SUMMARY OF PRODUCT CHARACTERISTICS

1 NAME OF THE MEDICINAL PRODUCT

Headache and Upset Stomach Relief.

Asda Headache and Upset Stomach Relief

Boots Headache and Upset Stomach Relief

Superdrug Headache and Upset Stomach Relief

Tesco Health Recovery Effervescent Powder

Boots Recovery Effervescent Powder

2 QUALITATIVE AND QUANTITATIVE COMPOSITION

Each sachet contains; 1000 mg Paracetamol, Anhydrous citric acid 1185 mg, Sodium bicarbonate 808 mg, Potassium bicarbonate 715 mg, Anhydrous sodium carbonate 153 mg.

Excipients with known effect

Each sachet contains:

Aspartame 50mg

Sunset Yellow (E110) 0.125mg

For full list of excipients, see section 6.1

3. PHARMACEUTICAL FORM

Effervescent powder.

A free-flowing creamy/white powder with a characteristic orange odour. Free from large aggregates and particulate contamination.

4. CLINICAL PARTICULARS

4.1. Therapeutic indications

Recommended for the relief of headache with gastric upset, particularly associated with over-indulgence in food or drink or both.

4.2 Posology and method of administration

For oral administration.

Dissolve the contents of the sachet in a glass of water (150 – 200 ml) before taking.

Adults, elderly and adolescents aged 16 years and over:
One sachet every 4 to 6 hours as required. Do not take more than 4 sachets in any 24 hours.

Not to be given to adolescents and children under 16 years of age.

Instructions for use:
The recommended daily dosage or the specified number of doses should not be exceeded because of the risk of liver damage (see section 4.4 and 4.9).
Minimum dosing interval: 4 hours.
If pain or fever persist for more than 3 days or get worse, or if any other symptoms occur, treatment should be discontinued and a physician should be consulted.

Elderly patients:
Elderly patients, especially those who are frail or immobile, may require a reduced dose or frequency of dosing.

Renal impairment:
Patients who have been diagnosed with renal impairment must seek medical advice before taking this medication. It is recommended, when giving paracetamol to patients with renal failure, to reduce the dose and to increase the minimum interval between each administration to at least 6 hours. The product is contraindicated in patients with severe renal impairment (see sections 4.3 and 4.4).

4.3 Contraindications

Known hypersensitivity to any of the ingredients listed in section 6.1.
Hepatic or severe renal impairment.
Patients on sodium-restricted diets.
Patients on potassium-restricted diets

4.4. Special warnings and special precautions for use

Underlying liver disease increases the risk of paracetamol-related liver damage. The hazard of overdose is greater in those with non-cirrhotic alcoholic liver disease.

Paracetamol should be administered only with particular caution under the following circumstances:

- Chronic alcoholism
- Renal failure (GFR≤50ml/min). The product is contraindicated in case of severe renal failure (see section 4.3).
- Gilbert's Syndrome (familial non-haemolytic jaundice)
- Concomitant treatment with medicinal products affecting hepatic function
- Glucose-6-phosphate dehydrogenase deficiency
- Haemolytic anaemia
- Glutathione deficiency
- Dehydration
- Chronic malnutrition
- The elderly, adults and adolescents weighing less than 50kg

Cases of high anion gap metabolic acidosis (HAGMA) due to pyroglutamic acidosis have been reported in patients with severe illness such as severe renal impairment and sepsis, or in patients with malnutrition or other sources of glutathione deficiency (e.g. chronic alcoholism) who were treated with paracetamol at therapeutic dose for a prolonged period or a combination of paracetamol and flucloxacillin. If HAGMA due to pyroglutamic acidosis is suspected, prompt discontinuation of paracetamol and close monitoringis recommended. The measurement of urinary 5-oxoproline may be useful to identify pyroglutamic acidosis as underlying cause of HAGMA in patients with multiple risk factors.

Prolonged use of any type of painkiller for headaches can make them worse. If this situation is experienced or suspected, medical advice should be obtained and treatment should be discontinued. The diagnosis of medication overuse headache should be suspected in patients who have frequent or daily headaches despite (or because of) the regular use of headache medications.

Precaution should be observed in patients with asthma who are sensitive to acetylsalicylic acid, since mild bronchospasms are reported in association with paracetamol (cross reaction).

The stated dose should not be exceeded.

Immediate medical advice should be sought in the event of an overdose, even if the patient feels well because the risk of irreversible liver damage (see section 4.9).

.Patients should be advised not to take paracetamol containing products concurrently.

This medicine contains 50 mg aspartame in each sachet. Aspartame is a source of phenylalanine. It may be harmful if patients have phenylketonuria (PKU), a rare genetic disorder in which phenylalanine builds up because the body

cannot remove it properly. Neither non-clinical nor clinical data are available to assess aspartame use in infants below 12 weeks of age.

Each sachet contains 0.125mg Sunset Yellow which may cause allergic reactions.

4.5. Interaction with other medicinal products and other forms of interaction

The anticoagulant effect of warfarin and other coumarins may be enhanced by prolonged regular use of paracetamol with increased risk of bleeding;occasional doses have no significant effect.

Paracetamol is metabolized in the liver and can therefore interact with other medicines that follow the same pathway or may inhibit or induce this route; causing hepatotoxicity, particularly in overdose (see Section 4.9). The hepatototoxicity of paracetamol may be potentiated by excessive intake of alcohol.

The speed of absorption of paracetamol may be increased by metaclopramide or domperidone and absorption reduced by colestyramine. These interactions are considered to be of unlikely clinical significance in acute use at the dosage regimen proposed.
Colestyramine should not be administered within one hour of taking paracetamol.

There is limited evidence suggesting that paracetamol may affect chloramphenicol pharmacokinetics but its validity has been criticised and evidence of a clinically relevant interaction appears to be lacking. Although no routine monitoring is needed, it is important to bear in mind this potential interaction when these two medications are concomitantly administered, especially in malnourished patients.

In case of concomitant treatment with probenecid, the dose of paracetamol should be reduced because probenecid reduces the clearance of paracetamol by 50% because it prevents the conjugation of paracetamol with glucuronic acid.

Caution should be taken when paracetamol is used concomitantly with flucloxacillin as concurrent intake has been associated with high anion gap metabolic acidosis due to pyroglutamic acidosis, especially in patients with risks factors (see section 4.4).

Concomitant use of other paracetamol-containing medicines should be avoided. Consult your doctor if you are taking warfarin.

4.6 Fertility, pregnancy and lactation

Pregnancy

A large amount of data on pregnant women indicate neither malformative, nor feto/neonatal toxicity. Epidemiological studies on neurodevelopment in children exposed to paracetamol in utero show inconclusive results. If clinically needed, paracetamol can be used during pregnancy however it should be used at the lowest effective dose for the shortest possible time and at the lowest possible frequency.

Lactation
Paracetamol is excreted in breast milk but not in a clinically significant amount. Available published data do not contraindicate breast feeding.

Fertility
There are no data available regarding the influence of this medicine on fertility.

4.7. Effects on ability to drive and use machines

None.

4.8. Undesirable effects

Adverse reactions reported from extensive post-marketing experience are tabulated below by System Organ Class and frequency. The following convention has been utilised for the classification of undesirable effects: very common (≥1/10), common (≥1/100, <1/10), uncommon (≥1/1,000, <1/100), rare (≥1/10,000, <1/1000), very rare (<1/10,000), not known (cannot be estimated from available data).

Organ System Class	Adverse reaction	Frequency
Blood and lymphatic system disorders	Thrombocytopaenia Agranulocytosis	Rare
Immune system disorders	Anaphylaxis	Very rare
	Allergies (not including angioedema)	Rare
Respiratory, thoracic and mediastinal disorders	Bronchospasm in patients sensitive to aspirin and other NSAIDs	Very rare
Hepatobiliary disorders	Hepatic dysfunction	Very rare
Skin and subcutaneous tissue disorders	Cutaneous hypersensitivity reactions including skin rashes, pruritus, sweating, purpura, urticaria and angioedema.	Very rare
	Very rare cases of serious skin reactions have been reported. Toxic epidermal necrolysis (TEN), drug-induced dermatitis, Stevens-Johnson syndrome, Acute generalized exanthematous pustulosis (AGEP).	Very rare
Renal and urinary disorders	Sterile pyuria (cloudy urine)	Very rare

Metabolism and nutrition disorders	High anion gap metabolic acidosis	Not known

Description of selected adverse reactions
High anion gap metabolic acidosis
Cases of high anion gap metabolic acidosis due to pyroglutamic acidosis have been observed in patients with risk factors using paracetamol (see section 4.4). Pyroglutamic acidosis may occur as a consequence of low glutathione levels in these patients.

Reporting of suspected adverse reactions
Reporting suspected adverse reactions after authorisation of the medicinal product is important. It allows continued monitoring of the benefit/risk balance of the medicinal product. Healthcare professionals are asked to report any suspected adverse reactions via the Yellow Card Scheme at: www.mhra.gov.uk/yellowcard or search for MHRA Yellow Card in the Google Play or Apple App Store.

4.9 Overdose

PARACETAMOL

Liver damage is possible in adults who have taken 10g or more of paracetamol. Ingestion

of 5g or more of paracetamol may lead to liver damage if the patient has risk factors (see below).

Risk factors

If the patient

a, Is on long term treatment with carbamazepine, phenobarbitone, phenytoin, primidone,

rifampicin, St John's Wort or other drugs that induce liver enzymes.

Or

b, Regularly consumes ethanol in excess of recommended amounts.

Or

c, Is likely to be glutathione deplete e.g. eating disorders, cystic fibrosis, HIV infection,

starvation, cachexia.

Symptoms

Symptoms of paracetamol overdosage in the first 24 hours are pallor, nausea, vomiting,

anorexia and abdominal pain. Liver damage may become apparent 12 to 48 hours after

ingestion. Abnormalities of glucose metabolism and metabolic acidosis may occur. In

severe poisoning, hepatic failure may progress to encephalopathy, haemorrhage,

hypoglycaemia, cerebral oedema, and death. On initial presentation, the patient's symptoms may be limited to nausea or vomiting and may not reflect the severity of overdose or the risk of organ damage.Acute renal failure with acute tubular necrosis, strongly suggested by loin pain, haematuria and proteinuria, may develop even in the absence of severe liver damage. Cardiac arrhythmias and pancreatitis have been reported.

Management

Immediate treatment is essential in the management of paracetamol overdose. Despite a

lack of significant early symptoms, patients should be referred to hospital urgently for

immediate medical attention. Symptoms may be limited to nausea or vomiting and may

not reflect the severity of overdose or the risk of organ damage. Management should be

in accordance with established treatment guidelines, see BNF overdose section.

Treatment with activated charcoal should be considered if the overdose has been taken

within 1 hour. Plasma paracetamol concentration should be measured at 4 hours or later

after ingestion (earlier concentrations are unreliable). Treatment with N-acetylcysteine

may be used up to 24 hours after ingestion of paracetamol, however, the maximum

protective effect is obtained up to 8 hours post-ingestion. The effectiveness of the

antidote declines sharply after this time. If required the patient should be given

intravenous N-acetylcysteine, in line with the established dosage schedule. If vomiting is

not a problem, oral methionine may be a suitable alternative for remote areas, outside

hospital. Management of patients who present with serious hepatic dysfunction beyond

24h from ingestion should be discussed with the NPIS or a liver unit.

5 PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic properties

Pharmacotherapeutic group: Analgesics, Anilides.
ATC code NO2B E01
Paracetamol – antipyretic and mild analgesic actions.

Mechanism of action
Paracetamol is an analgesic and antipyretic. Its mechanism of action is believed to include inhibition of prostaglandin synthesis, primarily within the central nervous system. The lack of peripheral prostaglandin inhibition confers important pharmacological properties such as the maintenance of the protective prostaglandins within the gastrointestinal tract

Citric acid
Sodium bicarbonate
Potassium bicarbonate
Sodium carbonate

Sodium and potassium citrates are formed by effervescent reaction with water. These provide acid neutralising and buffering actions against acidic gastric contents.

5.2 Pharmacokinetic properties

Paracetamol
Absorption: Paracetamol is readily and rapidly absorbed from the gastro-intestinal tract with maximum plasma concentrations being reached 10 to 60 minutes after oral ingestion.

Distribution: Paracetamol is relatively uniformly distributed throughout most bodily fluids and exhibits variable protein binding.

Biotransformation: Paracetamol is metabolised mainly in the liver, following two major metabolic pathways, glucuronic acid and sulphuric acid conjugates. The latter route is rapidly saturated at doses higher than the therapeutic dosages. A minor route, catalyzed by the Cytocrome P450 (mostly CYP2E1), results in the formation of an intermediate reagent
(N-acetyl-p-benzoquinoneimine) which under normal conditions of use, is rapidly detoxified by glutathione and eliminated in the urine, after conjugation with cysteine and mercapturic acid. Conversely, when massive intoxication occurs, the quantity of this toxic metabolite is
increased.

Elimination: Less than 5% is excreted as unmodified paracetamol; the elimination half-life varies from 1 to 4 hours. Elimination is essentially through the urine. 90% of the ingested dose is eliminated via the kidneys within 24 hours, principally as glucuronide (60-80%) and sulphate conjugates (20-30%). In cases of renal failure (GFR≤50ml/min), the elimination of paracetamol is slightly delayed, the elimination half-life ranging from 2 to 5.3 hours. For the glucuronide and sulfate conjugates, the elimination rate is 3 times slower in subjects with severe renal impairment than in healthy subjects.

Antacid combination provides an immediately available, local buffering effect in the stomach. Absorbed sodium, potassium and citrate ions will be handled and excreted by normal metabolic routes.

5.3 Preclinical safety data

Conventional studies using the currently accepted standards for the evaluation of toxicity to reproduction and development are not available.

6 PHARMACEUTICAL PARTICULARS

6.1 List of excipients

orange flavour (including malto-dextrin, gum Arabic (E414), triacetin and alpha-tocopherol (E307))
acesulfame potassium (E950)
aspartame (E951)
quinoline yellow (E104)
sunset yellow (E110)
ascorbic acid

6.2. Incompatibilities

Not applicable.

6.3. Shelf life

36 months.

6.4. Special precautions for storage

Not applicable.

6.5. Nature and Contents of Container

This product is packed in laminate sachets comprising paper/polyethylene/aluminium foil/ polyethylene or paper/polyethylene/aluminium foil/Surlyn.

Five or ten sachets are contained in a boxboard carton.

6.6. Instruction for use and handling

Not applicable.

7. MARKETING AUTHORISATION HOLDER

Wrafton Laboratories Limited
Wrafton
Braunton
North Devon EX33 2DL
United Kingdom

8. MARKETING AUTHORISATION NUMBER

PL 12063/0033

9. DATE OF FIRST AUTHORISATION/RENEWAL OF THE AUTHORISATION

12 June 2003

10 DATE OF REVISION OF THE TEXT

23/04/2025