SUMMARY OF PRODUCT CHARACTERISTICS

1.0 NAME OF THE MEDICINAL PRODUCT
RAGEZUGI TABLETS

(PARACETAMOL 500 mg & DICLOFENAC POTASSIUM 50 mg TABLETS)

2.0 QUALITATIVE AND QUANTITATIVE COMPOSITION:

Each film-coated tablet contains:
Paracetamol BP …… 500mg
Diclofenac Potassium BP …… 50mg
Excipients …… q.s
Colour: Fast Green F.C.F

3.0 PHARMACEUTICAL FORM:

Film coated tablet (caplets)

4.0 CLINICAL PARTICULARS

4.1 Therapeutic Indications:

Diclofenac Paracetamol Tablet is used for the treatment, control, prevention, & improvement of the following diseases, conditions and symptoms:

- Fever
- Joint pain
- Toothache
- Relief of the pain of osteoarthritis of knees and hands
- Sprains
- Ear pain
- Febrility
- Cephalalgia
- Headache
- Periods pain

4.2 Posology and Method of administration:

Per tab contains diclofenac K 50 mg and paracetamol 500 mg: 1 tab 3 times/day.

4.3 Contraindications

Hypersensitivity to the components of the formulation, peptic ulcers or GI bleeding.

4.4 Special warnings and precautions for use

Diclofenac General undesirable effects may be minimised by using the lowest effective dose for the shortest duration necessary to control symptoms (see section 4.2 and GI and cardiovascular risks below). The concomitant use of diclofenac with systemic NSAIDs including cyclooxygenase- 2 selective inhibitors should be avoided due to the absence of any evidence demonstrating synergistic benefits and the potential for additive undesirable effects. Caution is indicated in the elderly on the basis of medical grounds. In particular, it is recommended that the lowest effective dose be used in frail elderly patients or those with a low body weight. As with other NSAIDs, allergic reactions, including anaphylactic/anaphylactoid reactions, can also occur in rare cases with diclofenac without earlier exposure to the drug. Like other NSAIDs, Diclofenac may mask the signs and symptoms of infection due to its pharmacodynamic properties. Gastrointestinal effects: Gastrointestinal bleeding, ulceration and perforation, which can be fatal, has been reported with all NSAIDs, including diclofenac, and may occur at any time during treatment, with or without warning symptoms or a previous history of serious gastrointestinal events. They generally have more serious consequences in the elderly. If gastrointestinal bleeding or ulceration occurs in patients receiving Diclofenac, the medicinal product should be withdrawn. As with all NSAIDs, including Diclofenac, close medical surveillance is imperative and particular caution should be exercised when prescribing Diclofenac in patients with symptoms indicative of gastrointestinal (GI) disorders or with a history suggestive of gastric or intestinal ulceration, bleeding or perforation (see section 4.8). The risk of GI bleeding is higher with increasing NSAID doses and in patients with a history of ulcer, particularly if complicated with haemorrhage or perforation. The elderly have an increased frequency of adverse reactions to NSAIDs especially gastrointestinal bleeding and perforation which may be fatal. To reduce the risk of GI toxicity in patients with a history of ulcer, particularly if complicated with haemorrhage or perforation, and in the elderly, the treatment should be initiated and maintained at the lowest effective dose. Combination therapy with protective agents (e.g. proton pump inhibitors or misoprostol) should be considered for these patients, and also for patients requiring concomitant use of medicinal products containing low-dose acetylsalicylic acid (ASA)/aspirin or other medicinal

products likely to increase gastrointestinal risk. Patients with a history of GI toxicity, particularly the elderly, should report any unusual abdominal symptoms (especially GI bleeding). Caution is recommended in patients receiving concomitant medications which could increase the risk of ulceration or bleeding, such as systemic corticosteroids, anticoagulants, anti-platelet agents or selective serotonin-reuptake inhibitors (see section 4.5). Close medical surveillance and caution should be exercised in patients with ulcerative condition maybe co exacerbated (see section 4.8). Hepatic effects: Close medical surveillance is required when prescribing Diclofenac to patients with impaired hepatic function, as their condition may be exacerbated. As with other NSAIDs including diclofenac, values of one or more liver enzymes may increase. During prolonged treatment with Diclofenac, regular monitoring of hepatic function is indicated as a precautionary measure. If abnormal liver function tests persist or worsen, if clinical signs or symptoms consistent with liver disease develop, or if other manifestations occur (e.g. eosinophilia, rash), Diclofenac should bediscontinued. Hepatitis may occur with use of diclofenac without prodromal symptoms. Caution is called for when using Diclofenac in patients with hepatic porphyria, since it may trigger an attack. Renal effects: As fluid retention and oedema have been reported in association with NSAID therapy, including Diclofenac, particular caution is called for in patients with impaired cardiac or renal function, history of hypertension, the elderly, patients receiving concomitant treatment with diuretics or medicinal products that can significantly impact renal function, and in those patients with substantial extracellular volume depletion from any cause, e.g. before or after major surgery (see section 4.3). Monitoring of renal function is recommended as a precautionary measure when using Diclofenac in such cases. Discontinuation of therapy is usually followed by recovery to the pre-treatment state. Skin 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 higher risk for 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. Diclofenac should be discontinued at the first appearance of skin rash, mucosal lesions, or any other sign of hypersensitivity. Cardiovascular and cerebrovascular effects: 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 diclofenac (particularly at high doses, 150 mg daily and in long term treatment) may be associated with a small increased risk of arterial thrombotic

events (for example myocardial infarction or stroke). Patients with significant risk factors for cardiovascular events (e.g. hypertension, hyperlipidaemia, diabetes mellitus, smoking) should only be treated with diclofenac after careful consideration. As the cardiovascular risks of diclofenac may increase with dose and duration of exposure, the shortest duration possible and the lowest effective daily dose should be used. The patient's need for symptomatic relief and response to therapy should be reevaluated periodically.

Haematological Effects: During prolonged treatment with Diclofenac, as with other NSAIDs, monitoring of the blood count is recommended. Like other NSAIDs, Diclofenac may temporarily inhibit platelet aggregation. Patients with defects of haemostasis should be carefully monitored. Anaemia may occur as a result of water retention or effects on erythropoiesis. Consequently, it is advisable to monitor the levels of haemoglobin and haematocrit if symptoms of anaemia are detected. Hyperpotassemia may occur in diabetic patients or those who are also taking potassiumsparing drugs (see section 4.5). Pre-existing asthma: In patients with asthma, seasonal allergic rhinitis, swelling of the nasal mucosa (i.e. nasal polyps), chronic obstructive pulmonary diseases or chronic infections of the respiratory tract (especially if linked to allergic rhinitis-like symptoms), reactions on NSAIDs like asthma exacerbations (so-called intolerance to analgesics/analgesics asthma), Quincke'soedemaor urticaria are more frequent than in other patients. Therefore, special precaution is recommended in such patients (readiness for emergency). This is applicable as well for patients who are allergic to other substances, e.g. with skin reactions, pruritus or urticaria. SLE and mixed connective tissue disease: In patients with systemic lupus erythematosus (SLE) and mixed connective tissue disorders there may be an increased risk of aseptic meningitis (see section section 4.8).

Administration: Injections must be carried out following strict rules of asepsis and antisepsis. Duration of treatment Diclofenac must not be administered for longer than 2 days. After 2 days, the need for an alternative NSAID should be reviewed and if long-term treatment with an NSAID is required, patients should be monitored for evidence of renal and hepatic dysfunction and blood count abnormalities. This is particularly important in the elderly.

Paracetamol

Contains Benzyl Alcohol. This should not be administered to new born or infants. Paracetamol

should be given with care to patients with impaired kidney or liver function.

4.5 Interaction with other medicinal products and other forms of interaction

Diclofenac

The following interactions include those observed with Diclofenac gastro-resistant tablets and/or other pharmaceutical forms of diclofenac. Lithium: NSAIDs have been reported to increase blood lithium levels via decreased renal excretion of lithium. If this combination is considered necessary, lithium plasma concentrations should be monitored carefully during the initiation, adjustment and withdrawal of diclofenac treatment. Digoxin: If used concomitantly, diclofenac may raise plasma concentrations of digoxin. Monitoring of the serum digoxin level is recommended. Diuretics, ACE inhibitors and Angiotensin-II Antagonists: NSAIDs may reduce the antihypertensive effect of diuretics and other antihypertensive drugs (such as betablockers, angiotensin converting enzyme (ACE) inhibitors). 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 antagonists and agents that inhibit cyclo-oxygenase may result in further deterioration of renal function, including possible acute renal failure, which is usually reversible. 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 (see also section 4.4). Other NSAIDs, corticosteroids and acetylsalicylic acid: Concomitant administration of diclofenac and other systemic NSAIDs or corticosteroids or acetylsalicylic acid may increase the frequency of gastrointestinal undesirable effects (see section 4.4) and is not recommended. Anticoagulants and heparin (administered in the elderly or at curative doses): Caution is recommended since concomitant administration with NSAIDs could increase the risk of bleeding via inhibition of platelet function and damage to the gastroduodenal mucosa (see section 4.4). NSAIDs may enhance the effects of anticoagulants such as warfarin and heparin. Heparin is not recommended for administration to elderly patients or at curative doses. Careful monitoring of the international normalized ratio (INR) is required if co-administration cannot be avoided. Although clinical investigations do not appear to indicate that diclofenac affects the action of anticoagulants, there are isolated reports of an increased risk of haemorrhage in patients receiving diclofenac and anticoagulants concomitantly. Close monitoring of such patients is

therefore recommended. Thrombolytics and anti-platelet agents: Caution is recommended since concomitant administration with NSAIDs could cause increased risk of bleeding via inhibition of platelet function and damage to the gastro-duodenal mucosa. Selective serotoninreuptake inhibitors (SSRIs): Concomitant administration of systemic NSAIDs, includingdiclofenac, and SSRIs may increase the risk of gastrointestinal bleeding (see section 4.4). Anti-diabetics: Clinical studies have shown that diclofenac can be given together with oral anti-diabetic agents without influencing their clinical effect. However, there have been isolated reports of both hypoglycaemic and hyperglycaemic effects necessitating changes in the dosage of the anti-diabetic agents during treatment with diclofenac. For this reason, monitoring of the blood glucose level is recommended as a precautionary measure during concomitant therapy. Methotrexate: Diclofenac can inhibit the tubular renal clearance of methotrexate hereby increasing methotrexate levels. Caution is recommended when NSAIDs, including diclofenac, are administered less than 24 hours before or after treatment with methotrexate, since blood concentrations of methotrexate may rise and the toxicity of this substance be increased. Weekly blood count monitoring during the first few weeks of the combination is recommended. Monitoring should be increased in patients with impaired kidney function or in elderly subjects. Pemetrexed in patients with normal renal function, CLcr > 80 ml/min: Increased risk of pemetrexed toxicity due to decrease in pemetrexed clearance. Biological monitoring of renal function is recommended. Calcineurin inhibitors (e.g. Ciclosporin, tacrolimus): Nephrotoxicity of calcineurin inhibitors may be enhanced by NSAIDs via renal prostaglandin mediated effects. During combined treatment, monitoring of renal function is recommended, especially in the elderly. Deferasirox: The concomitant administration of NSAIDs and deferasirox may increase the risk of gastrointestinal toxicity. Close clinical monitoring should be performed when these drugs are combined. Quinolone anti-bacterials: There have been isolated reports of convulsions which may have been due to concomitant use of quinolones and NSAIDs. Phenytoin: When using phenytoin concomitantly with diclofenac, monitoring of phenytoin plasma concentrations is recommended due to an expected increase in exposure to phenytoin. Colestipol and cholestyramine: These agents can induce a delay or decrease in absorption of diclofenac. Therefore, it is recommended to administer diclofenac at least one hour before or 4 to 6 hoursafter administration of Colestipol/ cholestyramine. Potent CYP2C9 inhibitors: Caution is recommended when co-prescribing diclofenac with potent CYP2C9 inhibitors (such as sulfinpyrazone and voriconazole), which could result in a significant increase in peak plasma concentration and exposure to diclofenac due to inhibition of diclofenac metabolism.

Mifepristone: NSAIDs should not be used for 8 -12 days after mifepristone administration as NSAIDs can reduce the effect of mifepristone. Zidovudine: Increased risk of haematological toxicity when NSAIDs are given with zidovudine. There is evidence of an increased risk of haemarthroses and haematoma in HIV(+) haemophiliacs receiving concurrent treatment with zidovudine and ibuprofen. Despite being extensively bound to proteins, Diclofenac does not interfere with the protein binding of: salicylates, tolbutamide, and prednisolone.

Paracetamol

Paracetamol may enhance the activity of coumarin anticoagulants, but itseffect is not generally of clinical significance.

4.6 Pregnancy and lactation

Diclofenac

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, includingcardiovascular, have been reported in animals given a prostaglandin synthesis inhibitor during the organogenetic period. During the first and second trimester of pregnancy, diclofenac should not be given unless clearly necessary. If diclofenac 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. During the third trimester of pregnancy, all prostaglandin synthesis inhibitors may expose the foetus to: - cardiopulmonarytoxicity (with premature closure of the ductus arteriosus and pulmonary hypertension); - renal dysfunction, which may progress to renal failure with oligo-hydroamniosis - 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 and to inhibition of uterine contractions resulting in delayed or prolonged labour. Consequently, diclofenac is contraindicated during the third trimester of pregnancy

Lactation Like other NSAIDs, diclofenac passes into the breast milk in small amounts. Therefore, Diclofenac should not be administered during breast feeding in order to avoid undesirable effects in the infant. Fertility As with other NSAIDs, the use of diclofenac may impair female 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 diclofenac should be considered.

4.7 Effects on ability to drive and use machines

Patients experiencing visual disturbances, dizziness, vertigo, somnolence or other central nervous system disturbances while taking Diclofenac, should refrain from driving or using machines.

4.8 Adverse effects

The following is a list of possible side-effects that may occur from all constituting ingredients of Diclofenac Potassium & Paracetamol Tablet. This is not a comprehensive list. These side-effects are possible, but do not always occur. Some of the side-effects may be rare but serious. Consult your doctor if you observe any of the following side-effects, especially if they do not go away.

- Constipation
- Skin reddening
- Nausea
- Gas
- Diarrhea
- Headache
- Allergic reactions
- Acute renal tubular necrosis
- Dizziness
- Cephalalgia
- Diclofenac Potassium & Paracetamol Tablet may also cause side-effects not listed here.

4.9 Overdose

Diclofenac Symptoms

There is no typical clinical picture resulting from diclofenac over dosage. Overdosage can cause symptoms such as vomiting, gastrointestinal haemorrhage,diarrhoea, dizziness, tinnitus or convulsions. In the event of significant poisoning, acute renal failure and liver damage are possible. Therapeutic measures Management of acute poisoning with NSAIDs, including diclofenac, essentially consists of supportive measures and symptomatic treatment. Supportive measures and symptomatic treatment should be given for complications such as hypotension, renal failure, convulsions, gastrointestinal disorder, and respiratory depression. Special measures such as forced diuresis, dialysis or haemo-perfusion are probably of no help in eliminating NSAIDs, including diclofenac, due to the high protein binding and extensive metabolism.

Paracetamol

Symptoms of overdosage may include nausea, vomiting, abdominal pain, diaphoresis, generalized weakness & lethargy. If an overdose of Paracetamol is suspected, blood should be withdrawn immediately for Paracetamol plasma assay, without regard to the presence or absence of symptomatology. The acute hepatotoxicity, nephrotoxicity of paracetamol can be overcome by the administration of sulfinydryl donors, e.g, N- acetylcysteine which should be given as soon as possible after ingestion. Treatment after 12 hours is not effective. Paracetamol overdose should be treated with gastric lavage if the patient is seen within 24 hours of ingestion of the drug.

5. PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic Properties

Diclofenac

Pharmacotherapeutic category: non-steroidal anti-inflammatory drugs (NSAIDs):

It is therapeutic sub group classification: musculo-skeletal system/anti-inflammatory and anti-rheumatic products/ non-steroids/acetic acid derivatives and related substances Mechanism of action: Diclofenac as Injection is a nonsteroidal agent with marked analgesic/ antiinflammatory

properties. It is an inhibitor of prostaglandin synthetase, (cyclooxygenase).Diclofenac Potassium in vitro does not suppress proteoglycan biosynthesis in cartilage at concentrations equivalent to the concentrations reached in human beings. When used concomitantly with opioids for the management of post-operative pain, diclofenac Potassium often reduces the need for opioids. Clinical efficacy: The analgesic efficacy of Diclofenac 25, 50 and 75 mg injection was evaluated in two pivotal dental pain studies. Patients with moderate to severe pain following dental impaction surgery were included in these studies. In one study the analgesic efficacy of Diclofenac 25, 50 and 75 mg/ml subcutaneously administered was compared to placebo. Diclofenac at all strengths produced a statistically significant higher pain relief (as measured on the VAS) compared to placebo.

Paracetamol

Pharmacotherapeutic group: Other Analgesics and Antipyretics Paracetamol is a clinically proven analgesic and antipyretic. Paracetamol may act predominantly by inhibiting prostaglandin synthesis in the central nervous system and to a lesser extent through a peripheral action by blocking pain impulse generation. The peripheral action may also be due to inhibition of prostaglandin synthesis or inhibition of the synthesis or actions of other substances that sensitize pain receptors to mechanical or chemical stimulation. Paracetamol produces antipyresis by acting centrally on the hypothalamic heat-regulating center to produce peripheral vasodilation resulting in increased blood flow through the skin, sweating and heat loss. The central action probably involves inhibition of prostaglandin synthesis in thehypothalamus.

5.2 Pharmacokinetic properties

Diclofenac

Absorption

Intramuscular injection: After administration of Diclofenac 75 mg/ml as injection by the i.m. route, absorption is rapid and the mean peak ug/ml equals approximately 8 μmol/L)reached is after 34 minutes.

The area under the concentration curve AUC0-t is 250.07 ± 46.89 μg/ml.min. I mean peak

plasma concentration for intramuscular Diclofenac (75mg/3ml) is 2.242 ± 0.566 µg/ml which is reached - afteris246.70 ±27 39minutes. 74µg/mland AUC after i.m. administration is about twice as large as it is following oral or rectal administration as this route avoids "firstpass"metabolism.

Subcutaneous injection:

After administration of Diclofenac 75 mg/ml as injection by the s.c. route,absorption is rapid and the mean peak plasma concentrations of 2.138 ± 0.646 µg/ml(2.5 µg/mlequals Approximately 8 µmol/l) is-t reachedis261.94in ±4053min.2

In comparative clinical studies the mean peak plasma concentration for intramuscular Voltarol

is 2.242 ± 0.566 µg/mlat27 minutes and the AUC0-t is 246.70 ± 39.74 µg/mlA.min. subcutaneous dose of 75 mg of Diclofenac was bioequivalent to an intramuscularly administered dose of Voltarol 75 mg/3 ml in terms of AUC and Cmax. The AUC after subcutaneous administration is about twice as large as it is following oral or rectal administration as this route avoids "first-pass"metabolism. Dose linearity in terms of AUC has been demonstrated for diclofenac absorbed after subcutaneous administration. Cmax was found to be not proportional to dose, withmean Cmax values of 1090 ng/ml, 1648.9 ng/ml and 1851.1 ng/ml with the 25 mg, 50 mg and 75 mg dose of Diclofenac respectively.

Distribution

The active substance is 99.7% protein bound, mainly to albumin (99.4%). Diclofenac enters the synovial fluid, where maximum concentrations are measured 2-4 hours after the peak plasma values have been attained. The apparent half-life for elimination from the synovial fluid is 3-6 hours. Two hours after reaching the peak plasma values, concentrations of the active substance are already higher in the synovial fluid than they are in the plasma and remain higher for up to 12 hours.

Biotransformation

Biotransformation of diclofenac takes place partly by glucuronidation of the intact molecule, but mainly by single and multiple hydroxylation and methoxylation, resulting in several phenolic metabolites, most of which are converted to glucuronide conjugates. Two phenolic metabolites are biologically active, but to a much lesser extent than diclofenac.

Elimination

Total systemic clearance of diclofenac in plasma is 263 ± 56 mL/min (mean value ±SD). The terminal half-life in plasma is 1-2 hours. Four of the metabolites, including the two active ones, also have short plasma half-lives of 1-3 hours. About 60% of the administered dose is excreted in the urine in the form of the glucuronide conjugate of the intact molecule and as metabolites, most of which are also converted to glucuronide conjugates. Less than 1% is excreted as unchanged substance. The rest of the dose is eliminated as metabolites throughthe bile in the faeces.

Characteristics in patients Elderly:

No relevant age-dependent differences in the drug's absorption, metabolism or excretion have been observed. Patients with renal impairment: In patients suffering from renal impairment, no accumulation of the unchanged active substance can be inferred from the singledose kinetics when applying the usual dosage schedule. At a creatinine clearance of Paracetamol.

Paracetamol is distributed throughout most body tissues. About 25% of Paracetamol in blood is bound to plasma proteins. The plasma half-life is 1.25 to 3 hours but may be increased by liver damage and following overdose. Paracetamol is metabolized in the liver. About 85% of a dose of Paracetamol is excreted in urine as free and conjugated Paracetamol within24 hours.

5.3 Preclinical safety data

Diclofenac

No new preclinical safety studies have been performed on Potassium diclofenac. The safety profile of the medicinal product is well-established. The local tolerance study demonstrated that the formulation does not present any significant unexpected local 16th May 2016 Diclofenac & Paracetamol 19 toxicity by either the intramuscular or subcutaneous routes of administration.
Paracetamol

Not available

6. Pharmaceutical particulars

6.1 List of excipients

Ingredients (In Order Of Mixing)	Specification
Maize Starch	BP
Calcium Carbonate	USP
Di basic calcium Phosphate	BP
PASTE PREPARATION	
Maize Starch BP	BP
Methyl Paraben BP	BP
Propyl Paraben BP	BP
P.V. P- K 30 BP	BP
Gelatin	BP
Purified Water	BP

LUBRICATION

Ingredients (In Order Of Mixing)	Specification
Purified Talcum	BP
Magnesium Stearate	BP
Aerosil	BP
Potassium Lauryl Sulphate	BP
Sod. Starch Glycolate	BP
Cross Carmellose Potassium	BP

COATING MATERIAL

Ingredients	Specification
Red Mix Colour Fast Green F.C.F	In house
I.P.A	BP
Methylene dichloride (MDC)	BP
Purified Water	BP

6.2 Incompatibilities

Not applicable.

6.3 Shelf life

3 years from date of manufacture

6.4 Special precautions for storage

Store below 30°C.

Protect from light and moisture.

6.5 Nature and contents of container

10 tablets packed in one blister. Such 10 blister packed in unit printed duplex board carton along with its package insert. Such cartons packed in export worthy shipper.

6.6 Special precautions for disposal and other handling

No special requirements.

7.0 Manufactured by:

JIANGSU RUINIAN QUANJIN PHARM. CO. LTD.
Chuanbu Village, Dingshu Town, Yixing City,
Jiangsu Provide China.

8.0 Marketed by:

FRESHBORN INDUSTRIES LTD.
Plot 18, Jesus Estate, Ijegun Egba,
Satellite Town,
Lagos State,
Nigeria.