SUMMARY OF PRODUCT CHARACTERISTICS

1 NAME OF THE MEDICINAL PRODUCT

Disprin

2 QUALITATIVE AND QUANTITATIVE COMPOSITION

Active Ingredient	mg/Tablet	Specification
Aspirin	300.00	Ph Eur

Excipient(s) with known effect:
Sodium 0.12 mg (0.005 mmol) per tablet,
Soy protein 0.0625 – 0.125 mg per tablet

For the full list of excipients, see section 6.1.

3 PHARMACEUTICAL FORM

Dispersible tablet

4 CLINICAL PARTICULARS

4.1 Therapeutic indications

For the relief of mild to moderate pain in headaches, including migraine headaches, toothache, neuralgia, sciatica, period pains and sore throats.

Reduction of temperature in feverishness, influenza and colds.

Reduction of inflammation in rheumatism and lumbago.

4.2 Posology and method of administration

Oral administration after dissolution in water.

Adults (including children 16 years and over): Two to three tablets every 4 -6 hours. Do not exceed 12 tablets in 24 hours.

Do not give to children aged under 16 years unless specifically indicated (e.g. for Kawasaki's disease).

There is no indication that dosage need be modified in the elderly.

The lowest effective dose should be used for the shortest duration necessary to relieve symptoms.

The patient should consult a doctor if symptoms persist or worsen, or if the product is required for more than 3 days.

Hepatic Impairment: Patients with hepatic impairment should seek the advice of a doctor before taking this product (see section 4.4).

Renal Impairment: Patients with renal impairment should seek the advice of a doctor before taking this product (see section 4.4).

Elderly population: Non-steroidal anti-inflammatory drugs should be used in caution in elderly patients who are more prone to adverse events (see section 4.4).

4.3 Contraindications

Hypersensitivity to acetylsalicylic acid or to any of the excipients listed in section 6.1.

Hypersensitivity to other non-steroidal anti-inflammatory drugs.

Nasal polyps associated with asthma.

Should not be given to patients suffering from a previous history of peptic ulceration or active peptic ulceration or bleeding disorders including haemophilia.

Disprin contains **soy protein**. If you are allergic to **peanut** or **soya** do not use this medicinal product.

Severe hepatic impairment.

Severe renal impairment.

Severe heart failure.

Children aged under 16 years due to a possible risk of Reye's syndrome, unless specifically indicated (see section 4.4).

Doses >100 mg/day during the third trimester of pregnancy (see section 4.6).

Breast-feeding (see section 4.6).

4.4 Special warnings and precautions for use

This medicine contains less than 1mmol sodium (23 mg) per dose, that is to state essentially ‘sodium-free’.

Undesirable effects may be minimised by using the lowest effective dose for the shortest duration necessary to control symptoms.

Gastrointestinal effects: NSAIDs should be given with care to patients with a history of gastrointestinal disease (ulcerative colitis, Crohn’s disease) as their condition may be exacerbated (see section 4.8).

Gastrointestinal bleeding, ulceration or perforation which can be fatal, has been reported with all NSAIDs at any time during treatment, with or without warning symptoms or a previous history of serious GI events.

The risk of GI bleeding, ulceration or perforation is higher with increasing NSAID doses, in patients with a history of ulcer, particularly if complicated with haemorrhage or perforation and in the elderly. These patients should commence treatment on the lowest dose available.

Patients with a history of GI toxicity, particularly the elderly, should report any unusual abdominal symptoms (especially GI bleeding) particularly in the initial stages of treatment.

Acetylsalicylic acid decreases platelet adhesiveness and increases bleeding time. Caution should be advised in patients receiving concomitant medications which could increase the risk of ulceration or bleeding, such as oral corticosteroids, anticoagulants such as warfarin, selective serotonin-reuptake inhibitors or anti-platelet agents such as clopidogrel and ticlopidine (see section 4.5).

SLE and mixed connective tissue disease: Systemic lupus erythematosus and mixed connective tissue disease, due to increased risk of aseptic meningitis (see section 4.8).

Dermatological effects: Serious skin reactions, some of them fatal, including exfoliative dermatitis, Stevens-Johnson syndrome, and toxic epidermal necrolysis, have been reported very rarely in association with the use of NSAIDs (see section 4.8). Patients appear to be at highest risk of these reactions early in the course of therapy, the onset of the reaction occurring in the majority of cases within the first month of treatment. Acetylsalicylic acid should be discontinued at the first appearance of skin rash, mucosal lesions, or any other sign of hypersensitivity.

Paediatric use: There is a possible association between acetylsalicylic acid and Reye’s syndrome when given to children. Reye’s syndrome is a very rare disease, which affects the brain and liver, and can be fatal. For this reason acetylsalicylic acid should not be given to children aged under 16 years unless specifically indicated (e.g. for Kawasaki’s disease).

Cardiovascular effects: Caution (discussion with doctor or pharmacist) is required prior to starting treatment in patients with a history of hypertension and/or heart failure as fluid retention, hypertension, and oedema have been reported in association with NSAID therapy.

Respiratory effects: Bronchospasm may be precipitated in patients suffering from or with a previous history of bronchial asthma or allergic disease.

Renal: Caution is advised in patients with renal impairment (see section 4.8).

Hepatic: Caution is advised in patients with hepatic impairment (see section 4.8).

Elderly: The elderly have an increased frequency of adverse reactions to NSAIDs especially gastrointestinal bleeding and perforation which may be fatal.

Other NSAIDs: The use with concomitant NSAIDs including cyclooxygenase-2 selective inhibitors should be avoided.

Impaired female fertility: May cause impairment of female fertility (see section 4.6).

Patients with gout: The product should not be given to patients with gout, as serum urate may be increased, unless recommended by a healthcare professional.

Surgical procedures: Acetylsalicylic acid should be stopped several days before scheduled surgical procedures due to increased bleeding time.

Laboratory tests: Acetylsalicylic acid and other salicylates can interfere with thyroid function tests.

Caution should be implemented in patients with a history of other bleeding events such as intracranial haemorrhage, due to the increased risk of intercranial haemorrhage with acetylsalicylic acid use.

Overuse headaches: Use of NSAIDs or analgesics for 15 or more days per month, for 3 months or more, increases your risk of medication overuse headache.

4.5 Interaction with other medicinal products and other forms of interaction

Corticosteroids: Increased risk of gastrointestinal ulceration or bleeding (see section 4.4).

Selective serotonin reuptake inhibitors (SSRIs): Increased risk of gastrointestinal bleeding (see section 4.4).

Calcium channel blockers: Reduced hypotensive effects, increased anti-platelet effects rarely resulting in prolonged bleeding time.

Cardiac glycosides: NSAIDs may exacerbate cardiac failure, reduce GFR and increase plasma cardiac glycoside levels.

Varicella vaccine: Avoid use of acetylsalicylic acid in varicella vaccine recipients due to a possible association with Reye's syndrome.

Antihypertensives (ACE inhibitors and Angiotensin II Antagonists) and diuretics: NSAIDs may reduce the effect of diuretics and other antihypertensive drugs. In some patients with compromised renal function (e.g. dehydrated patients or elderly patients with compromised renal function) the co-administration of an ACE inhibitor or Angiotensin II antagonist and agents that inhibit cyclo-oxygenase may result in further deterioration of renal function, including possible acute renal failure, which is usually reversible. These interactions should be considered in patients taking a coxib concomitantly with ACE inhibitors or angiotensin II antagonists. Therefore,

the combination should be administered with caution, especially in the elderly. Patients should be adequately hydrated and consideration should be given to monitoring of renal function after initiation of concomitant therapy, and periodically thereafter. Diuretics can increase the risk of nephrotoxicity of NSAIDs.

Anti-coagulants: Aspirin may enhance the effects of anticoagulants and inhibit the effects of uricosurics.

Ibuprofen: Experimental data suggest that ibuprofen may inhibit the effect of low dose aspirin on platelet aggregation when they are dosed concomitantly. However, the limitations of these data and the uncertainties regarding extrapolation of *ex-vivo* data to the clinical situation imply that no firm conclusions can be made for regular ibuprofen use, and no clinically relevant effect is considered to be likely for occasional ibuprofen use (see section 5.1).

Other NSAIDs or other salicylates including cyclooxygenase-2 selective inhibitors: Avoid concomitant use of two or more NSAIDs as this may increase the risk of adverse effects (see section 4.4).

Ciclosporin: Increased risk of nephrotoxicity with NSAIDs.

Tacrolimus: Possible increased risk of nephrotoxicity when NSAIDs are given with tacrolimus.

Zidovudine: Increased risk of haematological toxicity when NSAIDs are given with zidovudine.

Metoclopramide & domperidone: May increase the rate of absorption of acetylsalicylic acid.

Valproate: Acetylsalicylic acid may increase valproate levels resulting in valproate toxicity.

Quinolone antibiotics: Animal data indicate that NSAIDs can increase the risk of convulsions associated with quinolone antibiotics. Patients taking NSAIDs and quinolones may have an increased risk of developing convulsions.

Uricosurics: Acetylsalicylic acid may inhibit the effects of uricosurics.

Mifepristone: NSAIDs can reduce the effect of mifepristone.

Methotrexate: Decreased elimination of methotrexate.

Carbonic anhydrase inhibitors e.g. Acetazolamide: May result in severe acidosis and increased central nervous system toxicity.

Metamizole: Metamizole may reduce the effect of acetylsalicylic acid on platelet aggregation when taken concomitantly. Therefore, this combination should be used with caution in patients taking low dose aspirin for cardio protection.

Sulfonylurea hypoglycaemic drugs: Acetylsalicylic acid may increase the activity of sulfonylurea hypoglycaemic drugs.

Zafirlukast: The activity of zafirlukast may be increased following acetylsalicylic acid administration.

Phenytoin: Acetylsalicylic acid may increase the effects of phenytoin.

4.6 Pregnancy and lactation

Pregnancy

Doses of above 100 mg/day and up to 500 mg/day:

There is insufficient clinical experience regarding the use of doses above 100 mg/day up to 500 mg/day. Therefore, the recommendations below for doses of 500 mg/day and above apply also for this dose range.

Doses of 500 mg/day and above:

The product is contraindicated in the third trimester of pregnancy (see section 4.3) and should be avoided during the first and second trimesters.

Inhibition of prostaglandin synthesis may adversely affect the pregnancy and/or the embryo/foetal development. Data from epidemiological studies suggest an increased risk of miscarriage and of cardiac malformation and gastroschisis after use of a prostaglandin synthesis inhibitor in early pregnancy. The absolute risk for cardiovascular malformation was increased from less than 1%, up to approximately 1.5%. The risk is believed to increase with dose and duration of therapy. In animals, administration of a prostaglandin synthesis inhibitor has been shown to result in increased pre- and post-implantation loss and embryo-foetal lethality. In addition, increased incidences of various malformations, including cardiovascular, have been reported in animals given a prostaglandin synthesis inhibitor during the organogenetic period. From the 20th week of pregnancy onward, acetylsalicylic acid use may cause oligohydramnios resulting from foetal renal dysfunction. This may occur shortly after treatment initiation and is usually reversible upon discontinuation. In addition, there have been reports of ductus arteriosus constriction following treatment in the second trimester, most of which resolved after treatment cessation. Therefore, during the first and second trimester of pregnancy, acetylsalicylic acid should not be given unless clearly necessary. If acetylsalicylic acid is used by a woman attempting to conceive, or during the first and second trimester of pregnancy, the dose should be kept as low and duration of treatment as short as possible. Antenatal monitoring for oligohydramnios and ductus arteriosus constriction should be considered after exposure to acetylsalicylic acid for several days from gestational week 20 onward. Acetylsalicylic acid should be discontinued if oligohydramnios or ductus arteriosus constriction are found.

During the third trimester of pregnancy, all prostaglandin synthesis inhibitors may expose the foetus to:

- cardiopulmonary toxicity (premature constriction/closure of the ductus arteriosus and pulmonary hypertension);
- renal dysfunction (see above);

the mother and the neonate, at the end of pregnancy, to:

- possible prolongation of bleeding time, an anti-aggregating effect which may occur even at very low doses;
- inhibition of uterine contractions resulting in delayed or prolonged labour.

Consequently, acetylsalicylic acid at doses higher than 100 mg/day is contraindicated during the third trimester of pregnancy (see section 4.3).

Breast-feeding

Use of the product is contraindicated in breast-feeding. Acetylsalicylic acid given in breast-feeding mothers may pose a risk of Reye's syndrome in nursing infants (see section 4.3).

Fertility

There is some evidence that drugs which inhibit cyclo-oxygenase / prostaglandin synthesis may cause impairment of female fertility by an effect on ovulation. This is reversible on withdrawal of treatment.

4.7 Effects on ability to drive and use machines

None known.

4.8 Undesirable effects

Adverse events which have been associated with acetylsalicylic acid are given below, tabulated by system organ class and frequency. Frequencies are defined as: Very common (≥1/10); Common (≥1/100 and <1/10); Uncommon (≥1/1000 and <1/100); Rare (≥1/10,000 and <1/1000); Very rare (< 1/10,000); Not known (cannot be estimated from the available data). Within each frequency grouping, adverse events are presented in order of decreasing seriousness.

System Organ Class	Frequency	Adverse Events
Blood and	Not known	Hypoprothrombinaemia,

System Organ Class	Frequency	Adverse Events
Lymphatic System Disorders		thrombocytopenia, aplastic anaemia, agranulocytosis, pancytopenia
Immune System Disorders	Not known	Hypersensitivity[1], pyrexia, urticaria, pruritus, angioedema[1]
Metabolism and Nutrition Disorders	Not known	Sodium retention, fluid retention
Nervous System Disorders	Not known	Aseptic meningitis
Cardiac Disorders	Not known	Cardiac failure, oedema
Vascular Disorders	Not known	Hypertension, haemorrhage intercranial
Respiratory, Thoracic and Mediastinal Disorders	Very rare	Aspirin-exacerbated respiratory disease
	Not known	Bronchospasm, asthma, dyspnoea, rhinitis[1]
Gastrointestinal Disorders	Not known	Gastrointestinal haemorrhage, gastrointestinal disturbances[2], peptic ulcer, melaena, haematemesis, mouth ulceration
Hepatobiliary Disorders	Not known	Hepatotoxicity
Skin and Subcutaneous Tissue Disorders	Not known	Stevens-Johnson syndrome, toxic epidermal necrolysis, rash[1]
Renal and Urinary Disorders	Not known	Blood uric acid increased
Investigations	Not known	Bleeding time prolonged, platelet adhesiveness decreased

[1] Hypersensitivity reactions may consist of (a) respiratory tract reactivity, including asthma, bronchospasm (potentially severe, even fatal) and dyspnoea; (b) various skin reactions, including urticaria, angioedema, pruritus, other skin eruptions, and more rarely bullous reactions including Stevens-Johnson syndrome and toxic epidermal necrolysis.

[2] Gastrointestinal effects may include nausea, vomiting, dyspepsia, gastritis, diarrhoea, and constipation.

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

Salicylate poisoning is usually associated with plasma concentrations >350 mg/L (2.5 mmol/L). Most adult deaths occur in patients whose concentrations exceed 700 mg/L (5.1 mmol/L). Single doses less than 100 mg/kg are unlikely to cause serious poisoning.

Symptoms

Common features include vomiting, dehydration, tinnitus, vertigo, deafness, sweating, warm extremities with bounding pulses, increased respiratory rate, fever, dizziness and hyperventilation. Some degree of acid-base disturbance is present in most cases. Headache, nausea, restlessness, and ketosis may also occur.

A mixed respiratory alkalosis and metabolic acidosis with normal or high arterial pH (normal or reduced hydrogen ion concentration) is usual in adults and children over the age of four years. In children aged four years or less, a dominant metabolic acidosis with low arterial pH (raised hydrogen ion concentration) is common. Acidosis may increase salicylate transfer across the blood brain barrier.

Uncommon features include haematemesis, hyperpyrexia, hypoglycaemia, hypokalaemia, thrombocytopaenia, increased INR/PTR, intravascular coagulation, renal failure and non-cardiac pulmonary oedema.

Central nervous system features including confusion, disorientation, coma and convulsions are less common in adults than in children. In severe cases, respiratory failure and cardiovascular collapse are also possible.

Management

Give activated charcoal if an adult presents within one hour of ingestion of more than 125 mg/kg. The plasma salicylate concentration should be measured, although the severity of poisoning cannot be determined from this alone and the clinical and biochemical features must be taken into account. Elimination is increased by urinary alkalinisation, which is achieved by the administration of 1.26% sodium bicarbonate. The urine pH should be monitored. Correct metabolic acidosis with intravenous 8.4% sodium bicarbonate (first check serum potassium). Forced diuresis should not be used since it does not enhance salicylate excretion and may cause pulmonary oedema. Fluid and electrolyte management should be used to correct acidosis, hyperprexia, hypokalaemia and dehydration.

Haemodialysis is the treatment of choice for severe poisoning and should be considered in patients with plasma salicylate concentrations >700 mg/L (5.1 mmol/L), or lower concentrations associated with severe clinical or metabolic features. Patients under ten years or over 70 have increased risk of salicylate toxicity and may require dialysis at an earlier stage.

5 PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic properties

Aspirin:

Aspirin inhibits the cyclo-oxygenase enzyme involved in conversion of phospholipids to prostaglandins and its effects on the body are believed to result primarily from prevention of prostaglandin production. These effects include peripheral analgesia, fever reduction, reduction in inflammation and inhibition of platelet aggregation.

Experimental data suggest that ibuprofen may inhibit the effect of low dose aspirin on platelet aggregation when they are dosed concomitantly. In one study, when a single dose of ibuprofen 400mg was taken within 8 hours before or within 30 minutes after immediate release aspirin dosing (81mg), a decreased effect of aspirin on the formation of thromboxane or platelet aggregation occurred. However, the limitations of these data and the uncertainties regarding extrapolation of *ex vivo* data to the clinical situation imply that no firm conclusions can be made for regular ibuprofen use, and no clinically relevant effect is considered to be like for occasional ibuprofen use.

5.2 Pharmacokinetic properties

Aspirin is rapidly absorbed from the stomach and upper gastrointestinal tract with peak levels after around 20-30 minutes following dissolution. It is subject to first-pass metabolism with an overall bioavailability of around 70%. Metabolism is by conversion to salicylic acid and then by further conversion to other metabolites. These are excreted by the kidneys in both free and conjugated form. The plasma half-life of aspirin is around 15-20 minutes and that of salicylic acid is 2-3 hours.

5.3 Preclinical safety data

No preclinical findings of relevance have been reported.

6 PHARMACEUTICAL PARTICULARS

6.1 List of excipients

Calcium carbonate, maize starch, citric acid, talc, sodium lauryl sulphate, saccharin, crospovidone and lime flavour.

6.2 Incompatibilities

None known.

6.3 Shelf life

Three years.

6.4 Special precautions for storage

Store below 25°C in a dry place.

6.5 Nature and contents of container

Cardboard carton containing tablets in strips of aluminium foil with vinyl heat seal. Pack sizes: 6, 8, 12, **16**, 24, **32**, 48, 96 and 500 tablets. (Those pack sizes printed in bold are currently sold).

6.6 Special precautions for disposal

Oral administration after dissolution in water.

7 MARKETING AUTHORISATION HOLDER

Reckitt Benckiser Healthcare (UK) Limited
Dansom Lane
Hull
HU8 7DS
United Kingdom.

8 MARKETING AUTHORISATION NUMBER(S)

PL 00063/0017.

9 DATE OF FIRST AUTHORISATION/RENEWAL OF THE AUTHORISATION

24/04/1995 / 23/02/2004

10 DATE OF REVISION OF THE TEXT

21/07/2025