



Cinnamic acid: Human health tier II assessment

02 March 2018

- Chemicals in this assessment
- Preface
- Grouping Rationale
- Import, Manufacture and Use
- Restrictions
- Existing Worker Health and Safety Controls
- Health Hazard Information
- Risk Characterisation
- NICNAS Recommendation
- References

Chemicals in this assessment

Chemical Name in the Inventory	CAS Number
2-Propenoic acid, 3-phenyl-, (Z)-	102-94-3
2-Propenoic acid, 3-phenyl-, (E)-	140-10-3
2-Propenoic acid, 3-phenyl-	621-82-9

Preface

This assessment was carried out by staff of the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) using the Inventory Multi-tiered Assessment and Prioritisation (IMAP) framework.

The IMAP framework addresses the human health and environmental impacts of previously unassessed industrial chemicals listed on the Australian Inventory of Chemical Substances (the Inventory).

The framework was developed with significant input from stakeholders and provides a more rapid, flexible and transparent approach for the assessment of chemicals listed on the Inventory.

Stage One of the implementation of this framework, which lasted four years from 1 July 2012, examined 3000 chemicals meeting characteristics identified by stakeholders as needing priority assessment. This included chemicals for which NICNAS already held exposure information, chemicals identified as a concern or for which regulatory action had been taken overseas, and chemicals detected in international studies analysing chemicals present in babies' umbilical cord blood.

Stage Two of IMAP began in July 2016. We are continuing to assess chemicals on the Inventory, including chemicals identified as a concern for which action has been taken overseas and chemicals that can be rapidly identified and assessed by using Stage One information. We are also continuing to publish information for chemicals on the Inventory that pose a low risk to

human health or the environment or both. This work provides efficiencies and enables us to identify higher risk chemicals requiring assessment.

The IMAP framework is a science and risk-based model designed to align the assessment effort with the human health and environmental impacts of chemicals. It has three tiers of assessment, with the assessment effort increasing with each tier. The Tier I assessment is a high throughput approach using tabulated electronic data. The Tier II assessment is an evaluation of risk on a substance-by-substance or chemical category-by-category basis. Tier III assessments are conducted to address specific concerns that could not be resolved during the Tier II assessment.

These assessments are carried out by staff employed by the Australian Government Department of Health and the Australian Government Department of the Environment and Energy. The human health and environment risk assessments are conducted and published separately, using information available at the time, and may be undertaken at different tiers.

This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

For more detail on this program please visit: www.nicnas.gov.au

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ACRONYMS & ABBREVIATIONS

Grouping Rationale

Cinnamic acid occurs naturally in many plants and traditional foods in *trans* and *cis* forms. The *trans*-form (CAS No. 140-10-3) is more stable than the *cis*-, and is in most of the commercially available products. The *trans*-form is required for lignin formation in plants, and is found in the essential oils of basil, balsam of Peru, Chinese cinnamon, *Melaleuca bracteata* and *Alpinia galangal*, Asian and American styrax and cocoa leaves. The *cis*-form (CAS No. 102-94-3) has been found in the oil of *Alpinia malacencis* (Opydke, 1979; Adams, 2004). The CAS No. 621-82-9 is the generic CAS for cinnamic acid of unspecified isomeric purity.

Import, Manufacture and Use

Australian

No specific Australian use, import, or manufacturing information has been identified.

International

The following international uses have been identified through: Galleria Chemica; the European Commission Cosmetic Ingredients and Substances (CosIng) database; the United States (US) Personal Care Products Council International Nomenclature of Cosmetic Ingredients (INCI) Dictionary; and other sources (PubMed).

The chemicals (CAS Nos. 140-10-3 and 621-82-9) have reported cosmetic and domestic uses as fragrance ingredients in skin conditioning agents, fine fragrances, and other cosmetic and personal care products, and in household cleaners and detergents.

The chemicals (CAS Nos. 140-10-3 and 621-82-9) have documented use in the Compilation of Ingredients used in Cosmetics in the United States (CIUCUS, 2011).

The chemicals (CAS Nos. 140-10-3 and 621-82-9) have reported site-limited uses as precursors for synthesising cinnamic esters which have a wide variety of applications. *Trans*-cinnamic acid (CAS No. 140-10-3) is used as a smoke producing agent to simulate fires for firefighting training.

The chemicals (CAS Nos. 102-94-3, 140-10-3 and 621-82-9) have reported non-industrial uses as food additives and in certain pharmaceuticals. *Cis*-cinnamic acid (CAS No. 102-94-3) has reported non-industrial use as a plant growth inhibitor and is also a precursor to the sweetener aspartame.

Cinnamic acid occurs naturally in storax, balsam of Peru, oil of cinnamon and coca leaves.

Restrictions

Australian

No known restrictions have been identified.

International

No known restrictions have been identified.

Existing Worker Health and Safety Controls

Hazard Classification

The chemicals are not listed on the Hazardous Chemical Information System (HCIS) (Safe Work Australia).

Exposure Standards

Australian

No specific exposure standards are available.

International

No specific exposure standards are available.

Health Hazard Information

Toxicokinetics

Studies in animals and humans have shown that cinnamic acid is rapidly absorbed from the gastrointestinal tract, metabolised and excreted primarily in the urine, and to a minor extent in the faeces.

Following oral administration in rats, cinnamic acid is excreted within 24 hours in the urine at 82 %, and in the faeces at 0.9 %, indicating high gastrointestinal absorption. When radiolabelled cinnamic acid was orally administered to rats at 0.0005–2.5 mmol, about 73–88 % was recovered in the urine and trace amounts were present in the carcasses after 3 days. This indicated that cinnamic acid is readily and quantitatively excreted at all dose levels (Bickers et al., 2005; Letizia et al., 2005).

Following oral administration, the metabolic profile of cinnamic acid is similar for both humans and rodents. The chemical is first converted to acyl coenzyme-A (CoA) esters which either undergo i) glycine conjugation via *N*-acetyl transferase catalysis, or ii) rapid beta-oxidation to benzoylCoA (primary pathway). BenzoylCoA can conjugate with glycine to form hippuric acid or the CoA thioester can be hydrolysed to free benzoic acid which is then excreted (Adams et al., 2004; Belsito et al., 2007).

In both rats and mice, the urinary metabolites identified are hippuric acid (major metabolite at 44–77 %), benzoyl glucuronide, 3-hydroxy-3-phenylpropionic acid, benzoic acid, *p*-hydroxyhippuric acid and unchanged cinnamic acid. Acetophenone and cinnamoylglycine were identified only in the urine of mice. At higher doses (>80 mg/kg bw), the amount of hippuric acid decreased, and the amount of benzoyl glucuronide and benzoic acid increased indicating saturation of the glycine conjugation pathway (Letizia et al., 2005; EFSA, 2017).

In mice at low doses, the glycine conjugation pathway competes with the beta-oxidation pathway. However with increasing dose levels, urinary hippuric acid increases while cinnamoylglycine levels decrease, indicating that *N*-acetyl transferase has high affinity but low capacity for cinnamic acid. Only a small proportion of benzoyl glucuronide was detected at all doses, indicating that this conjugation reaction is of minimal importance in mice (Adams et al., 2004; EFSA, 2017).

In humans, the major urinary metabolites identified are hippuric acid and glucuronic acid conjugates of benzoic acid (cinnamoyl glucuronide and benzoyl glucuronide) present in a ratio of 74: 24.5: 1.5, respectively. A small amount of the unchanged parent chemical was also observed. Patients with renal disease or hepatic disturbances excreted a higher ratio of urinary glucuronides (Letizia et al., 2005).

Cinnamic acid is moderately to highly absorbed dermally, with greater absorption occurring under occluded conditions. In a 24-hour percutaneous study in rhesus monkey, the absorption of radiolabelled cinnamic acid was measured following occluded and non-occluded applications, and urine was collected over 4 days. The amount absorbed was 38.6 % and 83.9 % for non-occluded and occluded conditions, respectively. In a 72-hour in vitro test in excised human abdominal skin, the absorption of radiolabelled cinnamic acid was 17.8 % and 60.8 % for non-occluded and occluded conditions, respectively (Letizia et al., 2005).

Studies in humans have shown that the chemical undergoes rapid systemic clearance following administration. In a test in healthy volunteers and those with hepatocellular disease (*n* = 125), serum cinnamic acid was measured at 5 and 20 minutes after injection of aqueous sodium cinnamate (0.1 cc/10 pounds). In normal subjects, the serum cinnamic acid level peaked at 5 minutes at 2.7–4.9 mg/100 mL, and rapidly declined to near control levels at 20 minutes at a rate of 4–6 %/minute. In subjects with liver damage, the average serum level at 5 minutes was 1.9–5.0 mg/100 mL, with a less significant rate of decline at 0.3–3.1 %/minute. In another study, the serum cinnamic acid levels of 11 healthy adult volunteers intravenously exposed to the chemical at 5 mg/kg bw peaked at 2.5 minutes and the chemical was cleared within 20 minutes (Letizia et al., 2005).

Cinnamic acid is the major intermediate metabolite of cinnamyl esters, cinnamyl alcohol and cinnamaldehyde, and was detected in several pharmacokinetic studies using these chemicals (Bickers et al., 2005).

Acute Toxicity

Oral

Based on the available data, the chemicals have low acute toxicity in animal tests following oral exposure.

The reported median lethal doses (LD50) values are 2000–5000 mg/kg bw in rats, and >5000 mg/kg bw in mice and guinea pigs (Bickers et al., 2005; Galleria Chemica).

Dermal

The chemicals have low acute toxicity in animal tests following dermal exposure.

The reported dermal LD50 was >5000 mg/kg bw in rabbits. The only clinical observation was slight erythema (Letizia et al., 2005; Galleria Chimica).

Inhalation

Based on the available acute toxicity and repeat dose toxicity data for animals (see **Repeated Dose Toxicity** section), the chemicals are not expected to have high acute toxicity.

In an acute inhalation study, rats and guinea pigs were exposed to pyrotechnically generated cinnamic smoke at concentrations of 920 mg/m³ for 30 minutes and 1630 mg/m³ for 45 minutes. Minimal pulmonary alveolar capillary congestion was observed in a few animals. The authors reported that these concentrations are 12 and 22 times higher than concentrations likely to be used in practical situations (~70 mg/m³) (Romano, Lukey & Salem, 2007).

Corrosion / Irritation

Skin Irritation

The chemicals are reported to be a slight skin irritants (at >10 %) in animal studies. The effects were not sufficient to warrant hazard classification.

Irritation was observed in 2 studies. In a 48-hour closed patch test, 5 female Ssc:AL guinea pigs were pre-treated with Freund's complete adjuvant, followed by treatment with 1–10 % of the chemical in propylene glycol. Slight irritation was observed at 10 % concentration (Letizia et al., 2005). The chemical (CAS No. 140-10-3) was slightly irritating when applied undiluted to intact or abraded rabbit skin for 24 hours, under occlusion (Opdyke, 1979).

No irritation was observed in 3 skin irritation studies. The chemical (CAS No. 140-10-3) was not irritating when applied at 20 µL aliquots of 15 % to the shaved backs, abdomen and flanks of 5 female Hartley guinea pigs up to 3 hours. In a maximisation pre-test, no irritation was observed when the chemical was applied (occlusively) on the clipped, shaved flank of albino female Ssc:AL guinea pigs (n = 3/dose) at doses of 2–200 mg/mL for 24 hours, with observation up to 72 hours. No irritation was observed in a pre-sensitisation study in 10 female Balb/c mice exposed to 15 % of the chemical six times over 14 days (Letizia et al., 2005).

In a photoallergy study, groups of male guinea pigs (n = 5/dose) were patch-tested (occlusively) with the chemical (CAS No. 140-10-3) at 0, 1, 10, 100 or 200 mg/mL (0.1, 1, 10 or 20 %) for 2 hours. The application sites were wiped after exposure and the animals were observed up to 4 days. No irritation was observed (Letizia et al., 2005).

Slight erythema in rabbits was observed as part of the acute dermal study described earlier (see **Acute Dermal Toxicity** section).

Eye Irritation

Only limited information is available. The chemical is not an eye irritant up to concentrations of 1 %. The effects observed were not sufficient to warrant hazard classification.

In an eye irritation study, the chemical (CAS No. 140-10-3) was instilled at 1 or 10 mg/mL (0.1–1 %) in distilled water into one eye of rabbits and guinea pigs (number of animals not stated). Observations were made for up to 24 hours. No irritation was observed (Bickers et al., 2005; Letizia et al., 2005).

Observation in humans

In a 48-hour closed patch test, the chemical (CAS No. 140-10-3) at 4 % in petrolatum did not irritate the backs of 25 healthy male volunteers (Letizia et al., 2005).

In an acute inhalation toxicity study, male volunteers were exposed to cinnamic acid smoke at 70 mg/m³ for 10 minutes. Transient minimal irritant symptoms (slight excess lacrimation, mild rhinorrhoea and bouts of non-productive coughing), and slight increases in respiratory rate, tidal volume and minute volume were reported. These effects returned to control values within 5–10 minutes after exposure. The pre- and post-exposure chest radiographs were normal (Romano, Lukey & Salem, 2007).

Sensitisation

Skin Sensitisation

Based on the available data, the chemicals were predominantly negative at 10 % concentration in guinea pig studies. Sensitisation was observed at 15 % concentration in guinea pig but not in mice; however, effects were reversible. No sensitisation was observed at up to 4 % in humans (see **Observation in Humans** section). Therefore, based on the weight of evidence, the chemicals are not likely skin sensitisers.

The chemical (CAS No. 140-10-3) was negative in several guinea pig sensitisation tests at a concentration of 10 %. This included up to 10 % in propylene glycol in a maximisation study, in ethanol/water in two Buehler tests, and in an unknown vehicle in a closed epicutaneous test (CET). Weak to moderate sensitisation was observed in a modified Freund's complete adjuvant test (FCAT) in guinea pigs with the chemical at 10 % in acetone (Bickers et al., 2005; Letizia et al., 2005).

In two guinea pig studies for non-immunologic contact urticaria, 10–15 female Hartley guinea pigs were challenged with the chemical (CAS No. 140-10-3) at doses up to 15 % in ethanol on both sides of the earlobes. Sensitisation (immediate erythema and swelling in the earlobe) was observed immediately. At the highest tested dose (15 %), maximum swelling was observed within 50 minutes, but subsided within 3 hours (Letizia et al., 2005).

In a mouse ear swelling assay, 10 female Balb/c mice were exposed epicutaneously to a 100 µL aliquot of the chemical (CAS No. 140-10-3) at 15 % on the abdomen and thorax, followed by topical application of the chemical at 15 % to the ears. The ear thickness was measured before challenge exposure and up to 48 hours after exposure. No sensitisation was observed (Letizia et al., 2005).

Observation in humans

Many studies are available for patch tests of the chemical (CAS No. 140-10-3) with patients with pre-existing allergies or sensitivities. The chemical itself does not cause sensitisation in healthy volunteers at up to 4 % concentration.

In a 48-hour closed patch test, no reactions were reported for the chemical at 4 % in petrolatum in 25 healthy male volunteers. The chemical was applied under occlusion on the forearms of all subjects for 5 alternate-day 48-hour periods (Letizia et al., 2005).

Cross-reactions of cinnamic acid were observed in patients who had previously reacted to balsam of Peru. In a 48-hour closed patch test, the chemicals were applied to eczema patients with pre-existing reactions. Positive results were observed in 51/200 patients exposed to 5 % cinnamic acid (isomer not stated) in petrolatum; in 1/42 patients exposed to 2 % *cis*-cinnamic acid; and in 32/128 patients exposed to 5 % *trans*-cinnamic acid. In a 24-hour study, 33/102 patients reacted to 5 % cinnamic acid in petrolatum. In patch test studies at 8 worldwide centers, reactions to the chemical (dose and vehicle not stated) were observed in 26/142 patients with pre-existing allergies to 25 % Peru balsam (Letizia et al., 2005).

In several contact urticaria studies, reactions were observed in a portion of the atopic and non-atopic dermatitis patients exposed to the chemical up to 5 % (Letizia et al., 2005).

Repeated Dose Toxicity

Oral

No repeat dose toxicity studies are available.

In 2-year repeat dose oral studies, cinnamaldehyde (CAS No. 104-55-2) which metabolises to cinnamic acid was administered to rats and mice. The reported no observed adverse effect levels (NOAEL) of 200–550 mg/kg bw/day from these studies are over 600 000 times the maximum daily exposure to cinnamic acid (Bickers et. al., 2005; Letizia et. al., 2005; NICNAS).

Dermal

No data are available.

Inhalation

Based on the available data, the chemicals are not considered to cause serious damage to health following repeated inhalation exposure.

In a 40-week repeat dose toxicity study, mice, rats and guinea pigs were exposed to cinnamic acid smoke at concentrations of 0, 31.6, 102.0 or 304.1 mg/m³ for 1 hour/day, 5 days/week. Minimal and non-specific histological changes in the respiratory tract were observed at all doses. Renal lesions were observed only in mice, and were considered to be incidental (Marrs et. al., 1989).

Rats and guinea pigs were exposed to pyrotechnically generated cinnamic smoke at concentrations of 920 mg/m³, 1 hour/day for 5 consecutive days. No mortality or toxicity effects in the larynx, trachea, lung, liver and kidney were observed (Romano, Lukey & Salem, 2007).

Genotoxicity

Based on the available in vitro data, the chemicals are generally negative in bacterial systems but positive in mammalian studies. Antimutagenicity was also reported in some in vitro studies. No in vivo studies are available. Cinnamaldehyde which metabolises to cinnamic acid is not considered to be genotoxic (NICNAS). Therefore, no classification is recommended.

The following results were reported in various in vitro assays (Bickers et. al., 2005; Letizia et. al., 2005):

- negative in an Ames and a modified Ames assay with strains of *Salmonella typhimurium* (TA1535, TA1537, TA1538, TA98 and TA100) up to 1000 µg/plate, with and without metabolic activation;
- negative in a rec assay using *Bacillus subtilis* strains H17 (rec+) and M45 (rec-);
- negative in cultures of *Escherichia coli* strain PQ37 at doses tested up to toxic levels, and in strain B/r WP2s (trpE65) (doses not reported);
- induced chromosomal aberrations in Chinese hamster ovary (CHO) cells at doses of 10 and 33.3 µM. The highest tested dose (100 µM) was toxic; and
- dose-related increases in an L5178Y mouse lymphoma cell forward mutation assay, with or without metabolic activation.

The chemical was also evaluated in several in vitro studies for antimutagenic activity. In general, no antimutagenic activity was reported in bacterial studies. However, when *E. coli* strain (WP2s) cells were irradiated with ultra-violet (UV), the chemical was observed to decrease UV-induced mutation by 12 % at a dose of 24 µmol/plate in one study and by 74.8 % in another study. When evaluated in the eukaryotic murine mammary tumour FM3A cell line, the chemical was reported to reduce ethyl methanesulfonate (EMS) and quinacrine-induced mutation frequencies by 40–55 % (Letizia et. al., 2005).

Carcinogenicity

No data are available.

Reproductive and Developmental Toxicity

No reproductive studies are available. No maternal or developmental toxicity was reported in the available developmental toxicity study. Cinnamaldehyde is not considered a reproductive or developmental toxicant (NICNAS). Therefore, no classification is recommended.

In a developmental toxicity study, groups of 14–15 pregnant female albino rats were administered the chemical orally at doses of 0, 5 or 50 mg/kg bw/day throughout pregnancy. On gestation day (GD) 20, 6–9 rats per group were euthanised and the foetuses examined. No significant differences for the treated groups were observed for developmental parameters (foetal body weight changes, number of survivors, bone development, skeletal ossification or liver nucleic acid) compared with controls. The offspring of the remaining animals were delivered on GD 22–23 and observed over one month. No significant differences between treated and control animals were observed (Letizia et. al., 2005).

In another study, the chemical was evaluated for potential oestrogen binding activity in ovariectomised Sprague-Dawley (SD) rats in an oestrogen receptor (ER) competitive binding assay. The uterine cytosol was incubated with radiolabelled beta-oestradiol and the chemical in increasing doses for 20 hours. The chemical at 1 mM did not bind to the ER and was considered inactive (Letizia et. al., 2005).

Risk Characterisation

Critical Health Effects

No critical health effects for risk characterisation were identified for the chemical. However, the chemical could possess hazardous properties such as skin and eye irritation when used at higher concentrations.

Public Risk Characterisation

The general public could be exposed through the skin or inhalation when using cosmetic and domestic products containing the chemicals. However, based on available international exposure information (see **Import, Manufacture and Use: International** section), the concentration in these products is not considered to be sufficiently high to cause adverse health effects. The estimated consumer dermal systemic exposure for cinnamic acid in cosmetic products is reported as 0.0005 mg/kg bw/day. For skin sensitisation, the calculated exposure concentration to cinnamic acid used in fine fragrances products is reported as 0.01 %, based on its use in consumer products at 20 % (Bickers et al., 2005). Therefore, the risk to public health is not considered to be unreasonable and further risk management is not considered necessary for public safety.

Occupational Risk Characterisation

During product formulation, dermal and ocular exposure of workers to the chemicals may occur, particularly where manual or open processes are used. These may include transfer and blending activities, quality control analysis, and cleaning and maintaining equipment. Worker exposure to the chemicals at lower concentrations may also occur while using formulated products containing the chemicals. The level and route of exposure will vary depending on the method of application and work practices employed.

At low concentrations, an unreasonable risk to workers is not expected. However, at high concentrations, the chemical may pose an unreasonable risk to workers unless adequate control measures to minimise dermal and ocular exposure are implemented. The chemical should be appropriately classified and labelled to ensure that a person conducting a business or undertaking (PCBU) at a workplace (such as an employer) has adequate information to determine the appropriate controls.

NICNAS Recommendation

The risk to workers and public from the use of the chemicals is not considered to be unreasonable. No recommendations or further assessment is required.

Regulatory Control

Work Health and Safety

The chemicals are not recommended for classification and labelling under the current adopted Globally Harmonised System of Classification and labelling of Chemicals (GHS). This report does not consider classification of physical hazards and environmental hazards.

Advice for consumers

Products containing the chemicals should be used according to the instructions on the label.

Advice for industry

Control measures

Control measures to minimise the risk from dermal and ocular exposure to the chemicals should be implemented in accordance with the hierarchy of controls. Approaches to minimise risk include substitution, isolation and engineering controls. Measures required to eliminate, or minimise risk arising from storing, handling and using a hazardous chemical depend on the physical form and the manner in which the chemicals are used. Examples of control measures that could minimise the risk include, but are not limited to:

- air monitoring to ensure control measures in place are working effectively and continue to do so;
- minimising manual processes and work tasks through automating processes;
- work procedures that minimise splashes and spills;
- regularly cleaning equipment and work areas; and
- using protective equipment that is designed, constructed, and operated to ensure that the worker does not come into contact with the chemicals.

Guidance on managing risks from hazardous chemicals are provided in the *Managing risks of hazardous chemicals in the workplace—Code of practice* available on the Safe Work Australia website.

Personal protective equipment should not solely be relied upon to control risk and should only be used when all other reasonably practicable control measures do not eliminate or sufficiently minimise risk. Guidance in selecting personal protective equipment can be obtained from Australian, Australian/New Zealand or other approved standards.

Obligations under workplace health and safety legislation

Information in this report should be taken into account to help meet obligations under workplace health and safety legislation as adopted by the relevant state or territory. This includes, but is not limited to:

- ensuring that hazardous chemicals are correctly classified and labelled;
- ensuring that (material) safety data sheets ((M)SDS) containing accurate information about the hazards (relating to both health hazards and physicochemical (physical) hazards) of the chemical are prepared; and

- managing risks arising from storing, handling and using a hazardous chemical.

Your work health and safety regulator should be contacted for information on the work health and safety laws in your jurisdiction.

Information on how to prepare an (M)SDS and how to label containers of hazardous chemicals are provided in relevant codes of practice such as the *Preparation of safety data sheets for hazardous chemicals—Code of practice* and *Labelling of workplace hazardous chemicals—Code of practice*, respectively. These codes of practice are available from the Safe Work Australia website.

A review of the physical hazards of these chemicals has not been undertaken as part of this assessment.

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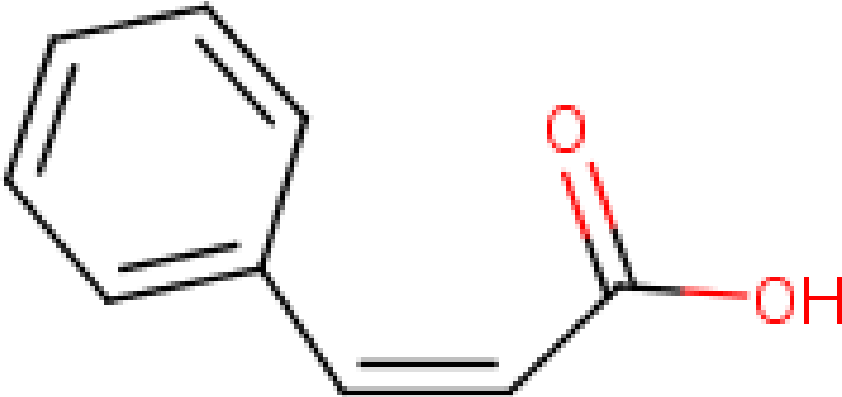
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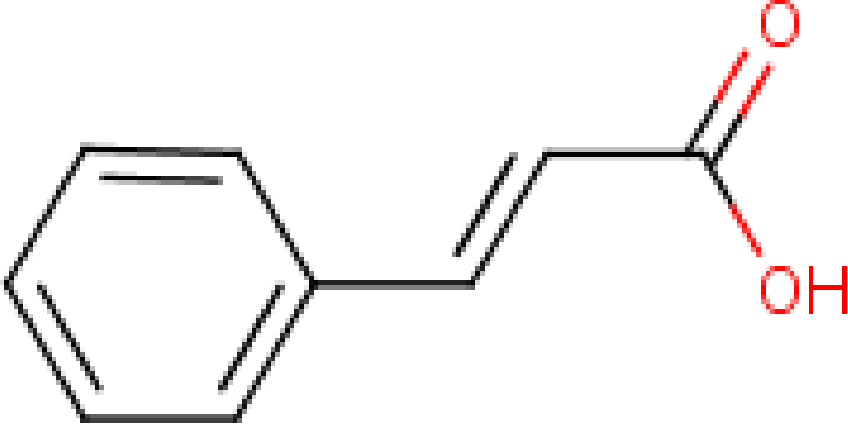
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Last Update 02 March 2018

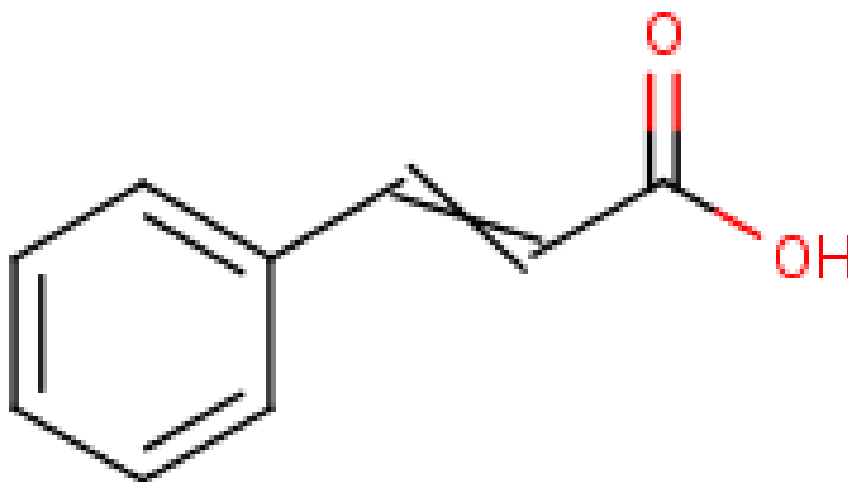
Chemical Identities

Chemical Name in the Inventory and Synonyms	2-Propenoic acid, 3-phenyl-, (Z)- allocinnamic acid cis-cinnamic acid cis-.beta.-carboxystyrene
CAS Number	102-94-3
Structural Formula	
Molecular Formula	C ₉ H ₈ O ₂
Molecular Weight	148.16

Chemical Name in the Inventory and Synonyms	2-Propenoic acid, 3-phenyl-, (E)- trans-cinnamic acid (E)-cinnamic acid trans-beta-carboxystyrene trans-3-phenylacrylic acid
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CAS Number	140-10-3
Structural Formula	
Molecular Formula	C9H8O2
Molecular Weight	148.16

Chemical Name in the Inventory and Synonyms	2-Propenoic acid, 3-phenyl- beta.-phenylacrylic acid cinnamic acid benzenepropenoic acid acidum cinnamylicum
CAS Number	621-82-9
Structural Formula	



Molecular Formula	C ₉ H ₈ O ₂
Molecular Weight	148.16

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