NEW ZEALAND DATA SHEET

Biodone 2 mg/mL Oral solution
Biodone Forte 5 mg/mL Oral solution
Biodone Extra Forte 10 mg/mL Oral solution

1 PRODUCT NAME

Biodone, 2 mg/mL, Oral solution
Biodone Forte, 5 mg/mL, Oral solution
Biodone Extra Forte, 10 mg/mL, Oral solution

References to “Biodone” in this data sheet refer to all strengths.

2 QUALITATIVE AND QUANTITATIVE COMPOSITION

Methadone hydrochloride 2 mg/mL, 5 mg/mL, 10 mg/mL

For the full list of excipients, see section 6.1.

3 PHARMACEUTICAL FORM

Biodone is an oral solution containing methadone hydrochloride BP and is presented in 200 mL amber glass bottles with tamper-evident caps or in amber plastic bottles with an induction seal under the cap as tamper evidence. Biodone is yellow (due to sunset yellow), Biodone Forte is colourless and Biodone Extra Forte is red (due to permicol red, a diluted form of amaranth). There are no other additives or excipients in Biodone solutions.

4 CLINICAL PARTICULARS

4.1 Therapeutic indications

Oral Methadone is used in detoxification and maintenance treatment as a substitute for heroin or other morphine-like drugs to suppress the opiate-agonist abstinence syndrome in patients who are dependent on these drugs.

4.2 Dose and method of administration

Biodone is to be administered by the oral route only. Initial doses should be based on the individual's history of quantity, frequency and route of administration of opiates and should also take into account the person's hepatic and renal functioning. There may be some withdrawal symptoms not covered by the first dose. Initial doses will generally be in the range of 15-35 mg per day and should never be higher than 40 mg.

Subsequent dosage should be adjusted according to the requirements and response of the patient. Stabilisation of maintenance dosage usually occurs at 60-120 mg daily although a higher dosage is sometimes required. A single dose of Biodone daily usually adequately maintains the patient and there generally is no apparent advantage to divided doses. However, rapid metabolisers of methadone may not maintain adequate plasma methadone concentrations with usual dosing regimens. Maintenance dosage requirements should be reviewed regularly and reduced as indicated.

The dose of Biodone required is to be measured accurately, using a calibrated dropper or other appropriate method.

Biodone should be used in accordance with “Opioid Substitution Treatment, New Zealand Practice Guidelines” published by the Ministry of Health, February 2003. Substantial portions of this data sheet have been prepared from this publication, which may be downloaded from the publications page of www.moh.govt.nz.

4.3 Contraindications

Biodone is contraindicated in the following situations:

- individuals who are hypersensitive to methadone or to either of the colours sunset yellow (Biodone) or permicol red (Biodone Extra Forte) which are the only components in the formulations.
- in patients with respiratory depression, especially in the presence of cyanosis and excessive bronchial secretions.
- during an attack of bronchial asthma.
- in the presence of acute alcoholism, head injury and raised intracranial pressure.
- in individuals receiving monoamine oxidise inhibitors or within 14 days of stopping such treatment.
- in patients with ulcerative colitis, since it may precipitate toxic dilation or spasm of the colon.
- in patients with severe hepatic impairment as it may precipitate hepatic encephalopathy.
- in biliary and renal tract spasm.

4.4 Special warnings and precautions for use

Biodone may interfere with evaluation of CNS function, thereby masking the patient's clinical course. Patients with reduced blood volume may be more sensitive to the hypotensive effects of Biodone than other patients. The use of Biodone in patients with chronic ulcerative colitis may stimulate motility in the colon; in patients with acute ulcerative colitis, toxic dilation may occur. Biodone induced increase in intraluminal pressure may endanger surgical anastomosis. Biodone may obscure the diagnosis or clinical course in patients with acute abdominal conditions. Plasma amylase and lipase determinations should not be performed within 24 hours after a Biodone dose, which may increase the activity of these markers. Patients with prostatic hypertrophy or urethral stricture may be more prone to urinary retention and oliguria than other patients. Biodone may increase the risk of water intoxication in postoperative patients because of the stimulation of the release of vasopressin, suppression of gonadotrophic function may cause impotence and a decline in libido. Biodone may have a prolonged duration and cumulative effect in patients with hepatic or renal dysfunction.

Profound sedation, respiratory depression, coma, and death may result from the concomitant use of Biodone with benzodiazepines or other CNS depressants (e.g., non-benzodiazepine sedatives/hypnotics, anxiolytics, tranquilizers, muscle relaxants, general anaesthetics, medicines with antihistamine-sedating actions such as antipsychotics, other opioids, alcohol). Because of these risks, reserve concomitant prescribing of these drugs for use in patients for whom alternative treatment options are inadequate.

Observational studies have demonstrated that concomitant use of opioid analgesics and benzodiazepines increases the risk of medicine-related mortality compared to use of opioid analgesics alone. Because of similar pharmacological properties, it is reasonable to expect similar risk with the concomitant use of other CNS depressant drugs with opioid analgesics [see Section 4.5].

If the decision is made to prescribe a benzodiazepine or other CNS depressant concomitantly with an opioid analgesic, prescribe the lowest effective dosages and minimum durations of concomitant use. In patients already receiving an opioid analgesic, prescribe a lower initial dose of the benzodiazepine or other CNS depressant than indicated in the absence of an opioid, and titrate based on clinical response. If an opioid analgesic is initiated

in a patient already taking a benzodiazepine or other CNS depressant, prescribe a lower initial dose of the opioid analgesic, and titrate based on clinical response. Follow patients closely for signs and symptoms of respiratory depression and sedation.

Advise both patients and caregivers about the risks of respiratory depression and sedation when Biodone is used with benzodiazepines or other CNS depressants (including alcohol and illicit drugs). Advise patients not to drive or operate heavy machinery until the effects of concomitant use of the benzodiazepine or other CNS depressant have been determined. Screen patients for risk of substance use disorders, including opioid abuse and misuse, and warn them of the risk for overdose and death associated with the use of additional CNS depressants including alcohol and illicit drugs [see Section 4.5].

Cardiac conduction effects
Laboratory studies, both in vitro and in vivo, have demonstrated that methadone inhibits cardiac potassium channels and prolongs the QT interval. Cases of QT interval prolongation and serious arrhythmia (torsades de pointes) have been observed during treatment with methadone. These cases appear to be more commonly associated with, but not limited to, higher dose treatment (>100 mg/day). Most cases involve patients being treated for pain with large, multiple daily doses of methadone although cases have been reported in patients receiving doses commonly used for maintenance treatment of opioid addiction.

Methadone should be administered with particular caution to patients already at risk for development of prolonged QT interval. These risks include cardiac hypertrophy, history of cardiac conduction abnormalities, advanced heart disease or ischaemic heart disease, liver disease, family history of sudden death, hypokalaemia, hypomagnesaemia), concomitant treatments with medicines that have a potential for QT prolongation, concomitant treatment with medicines which may cause electrolyte abnormalities (e.g. diuretics) and concomitant treatment with CYP3A4 inhibitors.

QT prolongation has also been reported in patients with no prior cardiac history who have received high doses of methadone. Patients developing QT prolongation while on methadone treatment should be evaluated for the presence of modifiable risk factors, such as concomitant medications with cardiac effects, drugs which might cause electrolyte abnormalities, and drugs which might act as inhibitors of methadone metabolism.

In patients with recognised risk factors of QT prolongation, or in case of concomitant treatment with medicines that have a potential for QT prolongation, ECG monitoring is recommended prior to methadone treatment, at dose stabilisation, after dose increases, or after any potentially interacting medicine. In patients without recognised risk factors for QT prolongation, ECG monitoring is recommended before dose titration above 100 mg/day, and at seven days after titration.

For use of methadone to treat pain, the risk of QT prolongation and development of dysrhythmias should be weighed against the benefit of adequate pain management and the availability of alternative therapies.

Effects on Children
The fatal dose of methadone in children is 10 – 20 mg. Symptoms of opioid overdose in children are similar to those in adults, with pupillary miosis; however, the pupils may be normoreactive or, rarely, fixed and dilated. Infants may have drowsiness, coma and apnoea.

Children are usually, but not always, symptomatic. Known or suspected methadone intoxicated children should be hospitalised, since respiratory depression may be observed as long as 48 hours after ingestion. Successful resuscitation with a narcotic antagonist may be followed by relapse. Treatment must include establishment of an airway, maintenance of adequate respiratory ventilation, precise supportive care to maintain fluid and electrolyte balance, naloxone, emptying of upper and lower gastrointestinal tracts, and prevention of aspiration of gastric contents.

Use in the Elderly
Methadone has a long plasma half-life, which may lead to accumulation, particularly if renal function is impaired (see Renal Impairment). In common with other opioids, methadone may cause confusion in this age group, therefore careful monitoring is advised.

Hepatic Impairment
Particular care should be taken when Biodone is to be used in patients with hepatic impairment as these patients metabolise methadone more slowly than normal patients. Where not contraindicated methadone should be given at less than the normal recommended dose and the patient's response used as a guide to further dosage requirements [see Section 4.3].

Renal Impairment
Biodone should be used with caution in patients with renal dysfunction.

Effects on laboratory tests
The serum BSP retention test may be increased (hepatotoxic effect or spasm of sphincter of Oddi). Plasma cortisol may be increased in response to cold to an extent not seen in controls. An increase in the serum albumin, prolactin and immunoglobulin IgG levels may be seen as a response to chronic administration. A significant decrease in serum indocyanine green level has been observed in a small series of patients with normal liver function tests. PCO2 may be increased due to decreased pulmonary ventilation. Physiological changes in thyroid hormones may be seen – decrease in serum thyroxine (T4), a decrease in free thyroxine and an increase in tri-iodothyronine (T3).

4.5 Interaction with other medicines and other forms of interaction

The drugs listed below are known to affect methadone metabolism and should therefore be used with caution by those being treated with Biodone.

NOTE: Those with hepatitis C may have impaired liver function. This needs to be taken into account when the use of drugs metabolised by the liver is considered. The dose of paracetamol, for example, needs to be well within the standard 4 g per day.

Drug	Status of effect	Interaction	Mechanism
Alcohol	Clinically important	Increased sedation, increased respiratory depression; combination may also have increased hepatotoxic potential	Additive central nervous system depression
Benzodiazepines	Clinically important	Enhanced sedative effect	Additive CNS depression
Buprenorphine	Clinically important	Antagonistic effect or	Partial agonist of

		enhanced sedative and respiratory depression.	opiate receptors.
Carbamazepine	Clinically important	Reduced methadone levels	Stimulates hepatic enzymes involved in methadone metabolism.
Chlormethiazole	Clinically important	Enhanced sedative effect.	Additive CNS depression
Cimetidine	Two cases have been shown in patients taking methadone as analgesia.	Possible increase in methadone plasma levels.	Inhibits hepatic enzymes involved in methadone metabolism.
Cisapride, domperidone, metoclopramide	Theoretical	Theoretically might increase the speed of onset of methadone absorption, but not the extent.	Possibly by reversing the delayed gastric emptying associated with opioids.
Cyclizine and other sedating antihistamines	Clinically important	Anecdotal reports of injection of cyclizine with opioids causing hallucinations.	Additive psychoactive effects; antimuscarinic effects at high doses.
Desipramine	Clinically important	Raised desipramine levels by up to a factor of two	Unknown interaction not seen with other tricyclic antidepressants.
Other tricyclic antidepressants	Theoretical	Enhanced sedative effect, which is dependent.	Additive CNS dose depression.
Erythromycin	In theory should interact but combination has not been studies.	Increase in methadone levels.	Decreased methadone metabolism.
Fluconazole	In theory the same as ketokonazole		
Fluoxetine	Clinically important	Raised methadone levels but not as significant as for fluvoxamine.	Decreased methadone metabolism.
Fluvoxamine, other SSRI	Clinically important theoretical	Raised plasma methadone levels.	Decreased methadone metabolism.
Grapefruit juice	Should interact in theory and there have been several anecdotal reports.	Raised methadone levels.	Decreased methadone metabolism.
Indinavir	Clinically important	Raised methadone levels	Decreased methadone metabolism.
Ketoconazole	Clinically important	Raised methadone	Decreased

		levels.	methadone metabolism.
MAOI (including selegiline and moclobemide.	Severe with pethidine though unlikely with methadone and has never been described.	CNS excitation delirium, hyperpyrexia, convulsions hypotension or respiratory depression.	Unclear; avoid the combination if possible.
Naltrexone	Clinically important	Blocks effect of methadone (long acting).	Opioid agonist – competes for opiate receptors.
Naloxone	Clinically important	Blocks effect of methadone (long acting), but may be needed if overdose suspected.	Opioid agonist – competes for opiate receptors.
Nevirapine	Clinically important	Decreased methadone levels.	Increased methadone metabolism.
Nifedipine	Has been demonstrated *in vitro* only.	Increased nifedipine levels; no effect on methadone levels.	Methadone decreases the metabolism of nifedipine.
Omeprazole	To date, demonstrated only in animals.	Increased methadone levels.	Possibly affects methadone absorption form the gut.
Phenobarbitone	Clinically important	Reduced methadone levels; increased sedation additive CNS depression	Barbiturates stimulate hepatic enzymes involved in methadone metabolism
Phenytoin	Clinically important	Reduced methadone levels	Phenytoin stimulates hepatic enzymes involved in methadone metabolism
Rifampicin	Very important: most patients are likely to be affected	Reduced methadone levels	Rifampicin stimulates hepatic enzymes involved in methadone metabolism
Rifabutin	Occasionally clinically important	Decreased methadone levels.	Increased methadone metabolism
Ritonavir	Clinically important	Ritonavir may increase plasma methadone levels	Inhibits methadone metabolism
Other protease inhibitors	Theoretical	May raise or lower plasma methadone	Inhibits methadone metabolism

		levels	
Urine acidifiers (e.g. ascorbic acid / vitamin C)	Clinically important	Reduced plasma methadone levels	Raised urinary excretion of methadone
Urine alkalinisers (e.g. sodium bicarbonate)	Clinically important	Increased plasma methadone levels	Reduced urinary excretion of methadone
Zidovudine	Clinically important	Raised plasma levels of zidovudine; no effect on methadone levels	Unknown
Zopiclone	Clinically important	Enhanced sedative effects	Additive CNS depression
Other opioids		Enhanced sedative effect	Additive CNS depression; enhanced respiratory depression
Other CNS depressant drugs (e.g. neuroleptics, hyoscine)	Clinically important	Enhanced sedative effects, which are dose dependent	Additive CNS depression

Benzodiazepines and other Central Nervous System (CNS) Depressants	
Clinical Impact	Due to additive pharmacologic effect, the concomitant use of benzodiazepines or other CNS depressants including alcohol, increases the risk of respiratory depression, profound sedation, coma, and death.
Intervention	Reserve concomitant prescribing of these drugs for use in patients for whom alternative treatment options are inadequate. Limit dosages and durations to the minimum required. Follow patients closely for signs of respiratory depression and sedation [see Section 4.4 Warnings and Precautions].
Examples	Benzodiazepines and other sedatives/hypnotics, anxiolytics, tranquilizers, muscle relaxants, general anaesthetics, drugs with antihistamine-sedating actions such as antipsychotics, other opioids, alcohol.

4.6 Fertility, pregnancy and lactation

Use in pregnancy
Methadone maintenance therapy during pregnancy can improve the health of the mother and the chances of a full-term healthy baby. The main risk to the health of the foetus is for the pregnant woman to start and stop opiate use, particularly where this precipitates the opioid withdrawal syndrome. Women receiving Biodone during their pregnancy should be under the care of a specialist midwifery drug and alcohol service or a General Practitioner approved or authorised to prescribe controlled drugs for the treatment of dependence under the Misuse of Drugs Act 1975. The clinician should ensure that pregnant women have information regarding the effect of methadone maintenance therapy and illicit opioid use with nicotine, alcohol and other drug use on the foetus. Neonates should be observed closely for signs of respiratory depression if the mother has received methadone during labour.

Use in lactation
Methadone is transferred into breast milk at very low levels. Caution should be exercised when methadone is administered to a breastfeeding mother due to the risks of sedation and respiratory depression in the infant.

Breastfeeding mothers receiving methadone for opioid substitution treatment should be under specialist care from obstetric and paediatric staff with experience in monitoring for neonatal abstinence syndrome. The baby should be monitored for sedation and poor feeding particularly during the first three weeks of life. Breastfeeding mothers should receive specific information on how to identify respiratory depression and sedation in their babies and when it may be necessary to seek immediate medical care.

Breastfeeding mothers should be advised to wean slowly off breastfeeding when they decide to stop to reduce the possibility of withdrawal symptoms in the baby.

The evaluation of the risks and benefits of breastfeeding while on methadone for opioid substitution treatment should be done jointly by the prescriber and patient.

4.7 Effects on ability to drive and use machines
Patients who are well stabilised may continue to drive and operate machinery as part of their normal activities. If Biodone dosage has not been stabilised or is changed for any reason caution must be exercised due to the potential for increased drowsiness and other side effects.

4.8 Undesirable effects
The most frequently observed adverse reactions include lightheadedness, dizziness, sedation, nausea, vomiting, and sweating. These effects seem to be more prominent in ambulatory patients and in those who are not suffering severe pain. In such individuals, lower doses are advisable.

The major hazards of methadone are respiratory depression and, to a lesser degree, systemic hypotension. Respiratory arrest, shock, cardiac arrest, and death have occurred.

Other adverse reactions include the following:

Body as a whole: Weakness, oedema, headache

Cardiovascular: Arrhythmias, bigeminal rhythms, bradycardia, cardiomyopathy, ECG abnormalities, extrasystoles, flushing, heart failure, hypotension, palpitations, phlebitis, QT interval prolongation, syncope, T-wave inversion, tachycardia, torsades de pointes, ventricular fibrillation, ventricular tachycardia.

Endocrine: Hypogonadism

Gastrointestinal: Abdominal pain, anorexia, biliary tract spasm, constipation, dry mouth, glossitits.

Heamtologic: Reversible thrombocytopenia has been described in opioid addicts with chronic hepatitis.

Metabolic: Hypokalaemia, hypomagnesaemia, weight gain.

Musculoskeletal: Decreased muscle mass and strength, osteoporosis, and fractures.

Nervous system disorders: raised intracranial pressure, sedation.

Psychiatric: agitation, changes of mood, dependence, disorientation, dysphoria, euphoria, hallucinations, insomnia.

Renal: Antidiuretic effect, urinary retention or hesitancy.

Reproductive: Amenorrhoea, reduced libido and/ or potency, reduced ejaculate volume, reduced seminal vesicle and prostate secretions, decreased sperm motility, abnormalities in sperm morphology.

Respiratory: Pulmonary oedema, respiratory depression

Skin and subcutaneous tissue: Pruritus, other skin rashes, and rarely, hemorrhagic urticaria.

Maintenance on a stabilised dose: During prolonged administration of methadone, as in a methadone maintenance programme, constipation and sweating often persist and hypogonadism, decreased serum testosterone and reproductive effects are thought to be related to chronic opioid use.

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. Healthcare professionals are asked to report any suspected adverse reactions https://nzphvc.otago.ac.nz/reporting/

4.9 Overdose
For non-tolerant adults, doses of 50 mg or less have been known to be fatal, including doses taken orally. Potentially lethal overdoses of methadone can occur within 30 minutes to six hours after ingestion by non-tolerant or partially tolerant individuals.

Signs of methadone intoxication in an adult include pinpoint pupils, hypothermia, respiratory depression, bradycardia, pulmonary oedema (not always), hypotension, coma and seizures. Rhabdomyolysis, myoglobinuria, muscle necrosis and renal failure may occur secondary to methadone intoxication and may result from muscle damage related to prolonged coma and immobilisation or from a direct toxic effect of methadone.

To treat overdose, ensure that the airway is clear and perform emergency cardiopulmonary resuscitation as necessary. Take the person to hospital as soon as possible where treatment with an infusion of naloxone can be commenced. Overdose patients should remain in hospital for 24 – 72 hours due to methadone's long half-life.

For advice on the management of overdose please contact the National Poisons Centre on 0800 POISON (0800 764766).

5 PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic properties

Pharmacotherapeutic group: Drugs used in opioid dependence ATC Code: N07BC02

Methadone hydrochloride is an opiate agonist and exerts its principal pharmacological effect on the CNS and on the intestines. It interacts at specific receptor binding sites in the CNS and other tissues. Several subtypes of opiate receptors have been described including the μ type. Methadone interacts with the μ receptor to suppress opiate withdrawal. Methadone acts at several sites within the CNS involving several systems of neurotransmitter to produce analgesia, but the precise mechanism of action has not been fully elucidated. Methadone alters the perception of pain at the spinal cord and higher levels in the CNS and the patient's emotional response to pain.

In addition to analgesia, the effects of Methadone on the CNS cause suppression of the cough reflex, respiratory depression, drowsiness, sedation, change in mood, euphoria, dysphoria, mental clouding, nausea and vomiting, and EEG changes. Methadone causes miosis which is antagonized by atropine.

Methadone increases smooth muscle tone in the urinary tract and induces spasms. In the urinary bladder, tone of the detrusor muscle is increased, possibly resulting in urinary urgency. Methadone also increases the tone of the vesical sphincter, which may make urination difficult. These effects, in conjunction with the central effects of the drug on the release of vasopressin, may produce oliguria. Methadone has little cardiovascular effect when given in therapeutic doses to supine patients. When the patient assumes a "head-up" position, orthostatic hypotension and fainting may occur as a result of peripheral vasodilation. This peripheral vasodilation may be caused by opiate agonist-induced release of histamine or by depression of the vasomotor centre in the medulla. Methadone exerts endocrinologic effects, some of which may be related to CNS effects. Methadone generally simulates the release of vasopressin and inhibits the release of corticotropin, gonadotropins and thyrotropin.

5.2 Pharmacokinetic properties

Methadone hydrochloride is readily absorbed after administration by mouth and has high oral bioavailability. Peak plasma concentrations occur at 4 hours, but this varies widely among individuals. Methadone undergoes considerable tissue distribution and protein binding is reported to be 60 to 90% with a1,-acid glycoprotein being the main binding protein in the plasma. The volume of distribution is 5 l/kg. Metabolism to the major metabolite 2-ethylidine-1,5-dimethyl, 3,3-diphenylpyrrolidine and the minor metabolite 2-ethyl-3,3-diphenyl-5-methyplyrrolidine, both of them inactive, occurs in the liver. These metabolites are excreted in the faeces and urine together with unchanged methadone. Other metabolites, including methadol and normethadol (reported to be pharmacologically active), have also been described but account for a small proportion of the dose. The liver may also serve as a major storage site of unchanged methadone, which is taken up, bound non-specifically by the liver and released again mainly unchanged.

Marked inter-individual variations in kinetics have been observed with methadone. Elimination half-lives vary considerably (a range of 15 to 60 hours has been reported) and careful adjustment of dosage is necessary with repeated administration, after which there is a gradual accumulation in the tissues.

Plasma concentrations have been found to vary widely during methadone maintenance therapy with large differences between patients and wide fluctuations in individual patients. Declining concentrations have been reported during methadone maintenance suggesting that tolerance occurs, possibly as a result of auto-induction of hepatic microsomal enzymes.

5.3 Preclinical safety data
No information held by the sponsor.

6 PHARMACEUTICAL PARTICULARS

6.1 List of excipients
Biodone - Sunset yellow FCF, Purified Water
Biodone Forte – Purified Water
Biodone Extra Forte – Amaranth, Purified Water

6.2 Incompatibilities
Biodone is incompatible with alkaline solutions, which cause precipitation.

6.3 Shelf life
Biodone – Amber glass bottle, 12 months from date of manufacture; Plastic HDPE bottle – 24 months from date of manufacture

Biodone Forte – Amber glass bottle, 12 months from date of manufacture; Plastic HDPE bottle – 24 months from date of manufacture

Biodone Extra Forte – Amber glass bottle, 12 months from date of manufacture, Plastic HDPE bottle – 24 months from date of manufacture

6.4 Special precautions for storage
Store Biodone in a cool, dry place below 25°C, in a controlled drug safe, but do not refrigerate. Pharmacists should emphasise the importance of storing takeaway doses in a cool place, out of sight from, and out of the reach of children. They should be locked away if possible. Always keep Biodone in the original container and do not use it if the expiry date on the container has run out.

6.5 Nature and contents of container
Biodone – 100 mL, 200 mL amber glass bottle; 200 mL Plastic HDPE bottle
Biodone Forte – 200 mL amber glass bottle; 200 mL Plastic HDPE bottle
Biodone Extra Forte – 200 mL amber glass bottle; 200 mL Plastic HDPE bottle

Not all pack sizes may be available.

6.6 Special precautions for disposal <and other handling>
Methadone hydrochloride is a controlled drug (B3) and must be stored in a controlled drug safe. All usage must be recorded in a controlled drug register.

Takeaway doses are to be dispensed as individual daily doses with each day's dose packed in appropriately labelled bottles with child-resistant closures. The requirement for child resistant closures may be omitted if the prescriber has endorsed (or the pharmacist annotates) the prescription not to be dispensed in a container with a safety cap because of

patient infirmity. Pharmacists should ensure that those receiving Biodone can open and close the CRCs correctly and are aware of the need for them.

7 MEDICINE SCHEDULE

Controlled Drug (B3)

8 SPONSOR

Biomed Limited
52 Carrington Road
Point Chevalier
Auckland

Phone: 0800 833 133

9 DATE OF FIRST APPROVAL

Date of publication in the New Zealand Gazette of consent to distribute the medicine:
Biodone – 17 April 1997
Biodone Forte, Biodone Extra Forte – 07 February 1997

10 DATE OF REVISION OF THE TEXT

9 May 2019

SUMMARY TABLE OF CHANGES

DATE	CHANGE
May-19	Update as per MARC recommendation letter dated 30/04/19 Section 4.6 - Use in Lactation