Summary of Product Characteristics

1 NAME OF THE MEDICINAL PRODUCT

Xefo Rapid 8 mg film-coated tablets

2 QUALITATIVE AND QUANTITATIVE COMPOSITION

One film-coated tablet contains 8 mg lornoxicam.

For the full list of excipients, see section 6.1.

3 PHARMACEUTICAL FORM

Film-coated tablet
White to yellowish round biconvex film-coated tablet.

4 CLINICAL PARTICULARS

4.1 Therapeutic Indications

Short-term relief of acute mild to moderate pain.

4.2 Posology and method of administration

Posology

For all patients the appropriate dosing regimen should be based upon individual response to treatment.

Acute pain
8-16 mg lornoxicam given in doses of 8 mg. An initial dose of 16 mg followed by 8 mg 12 hours later can be given on the first treatment day. After the first treatment day the maximum recommended daily dose is 16 mg.

Additional information on special populations

Children and adolescents
Lornoxicam is not recommended for use in children and adolescents below age 18 because of a lack of data on safety and efficacy.

Elderly
No special dosage modification is required for elderly patients above age 65 unless renal or hepatic function is impaired. Lornoxicam should be administered with precaution as gastrointestinal adverse effects are less well tolerated in this group (see section 4.4).

Renal impairment
Reduction of dose frequency of Xefo Rapid to once daily in patients suffering from renal impairment is recommended.

Hepatic impairment
Reduction of dose frequency of Xefo Rapid to once daily in patients suffering from hepatic impairment is recommended.

Undesirable effects may be minimised by using the lowest effective dose for the shortest duration necessary to control symptoms (see section 4.4.).

Method of Administration

Xefo Rapid film-coated tablets are supplied for oral administration and should be taken with a sufficient quantity of liquid.

4.3 Contraindications

- Hypersensitivity to the active substance or to any of the excipients listed in section 6.1
- Thrombocytopenia
- Hypersensitivity (symptoms like asthma, rhinitis, angioedema or urticaria) to other NSAIDs including acetylsalicylic acid
- Severe heart failure
- Gastro-intestinal bleeding, cerebrovascular bleeding or other bleeding disorders
- History of gastrointestinal bleeding or perforation, related to previous NSAIDs therapy
- Active or history of recurrent peptic ulcer/haemorrhage (two or more distinct episodes of proven ulceration or bleeding)
- Severe hepatic impairment
- Severe renal impairment (Serum creatinine > 700 µmol/l)
- The third trimester of pregnancy (see section 4.6).

4.4 Special warnings and precautions for use

For the following disorders, lornoxicam should only be administered after careful risk-benefit assessment:
- Renal impairment: Lornoxicam should be administered with precaution in patients with mild (serum creatinine 150-300 µmol/l) to moderate (serum creatinine 300 – 700 µmol/l) renal impairment due to dependency on renal prostaglandins for maintenance of renal blood flow. Treatment with lornoxicam should be discontinued if renal function deteriorates during treatment.
- Renal functions should be monitored in patients who undergo major surgery, with cardiac failure, receiving treatment with diuretics, receiving concomitant treatment with drugs that are suspected to or known to be able to cause kidney damage.
- Patients with blood coagulation disorders: Careful clinical monitoring and laboratory assessment is recommended (e.g. APTT).
- Hepatic impairment (e.g. liver cirrhosis): Clinical monitoring and laboratory assessments at regular intervals should be considered in patients with hepatic impairment as accumulation of lornoxicam (increase in AUC) may occur after treatment with daily doses of 12-16 mg. Apart from that, hepatic impairment does not seem to affect pharmacokinetic parameters of lornoxicam as compared to healthy subjects.
- Long term treatment (longer than 3 months): Regular laboratory assessments of haematology (haemoglobin), renal functions (creatinine) and liver enzymes are recommended.
- Elderly patients above 65 years: Monitoring of renal and hepatic function is recommended. Precaution is advised in elderly postoperative patients.
The use of lornoxicam with concomitant NSAIDs including cyclooxygenase-2 selective inhibitors should be avoided.

Undesirable effects may be minimised by using lowest effective dose for the shortest duration necessary to control symptoms (see section 4.2, and GI and cardiovascular risks below).

Gastrointestinal bleeding, ulceration and perforation: GI bleeding, ulceration or perforation, which can be fatal, has been reported with all NSAIDs at anytime 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 (see section 4.3), and in the elderly. These patients should commence treatment on the lowest dose available. Combination therapy with protective agents (e.g. misoprostol or proton pump inhibitors) should be considered for these patients, and also for patients requiring concomitant low dose acetylsalicylic acid or other active substances likely to increase gastrointestinal risk (see below and section 4.5). Clinical monitoring at regular intervals is recommended.

Patients with a history of GI toxicity, particularly when elderly, should report any unusual abdominal symptoms (especially GI bleeding) particularly in the initial stages of treatment.
Caution should be advised in patients receiving concomitant medicinal products, 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 acetylsalicylic acid (see section 4.5).

When GI bleeding or ulceration occurs in patients receiving lornoxicam, the treatment should be withdrawn.

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).

The elderly have an increased frequency of adverse reactions to NSAIDs especially gastrointestinal bleeding and perforation, which may be fatal (see section 4.3).

Caution is required in patients with a history of hypertension and/or heart failure, as fluid retention and oedema have been reported in association with NSAID therapy.

Appropriate monitoring and advice are required for patients with a history of hypertension and/or mild to moderate congestive heart failure, as fluid retention and oedema have been reported in association with NSAID therapy.

Clinical trial and epidemiological data suggest that use of some NSAIDs (particularly at high doses and in long term treatment) may be associated with a small increased risk of arterial thrombotic events (for example myocardial infarction or stroke). There are insufficient data to exclude such a risk for lornoxicam.

Patients with uncontrolled hypertension, congestive heart failure, established ischaemic heart disease, peripheral arterial disease, and/or cerebrovascular disease should only be treated with Lornoxicam after careful consideration. Similar consideration should be made before initiating longer-term treatment of patients with risk factors for cardiovascular disease (e.g. hypertension, hyperlipidaemia, diabetes mellitus, smoking).

Concomitant treatment with NSAIDs and heparin in the context of a spinal or epidural anaesthesia increases the risk of spinal/epidural haematoma (see section 4.5).

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. Lornoxicam should be discontinued at the first appearance of skin rash, mucosal lesions, or any other sign of hypersensitivity.

Caution is required if administered to patients suffering from, or with a previous history of, bronchial asthma since NSAIDs have been reported to precipitate bronchospasm in such
patients.

In patients with systemic lupus erythematosus (SLE) and mixed connective tissue disorders there may be an increased risk of aseptic meningitis.

Lornoxicam reduces platelet aggregation and prolongs bleeding time and consequently care should be taken when administering to patients with increased bleeding tendency.

Concomitant treatment of NSAIDs and tacrolimus may increase the risk of nephrotoxicity owing to reduced synthesis of prostacyclin in the kidney. Renal function must therefore be monitored closely in patients receiving combination therapy.

As with most NSAIDs occasional increase in serum transaminases level, increase in serum bilirubin or other liver function parameters, as well as increases in serum creatinine and blood urea nitrogen as well as other laboratory abnormalities have been reported. Should any such abnormality prove significant or persist the administration of lornoxicam should be stopped and appropriate investigations prescribed.

The use of lornoxicam, as with any drug known to inhibit cyclooxygenase/prostaglandin synthesis, may impair fertility and is not recommended in women attempting to conceive. In women who have difficulties conceiving or who are undergoing investigation of infertility, withdrawal of lornoxicam should be considered.

Exceptionally, varicella can be at the origin of serious cutaneous and soft tissues infectious complications.
To date, the contributing role of NSAIDs in the worsening of these infections cannot be ruled out. Thus, it is advisable to avoid use of lornoxicam in case of varicella.

4.5 Interaction with other medicinal products and other forms of interaction

Concomitant administration of lornoxicam and

- Cimetidine: Increased plasma concentrations of lornoxicam. (No interaction between lornoxicam and ranitidine, or lornoxicam and antacids has been demonstrated).
- Anti-coagulants: NSAIDs may enhance the effects of anti-coagulants, such as warfarin (see section 4.4). Careful monitoring of INR should be undertaken.
- Phenprocoumon: Decreased effect of phenprocoumon treatment.
- Heparin: NSAIDs increase the risk of spinal or epidural haematoma when given concomitantly to heparin in the context of spinal or epidural anaesthesia (see section 4.4.).
- ACE inhibitors: The antihypertensive effect of the ACE inhibitor may decrease.
- Diuretics: Decreased diuretic and antihypertensive effect of loop diuretics, thiazide diuretics, and potassium sparing diuretics.
- Beta-adrenergic blockers: Decreased antihypertensive efficacy.
- Angiotensin II receptor blocker: Decreased antihypertensive efficacy.
- Digoxin: Decreased renal clearance of digoxin.
- Corticosteroids: Increased risk of gastrointestinal ulceration or bleeding (see section 4.4).
- Quinolone antibiotics: Increased risk of seizures.
- Anti-platelet agents: Increased risk of gastrointestinal bleeding (see section 4.4).
- Other NSAIDs: Increased risk of gastrointestinal bleeding.
- Methotrexate: Increased serum concentration of methotrexate. Increased toxicity may result. When concomitant therapy has to be used careful monitoring should be undertaken.
- Selective serotonin reuptake inhibitors (SSRIs): Increased risk of gastrointestinal bleeding (see section 4.4).
- Lithium: NSAIDs inhibit renal clearance of lithium, thus the serum concentration of lithium may increase above toxicity limits. Therefore serum lithium levels require monitoring, especially during initiation, adjustment and withdrawal of treatment.
- Cyclosporine: Increased serum concentration of cyclosporine. Nephrotoxicity of cyclosporine may be enhanced via renal prostaglandin mediated effects. During combined treatment renal function should be monitored.
- Sulphonylureas (e.g. glibenclamide): Increased risk of hypoglycaemia.
- Known inducers and inhibitors of CYP2C9 isoenzymes: Lornoxicam (as other NSAIDs depending on the cytochrome P450 2C9 (CYP2C9 isoenzyme)) has interactions with known inducers and inhibitors of CYP2C9 isoenzymes (see section 5.2 Biotransformation).
- Tacrolimus: Increase the risk of nephrotoxicity owing to reduced synthesis of prostacyclin in the kidney. During combined treatment renal function should be monitored (see section 4.4).
- Pemetrexed: NSAIDs may reduce renal clearance of pemetrexed resulting in increased renal and gastrointestinal toxicity, and myelosuppression.

Xefo film-coated tablets show a delayed absorption of lornoxicam when given with food. Therefore, Xefo film-coated tablets should not be taken with food when a quick onset of efficacy (relief of pain) is required.

Food may decrease the absorption with about 20% and increase T_{max}.

4.6 Fertility, pregnancy and lactation

Pregnancy
Lornoxicam is contraindicated on the third trimester of pregnancy and should not be used during pregnancy in the first and second trimesters and delivery, as no clinical data on exposed pregnancies are available.

There are no adequate data from the use of lornoxicam in pregnant women. Studies in animals have shown reproductive toxicity (see section 5.3).

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 after use of a prostaglandin synthesis inhibitor in early pregnancy. 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. During the first and second trimester of pregnancy, prostaglandin synthesis inhibitors should not be given unless clearly necessary.

Prostaglandin synthesis inhibitors administered during the third trimester of pregnancy may expose the foetus to cardiopulmonary toxicity (premature closure of the ductus arteriosus and pulmonary hypertension) and renal dysfunction which may lead to renal failure and hence a reduced quantity of amniotic fluid. At the end of pregnancy, prostaglandin synthesis inhibitors may expose the mother and the foetus to increased bleeding time and inhibition of uterine contractions, which may delay or prolong the labour. Therefore, the use of lornoxicam is contraindicated during the third trimester of pregnancy (see section 4.3).

Breastfeeding
There are no data on the excretion of lornoxicam in human breast milk. Lornoxicam is excreted in milk of lactating rats in relatively high concentrations. Therefore lornoxicam should not be used in breastfeeding women.

4.7 Effects on ability to drive and use machines

Patients showing dizziness and/or sleepiness under treatment with lornoxicam should refrain from driving or operation machinery.

4.8 Undesirable effects

The most commonly observed adverse events of NSAIDs are gastrointestinal in nature. Peptic ulcers, perforation or GI bleeding, sometimes fatal, particularly in the elderly, may occur (see section 4.4). Nausea, vomiting, diarrhoea, flatulence, constipation, dyspepsia, abdominal pain, melaena, haematemesis, ulcerative stomatitis, exacerbation of colitis and Crohn’s disease (see section 4.4) have been reported following administration of NSAIDs. Less frequently, gastritis has been observed.

Approximately 20% of patients treated with lornoxicam can be expected to experience adverse reactions. The most frequent adverse effects of lornoxicam include nausea, dyspepsia, indigestion, abdominal pain, vomiting, and diarrhoea. These symptoms have generally occurred in less than 10% of patients in available studies.

Oedema, hypertension, and cardiac failure, have been reported in association with NSAID treatment.

Clinical trial and epidemiological data suggest that use of some NSAIDs (particularly at high doses and in long term treatment) may be associated with an increased risk of arterial thrombotic events (for example myocardial infarction or stroke) (see section 4.4).

Exceptionally, occurrence of serious cutaneous and soft tissues infectious complications during varicella.

Listed below are undesirable effects, which generally occurred in more than 0.05% of the 6.,17 patients treated in clinical phase II, III and IV trials.

Very common (≥1/10); Common (≥1/100 to <1/10); Uncommon (≥1/1,000 to <1/100); rare (≥1/10,000 to <1/1,000);

Very rare (<1/10,000), not known (cannot be estimated from the available data).

Infections and infestations
Rare: Pharyngitis.

Blood and lymphatic system disorders
Rare: Anaemia, thrombocytopenia, leukopenia, prolonged bleeding time
Very rare: Ecchymosis. NSAIDs have been reported to cause potentially severe hematological disorders like neutropenia, agranulocytosis, aplastic anaemia, and hemolytic anaemia as class effects.

Immune system disorders
Rare: Hypersensitivity , anaphylactoid reaction and anaphylaxis.

Metabolism and nutrition disorders
Uncommon: Anorexia, weight changes.

Psychiatric disorders
Uncommon: Insomnia, depression.
Rare: Confusion, nervousness, agitation.

Nervous system disorders
Common: Mild and transient headache, dizziness.
Rare: Somnolence, paraesthesia, dysgeusia, tremor, migraine.
Very rare: Aseptic meningitis in patients with SLE and mixed connective tissue disorder (see 4.4).

Eye disorders
Uncommon: Conjuctivitis.
Rare: Visual disturbances.

Ear and labyrinth disorders
Uncommon: Vertigo, tinnitus.

Cardiac disorders
Uncommon: Palpitations, tachycardia, oedema, cardiac failure.

Vascular disorders
Uncommon: Flushing, oedema.
Rare: Hypertension, hot flush, haemorrhage, haematoma.

Respiratory, thoracic and mediastinal disorders
Uncommon: Rhinitis.
Rare: Dyspnoea, cough, bronchospasm.

Gastrointestinal disorders
Common: Nausea, abdominal pain, dyspepsia, diarrhoea, vomiting.
Uncommon: Constipation, flatulence, eructation, dry mouth, gastritis, gastric ulcer, abdominal pain upper, duodenal ulcer, mouth ulceration.
Rare: Melaena, haematemesis, stomatitis, oesophagitis, gastrooesophageal reflux, dysphagia, aphthous stomatitis, glossitis, perforated peptic ulcer, gastrointestinal haemorrhage.

Hepatobiliary disorders
Uncommon: Increase in liver function tests, SGPT (ALT) or SGOT (AST).
Very rare: Hepatotoxicity resulting in e.g. hepatic failure, hepatitis, jaundice and cholestasis.

Skin and subcutaneous tissue disorders
Uncommon: Rash, pruritus, hyperhidrosis, rash erythematous, urticaria and angioedema, alopecia.

Rare: Dermatitis and eczema, purpura.
Very rare: Oedema and bullous reactions, Stevens-Johnson syndrome, toxic epidermal necrolysis.

Musculoskeletal and connective tissue disorders
Uncommon: Arthralgia.
Rare: Bone pain, muscle spasms, myalgia.

Renal and urinary disorders
Rare: Nocturia, micturition disorders, increase in blood urea nitrogen and creatinine levels.
Very rare: Lornoxicam may precipitate acute renal failure in patients with pre-existing renal impairment, who are dependent on renal prostaglandins for maintenance of renal blood flow
(see 4.4). Nephrotoxicity in various forms including nephritis and nephrotic syndrome has been associated with NSAIDs as class effect.

General disorders and administration site conditions
Uncommon: Malaise, face oedema.
Rare: Asthenia.

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 HPRA Pharmacovigilance, Earlsfort Terrace, IRL - Dublin 2; Tel: +353 1 6764971; Fax: +353 1 6762517. Website: www.hpra.ie; E-mail: medsafety@hpra.ie.

4.9 Overdose

At this time, there is no experience of overdose to permit definition of the consequence of an overdose, or to suggest specific managements. However, it can be expected that after an overdose with lornoxicam, the following symptoms can be seen: Nausea, vomiting, cerebral symptoms (dizziness, disturbances in vision). Severe symptoms are ataxia ascending to coma and cramps, liver and kidney damages and maybe coagulation disorders.

In the case of a real or suspected overdose, the medicinal product should be withdrawn. Due to its short half-life, lornoxicam is rapidly excreted. Lornoxicam is not dialysable. No specific antidote is known to date. The usual emergency measures including gastric lavage should be considered. Based on principles, only administering activated charcoal immediately after the intake of lornoxicam can lead to diminished absorption of the preparation. Gastrointestinal disorders can for example be treated with a prostaglandin analogue or ranitidine.

5 PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic properties

Pharmacotherapeutic group: Antiinflammatory and antirheumatic products, non-steroids, oxicams ATC code: M01 AC05

Mechanism of action
Lornoxicam is a non-steroidal anti-inflammatory drug with analgesic properties and belongs to the class of oxicams. Lornoxicams mode of action is mainly related to the inhibition of the prostaglandin synthesis (inhibition of the cyclooxygenase enzyme) leading to desensitisation of peripheral nociceptors and consequently inhibition of inflammation. A central effect on nociception, which seems to be independent of anti-inflammatory effects has also been suggested.

Pharmacodynamic effects
Lornoxicam has no effect on vital signs (e.g. body temperature, respiratory rate, heart rate, blood pressure, ECG, spirometry).

Clinical efficacy and safety
The analgesic properties of lornoxicam have been demonstrated successfully in several clinical trials during development of the drug.

Due to a local gastrointestinal irritation and a systemic ulcerogenic effect related to the inhibition of prostaglandin (PG)-synthesis, gastrointestinal sequela are common undesirable effects after treatment with lornoxicam as seen with other NSAIDs.

In a clinical study in patients with pain after surgical removal of an impacted third molar lornoxicam Rapid film-coated tablets showed a faster onset of action compared to lornoxicam film-coated tablets.

5.2 Pharmacokinetic properties

Absorption
Lornoxicam is absorbed rapidly and almost completely from the gastrointestinal tract. Maximum plasma concentrations are achieved after approximately 30 minutes. The C_{max} for Xefo Rapid film-coated tablets is higher than C_{max} for Xefo film-coated tablets and equivalent to C_{max} for the parenteral formulation of lornoxicam. The absolute bioavailability of Xefo Rapid film-coated tablets is 90-100% which is equivalent to Xefo film-coated tablet. No first-pass effect has been observed. The mean elimination half-life is 3-4 hours.

No data are available on simultaneous intake of Xefo Rapid film-coated tablets with meals, but based on data for Xefo film-coated tablets a reduction of C_{max}, an increase in T_{max}, and a reduction in the absorption (AUC) of lornoxicam may be expected.

Distribution
Lornoxicam is found in the plasma in unchanged form and as its hydroxylated metabolite. The plasma protein binding of lornoxicam is 99% and not concentration dependent.

Biotransformation
Lornoxicam is extensively metabolised in the liver, primarily to the inactive 5–hydroxylornoxicam by hydroxylation. CYP2C9 is involved in this biotransformation of lornoxicam. Due to genetic polymorphism, slow and extensive metabolisers exist for this enzyme, which could result in markedly, increased plasma levels of lornoxicam in slow metabolisers. The hydroxylated metabolite exhibits no pharmacological activity. Lornoxicam is metabolised completely, and approximately 2/3 is eliminated via the liver and 1/3 via the kidneys as inactive substance.

When tested in animal models, lornoxicam did not induce liver enzymes. From clinical trial data there is no evidence of accumulation of lornoxicam after repeated administrations, when given according to recommended dosage. This finding was supported by drug monitoring data from one year studies.

Elimination
The mean elimination half-life of the parent compound is 3 to 4 hours. After oral administration about 50% is excreted in the faeces and 42% through the kidneys, mainly as 5-hydroxylornoxicam. The elimination half-life of 5-hydroxylornoxicam is about 9 hours after a parenteral single or twice daily dose.

In elderly patients above age 65, the clearance is reduced with 30-40%. Apart from reduced clearance, there is no significant change in the kinetic profile of lornoxicam in elderly patients.

There is no significant change in the kinetic profile of lornoxicam in patients with renal or hepatic failure, except for accumulation in patients with chronic liver disease after 7 days of treatment with daily doses of 12 and 16 mg.

5.3 Preclinical safety data

Non-clinical data reveal no special hazard for humans based on conventional studies of safety pharmacology, repeated dose toxicity, genotoxicity, and carcinogenic potential.

Lornoxicam caused renal toxicity and gastrointestinal ulceration single- and repeat-dose toxicity studies in several species.

In animals, administration of 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.

In rat, lornoxicam impaired fertility (effects on ovulation and implantation), and affected the pregnancy and delivery. In rabbit and rat, lornoxicam caused premature closure of the ductus arteriosus due to inhibition of cyclooxygenase.

6 PHARMACEUTICAL PARTICULARS

6.1 List of excipients

Core:
Cellulose, microcrystalline
Sodium hydrogen carbonate
Calcium hydrogen phosphate, anhydrous
Low substituted hydroxypropylcellulose
Hydroxypropylcellulose
Calcium stearate

Film:
Titanium dioxide (E171)
Talc
Propylene glycol
Hypromellose

6.2 Incompatibilities

Not applicable.

6.3 Shelf life

2 years.

6.4 Special precautions for storage

Do not store above 30°C.

6.5 Nature and contents of container

OPA-Alu-PVC/Alu blister.
Pack sizes: 6, 10, 20, 30, 50, 100, 250 film-coated tablets

Not all pack sizes may be marketed

6.6 Special precautions for disposal

No special requirements.

7 MARKETING AUTHORISATION HOLDER

Takeda UK Limited
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8 MARKETING AUTHORISATION NUMBER

PA 1547/005/004

9 DATE OF FIRST AUTHORISATION/RENEWAL OF THE AUTHORISATION

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10 DATE OF REVISION OF THE TEXT

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