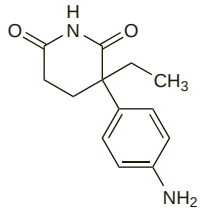
## Aminoglutethimide *(BAN, rINN)* ⊗

Aminoglutethimid; Aminoglutéthimide; Aminoglutethimidum; Aminoglutetimid; Aminoglutetimida; Aminoglutetimidas; Aminoglutetimidi; Aminoglythetimide; Ba-16038. 2-(4-Aminophenyl)-2-ethylglutarimide; 3-(4-Aminophenyl)-3-ethylpiperidine-2,6-dione.

Аминоглутетимид

$C_{13}H_{16}N_2O_2$ = 232.3.
*CAS — 125-84-8.*
*ATC — L02BG01.*
*ATC Vet — QL02BG01.*



**Pharmacopoeias.** In *Chin.*, *Eur.* (see p.vii), and *US*.

**Ph. Eur. 6.2** (Aminoglutethimide). A white or slightly yellow, crystalline powder. Practically insoluble in water; freely soluble in acetone; soluble in methyl alcohol.

**USP 31** (Aminoglutethimide). A white or creamy-white, fine, crystalline powder. Very slightly soluble in water; readily soluble in most organic solvents. It forms water-soluble salts with strong acids. The pH of a 0.1% solution in dilute methyl alcohol (1 in 20) is between 6.2 and 7.3.

### Adverse Effects

The most frequent adverse effects reported with aminoglutethimide include drowsiness, lethargy, and skin rashes (sometimes with fever); these generally diminish after the first 6 weeks of therapy. Dizziness and nausea occasionally occur. Leucopenia, thrombocytopenia, agranulocytosis, or severe pancytopenia have occurred rarely. Adrenal insufficiency may rarely occur, and there have been reports of other endocrine disturbances including hypothyroidism, and virilisation. Other rare effects include ataxia, headache, depression, gastrointestinal disturbances, hypercholesterolaemia, and orthostatic hypotension.

Overdosage may lead to CNS depression and impairment of consciousness, electrolyte disturbances, and respiratory depression.

**Effects on the liver.** Aminoglutethimide has been associated with reports of cholestatic jaundice, accompanied by rash[1,2] and fever,[2] and probably due to an idiosyncratic hypersensitivity reaction.[1] It has been suggested that liver function tests should be carried out in patients receiving aminoglutethimide who develop fever and eruptions.[2]

1. Gerber SB, Miller KB. Cholestatic jaundice and aminoglutethimide. *Ann Intern Med* 1982; **97:** 138.
2. Perrault DJ, Domovitch E. Aminoglutethimide and cholestasis. *Ann Intern Med* 1984; **100:** 160.

**Effects on the lungs.** Pulmonary infiltrates in a patient who developed progressive dyspnoea on starting therapy with aminoglutethimide were found to be due to diffuse alveolar damage and haemorrhage; thrombocytopenia was present but prothrombin and bleeding times were normal. The patient's gas exchange and chest radiographs improved on stopping aminoglutethimide and giving corticosteroids.[1] Blood and pulmonary eosinophilia, which resolved on stopping aminoglutethimide therapy, has also been reported.[2]

1. Rodman DM, *et al.* Aminoglutethimide, alveolar damage, and hemorrhage. *Ann Intern Med* 1986; **105:** 633.
2. Bell SC, Anderson EG. Pulmonary eosinophilia associated with aminoglutethimide. *Aust N Z J Med* 1998; **28:** 670–1.

**Lupus.** SLE occurred in a patient who received aminoglutethimide, and resolved when the drug was withdrawn.[1] In another report, however, a patient with a lupus-like syndrome had a reduction in disease activity when tamoxifen therapy was changed to aminoglutethimide.[2]

1. McCraken M, *et al.* Systemic lupus erythematosus induced by aminoglutethimide. *BMJ* 1980; **281:** 1254.
2. Etherington J, *et al.* Effect of aminoglutethimide on the activity of a case of a connective tissue disorder with features of systemic lupus erythematosus. *Lupus* 1993; **2:** 387.

### Precautions

Aminoglutethimide inhibits adrenal steroid production so supplementary glucocorticoid therapy with hydrocortisone must normally be given, although supplementation may not be necessary in patients with Cushing's syndrome. Some patients also require a mineralocorticoid. It has been suggested that aminoglutethimide should be temporarily withdrawn in patients who undergo shock or trauma, or develop intercurrent infection.

Blood pressure, blood counts, and serum electrolytes should be regularly monitored during aminoglutethimide therapy and periodic monitoring of liver and thyroid function is recommended.

Aminoglutethimide should not be given during pregnancy as pseudohermaphroditism may occur in the fetus.

Aminoglutethimide frequently causes drowsiness: patients so affected should not drive or operate machinery.

**Porphyria.** Aminoglutethimide has been associated with acute attacks of porphyria and is considered unsafe in porphyric patients.

### Interactions

The rate of metabolism of some drugs is increased by aminoglutethimide; patients also taking warfarin or other coumarin anticoagulants, theophylline, tamoxifen, medroxyprogesterone, or oral hypoglycaemics, may require increased dosages of these drugs. The metabolism of dexamethasone is also accelerated, which limits its value for corticosteroid supplementation in patients receiving aminoglutethimide. Use with diuretics may lead to hyponatraemia, while alcohol may potentiate the central effects of aminoglutethimide.

◊ See also references to aminoglutethimide's interactions with *digitoxin* (p.1259), *theophylline* (p.1144), *progestogens* (p.2126), *tamoxifen* (see Antineoplastics, p.774), and *anticoagulants* (under Warfarin, p.1429).

### Pharmacokinetics

Aminoglutethimide is well absorbed after oral doses, with peak plasma concentrations occurring after 1 to 4 hours. It is metabolised in the liver, primarily to *N*-hydroxylaminoglutethimide and *N*-acetylaminoglutethimide, and appears to induce its own metabolism. The half-life, which is reported to be about 13 hours after a single dose, is decreased to around 9 hours after about 2 weeks of continuous therapy. Aminoglutethimide is excreted in urine, about half a dose being excreted unchanged and the remainder as metabolites. Only about 20 to 25% of a dose is bound to plasma protein.

**Half-life.** A study in 17 patients showed that the plasma half-life of aminoglutethimide had a mean value of 15.5 hours after single doses but fell to 8.9 hours during multiple-dose therapy.[1] This marked reduction could largely be attributed to a decrease in the volume of distribution; auto-induction of metabolism might be of less importance in decreasing half-life than had been previously suggested.

1. Lønning PE, *et al.* Single-dose and steady-state pharmacokinetics of aminoglutethimide. *Clin Pharmacokinet* 1985; **10:** 353–64.

### Uses and Administration

Aminoglutethimide is an analogue of glutethimide (p.1000) and was formerly used for its weak anticonvulsant properties. Aminoglutethimide blocks the production of adrenal steroids and acts as an aromatase inhibitor to block the conversion of androgens to oestrogens (the major source of oestrogens in women without ovarian function). It was used in the treatment of metastatic breast cancer (p.661) in postmenopausal or oophorectomised women and as palliative treatment in men with advanced prostatic cancer (p.671).

Aminoglutethimide has also been used in the treatment of Cushing's syndrome (p.2344). Usual oral doses range from 1 to 2 g daily, in divided doses.

The *dextro*-isomer of aminoglutethimide, dexaminoglutethimide has been investigated.

### Preparations

***BP 2008:*** Aminoglutethimide Tablets;
***USP 31:*** Aminoglutethimide Tablets.

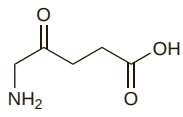
**Proprietary Preparations** (details are given in Part 3)
***Arg.:*** Orimeten†; ***Austral.:*** Cytadren; ***Austria:*** Orimeten†; ***Belg.:*** Orimeten†; ***Braz.:*** Orimeten†; ***Chile:*** Orimeten†; ***Cz.:*** Orimeten†; ***Fr.:*** Orimetene†; ***Ger.:*** Orimeten†; ***Hong Kong:*** Orimetene; ***Ital.:*** Orimeten†; ***Malaysia:*** Orimetene†; ***Neth.:*** Orimeten†; ***NZ:*** Cytadren†; ***Rus.:*** Mamomit (Мамомит); Orimeten (Оримотен)†; ***S.Afr.:*** Orimeten†; ***Spain:*** Orimeten†; ***Switz.:*** Orimetene†; ***UK:*** Orimeten†; ***USA:*** Cytadren†.

---

# 5-Aminolevulinic Acid

ALA; 5-ALA; δ-Aminolaevulinic Acid; 5-Aminolaevulinic Acid; 5-Aminolevulínico, ácido. 5-Amino-4-oxopentanoic acid.

$C_5H_9NO_3$ = 131.1.
*CAS — 106-60-5.*
*ATC — L01XD04.*
*ATC Vet — QL01XD04.*



## Aminolevulinic Acid Hydrochloride *(USAN)*

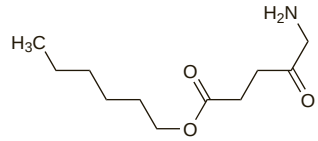
Aminolaevulinic Acid Hydrochloride; Aminolevulínico, hidrocloruro ácido. 5-Aminolevulinic acid hydrochloride.

$C_5H_9NO_3$, HCl = 167.6.
*CAS — 5451-09-2.*
*ATC — L01XD04.*
*ATC Vet — QL01XD04.*

## Hexaminolevulinate Hydrochloride *(USAN)*

P-1026. Hexyl 5-amino-4-oxopentanoate hydrochloride.

$C_{11}H_{21}NO_3$,HCl = 251.8.
*CAS — 140898-91-5.*



*(hexaminolevulinate)*

## Methyl Aminolevulinate Hydrochloride *(USAN)*

Methyl Aminolaevulinate Hydrochloride; Metilaminolevulinato, hidrocloruro de; P-1202. Methyl 5-amino-4-oxopentanoate hydrochloride.

$C_6H_{11}NO_3$,HCl = 181.6.
*CAS — 79416-27-6.*
*ATC — L01XD03.*
*ATC Vet — QL01XD03.*

### Adverse Effects and Precautions

The mechanism of action of topical 5-aminolevulinic acid or its derivatives generally results in local phototoxicity, manifest as a localised burning or stinging sensation, erythema, oedema, pruritus, scabbing, or pain. Symptoms are usually mild to moderate, and transient. During treatment, patients should be advised to avoid sunlight or prolonged exposure to bright light.

Other common adverse effects on the skin include scaling or crusting, ulceration, suppuration, blistering, bleeding, sensation of heat, erosion or exfoliation, and skin infection. Urticaria, rash, and changes in skin pigmentation may also occur. Application site discharge, eczema, and allergic contact dermatitis have been reported. Other common adverse effects include paraesthesia and headache. Nausea, fatigue, eye swelling or eye pain, and wound haemorrhage have been reported.

**Handling.** US licensed product information warns that nitrile gloves should be worn during application and removal of methyl aminolevulinate hydrochloride cream; vinyl or latex gloves do not provide adequate protection.

**Hypersensitivity.** Allergic reactions to aminolevulinic acid[1] and methyl aminolevulinate[2] have been reported.

1. Gniazdowska B, *et al.* Allergic contact dermatitis from δ-aminolevulinic acid used for photodynamic therapy. *Contact Dermatitis* 1998; **38:** 348–9.
2. Wulf HC, Philipsen P. Allergic contact dermatitis to 5-aminolaevulinic acid methylester but not to 5-aminolaevulinic acid after photodynamic therapy. *Br J Dermatol* 2004; **150:** 143–5.

**Porphyria.** 5-Aminolevulinic acid and its derivatives are considered to be unsafe in patients with porphyria.

### Interactions

Use with other known photosensitisers such as griseofulvin, thiazide diuretics, sulfonylureas, phenothiazines, sulfonamides, and tetracyclines might increase the photosensitivity reaction commonly seen with 5-aminolevulinic acid or its derivatives.

**St John's wort.** A patient taking St John's wort had a pronounced phototoxic reaction consisting of an erythematous rash and swelling of the face, neck, and hands, 6 hours after receiving oral aminolevulinic acid. Although both drugs have been associated with photosensitivity, the authors suggested a synergistic effect had occurred. Tests *in vitro* appeared to confirm this.[1]

1. Ladner DP, *et al.* Synergistic toxicity of δ-aminolaevulinic acid-induced protoporphyrin IX used for photodiagnosis and hypericum extract, a herbal antidepressant. *Br J Dermatol* 2001; **144:** 916–8.

### Pharmacokinetics

After intravenous and oral doses of aminolevulinic acid hydrochloride equivalent to 100 mg of aminolevulinic acid, the mean half-life of aminolevulinic acid is stated to be about 0.83 hours and 0.7 hours, respectively; oral bioavailability is about 50 to 60%. *In-vitro* studies of dermal absorption found that the mean cumulative absorption of methyl aminolevulinate

through healthy human skin after 24 hours was 0.26% of a dose and a skin depot containing 4.9% of the dose was formed.

◊ References.

1. Juzeniene A, *et al.* Topical application of 5-aminolaevulinic acid, methyl 5-aminolaevulinate and hexyl 5-aminolaevulinate on normal human skin. *Br J Dermatol* 2006; **155:** 791–9.

## Uses and Administration

5-Aminolevulinic acid is a naturally occurring haem precursor that is metabolised in the body to protoporphyrin IX, a photosensitiser, and then to haem. It has been formulated for topical use in photodynamic therapy (see under Porfimer Sodium, p.764). It is used to treat actinic keratoses and basal cell carcinoma (see below). Aminolevulinic acid hydrochloride is applied topically as a 20% solution in the treatment of non-hyperkeratotic actinic keratoses of the face or scalp. This is followed, 14 to 18 hours later, by illumination with blue wavelength light sufficient to supply a dose of 10 J/cm$^2$. Treatment may be repeated once after 8 weeks if necessary.

Methyl aminolevulinate hydrochloride is a derivative of 5-aminolevulinic acid that is applied topically for the treatment of non-hyperkeratotic, non-pigmented actinic keratoses of the face or scalp when other therapies are considered inappropriate. It is also used for the treatment of superficial and/or nodular basal cell carcinoma unsuitable for other therapies. In some countries, methyl aminolevulinate is licensed for the treatment of Bowen's disease (squamous cell carcinoma *in situ*) when surgical excision is inappropriate. A cream containing the equivalent of 16% methyl aminolevulinate is applied to the lesions and covered with an occlusive dressing. After 3 hours the cream is removed and the lesions are exposed to red wavelength light in a dose of 75 J/cm$^2$; exposure to the cream for more than 4 hours should be avoided. For actinic keratoses, one session of photodynamic therapy is given, and again after 3 months if necessary. For basal cell carcinoma or Bowen's disease two sessions are given a week apart.

Patients should avoid sunlight or bright light sources for about 2 days after application.

Hexaminolevulinate hydrochloride is used as a diagnostic agent for the detection of bladder cancer (p.659), and is under investigation for the photodynamic therapy of bladder cancer.

**Malignant neoplasms.** The use of topical aminolevulinic acid or methyl aminolevulinate in the photodynamic therapy (PDT) of *actinic keratosis* and *basal cell carcinoma* (p.673) has been reviewed.[1-5] Both aminolevulinic acid and methyl aminolevulinate are considered to be effective in clearing non-hyperkeratotic actinic keratoses of the face and scalp, with response rates comparable to topical 5-fluorouracil and cryotherapy; cosmetic response is superior to that with cryotherapy.[6,7] More than 80% of lesions cleared completely after 2 treatments with aminolevulinic acid PDT; a recurrence rate of 19% was reported over 12 months.[8] Single treatment with methyl aminolevulinate is effective for thin lesions but thicker, non-responsive lesions may benefit from repeated treatment.[9] No significant differences in efficacy were seen between PDT with aminolevulinic acid or methyl aminolevulinate in the treatment of scalp actinic keratosis.[10] Aminolevulinic acid is considered as effective as cryotherapy but with superior healing and cosmetic results for superficial basal cell carcinoma; it is less effective for nodular disease.[6] Intralesional use has been investigated to improve penetration of the photosensitiser, with good preliminary results.[11] Methyl aminolevulinate topical PDT may be preferable for difficult-to-treat basal cell carcinoma.[12,13] Aminolevulinic acid is as effective in *Bowen's disease* (squamous cell carcinoma *in situ*) as cryotherapy or 5-fluorouracil, but with fewer adverse effects.[6] Topical methyl aminolevulinate PDT was more effective and cosmetically acceptable compared with topical 5-fluorouracil for pre-malignant skin disease in a small study in organ transplant recipients.[14]

PDT using oral 5-aminolevulinic acid as the photosensitiser, at doses of 30 or 60 mg/kg, has been used to treat *Barrett's oesophagus*, which is a major risk factor for oesophageal adenocarcinoma (p.664).[15,16] A small study found similar clinical responses with low-dose (30 mg/kg) and high-dose (60 mg/kg) aminolevulinic acid protocols.[17] Good long-term results have been reported with aminolevulinic acid PDT in patients with early neoplasia or high-grade intraepithelial neoplasia.[18]

Aminolevulinic acid is also under investigation for the photodynamic detection and treatment of *brain tumours* (p.660).[19] It has also been used for the fluorescence detection of *pleural malignancies*.[20]

An intravesical solution of aminolevulinic acid has been instilled for the detection[21] and management[22] of superficial *bladder cancer* (p.659). Hexaminolevulinate is used for the diagnosis of bladder cancer, and it appears to be more effective than standard white light cystoscopy.[23-25]

A topical application of 5-aminolevulinic acid 3% has been tried in the treatment of *cervical intraepithelial neoplasia*, with poor response.[26]

1. Ormrod D, Jarvis B. Topical aminolevulinic acid HCl photodynamic therapy. *Am J Clin Dermatol* 2000; **1:** 133–9.
2. Gupta AK, Ryder JE. Photodynamic therapy and topical aminolevulinic acid: an overview. *Am J Clin Dermatol* 2003; **4:** 699–708.
3. Siddiqui MAA, *et al.* Topical methyl aminolevulinate. *Am J Clin Dermatol* 2004; **5:** 127–37.
4. Marmur ES, *et al.* A review of laser and photodynamic therapy for the treatment of nonmelanoma skin cancer. *Dermatol Surg* 2004; **30:** 264–71.
5. Lehmann P. Methyl aminolaevulinate-photodynamic therapy: a review of clinical trials in the treatment of actinic keratoses and nonmelanoma skin cancer. *Br J Dermatol* 2007; **156:** 793–801.
6. Morton CA, *et al.* Guidelines for topical photodynamic therapy: report of a workshop of the British Photodermatology Group. *Br J Dermatol* 2002; **146:** 552–67.
Also available at: http://www.bad.org.uk/healthcare/guidelines/topical_photodynamic_therapy.pdf (accessed 09/05/08)
7. Morton C, *et al.* AKtion Investigators. Intraindividual, right-left comparison of topical methyl aminolaevulinate-photodynamic therapy and cryotherapy in subjects with actinic keratoses: a multicentre, randomized controlled study. *Br J Dermatol* 2006; **155:** 1029–36.
8. Tschen EH, *et al.* Phase IV ALA-PDT Actinic Keratosis Study Group. Photodynamic therapy using aminolaevulinic acid for patients with nonhyperkeratotic actinic keratoses of the face and scalp: phase IV multicentre clinical trial with 12-month follow up. *Br J Dermatol* 2006; **155:** 1262–9.
9. Tarstedt M, *et al.* A randomized multicenter study to compare two treatment regimens of topical methyl aminolevulinate (Metvix )-PDT in actinic keratosis of the face and scalp. *Acta Derm Venereol* 2005; **85:** 424–8.
10. Moloney FJ, Collins P. Randomized, double-blind, prospective study to compare topical 5-aminolaevulinic acid methylester with topical 5-aminolaevulinic acid photodynamic therapy for extensive scalp actinic keratosis. *Br J Dermatol* 2007; **157:** 87–91.
11. Cappugi P, *et al.* New proposal for the treatment of nodular basal cell carcinoma with intralesional 5-aminolevulinic acid. *J Chemother* 2004; **16:** 491–3.
12. Horn M, *et al.* Topical methyl aminolaevulinate photodynamic therapy in patients with basal cell carcinoma prone to complications and poor cosmetic outcome with conventional treatment. *Br J Dermatol* 2003; **149:** 1242–9.
13. Vinciullo C, *et al.* Photodynamic therapy with topical methyl aminolaevulinate for 'difficult-to-treat' basal cell carcinoma. *Br J Dermatol* 2005; **152:** 765–72.
14. Perrett CM, *et al.* Treatment of post-transplant premalignant skin disease: a randomized intrapatient comparative study of 5-fluorouracil cream and topical photodynamic therapy. *Br J Dermatol* 2007; **156:** 320–8.
15. Ackroyd R, *et al.* Photodynamic therapy for dysplastic Barrett's oesophagus: a prospective, double blind, randomised, placebo controlled trial. *Gut* 2000; **47:** 612–17.
16. Barr H. Barrett's esophagus: treatment with 5-aminolevulinic acid photodynamic therapy. *Gastrointest Endosc Clin N Am* 2000; **10:** 421–37.
17. Kelty CJ, *et al.* Comparison of high- vs low-dose 5-aminolevulinic acid for photodynamic therapy of Barrett's esophagus. *Surg Endosc* 2004; **18:** 452–8.
18. Pech O, *et al.* Long-term results of photodynamic therapy with 5-aminolevulinic acid for superficial Barrett's cancer and high-grade intraepithelial neoplasia. *Gastrointest Endosc* 2005; **62:** 24–30.
19. Friesen SA, *et al.* 5-Aminolevulinic acid-based photodynamic detection and therapy of brain tumors (review). *Int J Oncol* 2002; **21:** 577–82.
20. Baas P, *et al.* Fluorescence detection of pleural malignancies using 5-aminolaevulinic acid. *Chest* 2006; **129:** 718–24.
21. Zaak D, *et al.* Role of 5-aminolevulinic acid in the detection of urothelial premalignant lesions. *Cancer* 2002; **95:** 1234–8. Correction. *ibid.*; 2580.
22. Kriegmair M, *et al.* Early clinical experience with 5-aminolevulinic acid for the photodynamic therapy of superficial bladder cancer. *Br J Urol* 1996; **77:** 667–71.
23. Anonymous. Hexyl aminolevulinate: 5-ALA hexylester, 5-ALA hexylesther, aminolevulinic acid hexyl ester, hexaminolevulinate, hexyl-5-aminolevulinate, P 1206. *Drugs R D* 2005; **6:** 235–8.
24. Jocham D, *et al.* Improved detection and treatment of bladder cancer using hexaminolevulinate imaging: a prospective, phase III multicenter study. *J Urol (Baltimore)* 2005; **174:** 862–6.
25. Frampton JE, Plosker GL. Hexyl aminolevulinate: in the detection of bladder cancer. *Drugs* 2006; **66:** 571–8.
26. Barnett AA, *et al.* A randomised, double-blind, placebo-controlled trial of photodynamic therapy using 5-aminolaevulinic acid for the treatment of cervical intraepithelial neoplasia. *Int J Cancer* 2003; **103:** 829–32.

**Skin disorders.** 5-Aminolevulinic acid has been used topically in the photodynamic therapy (PDT) of skin conditions such as psoriasis, recalcitrant viral warts, acne vulgaris, and cutaneous T-cell lymphoma.[1] Variable results have been reported with this therapy in patients with plaque psoriasis; in one study therapy was generally well-tolerated,[2] but in the other all patients reported stinging or burning during irradiation.[3] A study comparing 5-aminolevulinic acid and methyl aminolevulinate for PDT of acne found both to be equally effective, although methyl aminolevulinate seemed to be better tolerated.[4] Topical methyl aminolevulinate-based PDT has been found to be effective in the treatment of facial acne vulgaris.[5,6] PDT with 5-aminolevulinic acid has been reported to significantly improve an infected leg ulcer resistant to conventional therapies.[7] A topical 10% solution has been used with intraurethral PDT to treat condylomata acuminata.[8]

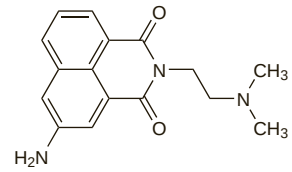
1. Ibbotson SH. Topical 5-aminolaevulinic acid photodynamic therapy for the treatment of skin conditions other than nonmelanoma skin cancer. *Br J Dermatol* 2002; **146:** 178–88.
2. Smits T, *et al.* A placebo-controlled randomized study on the clinical effectiveness, immunohistochemical changes and protoporphyrin IX accumulation in fractionated 5-aminolaevulinic acid-photodynamic therapy in patients with psoriasis. *Br J Dermatol* 2006; **155:** 429–36.
3. Radakovic-Fijan S, *et al.* Topical aminolaevulinic acid-based photodynamic therapy as a treatment option for psoriasis? Results of a randomized, observer-blinded study. *Br J Dermatol* 2005; **152:** 279–83.
4. Wiegell SR, Wulf HC. Photodynamic therapy of acne vulgaris using 5-aminolevulinic acid versus methyl aminolevulinate. *J Am Acad Dermatol* 2006; **54:** 647–51.
5. Wiegell SR, Wulf HC. Photodynamic therapy of acne vulgaris using methyl aminolaevulinate: a blinded, randomized, controlled trial. *Br J Dermatol* 2006; **154:** 969–76.
6. Hörfelt C, *et al.* Topical methyl aminolaevulinate photodynamic therapy for treatment of facial acne vulgaris: results of a randomized, controlled study. *Br J Dermatol* 2006; **155:** 608–13.
7. Clayton TH, Harrison PV. Photodynamic therapy for infected leg ulcers. *Br J Dermatol* 2007; **156:** 384–5.
8. Wang XL, *et al.* Topical 5-aminolaevulinic acid-photodynamic therapy for the treatment of urethral condylomata acuminata. *Br J Dermatol* 2004; **151:** 880–5.

## Preparations

**Proprietary Preparations** (details are given in Part 3)
***Austria:*** Metvix; ***Belg.:*** Metvix; ***Cz.:*** Hexvix; MC 506; Metvix; ***Denm.:*** Metvix; ***Fin.:*** Hexvix; Metvix; ***Fr.:*** Metvixia; ***Ger.:*** Metvix; ***Gr.:*** Hexvix; ***Ital.:*** Metvix; ***Neth.:*** Hexvix; Metvix; ***Norw.:*** Hexvix; Metvix; ***NZ:*** Metvix; ***Port.:*** Gliolan; Hexvix; Metvix; ***S.Afr.:*** Metvix; ***Spain:*** Metvix; ***Swed.:*** Hexvix; Metvix; ***Switz.:*** Metvix; ***UK:*** Metvix; Porphin; ***USA:*** Levulan Kerastick; Metvixia.

## Amonafide *(rINN)*

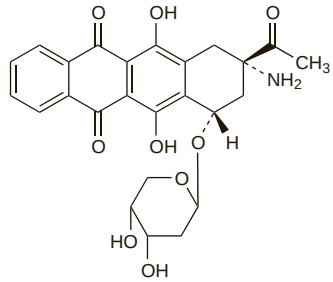
Amonafida; Amonafidum; M-FA-142; Nafidimide; NSC-308847.
3-Amino-*N*-[2-(dimethylamino)ethyl]naphthalimide.
Амонафид
$C_{16}H_{17}N_3O_2$ = 283.3.
*CAS — 69408-81-7.*



### Profile
Amonafide is a topoisomerase inhibitor. Amonafide malate is under investigation for the treatment of acute myeloid leukaemia. Amonafide hydrochloride has also been investigated, but studies were stopped due to serious adverse effects.

## Amrubicin *(USAN, rINN)*

Amrubicina; Amrubicine; Amrubicinum; SM-5887. (+)-(7*S*,9*S*)-9-Acetyl-9-amino-7-[(2-deoxy-β-D-*erythro*-pentopyranosyl)-oxy]-7,8,9,10-tetrahydro-6,11-dihydroxy-5,12-naphthacenedione.
Амрубицин
$C_{25}H_{25}NO_9$ = 483.5.
*CAS — 110267-81-7 (amrubicin); 110311-30-3 (amrubicin hydrochloride).*



### Profile
Amrubicin is a synthetic anthracycline derivative related to compounds such as doxorubicin (p.712). Amrubicin hydrochloride is used as an antineoplastic in the treatment of lung cancer, and is under investigation in the treatment of superficial bladder cancers, and lymphomas.