1 PRODUCT NAME
XIFAXAN®

2 QUALITATIVE AND QUANTITATIVE COMPOSITION
Rifaximin 550 mg Film-coated Tablets

3 PHARMACEUTICAL FORM
XIFAXAN tablets are oval biconvex pink film-coated tablets containing rifaximin 550 mg, marked RX on one side.

4 CLINICAL PARTICULARS
4.1 Therapeutic indications
Prevention of the recurrence of hepatic encephalopathy where other treatments have failed or are contraindicated.

4.2 Dose and method of administration
The recommended dose of XIFAXAN is one 550 mg tablet taken orally twice a day, with or without food.

In the pivotal trial of XIFAXAN for HE, 91% of the patients were using lactulose concomitantly.

Because of the limited systemic absorption of rifaximin, no specific dosing adjustment is recommended for patients with hepatic insufficiency.

4.3 Contraindications
XIFAXAN is contraindicated in patients with a hypersensitivity to rifaximin, any of the rifamycin antimicrobial agents, or any of the components in XIFAXAN. Hypersensitivity reactions have included exfoliative dermatitis, angioneurotic oedema, and anaphylaxis.

Cases of intestinal obstruction.

4.4 Special warnings and precautions for use
Use with P-glycoprotein inhibitors
Caution should be exercised when concomitant use of rifaximin and a P-glycoprotein inhibitor such as ciclosporin is needed (see Interactions).

Use with warfarin
Both decreases and increases in international normalized ratio (in some cases with bleeding events) have been reported in patients maintained on warfarin and prescribed rifaximin. If co-administration is necessary, the international normalized ratio should be carefully monitored with the addition or withdrawal of rifaximin. Adjustments in the dose of oral anticoagulants may be necessary (see ‘INTERACTIONS WITH OTHER MEDICINES’).

Paediatric use
The safety and effectiveness of XIFAXAN for the prevention of recurrence of hepatic encephalopathy have not been established in patients under 18 years of age.

Use in the elderly

XIFAXAN® 550 mg  Data Sheet September 2013
In the controlled trial with XIFAXAN 550 mg for hepatic encephalopathy, 19.4% were aged 65 years and over, while 2.3% were 75 and over. No overall differences in safety or effectiveness were observed between these subjects and younger subjects, and other reported clinical experience has not identified differences in responses between the elderly and younger patients, but greater sensitivity of some older individuals cannot be ruled out.

**Genotoxicity**
Rifaximin was not genotoxic in the bacterial reverse mutation assay, chromosomal aberration assay, rat bone marrow micronucleus assay, rat hepatocyte unscheduled DNA synthesis assay, or the CHO/HGPRT mutation assay.

**Carcinogenicity**
The carcinogenic potential of rifaximin was examined in a 2 year study with CD rats. Oral administration at doses up to 250 mg/kg/day (about twice the MRHD based on body surface area) produced no evidence of a carcinogenic effect except for an increased trend in malignant schwannomas in the heart in males but not females, at an incidence (5%) exceeding the maximum historical control incidence (1.7%). Despite lack of statistical significance of pairwise testing and absence of this finding in females, a possible relationship to treatment cannot be dismissed.

There was no increase in tumours in Tg.rasH2 mice treated orally with rifaximin for 26 weeks at doses up to 2000 mg/kg/day (mean plasma concentrations 2-5 times the clinical Cmax in healthy volunteers, less than clinical exposure in hepatically impaired patients).

**Clostridium difficile-Associated Diarrhoea**
*Clostridium difficile*-associated diarrhoea (CDAD) has been reported with use of nearly all antibacterial agents, including XIFAXAN, and may range in severity from mild diarrhoea to fatal colitis. Treatment with antibacterial agents alters the normal flora of the colon which may lead to overgrowth of *C. difficile*.

*C. difficile* produces toxins A and B which contribute to the development of CDAD. Hypertoxin producing strains of *C. difficile* cause increased morbidity and mortality, as these infections can be refractory to antimicrobial therapy and may require colectomy. CDAD must be considered in all patients who present with diarrhoea following antibiotic use. Careful medical history is necessary since CDAD has been reported to occur over two months after the administration of antibacterial agents.

If CDAD is suspected or confirmed, ongoing antibiotic use not directed against *C. difficile* may need to be discontinued. Appropriate fluid and electrolyte management, protein supplementation, antibiotic treatment of *C. difficile*, and surgical evaluation should be instituted as clinically indicated.

**Renal Impairment**
No clinical data are available on the use of rifaximin in patients with impaired renal function.

**Severe (Child-Pugh C) hepatic impairment**
There is increased systemic exposure in patients with hepatic impairment. The clinical trials were limited to patients with MELD scores <25. Therefore, caution should be exercised when administering XIFAXAN to patients with severe hepatic impairment (Child-Pugh C).

**Development of drug resistant bacteria**
Resistant strains of bacteria including *Staphylococcus aureus* are more likely to develop if patients are exposed to XIFAXAN long term. It is likely that strains resistant to rifaximin will also be resistant to rifampicin. Therefore XIFAXAN is not recommended for use in patients at
low risk for development of further episodes of HE or who have a satisfactory response to alternative treatments.

4.5 Interaction with other medicines and other forms of interaction
There is no experience regarding administration of rifaximin to subjects who are taking another rifamycin antibacterial agent to treat a systemic bacterial infection.

In vitro studies have shown that rifaximin did not inhibit cytochrome P450 isozymes 1A2, 2A6, 2B6, 2C9, 2C19, 2D6, 2E1 and CYP3A4 at concentrations up to 200 ng/mL (at least 10 times the clinical C\text{max}). Rifaximin is not expected to inhibit these enzymes in clinical use.

In healthy subjects, clinical drug interaction studies demonstrated that rifaximin did not significantly affect the pharmacokinetics of CYP3A4 substrates, however, in hepatic impaired patients it cannot be excluded that rifaximin may decrease the exposure of concomitant CYP3A4 substrates administered (e.g. warfarin, antiepileptics, antiarrhythmics and oral contraceptives), due to the higher systemic exposure with respect to healthy subjects.

An in vitro study suggested that rifaximin is a moderate substrate of P-glycoprotein (P-gp) and metabolized by CYP3A4. It is unknown whether concomitant drugs which inhibit CYP3A4 can increase the systemic exposure of rifaximin.

In healthy subjects, co-administration of a single dose of ciclosporin (600 mg), a potent P-glycoprotein inhibitor, with a single dose of rifaximin (550 mg) resulted in 83-fold and 124-fold increases in rifaximin mean C\text{max} and AUC. The clinical significance of this increase in systemic exposure is unknown.

Both decreases and increases in international normalized ratio have been reported in patients maintained on warfarin and prescribed rifaximin. If co-administration is necessary, the international normalized ratio should be carefully monitored with the addition or withdrawal of rifaximin. Adjustments in the dose of oral anticoagulants may be necessary.

The potential for drug-drug interactions to occur at the level of transporter systems has been evaluated in vitro and these studies suggest that a clinical interaction between rifaximin and other compounds that undergo efflux via P-gp and other transport proteins is unlikely (MRP2, MRP4, BCRP and BSEP).

There is no experience regarding administration of rifaximin to subjects who are taking another rifamycin antibacterial agent to treat a systemic bacterial infection.

4.6 Fertility, pregnancy and lactation
Effects on fertility
There were no effects on fertility in rats treated with rifaximin at oral doses up to 300 mg/kg/day (about 2.5 times the MRHD based on body surface area).

Use in pregnancy
Pregnancy Category B1
Nonclinical studies of placental transfer of rifaximin/metabolites have not been conducted. There was no evidence of teratogenicity in pregnant rats or rabbits treated with rifaximin during the period of organogenesis at respective oral doses up to 300 and 1000 mg/kg/day. The dose in rats was about 2.5 times the MRHD based on body surface area. Compared with clinical exposure (plasma AUC) at the MRHD, the exposure in rabbits was slightly greater than that in healthy volunteers but less than that in hepatically impaired patients.

Use in lactation
It is unknown whether rifaximin/metabolites are excreted in human milk. A risk to the child cannot be excluded. A decision must be made whether to discontinue breast-feeding or to discontinue/abstain from rifaximin therapy taking into account the benefit of breast feeding for the child and the benefit of therapy for the woman.

Oral administration of rifaximin to rats from early gestation to weaning at doses up to 300 mg/kg/day (about 2.5 times the MRHD based on body surface area) did not elicit any adverse effects on gestation or parturition, or on offspring viability, development and reproductive performance.

4.7 Effects on ability to drive and use machines
Dizziness and somnolence have been reported in clinical controlled trials. However, rifaxamin has negligible influence on the ability to drive and use machines.

4.8 Undesirable effects
The data described below reflect exposure to XIFAXAN 550 mg in 336 patients, including 257 exposed at least for 6 months and 114 exposed for more than a year (mean exposure was 274 days). The safety of XIFAXAN 550 mg taken two times a day for the maintenance of remission from hepatic encephalopathy recurrence in adult patients was evaluated in a 6-month placebo controlled clinical trial (n = 140) and in a long term follow-up study (n = 266). The population studied had a mean age of 56.5 (range: 21-82) years; approximately 20% of the patients were ≥ 65 years old, 58% were male, 89% were white, and 4.5% were black. Ninety-one percent of patients in the trial were taking lactulose concomitantly. All adverse events that occurred at an incidence ≥ 5% and at a higher incidence in XIFAXAN 550 mg-treated subjects than in the placebo group in the 6-month trial are provided in Table 1. (These include adverse events that may be attributable to the underlying disease).

<table>
<thead>
<tr>
<th>MedDRA System Organ Class</th>
<th>Event</th>
<th>Rifaximin N= 140 n (%)</th>
<th>Placebo N=159 n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood and lymphatic system disorders</td>
<td>Anaemia</td>
<td>11 (7.9)</td>
<td>6 (3.8)</td>
</tr>
<tr>
<td>Gastrointestinal disorders</td>
<td>Ascites</td>
<td>16 (11.4)</td>
<td>15 (9.4)</td>
</tr>
<tr>
<td></td>
<td>Nausea</td>
<td>20 (14.3)</td>
<td>21 (13.2)</td>
</tr>
<tr>
<td></td>
<td>Abdominal pain</td>
<td>12 (8.6)</td>
<td>13 (8.2)</td>
</tr>
<tr>
<td></td>
<td>Abdominal distension</td>
<td>11 (7.9)</td>
<td>12 (7.5)</td>
</tr>
<tr>
<td></td>
<td>Abdominal pain upper</td>
<td>9 (6.4)</td>
<td>8 (5.0)</td>
</tr>
<tr>
<td></td>
<td>Constipation</td>
<td>9 (6.4)</td>
<td>10 (6.3)</td>
</tr>
<tr>
<td>General disorders and administration site conditions</td>
<td>Fatigue</td>
<td>17 (12.1)</td>
<td>18 (11.3)</td>
</tr>
<tr>
<td></td>
<td>Oedema peripheral</td>
<td>21 (15.0)</td>
<td>13 (8.2)</td>
</tr>
<tr>
<td></td>
<td>Pyrexia</td>
<td>9 (6.4)</td>
<td>5 (3.1)</td>
</tr>
<tr>
<td>Infections &amp; infestations</td>
<td>Nasopharyngitis</td>
<td>10 (7.1)</td>
<td>10 (6.3)</td>
</tr>
<tr>
<td>Musculoskeletal and connective tissue disorders</td>
<td>Muscle spasms</td>
<td>13 (9.3)</td>
<td>11 (6.9)</td>
</tr>
<tr>
<td></td>
<td>Arthralgia</td>
<td>9 (6.4)</td>
<td>4 (2.5)</td>
</tr>
<tr>
<td></td>
<td>Back pain</td>
<td>9 (6.4)</td>
<td>10 (6.3)</td>
</tr>
<tr>
<td>Nervous system disorders</td>
<td>Dizziness</td>
<td>18 (12.9)</td>
<td>13 (8.2)</td>
</tr>
<tr>
<td>Psychiatric disorders</td>
<td>Insomnia</td>
<td>10 (7.1)</td>
<td>11 (6.9)</td>
</tr>
<tr>
<td></td>
<td>Depression</td>
<td>10 (7.1)</td>
<td>8 (5.0)</td>
</tr>
<tr>
<td>Respiratory, thoracic and mediastinal disorders</td>
<td>Cough</td>
<td>10 (7.1)</td>
<td>11 (6.9)</td>
</tr>
<tr>
<td></td>
<td>Dyspnoea</td>
<td>9 (6.4)</td>
<td>7 (4.4)</td>
</tr>
<tr>
<td>Skin and subcutaneous tissue disorders</td>
<td>Pruritus</td>
<td>13 (9.3)</td>
<td>10 (6.3)</td>
</tr>
<tr>
<td></td>
<td>Rash</td>
<td>7 (5.0)</td>
<td>6 (3.8)</td>
</tr>
</tbody>
</table>
The following adverse events, presented by body system, have also been reported in the placebo-controlled clinical trial in greater than 2% but less than 5% of patients taking XIFAXAN 550 mg orally two times a day for hepatic encephalopathy. The following includes adverse events occurring at a greater incidence than placebo.

Table 2 – Adverse events occurring in more than 2% and less than 5% in patients receiving XIFAXAN and at a higher incidence than placebo

<table>
<thead>
<tr>
<th>MedDRA System Organ Class</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ear and Labyrinth Disorders:</td>
<td>Vertigo</td>
</tr>
<tr>
<td>Gastrointestinal Disorders:</td>
<td>Abdominal pain lower, abdominal tenderness, dry mouth, oesophageal variceal bleed, stomach discomfort</td>
</tr>
<tr>
<td>General Disorders and Administration Site Conditions:</td>
<td>Chest pain, generalized oedema, influenza-like illness, pain NOS</td>
</tr>
<tr>
<td>Infections and Infestations:</td>
<td>Cellulitis, pneumonia, rhinitis, upper respiratory tract infection NOS</td>
</tr>
<tr>
<td>Injury, Poisoning and Procedural Complications:</td>
<td>Contusion, fall, procedural pain</td>
</tr>
<tr>
<td>Investigations:</td>
<td>Weight increased</td>
</tr>
<tr>
<td>Metabolic and Nutritional Disorders:</td>
<td>Anorexia, dehydration, hyperglycaemia, hyperkalaemia, hypoglycaemia, hyponatraemia</td>
</tr>
<tr>
<td>Musculoskeletal, Connective Tissue, and Bone Disorders:</td>
<td>Myalgia, pain in extremity</td>
</tr>
<tr>
<td>Nervous System Disorders:</td>
<td>Amnesia, disturbance in attention, hypoesthesia, memory impairment, tremor</td>
</tr>
<tr>
<td>Psychiatric Disorders</td>
<td>Confusional state</td>
</tr>
<tr>
<td>Respiratory, Thoracic, and Mediastinal Disorders:</td>
<td>Hypotension</td>
</tr>
</tbody>
</table>

Post-marketing Experience

The following adverse reactions have been identified during post approval use of rifaximin. The frequency of these reactions is not known (cannot be estimated from the available data).

Table 3 - Adverse reactions occurring at unknown frequency in patients receiving rifaximin in post-marketing experience

<table>
<thead>
<tr>
<th>MedDRA System Organ Class</th>
<th>MedDRA Preferred Term</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infections and infestations</td>
<td>Clostridial infections (C. difficile)</td>
</tr>
<tr>
<td>Blood and lymphatic system disorder</td>
<td>Thrombocytopenia</td>
</tr>
<tr>
<td>Immune system disorders</td>
<td>Anaphylactic responses, Angioedemas, Hypersensitivity</td>
</tr>
<tr>
<td>Nervous system disorders</td>
<td>Presyncope, Syncope</td>
</tr>
<tr>
<td>Hepatobiliary disorders</td>
<td>Liver function tests abnormalities</td>
</tr>
<tr>
<td>Skin and subcutaneous tissue disorders</td>
<td>Dermatitis, eczema, Erythemas, Pruritus NEC, Purpura, Urticarias</td>
</tr>
<tr>
<td>Investigations</td>
<td>International normalised ratio abnormalities</td>
</tr>
</tbody>
</table>
4.9 Overdose

No specific information is available on the treatment of overdosage with XIFAXAN.

In clinical trials with patients suffering from traveller’s diarrhoea doses of up to 1,800 mg/day have been tolerated without any severe clinical sign. Even in patients/subjects with normal bacterial flora, rifaximin in dosages of up to 2,400 mg/day for 7 days did not result in any relevant clinical symptoms related to the high dosage.

In case of accidental overdosage, symptomatic treatments and supportive care are suggested. For information on the management of overdose, contact the National Poisons Centre on 0800 764 766.

5 PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic properties

Pharmacotherapeutic group: intestinal, anti-infective - antibiotics - Class ATC A07AA11

Rifaximin is a non-aminoglycoside semi-synthetic, non-systemic antibiotic derived from rifamycin SV.

Rifaximin acts by binding to the beta-subunit of bacterial DNA-dependent RNA polymerase resulting in inhibition of bacterial RNA synthesis. Rifaximin has a broad antimicrobial spectrum against most of the Gram-positive and Gram-negative, aerobic and anaerobic bacteria responsible for intestinal infections.

Due to the very low absorption from the gastrointestinal tract, rifaximin is locally acting in the intestinal lumen and clinically not effective against invasive pathogens, even though these bacteria are susceptible in vitro.

In the prevention of recurrent hepatic encephalopathy, rifaximin is thought to have an effect on the gastrointestinal flora.

Clinical Trials

The efficacy of XIFAXAN 550 mg taken orally twice a day was evaluated in a randomised, placebo-controlled, double-blind, multi-centre 6-month trial of adult subjects from the U.S.A., Canada and Russia who were defined as being in remission (Conn score of 0 or 1) from hepatic encephalopathy (HE). Eligible subjects had ≥ 2 episodes of HE associated with chronic liver disease in the previous 6 months. A total of 299 subjects were randomized to receive either XIFAXAN (n = 140) or placebo (n = 159). Patients had a mean age of 56 years (range, 21-82 years), 81% < 65 years of age, 61% were male and 86% were white. At baseline, 67% of patients had a Conn score of 0 and 68% had an asterixis grade of 0. Patients had MELD scores of either ≤ 10 (27%) or 11 to 18 (64%) at baseline. No patients were enrolled with a MELD score of > 25. Lactulose was concomitantly used by 91% of the patients in each treatment arm of the study. Per the study protocol, patients were withdrawn from the study after experiencing a breakthrough HE episode. Other reasons for early study discontinuation included: adverse reactions (XIFAXAN 6%; placebo 4%), patient request to withdraw (XIFAXAN 4%; placebo 6%) and other (XIFAXAN 7%; placebo 5%).
The primary endpoint was the time to first breakthrough overt HE episode. A breakthrough overt HE episode was defined as a marked deterioration in neurological function and an increase of Conn score to Grade ≥ 2. In patients with a baseline Conn score of 0, a breakthrough overt HE episode was defined as an increase in Conn score of 1 and asterixis grade of 1.

Breakthrough overt HE episodes were experienced by 31 of 140 subjects (22%) in the XIFAXAN group and by 73 of 159 subjects (46%) in the placebo group during the 6 month treatment period. Comparison of Kaplan-Meier estimates of event-free curves showed XIFAXAN significantly reduced the risk of HE breakthrough by 58% during the 6-month treatment period.

Presented below in Figure 1 is the Kaplan-Meier event-free curve for all subjects (n = 299) in the study.

When the results were evaluated by the following demographic and baseline characteristics, the treatment effect of XIFAXAN 550 mg in reducing the risk of breakthrough overt HE recurrence was consistent for: sex, baseline Conn score, duration of current remission and diabetes. The differences in treatment effect could not be assessed in the following sub-populations due to small sample size: non-white (n = 42), baseline MELD > 19 (n = 26), and those without concomitant lactulose use (n = 26).

HE-related hospitalizations (hospitalizations directly resulting from HE, or hospitalizations complicated by HE) were reported for 19 of 140 subjects (14%) and 36 of 159 subjects (23%) in the XIFAXAN and placebo groups respectively. Comparison of Kaplan-Meier estimates of event-free curves showed XIFAXAN significantly reduced the risk of HE-related hospitalizations by 50% during the 6-month treatment period. Comparison of Kaplan-Meier estimates of event-free curves is shown in Figure 2.
Pharmacokinetic properties

Absorption
Pharmacokinetic studies in rats, dogs and humans demonstrated that after oral administration, rifaximin in the polymorph α form is virtually not absorbed (less than 1%). After a single dose and multiple doses of rifaximin 550 mg in healthy subjects, the mean time to reach peak plasma concentrations was about an hour. The pharmacokinetic (PK) parameters were highly variable and the accumulation ratio based on AUC was 1.37.

After repeated administration of therapeutic doses of rifaximin in healthy volunteers and patients with damaged intestinal mucosa (inflammatory bowel disease), plasma levels are negligible (less than 10 ng/mL). A clinically not relevant increase of rifaximin systemic absorption was observed when administered within 30 minutes of a high-fat breakfast.

In patients with hepatic encephalopathy (HE), rifaximin mean peak plasma concentrations of 13.5 ng/mL were detected after administration of 800 mg three times a day for 7 days. Less than 0.1% of the administered dose was recovered after 7 days.

The PK of rifaximin in patients with a history of HE was evaluated after administration of XIFAXAN 550 mg two times a day. The PK parameters were associated with a high variability and mean rifaximin exposure (AUCτ) in patients with a history of HE (147 ng•h/mL) was approximately 12-fold higher than that observed in healthy subjects following the same dosing regimen (12.3 ng•h/mL). When PK parameters were analysed based on Child-Pugh Class A and B, the mean AUCτ was 10 and 13-fold higher, respectively, compared to that in healthy subjects.

Distribution
Rifaximin is moderately bound to human plasma proteins. In vivo, the mean protein binding ratio was 67.5% in healthy subjects and 62% in patients with hepatic impairment when XIFAXAN 550 mg was administered.
Metabolism
A mass balance study carried out in healthy volunteers (see ‘Excretion’) suggests that absorbed rifaximin undergoes metabolism with minimal renal excretion of the unchanged drug. The enzymes responsible for metabolizing rifaximin are unknown.

Excretion
Rifaximin is almost exclusively excreted in faeces. In a mass balance study, after administration of 400 mg $^{14}$C-rifaximin orally to healthy volunteers, of the 96.94% total recovery, 96.62% of the administered radioactivity was recovered in faeces almost exclusively as the unchanged drug and 0.32% was recovered in urine mostly as metabolites with 0.03% as the unchanged drug. Rifaximin accounted for 18% of radioactivity in plasma.

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

In a rat embryofoetal development study, a slight and transient delay in ossification that did not affect the normal development of the offspring, was observed at 300 mg/kg/day. In the rabbit, following oral administration of rifaximin during gestation, an increase in the incidence of skeletal variations was observed.

The clinical relevance of these findings is unknown.

6 PHARMACEUTICAL PARTICULARS

6.1 List of excipients
Tablet core:
Sodium starch glycolate type A
Glycerol distearate
Colloidal anhydrous silica
Talc
Microcrystalline cellulose

Tablet coating:
Hyromellose,
Titanium dioxide E171
Disodium edetate
Propylene glycol
Red iron oxide E172

6.2 Incompatibilities
None known.

6.3 Shelf life
Shelf life is 36 months (3 years) from manufacture.

6.4 Special precautions for storage
Store below 25ºC.

6.5 Nature and contents of container
The tablets are packaged in PVC/PE/PVDC/Aluminium blisters in cartons containing 14, 28, 30, 56, or 60 tablets. (Not all pack sizes may be marketed).
6.6 Special precautions for disposal
No special precautions required.

7 MEDICINE SCHEDULE
Prescription Medicine

8 SPONSOR
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Fax: (09) 918 5101

9 DATE OF FIRST APPROVAL
1 August 2013

10 DATE OF REVISION OF THE TEXT
13 September 2017
### SUMMARY TABLE OF CHANGES

<table>
<thead>
<tr>
<th>Section changed</th>
<th>Summary of new information</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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</table>