Information for the patient

VIMPAT® 10 mg/ml
Solution for infusion
Lacosamide

1. NAME OF THE MEDICINAL PRODUCT
Vimpat 10 mg/ml solution for infusion

2. QUALITATIVE AND QUANTITATIVE COMPOSITION
Each ml of solution for infusion contains 10 mg lacosamide.
Each vial of 20 ml solution for infusion contains 200 mg lacosamide.

Excipients with known effect:
Each ml of solution for infusion contains 2.99 mg sodium.
For the full list of excipients, see section 6.1

3. PHARMACEUTICAL FORM
Solution for infusion.
Clear, colourless solution.

4. CLINICAL PARTICULARS

4.1 Therapeutic indications
Vimpat is indicated as adjunctive therapy in the treatment of partial-onset seizures with or without secondary generalisation in adult and adolescent (16-18 years) patients with epilepsy.

4.2 Posology and method of administration
Posology
Lacosamide therapy can be initiated with either oral or i.v. administration. Solution for infusion is an alternative for patients when oral administration is temporarily not feasible. The overall duration of treatment with i.v. lacosamide is at the physician's discretion; there is experience from clinical trials with twice daily infusions of lacosamide for up to 5 days
Lacosamide must be administered twice a day (usually once in the morning and once in the evening). The recommended starting dose is 50 mg twice a day which should be increased to an initial therapeutic dose of 100 mg twice a day after one week.
Lacosamide treatment may also be initiated with a single loading dose of 200 mg, followed approximately 12 hours later by a 100 mg twice daily (200 mg/day) maintenance dose regimen. A loading dose may be initiated in patients in situations when the physician determines that rapid attainment of lacosamide steady state plasma concentration and therapeutic effect is warranted. It should be administered under medical supervision with consideration of the potential for increased incidence of central nervous system adverse reactions (see section 4.8). Administration of a loading dose has not been studied in acute conditions such as status epilepticus.
Depending on response and tolerability, the maintenance dose can be further increased by 50 mg twice a day every week, to a maximum recommended daily dose of 400 mg (200 mg twice a day).
In accordance with current clinical practice, if lacosamide has to be discontinued, it is recommended this be done gradually (e.g. taper the daily dose by 200 mg/week).
Conversion to or from oral and intravenous administration can be done directly without titration. The total daily dose and twice daily administration should be maintained.

Special populations

Older people (over 65 years of age)
No dose reduction is necessary in elderly patients. The experience with lacosamide in elderly patients with epilepsy is limited. Age associated decreased renal clearance with an increase in AUC levels should be considered in elderly patients (see following paragraph 'renal impairment' and section 5.2).

Renal impairment
No dose adjustment is necessary in mildly and moderately renally impaired patients (CL_{CR} >30 ml/min). In patients with mild or moderate renal impairment a loading dose of 200 mg may be considered, but further dose titration (>200 mg daily) should be performed with caution. In patients with severe renal impairment (CL_{CR} ≤30 ml/min) and in patients with end-stage renal disease, a maximum maintenance dose of 250 mg/day is recommended. In these patients, the dose titration should be performed with caution. If a loading dose is indicated, an initial dose of 100 mg followed by a 50 mg twice daily regimen for the first week should be used. For patients requiring haemodialysis a supplement of up to 50% of the divided daily dose directly after the end of haemodialysis is recommended. Treatment of patients with end-stage renal disease should be made with caution as there is little clinical experience and accumulation of a metabolite (with no known pharmacological activity).

Hepatic impairment
No dose adjustment is needed for patients with mild to moderate hepatic impairment.
The dose titration in these patients should be performed with caution considering co-existing renal impairment. A loading dose of 200mg may be considered, but further dose titration (>200 mg daily) should be performed with caution. The pharmacokinetics of lacosamide has not been evaluated in severely hepatic impaired patients (see section 5.2).

Paediatric population
The safety and efficacy of lacosamide in children aged below 16 years have not yet been established. No data are available.

Method of administration
Product with particulate matter or discolouration should not be used.
The solution for infusion is infused over a period of 15 to 60 minutes twice daily. Vimpat solution for infusion can be administered intravenously without further dilution or can be diluted with sodium chloride 9 mg/ml (0.9%) solution for injection, glucose 50 mg/ml (5%) solution for injection or lactated Ringer's solution for injection.

4.3 Contraindications
Hypersensitivity to the active substance or to any of the excipients listed in section 6.1.
Known second- or third-degree atrioventricular (AV) block.

4.4 Special warnings and precautions for use
Suicidal ideation and behaviour
Suicidal ideation and behaviour have been reported in patients treated with anti-epileptic agents in several indications. A meta-analysis of randomised placebo controlled trials of anti-epileptics has also shown a small increased risk of suicidal ideation and behaviour. The mechanism of this risk is not known and the available data do not exclude the possibility of an increased risk for lacosamide.
Therefore patients should be monitored for signs of suicidal ideation and behaviours and appropriate treatment should be considered. Patients (and caregivers of patients) should be advised to seek medical advice should signs of suicidal ideation or behaviour emerge (see section 4.8).

Cardiac rhythm and conduction
Prolongations in PR interval with lacosamide have been observed in clinical studies. Lacosamide should be used with caution in patients with known conduction problems or severe cardiac disease such as a history of myocardial infarction or heart failure. Caution should especially be exerted when treating elderly patients as they may be at an increased risk of cardiac disorders or when lacosamide is used in combination with products known to be associated with PR prolongation.
Second degree or higher AV block has been reported in post-marketing experience. In the placebo-controlled trials of lacosamide in epilepsy patients, atrial fibrillation or flutter were not reported; however both have been reported in open-label epilepsy trials and in post-marketing experience (see section 4.8).
Patients should be made aware of the symptoms of second-degree or higher AV block (e.g. slow or irregular pulse, feeling of lightheaded and fainting) and of the symptoms of atrial fibrillation and flutter (e.g. palpitations, rapid or irregular pulse, shortness of breath). Patients should be counselled to seek medical advice should any of these symptoms occur.

Dizziness
Treatment with lacosamide has been associated with dizziness which could increase the occurrence of accidental injury or falls. Therefore, patients should be advised to exercise caution until they are familiar with the potential effects of the medicine (see section 4.8).
This medicinal product contains 2.6 mmol (or 59.8 mg) sodium per vial. To be taken into consideration for patients on a controlled sodium diet.

4.5 Interaction with other medicinal products and other forms of interaction
Lacosamide should be used with caution in patients treated with medicinal products known to be associated with PR prolongation (e.g. carbamazepine, lamotrigine, pregabalin) and in patients treated with class I antiarrhythmics. However, subgroup analysis did not identify an increased magnitude of PR prolongation in patients with concomitant administration of carbamazepine or lamotrigine in clinical trials.

In vitro data
Data generally suggest that lacosamide has a low interaction potential. In vitro studies indicate that the enzymes CYP1A2, 2B6, and 2C9 are not induced and that CYP1A1, 1A2, 2A6, 2B6, 2C8, 2C9, 2D6, and 2E1 are not inhibited by lacosamide at plasma concentrations observed in clinical trials. An in vitro study indicated that lacosamide is not transported by P glycoprotein in the intestine. In vitro data show that CYP2C9, CYP2C19 and CYP3A4 are capable of catalysing the formation of the O-desmethyl metabolite.

In vivo data
Lacosamide does not inhibit or induce CYP2C19 and CYP3A4 to a clinically relevant extent. Lacosamide did not affect the AUC of midazolam (metabolised by CYP3A4, lacosamide given 200 mg twice daily) but C_{max} of midazolam was slightly increased (30%). Lacosamide did not affect the pharmacokinetics of omeprazole (metabolised by CYP2C19 and 3A4, lacosamide given 300 mg twice daily).
The CYP2C19 inhibitor omeprazole (40 mg q.d.) did not give rise to a clinically significant change in lacosamide exposure. Thus moderate inhibitors of CYP2C19 are unlikely to affect systemic lacosamide exposure to a clinically relevant extent.
Caution is recommended in concomitant treatment with strong inhibitors of CYP2C9 (e.g.fluconazole) and CYP3A4 (e.g. itraconazole, ketoconazole, ritonavir, clarithromycin), which may lead to increased systemic exposure of lacosamide. Such interactions have not been established *in vivo* but are possible based on *in vitro* data.
Strong enzyme inducers such as rifampicin or St John's wort (Hypericum perforatum) may moderately reduce the systemic exposure of lacosamide. Therefore, starting or ending treatment with these enzyme inducers should be done with caution.

Antiepileptics
In interaction trials lacosamide did not significantly affect the plasma concentrations of carbamazepine and valproic acid. Lacosamide plasma concentrations were not affected by carbamazepine and by valproic acid. A population PK analysis estimated that concomitant treatment with other anti-epileptics known to be enzyme inducers (carbamazepine, phenytoin, phenobarbital, in various doses) decreased the overall systemic exposure of lacosamide by 25%.

Oral contraceptives
In an interaction trial there was no clinically relevant interaction between lacosamide and the oral contraceptives ethinylestradiol and levonorgestrel. Progesterone concentrations were not affected when the medicinal products were co-administered.

Others
Interaction trials showed that lacosamide had no effect on the pharmacokinetics of digoxin. There was no clinically relevant interaction between lacosamide and metformin.
Co-administration of warfarin with lacosamide does not result in a clinically relevant change in the pharmacokinetics and pharmacodynamics of warfarin.
Although no pharmacokinetic data on the interaction of lacosamide with alcohol are available, a pharmacodynamic effect cannot be excluded.
Lacosamide has a low protein binding of less than 15%. Therefore, clinically relevant interactions with other drugs through competition for protein binding sites are considered unlikely.

4.6 Fertility, pregnancy and lactation
Pregnancy

Risk related to epilepsy and antiepileptic medicinal products in general
For all anti-epileptic drugs, it has been shown that in the offspring of women treated with epilepsy, the prevalence of malformations is two to three times greater than the rate of approximately 3% in the general population. In the treated population, an increase in malformations has been noted with polytherapy, however, the extent to which the treatment and/or the illness is responsible has not been elucidated.
Moreover, effective anti-epileptic therapy must not be interrupted, since the aggravation of the illness is detrimental to both the mother and the foetus.

Risk related to lacosamide
There are no adequate data from the use of lacosamide in pregnant women. Studies in animals did not indicate any teratogenic effects in rats or rabbits, but embryotoxicity was observed in rats and rabbits at maternal toxic doses (see section 5.3). The potential risk for humans is unknown.
Lacosamide should not be used during pregnancy unless clearly necessary (if the benefit to the mother clearly outweighs the potential risk to the foetus). If women decide to become pregnant, the use of this product should be carefully re-evaluated.

Breastfeeding
It is unknown whether lacosamide is excreted in human breast milk. Animal studies have shown excretion of lacosamide in breast milk. For precautionary measures, breast-feeding should be discontinued during treatment with lacosamide.

Fertility
No adverse reactions on male or female fertility or reproduction were observed in rats at doses producing plasma exposures (AUC) up to approximately 2 times the plasma AUC in humans at the maximum recommended human dose (MRHD).

4.7 Effects on ability to drive and use machines
Lacosamide has minor to moderate influence on the ability to drive and use machines. Lacosamide treatment has been associated with dizziness or blurred vision.
Accordingly, patients should be advised not to drive a car or to operate other potentially hazardous machinery until they are familiar with the effects of lacosamide on their ability to perform such activities.

4.8 Undesirable effects
Summary of safety profile
Based on the analysis of pooled placebo-controlled clinical trials in adjunctive therapy in 1,308 patients with partial-onset seizures, a total of 61.9% of patients randomized to lacosamide and 35.2% of patients randomized to placebo reported at least 1 adverse reaction. The most frequently reported adverse reactions with lacosamide treatment were dizziness, headache, nausea and diplopia. They were usually mild to moderate in intensity. Some were dose-related and could be alleviated by reducing the dose. Incidence and severity of central nervous system (CNS) and gastrointestinal (GI) adverse reactions usually decreased over time.
Over all controlled studies, the discontinuation rate due to adverse reactions was 12.2% for patients randomized to lacosamide and 1.6% for patients randomized to placebo. The most common adverse reaction resulting in discontinuation of lacosamide therapy was dizziness.
Incidence of CNS adverse reactions such as dizziness may be higher after a loading dose.

Tabulated list of adverse reactions
The table below shows the frequencies of adverse reactions which have been reported in clinical trials and post-marketing experience. The frequencies are defined as follows: very common (≥1/10), common (≥1/100 to <1/10), uncommon (≥1/1,000 to <1/100) and not known (frequency cannot be estimated from available data). Within each frequency grouping, undesirable effects are presented in order of decreasing seriousness.

System organ class	Very common	Common	Uncommon	Not known
Blood and lymphatic disorders				Agranulocytosis(1)
Immune system disorders			Drug hypersensitivity(1)	Drug reaction with eosinophilia and systemic symptoms (DRESS) [1]
Psychiatric disorders		Depression Confusional state Insomnia(1)	Aggression[1], Agitation[1], Euphoric mood[1] Psychotic disorder[1] Suicide attempt [1] Suicidal ideation [1] Hallucination [1]	
Nervous system disorders	Dizziness Headache	Balance disorder Coordination abnormal Memory impairment Cognitive disorder Somnolence, Tremor Nystagmus, Hypoesthesia Dysarthria Disturbance in attention Paraesthesia		
Eye disorders	Diplopia	Vision blurred		
Ear and labyrinth disorders		Vertigo, Tinnitus		
Cardiac disorders			Atrioventricular block[1] Bradycardia[1] Atrial Fibrillation [1] Atrial Flutter [1]	
Gastrointestinal disorders	Nausea	Vomiting, Constipation Flatulence, Dyspepsia Dry mouth, Diarrhoea		
Hepatobiliary disorders			Liver function test abnormal[1]	
Skin and subcutaneous tissue disorders		Pruritus Rash[1]	Angioedema[1] Urticaria[1]	Stevens-Johnson syndrome[1] Toxic epidermal necrolysis[1]
Musculoskeletal and connective tissue disorders		Muscle spasms		
General disorders and administration site conditions		Gait disturbance, Asthenia, Fatigue, Irritability Feeling drunk, Injection site pain or discomfort[2], Irritation[2]	Erythema[2]	
Injury, poisoning and procedural complications		Fall, Skin laceration Contusion		

[1] adverse reactions reported in post marketing experience.

[2] local adverse events associated with intravenous administration

Description of selected adverse reactions
The use of lacosamide is associated with dose-related increase in the PR interval. Adverse reactions associated with PR interval prolongation (e.g. atrioventricular block, syncope, bradycardia) may occur.
In clinical trials in epilepsy patients the incidence rate of reported first degree AV Block is uncommon, 0.7%, 0%, 0.5% and 0% for lacosamide 200 mg, 400 mg, 600 mg or placebo, respectively. No second or higher degree AV Block was seen in these studies. However, cases with second and third degree AV Block associated with lacosamide treatment have been reported in post-marketing experience.
In clinical trials, the incidence rate for syncope is uncommon and did not differ between lacosamide treated epilepsy patients (0.1%) and placebo treated epilepsy patients (0.3%).
Atrial fibrillation or flutter were not reported in short term clinical trials; however both have been reported in open-label epilepsy trials and in post-marketing experience.

Laboratory abnormalities
Abnormalities in liver function tests have been observed in controlled trials with lacosamide in adult patients with partial-onset seizures who were taking 1 to 3 concomitant anti-epileptic drugs. Elevations of ALT to ≥3XULN occurred in 0.7% (7/935) of Vimpat patients and 0% (0/356) of placebo patients.

Multiorgan hypersensitivity reactions
Multiorgan hypersensitivity reactions (also known as Drug Reaction with Eosinophilia and Systemic Symptoms, DRESS) have been reported in patients treated with some antiepileptic agents. These reactions are variable in expression but typically present with fever and rash and can be associated with involvement of different organ systems. If multiorgan hypersensitivity reaction is suspected, lacosamide should be discontinued.

Paediatric Population
Frequency, type and severity of adverse reactions in adolescents aged 16-18 years are expected to be the same as in adults. The safety of lacosamide in children aged below 16 years has not yet been established. No data are available.

4.9 Overdose
There is limited clinical experience with lacosamide overdose in humans.

Symptoms
Clinical symptoms (dizziness and nausea) following doses of 1,200 mg/day were mainly related to the central nervous system and the gastrointestinal system and resolved with dose adjustments.
The highest reported overdose in the clinical development program for lacosamide was 12 g taken in conjunction with toxic doses of multiple other antiepileptics. The subject was initially comatose and then fully recovered without permanent sequelae.

Management
There is no specific antidote for overdose with lacosamide. Treatment of lacosamide overdose should include general supportive measures and may include haemodialysis if necessary (see section 5.2).

5. PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic properties
Pharmacotherapeutic group: antiepileptics, other antiepileptics, ATC code: N03AX18

Mechanism of action
The active substance, lacosamide (R-2-acetamido-N-benzyl-3-methoxypropionamide) is a functionalised amino acid.
The precise mechanism by which lacosamide exerts its antiepileptic effect in humans remains to be fully elucidated.
In vitro electrophysiological studies have shown that lacosamide selectively enhances slow inactivation of voltage-gated sodium channels, resulting in stabilization of hyperexcitable neuronal membranes.

Pharmacodynamic effects
Lacosamide protected against seizures in a broad range of animal models of partial and primary generalized seizures and delayed kindling development.
In non-clinical experiments lacosamide in combination with levetiracetam, carbamazepine, phenytoin, valproate, lamotrigine, topiramate or gabapentin showed synergistic or additive anticonvulsant effects.

Clinical efficacy and safety
The efficacy of Vimpat as adjunctive therapy at recommended doses (200 mg/day, 400 mg/day) was established in 3 multicenter, randomized, placebo-controlled clinical trials with a 12-week maintenance period. Vimpat 600 mg/day was also shown to be effective in controlled adjunctive therapy trials, although the efficacy was similar to 400 mg/day and patients were less likely to tolerate this dose because of CNS- and gastrointestinal-related adverse reactions. Thus, the 600 mg/day dose is not recommended. The maximum recommended dose is 400 mg/day. These trials, involving 1308 patients with a history of an average of 23 years of partial-onset seizures, were designed to evaluate the efficacy and safety of lacosamide when administered concomitantly with 1-3 antiepileptics in patients with uncontrolled partial-onset seizures with or without secondary generalisation. Overall the proportion of subjects with a 50% reduction in seizure frequency was 23%, 34%, and 40% for placebo, lacosamide 200 mg/day and lacosamide 400 mg/day.
There are insufficient data regarding the withdrawal of concomitant antiepileptic medicinal products to achieve monotherapy with lacosamide.
The pharmacokinetics and safety of a single loading dose of iv lacosamide were determined in a multicenter, open-label study designed to assess the safety and tolerability of rapid initiation of
lacosamide using a single iv loading dose (including 200 mg) followed by twice daily oral dosing (equivalent to the iv dose) as adjunctive therapy in adult subjects 16 to 60 years of age with
partial-onset seizures.

5.2 Pharmacokinetic properties
Absorption
After i.v. administration, C_{max} is reached at the end of infusion. The plasma concentration increases proportionally with dose after oral (100 800 mg) and i.v. (50 300 mg) administration.

Distribution
The volume of distribution is approximately 0.6 L/kg. Lacosamide is less than 15% bound to plasma proteins.

Biotransformation
95% of the dose is excreted in the urine as drug and metabolites. The metabolism of lacosamide has not been completely characterised.
The major compounds excreted in urine are unchanged lacosamide (approximately 40% of the dose) and its O-desmethyl metabolite less than 30%.
A polar fraction proposed to be serine derivatives accounted for approximately 20% in urine, but was detected only in small amounts (0-2%) in human plasma of some subjects. Small amounts (0.5-2%) of additional metabolites were found in the urine.
In vitro data show that CYP2C9, CYP2C19 and CYP3A4 are capable of catalysing the formation of the O desmethyl metabolite but the main contributing isoenzyme has not been confirmed *in vivo*. No clinically relevant difference in lacosamide exposure was observed comparing its pharmacokinetics in extensive metabolisers (EMs, with a functional CYP2C19) and poor metabolisers (PMs, lacking a functional CYP2C19). Furthermore an interaction trial with omeprazole (CYP2C19 inhibitor) demonstrated no clinically relevant changes in lacosamide plasma concentrations indicating that the importance of this pathway is minor.
The plasma concentration of O-desmethyl lacosamide is approximately 15% of the concentration of lacosamide in plasma. This major metabolite has no known pharmacological activity.

Elimination
Lacosamide is primarily eliminated from the systemic circulation by renal excretion and biotransformation. After oral and intravenous administration of radiolabeled lacosamide, approximately 95% of radioactivity administered was recovered in the urine and less than 0.5% in the feces. The elimination half-life of the unchanged drug is approximately 13 hours. The pharmacokinetics is dose-proportional and constant over time, with low intra- and inter-subject variability. Following twice daily dosing, steady state plasma concentrations are achieved after a 3 day period. The plasma concentration increases with an accumulation factor of approximately 2.
A single loading dose of 200 mg approximates steady-state concentrations comparable to 100 mg twice daily oral administration.

Pharmacokinetics in special patient groups

Gender
Clinical trials indicate that gender does not have a clinically significant influence on the plasma concentrations of lacosamide.

Renal impairment
The AUC of lacosamide was increased by approximately 30% in mildly and moderately and 60% in severely renal impaired patients and patients with end-stage renal disease requiring hemodialysis compared to healthy subjects, whereas c_{max} was unaffected.
Lacosamide is effectively removed from plasma by haemodialysis. Following a 4 hour haemodialysis treatment, AUC of lacosamide is reduced by approximately 50%. Therefore dosage supplementation following haemodialysis is recommended (see section 4.2). The exposure of the O-desmethyl metabolite was several-fold increased in patients with moderate and severe renal impairment. In absence of haemodialysis in patients with end-stage renal disease, the levels were increased and continuously rising during the 24 hour sampling. It is unknown whether the increased metabolite exposure in endstage renal disease subjects could give rise to adverse effects but no pharmacological activity of the metabolite has been identified.

Hepatic impairment
Subjects with moderate hepatic impairment (Child-Pugh B) showed higher plasma concentrations of lacosamide (approximately 50% higher AUC_{norm}). The higher exposure was partly due to a reduced renal function in the studied subjects. The decrease in non-renal clearance in the patients of the study was estimated to give a 20% increase in the AUC of lacosamide. The pharmacokinetics of lacosamide has not been evaluated in severe hepatic impairment (see section 4.2).

Older people (over 65 years of age)
In a study in elderly men and women including 4 patients >75 years of age, AUC was about 30 and 50% increased compared to young men, respectively. This is partly related to lower body weight. The body weight normalized difference is 26 and 23%, respectively. An increased variability in exposure was also observed. The renal clearance of lacosamide was only slightly reduced in elderly subjects in this study.
A general dose reduction is not considered to be necessary unless indicated due to reduced renal function (see section 4.2).

5.3 Preclinical safety data
In the toxicity studies, the plasma concentrations of lacosamide obtained were similar or only marginally higher than those observed in patients, which leaves low or non-existing margins to human exposure
A safety pharmacology study with intravenous administration of lacosamide in anesthetized dogs showed transient increases in PR interval and QRS complex duration and decreases in blood pressure most likely due to a cardiodepressant action. These transient changes started in the same concentration range as after maximum recommended clinical dosing. In anesthetized dogs and Cynomolgus monkeys, at intravenous doses of 15-60 mg/kg, slowing of atrial and ventricular conductivity, atrioventricular block and atrioventricular dissociation were observed.
In the repeated dose toxicity studies, mild reversible liver changes were observed in rats starting at about 3 times the clinical exposure. These changes included an increased organ weight, hypertrophy of hepatocytes, increases in serum concentrations of liver enzymes and increases in total cholesterol and triglycerides. Apart from the hypertrophy of hepatocytes, no other histopathologic changes were observed.
In reproductive and developmental toxicity studies in rodents and rabbits, no teratogenic effects but an increase in numbers of stillborn pups and pup deaths in the peripartum period, and slightly reduced live litter sizes and pup body weights were observed at maternal toxic doses in rats corresponding to systemic exposure levels similar to the expected clinical exposure. Since higher exposure levels could not be tested in animals due to maternal toxicity, data are insufficient to fully characterise the embryofetotoxic and teratogenic potential of lacosamide.
Studies in rats revealed that lacosamide and/or its metabolites readily crossed the placental barrier.

6. PHARMACEUTICAL PARTICULARS

6.1 List of excipients
water for injections
sodium chloride
hydrochloric acid (for pH adjustment)

6.2 Incompatibilities
This medicinal product must not be mixed with other medicinal products except those mentioned in section 6.5.

6.3 Special precautions for storage
Do not store above 30°C.
For storage conditions after dilution of the medicinal product, see section 6.5.

6.4 Nature and contents of container
Colourless type I glass vial with a chlorobutyl rubber closure coated with a fluoropolymer.
Packs of 1x20 ml and 5x20 ml.
Not all packsizes may be marketed.

6.5 Special precautions for disposal and other handling
This medicinal product is for single use only, any unused solution should be discarded.
Chemical and physical in-use stability has been demonstrated for 24 hours at temperatures up to 25° C for product mixed with the diluents mentioned in 6.5 and stored in glass or PVC bags.
From a microbiological point of view, the product should be used immediately. If not used immediately, in-use storage times and conditions prior to use are the responsibility of the user and would not be longer than 24 hours at 2 to 8°C, unless dilution has taken place in controlled and validated aseptic conditions.
Vimpat solution for infusion was found to be physically compatible and chemically stable when mixed with the following diluents for at least 24 hours and stored in glass or PVC bags at temperatures up to 25°C.
Diluents:
sodium chloride 9 mg/ml (0.9%) solution for injection
glucose 50 mg/ml (5%) solution for injection
lactated Ringer's solution for injection.

Manufactured by
UCB Pharma SA
Chemin du Foriest
1420 Braine L'Alleud
Belgium

Batch released by
Aesica Pharmaceuticals GmbH
Alfred Nobel Strasse 10
40789 Monheim Am Rhein,
Germany

MARKETING AUTHORISATION HOLDER
UCB Pharma SA
Allée de la Recherche 60
B 1070 Bruxelles
Belgium

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This is a Medicament
- A Medicament is a product, which affects your health, and its consumption, contrary to instruction, is dangerous for you.
- Follow strictly the doctor's prescription .the method of use and the instructions of the pharmacist who sold the medicament.
- The doctor and the pharmacist are experts in medicine, its benefits and risks.
- Do not, by yourself, interrupt the period of treatment prescribed for you.
- Do not repeat the same prescription without consulting your doctor
- Keep medicaments out of the reach of children.

Council of Arab Health Ministers
Union of Arab Pharmacists

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