5.0, 7.4, 9.5 and 11.3 were 6.5X10-7/sec, 1.5X10-6/sec, 1.3X10-6/sec, 3.7X10-6/sec and 1.6X10-4/sec, which correspond to half-lives of 12.5 days, 5.4 days, 6.3 days, 2.2 days and 1.2 hrs, respectively(2). Acetylsalicylic acid contains chromophores that absorb at wavelengths >290 nm(3) and therefore may be susceptible to direct photolysis by sunlight(SRC).

(1) Meylan WM, Howard PH; Chemosphere 26: 2293-99 (1993) (2) Organ Econ Coop Develop; OECD Guidelines for Testing of Chemicals. OECD. Berlin: Umweltbundesant pp. 842 (1981) (3) Lyman WJ et al; Handbook of Chemical Property Estimation Methods. Washington, DC: Amer Chem Soc pp. 8-12 (1990)

from HSDB

12.2.6 Bioconcentration

An estimated BCF of 3 was calculated in fish for acetylsalicylic acid (SRC), using a log Kow of 1.19(1) and a regression-derived equation(2). According to a classification scheme(3), this BCF suggests the potential for bioconcentration in aquatic organisms is low(SRC).

(1) Hansch C et al; Exploring QSAR. Hydrophobic, Electronic, and Steric Constants. ACS Prof Ref Book. Heller SR, consult. ed., Washington, DC: Amer Chem Soc p. 54 (1995) (2) Meylan WM et al; Environ Toxicol Chem 18: 664-72 (1999) (3) Franke C et al; Chemosphere 29: 1501-14 (1994)

▸ *from HSDB*

12.2.7 Soil Adsorption/Mobility

The Koc of acetylsalicylic acid is estimated as 100(SRC), using a log Kow of 1.19(1) and a regression-derived equation(2). According to a classification scheme(3), this estimated Koc value suggests that acetylsalicylic acid is expected to have high mobility in soil(SRC). The pKa of acetylsalicylic acid is estimated as 3.49(4), indicating that this compound will primarily exist as an anion in the environment and anions generally do not adsorb as strongly to soils containing organic carbon and clay than their neutral counterparts(5).

(1) Hansch C et al; Exploring QSAR. Hydrophobic, Electronic, and Steric Constants. ACS Prof Ref Book. Heller SR, consult. ed., Washington, DC: Amer Chem Soc p. 54 (1995) (2) Lyman WJ et al; Handbook of Chemical Property Estimation Methods. Washington, DC: Amer Chem Soc pp. 4-9 (1990) (3) Swann RL et al; Res Rev 85: 17-28 (1983) (4) O'Neil MJ, ed; The Merck Index. 14th ed Whitehouse Station, NJ: Merck and Co Inc pp. 140 (2006) (5) Doucette WJ; pp. 141-188 in Handbook of Property Estimation Methods for Chemicals. Boethling RS, Mackay D, eds. Boca Raton, FL: Lewis Publ (2000)

▸ *from HSDB*

12.2.8 Volatilization from Water/Soil

The pKa of acetylsalicylic acid is 3.49(1). This pKa indicates that acetylsalicylic acid is expected to primarily exist in anionic form in moist soil and water(SRC). Volatilization from moist soil and water is not expected since anions do not volatilize(SRC). Acetylsalicylic acid is not expected to volatilize from dry soil surfaces(SRC) based upon a vapor pressure of 2.5X10-5 mm Hg(2).

(1) O'Neil MJ, ed; The Merck Index. 14th ed Whitehouse Station, NJ: Merck and Co Inc pp. 140 (2006) (2) Eisenreich SJ et al; Environ Sci Technol 15: 30-38 (1981)

▸ *from HSDB*

12.2.9 Water Concentrations

SURFACE WATER: Acetylsalicylic acid was detected in surface water at a max concn of 0.34 ug/L(1).[(1) Daughton CG, Ternes TA; Environ Health Perspect 107: 907-938 (1999)] Full text: PMC1566206
Abstract: PubMed

▸ *from HSDB*

12.2.10 Effluents Concentrations

That pharmaceutically active compounds such as acetylsalicylic acid enter the environment via sewage has been known for over 20 years(1). Acetylsalicylic acid was detected in 22 effluent samples from a German sewage treatment facility at a max concn of 1.5 ug/L(2). Acetylsalicylic acid was detected in effluent from an unspecified sewage treatment plant in Germany at a max concn of 95.62 ug/L(3). Concentrations of approximately 1 ug/L in sewage effluent to a range of <50 to 1,510 ug/L in effluent from sedimentation tanks have also been reported(4).

(1) Daughton CD, Ternes TA; Environ Health Pesrpect 107: 907-38 (1999) (2) Ternes T; Wasser Boden 53: 9-14 (2001) (3) Stan HJ, Heberer T; Analusis 25: M20-M23(1997) (4) Halling-Sorensen B et al; Chemosphere 36: 357-93 (1998)

▸ *from HSDB*

12.2.11 Milk Concentrations

EXPERIMENTAL: Acetylsalicylic acid and the acetylsalicylate ion were detected in the breast milk of nursing mothers within 1 hour of aspirin (acetylsalicylic acid) ingestion(1).

(1) Cone MV et al; Chemicals identified in Human Biological Materials: A Database. USEPA-560/13-80-036a (1981)

▸ *from HSDB*

Peak salicylate concentrations of 173 to 483 ug/mL have been measured in /breast milk/ 5 to 8 hours after maternal ingestion of a single 650 mg dose /of aspirin/.

Thomson/Micromedex. Drug Information for the Health Care Professional. Volume 1, Greenwood Village, CO. 2007., p. 2576

▸ *from HSDB*

12.2.12 Probable Routes of Human Exposure

NIOSH (NOES Survey 1981-1983) has statistically estimated that 191 workers (81 of these are female) are potentially exposed to acetylsalicylic acid in the US(1). Occupational exposure to acetylsalicylic acid may occur through inhalation of dust and dermal contact with this compound at workplaces where acetylsalicylic acid is produced or used(SRC). The general population is exposed to acetylsalicylic acid through its ingestion as an over-the-counter medication.

(1) NIOSH; NOES. National Occupational Exposure Survey conducted from 1981-1983. Estimated numbers of employees potentially exposed to specific agents by 2-digit standard industrial classification (SIC). Available at http://www.cdc.gov/noes/ as of Dec 17, 2007.

▸ *from HSDB*

12.2.13 Body Burdens

Acetylsalicylic acid and the acetylsalicylate ion were detected in the breast milk of nursing mothers within 1 hour of aspirin (acetylsalicylic acid) ingestion(1).

(1) Cone MV et al; Chemicals identified in Human Biological Materials: A Database. USEPA-560/13-80-036a (1981)

from HSDB

13 Literature

13.1 Depositor Provided PubMed Citations

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13.2 NLM Curated PubMed Citations

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13.3 Synthesis References

Synthesis Reference

Marino Gobetti, Guido Vandoni, "Acetylsalicylic acid thioesters, a process for their preparation and pharmaceutical compositions containing them." U.S. Patent US4563443, issued March, 1981.

from DrugBank

Chen, Hong; Long, Xiang; Huang, Siqing. Synthesis of aspirin with vitamin C as catalyst. Huaxue Shijie (2004), 45(12), 642-643.

from Human Metabolome Database (HMDB)

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Macdonald S: Aspirin use to be banned in under 16 year olds. BMJ. 2002 Nov 2;325(7371):988.
Abstract: PubMed

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Sneader W: The discovery of aspirin: a reappraisal. BMJ. 2000 Dec 23-30;321(7276):1591-4.
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Aukerman G, Knutson D, Miser WF: Management of the acute migraine headache. Am Fam Physician. 2002 Dec 1;66(11):2123-30.
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Authors unspecified: Randomised trial of intravenous streptokinase, oral aspirin, both, or neither among 17,187 cases of suspected acute myocardial infarction: ISIS-2. ISIS-2 (Second International Study of Infarct Survival) Collaborative Group. Lancet. 1988 Aug 13;2(8607):349-60.
Abstract: PubMed

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General Reference

Dorsch MP, Lee JS, Lynch DR, Dunn SP, Rodgers JE, Schwartz T, Colby E, Montague D, Smyth SS: Aspirin resistance in patients with stable coronary artery disease with and without a history of myocardial infarction. Ann Pharmacother. 2007 May;41(5):737-41. Epub 2007 Apr 24.
Abstract: PubMed

from DrugBank

13.5 Metabolite References

Download

1 to 5 of 25 View More

PMID	Reference
16635110	Markuszewski L, Rosiak M, Golanski J, Rysz J, Spychalska M, Watala C: Reduced blood platelet sensitivity to aspirin in coronary artery disease: are dyslipidaemia and inflammatory states possible factors predisposing to sub-optimal platelet response to aspirin? Basic Clin Pharmacol Toxicol. 2006 May;98(5):503-9.
16785341	Frelinger AL 3rd, Furman MI, Linden MD, Li Y, Fox ML, Barnard MR, Michelson AD: Residual arachidonic acid-induced platelet activation via an adenosine diphosphate-dependent but cyclooxygenase-1- and cyclooxygenase-2-independent pathway: a 700-patient study of aspirin resistance. Circulation. 2006 Jun 27;113(25):2888-96. Epub 2006 Jun 19.
16359503	Eikelboom JW, Hankey GJ, Thom J, Claxton A, Yi Q, Gilmore G, Staton J, Barden A, Norman PE: Enhanced antiplatelet effect of clopidogrel in patients whose platelets are least inhibited by aspirin: a randomized crossover trial. J Thromb Haemost. 2005 Dec;3(12):2649-55.
16369302	Eikelboom J, Feldman M, Mehta SR, Michelson AD, Oates JA, Topol E: Aspirin resistance and its implications in clinical practice. MedGenMed. 2005 Jul 11;7(3):76.
16517326	Konrad CJ, Schuepfer GK, Gerber H, Rukwied R, Schmelz M, Schley M: Duration of effects of aspirin on platelet function in healthy volunteers: an analysis using the PFA-100. J Clin Anesth. 2006 Feb;18(1):12-7.

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13.6 Springer Nature References

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from Springer Nature

13.7 Thieme References

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from Thieme Chemistry

13.8 Chemical Co-Occurrences in Literature

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13.9 Chemical-Disease Co-Occurrences in Literature

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from PubChem

13.10 Chemical-Gene Co-Occurrences in Literature

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from PubChem

14 Patents

1. US5972916
2. US6015577
3. US6926907
4. US9101637
5. US9226892
6. US8865187
7. US9216150
8. US9351984
9. US9539214
10. US9364439
11. US8206741
12. US9987231

from DrugBank

14.1 Depositor-Supplied Patent Identifiers

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14.2 FDA Orange Book Patents

FDA Orange Book Patents: 1 of 10 (FDA Orange Book Patent ID)

Patent	9216150
Expiration	Sep 29, 2032
Applicant	PLX PHARMA
Drug Application	N203697 (Prescription Drug: VAZALORE. Ingredients: ASPIRIN)

from FDA Orange Book

FDA Orange Book Patents: 2 of 10 (FDA Orange Book Patent ID)

Patent	9226892
Expiration	Sep 29, 2032
Applicant	PLX PHARMA
Drug Application	N203697 (Prescription Drug: VAZALORE. Ingredients: ASPIRIN)

from FDA Orange Book

FDA Orange Book Patents: 3 of 10 (FDA Orange Book Patent ID)

Patent	9987231
Expiration	Jan 2, 2033
Applicant	GENUS LIFESCIENCES
Drug Application	1. N205103 (Prescription Drug: YOSPRALA. Ingredients: ASPIRIN 2. OMEPRAZOLE) 3. N205103 (Prescription Drug: YOSPRALA. Ingredients: ASPIRIN 4. OMEPRAZOLE)

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15 Biomolecular Interactions and Pathways

15.1 Protein Bound 3-D Structures

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15.2 Biosystems and Pathways

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15.3 DrugBank Interactions

Target	Prostaglandin G/H synthase 1
Action	inhibitor
PubChem Protein Target	P23219
PubChem Gene Target	PTGS1
General Function	Prostaglandin-endoperoxide synthase activity
Specific Function	Converts arachidonate to prostaglandin H2 (PGH2), a committed step in prostanoid synthesis. Involved in the constitutive production of prostanoids in particular in the stomach and platelets. In gastric epithelial cells, it is a key step in the generation of prostaglandins, such as prostaglandin E2 (PGE2), which plays an important role in cytoprotection. In platelets, it is involved in the generation of thromboxane A2 (TXA2), which promotes platelet activation and aggregation, vasoconstriction and proliferation of vascular smooth muscle cells.
Reference	Stevenson DD, Szczeklik A: Clinical and pathologic perspectives on aspirin sensitivity and asthma. J Allergy Clin Immunol. 2006 Oct;118(4):773-86; quiz 787-8. Epub 2006 Sep 1. Abstract: PubMed
Reference	Flipo RM: [Are the NSAIDs able to compromising the cardio-preventive efficacy of aspirin?]. Presse Med. 2006 Sep;35(9 Spec No 1):1S53-60. Abstract: PubMed
Reference	Schwartz KA: Aspirin resistance: a review of diagnostic methodology, mechanisms, and clinical utility. Adv Clin Chem. 2006;42:81-110. Abstract: PubMed
Reference	Birnbaum Y, Ye Y, Lin Y, Freeberg SY, Huang MH, Perez-Polo JR, Uretsky BF: Aspirin augments 15-epi-lipoxin A4 production by lipopolysaccharide, but blocks the pioglitazone and atorvastatin induction of 15-epi-lipoxin A4 in the rat heart. Prostaglandins Other Lipid Mediat. 2007 Feb;83(1-2):89-98. Epub 2006 Nov 7. Abstract: PubMed
Reference	Guthikonda S, Lev EI, Patel R, DeLao T, Bergeron AL, Dong JF, Kleiman NS: Reticulated platelets and uninhibited COX-1 and COX-2 decrease the antiplatelet effects of aspirin. J Thromb Haemost. 2007 Mar;5(3):490-6. Abstract: PubMed
Reference	Chen X, Ji ZL, Chen YZ: TTD: Therapeutic Target Database. Nucleic Acids Res. 2002 Jan 1;30(1):412-5. Abstract: PubMed

from DrugBank

Enzyme	Cytochrome P450 2C9
Action	substrate
PubChem Protein Target	P11712
PubChem Gene Target	CYP2C9
General Function	Steroid hydroxylase activity
Specific Function	Cytochromes P450 are a group of heme-thiolate monooxygenases. In liver microsomes, this enzyme is involved in an NADPH-dependent electron transport pathway. It oxidizes a variety of structurally unrelated compounds, including steroids, fatty acids, and xenobiotics. This enzyme contributes to the wide pharmacokinetics variability of the metabolism of drugs such as S-warfarin, diclofenac, phenytoin, tolbutamide and losartan.
Reference	Preissner S, Kroll K, Dunkel M, Senger C, Goldsobel G, Kuzman D, Guenther S, Winnenburg R, Schroeder M, Preissner R: SuperCYP: a comprehensive database on Cytochrome P450 enzymes including a tool for analysis of CYP-drug interactions. Nucleic Acids Res. 2010 Jan;38(Database issue):D237-43. doi: 10.1093/nar/gkp970. Epub 2009 Nov 24. Abstract: PubMed
Reference	Rendic S: Summary of information on human CYP enzymes: human P450 metabolism data. Drug Metab Rev. 2002 Feb-May;34(1-2):83-448. Abstract: PubMed

from DrugBank

Transporter	Solute carrier family 22 member 6
Action	inhibitor
PubChem Protein Target	Q4U2R8
PubChem Gene Target	SLC22A6
General Function	Sodium-independent organic anion transmembrane transporter activity
Specific Function	Involved in the renal elimination of endogenous and exogenous organic anions. Functions as organic anion exchanger when the uptake of one molecule of organic anion is coupled with an efflux of one molecule of endogenous dicarboxylic acid (glutarate, ketoglutarate, etc). Mediates the sodium-independent uptake of 2,3-dimercapto-1-propanesulfonic acid (DMPS) (By similarity). Mediates the sodium-independent uptake of p-aminohippurate (PAH), ochratoxin (OTA), acyclovir (ACV), 3'-azido-3-'deoxythymidine (AZT), cimetidine (CMD), 2,4-dichloro-phenoxyacetate (2,4-D), hippurate (HA), indoleacetate (IA), indoxyl sulfate (IS) and 3-carboxy-4-methyl-5-propyl-2-furanpropionate (CMPF), cidofovir, adefovir, 9-(2-phosphonylmethoxyethyl) guanine (PMEG), 9-(2-phosphonylmethoxyethyl) diaminopurine (PMEDAP) and edaravone sulfate. PAH uptake is inhibited by p-chloromercuribenzenesulphonate (PCMBS), diethyl pyrocarbonate (DEPC), sulindac, diclofenac, carprofen, glutarate and okadaic acid (By similarity). PAH uptake is inhibited by benzothiazolylcysteine (BTC), S-chlorotrifluoroethylcysteine (CTFC), cysteine S-conjugates S-dichlorovinylcysteine (DCVC), furosemide, steviol, phorbol 12-myristate 13-acetate (PMA), calcium ionophore A23187, benzylpenicillin, furosemide, indomethacin, bumetamide, losartan, probenecid, phenol red, urate, and alpha-ketoglutarate.
Reference	Apiwattanakul N, Sekine T, Chairoungdua A, Kanai Y, Nakajima N, Sophasan S, Endou H: Transport properties of nonsteroidal anti-inflammatory drugs by organic anion transporter 1 expressed in Xenopus laevis oocytes. Mol Pharmacol. 1999 May;55(5):847-54. Abstract: PubMed

from DrugBank

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16 Biological Test Results

16.1 BioAssay Results

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17 Classification

17.1 Ontologies

17.1.1 MeSH Tree

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17.1.2 ChEBI Ontology

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17.1.3 KEGG: Drug

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17.1.4 KEGG: ATC

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17.1.5 KEGG: Target-based Classification of Drugs

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17.1.6 KEGG: JP15

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17.1.7 KEGG: Risk Category of Japanese OTC Drugs

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17.1.8 KEGG: OTC drugs

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17.1.9 KEGG: Drug Classes

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17.1.10 WHO ATC Classification System

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17.1.11 FDA Pharm Classes

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17.1.12 WIPO IPC

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17.1.13 ChemIDplus

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17.1.14 CAMEO Chemicals

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17.1.15 Guide to PHARMACOLOGY Target Classification

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17.1.16 ChEMBL Target Tree

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18 Information Sources

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   https://cameochemicals.noaa.gov/chemical/19712 https://cameochemicals.noaa.gov/chemical/19712
   CAMEO Chemical Reactivity Classification
   https://cameochemicals.noaa.gov/browse/react https://cameochemicals.noaa.gov/browse/react

2. ChemIDplus /source/ChemIDplus
   Aspirin [USP:BAN:JAN]
   https://chem.nlm.nih.gov/chemidplus/sid/0000050782 https://chem.nlm.nih.gov/chemidplus/sid/0000050782
   ChemIDplus Chemical Information Classification
   https://chem.sis.nlm.nih.gov/chemidplus/chemidheavy.jsp https://chem.sis.nlm.nih.gov/chemidplus/chemidheavy.jsp

3. DTP/NCI /source/DTP/NCI
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   https://dtp.cancer.gov/dtpstandard/servlet/dwindex?searchtype=NSC&outputformat=html&searchlist=406186 https://dtp.cancer.gov/dtpstandard/servlet/dwindex?searchtype=NSC&outputformat=html&searchlist=406186
   aspirin
   https://dtp.cancer.gov/dtpstandard/servlet/dwindex?searchtype=NSC&outputformat=html&searchlist=755899 https://dtp.cancer.gov/dtpstandard/servlet/dwindex?searchtype=NSC&outputformat=html&searchlist=755899
   aspirin
   https://dtp.cancer.gov/dtpstandard/servlet/dwindex?searchtype=NSC&outputformat=html&searchlist=27223 https://dtp.cancer.gov/dtpstandard/servlet/dwindex?searchtype=NSC&outputformat=html&searchlist=27223

4. DrugBank /source/DrugBank
   Acetylsalicylic acid
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   http://www.drugbank.ca/drugs/DB00945#targets http://www.drugbank.ca/drugs/DB00945#targets
   http://www.drugbank.ca/drugs/DB00945#enzymes http://www.drugbank.ca/drugs/DB00945#enzymes
   http://www.drugbank.ca/drugs/DB00945#transporters http://www.drugbank.ca/drugs/DB00945#transporters
   http://www.drugbank.ca/drugs/DB00945#carriers http://www.drugbank.ca/drugs/DB00945#carriers

5. EPA Chemicals under the TSCA /source/EPA Chemicals under the TSCA
   Benzoic acid, 2-(acetyloxy)-
   http://www.epa.gov/chemical-data-reporting http://www.epa.gov/chemical-data-reporting

6. EPA DSStox /source/EPA DSStox
   Aspirin
   https://comptox.epa.gov/dashboard/dsstoxdb/results?search=DTXSID5020108 https://comptox.epa.gov/dashboard/dsstoxdb/results?search=DTXSID5020108

7. European Chemicals Agency (ECHA) /source/European Chemicals Agency (ECHA)
   O-acetylsalicylic acid
   https://echa.europa.eu/substance-information/-/substanceinfo/100.000.059 https://echa.europa.eu/substance-information/-/substanceinfo/100.000.059
   O-acetylsalicylic acid
   https://echa.europa.eu/information-on-chemicals/cl-inventory-database/-/discli/details/88331 https://echa.europa.eu/information-on-chemicals/cl-inventory-database/-/discli/details/88331

8. Human Metabolome Database (HMDB) /source/Human Metabolome Database (HMDB)
   Aspirin
   http://www.hmdb.ca/metabolites/HMDB0001879 http://www.hmdb.ca/metabolites/HMDB0001879

9. ILO-ICSC /source/ILO-ICSC
   2-(ACETYLOXY)BENZOIC ACID
   http://www.ilo.org/dyn/icsc/showcard.display?p_version=2&p_card_id=0822 http://www.ilo.org/dyn/icsc/showcard.display?p_version=2&p_card_id=0822

10. OSHA Occupational Chemical DB /source/OSHA Occupational Chemical DB
    ACETYLSALICYLIC ACID
    http://www.osha.gov/chemicaldata/chemResult.html?RecNo=384 http://www.osha.gov/chemicaldata/chemResult.html?RecNo=384

11. The National Institute for Occupational Safety and Health (NIOSH) /source/The National Institute for Occupational Safety and Health (NIOSH)
    Acetylsalicylic acid
    https://www.cdc.gov/niosh/npg/npgd0010.html https://www.cdc.gov/niosh/npg/npgd0010.html
    Salicylic acid, acetate
    https://www.cdc.gov/niosh-rtecs/VOAAE60.html https://www.cdc.gov/niosh-rtecs/VOAAE60.html

12. ChEBI /source/ChEBI
    Acetylsalicylic acid
    http://www.ebi.ac.uk/chebi/searchId.do?chebiId=CHEBI:15365 http://www.ebi.ac.uk/chebi/searchId.do?chebiId=CHEBI:15365
    ChEBI Ontology
    http://www.ebi.ac.uk/chebi/userManualForward.do#ChEBI%20Ontology http://www.ebi.ac.uk/chebi/userManualForward.do#ChEBI%20Ontology

13. FDA Pharm Classes /source/FDA Pharm Classes
    ASPIRIN
    https://www.accessdata.fda.gov/spl/data/890dd4fd-5fdc-45e8-a38a-bcf005793ef8/890dd4fd-5fdc-45e8-a38a-bcf005793ef8.xml https://www.accessdata.fda.gov/spl/data/890dd4fd-5fdc-45e8-a38a-bcf005793ef8/890dd4fd-5fdc-45e8-a38a-bcf005793ef8.xml
    ASPIRIN
    https://www.accessdata.fda.gov/spl/data/82e04776-4e1d-4a45-8d81-8662176ae54d/82e04776-4e1d-4a45-8d81-8662176ae54d.xml https://www.accessdata.fda.gov/spl/data/82e04776-4e1d-4a45-8d81-8662176ae54d/82e04776-4e1d-4a45-8d81-8662176ae54d.xml
    FDA Pharmacological Classification
    https://www.fda.gov/ForIndustry/DataStandards/StructuredProductLabeling/ucm162549.htm https://www.fda.gov/ForIndustry/DataStandards/StructuredProductLabeling/ucm162549.htm

14. NCIt /source/NCIt

Aspirin
https://ncit.nci.nih.gov/ncitbrowser/ConceptReport.jsp?dictionary=NCI_Thesaurus&ns=NCI_Thesaurus&code=C287 https://ncit.nci.nih.gov/ncitbrowser/ConceptReport.jsp?dictionary=NCI_Thesaurus&ns=NCI_Thesaurus&code=C287

15. HSDB /source/HSDB

ACETYLSALICYLIC ACID
https://toxnet.nlm.nih.gov/cgi-bin/sis/search/r?dbs+hsdb:@term+@rn+@rel+50-78-2 https://toxnet.nlm.nih.gov/cgi-bin/sis/search/r?dbs+hsdb:@term+@rn+@rel+50-78-2

16. ClinicalTrials.gov /source/ClinicalTrials.gov

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17. DailyMed /source/DailyMed

ACETAMINOPHEN; ASPIRIN; CAFFEINE
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ASPIRIN; OMEPRAZOLE
https://dailymed.nlm.nih.gov/dailymed/search.cfm?labeltype=all&query=YOSPRALA https://dailymed.nlm.nih.gov/dailymed/search.cfm?labeltype=all&query=YOSPRALA
ASPIRIN; CARISOPRODOL; CODEINE PHOSPHATE
https://dailymed.nlm.nih.gov/dailymed/search.cfm?labeltype=all&query=CARISOPRODOL,+ASPIRIN+AND+CODEINE+PHOSPHATE https://dailymed.nlm.nih.gov/dailymed/search.cfm?labeltype=all&query=CARISOPRODOL,+ASPIRIN+AND+CODEINE+PHOSPHATE
ASPIRIN; OXYCODONE HYDROCHLORIDE
https://dailymed.nlm.nih.gov/dailymed/search.cfm?labeltype=all&query=OXYCODONE+AND+ASPIRIN https://dailymed.nlm.nih.gov/dailymed/search.cfm?labeltype=all&query=OXYCODONE+AND+ASPIRIN
ASPIRIN
https://dailymed.nlm.nih.gov/dailymed/search.cfm?labeltype=all&query=ASPIRIN https://dailymed.nlm.nih.gov/dailymed/search.cfm?labeltype=all&query=ASPIRIN
ASPIRIN; BUTALBITAL; CAFFEINE
https://dailymed.nlm.nih.gov/dailymed/search.cfm?labeltype=all&query=BUTALBITAL,+ASPIRIN+AND+CAFFEINE https://dailymed.nlm.nih.gov/dailymed/search.cfm?labeltype=all&query=BUTALBITAL,+ASPIRIN+AND+CAFFEINE

18. EU Community Register of Medicinal Products /source/EU Community Register of Medicinal Products

Acetylsalicylic acid
https://ec.europa.eu/health/documents/community-register/html/o208.htm https://ec.europa.eu/health/documents/community-register/html/o208.htm

19. NITE-CMC /source/NITE-CMC

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20. FDA Orange Book /source/FDA Orange Book

ACETAMINOPHEN; ASPIRIN; CAFFEINE
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21. FDA/SPL Indexing Data /source/FDA/SPL Indexing Data

R16CO5Y76E
https://www.fda.gov/ForIndustry/DataStandards/SubstanceRegistrationSystem-UniqueIngredientIdentifierUNII/ https://www.fda.gov/ForIndustry/DataStandards/SubstanceRegistrationSystem-UniqueIngredientIdentifierUNII/

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https://spectrabase.com/spectrum/67V44jWOjef https://spectrabase.com/spectrum/67V44jWOjef
https://spectrabase.com/spectrum/24gz6aGgI8L https://spectrabase.com/spectrum/24gz6aGgI8L
https://spectrabase.com/spectrum/5GijQrTxjbM https://spectrabase.com/spectrum/5GijQrTxjbM
https://spectrabase.com/spectrum/GzZpuLYomRj https://spectrabase.com/spectrum/GzZpuLYomRj
https://spectrabase.com/spectrum/2kIORiNyKde https://spectrabase.com/spectrum/2kIORiNyKde
https://spectrabase.com/spectrum/4IMNy32V8sS https://spectrabase.com/spectrum/4IMNy32V8sS
https://spectrabase.com/spectrum/F2hhMNoooB3 https://spectrabase.com/spectrum/F2hhMNoooB3
https://spectrabase.com/spectrum/ASYdzmZ0naO https://spectrabase.com/spectrum/ASYdzmZ0naO
https://spectrabase.com/spectrum/VEyU80HVqz https://spectrabase.com/spectrum/VEyU80HVqz
https://spectrabase.com/spectrum/21xlEEGjoVo https://spectrabase.com/spectrum/21xlEEGjoVo
https://spectrabase.com/spectrum/DmSZUBw2tf3 https://spectrabase.com/spectrum/DmSZUBw2tf3
https://spectrabase.com/spectrum/7ZdLb5TGbEA https://spectrabase.com/spectrum/7ZdLb5TGbEA
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acetylsalicylate
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Bufferin
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24. LiverTox /source/LiverTox

Aspirin
https://livertox.nlm.nih.gov/Aspirin.htm https://livertox.nlm.nih.gov/Aspirin.htm

25. MassBank of North America (MoNA) /source/MassBank of North America (MoNA)
BSYNRYMUTXBXSQ-UHFFFAOYSA-N
http://mona.fiehnlab.ucdavis.edu/spectra/browse?inchikey=BSYNRYMUTXBXSQ-UHFFFAOYSA-N http://mona.fiehnlab.ucdavis.edu/spectra/browse?inchikey=BSYNRYMUTXBXSQ-UHFFFAOYSA-N

26. NIST /source/NIST
Acetylsalicylic acid
http://www.nist.gov/srd/nist1a.cfm http://www.nist.gov/srd/nist1a.cfm

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The Protein Data Bank (PDB) is a crystallographic database for the three-dimensional structural data of large biological molecules, such as proteins and nucleic acids
http://www.rcsb.org/ligand/AIN http://www.rcsb.org/ligand/AIN

28. Springer Nature /source/Springer Nature
Literature references related to scientific contents from Springer Nature journals and books. Read more ... https://link.springer.com/

29. The Cambridge Structural Database /source/The Cambridge Structural Database
The Cambridge Structural Database provides access to 3D structures of molecules determined experimentally using diffraction techniques.
http://www.ccdc.cam.ac.uk/pages/Home.aspx http://www.ccdc.cam.ac.uk/pages/Home.aspx

30. Thieme Chemistry /source/Thieme Chemistry
Literature references related to scientific contents from Thieme journals and books. Read more: http://www.thieme-chemistry.com

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32. PubChem
Data deposited in or computed by PubChem
https://pubchem.ncbi.nlm.nih.gov https://pubchem.ncbi.nlm.nih.gov

33. MeSH /source/MeSH
Aspirin
https://www.ncbi.nlm.nih.gov/mesh/68001241 https://www.ncbi.nlm.nih.gov/mesh/68001241
MeSH Tree
http://www.nlm.nih.gov/mesh/meshhome.html http://www.nlm.nih.gov/mesh/meshhome.html
Antipyretics
https://www.ncbi.nlm.nih.gov/mesh/68058633 https://www.ncbi.nlm.nih.gov/mesh/68058633
Anti-Inflammatory Agents, Non-Steroidal
https://www.ncbi.nlm.nih.gov/mesh/68000894 https://www.ncbi.nlm.nih.gov/mesh/68000894
Fibrinolytic Agents
https://www.ncbi.nlm.nih.gov/mesh/68005343 https://www.ncbi.nlm.nih.gov/mesh/68005343
Cyclooxygenase Inhibitors
https://www.ncbi.nlm.nih.gov/mesh/68016861 https://www.ncbi.nlm.nih.gov/mesh/68016861
Platelet Aggregation Inhibitors
https://www.ncbi.nlm.nih.gov/mesh/68010975 https://www.ncbi.nlm.nih.gov/mesh/68010975

34. KEGG /source/KEGG
Therapeutic category of drugs in Japan
http://www.genome.jp/kegg-bin/get_htext?br08301.keg http://www.genome.jp/kegg-bin/get_htext?br08301.keg
Anatomical Therapeutic Chemical (ATC) classification
http://www.genome.jp/kegg-bin/get_htext?br08303.keg http://www.genome.jp/kegg-bin/get_htext?br08303.keg
Target-based classification of drugs
http://www.genome.jp/kegg-bin/get_htext?br08310.keg http://www.genome.jp/kegg-bin/get_htext?br08310.keg
Drugs listed in the Japanese Pharmacopoeia
http://www.genome.jp/kegg-bin/get_htext?br08311.keg http://www.genome.jp/kegg-bin/get_htext?br08311.keg
Risk category of Japanese OTC drugs
http://www.genome.jp/kegg-bin/get_htext?br08312.keg http://www.genome.jp/kegg-bin/get_htext?br08312.keg
Classification of Japanese OTC drugs
http://www.genome.jp/kegg-bin/get_htext?br08313.keg http://www.genome.jp/kegg-bin/get_htext?br08313.keg
Drug Classes
http://www.genome.jp/kegg-bin/get_htext?br08330.keg http://www.genome.jp/kegg-bin/get_htext?br08330.keg

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http://www.guidetopharmacology.org/ http://www.guidetopharmacology.org/

38. NCBI
LinkOut is a service that allows one to link directly from NCBI databases to a wide range of information and services beyond NCBI systems.
https://www.ncbi.nlm.nih.gov/projects/linkout https://www.ncbi.nlm.nih.gov/projects/linkout