Australian Government
Department of Health and Aged Care
Australian Industrial Chemicals Introduction Scheme

Phenol, 4,4'-(1-methylethylidene)bis- (Bisphenol A)

Evaluation statement

1 October 2024

Draft

Table of contents

Contents

AICIS evaluation statement

Subject of the evaluation

Phenol, 4,4'-(1-methylethylidene)bis- (Bisphenol A)

Chemical in this evaluation

CAS name	CAS number
Phenol, 4,4'-(1-methylethylidene)bis-	80-05-7

Reason for the evaluation

New information is available about human health risks.

Parameters of evaluation

The chemical is listed on the Australian Inventory of Industrial Chemicals (the Inventory). It was previously assessed under the Inventory Multi-tiered Assessment and Prioritisation (IMAP) framework, under the former National Industrial Chemicals Introduction and Assessment Scheme (NICNAS). This evaluation statement should be read in conjunction with the IMAP assessment for bisphenol A (NICNAS 2016).

This evaluation will:

- review the weight of evidence on reproductive toxicity including new information
- consider whether amendments to the classification for reproductive toxicity are warranted.

Summary of evaluation

Summary of introduction, use and end use

The chemical has reported domestic use in Australia in adhesives (binding agents) and site-limited uses in stabilisers with an introduction volume between 100 and 1000 tonnes (NICNAS 2016).

Based on international use information, bisphenol A primarily has site limited use in the manufacture of plastic and polymer products, including use as a monomer in the production of some food contact plastics (epoxy and polycarbonates). The chemical is reported to be used in heat sensitive paper, for example, credit card receipts. It is also used in flame retardants, rubber chemicals and stabilisers (NICNAS 2016).

It has commercial use in:

- adhesives and binding agents
- anti-static agents

- cleaning and washing agents
- construction materials
- corrosion inhibitors
- fillers
- hydraulic fluids and additives
- insulating materials
- lubricants and additives
- paints, lacquers and varnishes
- process regulators
- reprographic agents
- softeners
- surface treatment
- surface-active agents
- viscosity adjustors (NICNAS 2016).

Human health

Summary of health hazards

The critical health effects for risk characterisation include systemic long term effects of reproductive toxicity and general toxicity (liver and kidney effects), and local effects of skin sensitisation and eye damage and respiratory irritation. This evaluation focuses on reproductive toxicity for which new studies are available.

Multi-generational studies in rats and mice reported adverse effects at high doses (600 mg/kg bw/day), including reduction in number of litters and litter size, fewer live pups born and effects on reproductive organs. These effects were supported by non-guideline studies in animals. These effects occurred in the absence of marked maternal toxicity. Currently the evidence is not sufficient to infer a causal link between BPA exposure and reproductive and developmental effects in humans.

Hazard classifications relevant for worker health and safety

The chemical satisfies the criteria for classification according to the Globally Harmonized System of Classification and Labelling of Chemicals (GHS) (UNECE 2017) for hazard classes relevant for worker health and safety as follows. This evaluation does not consider classification of physical hazards and environmental hazards. Apart from the amended reproductive toxicity classification, this is the current classification in the Hazardous Chemicals Information System (HCIS).

Health hazards	Hazard category	Hazard statement
Serious eye damage	Eye Damage 1	H318: Causes serious eye damage
Skin sensitisation	Skin Sens. 1	H317: May cause an allergic skin reaction
Reproductive toxicity	Repr. 1B	H360F: May damage fertility
Specific target organ toxicity (single exposure)	STOT Single Exp. 3	H335: May cause respiratory irritation

Summary of health risk

Workers

During product formulation, oral, dermal, inhalation and ocular exposure may occur, particularly where manual or open processes are used. These could include transfer and blending activities, quality control analysis, and cleaning and maintaining equipment. Worker exposure to the chemical at lower concentrations could 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 and local health effects, the chemical could pose an unreasonable risk to workers. Control measures to minimise oral, dermal, ocular and inhalation exposure are needed to manage the risk to workers (see **Proposed means for managing risk** section).

Proposed means for managing risk

Workers

Recommendation to Safe Work Australia

It is recommended that Safe Work Australia (SWA) update the HCIS to include classifications relevant to work health and safety.

Information relating to safe introduction and use

The information in this statement including recommended hazard classifications, should be used by a person conducting a business or undertaking at a workplace (such as an employer) to determine the appropriate controls under the relevant jurisdiction Work Health and Safety laws.

Control measures that could be implemented to manage the risk arising from oral, dermal, ocular and inhalation exposure to this chemical includes, but is not limited to:

- using closed systems or isolating operations
- using local exhaust ventilation to prevent the chemical from entering the breathing zone of any worker
- minimising manual processes and work tasks through automating processes
- adopting work procedures that minimise splashes and spills
- cleaning equipment and work areas regularly
- using protective equipment that is designed, constructed, and operated to ensure that the worker does not come into contact with the chemical.

Measures required to eliminate or manage risk arising from storing, handling and using this hazardous chemical depends on the physical form and how this chemical is used.

These control measures may need to be supplemented with conducting health monitoring for any worker who is at significant risk of exposure to the chemical, if valid techniques are available to monitor the effect on the worker's health.

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.

Model codes of practice, available from the Safe Work Australia website, provide information on how to manage the risks of hazardous chemicals in the workplace, prepare an SDS and label containers of hazardous chemicals. Your Work Health and Safety regulator should be contacted for information on Work Health and Safety laws and relevant Codes of Practice in your jurisdiction.

Conclusions

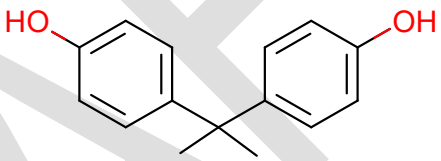
The Executive Director proposes to be satisfied that the identified risks to human health from the introduction and use of the industrial chemical can be managed.

Note:

1. Obligations to report additional information about hazards under *Section 100* of the *Industrial Chemicals Act 2019* apply.
2. You should be aware of your obligations under environmental, workplace health and safety and poisons legislation as adopted by the relevant state or territory

Supporting information

Chemical identity

CAS number	80-05-7
CAS name	Phenol, 4,4'-(1-methylethylidene)bis-
Molecular formula	$C_{15}H_{16}O_2$
Associated names	Bisphenol A (BPA) 2,2-Bis(4-hydroxyphenyl)propane 4,4'-(1-Methylethylidene)bisphenol
Molecular weight (g/mol)	228.29
SMILES (canonical)	OC1=CC=C(C=C1)C(C2=CC=C(O)C=C2)(C)C
Structural formula	

Relevant physical and chemical properties

The following properties were retrieved from Galleria Chemica and SciFinder (Chemwatch n.d.; CAS n.d.).

Physical form	White solid
Melting point	155°C
Boiling point	250–252°C
Vapour pressure	4.12E-09 hPa at 25°C
Water solubility	Moderately soluble (300 mg/L at 25°C)
pK_a	10.29±0.10, most acidic temp: 25°C
log K_{ow}	3.4 at 21.5 °C and pH 6.4

Existing Australian regulatory controls

AICIS

No specific controls are currently available for the chemical.

Workers

The chemical is listed on the HCIS (Safe Work Australia, SWA) with the following hazard category and statements for human health:

Health hazards	Hazard category	Hazard statement
Serious eye damage	Eye Damage 1	H318: Causes serious eye damage
Skin sensitisation	Skin Sens. 1	H317: May cause an allergic skin reaction
Reproductive toxicity	Repr. 2	H361f: Suspected of damaging fertility
Specific target organ toxicity (single exposure)	STOT Single Exp. 3	H335: May cause respiratory irritation

From 1 December 2026 and following implementation into the work health and safety laws of the Commonwealth, states and territories, new Workplace exposure limits (WEL) for airborne contaminants (WEL list) will be adopted throughout Australia. The WEL for the chemical is 2 mg/m^3 time weighted average (TWA) with a notation of DSEN (SWA 2024.

International regulatory status

Exposure standards

The following exposure standards are identified (Chemwatch n.d.):

Bisphenol A (CAS No. 80-05-7) has an exposure limit of 2–10 mg/m^3 TWA in countries such as Austria, China, Finland, Ireland, the Netherlands, Norway, Poland, Russia, Thailand, and the United Kingdom.

The chemical also has a short-term exposure limit (STEL) of 4–5 mg/m^3 in countries such as Austria, the Czech Republic and Denmark.

European Union

The European Commission prohibited placing thermal paper on the market if it contains bisphenol A equal to or greater than 0.02% by weight after 2 January 2020 (EC 2016).

The chemical is listed on the candidate list of Substances of Very High Concern (SVHC) for eventual inclusion in Annex XIV (ECHA 2017). The reason for inclusion is its toxicity for reproduction (Article 57c). In the EU, companies could have legal obligations if the chemical that they produce, supply, or use is included on the candidate list whether it is used on its own, in mixtures, or present in articles.

Health hazard information

Toxicokinetics

In humans, orally administered BPA is well absorbed and undergoes complete first-pass metabolism in the liver to BPA-glucuronide as major metabolite, which is rapidly excreted in the urine, with a half life of less than 6 hours (EFSA 2008). In humans, non-human primates and rodents, limited sulphate conjugation can also occur. Comparison of oral and intravenous toxicokinetic data indicated that the available unconjugated bisphenol A in adults is 2.8% in rats, 0.45% in mice and 0.9% in monkeys. Dermal absorption is low; available experimental evidence indicates a 24 hour percutaneous penetration of bisphenol A across human skin of 2.3–8.6% (NICNAS 2016).

Reproductive and development toxicity

The chemical is classified as 'Reproductive toxicity – Category 2 (H361f): 'Suspected of damaging fertility' in the HCIS (SWA n.d.). Consideration of the following evidence supports amending this classification to the hazard category 'Reproductive toxicity – Category 1B' and the hazard statement 'H360F – May cause adverse effects on fertility':

- adverse effects on fertility in multi-generational studies in rats and mice occurring at medium or high doses
- supporting results from non-guideline studies that found effects on female reproductive capacity, sperm parameters and male and female reproductive organs
- effects were not co-occurring with marked systemic toxicity.

Based on the evidence and given the uncertainties associated with effects on developmental toxicity (see EFSA comments below), this evaluation is focused on reproductive toxicity (effects on fertility and sexual function).

In the 2015 European Food Safety Authority (EFSA) opinion (EFSA 2015), the (Scientific Panel on Food Contact Materials (CEF) concluded that the evidence was not sufficient to infer a causal link between BPA exposure and reproductive and developmental effects in humans, although in experimental animal studies BPA was a reproductive toxicant at high doses (above a human equivalent dose factor (HED) of 3.6 mg/kg bw/day. In the 2023 evaluation (EFSA 2023) new data from animal and human studies were reviewed. The likelihood of a health effect in the overall body of evidence was evaluated and classified as:

- 'Very Likely: There is very high confidence in the body of evidence for an association between exposure to the substance and health effect/s (e.g. there is much evidence showing consistent effect/s).
- Likely: There is high confidence in the body of evidence for an association between exposure to the substance and health effect/s (e.g. there is evidence showing consistent effects).
- As Likely As Not (ALAN): There is low confidence in the body of evidence for an association between exposure to the substance and health effect/s (e.g. there is evidence showing inconsistent effects).
- Not Likely: There is very low confidence in the body of evidence for an association between exposure to the substance and health effect/s (e.g. there is evidence showing consistent no effects).
- Inadequate evidence: There is insufficient evidence available to assess if the exposure to the substance is associated with and health effect/s or data are missing.'

In the animal studies, the likelihood of reproductive effects was assessed by weight of evidence (WoE) in three clusters. In the female reproductive toxicity cluster, the following were concluded to be **Likely** effects on:

- ovary weight and histology after developmental exposure
- ovary histology after developmental and adult exposure
- implantation rate after growth phase/young age exposure
- follicle counts after adult exposure.

In the male reproductive toxicity cluster, the following effects were concluded to be **Likely** effects on:

- epididymis histology (exfoliated germ cells and inflammation) after developmental and adult exposure
- testes histology (increased seminiferous tubules with lumen and acrosomal vesicles) after growth phase/young age exposure
- sperm motility, morphology, viability and acrosome reaction after adult exposure.

In the developmental toxicity cluster, although effects were also noted, the results were less consistent. These effects were judged as **As Likely As Not**.

Multi-generational studies – mice

In a continuous breeding study, CD-1 mice (20/group/sex and 40/sex controls) were administered the chemical in feed at 0, 0.25, 0.5 or 1.0% (daily intakes of BPA were estimated to be 0, 300 600 or 1200, and 0, 325, 650 or 1300 for males and females, respectively). The work included 4 components:

- a dose range finding study (results not discussed)
- a continuous breeding phase
- cross over mating
- offspring assessment and assessment of reproductive capacity.

A 1 week premating period was followed by a 14 week mating period (**the continuous breeding phase**) when cohabiting pairs were allowed to breed. Reproductive performance was monitored by recording:

- the total number of F1 litters produced in this period per breeding pair
- for each litter–the litter size, number of live pups and sex ratio on the day of birth.

The pups were discarded immediately and the couple allowed to breed again for the rest of the 14 week period. The litters born after the cohabiting period remained with their mothers until weaning on PND21 (postnatal day) and were used in the final phase assessing offspring and reproductive capacity.

Twenty F0 males and 20 F0 females from the top dose group were then mated with 20 control females and 20 control males, respectively (the cross over mating phase) to determine whether one sex was more affected following exposure to the chemical. Treatment with BPA was discontinued in the diet during this 7 day cohabitation period and resumed for 21 days after the breeding pairs were separated. The same reproductive assessment was conducted as described for the continuous breeding phase (including litter size, number of live pups and sex ratio on the day of birth). Parental animals were sacrificed within 1 week of the delivery of the pups.

A maximum of 20 male and 20 female F1 generation offspring (from the final litters of the control and high dose groups in the continuous breeding phase) were retained after weaning for assessment of their reproductive capacity. After rearing the rats to sexual maturity, each F1 female was paired with a F1 male from the same dose group (control or high dose) for 7 days. The resulting litters were evaluated and discarded on the day of birth as described for the litters produced during the F0 generation cohabitation phase (and cross over mating phase).

For all control and high dose F0 and all reared F1 animals, liver, kidneys, adrenal glands and reproductive organs were weighed and subjected to histopathological examination. In males, sperm analysis (concentration, motility and morphology) was undertaken, and effects on the oestrous cycle assessed in females.

The following results were reported for the continuous breeding phase, crossover phase and assessment of the reproductive capacity of F1 animals:

Continuous Breeding Phase

- A statistically significant decrease compared to controls was observed in the number of litters produced per pair, litter size and the number of live pups per litter in the high and mid-dose group. The litter size reductions occurred across all matings and the magnitude of all these decreases were dose related.
- A statistically significant decrease in live pup weight on PND 0 was observed in females at the top dose after adjustment for litter size, including live and still births.
- General toxicity was observed as:
    - A statistically significant decrease in maternal body weight was observed after each litter on postnatal day (PND) 0 at the top dose in the continuous breeding phase.
    - At necropsy of the F0 generation (controls and top dose group only), treatment related effects were seen at the highest dose level; for both sexes relative liver weight was significantly increased and relative combined kidney/adrenal weight was significantly increased compared to controls. Similar effects were observed in the F1 generation.

Crossover Phase

- At study termination for the crossover phase, a small but statistically significant decrease in **body weight** was observed in treated females.
- A statistically significant decrease in **litter size** and **number of live pups** per litter were observed. More significant effects were noted in exposed females.

Offspring assessment and assessment of reproductive capacity

- Deaths among F1 generation were observed during lactation (day 0-21) and post weaning (day 21–74) 6%, 4%, 14% and 38% animals up to day 74 in the control, low dose, mid dose and high dose groups, respectively.
- No effects on fertility index, litter size or number of live pups were reported. However, at the top dose there were only 8 litters that had at least one male and one female for the mating phase. Therefore, there were only 11 breeding pairs at the top dose compared to 19–20 breeding pairs in the control, low dose and mid dose groups.

Histological examination was conducted on all F1 animals, and the only effects observed were toxicity to the liver and kidney at all doses. The lowest observed adverse effects level for reproductive toxicity was 600 mg/kg bw/day based on adverse effects on fertility (ECHA 2014a; ECHA 2014b).

In a two generation GLP compliant study conducted in accordance with OECD TG 416, CD-1 mice (28/sex/dose) were administered the chemical (purity 99.7%) by gavage at 0, 0.018, 0.18, 1.8, 30, 300 or 3500 ppm (equivalent to approximately 0, 0.003, 0.03, 0.3, 5, 50 or 600 mg/kg bw/day). The positive control group was exposed to 17β-oestradiol, and the negative control group received the vehicle only. Mice were exposed to the chemical 8 weeks prior to mating, and then from conception to adulthood (F1)(chronic exposure). In females, most of the reproductive parameters (i.e. reproductive organ weights, ovarian primordial follicles count, histopathology of ovaries and uterus, mating and fertility indices, litter size at birth, sex ratio, percent of post-implantation loss) were unaffected by the treatment. Effects of BPA on reproduction and the offspring were only observed at the highest dose (600 mg/kg bw/day). The following observations were reported:

- In females, the vaginal patency was significantly accelerated at 600 mg/kg bw/day when adjusted to the body weight on PND21.
- F0 treated females were twice more frequently in oestrus compared to controls.
- The length of the gestation period was significantly increased by 0.3 days in F0 and F1 generations.
- The body weight of the F1 pups was significantly lower during lactation.
- Epididymal sperm concentration was significantly decreased at 600 mg/kg bw/day in F0 males.
- In F1 males, there was a significant reduction in anogenital distance (AGD) on PND21 when adjusted to the body weight when exposed to 50 or 600 mg/kg bw/day.
- In F1 and F2 males exposed to 600 mg/kg bw/day, testes weight was significantly reduced with histopathology findings including a significantly increased incidence of minimal to mild hypoplasia of the seminiferous tubules.
- The incidence of undescended testes was significantly increased in F1 and F2 weanlings exposed to 600 mg/kg bw/day.
- General toxicity included significantly increased kidney and liver weight as well as reduced body weight gain in F1 mice.

The LOAEL for reproductive toxicity was 50 mg/kg bw/day based on effects on the reduction in the AGD (ECHA 2014a; ECHA 2014b; REACH n.d.).

Multi-generational studies – rats

In a 3 generation GLP compliant reproductive toxicity study conducted in accordance with U.S. EPA guidelines (U.S EPA OPPTS 837.3800, 1998), CD Sprague Dawley (SD) rats (30/sex/dose) were administered BPA (purity at 99.5%) in the diet at 0, 0.015, 0.3, 4.5, 75, 750 or 7500 ppm corresponding to approximately 0, 0.001, 0.02, 0.3, 5, 50 and 500 mg/kg bw/day. The animals were exposed for 10 weeks before mating, and this continued for males through a 2 week mating period and for an additional 3 weeks after mating. Females were exposed from conception through to gestation and lactation. Males and females from thesame group were mated together and 3 generations of males and females were then studied. For each generation, 30 weanling animals per sex and per dose were selected in order to become the parents of the next generation, and 3 animals per sex and per litter were necropsied and underwent further analysis.

The following observations were reported:

- There was a significant reduction in the average number of live pups per litter at 500 mg/kg bw/day in all generations on PND0. The decrease was reported without a statistically significant effect on post-implantation loss or on the number of dead pups per litter.
- The absolute and relative paired ovary weights were significantly decreased in adult F1, F2 and F3 females at 500 mg/kg bw/day.
- In the F1, F2 and F3 offspring, the body weight was significantly lower per litter (12-27%) at 500 mg/kg bw/day in all animals on PND7–21.
- The AGD was significantly increased in F2 females (measured only in F2 and F3 offspring) on PND0 in groups exposed to 0.001, 0.02, 0.3 and 50 mg/kg bw/day.
- The onset of puberty (evaluated as the age of vaginal patency) was significantly delayed at 500 mg/kg bw/day in F1, F2 and F3 females.
- In males, preputial separation was significantly delayed in F1, F2 and F3 generations.
- General toxicity included a significant reduction in body weight gain in all exposed generations and kidney effects (renal tubular degeneration) in females (not in F3) at 500 mg/kg bw/day.

The LOAEL for reproductive toxicity was 500 mg/kg bw/day based on effects on the offspring body weight, ovary weights and number of pups born (ECHA 2014a; REACH n.d).

In a two generation reproduction toxicity study, Crj;CD (SD) IGS rats (25/sex/group) were orally administered the chemical at 0, 0.2, 2, 20 and 200 µg/kg bw/day by gavage for 2 generations. The study protocol was similar to the OECD TG 416 but females were treated for only 2 weeks before mating whereas males were exposed during a 10 week premating period. No effects were observed apart from a decrease in the absolute and relative weight of seminal vesicles in F2 males at the lowest dose. No general toxicity or effects on fertility were reported in this study (ECHA 2014a; REACH n.d.).

Other supporting animal studies

In a GLP compliant oral gavage study investigating the toxicologic potential of BPA following perinatal only (gestation day (GD) 6–PND 21) or chronic exposure (GD6–1 or 2 years) in SD rats, the following treatment related effects, only at the highest dose (at 25000 µg BPA/kg bw/day), were reported:

- lesions in the epididymis (exfoliated germ cells and lymphocyte cellular infiltration)
- hyperplasia in the pars distalis of the pituitary gland (males)
- increased vaginal epithelial hyperplasia
- lesions in the uterus (cystic endometrial hyperplasia, squamous metaplasia, apoptosis of the endometrial luminal epithelial cells)
- increase in follicular cysts (also observed at 2500 µg BPA/kg bw/day)
- no significant effects on sperm parameters or testicular histopathology in the BPA dose groups
- no adverse effects on the oestrous cycle following BPA treatment (REACH n.d.).

In several reproductive studies described in the ECHA Committee of Risk Assessment and the CLH Report (ECHA 2014a; ECHA 2014b), various effects of BPA have been reported on female reproductive tract morphology and function and on fertility including:

- reduced ovarian weight
- increase in ovarian follicular cysts
- depletion of corpora lutea

- benign lesion like endometrial hyperplasia
- adverse effect of BPA on the oestrous cycle, including irregular and prolonged cycles

A significant proportion of non-guideline studies reported effects on male sexual parameters (effects on sperm, hormone levels or sexual function) and histopathogical changes in the testes and epididymis (ECHA 2014a, EFSA 2023).

Mechanisms of action for the identified BPA reproductive toxicity endpoints have been non-systematically explored in the literature. They include oestrogen and androgen receptor (AR) interactions and associated adult and indirect (germline) exposure (EFSA 2023). The ECHA RAC opinion concluded that the mode of action for disruption of the reproductive tract may be caused by a direct and indirect disruption of hypothalamic–pituitary–gonadal (HPG) axis or by direct organ specific toxicity and is considered relevant to humans (ECHA 2024a).

Human studies

A number of epidemiological studies investigated the potential effects of the chemical on sexual function and fertility in humans. The findings suggested that BPA could become systemically available and may have an effect on fertility in men and women (ECHA 2014a).

EFSA recently reviewed the human data relating to female reproductive toxicity, male reproductive toxicity and developmental toxicity. The likelihood of a health effect in the overall body of evidence was evaluated. No effects were judged as **Likely** or **Very Likely**. An association between maternal BPA exposure and impaired pre- and post-natal growth, shorter duration of gestation or preterm delivery, reduced male fertility and pubertal development when exposed during childhood, was judged as **Not Likely** (EFSA, 2023).

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