AUSTRALIAN PRODUCT INFORMATION

NICOTINIC ACID

(nicotinic acid) tablets

1 NAME OF THE MEDICINE

Nicotinic acid 250 mg

2 QUALITATIVE AND QUANTITATIVE COMPOSITION

Each tablet contains 250 mg of nicotinic acid as the active ingredient.

Excipients with known effect: gluten, sulfites and sugars as lactose.

For the full list of excipients, see Section 6.1 LIST OF EXCIPIENTS.

3 PHARMACEUTICAL FORM

NICOTINIC ACID tablets are white, bevelled edge tablet with breakline on one side.

4 CLINICAL PARTICULARS

4.1 THERAPEUTIC INDICATIONS

1. The treatment of hyperlipidaemia, hypertriglyceridaemia and Frederickson-Lees Levy hyperlipoproteinaemia type II, IIB, III, IV and V (as adjunctive therapy in addition to diet and other measures); and

2. Pellagra (note: pellagra in Australia is limited to special situations not typical of the general lifestyle. A variety of other non-nutritional factors may also lead to the development of the disease).

4.2 DOSE AND METHOD OF ADMINISTRATION

In adults

Pellagra: 250 mg (one tablet) twice a day.

Hypercholesterolaemia, hypertriglyceridaemia: 250 mg three times daily increased by 250 mg increments every fourth day until a final daily dose of 3 to 4.5 g is reached.

Individual dosage is recommended because lipid reduction is dose related. Initially plasma cholesterol and triglyceride levels should be monitored.

Tablets should be taken orally after meals.

Following oral administration, nicotinic acid induced vasodilation occurs within 20 minutes and persists for about 20 to 60 minutes.

4.3 CONTRAINDICATIONS

NICOTINIC ACID may exacerbate hepatic dysfunction and large doses may exacerbate peptic ulcer, overt diabetes mellitus, gout or hyperuricaemia.

Large doses of NICOTINIC ACID should not be used by persons with heart or gallbladder disease, arterial bleeding or glaucoma.

Contraindicated in cases of recent myocardial infarction.

NICOTINIC ACID is contraindicated in patients with severe idiosyncratic reactions to it or those who exhibit a sudden fall in peripheral vascular resistance.

4.4 SPECIAL WARNINGS AND PRECAUTIONS FOR USE

Identified Precautions

Antihypertensive Drugs

Patients taking antihypertensive drugs should consult a physician before taking NICOTINIC ACID. Antihypertensive drugs may have an additive vasodilating effect and produce postural hypotension.

Liver Function

Frequent monitoring of liver function should be performed during therapy to ascertain that the drug has no adverse effects.

Glucose Tolerance

As decreased glucose tolerance may occur, glucose tolerance tests should be performed regularly. Adjustment of diet and/or hypoglycaemic therapy may be necessary.

Serum Uric Acid Levels

Frequent monitoring of serum uric acid levels is advised as elevated uric acid levels may occur during long-term therapy.

Gastrointestinal Irritation or Peptic Ulcer History

Nicotinic acid causes release of histamine from the mast cells to stimulate gastric secretion of hydrochloric acid. Therefore, patients prone to gastrointestinal irritation or with a history of peptic ulcer should be closely supervised.

Myocardial Infarction

Nicotinic acid therapy should be withdrawn if the patient has a myocardial infarction.

Use in the Elderly

No data available.

Paediatric Use

No data available.

Effects on Laboratory Tests

Nicotinic acid may cause false elevation in fluorometric determinations of urinary catecholamines and false positive tests for urinary glucose when Benedict's reagent is used. Nicotinic acid has also been reported to give false positive results for blood bilirubin tests.

4.5 INTERACTIONS WITH OTHER MEDICINES AND OTHER FORMS OF INTERACTIONS

Adrenergic blocking agents

Due to an additive vasodilating effect, postural hypotension may occur when nicotinic acid is added to the regimen of patients taking adrenergic blocking agents.

Anti-hyperglycaemic therapy

Because nicotinic acid can cause hyperglycaemic, dosage adjustment of insulin or oral anti-hyperglycaemic therapy may be required in diabetic patients.

Aspirin

Concurrent use of aspirin and nicotinic acid may result in a reduction of the warmth and flushing associated with nicotinic acid use. Also, concurrent use of aspirin may result in an increased and prolonged nicotinic acid concentration, and so the potential for nicotinic acid toxicity may exist.

Clonidine

Concomitant nicotinic acid and clonidine has been reported to result in reduction in flushing of skin secondary to nicotinic acid.

Colestipol

Nicotinic acid absorption may be affected by administration with colestipol. Combined use of these two drugs resulted in lower plasma cholesterol concentrations than were achieved with colestipol alone.

Glipizide

Concomitant administration of glipizide and nicotinic acid may result in loss of blood glucose control since nicotinic acid can cause hyperglycaemia.

Isoniazid

Concomitant administration of isoniazid and nicotinic acid may cause nicotinic acid requirements to be increased, but pellagra is rare, only occurring in patients with an underlying nicotinic acid deficiency.

Lovastatin/Pravastatin/Simvastatin

The concurrent use of lovastatin, pravastatin or simvastatin and nicotinic acid may be associated with myopathy and an increased risk of rhabdomyolysis, and acute renal failure. Symptoms of myopathy and rhabdomyolysis should be monitored.

Nicotine

If nicotinic acid and transdermal nicotine are used concurrently, flushing and dizziness after each nicotinic acid dose may occur.

Alcohol

In one case report concomitant alcohol and nicotinic acid therapy resulted in delirium (paranoid ideation and asterixis) and lactic acidosis.

4.6 FERTILITY, PREGNANCY AND LACTATION

Use in Pregnancy

Pregnancy Category: B2

Category B2: Drugs which have been taken by only a limited number of pregnant women and women of childbearing age, without an increase in the frequency of malformation or other direct or indirect harmful effects on the human fetus having been observed.

Studies in animals are inadequate or may be lacking, but available data show no evidence of an increased occurrence of fetal damage.

Contraindicated.

Use in Lactation

Contraindicated.

4.7 EFFECTS ON ABILITY TO DRIVE AND USE MACHINES

The effects of this medicine on a person's ability to drive and use machines were not assessed as part of its registration.

4.8 ADVERSE EFFECTS (UNDESIRABLE EFFECTS)

Cardiovascular

Acute flush, pounding in the head, sensation of heat, headache, hypotension. Atrial fibrillation and other arrhythmias in patients with CHD.

Dermatological

Pruritus, dryness with mild epidermal exfoliation, brown pigmentation, hyperkeratosis, urticaria, furunculosis, rash. All these adverse reactions are reversible on cessation of drug therapy.

Endocrine

Increased insulin requirements in diabetic patients, hypothyroidism.

Gastrointestinal

Nausea, vomiting, diarrhoea, heartburn, flatulence, activation of peptic ulcer.

Hepatic

Cholestatic jaundice, elevated liver function tests, ascites, hepatomegaly, patchy fibrosis, areas of necrosis, cholestasis and lymphocyte infiltration around the bile ducts.

Nervous system

Nervousness.

Others

Hyperuricemia, toxic amblyopia.

Reporting Suspected Adverse Effects

Reporting suspected adverse reactions after registration of the medicinal product is important. It allows continued monitoring of the benefit-risk balance of the medicinal product. Healthcare professionals are asked to report any suspected adverse reactions at www.tga.gov.au/reporting-problems.

4.9 OVERDOSE

Symptoms: Cutaneous flush, pruritus, vomiting, diarrhoea, dyspepsia, syncope, severe abdominal cramps.

Treatment: Discontinue drug and institute general supportive measures.

For information on the management of overdose, contact the Poisons Information Centre on 13 11 26 (Australia).

5 PHARMACOLOGICAL PROPERTIES

Nicotinic acid is a water-soluble B complex vitamin which is able to reduce serum lipids.

5.1 PHARMACODYNAMIC PROPERTIES

Mechanism of Action

NICOTINIC ACID lowers serum cholesterol and triglyceride concentrations by inhibiting the synthesis of very low density lipoproteins (VLDL) which are the precursors to the formation of low-density lipoproteins, the principal carrier of blood cholesterol. Several possible modes of action have been proposed, including inhibition of hepatic synthesis of lipoproteins containing apolipoprotein B-100, promotion of lipoprotein lipase activity, and reduction of free fatty acid mobilisation from adipose tissue with an increase in faecal output of sterols. Oral therapy produces reduced triglyceride concentrations within several hours and reduced cholesterol concentrations within several days.

NICOTINIC ACID also has a vasodilation effect when administered in large doses, identified by flushing of the skin while plasma nicotinic acid levels are rising. This process is believed to be mediated by prostacyclin. Vasodilation occurs within 20 minutes of an oral dose and persists for about 20-60 minutes.

NICOTINIC ACID has been reported to stimulate histamine release resulting in increased gastric motility and acid production which may activate peptic ulcer. Reports have also indicated that large doses of nicotinic acid may decrease uric acid excretion and impair glucose tolerance. These effects may result in precipitation of an episode of gout in susceptible patients and may necessitate adjustment of diet and anti-hyperglycaemic therapy in diabetic patients.

The normal physiological role of nicotinic acid is as a component of the coenzymes NAD and NADP which are essential for oxidation-reduction reactions in tissue respiration. Nicotinamide, a metabolite of nicotinic acid, possesses similar function as a vitamin but has no pharmacological value in reducing lipids.

Clinical Trials

No data available.

5.2 PHARMACOKINETIC PROPERTIES

Absorption and distribution

Nicotinic acid is readily absorbed from the gastrointestinal tract following oral administration and is widely distributed in the body tissues.

Metabolism and excretion

It is metabolised in the liver to nicotinamide when taken in physiological doses but when therapeutic doses are taken only a portion is converted to nicotinamide with the remainder eventually being excreted unchanged in the urine. Nicotinamide is widely distributed in the body and is further metabolised in the liver to N-methylnicotinamide and the 2-pyridone and 4-pyridone derivatives with some nicotinuric acid also being formed before being excreted in the urine. The elimination half-life is approximately 45 minutes, and time to peak serum concentration after oral administration is also 45 minutes.

5.3 PRECLINICAL SAFETY DATA

Genotoxicity

No data available.

Carcinogenicity

No data available.

6 PHARMACEUTICAL PARTICULARS

6.1 LIST OF EXCIPIENTS

Lactose monohydrate, wheat starch, povidone, purified talc and magnesium stearate.

6.2 INCOMPATIBILITIES

Incompatibilities were either not assessed or not identified as part of the registration of this medicine.

6.3 SHELF LIFE

In Australia, information on the shelf life can be found on the public summary of the Australian Register of Therapeutic Goods (ARTG). The expiry date can be found on the packaging.

6.4 SPECIAL PRECAUTIONS FOR STORAGE

Store below 30ºC.

6.5 NATURE AND CONTENTS OF CONTAINER

Container type: glass bottle

Pack sizes: Available in bottles of 100’s or 200’s.

Some strengths, pack sizes and/or pack types may not be marketed.

Australian Register of Therapeutic Goods (ARTG)

AUST R 27193 - NICOTINIC ACID 250mg

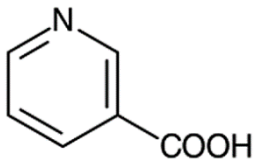
6.6 SPECIAL PRECAUTIONS FOR DISPOSAL

In Australia, any unused medicine or waste material should be disposed of by taking it to your local pharmacy.

6.7 PHYSICOCHEMICAL PROPERTIES

Chemical Structure

Structural formula:



Molecular weight: 123.1

CAS Number

59-67-6

7 MEDICINE SCHEDULE (POISONS STANDARD)

S3 (Pharmacist only medicine)

8 SPONSOR

Alphapharm Pty Ltd trading as Viatris

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30 – 34 Hickson Road

Millers Point NSW 2000

www.viatris.com.au

Phone: 1800 274 276

9 DATE OF FIRST APPROVAL

21/10/1991

10 DATE OF REVISION

09/01/2025

Summary Table of Changes

Section Changed	Summary of New Information
All	Minor editorial changes
1	Revised name of medicine in alignment to the ARTG record
2	Update to S1 declaration

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