increased every 15 minutes during the first hour, as tolerated, to a maximum of 43 units/kg per hour such that the infusion is completed in about 3 to 4 hours (but see also under Adverse Effects, Treatment, and Precautions, above). In some countries, the dose is expressed as mg/kg: 100 units is equivalent to about 580 micrograms of laronidase.

Mucopolysaccharidosis I. Mucopolysaccharidosis I is a progressive disorder characterised by deficiency of the enzyme α-L-iduronidase, which is necessary to catalyse the hydrolysis of terminal α-L-iduronic residues of the glycosaminoglycans, dermatan sulfate and heparan sulfate. This results in their accumulation in tissues, with many clinical manifestations including hepatomegaly, skeletal abnormalities, pulmonary disease, eye disease, and progressive deterioration of the CNS. Mucopolysaccharidosis I has traditionally been classified into three main forms based on clinical symptoms and severity: Hurler syndrome, Hurler-Scheie syndrome, and Scheie syndrome. Hurler syndrome is the most severe form with a life expectancy of less than 10 years. However, there is a degree of overlap between the syndromes and they are indistinguishable by routine enzyme or urine tests.

Treatment was previously limited to symptomatic management but other options to halt disease progression are now available. Haematopoietic stem-cell transplantation using bone marrow or umbilical cord blood is of benefit in systemic disease and can prevent (but not usually reverse) CNS deterioration. However, substantial adverse effects limit its use to patients with severe disease. Enzyme replacement therapy with laronidase has been reported to confer benefit on the systemic manifestations of the disease, but since it does not cross the blood-brain barrier in appreciable amounts, beneficial effect on CNS symptoms is again predicted to be unlikely. However, the improvements conferred by enzyme replacement therapy might make haematopoietic stem-cell transplantation easier to tolerate.

References.

1. Kakkis ED, *et al.* Enzyme-replacement therapy in mucopolysaccharidosis I. *N Engl J Med* 2001; **344:** 182–8.
2. Wraith JE. Enzyme replacement therapy in mucopolysaccharidosis type I: progress and emerging difficulties. *J Inherit Metab Dis* 2001; **24:** 245–50.
3. Kakkis ED. Enzyme replacement therapy for the mucopolysaccharide storage disorders. *Expert Opin Invest Drugs* 2002; **11:** 675–85.
4. Kakavanos R, *et al.* Immune tolerance after long-term enzyme-replacement therapy among patients who have mucopolysaccharidosis I. *Lancet* 2003; **361:** 1608–13.
5. Muenzer J, Fisher A. Advances in the treatment of mucopolysaccharidosis type I. *N Engl J Med* 2004; **350:** 1932–4.
6. Staba SL, *et al.* Cord-blood transplants from unrelated donors in patients with Hurler's syndrome. *N Engl J Med* 2004; **350:** 1960–9.
7. Wraith JE, *et al.* Enzyme replacement therapy for mucopolysaccharidosis I: a randomized, double-blinded, placebo-controlled, multinational study of recombinant human α-L-iduronidase (laronidase). *J Pediatr* 2004; **144:** 581–8.
8. Grewal SS, *et al.* Safety and efficacy of enzyme replacement therapy in combination with hematopoietic stem cell transplantation in Hurler syndrome. *Genet Med* 2005: **7:** 143–6.
9. Miebach E. Enzyme replacement therapy in mucopolysaccharidosis type I. *Acta Paediatr Suppl* 2005; **94** (suppl 447): 58–60.
10. Wraith JE, *et al.* Enzyme replacement therapy in patients who have mucopolysaccharidosis I and are younger than 5 years: results of a multinational study of recombinant human α- -iduronidase (laronidase). Abstract: *Pediatrics* 2007; **120:** 158. Full version: http://pediatrics.aappublications.org/cgi/reprint/120/1/e37 (accessed 07/02/08)

Preparations

Proprietary Preparations (details are given in Part 3)

Belg.: Aldurazyme; ***Canad.:*** Aldurazyme; ***Cz.:*** Aldurazyme; ***Denm.:*** Aldurazyme; ***Fin.:*** Aldurazyme; ***Fr.:*** Aldurazyme; ***Ger.:*** Aldurazyme; ***Israel:*** Aldurazyme; ***Ital.:*** Aldurazyme; ***Neth.:*** Aldurazyme; ***Norw.:*** Aldurazyme; ***NZ:*** Aldurazyme; ***Pol.:*** Aldurazyme; ***Spain:*** Aldurazyme; ***Swed.:*** Aldurazyme; ***UK:*** Aldurazyme; ***USA:*** Aldurazyme.

Lavender

English Lavender; Kwiat lawendy (lavender flower); Lavande, fleur de (lavender flower); Lavande Vrai; Lavandulae flos (lavender flower); Lavendelblomma (lavender flower); Lavendelblüten; Laventelinkukka (lavender flower); Levandų žiedai (lavender flower); Levandulový květ (lavender flower); Levendulavirág (lavender flower).

Pharmacopoeias. *Eur.* (see p.vii) includes lavender flower.
Ph. Eur. 6.2 (Lavender Flower; Lavandulae flos). It consists of the dried flower of *Lavandula angustifolia* (*L. officinalis*). It contains not less than 1.3% v/w of essential oil, calculated with reference to the anhydrous drug. Protect from light.

Profile

Lavender flower is used as a sedative. It has also been used as a cholagogue. It is an ingredient of herbal remedies used for a variety of disorders.

Lavender flowers are the source of lavender oil (below).

Preparations

Proprietary Preparations (details are given in Part 3)

Multi-ingredient: ***Arg.:*** Lavandula Oligoplex; ***Austral.:*** Cimicifuga Compound; ***Austria:*** Euka; Mentopin; ***Braz.:*** Balsamo Branco; Traumac; ***Cz.:*** Melaton†; Schlaf-Nerventee N†; Valofyt Neo; ***Fr.:*** Mediflor Tisane Digestive No 3; ***Ger.:*** Presselin Dyspeptikum†; ***NZ:*** Botanica Hayfever; ***Pol.:*** Lumewal; Nervinolum; Nervosol; Reumobonisol; ***Port.:*** Cholagutt†; Erpecalm; ***S.Afr.:*** Krampdruppels; ***Spain:*** Linimento Naion; ***Switz.:*** Tisane relaxante N†; ***UK:*** Vital Eyes.

Lavender Oil

English Lavender Oil (from *L. intermedia*); Esencia de Alhucema; Esencia de Espliego; Essência de Alfazema; Foreign Lavender Oil (from *L. officinalis*); Huile Essentielle de Lavande; Lavanda, aceite esencial de; Lavande, huile essentielle de; Lavandulae aetheroleum; Lavandulae Etheroleum; Lavanta Yağı; Lavendelöl; Lavendelolja; Lavender Flower Oil; Laventeliöljy; Levandų eterinis aliejus; Levandulová silice; Olejek lawendowy; Oleum Lavandulae.

Pharmacopoeias. In *Eur.* (see p.vii).
Ph. Eur. 6.2 (Lavender Oil). An essential oil obtained by steam distillation from the flowering tops of *Lavandula angustifolia* (*L. officinalis*). A colourless or pale yellow clear liquid with a characteristic odour. Relative density 0.878 to 0.892. Store in well-filled airtight containers at a temperature not exceeding 25°. Protect from light.

Profile

Lavender oil has been used as a carminative and as a flavour. It is sometimes applied externally as an insect repellent. Its chief use is in perfumery and it is occasionally used in ointments and other pharmaceutical preparations to cover disagreeable odours. It has been suggested that lavender oil may have sedative properties after inhalation. It is also used in aromatherapy.

Lavender oil has been reported to produce nausea, vomiting, headache, and chills when inhaled or absorbed through the skin. It may cause contact allergy and phototoxicity.

Adverse effects. There have been reports of contact dermatitis associated with lavender oil in a shampoo,[1] and facial dermatitis after application of the oil to pillows for its sedative properties.[2]

1. Brandão FM. Occupational allergy to lavender oil. *Contact Dermatitis* 1986; **15:** 249–50.
2. Coulson IH, Khan ASA. Facial 'pillow' dermatitis due to lavender oil allergy. *Contact Dermatitis* 1999; **41:** 111.

Insomnia. Ambient exposure to lavender oil produced similar sleep patterns to conventional sedatives in 4 elderly patients.[1]

1. Hardy M, *et al.* Replacement of drug treatment for insomnia by ambient odour. *Lancet* 1995; **346:** 701.

Preparations

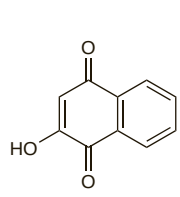
Proprietary Preparations (details are given in Part 3)

Pol.: Lawenol.

Multi-ingredient: ***Austral.:*** Apex Repel Natural; Neutralice; ***Austria:*** Berggeist; Rowalind; ***Belg.:*** Mouskito Travel Milk; ***Braz.:*** Aliviol; Analgen†; Benegel; Gelflex; Gelol†; Gelonevral†; Inhalante Yatropan; Mentalol†; Mialgex†; Nevrol; Salimetin†; ***Cz.:*** Amol; Ondrejova Mast; Tiger Ol†; ***Fr.:*** Aromasol; Balsofumine; Balsofumine Mentholee; Ephydrol; Gouttes aux Essences; Maghora; Moustidose; Moustidose Bebe-Nourrisson; Paps; Pectoderme†; Perubore; Poudre du Marcheur; Resistim; ***Ger.:*** Amol Heilkrautergeist N; Dolo-cyl; Leber-Galle-Tropfen 83†; Solum Ol; ***Hong Kong:*** Calmiderm; ***Hung.:*** Opodeldok†; ***Israel:*** Headache Pads; ***Ital.:*** Citrosystem; Controller; Mistick Verde; Venalta; Vicks Baby Balsam†; ***NZ:*** Apex Repel Natural; Electric Blue Headlice; Vicks Baby Balsam; ***Pol.:*** Amol; Aromatol; Carmolis; ***Port.:*** Solubeol†; Vaporil; ***Rus.:*** Carmolis (Кармолис)†; Carmolis Fluid (Кармолис Жидкость)†; Espol (Эспол); ***S.Afr.:*** Arnica Massage Oil; Entressdruppels HM; Rooilavental; ***Spain:*** Dolokey; Termosan; ***Switz.:*** Baume du Chalet; Carmol; Dolo-Arthrosenex sine Heparino†; Hygiodermil; Liberol Bain†; Massorax†; Muco-Sana†; Nasobol†; Oculosan; Perskindol Classic; Perubare†; Pommade Nasale Radix†; Pulmex; Saltrates†; Spagyrom; Ziegella; ***Turk.:*** Algo-Wax; ***UK:*** Arnica Massage Balm; Eucanol; Larch Resin comp.; Massage Balm with Calendula; Migrastick; ***USA:*** Nasal Jelly; ***Venez.:*** Friccion Aromatica.

Lawsone

Lawsonia. 2-Hydroxy-1,4-naphthoquinone.
$C_{10}H_6O_3$ = 174.2.
CAS — 83-72-7.



Profile

Lawsone is a dye present in henna (p.2318), the leaves of *Lawsonia* spp., and may also be prepared synthetically. It has been used with dihydroxyacetone in sunscreen preparations. There appears to be no evidence that it has any sunscreening properties when used alone.

Adverse effects. Observation that lawsone causes oxidative damage to red blood cells *in vitro* supported a suggestion that percutaneous absorption of henna could contribute to unexplained neonatal hyperbilirubinaemia in countries where the ceremonial use of henna is widespread.[1]

1. Zinkham WH, Oski FA. Henna: a potential cause of oxidative hemolysis and neonatal hyperbilirubinemia. *Pediatrics* 1996; **97:** 707–9.

Lead

Blei; Plomb; Plomo; Plumbum.
Pb = 207.2.
CAS — 7439-92-1.

Description. Lead is a grey, malleable and ductile metal.

Adverse Effects

Lead poisoning (plumbism) may be due to inorganic or organic lead and may be acute or, more often, chronic. It has followed exposure to a wide range of compounds and objects from which lead may be absorbed following ingestion or inhalation. Some of those that have been incriminated include paint, pottery glazes, crystal glassware, domestic water supplies, petrol, poteen, cosmetics (particularly home-made or traditional forms such as kohl or surma), herbal or folk remedies, including traditional Chinese medicines, newsprint, and retained bullets. Children are often the victims of accidental poisoning and may be vulnerable to chronic exposure to lead from environmental pollution.

Acute effects of lead poisoning include metallic taste, abdominal pain, diarrhoea, vomiting, hypotension, muscle weakness and cramps, fatigue, abnormal liver function tests, and acute interstitial nephritis. Encephalopathy may occur and is more common in children. Symptoms of chronic poisoning with inorganic lead include anorexia, abdominal pain, constipation, anaemia, headache, fatigue, irritability, peripheral neuropathy, and encephalopathy with convulsions and coma. There may be kidney damage and impairment of mental function. Children with elevated lead concentrations may be asymptomatic apart from intellectual deficits and behavioural disorders.

Organic lead poisoning produces mainly CNS symptoms; there can be gastrointestinal and cardiovascular effects, and renal and hepatic damage.

◊ General references to lead exposure, adverse effects, screening, and management,[1-15] and case reports of specific sources of lead exposure.[16-21]

1. WHO. Recommended health-based limits in occupational exposure to heavy metals: report of a WHO study group. *WHO Tech Rep Ser 647* 1980. Available at: http://whqlibdoc.who.int/trs/WHO_TRS_647.pdf (accessed 08/07/08)
2. WHO. Lead—environmental aspects. *Environmental Health Criteria 85*. Geneva: WHO, 1989. Available at: http://www.inchem.org/documents/ehc/ehc/ehc85.htm (accessed 08/07/08)
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4. Wolf AW, *et al.* Effects of iron therapy on infant blood lead levels. *J Pediatr* 2003; **143:** 789–95.
5. American Academy of Pediatrics Committee on Environmental Health. Lead exposure in children: prevention, detection, and management. *Pediatrics* 2005; **116:** 1036–46.
6. Kordas K, *et al.* Iron and zinc supplementation does not improve parent or teacher ratings of behavior in first grade Mexican children exposed to lead. *J Pediatr* 2005; **147:** 632–9.
7. Stewart WF, *et al.* Past adult lead exposure is linked to neurodegeneration measured by brain MRI. *Neurology* 2006; **66:** 1476–84.
8. Zimmermann MB, *et al.* Iron fortification reduces blood lead levels in children in Bangalore, India. *Pediatrics* 2006; **117:** 2014–21.
9. Téllez-Rojo MM, *et al.* Longitudinal associations between blood lead concentrations lower than 10 μg/dL and neurobehavioral development in environmentally exposed children in Mexico City. *Pediatrics* 2006; **118:** e323–e330. Available at: http://pediatrics.aappublications.org/cgi/content/full/118/2/e323 (accessed 08/07/08)
10. Yuan W, *et al.* The impact of early childhood lead exposure on brain organization: a functional magnetic resonance imaging study of language function. *Pediatrics* 2006; **118:** 971–7.
11. Menke A, *et al.* Blood lead below 0.48 μmol/L (10 μg/dL) and mortality among US adults. *Circulation* 2006; **114:** 1388–94.
12. Rischitelli G, *et al.* Screening for elevated lead levels in childhood and pregnancy: an updated summary of evidence for the US Preventive Services Task Force. *Pediatrics* 2006; **118:** e1867–e1895. Available at: http://pediatrics.aappublications.org/cgi/content/full/118/6/e1867 (accessed 08/07/08)
13. Gracia RC, Snodgrass WR. Lead toxicity and chelation therapy. *Am J Health-Syst Pharm* 2007; **64:** 45–53.
14. Chen A, *et al.* Lead exposure, IQ, and behavior in urban 5- to 7-year-olds: does lead affect behavior only by lowering IQ? *Pediatrics* 2007; **119:** e650–e658. Available at: http://pediatrics.aappublications.org/cgi/content/full/119/3/e650 (accessed 08/07/08)
15. CDC Advisory Committee on Childhood Lead Poisoning Prevention. Interpreting and managing blood lead levels <10 μg/dL in children and reducing childhood exposures to lead: recommendations of CDC's Advisory Committee on Childhood Lead Poisoning Prevention. *MMWR* 2007; **56 (RR-8)**: 1–16. Correction. *ibid.*; **56 (47)**: 1241. Also available at: http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5608a1.htm (accessed 05/08/08)
16. Powell ST, *et al.* Succimer therapy for congenital lead poisoning from maternal petrol sniffing. *Med J Aust* 2006; **184:** 84–5.
17. Coon T, *et al.* Lead toxicity in a 14-year-old female with retained bullet fragments. *Pediatrics* 2006; **117:** 227–30.

18. Frith D, *et al*. Lead poisoning—a differential diagnosis for abdominal pain. *Lancet* 2005; **366:** 2146.
19. VanArsdale JL, *et al*. Lead poisoning from a toy necklace. *Pediatrics* 2004; **114:** 1096–9.
20. Guillard O, *et al*. A case of acute lead poisoning in a 2-year-old child. *Br J Clin Pharmacol* 2006; **62:** 246–7.
21. Berkowitz S, Tarrago R. Acute brain herniation from lead toxicity. *Pediatrics* 2006; **118:** 2548–51.

Treatment of Adverse Effects

The main aim in the management of both acute and chronic lead poisoning is to control the symptoms and reduce the concentration of lead in the body. Patients should be removed from the source of exposure and iron and calcium deficiencies corrected.

Treatment of acute symptomatic poisoning entails supportive therapy including intravenous fluids. Renal and hepatic function should also be monitored and convulsions controlled with a benzodiazepine. Encephalopathy, which is rare in adults but more common in children, requires urgent treatment.

Acute ingestion of lead or its salts should be treated if appropriate by activated charcoal or gastric lavage if within 1 hour of ingestion of a potentially life-threatening dose. Emesis is not recommended for organic lead compounds.

In severe cases of inorganic or organic lead poisoning, chelation therapy may be required to facilitate removal of lead from the body. A lead mobilisation test that measures urinary excretion of lead after a standard dose of sodium calcium edetate (p.1462) has been widely used as a means of assessing the need for therapy. However, because of difficulties in administering the test and uncertainties in interpreting results, some authorities have recommended blood-lead concentrations as a guide to treatment. The lead mobilisation test in addition to measurement of blood-lead concentrations may be useful in determining the necessity for chelation therapy in children (see below). Blood-lead concentrations should be obtained before starting chelation therapy, and then again after the first course since mobilisation of stored lead from body tissues may cause blood-lead concentrations to rebound after an initial drop; a second course of chelator may then be required. Raised blood-lead concentrations despite chelation therapy could also indicate continued exposure.

Chelation therapy is not indicated for children with a blood-lead concentration less than 25 micrograms per 100 mL. Children with a blood-lead concentration of 25 to 44 micrograms per 100 mL and a positive sodium calcium edetate mobilisation test should be considered for treatment with oral succimer (p.1466); alternative chelators are oral penicillamine (p.1456), and in the USA but not UK, dimercaprol (p.1444). For blood-lead concentrations of 45 to 70 micrograms per 100 mL, oral succimer should be given, followed by a second course if necessary. If encephalopathy is present or blood-lead concentrations are over 70 micrograms per 100 mL, parenteral sodium calcium edetate should be given with monitoring of renal and hepatic function, followed by a second course of chelator if necessary. In the USA, dimercaprol is given with sodium calcium edetate and 4 hours before the first dose; While dimercaprol has been used in the UK, it is not currently recommended as first-line treatment for lead poisoning.

It is considered that asymptomatic adults do not generally require chelation therapy. Symptomatic adults without encephalopathy may be treated with succimer; alternatives are unithiol, penicillamine or sodium calcium edetate. Adults with severe toxicity or encephalopathy should be treated with parenteral sodium calcium edetate followed by a second course if necessary. As with children (see above), dimercaprol is given with sodium calcium edetate in the USA but not in the UK.

Lead foreign bodies may need to be removed surgically or endoscopically to prevent further exposure. Long-term management of chronic lead poisoning involves eliminating environmental lead exposure. Chelation therapy is not a substitute for environmental controls in those suffering occupational exposure.

◊ Results from one study[1] indicated that succimer did not improve scores on tests of cognition, behaviour, or neuropsychological function despite lowering blood levels of lead in children who had initial levels of less than 45 micrograms per 100 mL. The authors suggested that, since succimer is as effective a chelator as any other currently available, chelation therapy in general may not be beneficial in children with these blood-lead levels.

1. Rogan WJ, *et al*. The effect of chelation therapy with succimer on neuropsychological development in children exposed to lead. *N Engl J Med* 2001; **344:** 1421–6.

Lead in the Environment

Many countries have taken action to reduce lead exposure from environmental sources, including food, paint, and petrol, by limiting or banning altogether the use of lead compounds in such sources. Such measures have been of value in reducing childhood exposure to lead. Screening of all children to detect those at risk of chronic lead poisoning and developmental deficit has been advocated, but selective screening in areas perceived as high risk may be more appropriate in countries where the overall level of lead contamination is low.

Pharmacokinetics

Lead is absorbed from the gastrointestinal tract. It is also absorbed by the lungs from dust particles or fumes.

Inorganic lead is not absorbed through intact skin, but organic lead compounds may be absorbed rapidly.

Lead is distributed in the soft tissues, with higher concentrations in the liver and kidneys. In the blood it is associated with the erythrocytes. Over a period of time lead accumulates in the body and is deposited in calcified bone, hair, and teeth. Lead crosses the placental barrier. It is excreted in the faeces, urine, and sweat, and also appears in breast milk.

Uses and Administration

Lead compounds were formerly used as astringents, but the medicinal use of preparations containing lead is no longer recommended. The lead salts or compounds that have been used have included lead acetate and lead subacetate (for lead lotion, still known sometimes as lotio plumbi), lead carbonate, lead monoxide, and lead oleate (for lead plaster-mass).

Preparations

Proprietary Preparations (details are given in Part 3)

Multi-ingredient: ***Austria:*** Vulpuran; ***Mex.:*** Emplasto Monopolis.

Lecithin

E322; E442 (ammonium phosphatides); Lécithine; Lecithinum; Lecitina; Lesitinler.

Pharmacopoeias. In *Ger.* Also in *USNF*.

USNF 26 (Lecithin). A complex mixture of acetone-insoluble phosphatides, which consists chiefly of phosphatidyl choline, phosphatidyl olamine, phosphatidyl serine, and phosphatidyl inositol, combined with various amounts of other substances such as triglycerides, fatty acids, and carbohydrates, as separated from the crude vegetable oil source. It contains not less than 50% of acetone-insoluble matter.

The consistency of both natural grades and refined grades of lecithin may vary from plastic to fluid, depending upon the content of free fatty acid and oil, and upon the presence or absence of other diluents. Its colour varies from light yellow to brown, depending on the source, on crop variations, and on whether it is bleached or unbleached.

It is odourless or has a characteristic, slight nutlike odour. It is partially soluble in water, but readily hydrates to form emulsions. The oil-free phosphatides are soluble in fatty acids, but are practically insoluble in fixed oils. When all phosphatide fractions are present, lecithin is partially soluble in alcohol and practically insoluble in acetone.

Profile

Lecithin is an emulsifying and stabilising agent used in both the pharmaceutical and the food industries.

Lecithin has also been used as a source of choline in the treatment of dementia (p.362) but with little evidence of clinical benefit. Phosphatidyl serine (p.2367) has been used similarly. Other constituents of lecithin such as phosphatidyl olamine and phosphatidyl inositol may be found in natural pulmonary surfactants (p.2375).

Lecithin is also an ingredient of preparations promoted as tonics and dietary supplements in an enormous range of disorders.

◊ References.

1. Higgins JPT, Flicker L. Lecithin for dementia and cognitive impairment. Available in The Cochrane Database of Systematic Reviews; Issue 4. Chichester: John Wiley; 2000 (accessed 14/02/06).

Preparations

Proprietary Preparations (details are given in Part 3)

Arg.: Herbaccion Lecitina; Reducin; ***Austral.:*** Buerlecithin; ***Austria:*** Buerlecithin Compact; Dermo WAS; ***Cz.:*** Buerlecithin†; Essentiale N†; ***Ger.:*** Buerlecithin; ***India:*** Essentiale-L; ***Indon.:*** Neurochol; ***Mex.:*** Leciderm; ***Pol.:*** Lecitan; ***Port.:*** Pansebase Solido†; ***Switz.:*** Buerlecithin Compact†; ***Venez.:*** Lecivar.

Multi-ingredient: ***Arg.:*** Ayton; Cholesterol Reducing Plan†; Herbaccion Memory; KLB6 Fruit Diet; No-Gras; Prueboi†; Sojasterol†; Top Life Diet†; ***Austral.:*** Berberis Complex; Bioglan Zellulean with Escin; Extralife Arthri-Care; Extralife Extra-Brite; Extralife Liva-Care; ML 20†; Plantiodine Plus†; ***Austria:*** Bilatin; Buerlecithin; Lecikur; ***Canad.:*** Complex 15†; Kyolic 104†; ***Chile:*** Cartilago T-500; ***Cz.:*** Vita Buerlecithin†; ***Fr.:*** Cholegerol†; ***Ger.:*** Hicoton†; Lipidavit; Tears Again; Vita Buerlecithin†; ***Hong Kong:*** Apaisac; Ginkgo-PS†; Wari-Procomil; ***India:*** Livage; ***Indon.:*** BIO-EPL; Cholesvit; Curson; Epatin; Hepachol; Lanagogum; Lanaven Plus; Lesichol; Nutriflam; Verona; ***Ital.:*** Nutrigel†; Ottovis; Solecin; Tricortin; ***Malaysia:*** Livguard; ***Mex.:*** Lecifar-K†; ***Philipp.:*** Korgivit-E; Liverine; Memori Plus; Memory DD; ***Pol.:*** Lecigal†; Lecytyna E; ***Port.:*** Pansebase; Pansebase Composto; Secpel; Secpel Composto; ***Singapore:*** Ginkgo-PS; ***Switz.:*** Biovital Ginseng; Vita Buerlecithin; ***Thai.:*** Wari-Procomil†; ***UK:*** Kelp Plus 3; S.P.H.P.; ***USA:*** KLB6; ***Venez.:*** Lecivar Plus.

Leishmanin

Leishmanina.

Profile

Leishmanin is a suspension of *Leishmania* promastigotes used in an intradermal test to indicate previous exposure to leishmanial antigens. Its chief use is in epidemiological studies of leishmaniasis (p.824). The leishmanin skin test has also been known as the Montenegro test.

Lemon

Pharmacopoeias. *Br.* includes dried lemon peel and *Swiss* includes fresh lemon peel.

BP 2008 (Dried Lemon Peel). The dried outer part of the pericarp of the ripe, or nearly ripe, fruit of *Citrus limon*. It contains not less than 2.5% v/w of volatile oil.

Profile

Lemon, *Citrus limon* (*Citrus limonum*) (Rutaceae), is an ingredient of herbal remedies used for gastrointestinal disorders and as tonics. The juice is traditionally included in preparations for colds and coughs. Lemon is a source of bioflavonoids used to improve capillary function (see Flavonoid Compounds, p.2304). The peel is the source of lemon oil (p.2332). Citrus fruits are a source of vitamin C (p.1983).

Photosensitivity is associated with citrus oils.

Preparations

BP 2008: Concentrated Compound Gentian Infusion;
USNF 26: Lemon Tincture.

Proprietary Preparations (details are given in Part 3)

Multi-ingredient: ***Austria:*** Gencydo; ***Braz.:*** Balsamo Branco; ***Ger.:*** Doppelherz Melissengeist†; Gencydo; ***Ital.:*** Altadrine; ***Port.:*** Erpecalm; ***Rus.:*** Doppelherz Melissa (Доппельгерц Мелисса); ***Switz.:*** Gencydo.

Lemon Oil

Aetheroleum Citri; Citri Etheroleum; Citrinų eterinis aliejus; Citromolaj; Citron, huile essentielle de; Citronenöl; Citronolja; Citronová silice; Esencia de Cidra; Essence de Citron; Essência de Limão; Limón, aceite esencial de; Limonis aetheroleum; Ol. Limon.; Olejek cytrynowy; Oleum Citri; Oleum Limonis; Sitruunaöljy.

Pharmacopoeias. In *Eur.* (see p.vii). Also in *USNF*.

Ph. Eur. 6.2 (Lemon Oil). The essential oil obtained by suitable mechanical means without the aid of heat from the fresh peel of *Citrus limon*. It contains a maximum of 0.5% β-caryophyllene, 0.5 to 2.3% geranial, 0.1 to 0.8% geranyl acetate, 56.0 to 78.0% limonene, 0.3 to 1.5% neral, 0.2 to 0.9% neryl acetate, 7.0 to 17.0% β-pinene, 1.0 to 3.0% sabinene, 6.0 to 12.0% γ-terpinene, and a maximum of 0.6% α-terpineol.

A clear mobile pale yellow to greenish-yellow liquid with a characteristic odour. It may become cloudy at low temperatures. Store in well-filled airtight containers at a temperature not exceeding 25°. Protect from light. Where applicable the label should state that the contents are Italian-type lemon oil.

USNF 26 (Lemon Oil). The volatile oil obtained by expression, without the aid of heat, from the fresh peel of the fruit of *Citrus* × *limon* (Rutaceae), with or without the previous separation of the pulp and the peel. The total aldehyde content, calculated as citral, is not less than 2.2% and not more than 3.8% for California-type lemon oil, and not less than 3.0% and not more than 5.5% for Italian-type lemon oil. Store in well-filled airtight containers.

Profile

Lemon oil is chiefly used in perfumery and as a flavour. It is used in the preparation of terpeneless lemon oil (below). It has also been used with other volatile agents in rubefacient preparations and preparations for respiratory-tract disorders. Both lemon oil and lemon petitgrain oils (prepared from the leaves and twigs) are used in aromatherapy.

Photosensitivity reactions and contact dermatitis have been reported.

Preparations

BP 2008: Aromatic Ammonia Spirit;
USNF 26: Compound Orange Spirit.

Proprietary Preparations (details are given in Part 3)

Multi-ingredient: ***Austral.:*** Genuine Australian Eucalyptus Drops†; ***Austria:*** Spasmo Claim; ***Canad.:*** SH-206; ***Chile:*** Agua del Carmen; Agua Melisa Carminativa; ***Cz.:*** Amol; Coldastop; ***Fr.:*** Ephydrol; Poudre du Marcheur; ***Ger.:*** Amol Heilkrautergeist N; Babix-Wundsalbe N†; GeloSitin; Melissengeist; ***Indon.:*** OBH; ***Israel:*** Garonsept; ***Ital.:*** Esoklin; Valda Timo e Limone; Venalta; ***NZ:*** Electric Blue Headlice; Lemsip Dry Cough†; ***Pol.:*** Amol; Argol Essenza Balsamica; Argol Grip; Argol Rheuma; Aromatol; Carmolis; ***Rus.:*** Carmolis (Кармолис)†; ***S.Afr.:*** Balsem Vita GEEL; Balsem Vita ROOI; Spiritus Contra Tussim Drops; ***Switz.:*** Alcoolat de Melisse†; Carmol; Neo-Angin au miel et citron; Perskindol Classic; Pirom; Sansilla; Sibrovita; ***UK:*** Melissa Comp.; ***USA:*** Mexsana.

Terpeneless Lemon Oil

Limón exento de terpeno, aceite esencial de; Oleum Limonis Deterpenatum.

Pharmacopoeias. In *Br.*

BP 2008 (Terpeneless Lemon Oil). A clear colourless or pale yellow liquid, visibly free from water, with the characteristic odour and taste of lemon, prepared by concentrating lemon oil under reduced pressure until most of the terpenes have been removed, or by solvent partition. It contains not less than 40% w/w of aldehydes calculated as citral. Soluble 1 in 1 of alcohol (80%). Store in well-filled containers at a temperature not exceeding 25°. Protect from light.

Profile

Terpeneless lemon oil is used as a flavour. It has the advantages of being stronger in taste and odour and more readily soluble