SUMMARY OF PRODUCT CHARACTERISTICS

1 NAME OF THE MEDICINAL PRODUCT

Monofer 100 mg/ml solution for injection/infusion

2 QUALITATIVE AND QUANTITATIVE COMPOSITION

One millilitre of solution contains 100 mg iron as ferric derisomaltose

1 ml vial/ampoule contains 100 mg iron as ferric derisomaltose
2 ml vial/ampoule contains 200 mg iron as ferric derisomaltose
5 ml vial/ampoule contains 500 mg iron as ferric derisomaltose
10 ml vial/ampoule contains 1,000 mg iron as ferric derisomaltose

An iron dose of 100 mg contains up to 9.4 mg (0.41 mmol) sodium, see section 4.4.

For the full list of excipients, see section 6.1.

3 PHARMACEUTICAL FORM

Solution for injection/infusion.

Dark brown, non transparent solution.

4 CLINICAL PARTICULARS

4.1 Therapeutic indications

Monofer is indicated for the treatment of iron deficiency in the following conditions:

- When oral iron preparations are ineffective or cannot be used
- Where there is a clinical need to deliver iron rapidly

The diagnosis must be based on laboratory tests. In relation to acute blood loss, haemoglobin is sufficient as a diagnostic laboratory test.

4.2 Posology and method of administration

Monitor carefully patients for signs and symptoms of hypersensitivity reactions during and following each administration of Monofer. Monofer should only be administered when staff trained to evaluate and manage anaphylactic reactions is immediately available, in an environment where full resuscitation facilities can be assured. The patient should be observed for adverse effects for at least 30 minutes following each Monofer injection (see section 4.4).

Each IV iron administration is associated with a risk of a hypersensitivity reaction. Thus, to minimise risk the number of single IV iron administrations should be kept to a minimum.

Posology

The posology of Monofer follows a stepwise approach: [1] determination of the individual iron need and [2] calculation and administration of the iron dose(s). The steps can be repeated after [3] post-iron repletion assessments.

Step 1: Determination of the iron need:

The iron need can be determined using either the Simplified Table (i) or the Ganzoni formula below (ii).

The iron need is expressed in mg elemental iron.

i. Simplified Table:

Table 1. Simplified Table

Hb (g/dl)	Hb (mmol/l)	Patients with bodyweight <50 kg	Patients with bodyweight 50 kg to <70 kg	Patients with body weight ≥70 kg
≥10	≥6.2	500 mg	1000 mg	1500 mg
<10	<6.2	500 mg	1500 mg	2000 mg

ii. Ganzoni formula:

Table 2. Ganzoni formula

$$\underset{\text{[mg iron]}}{\text{Iron need}} = \underset{\text{[kg]}}{\text{Body weight}^{(A)}} \times (\underset{\text{[g/dl]}}{\text{Target Hb}^{(D)}} - \text{Actual Hb})^{(B)} \times 2.4 + \underset{\text{[mg iron]}}{\text{Iron for iron stores}^{(C)}}$$

(A) It is recommended to use the patient's ideal body weight for obese patients or pre-pregnancy weight for pregnant women. For all other patients use actual body weight. Ideal body weight may be calculated in a number of ways e.g. by calculating weight at BMI 25 i.e. ideal body weight = 25 * (height in m)^2

(B) To convert Hb [mM] to Hb [g/dl] you should multiply Hb [mM] by factor 1.61145

(C) For a person with a body weight above 35 kg, the iron stores are 500 mg or above. Iron stores of 500 mg are at the lower limit normal for small women. Some guidelines suggest using 10-15 mg iron /kg body weight.

(D) Default Hb target is 15 g/dl in the Ganzoni formula. In special cases such as pregnancy consider using a lower haemoglobin target.

iii. Fixed iron need:

A fixed dose of 1000 mg is given and the patient is re-evaluated for further iron need according to “Step 3: Post-iron repletion assessments”. For patients weighing less than 50 kg use the Simplified table or Ganzoni formula for iron need calculation.

Step 2: Calculation and administration of the maximum individual iron dose(s):

Based on the iron need determined above the appropriate dose(s) of Monofer should be administered taking into consideration the following:

The total dose per week should not exceed 20 mg iron/kg bodyweight.
A single Monofer infusion should not exceed 20 mg iron/kg body weight.
A single Monofer bolus injection should not exceed 500 mg iron.

Step 3: Post-iron repletion assessments:

Re-assessment including blood tests should be performed by the clinician based on the individual patient's condition. To evaluate the effect of IV iron treatment, the Hb level should be re-assessed no earlier than 4 weeks post final Monofer administration to allow adequate time for erythropoiesis and iron utilisation. In the event the patient requires further iron repletion, the iron need should be recalculated.

Children and adolescents:

Monofer is not recommended for use in children and adolescents < 18 years due to insufficient data on safety and efficacy.

Method of administration:

Monofer must be administered by the intravenous route either by injection or by infusion.

Monofer should not be administered concomitantly with oral iron preparations, since the absorption of oral iron might be decreased (see section 4.5).

Intravenous bolus injection:

Monofer may be administered as an intravenous bolus injection up to 500 mg up to three times a week at an administration rate of up to 250 mg iron/minute. It may be administered undiluted or diluted in maximum 20 ml sterile 0.9% sodium chloride.

Table 3: Administration rates for intravenous bolus injection

Volume of Monofer	Equivalent iron dose	Administration rate/ Minimum administration time	Frequency
≤5 ml	≤500 mg	250 mg iron/minute	1-3 times a week

Intravenous infusion:

The iron need required may be administered in a single Monofer infusion up to 20 mg iron/kg body weight or as weekly infusions until the cumulative iron need has been administered.

If the iron need exceeds 20 mg iron/kg body weight, the dose must be split in two administrations with an interval of at least one week. It is recommended whenever possible to give 20 mg iron/kg body weight in the first administration. Dependent on clinical judgement the second administration could await follow-up laboratory tests.

Table 4: Administration rates for intravenous infusion

Iron dose	Minimum administration time
≤1000 mg >1000 mg	More than 15 minutes 30 minutes or more

Monofer should be infused undiluted or diluted in sterile 0.9% sodium chloride. For stability reasons, Monofer should not be diluted to concentrations less than 1 mg iron/ml (not including the volume of the ferric derisomaltose solution) and never diluted in more than 500 ml. Please refer to section 6.6.

Injection into dialyser:

Monofer may be administered during a haemodialysis session directly into the venous limb of the dialyser under the same procedures as outlined for intravenous bolus injection.

4.3 Contraindications

- Hypersensitivity to the active substance, to Monofer or any of its excipients listed in section 6.1
- Known serious hypersensitivity to other parenteral iron products
- Non-iron deficiency anaemia (e.g. haemolytic anaemia)
- Iron overload or disturbances in utilisation of iron (e.g. haemochromatosis, haemosiderosis)
- Decompensated liver disease

4.4 Special warnings and precautions for use

Parenterally administered iron preparations can cause hypersensitivity reactions including serious and potentially fatal anaphylactic/anaphylactoid reactions. Hypersensitivity reactions have also been reported after previously uneventful doses of parenteral iron complexes. There have been reports of hypersensitivity reactions which progressed to Kounis syndrome (acute allergic coronary arteriospasm that can result in myocardial infarction, see section 4.8).

The risk is enhanced for patients with known allergies including drug allergies, including patients with a history of severe asthma, eczema or other atopic allergy. There is also an increased risk of hypersensitivity reactions to parenteral iron complexes in patients with immune or inflammatory conditions (e.g. systemic lupus erythematosus, rheumatoid arthritis).

Monofer should only be administered when staff trained to evaluate and manage anaphylactic reactions is immediately available, in an environment where full resuscitation facilities can be assured. Each patient should be observed for adverse effects for at least 30 minutes following each Monofer injection. If hypersensitivity reactions or signs of intolerance occur during administration, the treatment must be stopped immediately. Facilities for cardio respiratory resuscitation and equipment for handling acute anaphylactic/anaphylactoid reactions should be available, including an injectable 1:1000 adrenaline solution. Additional treatment with antihistamines and/or corticosteroids should be given as appropriate.

In patients with compensated liver dysfunction, parenteral iron should only be administered after careful benefit/risk assessment. Parenteral iron administration should be avoided in patients with hepatic dysfunction (alanine aminotransferase and/or aspartate aminotransferase > 3 times upper limit of normal) where iron overload is a precipitating factor, in particular Porphyria Cutanea Tarda (PCT). Careful monitoring of iron status is recommended to avoid iron overload.

Parenteral iron should be used with caution in case of acute or chronic infection.

Monofer should not be used in patients with ongoing bacteraemia.

Hypotensive episodes may occur if intravenous injection is administered too rapidly.

Caution should be exercised to avoid paravenous leakage when administrating Monofer. Paravenous leakage of Monofer at the injection site may lead to irritation of the skin and potentially long lasting brown discolouration at the site of injection. In case of paravenous leakage, the administration of Monofer must be stopped immediately.

Monofer contains up to 9.4 mg (0.41 mmol) sodium per iron dose of 100 mg, equivalent to 0.47% of the WHO recommended maximum daily intake of 2 g sodium for an adult.

4.5 Interaction with other medicinal products and other forms of interaction

As with all parenteral iron preparations the absorption of oral iron is reduced when administered concomitantly.

Large doses of parenteral iron (5 ml or more) have been reported to give a brown colour to serum from a blood sample drawn four hours after administration.

Parenteral iron may cause falsely elevated values of serum bilirubin and falsely decreased values of serum calcium.

4.6 Fertility, pregnancy and lactation

Pregnancy

There is only limited data from the use of Monofer in pregnant women from one study with 100 exposed pregnant women. A careful risk/benefit evaluation is therefore required before use during pregnancy.

Iron deficiency anaemia occurring in the first trimester of pregnancy can in many cases be treated with oral iron. Treatment with Monofer should be confined to second and third trimester if the benefit is judged to outweigh the potential risk for both the mother and the foetus.

Foetal bradycardia may occur following administration of parenteral irons. It is usually transient and a consequence of a hypersensitivity reaction in the mother. The unborn baby should be carefully monitored during intravenous administration of parenteral irons to pregnant women.

Breast-feeding
A clinical study showed that transfer of iron from Monofer to human milk was very low. At therapeutic doses of Monofer no effects on the breastfeed newborns/infants are anticipated.

Fertility
There are no data on the effect of Monofer on human fertility. Fertility was unaffected following Monofer treatment in animal studies (see section 5.3).

4.7 Effects on ability to drive and use machines

No studies on the effects on the ability to drive and use machines have been performed.

4.8 Undesirable effects

The table presents the adverse drug reactions (ADRs) reported during Monofer treatment in clinical trials and in-market experience.

Acute severe hypersensitivity reactions may occur with parenteral iron preparations. They usually occur within the first few minutes of administration and are generally characterised by the sudden onset of respiratory difficulty and/or cardiovascular collapse; fatalities have been reported. Other less severe manifestations of immediate hypersensitivity, such as urticaria and itching may also occur. In pregnancy, associated foetal bradycardia may occur with parenteral iron preparations.

Fishbane reaction characterised by flushing in the face, acute chest and/or back pain and tightness sometimes with dyspnea in association with IV iron treatment may occur (frequency uncommon). This may mimic the early symptoms of an anaphylactoid/anaphylactic reaction. The infusion should be stopped and the patient's vital signs should be assessed. These symptoms disappear shortly after the iron administration is stopped. They typically do not reoccur if the administration is restarted at a lower infusion rate.
Distant skin discolouration has also been reported post marketing following IV iron administration.

Adverse drug reactions observed during clinical trials and post-marketing experience

System Organ Class	Common (≥1/100 to <1/10)	Uncommon (≥1/1000 to <1/100)	Rare (≥1/10000 to <1/1000)	Not known
Immune system disorders		Hypersensitivity, including severe reactions	Anaphylactoid/ anaphylactic reactions	
Nervous system disorders		Headache, paraesthesia, dysgeusia, blurred vision, loss of consciousness, dizziness, fatigue	Dysphonia, seizure, tremor, altered mental status	
Cardiac disorders		Tachycardia	Arrhythmia	Kounis syndrome
Vascular disorders		Hypotension, hypertension		
Respiratory, thoracic and mediastinal disorders		Chest pain, dyspnoea, bronchospasm		
Gastrointestinal disorders	Nausea	Abdominal pain, vomiting, dyspepsia, constipation, diarrhoea		
Skin and subcutaneous tissue disorders	Rash	Pruritus, urticaria, flushing, sweating, dermatitis	Angioedema	Distant skin discolouration
Metabolism and nutritional disorders		Hypophosphataemia		
Musculoskeletal and connective tissue disorders		Back pain, myalgia, arthralgia, muscle spasms		
General disorders and	Injection site reactions*	Pyrexia, chills/shivering,	Malaise, influenza like	

administration site conditions		infection, local phlebitic reaction, skin exfoliation	illness**	
Investigations		Hepatic enzyme increased		

* Includes the following preferred terms, i.e. injection site erythema, -swelling, -burning, -pain, -bruising, -discolouration, -extravasation, -irritation, -reaction.
** Influenza like illness whose onset may vary from a few hours to several days.

Description of selected adverse reactions
Delayed reactions may also occur with parenteral iron preparations and can be severe. They are characterised by arthralgia, myalgia and sometimes fever. The onset varies from several hours up to four days after administration. Symptoms usually last two to four days and settle spontaneously or following the use of simple analgesics.

Reporting of suspected adverse reactions
Reporting suspected adverse reactions after authorisation of the medicinal product is important. It allows continued monitoring of the benefit/risk balance of the medicinal product. Healthcare professionals are asked to report any suspected adverse reactions via the Yellow Card Scheme, Website: www.mhra.gov.uk/yellowcard.

4.9 Overdose

The active substance in Monofer has a low toxicity. The preparation is well tolerated and has a minimal risk of accidental overdosing.

Overdose may lead to accumulation of iron in storage sites eventually leading to haemosiderosis. Monitoring of iron parameters such as serum ferritin may assist in recognising iron accumulation. Supportive measures such as chelating agents can be used.

5 PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic properties

Pharmacotherapeutic group: Iron parenteral preparation, ATC code: B03AC

Monofer solution for injection is a colloid with strongly bound iron in spheroidal iron-carbohydrate particles.

The Monofer formulation contains iron in a complex that enables a controlled and slow release of bioavailable iron to iron-binding proteins with little risk of free iron.

Each particle consists of a matrix of iron(III) atoms and derisomaltose with an average molecular weight of 1000 Da and a narrow molecular weight distribution that is almost devoid of mono- and disaccharides.
INN name: Ferric derisomaltose (also known as iron(III) isomaltoside 1000).

The chelation of iron(III) with carbohydrate confers to the particles a structure resembling ferritin that is suggested to protect against the toxicity of unbound inorganic iron(III).

The iron is available in a non-ionic water-soluble form in an aqueous solution with pH between 5.0 and 7.0.

Evidence of a therapeutic response can be seen within a few days of administration of Monofer as an increase in the reticulocyte count. Due to the slow release of bioavailable iron serum ferritin peaks within days after an intravenous dose of Monofer and slowly returns to baseline after weeks.

Clinical efficacy

The efficacy of Monofer has been studied in the different therapeutic areas necessitating IV iron to correct iron deficiency. The main trials are described in more detail below.

Iron deficiency anaemia outside CKD

The P-Monofer-IDA-01 trial was an open-label, comparative, randomised, multi-centre, non-inferiority trial conducted in 511 patients with IDA randomised 2:1 to either Monofer or iron sucrose. 90 % of recruited patients were females. The dosing of Monofer was performed according to the Simplified Table as described in section 4.2 above and dosing of iron sucrose was calculated according to Ganzoni and administered as 200 mg infusions. The primary endpoint was the proportion of patients with an Hb increase ≥2 g/dl from baseline at any time between weeks 1 to 5. A higher proportion of patients treated with Monofer compared to iron sucrose reached the primary endpoint, 68.5% vs 51.6%, respectively (FAS, $p < 0.0001$).

The P-Monofer-IDA-03 trial was an open-label, comparative, randomised, multi-centre trial conducted in 1512 patients with IDA randomised 2:1 to either Monofer 1000 mg infused over 20 min (1009 subjects) or iron sucrose administered as 200 mg IV injections repeated up to a cumulative dose of 1000 mg (503 subjects). For the co-primary efficacy endpoint the change from baseline to week 8 in Hb was 2.49 g/dL in the Monofer fer group and 2.49 g/dL in the iron sucrose group. The estimated treatment difference [95 % CI] of Monofer - iron sucrose was 0.00 g/dL [-0.13;0.13]. Since the lower bound of the 95 % CI for the treatment difference was above -0.5 g/dL, non-inferiority was concluded. For the co-primary safety endpoint, a total of 3 treatment emergent serious or severe hypersensitivity reactions in 989 subjects (0.3 %) were adjudicated and confirmed by the adjudication committee in the Monofer group. The 95 % CI was [0.06 %;0.88 %] and as the upper bound was <3 %, the primary safety objective was considered met. In the iron sucrose group 2 treatment emergent serious or severe hypersensitivity reactions in 494 subjects (0.4 %) were adjudicated and confirmed by the adjudication committee. The risk difference between Monofer and iron sucrose was estimated to -0.10 % [95% CI: -0.91;0.71].

Nephrology

Non-dialysis-dependent chronic kidney disease

The P-Monofer-CKD-02 trial was an open-label, comparative, randomised, multi-centre, non-inferiority trial conducted in 351 iron deficient non-dialysis dependent (NDD) chronic kidney disease (CKD) patients, randomised 2:1 to either Monofer or oral iron sulphate administered as 100 mg elemental oral iron twice daily (200 mg daily) for 8 weeks. The patients in the Monofer group were randomised to infusion of 1000 mg single dose or bolus injections of 500 mg. Monofer was non-inferior to oral iron at week 4 ($p<0.001$) and also sustained a superior increase in Hb compared to oral iron from week 3 until the end of trial at week 8 ($p=0.009$ at week 3).

The P-Monofer-CKD-04 trial was an open-label, comparative, randomised, multi-centre trial conducted in 1538 NDD-CKD patients with IDA randomised 2:1 to either Monofer 1000 mg infused over 20 min (1027 subjects) or iron sucrose administered as 200 mg IV injections repeated up to a cumulative dose of 1000 mg (511 subjects). For the co-primary efficacy endpoint, the change from baseline to week 8 in Hb was 1.22 g/dL in the Monofer group and 1.14 g/dL in the iron sucrose group. The estimated treatment difference was 0.08 g/dL [95% CI: -0.06;0.23]. Since the lower bound of the 95 % CI was above -0.5 g/dL, non-inferiority was concluded. For the co-primary safety endpoint, a total of 3 treatment emergent serious or severe hypersensitivity reactions in 1019 subjects (0.3 %) were adjudicated and confirmed by the adjudication committee in the Monofer group. The 95 % CI was [0.06 %;0.86 %] and as the upper bound was <3 %, the primary safety objective was considered met. No treatment emergent serious or severe hypersensitivity reactions were adjudicated and confirmed by the adjudication committee in the iron sucrose group. The risk difference between Monofer and iron sucrose was estimated to 0.29 % [95% CI: -0.19;0.77].

Haemodialysis-dependent chronic kidney disease
The P-Monofer-CKD-03 trial was an open-label, comparative, randomised, multi-centre, non-inferiority trial conducted in 351 haemodialysis patients randomised 2:1 to either Monofer or iron sucrose. Patients were randomised to either a single injection of 500 mg or 500 mg in split doses of Monofer or 500 mg iron sucrose in split doses. Both treatments showed similar efficacy with more than 82% of patients with Hb in the target range (non-inferiority, p=0.01).

Oncology
Cancer related anaemia
The P-Monofer-CIA-01 trial was an open-label, comparative, randomised, multi-centre, non-inferiority trial conducted in 350 cancer patients with anaemia randomised 2:1 to either Monofer or oral iron sulphate administered as 100 mg elemental oral iron twice daily (200 mg daily) for 12 weeks. The patients in the Monofer group were randomised to either an infusion of max 1000 mg single doses over 15 min or bolus injections of 500 mg over 2 min. The primary endpoint was change in Hb concentrations from baseline to week 4. Monofer was non-inferior to oral iron at week 4 ($p<0.001$) and a faster onset of the Hb response was observed with infusion of Monofer.

Gastroenterology
Inflammatory bowel disease
The P-Monofer-IBD-01 trial was an open-label, comparative, randomised, multi-centre, non-inferiority trial conducted in 338 inflammatory bowel disease (IBD) patients randomised 2:1 to receive Monofer or oral iron sulphate administered as 100 mg elemental oral iron twice daily for 8 weeks (200 mg daily). The patients in the Monofer group were randomised to either an infusion of max 1000 mg single doses over 15 min or bolus injections of 500 mg over 2 min. A modified Ganzoni formula was used to calculate the IV iron need with a target Hb of only 13 g/dl resulting in an average iron dose of 884 mg elemental iron compared to oral iron administered as 200 mg oral iron sulfate once daily for 8 weeks (11,200 mg elemental oral iron in total). The primary endpoint was change in Hb concentrations from baseline to week 8. The patients had mild to moderate disease activity. Non-inferiority in change of Hb to week 8 could not be demonstrated. The dose-response relationship observed with Monofer suggests that the true iron demand of IV iron was underestimated by the modified Ganzoni formula. The Hb response (Hb increase ≥2 g/dl) rate was 93% for patients receiving > 1000 mg Monofer.

Women's health
Postpartum

The P-Monofer-PP-01 trial was an open-label, comparative, randomised, single-centre trial conducted in 200 healthy women with postpartum haemorrhage exceeding 700 mL and ≤1000 ml or PPH >1000 ml and Hb >6.5 g/dl measured >12 hours after delivery. The women were randomised 1:1 to receive either a single dose of 1200 mg Monofer or standard medical care. The primary endpoint was the aggregated change in physical fatigue within 12 weeks postpartum. The difference in aggregated change in physical fatigue score within 12 weeks postpartum was -0.97 (p=0.006), in favour of Monofer.

5.2 Pharmacokinetic properties

The Monofer formulation contains iron in a strongly bound complex that enables a controlled and slow release of bioavailable iron to iron-binding proteins with little risk of free iron toxicity. After administration of a single dose of Monofer of 100 to 1000 mg of iron in pharmacokinetic studies, the iron injected or infused was cleared from the plasma with a half-life that ranged from 1 to 4 days. Renal elimination of iron was negligible.

Following intravenous administration, ferric derisomaltose is rapidly taken up by the cells in the reticuloendothelial system (RES), particularly in the liver and spleen from where iron is slowly released.

Circulating iron is removed from the plasma by cells of the reticuloendothelial system which split the complex into its components of iron and derisomaltose. The iron is immediately bound to the available protein moieties to form hemosiderin or ferritin, the physiological storage forms of iron, or to a lesser extent, to the transport molecule transferrin. This iron, which is subject to physiological control, replenishes haemoglobin and depleted iron stores.

Iron is not easily eliminated from the body and accumulation can be toxic. Due to the size of the complex, Monofer is not eliminated via the kidneys. Small quantities of iron are eliminated in urine and faeces.

Derisomaltose is either metabolised or excreted.

5.3 Preclinical safety data

Iron complexes have been reported to be teratogenic and embryocidal in non-anaemic pregnant animals at high single doses above 125 mg iron/kg body weight. The highest recommended dose in clinical use is 20 mg iron/kg body weight.

In a fertility study with Monofer in rats no effects on male reproductive performance and spermatogenic parameters were found at dose level tested.

6 PHARMACEUTICAL PARTICULARS

6.1 List of excipients

Water for injections

Sodium hydroxide (for pH adjustment)

Hydrochloric acid (for pH adjustment)

6.2 Incompatibilities

This medicinal product must not be mixed with other medicinal products except those mentioned in section 6.6

6.3 Shelf life

Shelf life of ampoules as packaged for sale
3 years

Shelf life of vials as packaged for sale
3 years

Shelf life after first opening of the container (undiluted):

From a microbiological point of view, unless the method of opening precludes the risk of microbial contamination, the product should be used immediately.

If not used immediately, in-use storage times and conditions are the responsibility of the user.

Shelf life after dilution with sterile 0.9% sodium chloride:

From a microbiological point of view, the product should be used immediately.

6.4 Special precautions for storage

This medicinal product does not require any special storage conditions.

For storage conditions of the reconstituted and diluted solution see section 6.3.

6.5 Nature and contents of container

Type 1 glass ampoule.
Pack sizes: 5 x 1 ml, 10 x 1 ml, 5 x 2 ml, 10 x 2 ml, 2 x 5 ml, 5 x 5 ml, 2 x 10 ml, 5 x 10 ml

Type 1 glass vial with chlorobutyl rubber stopper and aluminium cap.

Pack sizes: 1 x 1 ml, 5 x 1 ml, 10 x 1 ml, 5 x 2 ml, 10 x 2 ml, 1 x 5 ml, 2 x 5 ml, 5 x 5 ml, 1 x 10 ml, 2 x 10 ml, 5 x 10 ml

Not all pack sizes may be marketed.

6.6 Special precautions for disposal

Inspect vials/ampoules visually for sediment and damage before use. Use only those containing sediment-free, homogeneous solution.

Monofer is for single use only and any unused solution should be disposed of in accordance with local requirements.

Monofer must only be mixed with sterile 0.9% sodium chloride. No other intravenous dilution solutions should be used. No other therapeutic agents should be added. For dilution instructions, see section 4.2.

The reconstituted solution for injection should be visually inspected prior to use. Use only clear solutions without sediment.

7 MARKETING AUTHORISATION HOLDER

Pharmacosmos A/S

Roervangsvej 30

DK-4300 Holbaek

Denmark

8 MARKETING AUTHORISATION NUMBER(S)

PL 18380/0001

9 DATE OF FIRST AUTHORISATION/RENEWAL OF THE AUTHORISATION

Date of first authorisation: 18/01/2010
Date of latest renewal: 26/11/2014

10 DATE OF REVISION OF THE TEXT

28/03/2025