SCHEDULING STATUS

S5

1. NAME OF THE MEDICINE

DORMICUM® 7,5 mg tablets

DORMICUM® 15 mg tablets

2. QUALITATIVE AND QUANTITATIVE COMPOSITION

DORMICUM tablets contain midazolam maleate equivalent to 7,5 or 15 mg midazolam as the active substance.

Excipients with known effect:

DORMICUM contains sugar (lactose anhydrous).

For the full list of excipients, see section 6.1.

3. PHARMACEUTICAL FORM

DORMICUM 7,5 mg: White to almost white, oval, cylindrical, biconvex film-coated tablets. Imprint: upper surface '7,5'; lower surface scored.

DORMICUM 15 mg: Grey-blue, oval, cylindrical, biconvex, film-coated tablets. Imprint: upper surface '15'; lower surface scored.

4. CLINICAL PARTICULARS

4.1. Therapeutic Indications

DORMICUM is indicated:

- In short-term treatment of insomnia, when the disorder is severe, disabling, or subjecting the individual to extreme distress.
- In anaesthetic premedication.

4.2. Posology and method of administration

Posology

Patients should be advised to take DORMICUM for short-term treatment only.

Generally, the duration of treatment varies from a few days to a maximum of two weeks. The tapering-off process should be tailored to the individual. Treatment with DORMICUM should not be terminated abruptly.

In certain cases, extension beyond the maximum treatment period may be necessary; if so, it should not take place without re-evaluation of the patient's status. DORMICUM should be taken just before going to bed.

Dose for insomnia

Adults - Initial dose: 7,5 mg.

Dosage range: 7,5 mg - 15 mg.

Treatment should be started with the lowest recommended dose. The maximum dose should not be exceeded because of the increased risk of unacceptable CNS adverse effects, possibly including clinically relevant respiratory and cardiovascular depression. Patients with impaired liver function should have a reduced dose.

In premedication, a 15 mg DORMICUM tablet should be given orally, 30 - 60 minutes before the operation, unless the parenteral route is preferred.

Special populations

Elderly and/or debilitated patients

In elderly and/or debilitated patients the recommended dose is 7,5 mg. Elderly patients showed a larger sedative effect, therefore they may be at increased risk of cardio-respiratory depression as well. Thus, DORMICUM should be used very carefully in elderly patients, and if needed, a lower dose should be considered.

Patients with hepatic impairment

Patients with severe hepatic impairment should not be treated with DORMICUM (see section 4.3). In patients with impaired liver function the recommended dose is 7,5 mg. In patients with mild to

moderate hepatic impairment, the lowest dose possible should be considered, not exceeding 7,5 mg. DORMICUM should be used very carefully in patients with hepatic impairment with the dosage reduced by as much as 50 % as deemed necessary.

Patients with renal impairment

In patients with severe renal impairment, accumulation of the major midazolam metabolite, 1'-hydroxymidazolam glucuronide, may occur resulting in more apparent and prolonged sedation, possibly including clinically relevant respiratory and/or cardiovascular depression. DORMICUM should therefore be dosed carefully in this patient population. The recommended dose is 7,5 mg and when needed a lower dose should be considered.

4.3. Contraindications

- Use in patients with known hypersensitivity to midazolam or any medicine from the benzodiazepines group or to any excipient within the medicine.
- Myasthenia gravis.
- DORMICUM is not recommended for the primary treatment of psychotic illness.
- DORMICUM, as a single treatment regime, should not be used to treat depression or anxiety associated with depression (suicide may be precipitated in such patients).
- Severe hepatic impairment. DORMICUM is not indicated to treat patients with severe hepatic impairment as it may cause encephalopathy.
- Children below 18 years.
- DORMICUM tablets should not be given to children 12 years or younger, because the available strengths do not allow for appropriate dosing in this patient population.
- Respiratory insufficiency.
- Sleep apnoea syndrome.
- Concomitant therapy with ketoconazole, itraconazole, voriconazole, HIV protease inhibitors including ritonavir boosted protease inhibitors formulations and the HCV protease inhibitors boceprevir and telaprevir (see section 4.5).

- Galactose intolerance.

4.4. Special warnings and precautions for use

Tolerance

Some loss of efficacy of the hypnotic effects of short-acting benzodiazepines, such as DORMICUM, may develop after repeated use for a few weeks.

Dependence

Use of DORMICUM may lead to physical and psychological dependence. Discontinuation of therapy may result in withdrawal or rebound phenomena. Psychological dependence may occur. Abuse has been reported in poly-drug abusers.

Once physical dependence has developed, abrupt termination will be accompanied by withdrawal symptoms. These may consist of headaches, diarrhoea, muscle pain, extreme anxiety, tension, restlessness, confusion and irritability. In severe cases the following symptoms may occur: derealisation, depersonalisation, hyperacusis, numbness and tingling of the extremities, hypersensitivity to light, noise and physical contact, hallucinations or epileptic seizures.

Since the risk of withdrawal phenomena/rebound insomnia is higher after abrupt discontinuation of treatment, it is recommended that the dosage be decreased gradually (see sections 4.2 and 4.5).

Rebound insomnia

A transient syndrome, whereby the symptoms that led to treatment with DORMICUM, recur in an enhanced form, may occur on withdrawal of hypnotic treatment. It may be accompanied by other reactions including mood changes, anxiety and restlessness.

Since the risk of withdrawal phenomena/rebound phenomena is greater after abrupt discontinuation of treatment, it is recommended that the dosage be decreased gradually.

Duration of treatment

The duration of treatment should be as short as possible (see section 4.2), and should not exceed 2 weeks. The tapering-off process should be tailored to the individual. Extension beyond this period should not take place without re-evaluation of the situation.

It may be useful to inform the patient when treatment is started that it will be of limited duration and explain precisely how the dosage will be progressively decreased. Moreover, it is important that the patient be aware of the possibility of rebound phenomena, thereby minimising anxiety over such symptoms should they occur while DORMICUM is being discontinued.

There are indications that, in the case of DORMICUM, withdrawal phenomena can become manifest within the dosage interval, especially when the dosage is high.

Amnesia

DORMICUM may induce anterograde amnesia. The condition occurs most often several hours after ingesting the medicine and therefore, to reduce the risk, patients should ensure that they will be able to have an uninterrupted sleep of 7 - 8 hours after taking the medication (see section 4.8).

Psychiatric and "paradoxical" reactions

Reactions like restlessness, agitation, irritability, aggressiveness, anxiety, delusion, anger, nightmares, hallucinations, psychosis, inappropriate behaviour and other adverse behavioural effects are known to occur when using DORMICUM and other benzodiazepine-like agents. Should this occur, DORMICUM should be discontinued. They are more likely to occur in children and in the elderly.

Specific patient groups

In elderly and/or debilitated patients, as well as in patients with respiratory or cardiovascular impairment, the recommended dose is 7,5 mg. These patients may be more sensitive to the clinical side effects of DORMICUM, like cardio-respiratory depression. Thus DORMICUM should be used very carefully in these patient populations and if needed a lower dose should be considered.

Benzodiazepines, including DORMICUM, are not indicated for the treatment of patients with severe hepatic insufficiency, as it may precipitate encephalopathy.

DORMICUM is not recommended for the primary treatment of psychotic illness.

DORMICUM should not be used alone to treat depression or anxiety associated with depression, as suicide may occur in such patients.

Concomitant use of alcohol/CNS depressants

The concomitant use of DORMICUM with alcohol and/or central nervous system depressants should be avoided. Such concomitant use increases the clinical effects of DORMICUM, possibly including severe

sedation that could result in coma or death and clinically relevant respiratory and/or cardio-vascular depression (see section 4.5).

DORMICUM should be used with extreme caution in patients with a history of alcohol or drug abuse.

Co-medication with medicines that alter CYP3A activity

DORMICUM pharmacokinetics is altered in patients receiving concomitant medicines that inhibit or induce CYP3A. Consequently the clinical and adverse effects may be increased or decreased respectively (see section 4.5).

Lactose intolerance

Contains lactose. Patients with rare hereditary conditions of galactose intolerance e.g. galactosaemia, lactase deficiency, glucose-galactose malabsorption or fructose intolerance, should not take DORMICUM (see section 4.3).

4.5. Interaction with other medicinal products and other forms of interaction

Pharmacokinetic interaction*:* See sections 4.3 and 4.4.

Because DORMICUM is almost exclusively metabolised by cytochrome P450 3A (CYP3A), modulators of CYP3A have the potential to alter the plasma concentrations and subsequently the clinical effects of DORMICUM.

When co-administered with a CYP3A inhibitor, the clinical effects of oral DORMICUM may be stronger and also last longer. As a result, a lower dose of DORMICUM may be required. Conversely the effect of DORMICUM may be weaker and last shorter when co-administered with a CYP3A inducer and a higher dose may be required.

In case of CYP3A induction and reversible inhibition (so-called mechanism-based inhibition), the effect of the pharmacokinetics of DORMICUM may persist for several days up to a few weeks after administration of the CYP3A modulator. Examples include: clarithromycin, erythromycin, HIV protease inhibitors, delavirdine, calcium channel blockers (e.g. verapamil, diltiazem), kinase inhibitors (e.g. imatinib, lapatinib, idelalisib), or the oestrogen receptor modulator, raloxifene. Ethinylestradiol combined with norgestrel or gestodene did not modify exposure to DORMICUM to a clinically significant degree.

Medicines that inhibit CYP3A

Classification of CYP3A inhibitors: CYP3A inhibitors can be classified according to the strength of their inhibitory effect and to the importance of the clinical modifications when they are administered concomitantly with DORMICUM.

Very strong inhibitors: DORMICUM AUC increased > 10-fold and C_{max} increased > 3-fold. The following medicines fall into this category: ketoconazole, itraconazole, voriconazole, HIV protease inhibitors, including ritonavir boosted protease inhibitors.

Combination of DORMICUM administered orally with very strong CYP3A inhibitors is contraindicated (see section 4.3).

Strong inhibitors: DORMICUM AUC increased by 5 to 10-fold. The following medicines fall into this category: high dose clarithromycin, tyrosine kinase inhibitors (e.g. idelalisib) and the HCV protease inhibitors boceprevir and telaprevir. Concomitant administration of oral DORMICUM with boceprevir or telaprevir is contraindicated (see section 4.3).

Moderate inhibitors: DORMICUM AUC increased by 2 to 5-fold. The following medicines are identified as moderate inhibitors: fluconazole, clarithromycin, telithromycin, erythromycin, diltiazem, verapamil, nefazodone, neurokinin-1 (NK1) receptor antagonists (aprepitant, netupitant, casopitant), tabimoreline and posaconazole.

Combination of DORMICUM with strong and moderate CYP3A inhibitors requires a careful clinical evaluation of conditions that could make the patient particularly sensitive to the potential side effects of DORMICUM (see section 4.4).

Weak inhibitors: DORMICUM AUC increased by 1,25 to < 2-fold. The following medicines and herbals are included in this category: fentanyl, roxithromycin, cimetidine, ranitidine, fluvoxamine, bicalutamide, propiverine, everolimus, ciclosporin, simeprevir, grapefruit juice, echinacea purpurea and berberine as contained in goldenseal.

Concomitant administration of DORMICUM with weak CYP3A inhibitors does usually not lead to a relevant change of DORMICUM clinical effect.

Medicines that induce CYP3A

Patients receiving a combination of DORMICUM with CYP3A inducers may require a higher DORMICUM dose in particular if DORMICUM is co-administered with strong CYP3A inducers. Well known strong CYP3A inducers (≥ 80 % decrease in AUC) include: rifampicin, carbamazepine, phenytoin, enzalutamide and mitotane with its long lasting CYP3A inducing effect, while moderate CYP3A inducers (50 - 80 % decrease in AUC) include St. John's wort and weak inducers (20 - 50 % decrease in AUC) include efavirenz, clobazam, ticagrelor, vemurafenib, quercetin and Panax ginseng.

Pharmacodynamic interactions

The co-administration of DORMICUM with other sedative/hypnotic agents is likely to result in increased sedative/hypnotic effects. Such sedative/hypnotic agents include alcohol, opiates/opioids (when they are used as analgesics, antitussives or substitutive treatments), antipsychotics, other benzodiazepines used as anxiolytics or hypnotics, barbiturates, propofol, ketamine, etomidate; sedative antidepressants, antihistamines and centrally acting antihypertensive medicines. DORMICUM decreases the minimum alveolar concentration (MAC) of inhalational anaesthetics.

Enhanced side effects such as sedation and cardio-respiratory depression may also occur when DORMICUM is co-administered with any centrally acting depressants including alcohol. The combination of alcohol and DORMICUM should be avoided (see section 4.4).

See section 4.9 for warnings on other central nervous system depressants, including alcohol.

Medicines increasing alertness/memory like the AchE inhibitor physostigmine reversed the hypnotic effects of DORMICUM. Similarly, 250 mg of caffeine partly reversed the sedative effect of DORMICUM.

4.6. Fertility, pregnancy and lactation

Pregnancy

Insufficient data is available on DORMICUM to assess its safety during pregnancy. Benzodiazepines, including DORMICUM, should be avoided in pregnancy unless there is no safer alternative.

An increased risk of congenital malformation associated with the use of DORMICUM during the first trimester of pregnancy may occur.

If DORMICUM is prescribed to a woman of childbearing potential, she should contact her medical practitioner regarding discontinuation of DORMICUM, if she intends to become or suspects that she may be pregnant.

If, exceptionally, it is considered by a medical practitioner that administration of DORMICUM during the last three months of pregnancy, or during labour, is essential, effects on the neonate such as irregularities in the foetal heart rate, hypothermia, hypotonia, poor sucking and moderate respiratory depression can be expected, due to the pharmacological action of the product.

Moreover, infants born to mothers who took benzodiazepines chronically during the latter stages of pregnancy may have developed physical dependence and may be at some risk of developing withdrawal symptoms in the postnatal period.

Lactation

DORMICUM is excreted in breast milk. Do not use during lactation.

4.7. Effects on ability to drive and use machines

Sedation, amnesia, impaired concentration and impaired muscular function may adversely affect the ability to drive or use machines.

Prior to receiving DORMICUM, the patient should be warned not to drive a vehicle or operate a machine until completely recovered. The medical practitioner should decide when these activities may be resumed.

If sleep duration is insufficient or if alcohol is consumed, the likelihood of impaired alertness may be increased (see section 4.5).

4.8. Undesirable effects

a. Summary of the safety profile: No text

b. Tabulated list of adverse reactions

The side effects in the following table were reported post-marketing and are presented according to the MedDRA system organ classification. The frequency with which these side effects occur is not known (cannot be estimated from the available data).

Frequency categories are as follows: Very common (≥1/10); common (≥1/100 to <1/10); uncommon (≥1/1,000 to <1/100); rare (≥1/10,000 to <1/1,000); very rare (<1/10,000); not known (cannot be estimated from the available data)

MedDRA SOC	**Adverse reaction (Frequency not known)**
Immune system disorders	Hypersensitivity, Angioedema
Psychiatric disorders	Confusional state, Disorientation, Emotional and mood disturbances, Libido disorder*, Depression Paradoxical reactions: Restlessness, Agitation, Psychomotor hyperactivity, Irritability, Aggression, Delusion, Anger, Nightmare Hallucination, Psychotic disorder, Abnormal behaviour Dependence: Drug dependence, Withdrawal syndrome, Substance abuse
Nervous system disorders	Somnolence (during the day), Headache, Dizziness, Depressed level of consciousness, Ataxia, Anterograde amnesia Premedication: Impact on post-operative sedation
Cardiac disorders	Cardiac failure, Cardiac arrest
Respiratory, thoracic and mediastinal disorders	Respiratory depression
Eye disorders	Diplopia
Gastrointestinal disorders	Gastrointestinal disorder
Skin and subcutaneous tissue disorders	Skin reaction
Musculoskeletal and connective tissue disorders	Muscular weakness
General disorders and administration site conditions	Fatigue
Injury, poisoning and procedural complications	Fall, Fracture

c. Description of selected adverse reactions:

Psychiatric Disorders: The use of the product should be discontinued if paradoxical reactions occur. These effects are more likely to occur in the elderly. Abuse has been reported in poly-drug abusers (see section 4.4).

Nervous System Disorders: These adverse events occur predominantly at the start of therapy and usually disappear with repeated administration. Anterograde amnesia may occur with therapeutic doses, the risk increasing at higher dosages. Amnestic effects may be associated with inappropriate behaviour (see section 4.4).

Eye Disorders: Diplopia occurs predominantly at the start of therapy and usually disappears with repeated use.

Musculoskeletal and Connective Tissue Disorders: Muscle weakness usually occurs predominantly at the start of therapy and disappears with repeated administration.

General Disorders and Administration Site Conditions: Fatigue usually occurs predominantly at the start of therapy and disappears with repeated administration.

Injury, Poisoning and Procedural Complications: The risk of falls and fractures is increased in DORMICUM users taking concomitant sedatives (including alcoholic beverages) and in the elderly.

Reporting of suspected adverse reactions

Reporting suspected adverse reactions after authorisation of the medicine is important. It allows continued monitoring of the benefit/risk balance of the medicine. Health care providers are asked to report any suspected adverse reactions to SAHPRA via the “6.04 Adverse Drug Reactions Reporting Form”, found online under SAHPRA’s publications: https://www.sahpra.org.za/Publications/Index/8.

Patients and Healthcare professionals can contact Pharmaco directly for assistance with reporting adverse events on +27 (0) 11 784 00 77.

4.9. Overdose

Symptoms: Benzodiazepines, including DORMICUM, commonly cause drowsiness, ataxia, dysarthria and nystagmus. Overdose of DORMICUM is seldom life-threatening, if the medicine is taken alone, but

may lead to areflexia, apnoea, hypotonia, hypotension, cardio-respiratory depression and rare cases to coma. Coma, if it occurs, usually lasts a few hours but it may be more protracted and cyclical, particularly in elderly patients. Benzodiazepine respiratory depressant effects are more serious in patients with respiratory disease.

Benzodiazepines, including DORMICUM, increase the effects of other central nervous system depressants, including alcohol.

Treatment: Monitor the patient's vital signs and institute supportive measures as indicated by the patient's clinical state. In particular, patients may require symptomatic treatment for cardio-respiratory effects or central nervous system effects.

If taken orally further absorption should be prevented using an appropriate method (e.g. treatment within 1 - 2 hours with activated charcoal). If activated charcoal is used, airway protection is imperative for drowsy patients. In case of mixed ingestion gastric lavage may be considered, however not as a routine measure.

If CNS depression is severe, consider the use of flumazenil, a benzodiazepine antagonist. This should only be administered under closely monitored conditions. It has a short half-life (about an hour), therefore patients administered flumazenil will require monitoring after its affects have worn off. Flumazenil is to be used with extreme caution in the presence of medicines that reduce seizure threshold (e.g. tricyclic antidepressants). Refer to the professional information for flumazenil, for further information on the correct use of this medicine.

5. PHARMACOLOGICAL PROPERTIES

5.1. Pharmacodynamic properties

Pharmacotherapeutic group: Hypnotics and sedatives (benzodiazepine derivatives), ATC code: N05CD08.

Midazolam is a benzodiazepine. Midazolam has anxiolytic, sedative and hypnotic characteristics as well as possible muscle relaxant and anticonvulsant characteristics.

Mechanism of action

Midazolam is a sleep-inducing agent characterised by a rapid onset and short duration of action. It

also exerts anxiolytic, hypnotic, anticonvulsant and muscle-relaxant effects. Midazolam impairs psychomotor function after single and/or multiple doses.

The central actions of benzodiazepines are mediated through an enhancement of the GABAergic neurotransmission at inhibitory synapses. In the presence of benzodiazepines, the affinity of the gamma-aminobutyric acid (GABA) receptor for the neurotransmitter is enhanced through positive allosteric modulation resulting in an increased action of released GABA on the post-synaptic transmembrane chloride ion flux.

5.2. Pharmacokinetic properties

Absorption

Due to the substantial first-pass effect, the absolute bioavailability of oral midazolam is linear only in the 7,5 - 20 mg oral dose range.

After a single administration of a midazolam 15 mg tablet, maximum plasma concentrations of 70 - 120 ng/mL are reached within one hour. Food prolongs the time to peak plasma concentration by around one hour, pointing to a reduced absorption rate of midazolam. The absorption half-life is 5 - 20 minutes.

Distribution

The tissue distribution of midazolam is very rapid and in most cases a distribution phase is not apparent or is essentially finished within 1 - 2 hours after oral administration. The volume of distribution at steady state is 0,7 - 1,2 L/kg. 96 - 98 % of midazolam is bound to plasma proteins. The major fraction of plasma protein binding is due to albumin. There is a slow and minimal passage of midazolam into cerebrospinal fluid. In humans, midazolam has been shown to cross the placenta and to enter foetal circulation. Midazolam is found in human milk. Midazolam is not a substrate for drug transporters.

Metabolism

Midazolam is almost entirely eliminated by biotransformation. Less than 1 % of the dose is recovered in the urine as unchanged drug. Midazolam is hydroxylated by cytochrome P450, CYP3A isozymes. Both isozymes, CYP3A4 and also CYP3A5 are actively involved in the hepatic oxidative metabolism of midazolam.

The metabolism of midazolam after oral administration relies to a comparable extent on intestinal CYP3A and on hepatic CYP3A.

There are two main oxidised metabolites 1'-hydroxymidazolam (also named α-hydroxymidazolam) and 4-hydroxymidazolam. 1'-hydroxymidazolam is the major urinary and plasma metabolite. 60 - 80 % of the dose is glucuronidated and excreted in the urine form of 1'-hydroxymidazolam conjugate. Plasma concentrations of 1'-hydroxymidazolam may reach 30 - 50 % those of the parent compound. 1'-hydroxymidazolam is pharmacologically active and contributes significantly (about 34 %) to the effects of oral midazolam.

Previous investigation did not show a clinically relevant genetic polymorphism in the oxidative metabolism of midazolam.

Elimination

In healthy young volunteers, the elimination half-life of midazolam ranges between 1,5 to 2,5 hours. Repeated administrations of midazolam do not induce drug-metabolising enzymes. The elimination half-life of 1'-hydroxymidazolam is shorter than 1 hour.

Pharmacokinetics in special populations

Elderly

In elderly male subjects over 60 years of age, the elimination half-life of midazolam was significantly prolonged by a factor 2,5 as compared with younger male subjects. Total midazolam clearance was significantly reduced in male elderly subjects and the bioavailability of the oral tablet was significantly increased.

Patients with hepatic impairment

The pharmacokinetics of midazolam were significantly modified in patients with chronic liver disease including advanced liver cirrhosis. In particular, as a consequence of decreased liver clearance, the elimination half-life was prolonged and the absolute bioavailability of oral midazolam was significantly increased in cirrhotic patients compared to control.

Patients with renal impairment

The pharmacokinetics of midazolam are not altered in patients with severe renal impairment. However the major midazolam metabolite, 1'-hydroxymidazolam glucuronide, which is excreted through the kidney, accumulates in patients with severe renal impairment. This accumulation produces a prolonged sedation. Oral midazolam should therefore be administered carefully in patients with renal impairment and titrated to the desired effect.

Obese patients

In obese patients the volume of distribution of midazolam is increased. As a consequence, the main elimination half-life of midazolam is longer in obese than in non-obese patients (5,9 hours vs 2,3 hours). The oral bioavailability of the midazolam tablet was not different in obese patients compared to non-obese patients.

6. PHARMACEUTICAL PARTICULARS

6.1. List of excipients

DORMICUM tablets contain midazolam maleate equivalent to 7,5 mg or 15 mg midazolam.

Excipients:

DORMICUM 7,5 mg Tablets: lactose anhydrous, hypromellose, magnesium stearate, microcrystalline cellulose, pregelatinised starch, purified water, talc and titanium dioxide (CI 77891).

DORMICUM 15 mg Tablets: carmellose sodium, lactose anhydrous, hypromellose, indigo carmine (CI 73015), macrogol, magnesium stearate, maize starch, microcrystalline cellulose, polyacrylate dispersion, purified water, talc and titanium dioxide (CI 77891).

6.2. Incompatibilities

Not applicable

6.3. Shelf life

DORMICUM 7,5 mg: 60 months

DORMICUM 15 mg: 60 months

6.4. Special precautions for storage

Store at or below 30 °C. Protect from light and moisture.

Store in outer container until required for use.

Store out of reach of children.

Any unused product or waste material should be disposed of.

6.5. Nature and contents of container

DORMICUM 7,5 mg: blister packs of 20's, 30's and 90's.

DORMICUM 15 mg: blister packs of 20's, 30's and 90's.

Not all packs may be marketed.

6.6. Special precautions for disposal and other handling

Any unused product or waste material should be disposed of in accordance with local requirements.

7. HOLDER OF THE CERTIFICATE OF REGISTRATION

Pharmaco Distribution (Pty) Ltd.

3 Sandown Valley Crescent

South Tower, First Floor

Sandton 2196, Gauteng

South Africa

Ethical assistance Line: +27 (0) 784 00 77

8. REGISTRATION NUMBER(S)

DORMICUM 7,5 mg: 27/2.2/0078

DORMICUM 15 mg: R/2.2/123

9. DATE OF FIRST AUTHORISATION/RENEWAL OF THE AUTHORISATION

Registration: DORMICUM 7,5 mg: 18 January 1993; DORMICUM 15 mg: 14 August 1984

10. DATE OF REVISION OF THE TEXT

Last revision: 2022-08-26

DORMICUM 15 mg	
NAMIBIA Reg. No. 90/2.2/001403	NS3