# Benzoic acid, 4-(1,1-dimethylethyl)-: Human health tier II assessment

**22 November 2013**

## CAS Number: 98-73-7



## 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.

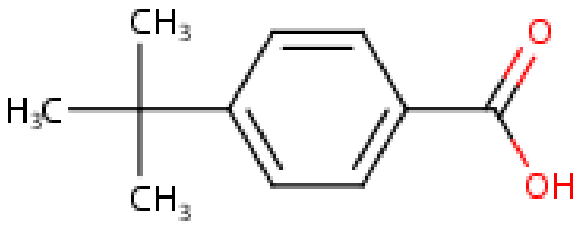
For more detail on this program please visit:www.nicnas.gov.au

**Disclaimer**



Acronyms & Abbreviations

## Chemical Identity

| | |
|---|---|
| Synonyms | p-tert-Butylbenzoic acid<br>4-tert-Butylbenzoic acid<br>PTBBA<br>Butylbenzoicacid<br>4-TBBA |
| Structural Formula |  |
| Molecular Formula | C11H14O2 |
| Molecular Weight (g/mol) | 178.23 |
| Appearance and Odour (where available) | White crystals to slightly yellow flakes. |
| SMILES | C(=O)(O)c1ccc(C(C)(C)C)cc1 |

# 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 European Union Registration, Evaluation and Authorisation of Chemicals (EU REACH) dossiers; EU Risk Assessment Report (EURAR), the Organisation for Economic Cooperation and Development Screening information data set International Assessment Report (OECD SIAR); Galleria Chemica; Substances and Preparations in the Nordic countries (SPIN) database; the European Commission Cosmetic Ingredients and Substances (CosIng) database; United States (US) Personal Care Product Council International Nomenclature of Cosmetic Ingredients (INCI) Dictionary; and eChemPortal: OECD High Production Volume chemical program—OECD HPV, the US Environmental Protection Agency's Aggregated Computer Toxicology Resource—ACToR, and the US National Library of Medicine's Hazardous Substances Data Bank—HSDB.

The chemicals are included in the CosIng database with masking and antimicrobial functions. However, there is currently no documented use of the chemical in cosmetic products in the United States (Personal Care Products Council, 2011).

The chemical has reported potential domestic use in varnishes, lacquers and paints. However, available European and North American databases do not give evidence for the use of PTBBA in consumer products, indicating the chemical is not likely to be widely available for domestic use.

The chemical has reported commercial use including:

- as an intermediate in organic synthesis;
- as an additive for  metalworking, cutting and lubricating oils;
- as a PVC stabiliser (salts of PTTBA); and
- as an alkyd resin modifier.

When PTTBA salts are used as PVC stabilisers, the p-tert-butylbenzoate ion is expected to remain unchanged in the polymer matrix. The PTTBA salt is reported to be <0.2 % of the total content (EURAR, 2009).

In alkyd resins, most of the PTBBA reacts with the hydroxyl groups. Less than 0.1 % PTBBA is reported to be left unreacted in the solution and in the hardened form of resin (EURAR, 2009).

Potential exposure to the chemical by personal contact with sex toys of polymeric materials as soft vinyl or thermoplastic rubber has been reported (EURAR, 2009).

# Restrictions

## Australian

No known restrictions have been identified.

## International

No known restrictions have been identified.

# Existing Work Health and Safety Controls

## Hazard Classification

The chemical is not listed on the Hazardous Substances Information System (HSIS) (Safe Work Australia).

## Exposure Standards

### Australian

No specific exposure standards are available.

### International

The following exposure standards are identified (Galleria Chemica):

- An occupational exposure limit (OEL) of 2 mg/m³ (inhalable dust) in different countries such as Netherlands, Germany and Switzerland.
- The short term exposure levels (STEL) of 4 mg/m³ (inhalable dust) in Germany and Switzerland.

# Health Hazard Information

## Toxicokinetics

There are no data available on the toxicokinetics, metabolism and distribution of the chemical after inhalation, oral and dermal exposure in animals or humans. However, based on the physical and chemical properties of PTBBA (molecular weight <200 g/mol and log Kow of 3.4) and effects observed in toxicological studies, extensive absorption and distribution can be assumed.

## Acute Toxicity

### Oral

The chemical had moderate acute toxicity in animal tests following oral exposure. A median lethal dose (LD50) of 550–800 mg/kg bw was reported in rats and mice. Observed sub-lethal effects included hypoactivity, ataxia, hypothermia to touch, respiratory congestion, loss of pain reflex, presence of yellow stains in the abdominal and rectal areas, impairment of forelimbs and hypospermatogenesis of the testes. In one study, the male rats displayed testicular atrophy following exposure to a single dose of 500 mg/kg bw (Hunter et al, 1965; EURAR, 2009).

### Dermal

The chemical was of low to moderate acute toxicity in animal tests following dermal exposure. The data are not sufficient to warrant a hazard classification.

Studies in New Zealand White rabbits reported a dermal LD50 of >900 mg/kg bw (30 % w/v solution) and >2000 mg/kg bw for the dry powder. No pathological lesions were observed in the latter study (EURAR, 2009; REACH). An LD50 of >2000 mg/kg bw was also reported for Wistar rats (REACH).

In another study, the LD50 value of the chemical (no data on purity), as a 30 % w/v solution in dimethyl sulfoxide (DMSO) was found to be approximately 300 mg/kg in Carworth Farm E strain (CFE) rats (REACH). However, this study is not suitable for classification purposes due to the likelihood of dermal penetration enhancement by the DMSO.

### Inhalation

In an acute inhalation study, Fischer 344 (F344) rats were exposed to PTBBA dust (purity 99.4 %) at concentrations of 0, 0.495 , 0.668, 0.958 or 1.802 mg/L for four hours. Results indicated that the LC50 is >1.802 mg dust/L, although major toxicological effects were observed at all doses. The lowest concentration of 0.495 mg/L induced changes in the testes, spinal cord and the rate of body weight gain. Moreover, testicular effects were also observed, which included reduction of sperm count and the number of tubular multinucleated cells, and presence of microscopic lesions. Other pathological signs noted were neuropathy of the forelimb and a decrease in the body weight gain in both male and females rats (EURAR, 2009).

## Corrosion / Irritation

### Skin Irritation

In a test performed according to the EU Test Guideline (TG) B4, no signs of skin irritation were observed after four hours of direct skin contact with 500 mg of the chemical (EURAR, 2009).

### Eye Irritation

In a study in New Zealand White rabbits, which was conducted in accordance with the EU TG B5, exposure to indiluted 100 mg PTBBA resulted only in mild eye irritation, which was reversible within three days (EURAR, 2009).

## Sensitisation

### Skin Sensitisation

The negative result observed for the chemical in a guinea pig maximisation test indicated that the chemical is not a skin sensitiser (EURAR, 2009).

## Repeated Dose Toxicity

### Oral

Considering the lowest observed adverse effect level (LOAEL) available from a 90-day rat study (6 mg/kg bw/d for male and 8 mg/kg for female rats), and based on the treatment-related effects reported, the chemical is considered to cause serious damage to health from repeated oral exposure. Target organs included the central nervous system, liver, kidneys, urinary tract and male reproductive system. Growth retardation was also observed.

In an oral 90-day study, albino Carworth Farm E (CFE) rats (10 animals/sex/group) were orally administered a diet containing doses of 0, 100, 316, 1000, 3160 and 10000 ppm of the chemical (calculated from food intake 0, 6, 21, and 75 mg/kg bw/d for males; 0, 8, 27, 89 mg/kg bw/d for females, for doses up to 1000 ppm; no calculation was available on the top two doses).

Mortality occurred at the top two doses. At 10000 ppm, 90 % of males died by day 34 and 100 % of females by day 53. Bilateral atrophy of the testes, particularly degenerated epithelium of seminiferous tubules, was found in males of all dose groups. Renal tubular necrosis and papillary necrosis were evident in treated male and female rats of all dose groups. At the top two doses, effects were also observed in the liver (sinusoidal congestion and fatty degeneration of centrilobular hepatocytes) and urinary tract (hydronephrosis, hydroureter, ureteral obstructions and haematuria). In addition, effects on the central nervous system (hind limb paralysis) were observed at the top doses. A no observed adverse effect level (NOAEL) could not be established in this study. The LOAEL was identified to be 6 mg/kg bw/d for males and 8 mg/kg for female rats (EURAR, 2009).

### Dermal

Considering the lowest observed adverse effect level (LOAEL) available from 28-day and 13-week rat studies (7.5–17.5 mg/kg bw/d), and based on the treatment-related effects reported, the chemical is considered to cause serious damage to health from repeated dermal exposure. Target organs included the liver, kidneys, peripheral blood and male reproductive system. Growth retardation was also observed (EURAR, 2009).

In a 13-week study, groups of 20 male and 20 female F344 rats were exposed to PTBBA topically (five days a week), with dosing solutions containing the diethanolamine (DEA) salt of the chemical. This resulted in daily exposures of 0, 17.5, 35, 70, or 140 mg/kg of the chemical. The LOAEL for this study was 17.5 mg/kg bw/day based on effects in the liver, kidney, testes and peripheral blood. Although the contribution of DEA (especially on the liver metabolism) cannot be ruled out, given the similarity of effects observed after repeated oral and inhalation exposure, effects observed are attributed to the chemical.

Dose-related significant increases in relative and absolute hepatic and renal weights were seen at all concentrations. Accompanying changes in clinical chemistry values suggested altered hepatic and renal functions. Histopathological changes in these organs including cytoplasmic vacuolation in the liver, and interstitial nephritis and papillary necrosis of the kidneys were detected at the top two doses. In these groups, signs of microcytic hypochromic anaemia were also observed. Liver cell vacuolation was also evident in female rats treated with 17.5 and 35 mg/kg of PTBBA.

Exposure of males to the two highest concentrations of PTBBA caused significantly reduced relative testis weights and sperm counts. Similarly, LDH-X enzyme activity, a measure of semen integrity, also showed decline in these PTBBA-treated groups. Histopathological changes including degeneration and regeneration of distal convoluted tubular epithelium, tubular casts, tubular degeneration with an absence of late spermatids, a reduced number of spermatogenic cell types, and giant cell formation were observed at the top two doses.

In another study, the chemical at various concentrations (0, 7.5, 15, 30 and 60 mg/kg bw) was topically applied on the shaved skin of CFE rats for 28 days. The histopathological evaluation revealed a level of germinal epithelium degeneration in male rats that received 60 mg/kg of the chemical. Dose-related significant increases in absolute and relative liver weights were seen in female rats of all dose groups and in male rats exposed to 15 mg/kg bw/d and above. The reported LOAEL in this study was 7.5 mg/kg bw/d, but was considered to be uncertain due to limited test parameters and insufficient numbers of animal subjects.

### Inhalation

Considering the lowest observed adverse effect concentration (LOAEC) available from 11–28 day rat studies (5–12.5 mg/m³), and based on the treatment-related effects reported, the chemical is considered to cause serious damage to health from repeated inhalation exposure. Target organs included the liver, kidneys, peripheral blood, central nervous system and male reproductive system. Growth retardation was also observed (EURAR, 2009).

In an 11-day inhalation study, groups of eight male and eight female F344 rats were repeatedly exposed to the chemical (dust) at achieved mean concentrations of 0, 12.5, 106 and 525 mg/m³ for six hours a day on four exposure days followed by three days (males) or four days (females) rest and another three-day period with exposure. The no observed adverse effect concentration (NOAEC) could not be established in this study. The LOAEC was 12.5 mg/m³ (six hours a day, seven exposure days) based on a dose-related reduction in the mean number of sperm in each testis and treatment-related lesions seen in the kidneys of rats from all dose groups.

Neurotoxicity was noted at concentrations of ≥ 106 mg/m³. Behavioural anomalies were observed in the exposed males, including limb paralysis, hunched posture, convulsions, hypoactivity, prolapsed penis and abnormal respiration. Neurodegenerative changes such as degeneration and loss of neurons, vacuolation, microgliosis and congestion were

localised in the central area of the grey matter lesions and in the ventral funiculi region of white matter. These animals also showed clinical signs of paraplegia.

Microscopically, treatment-related lesions were seen in the livers, spinal cord, testis, epididymides, and thymus of rats from mid and high dose groups. In animals exposed to 106 mg/m³ PTBBA, severe tubular changes were observed, consisting of absence of late spermatids, reduction in spermatogenic cell types, atrophy and inflammation of epididymides. Female rats exposed to high doses exhibited abnormalities in blood count. Significant loss of body weight was noted in both sexes.

In 28-day repeat dose inhalation toxicity study, three groups of Sprague Dawley (SD) rats of both sexes were exposed to PTBBA at low concentrations ranging from 1.5 to 15.7 mg/m³ (snout-only) for 6 h/day, 5days per week. The NOAEC for the chemical was reported to be 5 mg/m³ based on an increased incidence of body tremor in males exposed to 15.7 mg/m³. Liver weights were also significantly increased in females exposed at this dose.

Although the above studies have weaknesses in relation to OECD TG for repeated inhalation, the data provided valid information on the toxicity of the chemical and the identified targets were consistent with the results reported by other authors.

***Overall***

The target organs for repeat-dose toxicity of the chemical were the central nervous system, liver, kidneys, testes, epididymides, haematopoietic system and the thymus. Similar lesions in the liver, kidney, male reproductive organs and peripheral blood were identified across all studies regardless of the route of exposure. Neurotoxicity was observed after repeated inhalation and oral administration. Based on the data, the chemical is considered to meet the criteria to be classified as T (toxic) for all routes. This is supported by the opinion of the Committee for Risk Assessment (RAC) proposing harmonised classification and labelling of the chemical at a community level (ECHA, 2011).

## Genotoxicity

The chemical did not induce mutagenic effect in *Salmonella typhimurium* strains, but was weakly positive in an in vitro micronucleus test in Chinese Hamster V79 cells.

In an in vivo chromosomal aberration test, which was conducted in accordance to OECD TG 475, rats were tested using single oral gavage doses of 600 mg/kg bw for males and 300 mg/kg bw for females. The results were negative. Whilst the results of this study support that the clastogenic potential of the chemical observed in vitro is unlikely to be expressed in germ cells in vivo, there is an uncertainty regarding the potential for local clastogenic effects and chromosomal abberation (EURAR, 2009).

## Carcinogenicity

No data are available on the carcinogenicity of the chemical.

## Reproductive and Developmental Toxicity

The data available provide clear evidence of an adverse effect on male fertility and support classification (see **Recommendation** section). Classification is supported by the opinion of the RAC which proposed harmonised classification and labelling of the chemical (as above) at a community level (ECHA, 2011).

In a fertility study using Wistar rats, males were fed diets containing 0, 20,100 or 500 ppm PTBBA for a 70-day period before mating trials. Successful impregnation of at least one of the two females was used as fertility indicator. Unsuccessful males from the first mating trial were kept for another 70 days, but without exposure to PTBBA. These males were mated again (second mating trial) to two non-exposed female virgins for one week. The results showed that, in the first mating trial, the lowest levels of dietary PTBBA (20 ppm) did not cause fertility impairment. However, one male from the 100 ppm group was not successful in impregnating and no pregnancies occurred in the 500 ppm dosing group. The impairment was reversed in the second mating trial, although minor lesions were found in the germinal epithelium and were confined to a few tubules only at 500 ppm. A NOAEL for male fertility of 20 ppm (1.6 mg/kg/bw) was reported from this study.

In addition, repeated exposure by oral, skin and inhalation routes, and acute inhalation exposure has been shown to induce testicular abnormalities. The effects were primarily characterised by the degeneration of germinal epithelium resulting in disrupted spermatogenesis, damage in the epididymides, including atrophy and inflammation. The reported effect levels for testis toxicity were 6 mg/kg bw/day (oral LOAEL), 30 mg/kg bw/day (dermal NOAEL) and 12.5 mg/m³ inhalation LOAEC (see **Acute toxicity** and **Repeated dose toxicity** sections**)**.

The potential testicular effects of occupational exposure of PTBBA in humans were investigated in a cohort of 90 male workers in a PTBBA production facility in the early 1980. However, the results of the study were inconclusive due to the small sample size (Whorton et al, 1981).

### Other Health Effects

Neurotoxicity

Results from animal studies have indicated neurotoxic effects of PTBBA exposure following oral and inhalation exposure (see **Repeated dose toxicity** section).

## Risk Characterisation

### Critical Health Effects

The critical health effects for risk characterisation include systemic long-term effects

(reproductive toxicity and serious damage to health following repeated exposure). Similar effects are observed regardless of the route of exposure. The chemical may also cause harmful effects following acute exposure.

### Public Risk Characterisation

Given the uses identified for the chemical, public exposure to the chemical is expected to be minimal. Although the use of this chemical as a direct ingredient in cosmetic and domestic products in Australia is not known, international information indicates that it is not likely to be widely available for domestic and cosmetic use.

The chemical is used in the manufacture of consumer products and exposure may occur due to migration from articles, particularly when used as a stabiliser. Data indicate that residual traces of the chemical would only be present in minimal amounts (EURAR, 2009). The margin of exposure for dermal exposure of humans due to migration from sex toys was considered to be sufficient in a risk assessment conducted internationally for the chemical (EU RAR 2009). The scenario used, and therefore the estimated margins of exposure, are considered applicable in the Australian context, indicating that the presence of the chemical in sex toys does not pose an unreasonable risk to the public.

However, considering the potential for serious health effects at relatively low levels of exposure (regardless of route), a Tier III assessment should be undertaken to determine if the chemical is used in cosmetic and domestic products in Australia and, if used, to quantify the risk. In order to quantify the risk, detailed exposure information from industry is required.

### Occupational Risk Characterisation

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

Given the critical systemic long-term health effects, the chemical may pose an unreasonable risk to workers unless adequate control measures to minimise dermal, ocular and inhalation exposure to the chemical 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 appropriate controls.

Current use of the chemical is not known in Australia. However, a risk assessment conducted internationally for the chemical (EURAR, 2009) concluded that, for two occupational exposure scenarios: (1) production and further processing of PTBBA (2) production of alkyd resins in the polymers industry, 'there is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account' (EURAR, 2009).

The data available support an amendment to the hazard classification in HSIS (refer to **Recommendation section**).

# NICNAS Recommendation

Safe Work Australia should consider whether current controls are adequate to minimise the risk to workers. A Tier III assessment may be necessary to provide further information as to whether the current exposure controls are appropriate to offer adequate protection to workers. In addition, the chemical is recommended for Tier III assessment to determine whether the chemical is used in cosmetic and domestic products in Australia. If the chemical is used in this country, a quantitative risk assessment should be undertaken to characterise the risk.

All other risks are considered to have been sufficiently assessed at the Tier II level, provided that the recommended amendment to the classification is adopted, and labelling and all other requirements are met under workplace health and safety and poisons legislation as adopted by the relevant state or territory.

## Regulatory Control

### Public Health

The need for further regulatory control for public health will be determined as part of the Tier III assessment.

### Work Health and Safety

The chemical is recommended for classification and labelling under the current approved criteria and adopted GHS as below. This assessment does not consider classification of physical hazards and environmental hazards.

| Hazard | Approved Criteria (HSIS)[a] | GHS Classification (HCIS)[b] |
|---|---|---|
| Acute Toxicity | Harmful if swallowed (Xn; R22) | Harmful if swallowed - Cat. 4 (H302) |
| Repeat Dose Toxicity | Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed (T; R48/23/24/25) | Causes damage to organs through prolonged or repeated exposure - Cat. 1 (H372) |
| Reproductive and Developmental Toxicity | Repro. Cat 2 - May impair fertility (T; R60) | May damage fertility - Cat. 1B (H360F) |

[a] Approved Criteria for Classifying Hazardous Substances [NOHSC:1008(2004)].

[b] Globally Harmonized System of Classification and Labelling of Chemicals (GHS) United Nations, 2009. Third Edition.

* Existing Hazard Classification. No change recommended to this classification

## Advice for industry

***Control measures***

Control measures to minimise the risk from skin, eye and inhalation exposure to the chemical 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 storage, handling and use of a hazardous chemical are dependent on the physical form and the manner in which the chemical is used. Examples of control measures which may minimise the risk include, but are not limited to:

- use of closed systems or isolation of operations;
- use of local exhaust ventilation to prevent the chemical from entering the breathing zone of any worker;
- health monitoring for any worker who is at risk of exposure to the chemical if valid techniques are available to monitor the effect on the worker's health;
- air monitoring to ensure control measures in place are working effectively and continue to do so;
- minimisation of manual processes and work tasks through automation of processes;
- work procedures that minimise splashes and spills;
- regular cleaning of equipment and work areas; and
- use of protective equipment that is designed, constructed, and operated to ensure that, the worker does not come into contact with the chemical.

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 be relied upon on its own to control risk and should only be used when all other reasonably practicable control measures do not eliminate or sufficiently minimise risk. Guidance in selection of 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 assist with meeting 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 hazardous chemical are prepared; and
- management of risks arising from storage, handling and use of 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 physical hazards of the chemical has not been undertaken as part of this assessment.

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Last update 22 November 2013

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