CMEC 36
Complementary Medicines Evaluation Committee

Extracted Ratified Minutes
Thirty Sixth Meeting
26 July 2002

Abbreviations:
ADEC Australian Drug Evaluation Committee
ADRA Adverse Drug Reactions Advisory Committee
ADRU Adverse Drug Reactions Unit (of TGA)
ANZFA Australia New Zealand Food Authority
AQIS Australian Quarantine Inspection Service
ARTG Australian Register of Therapeutic Goods
ASMI Australian Self Medication Industry
BP British Pharmacopoeia
BSE Bovine spongiform encephalopathy
CHC Complementary Healthcare Council of Australia
CK Creatine kinase
CPK Creatine phosphokinase
CMEC Complementary Medicines Evaluation Committee
DSEB Drug Safety and Evaluation Branch
ELF Electronic Lodgement Facility
EP European Pharmacopoeia
GRB Geographical Risk of BSE
JHTF Joint TGA/Industry Herbal Task Force
L0AEL Lowest Observable Adverse Effect Level
MEC Medicines Evaluation Committee
NDPSC National Drugs and Poisons Schedule Committee
NOAEL No Observable Adverse Effect Level
OCM Office of Complementary Medicines
PBS Pharmaceutical Benefits Scheme
SUSDP Standard for the Uniform Scheduling of Drugs and Poisons
TGA Therapeutic Goods Administration
TGAL Therapeutic Goods Administration Laboratory Branch
TSE Transmissible spongiform encephalopathies
The thirty sixth meeting of the Complementary Medicines Evaluation Committee was held at the Kingsford Room, Stamford Hotel Sydney Airport, Sydney between 9:35 am and 4.30 pm on Friday 26 July 2002.

Members of CMEC present were:

Professor Tony Smith (Chair)
Mr Nick Burgess
Dr Roberta Chow
Dr Colin Duke
Ms Val Johanson
Professor Stephen Myers
Mr Kevin Ryan
Professor Bill Webster

Present from the TGA were:

Dr Fiona Cumming
Dr John Hall
Dr John McEwen
Dr Jennifer Elijah

1. **Procedural Matters**

1.1 **Opening of Meeting**

The Chairman opened the meeting at 9:35 am and welcomed members and TGA staff.

1.2 **Apologies**

Dr Joachim Fluhrer and Dr Heather Yeatman

1.3 **Conflict of Interest**

Members submitted conflict of interest declarations specific to agenda items for this meeting.

2. **Confirmation of Minutes of CMEC 35 (14 June 2002)**

The minutes of the thirty-fifth meeting of CMEC as amended were accepted as an accurate record of proceedings.

*CMEC Recommendation:*

Members made the following recommendation:

<table>
<thead>
<tr>
<th>Recommendation 36.1</th>
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<tbody>
<tr>
<td>CMEC confirms that the draft Minutes of its previous meeting (CMEC 35, 14 June 2002), as amended, are a true and accurate record of that previous meeting.</td>
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</table>
3. **Guidelines on levels and kinds of evidence to support claims for therapeutic goods (Guidelines)**

No matters were considered under this agenda item.

4. **CMEC Working Party on Herbal Medicine Issues**

No matters were considered under this agenda item.

5. **Action Arising from Previous Meetings**

5.2 **Caffeine labelling – Progress report**

*Background*

At CMEC 34 (May 2002), the Committee was provided with details of the caffeine content of a number of listable herbal ingredients. CMEC members considered the contents of caffeine in a range of herbal plants and discussed the implications of caffeine content in homoeopathic medicines and made the following recommendation:

**Recommendation 34.4**

CMEC recommends to the TGA that labels on products containing those herbs identified as containing caffeine be required to declare:

- that the goods contain caffeine; and
- the total quantity of caffeine per dosage unit.

**Homoeopathic medicines more dilute than 1X (mother tinctures) are exempt from this requirement.**

*Present discussion*

The Committee was advised that approximately 700 products were identified as containing caffeine-containing herbal ingredients on the ARTG. There were a number of issues that needed to be considered to ensure Recommendation 34.4 was efficiently and effectively implemented:

- *Identification of products with caffeine-containing herbal ingredients:*
  
  It was difficult to know which herbs contain or do not contain caffeine. Although, a list of plant sources that are reported to contain caffeine was included in the papers presented to CMEC 34, no such list can be exhaustive.

  CMEC members considered the question of whether the sponsors of all herbal products should be required to test their product(s) to ensure they do not contain caffeine, even if they do not contain herbs from the above mentioned list. It was noted that caffeine-containing plants are well known and it would not be difficult to limit testing to a few plant families. Members agreed that there was no need for all herbal products to be tested for caffeine, but it was suggested that there be ongoing vigilance to ensure an awareness of all of the plants that are known to contain caffeine.
• **Identifying homoeopathic products not exempt from the proposed caffeine labelling requirement**

It was noted that homoeopathic medicines more concentrated than a 1/10 dilution, (mother tincture in the Homoeopathic Pharmacopoeia of the United States [HPUS]), which have caffeine as a constituent require identification for labelling purposes. This could result in a large number of these products requiring testing to ensure that they do not contain caffeine.

The term ‘mother tincture’ was clarified. Homoeopathic remedies begin with part of the original substance dissolved in alcohol, called the mother tincture. The mother tincture is then diluted many times (under the French Pharmacopoeia). A 1X potency may be created by mixing one part of the mother tincture and nine parts alcohol or distilled water.

The recommendation made in relation to homoeopathic preparations by CMEC at the last meeting needed further clarification since there is a divergence of views as to whether 1X always constitutes a ‘mother tincture’.

Members agreed that it would be appropriate to amend this recommendation for homoeopathic medicines to ‘Homoeopathic medicines more dilute than 1X are exempt from this requirement’.

The Committee was advised that approved homoeopathic products containing caffeine were very few and well known and Members agreed that there was no need for all homoeopathic medicines to be tested for caffeine.

• **Quantification of caffeine/Production of new labels**

Members suggested that the label of caffeine containing herbal medicine state that ‘This product contains caffeine. Contact the sponsor if you need further information’. Consumers could then make a decision about consuming products that contain caffeine.

Members agreed that the labelling requirement on products containing those herbs identified as containing caffeine, need not include the total quantity of caffeine per dosage unit. However, the label of the products containing *Paullinina cupana* (guarana) should continue to include a statement alerting consumers to the fact that *Paullinina cupana* contain caffeine and the amount of caffeine, in milligrams, per unit dose.

CMEC decided in the light of this discussion to rescind Recommendation 34.4.

**CMEC Recommendation**

The CMEC resolved to recommend that:

<table>
<thead>
<tr>
<th>Recommendation 36.2</th>
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<tbody>
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<td>CMEC rescinded Recommendation 34.4 and made the following recommendation:</td>
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6. Evaluation of New Substances

6.1 Boron

Background
To assist with Trans Tasman harmonisation initiatives the National Drugs and Poisons Schedule Committee (NDPSC) recently amended the scheduling for boron. This amended Schedule 4 entry for boron for human therapeutic use now excludes preparations for:
- internal use containing 3 milligrams or less per recommended daily dose;
- paediatric dermal use containing 0.35 per cent or less of boron; and
- dermal use containing 0.35 per cent or less of boron except antifungal preparations.
This means that upper limits for boron content in listable complementary medicines are already in place by virtue of the revised scheduling arrangements.

Summary of evaluation report

Seven products containing boric acid, B(OH)₃, and two products containing borax, Na₂B₄O₇·10H₂O, as active ingredients are registered on the Australian Register of Therapeutic Goods (ARTG). The products are in the form of liquids, creams, ointments, tablet and powder.

Boron in the form of borates occurs naturally in the Earth's crust and oceans. Boron is not found in the elemental form in nature. Borates have wide industrial uses. Boric acid and borax are the main compounds used in cosmetics and therapeutic goods. Monomeric boric acid, B(OH)₃, is the predominant species in biological tissues and is a very weak acid (pKₐ = 9.15).

The chemical and toxicological properties of borax pentahydrate (Na₂B₄O₇·5H₂O), borax (Na₂B₄O₇·10H₂O), boric acid, and other borates are expected to be similar on a molar boron equivalent basis when dissolved in water or biological fluids at the same pH and at low concentration.

Boron compounds are well characterised and monographs for boric acid, sodium perborate, and borax are available in the British Pharmacopoeia 2001.

In terms of history and pattern of use, available evidence suggested that boron is an essential nutrient for human beings with food providing the greatest level of exposure to boron for most populations. An average intake of boron for humans is 0.44 µg/day from ambient air, 0.2–0.6 mg/day from drinking water, and 1.2 mg/day from the diet. The Australian dietary intake of boron in a small study was ~2.2 mg/day (with a urinary excretion of ~1.9 mg/day). Coffee, tea, and other beverages along with certain fruits, vegetables and cereals are the main sources.

Boron compounds are widely used in therapeutic and cosmetic products often in an excipient role. Boric acid is a weak topical bacteriostatic, fungistatic, and astringent agent. In cosmetics, boric acid serves as a buffering agent, biocide, and denaturant; borax functions as a pH adjuster.
Dietary supplement products available in other countries or via the Internet contain up to the recommended daily dose of 3 mg of boron in the form of borax, boron amino acid chelate or boron salts. Recommended daily maximum intakes for boron over a lifetime without appreciable health risks vary from 0.2 to 0.4 mg/kg.

Boron in its various dietary forms is rapidly absorbed and is excreted largely in the urine. Absorption appears to be virtually complete (95% in humans and rats), and boron appears rapidly in the blood and body tissues of several mammalian species following ingestion. Absorption across intact skin is negligible in all species studied but dermal absorption of boric acid is known to occur after application to broken or damaged skin.

Distribution of boron appears to take place by passive diffusion through the body fluids. Boron is distributed throughout the tissues and organs of animals and humans at concentrations normally between 0.05 and 0.6 µg/g fresh weight, and several times these concentrations in bones. Borate compounds are not metabolised by biological systems because of the considerable energy required to break the boron-oxygen bond. Boron appears to be eliminated largely in the urine. Boron interacts with other nutrients and plays a regulatory role in the metabolism of minerals, such as calcium, and subsequently bone metabolism.

Dietary deprivation of boron consistently results in adverse changes to biological functions and thus boron appears to be an essential nutrient for humans although no specific biochemical function for boron has been discovered. Variables affected by dietary boron include plasma and organ calcium and magnesium concentrations, plasma alkaline phosphatase and bone calcification. Consistent signs of deficiency include depressed growth and a reduction in some blood indices, particularly steroid hormone concentrations.

Boric acid and borax have a low acute oral toxicity in animals. Lethal dose (LD₅₀) values for mice, rats and dogs range from 2000 to >6000 mg/kg body weight. Acute toxicity signs include depression, ataxia, convulsions and death. From developmental toxicity studies, a No Observable Adverse Effect Level (NOAEL) of 9.6 mg/kg/day and a Lowest Observable Adverse Effect Level (LOAEL) of 13.3 mg/kg/day have been reported. Potential lethal doses for humans are usually cited as 3-6 g total for infants and 15-20 g total for adults.

Data regarding subchronic or chronic exposure to boron in the general population are limited. Effects on the male reproductive system have been reported following long-term exposure in animal studies.

No studies have been carried out on the possible carcinogenicity of boron or boron compounds in humans. Current data from both in vitro and in vivo studies suggest that boron compounds are neither mutagenic nor carcinogenic.

Nineteen adverse reaction reports to boric acid or borax-containing products are listed in the Australian Adverse Drug Reactions database. All of these products contained multiple ingredients. Other than burning or stinging reactions to topical application, the other adverse reactions appear unlikely to have been caused by boron compounds alone.
There were 64 adverse reaction reports in the US Special Nutritional Adverse Event Monitoring System (US SN/AEMS) web report for products containing boron. All of the products are multi-ingredient formulations and 34 cases were recorded for one product. The adverse reactions do not fit those expected for boric acid or borates.

Vomiting, abdominal pain and diarrhoea are among the main symptoms of acute boric acid poisoning.

Anaemia, anorexia, alopecia, dermatitis, confusion, and convulsions are among the reported symptoms of chronic boric acid intoxication. Headache, lethargy, restlessness, weakness, CNS irritation, and/or seizures may occur with high doses over longer term.

**Present discussion:**
CMEC Members noted that the claims for boron containing products on the Internet included:

- Helps maintain normal calcium balance and oestrogen levels.
- Boron helps to prevent the loss of calcium, phosphorus and magnesium through the urine.
- Helps to maintain proper blood levels of oestradiol which is a precursor of oestrogen.
- Studies indicate that boron improves calcium metabolism and utilisation.

Members discussed the toxicological properties of boron, noting that the major toxicities are reproductive (testicular atrophy) and developmental.

It was noted that if the Committee recommended boron to be suitable for use as an active ingredient in listed therapeutic goods, there was the potential for boron to be part of a wider range of therapeutic goods including products like multi ingredient drops for infants.

The National Drugs and Poisons Schedule Committee (NDPSC) recently amended the scheduling for boron based on a review of three recent major reviews:

- Integrated Risk Information System (IRIS 2002) *Toxicological review of boron and compounds* through the US Environmental Protection Agency;
- National Academy of Sciences (NAS 2002) *Dietary reference intake for boron*;

Members discussed the apparently wide margin for safety of boron as indicated by the available quantitative data in the form of NOAEL and LOAEL estimates for animal and human studies.

**CMEC Recommendation**
The CMEC resolved to recommend that:

**Recommendation 36.3**
CMEC recommends to the TGA that boron compounds (i.e. boric acid, borax, sodium perborate and sodium tetraborate pentahydrate [sodium borate pentahydrate]) are suitable for use as ingredients in listable therapeutic goods, with maximum listable amounts limited to those below amounts included on the *Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP).*
6.2 Molybdenum trioxide

Summary of evaluation report
Members considered an application for approval of molybdenum trioxide as an ingredient in Listable therapeutic goods. The proposed daily dose is 300 µg of molybdenum trioxide per tablet, providing a molybdenum dose of 200 µg/day.

Molybdenum occurs in nature chiefly as molybdenite (MoS₂). Molybdenum trioxide is formed by ‘roasting’ concentrated molybdenite in air. The substance is likely to be highly stable since it is in its highest oxidation state, and it is not hygroscopic.

Molybdenum is an essential micronutrient, forming part of a cofactor for critical enzymatic processes associated with detoxification and metabolism. As soluble molybdates, molybdenum, is found in most foods, with the richest sources including legumes, cereal grains, sorghum, seeds, some green leafy vegetables, milk and organ meats. Estimates of mean dietary molybdenum intake in a number of different countries range from 80-250 µg/day, but no Australian data are available.

There is no recommended daily intake (RDI) for molybdenum in Australia but the adult recommended daily allowance (RDA) for adults in the USA is 45 µg/day, and the US National Academy of Sciences has established a safe and adequate intake level of 75-250 µg/day for individuals over the age of ten.

There are currently 17 grandfathered, registered goods containing molybdenum trioxide on the ARTG with doses ranging from 33 µg to 1 mg. Under the Australian Food Standards Code, molybdenum (as sodium molybdate, or as high molybdenum yeast) can be added to formulated supplementary sports foods up to a total claimable daily amount of 125 µg and 62.5 µg for the inorganic and organic forms, respectively.

Promotional material for molybdenum supplements includes statements concerning possible effects related to deficiency, including oesophageal cancer, detoxification of dietary sulfites (used as preservatives in foods), prevention of cavities, prevention of anaemia and also as an aid in the detoxification of excess aldehydes produced by Candida species.

The effect of dietary molybdenum on metalloenzyme activity in pregnant and non-pregnant rats demonstrated that sodium molybdate significantly increased the plasma activity of the copper-containing enzyme, ceruloplasmin in pregnant but not in non-pregnant rats and increased xanthine oxidase and sulfite oxidase levels in both pregnant and non-pregnant rats.
Pharmacokinetic information and much of the safety data for molybdenum trioxide is based on studies using molybdenum compounds other than the trioxide salt. The toxicity of molybdenum compounds is dependent upon the extent of systemic absorption and therefore on their solubility. Hexavalent molybdenum is much more readily absorbed following oral administration than the tetravalent forms. Toxicity studies with the various molybdates are likely to be highly relevant to a consideration of molybdenum trioxide toxicity, since they form immediately upon addition of molybdenum trioxide to aqueous buffered solutions.

The rate of absorption of molybdenum is species dependent with the extent of absorption related not only to the level of molybdenum intake but also to the presence of dietary copper and sulfate, although the precise mechanisms underlying these interactions are not fully understood. Human studies have reported dietary ammonium molybdate absorption levels of up to 93%.

Molybdenum is distributed in the kidneys, liver and bone of animals. In animals and humans, molybdenum is eliminated rapidly by the kidneys and in humans, significant amounts are also found in the faeces. Studies in humans suggest that the kidney is the primary site of molybdenum homeostatic regulation.

There was a wide variation in the reported oral lethal dose (LD₅₀) value for molybdenum trioxide in rats ranging from 83 mg/kg to 2689 mg/kg for reasons not able to be determined. LD₅₀ values in mice were 141 mg/kg by the intraperitoneal (IP) and 94 mg/kg by the subcutaneous (SC) route, with clinical effects noted in the lungs, thorax and on respiration following IP administration. The guinea-pig LD₅₀ following IP administration was 400 mg/kg.

Many of the effects of molybdenum toxicity in longer term studies in animals resemble those of copper deficiency, and treatment with supplemental copper may reverse them. It is proposed that the mechanism for many of the toxic effects of molybdenum, at least in animals, is due to an interaction with copper.

In rats, molybdenum depressed growth at daily doses of 2 mg/kg or higher and induced bone deformities from 8 mg/kg. The lowest observed adverse effect level (LOAEL) for anaemia and diarrhoea was 50 mg/kg/day, while renal toxicity was observed with a LOAEL of 80 mg/kg/day.

Rabbits were more sensitive to molybdenum toxicity, with death, growth depression, anaemia, bone abnormalities and hair loss evident with daily ammonium molybdate doses of 2 or 2.35 mg/kg.

Dietary administration of sodium molybdate to guinea-pigs induced growth depression from 40 mg/kg/day, and hair discoloration at doses > 40 mg/kg/day. The effects of molybdenum on hair colour in this species could be alleviated by the addition of dietary copper, but body weight gain was only partially restored.

Results from both in vitro and in vivo mutagenicity studies indicate that molybdenum compounds, including molybdenum trioxide, are moderately genotoxic at high concentrations.
Molybdenum trioxide, or other molybdenum compounds, have not been investigated for oral carcinogenicity. According to the Register of Toxic Effects of Chemical Substances (RTECS) criteria, molybdenum trioxide is carcinogenic by the inhalational route (based on studies in rats) but this effect is most probably related to the route of exposure and is not considered to be relevant to oral administration. Molybdenum trioxide at very high doses weakly increased the incidence of lung tumours in strain A mice following its IP administration, but the relevance of this effect to oral administration is not known.

There is some epidemiological evidence that molybdenum may reduce the incidence of oesophageal cancer, suggesting that molybdenum may be protective against this form of cancer. However, this was not confirmed in a large Chinese study but some animal data are supportive.

There are no adequate data concerning the effects of molybdenum on human reproduction, but a range of adverse reproductive effects have been observed in experimental animals but only at high doses. These included increased neonatal mortality in mice, an increased rate of post-implantation loss after dosing of male mice, and prolonged oestrous cycles in rats with increased numbers of resorption and decreased fetal bodyweight gain. The no observable adverse effect levels (NOAEL) and LOAEL in this study were 0.9 and 1.6 mg/kg/day, respectively.

In the absence of consistent human data, a number of authorities have used this particular rat reproductive study to determine a Tolerable Daily Intake (TDI) or Tolerable Upper Intake Level (UL) for chronic exposure to molybdenum. After taking into account relevant “uncertainty factors” a TDI (or UL) was established at 9-10 µg/kg/day (630-700 µg/day in a 70 kg individual).

In terms of clinical data, clinical and biochemical effects of excess molybdenum exposure include gout-like symptoms, increased blood concentrations of uric acid, increased serum xanthine oxidase activity and decreased serum concentrations of copper. One particular Armenian study, which assisted in developing this information, has been criticised on methodological grounds and so it does not prove unequivocally that the clinical and biochemical effects were solely due to molybdenum ingestion. It is of note that the high molybdenum intake was accompanied by ingestion of lower than normal levels of copper, and this may have exacerbated the toxic effects of molybdenum. Nevertheless, this study has been used by the US Environmental Protection Agency (EPA) to assist in establishing an oral reference dose at 5 µg/kg/day (350 µg/day for a 70 kg individual).

The possibility of mineral imbalance, particularly involving copper levels in humans following ingestion of molybdenum supplements, is of key importance in establishing the suitability of molybdenum trioxide for inclusion in listable therapeutic goods. However the mechanisms of interaction are poorly understood.

There were no reports on the Australian Adverse Drug Reactions Database relating to molybdenum trioxide or any other molybdenum compounds. Adverse reactions reported in the medical literature include reports of increased blood uric acid concentrations and gout-like symptoms following dietary or occupational or exposure.
Members were advised that the present evaluation had found no evidence that molybdenum trioxide is not safe at the levels consistent with the generally recommended doses of minerals in therapeutic goods or in foods.

*Current discussion*

Members did not have significant concerns about the safety of the product noting that the tolerable daily intake/tolerable upper intake level/reference dose calculated by various authorities ranged from 350 µg/day to 2100 µg/day.

It was noted that there was little data on the clinical effects of molybdenum deficiency

Members were advised that the estimated safe and acceptable daily dietary intake (ESADDI) level in the United States has been superseded by the setting of an RDI and the limit for molybdenum of 100% of the RDI is 45 µg/day. The fact that molybdenum trioxide was already available in Australia in grandfathered, registered therapeutic goods, with a mean dose/day of 361 µg (range 33 µg – 1 mg), was noted.

After some discussion as to an appropriate and safe daily level of molybdenum intake, Members agreed to limit the maximum daily dose for molybdenum trioxide to 125 µg/day. This recommendation was assisted by the fact that the claimable limit for molybdenum in supplementary sports food was 125 µg/day and that the sponsor had not provided any data to support the proposed dose of 200 µg/day of molybdenum.

*CMEC Recommendation*

The CMEC resolved to recommend that:

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<thead>
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<th>Recommendation 36.4</th>
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<tbody>
<tr>
<td>CMEC recommends to the TGA that molybdenum trioxide is suitable for use as an active ingredient in listable therapeutic goods, with a limit on the maximum daily dose for molybdenum of 125 microgram/day.</td>
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</tbody>
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7. **Safety or Efficacy Reviews**

No matters were considered under this agenda item.

8. **Registration Applications**

No matters were considered under this agenda item.

9. **Variation to a Registered Product**

No matters were considered under this agenda item.
10. Matters referred from within the TGA

10.1 ADRAC Report

Members noted the Report from the 261st meeting of the Adverse Drug Reaction Advisory Committee (ADRAC) and discussed several reports:

11. Recommendation record

CMEC members adopted the following as a summary of the recommendations made at its 36th meeting:

Item 2 Minutes of CMEC’s 35th Meeting

Recommendation 36.1

CMEC confirms that the draft Minutes of its previous meeting (CMEC 35, 14 June 2002), as amended, are a true and accurate record of that previous meeting.

Item 5.2 Caffeine labelling – Progress Report

Recommendation 36.2

CMEC rescinded Recommendation 34.4 and made the following recommendation:

CMEC recommends to the TGA that labels on products containing those herbs known to contain caffeine be required to declare that the goods contain, or may contain caffeine, unless the sponsor can demonstrate that such products do not contain caffeine.

Homoeopathic medicines more dilute than 1X are exempt from this requirement.

Item 6.1 Boron

Recommendation 36.3

CMEC recommends to the TGA that boron compounds (i.e. boric acid, borax, sodium perborate and sodium tetraborate pentahydrate [sodium borate pentahydrate]) are suitable for use as ingredients in listable therapeutic goods, with maximum listable amounts limited to those below amounts included on the Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP).
Item 6.2 Molybdenum trioxide

Recommendation 36.4

CMEC recommends to the TGA that molybdenum trioxide is suitable for use as an active ingredient in listable therapeutic goods, with a limit on the maximum daily dose for molybdenum of 125 microgram/day.

12. For Information

No matters were considered under this agenda item.

13. Other business

The meeting closed at 4.30 pm on Friday 26 July 2002. The next meeting is to be held on Thursday 29 August and Friday 30 August 2002.