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

Catalogue Number	CS-SS-24003
Product Name	Paracetamol(Acetaminophen)
CAS No.	103-90-2
Category	Secondary Standards
Synonyms	4-(N-Acetylamino) phenol); Acetaminophen; Abensanil; Cetamol
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.
H412	Not available
H401	Not available
H411	Toxic to aquatic life with long lasting effects.
H341	Not available
H370	Not available
H371	Not available
H372	Not available
H373	Not available
H335	Not available

Precautionary Statement(s)

Code	Statement
P264	Wash hands thoroughly after handling.
P264+P265	Not available
P270	Not available
P273	Not available
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.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if presen
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.
P501	Dispose of contents/container in accordance with local/regional/national/international regulatio
P391	Not available
P203	Not available
P260	Not available

P308+P316	Not available
P318	Not available
P319	Get medical help if you feel unwell.
P405	Store locked up.
P261	Avoid breathing dust/fume/gas/mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

SECTION 3: Composition / information on ingredients

3.1 Substance

Component : Paracetamol(Acetaminophen)
CAS Number : 103-90-2
Molecular Formula : C■H■NO■
Molecular Weight : 151.16
Parent Chemical : Paracetamol
Synonyms : 4-(N-Acetylamino) phenol); Acetaminophen; Abensanil; Cetamol
Concentration : Not available

SECTION 4: First aid measures

SECTION 4: First-aid measures

4.1 Description of first aid measures
General advice: Seek medical attention if symptoms persist or if large exposure is suspected. Show this SDS to the physician.
Inhalation: Move person to fresh air. If breathing is difficult, seek medical attention.
Skin contact: Wash with soap and water. Remove contaminated clothing and wash before reuse. 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. Thermal decomposition may produce irritating and/or toxic fumes.

5.3 Advice for firefighters
Wear self-contained breathing apparatus (SCBA) and full protective gear. Use water spray to cool unopened containers. Avoid generating dust during fire-fighting operations.

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 eyes and skin. Provide adequate ventilation. Use appropriate personal protective equipment.

6.2 Environmental precautions
Avoid release to the environment. Prevent entry into drains, surface water, or soil.

6.3 Methods and material for containment and cleaning up
Avoid raising dust. Sweep up or vacuum using equipment fitted with HEPA filtration where possible. Place in a suitable, closed container for disposal. Clean spill area with water after material pickup if appropriate.

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 of dust and aerosols. Avoid breathing dust. Avoid contact with eyes, skin, and clothing. Use with adequate ventilation.

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. Keep away from incompatible materials.
Incompatible materials: Not available.

7.3 Specific end use(s)
Secondary standard / laboratory use. Not available for other specific uses.

SECTION 8: Exposure controls / personal protection

SECTION 8: Exposure controls/personal protection

8.1 Control parameters
Occupational exposure limits: Not available.
Biological limit values: Not available.

8.2 Exposure controls
Engineering controls: Provide appropriate exhaust ventilation to control dust levels.
Personal protective equipment (PPE):
- Eye/face protection: Safety glasses with side shields or chemical splash goggles.
- Skin protection: Protective gloves. Protective clothing as appropriate.
- Respiratory protection: If ventilation is inadequate or dust is generated, use a suitable particulate respirator in accordance with applicable regulations.
- Hygiene measures: Wash hands after handling. Do not eat, drink, or smoke when using this product.

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
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

Property	Value
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. Moisture (if applicable).

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: IDENTIFICATION AND USE: Acetaminophen is an odorless compound with a slightly bitter taste. It is a common analgesic and antipyretic agent used for the relief of fever as well as aches and pains associated with many conditions. HUMAN EXPOSURE AND TOXICITY: Nausea, vomiting, and abdominal pain usually occur within 2-3 hours after ingestion of toxic doses of the drug. In severe poisoning, CNS stimulation, excitement, and delirium may occur initially. This may be followed by CNS depression, stupor, hypothermia, marked prostration, rapid shallow breathing, rapid weak irregular pulse, low blood pressure, and circulatory failure. When an individual has ingested a toxic dose of acetaminophen, the individual should be hospitalized for several days of observation, even if there are no apparent ill effects, because maximum liver damage and/or cardiotoxic effects usually do not become apparent until 2-4 days after ingestion of the drug. Other symptoms of acute poisoning include cerebral edema and nonspecific myocardial depression. Vascular collapse results from the relative hypoxia and from a central depressant action that occurs only with massive doses. Shock may develop if vasodilation is marked. Fatal seizures may occur. Coma usually precedes death, which may occur suddenly or may be delayed for several days. Biopsy of the liver reveals centralobular necrosis with sparing of the periportal area. There have been reports of acute myocardial necrosis and pericarditis in individuals with acetaminophen poisoning. Hypoglycemia, which can progress to coma have been reported in patients ingesting toxic doses of acetaminophen. Low prothrombin levels and thrombocytopenia have been reported in patients with acetaminophen poisoning. Skin reactions of an

erythematous or urticarial nature which may be accompanied by fever and oral mucosal lesions also have been reported. For use anytime during pregnancy, 781 exposures were recorded, and possible associations with congenital dislocation of the hip (eight cases) and clubfoot (six cases) were found. There is inadequate evidence in humans for the carcinogenicity of acetaminophen. ANIMAL TOXICITY STUDIES: There is inadequate evidence in experimental animals for the carcinogenicity of acetaminophen. In rats fasted 24 hours and given a single dose of acetaminophen (2 g/kg) by gavage, liver necrosis around the central vein was noted at 9-12 hours and was much more extensive at 24 hours after treatment. In mice after dietary exposure to acetaminophen up to 6400 mg/kg daily for 13 weeks hepatotoxicity, organ weight changes and deaths were observed. Cats are particularly susceptible to acetaminophen intoxication, developing more diffuse liver changes, while hepatic centrilobular lesions found in dogs. High doses of acetaminophen caused testicular atrophy and delay in spermiogenesis in mice. Furthermore, reductions in the fertility and neonatal survival in mice were seen in the F0 generation and decreases in F1 pup weights were found at acetaminophen dose 1430 mg/kg. Acetaminophen was not mutagenic in Salmonella typhimurium assay with or without metabolic activation in six strains: TA1535, TA1537, TA1538, TA100, TA97 and TA98. In vitro and animal data indicate that small quantities of acetaminophen are metabolized by a cytochrome P-450 microsomal enzyme to a reactive intermediate metabolite (N-acetyl-p-benzoquinoneimine, N-acetylimidoquinone, NAPQI) which is further metabolized via conjugation with glutathione and ultimately excreted in urine as a mercapturic acid. It has been suggested that this intermediate metabolite is responsible for acetaminophen-induced liver necrosis in cases of overdose. Excipients found in liquid formulations of acetaminophen may decrease its liver toxicity. ECOTOXICITY STUDIES: Daphnia magna was the most susceptible among the test organisms to the environmental effects of acetaminophen. Acetaminophen has recently been identified as a promising snake toxicant to reduce brown tree snake populations on Guam, while posing only the minimal risks to non-target rodents, cats, pigs and birds. Each year, approximately 500 fatalities and 50,000 emergency department admissions in the United States are linked to acetaminophen. In 2021, US poison control centers recorded over 80,000 cases. Acetaminophen is the most prevalent drug-related cause of acute liver failure, with hepatic injury occurring as a consequence of the drug's metabolism properties. After reaching therapeutic concentrations of oral acetaminophen, 60% to 90% of the drug undergoes metabolism in the liver, forming glucuronic acid- and sulfate-conjugate metabolites. A smaller fraction, approximately 5% to 15%, undergoes metabolism via the cytochrome P450 system (CYP450)—metabolism primarily through CYP2E1 results in the formation of the toxic intermediate NAPQI. Under normal circumstances, NAPQI is neutralized by glutathione to form nontoxic metabolites. However, in the case of excessive doses of acetaminophen, the normal phase II drug metabolism pathways become depleted. The CYP450 pathway metabolizes a more significant portion of the acetaminophen, leading to elevated concentrations of NAPQI formation, and the limited glutathione stores can deplete. When there is a shortage of glutathione, NAPQI concentrations increase, and, as a reactive intermediate, it can react with essential cellular macromolecules such as proteins, lipids, and nucleic acids. This interaction can result in centrilobular (zone 3) hepatic injury and hepatocellular death, along with the potential for nephrotoxicity. The only approved antidote for acetaminophen overdose and toxicity is N-acetylcysteine (NAC). NAC acts as a precursor to glutathione synthesis, aiding in restoring intracellular glutathione stores to neutralize the NAPQI compound, directly inactivating NAPQI. NAC can be administered orally or via the IV route. The IV administration of NAC is typically preferred because vomiting is common with acetaminophen overdose. NAC administration follows a 20-hour IV or 72-hour oral protocol, and clinicians must monitor aspartate aminotransferase/alanine aminotransferase (AST/ALT) levels during treatment. Notably, the majority of patients do not exhibit symptoms in the initial hours after ingesting toxic doses of acetaminophen. During this early period, symptoms may be limited to abdominal pain and nausea, persisting for the first 12 to 24 hours. Although these symptoms may alleviate between 24 and 72 hours, AST/ALT concentrations may remain abnormal. Patients presenting more than 24 hours after ingesting toxic doses of acetaminophen may manifest symptoms including nausea, vomiting, jaundice, abdominal pain, and hypotension. The management of these patients may involve interventions such as airway management,

IV fluids, vasopressors, and addressing symptoms such as cerebral edema as they arise. A recent consensus statement published by America's Poison Centers, American Academy of Clinical Toxicology, American College of Medical Toxicology, and Canadian Association of Poison Control Centers addresses acetaminophen toxicity. NAC is administered through oral or IV routes, with the initial dose administered promptly upon identifying the need for treatment. The panel recommends a regimen providing a minimum of 300 mg/kg, either orally or IV, within the initial 20 to 24 hours of treatment. Nevertheless, the comparative effectiveness of various regimens still requires evaluation. The guidelines emphasize the importance of continuous assessment and caution against prematurely discontinuing treatment. Notably, a common clinical error involves administering NAC for 20 or 21 hours and then discontinuing without reassessing the patient. The panel chose to refine the Rumack-Matthew nomogram by retaining only the lines indicating clinical action. In this refined approach, the blood concentration of APAP is directly plotted on the nomogram, and NAC is administered to patients whose concentration exceeds the treatment line. Stopping criteria for NAC include an acetaminophen concentration below 10 μg/dL, an INR level below 2, normal levels of AST/ALT, or a decrease in AST/ALT by 25% to 50%, provided the patient is clinically stable. High-risk ingestion involves the consumption of at least 30 g of acetaminophen or an acetaminophen concentration surpassing the high-risk line on the nomogram. These cases are managed similarly to other acetaminophen overdoses, with consideration for the extended administration of activated charcoal, especially if the ingestion occurred more than 4 hours prior due to prolonged absorption. In addition, consultation with a clinical toxicologist may be required for an increased NAC dosage. In managing repeated supratherapeutic ingestion in 24 hours, unlike acute ingestion cases, treatment is based on signs and symptoms. If the acetaminophen concentration exceeds 20 μg/mL or AST levels or ALT are abnormal, NAC should be administered until the established stopping criteria are met. Extended-release acetaminophen products, intended for 8-hour use in the United States or Canada, are managed similarly to other acetaminophen products. Activated charcoal may continue to be effective for longer than 4 hours after ingestion, especially when evidence indicates ongoing absorption, such as an increasing acetaminophen concentration. NAC is required if the acetaminophen concentration from samples drawn 4 to 24 hours after ingestion surpasses the nomogram treatment line. In cases where the concentration from samples drawn 4 to 12 hours after ingestion falls below the treatment line but remains above 10 μg/mL, a follow-up measurement should be taken 4 to 6 hours after the initial assessment. In simultaneous ingestion with anticholinergic or opioid agonists, the concern is the potential for delays or prolongation of acetaminophen absorption. The management approach aligns with that of other acetaminophen products. If the initial acetaminophen concentration measured 4 to 24 hours after ingestion is 10 μg/mL or lower, further measurements are unnecessary, and N-acetylcysteine (NAC) treatment is not required. Conversely, if any concentration exceeds the treatment line, NAC is indicated. If the acetaminophen concentration measured within the same time frame falls between 10 μg/mL and the treatment line on the revised nomogram, and clinical signs indicating anticholinergic or opioid toxicity are present, a reevaluation should be scheduled 4 to 6 hours following the initial measurement. Notably, the dosing and duration of NAC treatment strictly follow the established standard protocol for acetaminophen ingestions. The panel recommends hemodialysis with NAC in massive acetaminophen toxicity with a concentration exceeding 900 μg/mL, accompanied by acidosis or altered consciousness.

- Skin corrosion/irritation: No data available.
- Serious eye damage/eye irritation: /LABORATORY ANIMALS: Acute Exposure/ Acetaminophen ... an analgesic and antipyretic, is without known ocular side effects, with the exception that in genetically very special mice it can cause irreversible opacification of the anterior portion of the lens when a large dose is given ip.
- Respiratory or skin sensitization: No data available.
- Germ cell mutagenicity: IDENTIFICATION AND USE: Acetaminophen is an odorless compound with a slightly bitter taste. It is a common analgesic and antipyretic agent used for the relief of fever as well as aches and pains associated with many conditions. HUMAN EXPOSURE AND TOXICITY: Nausea, vomiting, and abdominal pain usually occur within 2-3 hours after ingestion of toxic doses of the drug. In severe poisoning, CNS stimulation,

excitement, and delirium may occur initially. This may be followed by CNS depression, stupor, hypothermia, marked prostration, rapid shallow breathing, rapid weak irregular pulse, low blood pressure, and circulatory failure. When an individual has ingested a toxic dose of acetaminophen, the individual should be hospitalized for several days of observation, even if there are no apparent ill effects, because maximum liver damage and/or cardiotoxic effects usually do not become apparent until 2-4 days after ingestion of the drug. Other symptoms of acute poisoning include cerebral edema and nonspecific myocardial depression. Vascular collapse results from the relative hypoxia and from a central depressant action that occurs only with massive doses. Shock may develop if vasodilation is marked. Fatal seizures may occur. Coma usually precedes death, which may occur suddenly or may be delayed for several days. Biopsy of the liver reveals centralobular necrosis with sparing of the periportal area. There have been reports of acute myocardial necrosis and pericarditis in individuals with acetaminophen poisoning. Hypoglycemia, which can progress to coma have been reported in patients ingesting toxic doses of acetaminophen. Low prothrombin levels and thrombocytopenia have been reported in patients with acetaminophen poisoning. Skin reactions of an erythematous or urticarial nature which may be accompanied by fever and oral mucosal lesions also have been reported. For use anytime during pregnancy, 781 exposures were recorded, and possible associations with congenital dislocation of the hip (eight cases) and clubfoot (six cases) were found. There is inadequate evidence in humans for the carcinogenicity of acetaminophen. ANIMAL TOXICITY STUDIES: There is inadequate evidence in experimental animals for the carcinogenicity of acetaminophen. In rats fasted 24 hours and given a single dose of acetaminophen (2 g/kg) by gavage, liver necrosis around the central vein was noted at 9-12 hours and was much more extensive at 24 hours after treatment. In mice after dietary exposure to acetaminophen up to 6400 mg/kg daily for 13 weeks hepatotoxicity, organ weight changes and deaths were observed. Cats are particularly susceptible to acetaminophen intoxication, developing more diffuse liver changes, while hepatic centrilobular lesions found in dogs. High doses of acetaminophen caused testicular atrophy and delay in spermiogenesis in mice. Furthermore, reductions in the fertility and neonatal survival in mice were seen in the F0 generation and decreases in F1 pup weights were found at acetaminophen dose 1430 mg/kg. Acetaminophen was not mutagenic in Salmonella typhimurium assay with or without metabolic activation in six strains: TA1535, TA1537, TA1538, TA100, TA97 and TA98. In vitro and animal data indicate that small quantities of acetaminophen are metabolized by a cytochrome P-450 microsomal enzyme to a reactive intermediate metabolite (N-acetyl-p-benzoquinoneimine, N-acetylimidoquinone, NAPQI) which is further metabolized via conjugation with glutathione and ultimately excreted in urine as a mercapturic acid. It has been suggested that this intermediate metabolite is responsible for acetaminophen-induced liver necrosis in cases of overdose. Excipients found in liquid formulations of acetaminophen may decrease its liver toxicity. ECOTOXICITY STUDIES: Daphnia magna was the most susceptible among the test organisms to the environmental effects of acetaminophen. Acetaminophen has recently been identified as a promising snake toxicant to reduce brown tree snake populations on Guam, while posing only the minimal risks to non-target rodents, cats, pigs and birds.

- Carcinogenicity: IDENTIFICATION AND USE: Acetaminophen is an odorless compound with a slightly bitter taste. It is a common analgesic and antipyretic agent used for the relief of fever as well as aches and pains associated with many conditions. HUMAN EXPOSURE AND TOXICITY: Nausea, vomiting, and abdominal pain usually occur within 2-3 hours after ingestion of toxic doses of the drug. In severe poisoning, CNS stimulation, excitement, and delirium may occur initially. This may be followed by CNS depression, stupor, hypothermia, marked prostration, rapid shallow breathing, rapid weak irregular pulse, low blood pressure, and circulatory failure. When an individual has ingested a toxic dose of acetaminophen, the individual should be hospitalized for several days of observation, even if there are no apparent ill effects, because maximum liver damage and/or cardiotoxic effects usually do not become apparent until 2-4 days after ingestion of the drug. Other symptoms of acute poisoning include cerebral edema and nonspecific myocardial depression. Vascular collapse results from the relative hypoxia and from a central depressant action that occurs only with massive doses. Shock may develop if vasodilation is marked. Fatal seizures may occur. Coma usually precedes death, which may occur suddenly or may be delayed for several days. Biopsy of

the liver reveals centralobular necrosis with sparing of the periportal area. There have been reports of acute myocardial necrosis and pericarditis in individuals with acetaminophen poisoning. Hypoglycemia, which can progress to coma have been reported in patients ingesting toxic doses of acetaminophen. Low prothrombin levels and thrombocytopenia have been reported in patients with acetaminophen poisoning. Skin reactions of an erythematous or urticarial nature which may be accompanied by fever and oral mucosal lesions also have been reported. For use anytime during pregnancy, 781 exposures were recorded, and possible associations with congenital dislocation of the hip (eight cases) and clubfoot (six cases) were found. There is inadequate evidence in humans for the carcinogenicity of acetaminophen. ANIMAL TOXICITY STUDIES: There is inadequate evidence in experimental animals for the carcinogenicity of acetaminophen. In rats fasted 24 hours and given a single dose of acetaminophen (2 g/kg) by gavage, liver necrosis around the central vein was noted at 9-12 hours and was much more extensive at 24 hours after treatment. In mice after dietary exposure to acetaminophen up to 6400 mg/kg daily for 13 weeks hepatotoxicity, organ weight changes and deaths were observed. Cats are particularly susceptible to acetaminophen intoxication, developing more diffuse liver changes, while hepatic centrilobular lesions found in dogs. High doses of acetaminophen caused testicular atrophy and delay in spermiogenesis in mice. Furthermore, reductions in the fertility and neonatal survival in mice were seen in the F0 generation and decreases in F1 pup weights were found at acetaminophen dose 1430 mg/kg. Acetaminophen was not mutagenic in Salmonella typhimurium assay with or without metabolic activation in six strains: TA1535, TA1537, TA1538, TA100, TA97 and TA98. In vitro and animal data indicate that small quantities of acetaminophen are metabolized by a cytochrome P-450 microsomal enzyme to a reactive intermediate metabolite (N-acetyl-p-benzoquinoneimine, N-acetylimidoquinone, NAPQI) which is further metabolized via conjugation with glutathione and ultimately excreted in urine as a mercapturic acid. It has been suggested that this intermediate metabolite is responsible for acetaminophen-induced liver necrosis in cases of overdose. Excipients found in liquid formulations of acetaminophen may decrease its liver toxicity. ECOTOXICITY STUDIES: Daphnia magna was the most susceptible among the test organisms to the environmental effects of acetaminophen. Acetaminophen has recently been identified as a promising snake toxicant to reduce brown tree snake populations on Guam, while posing only the minimal risks to non-target rodents, cats, pigs and birds. Evaluation: There is inadequate evidence in humans for the carcinogenicity of paracetamol. There is inadequate evidence in experimental animals for the carcinogenicity of paracetamol. Overall evaluation: Paracetamol is not classifiable as to its carcinogenicity to humans (Group 3).
- Reproductive toxicity: IDENTIFICATION AND USE: Acetaminophen is an odorless compound with a slightly bitter taste. It is a common analgesic and antipyretic agent used for the relief of fever as well as aches and pains associated with many conditions. HUMAN EXPOSURE AND TOXICITY: Nausea, vomiting, and abdominal pain usually occur within 2-3 hours after ingestion of toxic doses of the drug. In severe poisoning, CNS stimulation, excitement, and delirium may occur initially. This may be followed by CNS depression, stupor, hypothermia, marked prostration, rapid shallow breathing, rapid weak irregular pulse, low blood pressure, and circulatory failure. When an individual has ingested a toxic dose of acetaminophen, the individual should be hospitalized for several days of observation, even if there are no apparent ill effects, because maximum liver damage and/or cardiotoxic effects usually do not become apparent until 2-4 days after ingestion of the drug. Other symptoms of acute poisoning include cerebral edema and nonspecific myocardial depression. Vascular collapse results from the relative hypoxia and from a central depressant action that occurs only with massive doses. Shock may develop if vasodilation is marked. Fatal seizures may occur. Coma usually precedes death, which may occur suddenly or may be delayed for several days. Biopsy of the liver reveals centralobular necrosis with sparing of the periportal area. There have been reports of acute myocardial necrosis and pericarditis in individuals with acetaminophen poisoning. Hypoglycemia, which can progress to coma have been reported in patients ingesting toxic doses of acetaminophen. Low prothrombin levels and thrombocytopenia have been reported in patients with acetaminophen poisoning. Skin reactions of an erythematous or urticarial nature which may be accompanied by fever and oral mucosal lesions also have been reported. For use anytime during pregnancy, 781 exposures were recorded, and possible associations

with congenital dislocation of the hip (eight cases) and clubfoot (six cases) were found. There is inadequate evidence in humans for the carcinogenicity of acetaminophen. ANIMAL TOXICITY STUDIES: There is inadequate evidence in experimental animals for the carcinogenicity of acetaminophen. In rats fasted 24 hours and given a single dose of acetaminophen (2 g/kg) by gavage, liver necrosis around the central vein was noted at 9-12 hours and was much more extensive at 24 hours after treatment. In mice after dietary exposure to acetaminophen up to 6400 mg/kg daily for 13 weeks hepatotoxicity, organ weight changes and deaths were observed. Cats are particularly susceptible to acetaminophen intoxication, developing more diffuse liver changes, while hepatic centrilobular lesions found in dogs. High doses of acetaminophen caused testicular atrophy and delay in spermiogenesis in mice. Furthermore, reductions in the fertility and neonatal survival in mice were seen in the F0 generation and decreases in F1 pup weights were found at acetaminophen dose 1430 mg/kg. Acetaminophen was not mutagenic in Salmonella typhimurium assay with or without metabolic activation in six strains: TA1535, TA1537, TA1538, TA100, TA97 and TA98. In vitro and animal data indicate that small quantities of acetaminophen are metabolized by a cytochrome P-450 microsomal enzyme to a reactive intermediate metabolite (N-acetyl-p-benzoquinoneimine, N-acetylimidoquinone, NAPQI) which is further metabolized via conjugation with glutathione and ultimately excreted in urine as a mercapturic acid. It has been suggested that this intermediate metabolite is responsible for acetaminophen-induced liver necrosis in cases of overdose. Excipients found in liquid formulations of acetaminophen may decrease its liver toxicity. ECOTOXICITY STUDIES: Daphnia magna was the most susceptible among the test organisms to the environmental effects of acetaminophen. Acetaminophen has recently been identified as a promising snake toxicant to reduce brown tree snake populations on Guam, while posing only the minimal risks to non-target rodents, cats, pigs and birds. /HUMAN EXPOSURE STUDIES/ The Rocky Mountain Poison and Drug Center reported the results of a nationwide study on acetaminophen overdose during pregnancy involving 113 women. Of the 60 cases that had appropriate laboratory and pregnancy outcome data, 19 occurred in the 1st trimester, 22 during the 2nd trimester, and 19 during the 3rd trimester. In those cases with a potentially toxic serum level of acetaminophen, early treatment with N-acetylcysteine was statistically associated with an improved pregnancy outcome by lessening the incidence of spontaneous abortion and fetal death. Only one congenital anomaly was observed in the series and that involved a 3rd trimester overdose with nontoxic maternal acetaminophen serum levels.

- STOT-single exposure: /AQUATIC SPECIES/ The increasing presence of pharmaceutical drugs in nature is cause of concern due to the occurrence of oxidative stress in non-target species. Acetaminophen is widely used in human medicine as an analgesic and antipyretic drug, and it is one of the most sold non-prescription drugs. The present study aimed to assess the toxic effects of acetaminophen (APAP) in Oncorhynchus mykiss following acute and chronic exposures in realistic levels. In order to evaluate the APAP effects in the rainbow trout, gills and liver were analyzed with biochemical biomarkers, such as catalase (CAT), total and selenium-dependent glutathione peroxidase (GPx), glutathione reductase (GRed) and glutathione-S-transferases (GSTs) activity and also lipid peroxidation levels (TBARS). The results obtained in all tests indicate that a significant response of oxidative stress was established, along with the increase of APAP concentrations. The establishment of an oxidative stress scenario occurred with the involvement of all tested biomarkers, sustaining a generalized set of pro-oxidative effects elicited by APAP. Additionally, the occurrence of oxidative damage strongly suggests the impairment of the antioxidant defense mechanism of O. mykiss. It is important to note that the occurrence of oxidative deleterious effects and peroxidative damages occurred for concentrations similar to those already reported for several freshwater ecosystems. The importance of these assumptions is further discussed under the scope of ecological relevance of the assessment of effects caused by pharmaceuticals in non-target organisms.
- STOT-repeated exposure: Paracetamol toxicity is one of the most common causes of poisoning worldwide. The toxic effects of acetaminophen are due to a minor alkylating metabolite (N-acetyl-p-benzo-quinone imine – also known as NAPQI), not acetaminophen itself nor any of the other major metabolites. Cytochromes P450 2E1 and 3A4 convert approximately 5% of paracetamol to NAPQI. This toxic metabolite reacts with sulfhydryl groups on

proteins and with glutathione (GSH). NAPQI depletes the liver's natural antioxidant glutathione and directly damages cells in the liver, leading to liver failure. In animal studies, hepatic glutathione must be depleted to less than 70% of normal levels before hepatotoxicity occurs. More specifically, oxidation by NAPQI of GSH to GSSG (oxidized glutathione) and the reduction of GSSG back to GSH by the NADPH-dependent glutathione reductase appear to be responsible for the rapid oxidation of NADPH that occurs in hepatocytes incubated with NAPQI. Risk factors for toxicity include excessive chronic alcohol intake, fasting or anorexia nervosa, and the use of certain drugs such as isoniazid. At usual doses, paracetamol is quickly detoxified by combining irreversibly with the sulfhydryl group of glutathione to produce a non-toxic conjugate that is eventually excreted by the kidneys. The toxic dose of paracetamol is highly variable. Chronic therapy with acetaminophen in doses of 4 grams daily has been found to lead to transient elevations in serum aminotransferase levels in a proportion of subjects, generally starting after 3 to 7 days, and with peak values rising above 3-fold elevated in 39% of persons. These elevations are generally asymptomatic and resolve rapidly with stopping therapy or reducing the dosage, and in some instances resolve even with continuation at full dose (Case 1). While acetaminophen has few side effects when used in therapeutic doses, recent reports suggest that its standard use can result in severe hypersensitivity reactions including Stevens Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN). Both of these syndromes can be life-threatening and both may be accompanied by evidence of liver injury. However, the hepatic involvement is usually mild and marked only by asymptomatic mild-to-moderate elevations in serum aminotransferase levels. The best known form of hepatoxicity from acetaminophen is an acute, serious hepatocellular injury as a result of intentional or unintentional overdose. The injury is due to a direct, toxic effect of the high doses of acetaminophen. Acetaminophen hepatotoxicity most commonly arises after a suicide attempt using more than 7.5 grams (generally more than 15 grams) as a single overdose (Case 2). Hepatic injury generally starts 24 to 72 hours after the ingestion with marked elevations in serum ALT and AST (often to above 2000 U/L), followed at 48 to 96 hours by clinical symptoms: jaundice, confusion, hepatic failure and in some instances death. Evidence of renal insufficiency is also common. Serum aminotransferase levels fall promptly and recovery is rapid if the injury is not too severe. Similar injury can occur with high therapeutic or supratherapeutic doses of acetaminophen given over several days for treatment of pain and not as a purposeful suicidal overdose (Case 3). This form of acetaminophen hepatotoxicity is referred to as accidental or unintentional overdose, and usually occurs in patients who have been fasting, or are critically ill with a concurrent illness, alcoholism or malnutrition, or have preexisting chronic liver disease. Some cases of unintentional overdose occur in patients taking acetaminophen in combinations with controlled substances (oxycodone, codeine), who take more than recommended amounts over several days in attempts to control pain or withdrawal symptoms. Instances of unintentional overdose in children are often due to errors in calculating the correct dosage or use of adult sized tablets instead of child or infant formulations. Because acetaminophen is present in many products, both by prescription and over-the-counter, another problem occurs when a patient ingests full or high doses of several products unaware that several contain acetaminophen. Likelihood score: A[HD] (well established cause of liver injury, but severe cases occur only with high doses).
- Aspiration hazard: No data available.

Likely routes of exposure
- IDENTIFICATION AND USE: Acetaminophen is an odorless compound with a slightly bitter taste. It is a common analgesic and antipyretic agent used for the relief of fever as well as aches and pains associated with many conditions. HUMAN EXPOSURE AND TOXICITY: Nausea, vomiting, and abdominal pain usually occur within 2-3 hours after ingestion of toxic doses of the drug. In severe poisoning, CNS stimulation, excitement, and delirium may occur initially. This may be followed by CNS depression, stupor, hypothermia, marked prostration, rapid shallow breathing, rapid weak irregular pulse, low blood pressure, and circulatory failure. When an individual has ingested a toxic dose of acetaminophen, the individual should be hospitalized for several days of observation, even if there are no apparent ill effects, because maximum liver damage and/or cardiotoxic effects usually do not become apparent until 2-4 days after ingestion of the drug. Other symptoms of acute poisoning include cerebral edema and

nonspecific myocardial depression. Vascular collapse results from the relative hypoxia and from a central depressant action that occurs only with massive doses. Shock may develop if vasodilation is marked. Fatal seizures may occur. Coma usually precedes death, which may occur suddenly or may be delayed for several days. Biopsy of the liver reveals centralobular necrosis with sparing of the periportal area. There have been reports of acute myocardial necrosis and pericarditis in individuals with acetaminophen poisoning. Hypoglycemia, which can progress to coma have been reported in patients ingesting toxic doses of acetaminophen. Low prothrombin levels and thrombocytopenia have been reported in patients with acetaminophen poisoning. Skin reactions of an erythematous or urticarial nature which may be accompanied by fever and oral mucosal lesions also have been reported. For use anytime during pregnancy, 781 exposures were recorded, and possible associations with congenital dislocation of the hip (eight cases) and clubfoot (six cases) were found. There is inadequate evidence in humans for the carcinogenicity of acetaminophen. ANIMAL TOXICITY STUDIES: There is inadequate evidence in experimental animals for the carcinogenicity of acetaminophen. In rats fasted 24 hours and given a single dose of acetaminophen (2 g/kg) by gavage, liver necrosis around the central vein was noted at 9-12 hours and was much more extensive at 24 hours after treatment. In mice after dietary exposure to acetaminophen up to 6400 mg/kg daily for 13 weeks hepatotoxicity, organ weight changes and deaths were observed. Cats are particularly susceptible to acetaminophen intoxication, developing more diffuse liver changes, while hepatic centrilobular lesions found in dogs. High doses of acetaminophen caused testicular atrophy and delay in spermiogenesis in mice. Furthermore, reductions in the fertility and neonatal survival in mice were seen in the F0 generation and decreases in F1 pup weights were found at acetaminophen dose 1430 mg/kg. Acetaminophen was not mutagenic in Salmonella typhimurium assay with or without metabolic activation in six strains: TA1535, TA1537, TA1538, TA100, TA97 and TA98. In vitro and animal data indicate that small quantities of acetaminophen are metabolized by a cytochrome P-450 microsomal enzyme to a reactive intermediate metabolite (N-acetyl-p-benzoquinoneimine, N-acetylimidoquinone, NAPQI) which is further metabolized via conjugation with glutathione and ultimately excreted in urine as a mercapturic acid. It has been suggested that this intermediate metabolite is responsible for acetaminophen-induced liver necrosis in cases of overdose. Excipients found in liquid formulations of acetaminophen may decrease its liver toxicity. ECOTOXICITY STUDIES: Daphnia magna was the most susceptible among the test organisms to the environmental effects of acetaminophen. Acetaminophen has recently been identified as a promising snake toxicant to reduce brown tree snake populations on Guam, while posing only the minimal risks to non-target rodents, cats, pigs and birds.

Symptoms related to the physical, chemical and toxicological characteristics
- IDENTIFICATION AND USE: Acetaminophen is an odorless compound with a slightly bitter taste. It is a common analgesic and antipyretic agent used for the relief of fever as well as aches and pains associated with many conditions. HUMAN EXPOSURE AND TOXICITY: Nausea, vomiting, and abdominal pain usually occur within 2-3 hours after ingestion of toxic doses of the drug. In severe poisoning, CNS stimulation, excitement, and delirium may occur initially. This may be followed by CNS depression, stupor, hypothermia, marked prostration, rapid shallow breathing, rapid weak irregular pulse, low blood pressure, and circulatory failure. When an individual has ingested a toxic dose of acetaminophen, the individual should be hospitalized for several days of observation, even if there are no apparent ill effects, because maximum liver damage and/or cardiotoxic effects usually do not become apparent until 2-4 days after ingestion of the drug. Other symptoms of acute poisoning include cerebral edema and nonspecific myocardial depression. Vascular collapse results from the relative hypoxia and from a central depressant action that occurs only with massive doses. Shock may develop if vasodilation is marked. Fatal seizures may occur. Coma usually precedes death, which may occur suddenly or may be delayed for several days. Biopsy of the liver reveals centralobular necrosis with sparing of the periportal area. There have been reports of acute myocardial necrosis and pericarditis in individuals with acetaminophen poisoning. Hypoglycemia, which can progress to coma have been reported in patients ingesting toxic doses of acetaminophen. Low prothrombin levels and thrombocytopenia have been reported in patients with acetaminophen poisoning. Skin reactions of an

erythematous or urticarial nature which may be accompanied by fever and oral mucosal lesions also have been reported. For use anytime during pregnancy, 781 exposures were recorded, and possible associations with congenital dislocation of the hip (eight cases) and clubfoot (six cases) were found. There is inadequate evidence in humans for the carcinogenicity of acetaminophen. ANIMAL TOXICITY STUDIES: There is inadequate evidence in experimental animals for the carcinogenicity of acetaminophen. In rats fasted 24 hours and given a single dose of acetaminophen (2 g/kg) by gavage, liver necrosis around the central vein was noted at 9-12 hours and was much more extensive at 24 hours after treatment. In mice after dietary exposure to acetaminophen up to 6400 mg/kg daily for 13 weeks hepatotoxicity, organ weight changes and deaths were observed. Cats are particularly susceptible to acetaminophen intoxication, developing more diffuse liver changes, while hepatic centrilobular lesions found in dogs. High doses of acetaminophen caused testicular atrophy and delay in spermiogenesis in mice. Furthermore, reductions in the fertility and neonatal survival in mice were seen in the F0 generation and decreases in F1 pup weights were found at acetaminophen dose 1430 mg/kg. Acetaminophen was not mutagenic in Salmonella typhimurium assay with or without metabolic activation in six strains: TA1535, TA1537, TA1538, TA100, TA97 and TA98. In vitro and animal data indicate that small quantities of acetaminophen are metabolized by a cytochrome P-450 microsomal enzyme to a reactive intermediate metabolite (N-acetyl-p-benzoquinoneimine, N-acetylimidoquinone, NAPQI) which is further metabolized via conjugation with glutathione and ultimately excreted in urine as a mercapturic acid. It has been suggested that this intermediate metabolite is responsible for acetaminophen-induced liver necrosis in cases of overdose. Excipients found in liquid formulations of acetaminophen may decrease its liver toxicity. ECOTOXICITY STUDIES: Daphnia magna was the most susceptible among the test organisms to the environmental effects of acetaminophen. Acetaminophen has recently been identified as a promising snake toxicant to reduce brown tree snake populations on Guam, while posing only the minimal risks to non-target rodents, cats, pigs and birds.

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
No data 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.
Waste treatment: Not available.
Contaminated packaging: Dispose of as unused product or according to local requirements.

SECTION 14: Transport information

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14.1 UN number
Not available.

14.2 UN proper shipping name
Not available.

14.3 Transport hazard class(es)
Not available.

14.4 Packing group
Not available.

14.5 Environmental hazards
Not available.

14.6 Special precautions for user
Not available.

14.7 Maritime 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
Not available.

15.2 Chemical safety assessment
No data available.

SECTION 16: Other information

SECTION 16: Other information

Product name: Paracetamol (Acetaminophen)
Catalog No.: CS-SS-24003
CAS No.: 103-90-2
Molecular weight: 151.16
Synonyms: 4-(N-Acetylamino) phenol); Acetaminophen; Abensanil; Cetamol
Supplier: Clearsynth Labs Ltd., Mumbai, India

Emergency phone: +91-22-245045900

Revision date: 2026-03-19

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