SECTION-1: Identification of the substance / mixture and the company / undertaking

Catalogue Number	CS-ER-02340
Product Name	Acetylsalicylic acid for peak identification(Secondary Standards traceble to EP)
CAS No.	50-78-2
Category	Secondary Standards
Synonyms	2-(Acetyloxy)benzoic acid (ACI); Rhodine (7CI); Salicylic acid acetate (8CI)
Brand	Clearsynth Labs Ltd.
Identified uses	Laboratory Chemicals
Uses advised against	Not available
Company	Clearsynth Labs Ltd. Mumbai, India
Emergency Phone #	+91-22-245045900
REACH No.	Not available

SECTION 2: Hazards identification

Disclaimer: This is sample MSDS. Please email **sales@clearsynth.com** for more details.

2.1 Classification of the substance or mixture-Regulation (EC) No 1272/2008:

Skin irritation (Category 2)
Serious eye damage/eye irritation (Category 2)
Acute toxicity (Category 4)

2.2 Label Elements

Signal Word: Warning



Hazard Statement(s)

Code	Statement
H302	Harmful if swallowed.
H315	Causes skin irritation.

H319	Causes serious eye irritation.
H335	Not available
H334	Not available
H412	Not available
H360	Not available
H370	Not available
H372	Not available
H316	Not available
H371	Not available
H373	Not available

Precautionary Statement(s)

Code	Statement
P261	Avoid breathing dust/fume/gas/mist/vapours/spray.
P264	Wash hands thoroughly after handling.
P264+P265	Not available
P270	Not available
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P301+P317	Not available
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if presen
P319	Get medical help if you feel unwell.
P321	Specific treatment (see … on this label).
P330	Not available
P332+P317	If skin irritation occurs: Get medical help.
P337+P317	If eye irritation persists: Get medical help.
P362+P364	Take off contaminated clothing and wash it before reuse.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

P405	Store locked up.
P501	Dispose of contents/container in accordance with local/regional/national/international regulatio
P233	Not available
P260	Not available
P273	Not available
P284	Not available
P342+P316	Not available
P403	Not available
P203	Not available
P308+P316	Not available
P318	Not available

SECTION 3: Composition / information on ingredients

3.1 Substance

Component : Acetylsalicylic acid for peak identification(Secondary Standards traceble to EP)
CAS Number : 50-78-2
Molecular Formula : C9H8O4
Molecular Weight : 180.16
Parent Chemical : Acetylsalicylic acid
Synonyms : 2-(Acetyloxy)benzoic acid (ACI); Rhodine (7CI); Salicylic acid acetate (8CI)
Concentration : Not available

SECTION 4: First aid measures

SECTION 4: First-aid measures

4.1 Description of first aid measures
- General advice: Remove contaminated clothing and shoes. Seek medical attention if symptoms persist or are severe.
- Inhalation: Move person to fresh air. If breathing is difficult, seek medical attention.
- Skin contact: Wash with plenty of soap and water. Get medical attention if irritation develops.
- Eye contact: Rinse cautiously with water for several minutes. Remove contact lenses if present and easy to do. Continue rinsing. Seek medical attention if irritation persists.
- Ingestion: Rinse mouth. Do not induce vomiting unless directed by medical personnel. Seek medical attention.

4.2 Most important symptoms and effects, both acute and delayed
- Not available.

4.3 Indication of any immediate medical attention and special treatment needed
- Treat symptomatically. No data available.

SECTION 5: Firefighting measures

SECTION 5: Fire-fighting measures

5.1 Extinguishing media
- Suitable extinguishing media: Water spray, alcohol-resistant foam, dry chemical, carbon dioxide.
- Unsuitable extinguishing media: Not available.

5.2 Special hazards arising from the substance or mixture
- May form combustible dust concentrations in air.
- Hazardous combustion products: Not available.

5.3 Advice for firefighters
- Wear self-contained breathing apparatus (SCBA) and full protective gear.
- Use water spray to cool unopened containers.
- Avoid inhalation of combustion products.

SECTION 6: Accidental release measures

SECTION 6: Accidental release measures

6.1 Personal precautions, protective equipment and emergency procedures
- Avoid breathing dust. Avoid contact with skin and eyes.
- Provide adequate ventilation.
- Use appropriate personal protective equipment (see Section 8).

6.2 Environmental precautions
- Prevent further leakage or spillage if safe to do so.
- Avoid release to the environment. Prevent entry into drains, surface waters, or soil.

6.3 Methods and material for containment and cleaning up
- Avoid dust formation.
- Collect spilled material using methods that minimize dust generation (e.g., dampen with water if compatible).
- Place in a suitable, closed container for disposal.
- Clean contaminated area with water and detergent.

6.4 Reference to other sections
- See Section 8 for personal protective equipment and Section 13 for disposal considerations.

SECTION-7: Handling and storage

SECTION 7: Handling and storage

7.1 Precautions for safe handling
- Handle in accordance with good industrial hygiene and safety practice.
- Avoid formation and inhalation of dust.
- Avoid contact with skin, eyes, and clothing.
- Use with adequate ventilation.
- Wash hands thoroughly after handling.

7.2 Conditions for safe storage, including any incompatibilities

- Store in a tightly closed container in a cool, dry, well-ventilated place.
- Protect from moisture.
- Incompatible materials: Not available.

7.3 Specific end use(s)
- Laboratory/analytical standard. No further information available.

SECTION 8: Exposure controls / personal protection

SECTION 8: Exposure controls/personal protection

8.1 Control parameters
- Occupational exposure limits: Not available.

8.2 Exposure controls
- Engineering controls: Use local exhaust ventilation or general ventilation to minimize dust exposure.
- Personal protective equipment (PPE):
- Eye/face protection: Safety glasses with side shields or chemical splash goggles.
- Skin protection: Protective gloves (material not specified). Wear lab coat or protective clothing.
- Respiratory protection: If dust is generated and ventilation is inadequate, use an appropriate particulate respirator.
- Hygiene measures: Wash hands after handling. Do not eat, drink, or smoke when using this product.
- Environmental exposure controls: Avoid release to the environment.

SECTION 9: Physical and chemical properties

9.1 Information on basic physical and chemical properties

Test	Result
Appearance	No data available
IR spectrum	No data available
pH	No data available
Solubility	No data available

Property	Value
a) Physical State	No data available
b) Color	No data available
c) Odor	No data available
d) pH	No data available
e) Vapour Pressure	No data available
f) Viscosity	No data available

Property	Value
g) Initial Boiling Point and boiling range	No data available
h) Melting Point / Freezing Point	No data available
i) Auto Ignition Temperature	No data available
j) Flash Point	No data available
k) Explosion Limit, Lower	No data available
l) Explosion Limit, Upper	No data available
m) Decomposition Temperature	No data available
n) Loss on Drying	No data available
o) Relative Density	No data available
p) Solubility (in DMSO)	No data available
q) Oxidizing Properties	No data available

SECTION 10: Stability and reactivity

SECTION 10: Stability and reactivity

10.1 Reactivity
- No data available.

10.2 Chemical stability
- Stable under recommended storage conditions.

10.3 Possibility of hazardous reactions
- No data available.

10.4 Conditions to avoid
- Excessive heat. Dust generation.

10.5 Incompatible materials
- Not available.

10.6 Hazardous decomposition products
- Not available.

SECTION 11: Toxicological information

11.1 Information on toxicological effects
- Acute toxicity: Aspirin is an acute irritant to ... the skin and eyes. Direct contact with the eye is painful ... 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.

- Skin corrosion/irritation: No data available.
- Serious eye damage/eye irritation: No data available.
- Respiratory or skin sensitization: No data available.
- Germ cell mutagenicity: /PLANTS/ PHYTOTOXICITY: CHROMOSOMAL ABERRATIONS & ALTERATIONS IN ROOT CELLS OF BROAD BEAN PLANTS TREATED WITH ACETYLSALICYLIC ACID (1.0 GRAIN).
- Carcinogenicity: /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.
- Reproductive toxicity: /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. /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.
- STOT-single exposure: 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. /AQUATIC SPECIES/ Metabolic products are often more toxic than their pharmacological parent compounds. Therefore, the acute and chronic effects of the main metabolites--salicylic acid (SAL), gentisic acid (GEN), and o-hydroxyhippuric acid (HDP)--of acetylsalicylic acid (ASA), the active ingredient in Aspirin and many other pharmaceuticals, were assessed using standard (Daphnia magna) and autochthonous (Daphnia longispina) cladocerans. The sequence of decreasing levels of acute and chronic toxicity of ASA metabolites to daphnids was GEN > SAL > HDP. HDP did not present acute toxicity, but chronic exposures enabled the production of abnormal neonates and, in particular, egg abortion. Thus, reproduction was the end point most susceptible to HDP. On the other hand, SAL and GEN induced changes in the normal patterns of reproduction and growth of both species. In general, D. longispina was more sensitive than was D. magna, although the population growth of the autochthonous species was superior under SAL exposures than that of the standard test species. Although the concentrations that were determined to have a toxic effect were above the levels detected in aquatic environmental samples, exposure to low levels of pharmacologically active substances for a duration longer than the test period may induce changes in nontarget organisms.

- STOT-repeated exposure: 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. /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/

- Aspiration hazard: No data available.

Likely routes of exposure

- 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.

Symptoms related to the physical, chemical and toxicological characteristics
- 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.

SECTION 12: Ecological information

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12.1 Toxicity
- No data available.

12.2 Persistence and degradability
- No data available.

12.3 Bioaccumulative potential
- No data available.

12.4 Mobility in soil
- No data available.

12.5 Results of PBT and vPvB assessment
- Not available.

12.6 Endocrine disrupting properties
- Not available.

12.7 Other adverse effects
- No data available.

SECTION 13: Disposal considerations

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13.1 Waste treatment methods
- Dispose of contents/container in accordance with local/regional/national/international regulations.
- Do not discharge to drains.
- Contaminated packaging: Dispose of as unused product or according to local requirements.
- Waste codes: Not available.

SECTION 14: Transport information

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- UN number: Not available.
- UN proper shipping name: Not available.
- Transport hazard class(es): Not available.
- Packing group: Not available.
- Environmental hazards: Not available.
- Special precautions for user: Not available.
- Transport in bulk according to IMO instruments: Not available.

SECTION 15: Regulatory information

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15.1 Safety, health and environmental regulations/legislation specific for the substance or mixture
- Regulatory listings: Not available.

15.2 Chemical safety assessment
- Not available.

SECTION 16: Other information

SECTION 16: Other information

- Product name: Acetylsalicylic acid for peak identification (Secondary Standards traceble to EP)
- Catalog No.: CS-ER-02340
- CAS No.: 50-78-2
- Molecular weight: 180.16
- Synonyms: 2-(Acetyloxy)benzoic acid (ACI); Rhodine (7CI); Salicylic acid acetate (8CI)
- Supplier: Clearsynth Labs Ltd., Mumbai, India
- Emergency phone: +91-22-245045900

Disclaimer
- The information provided is based on available product information and is intended for guidance in safe handling. It does not constitute a guarantee of specific properties. Users are responsible for compliance with applicable regulations and for determining suitability for a particular purpose.

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