

HEALTH ASSESSMENT: NAPHTHALENE AND PARA- DICHLOROBENZENE IN MOTHBALLS

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ABBREVIATIONS

ABG	Arterial blood gas
ATSDR	Agency for Toxic Substances and Disease Registry
BMD	Benchmark dose
bw	Body weight
CAS RN	Chemical abstract service registry number
ECHA	European Chemicals Agency
ED	Emergency department
EFSA	European Food Safety Authority
EU	European Union
GSH	Glutathione
G6PD	Glucose-6-phosphate dehydrogenase
Hb	Haemoglobin
HBGV	Health-based guidance value
HEC	Human equivalent concentration
HNZ	Health New Zealand – Te Whatu Ora
IARC	International Agency for Research on Cancer
LC ₅₀	Median lethal concentration (causes death in 50% of animals)
LD ₅₀	Median lethal dose (causes death in 50% of animals)
LOAEL	Lowest observed adverse effect level
MetHb	Methaemoglobin
NICNAS	National Industrial Chemicals Notification and Assessment Scheme
NOAEL	No observed adverse effect level

NPC	National Poisons Centre
NZ EPA	New Zealand Environmental Protection Authority
PAH	Polycyclic aromatic hydrocarbon
pCO ₂	Partial pressure of carbon dioxide
PDCB	Para-dichlorobenzene
pO ₂	Partial pressure of oxygen
POD	Point of departure
PTMI	Provisional tolerable monthly intake
PTWI	Provisional tolerable weekly intake
RBC	Red blood cell
REACH	Registration, Evaluation, Authorisation and Restriction of Chemicals
RfC	Reference concentration
RfD	Reference dose
SUSMP	Standard for the Uniform Scheduling of Medicines and Poisons
TDI	Tolerable daily intake
TWI	Tolerable weekly intake
UF	Uncertainty factor
USA	United States of America
US EPA	United States Environmental Protection Agency
US FDA	United States Food and Drug Administration
WBC	White blood cell
WHO	World Health Organization

1. EXECUTIVE SUMMARY

The purpose of this report is to summarise adverse health effects from acute oral exposure to mothballs containing naphthalene or para-dichlorobenzene (PDCB) in children and toddlers. This report will only consider domestic, non-occupational and accidental oral exposure to mothballs.

Mothballs have been widely used as a domestic control method against adult clothes moths responsible for fabric damage in long-stored garments. Naphthalene and PDCB are the most common ingredients found in mothballs. These have now been replaced with safer alternatives such as red cedar blocks, lavender sachets, or pheromone moth traps.

Despite the ban of naphthalene and PDCB mothballs in 2014 by New Zealand's Environmental Protection Authority, they are easily obtainable through online stores and ethnic retail stores across New Zealand. This is evident in data shared by the National Poison Centre showing that there were 18 cases of ingestion of naphthalene mothballs from 2017 to 2025, of which 12 required hospital referrals. Nine of the 18 cases were recorded in the age group 0 to 2 years, consistent with normal mouthing/exploratory behaviour in this age group.

Health New Zealand - Te Whatu Ora has raised concerns regarding the availability of mothballs in New Zealand and requested that PHF Science prepare a report examining the adverse health effects of accidental ingestion of mothballs in children and toddlers.

Accidental ingestion of mothballs can cause serious adverse health effects in children and toddlers. Clinical reports show that naphthalene mothball ingestion can result in severe poisoning whereas data on PDCB poisoning in children are scarce. Notably, no naphthalene or PDCB-related poisoning case reports have been published in New Zealand to date, despite confirmed exposure incidents.

There are no health-based guidance values suitable for characterising the acute oral risk posed by accidental human ingestion of mothballs. The existing US EPA's acute reference dose for naphthalene is based on neurotoxic endpoints in rats, which do not reflect the haematological effects that are the actual concern in paediatric poisoning. Consequently, this risk assessment is qualitative rather than quantitative, based on the integration of clinical case-report evidence, incident surveillance data and hazard/toxicological profiling.

Clinical data from the literature suggest that accidental ingestion of at least one naphthalene mothball (up to 5 g and 99% purity) can cause haemolytic anaemia and methaemoglobinaemia leading to cyanosis, reduced oxygen saturation, tissue hypoxia and metabolic acidosis. Individuals with a glucose-6-phosphate dehydrogenase (G6PD) deficiency are more prone to haemolysis after exposure to naphthalene mothballs. Non-specific symptoms that generally appear before acute haemolytic anaemia include discoloured urine, headache, anorexia, vomiting, diarrhoea and other gastrointestinal disturbances. Data on PDCB poisoning in children are still very limited but symptoms of exposure typically manifest differently, with less severe haemolytic consequences. Other risks associated with mothballs include mechanical or obstructive problems due to their slippery nature particularly for PDCB mothballs.

Although the frequency of mothball exposure incidents in New Zealand is relatively low, their availability in the market remains a concern for public health, especially for children, toddlers and individuals with G6PD deficiency.

2. INTRODUCTION

Mothballs have been commonly used for many years to repel adult clothes moths (eg, *Tineola bisselliella*) and kill larvae and eggs responsible for damaging clothes in homes, particularly those that are stored for long periods of time. Traditionally mothballs consisted of naphthalene but a second active ingredient PDCB was developed, due to the high flammability of naphthalene (Dubey et al 2014; Kim 2015). Although PDCB is less flammable than naphthalene, it generates toxic vapours similar to naphthalene making it an effective moth repellent (Dubey et al 2014).

Since mothballs containing naphthalene and PDCB were extensively used in homes, they pose a potential poisoning hazard following accidental ingestion in toddlers (1 to < 3 years old) and children (under 18 years old). The accidental ingestion of mothballs by children is also well documented in published studies and surveys. A study in Iran investigated naphthalene poisoning cases from mothballs using the archived medical records of patients from 2008 to 2018 (Memar et al 2021) and found that the highest prevalence of ingestion was observed in the age groups of below five years (accidental ingestion) and 20 – 25 years (intentional ingestion) (Memar et al 2021). Similarly, a 2024 annual report of the National Poison Data System from America's Poison Centers reported 1,268 cases of naphthalene mothball ingestion in different age groups (Comstock et al 2026). Overall, 36% (n=457) of the 1,268 cases (accidental ingestion) were reported in children below five years of age and a similar percentage (37%, n=464) was observed in the adult age group (> 20 years).

Health New Zealand – Te Whatu Ora raised concerns regarding the availability of mothballs in New Zealand and adverse health effects associated with accidental ingestion in toddlers and children.

As repeated ingestion of mothballs is highly unlikely, this report will only consider acute oral exposure to naphthalene and PDCB through domestic use of mothballs.

The objectives of this project are to:

- 1) Identify routes of exposure to naphthalene and PDCB from mothballs.
- 2) Carry out a comprehensive review of adverse human health effects of naphthalene and PDCB after accidental ingestion in children and toddlers.
- 3) Carry out a risk assessment for acute exposure to naphthalene and PDCB mothballs in domestic settings.

Scope of the report

In scope:

- Mothballs used in domestic settings only.
- Acute risks associated with accidental ingestion of naphthalene and PDCB from mothballs in children and toddlers.

Out of scope:

- Chronic risks associated with exposure to naphthalene and PDCB mothballs.
- Acute risks associated with intentional use (recreational use and suicides) of naphthalene and PDCB from mothballs in adults.

- Risks associated with any other chemical components of the mothballs (eg, colourants, fragrances etc).
- Occupational exposure to naphthalene and PDCB.
- Exposure to naphthalene and PDCB from other sources (eg, paints, gasoline, tobacco smoke etc)

2.1 PRODUCT DESCRIPTION – NAPHTHALENE AND PARA-DICHLOROBENZENE MOTHBALLS

Mothballs are small white balls or flakes that contain either naphthalene or PDCB with or without fragrance and are available in different sizes. The naphthalene or PDCB content in mothballs is generally 99.9% and mothballs can range in weight from 0.5 g to 5 g (Avila et al 2006; Price and Jayjock 2008). Some mothball products may also contain colourants and small amount of camphor (0.5%).



Figure 1: Examples of mothballs available in New Zealand market; Source: Ubuy NZ¹ and Fruugo NZ²

2.2 PHYSICO-CHEMICAL PROPERTIES OF NAPHTHALENE AND PARA-DICHLOROBENZENE

Naphthalene is the most volatile polycyclic aromatic hydrocarbon (PAH) which is produced from coal tar fractions by distillation and crystallisation (RAR 2003; WHO 2010). Naphthalene and other PAHs are also generated from incomplete combustion processes originating in industry, domestic sources including cigarette smoke and motor vehicle exhaust, as well as natural events such as forest fires (Beygisangchin et al 2026; NPIC 2010b). Naphthalene has a number of industrial uses such as in synthesis of phthalate plasticisers and synthetic resins; as ingredients for plasterboards and paints; as dispersants in synthetic and natural rubbers;

¹ [International Online Shopping Store for Premium & Luxury Brands | Buy Imported Goods at Ubuy New Zealand](#): Assessed on 4 August 2026

² [Mould Proof Mothballs Mothproof Anti mite White Mothballs for Home Closet Wardrobe | Fruugo NZ](#): Assessed on 4 August 2026

and as tanning agents in the leather industry (WHO 2010). Wood smoke, fuel oil and gasoline also contain naphthalene (WHO 2010).

PDCB is a chlorinated aromatic compound used as an insecticide and repellent (NPIC 2010a). It was first registered for use by the United States Department of Agriculture in 1947. PDCB was one of the first replacements for naphthalene in mothballs, due to its lower flammability (NPIC 2010a). It is also used in the manufacture of 2,5-dichloroaniline and certain pharmaceuticals; in the manufacture of polyphenylene sulphide resins used for surface coatings and moulding resins; and to control mildew (IARC 1999)

The physico-chemical properties of both compounds are presented in Table 1.

Table 1: Physico-chemical properties of naphthalene and para-Dichlorobenzene

Property	Naphthalene ^{1,2}	para-Dichlorobenzene ^{1,2}
CAS RN	91-20-3	106-46-7
Molecular weight	128.17 g/mol	147 g/mol
Appearance	White crystalline or colourless to brown solid	Colourless to white solid crystal
Boiling point	217.9 °C	174 °C
Melting point	80 °C	53 °C
Odour	Distinct aromatic odour	Strong pungent
Odour threshold	0.0075 to 0.42 mg/m ³	1.1 mg/m ³
Solubility	31 mg/L at 25 °C (in water) soluble in benzene, alcohol, ether, acetone	80 mg/L at 25 °C (in water) soluble in chloroform, carbon disulfide, benzene, ether, alcohol
Partition coefficients	Log K _{ow} : 3.30	Log K _{ow} : 3.52
Flashpoint	open cup 79°C closed cup 88°C	closed cup 66°C
Vapour pressure at 25°C	0.085 mm Hg	1.3 mm Hg

CAS RN: Chemical abstract service registry number; Log K_{ow}: Logarithm of the octanol-water partition coefficient

¹ (NPIC 2010b; Pubchem 2026b)

² (Pubchem 2026a; US EPA 2020),

2.3 REGULATION OF MOTHBALLS

2.3.1 European Union (EU)

In the EU, the marketing and distribution of mothball products containing naphthalene and PDCB is banned under the Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) regulation (EU 2007; 2008; 2026; Poulsen et al 2015). Naphthalene and PDCB are not included as substances in Annex I, IA, or IB of the biocidal goods directive regarding the marketing of biocidal products (EU 2007; 2008; Poulsen et al 2015). This means that the biocidal goods cannot contain naphthalene and PDCB in product category 19 ("Repellent and Attractants"). Therefore, as of August 2007 and July 2008, the EU prohibited the use of PDCB and naphthalene in mothballs, respectively (EU 2007; 2008).

2.3.2 New Zealand

Mothball products containing naphthalene and PDCB have been banned by New Zealand's Environmental Protection Authority (NZ EPA) since June 2014, because of the risk of children being poisoned by ingesting them (NZ EPA 2020).

2.3.3 Australia

Mothballs containing naphthalene and PDCB are legally permitted but are loosely regulated in Australia as compared to the EU and New Zealand. However, there are some requirements to manage risks due to mothballs. In Australia, naphthalene is classified as a

Schedule 6 poison and is listed in the Poisons Standard—the Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) as 'Naphthalene (excluding its derivatives) except in liquid hydrocarbons as an impurity' (NICNAS 2015; SUSMP 2026). This means products containing these chemicals must carry warnings and be used exactly according to label directions. Therefore, mothballs must be sold in child-resistant packaging with proper labels.

2.4 HUMAN HEALTH HAZARD CLASSIFICATION OF NAPHTHALENE AND PARA-DICHLOROBENZENE

The hazard classification of naphthalene and PDCB are summarised in Table 2.

Table 2: Hazard classification of naphthalene and para-dichlorobenzene

Compound	Hazard classification			
	EU	NZ EPA	Australia	IARC
Naphthalene (CAS RN 91-20-3)	Acute Tox. 4 (Oral): harmful if swallowed Carcinogen 2: suspected of causing cancer	Acute Tox. 4 (oral): harmful if swallowed Acute Tox. 4 (dermal): harmful in contact with skin Eye Irritation. 2: causes eye irritation Carcinogen 2: suspected of causing cancer STOT Rep. Exp.1: causes damage to organs	Acute Tox. 4 (oral): harmful if swallowed Carcinogen 2: suspected of causing cancer STOT SE.3 ^a : causes damage to organs	Group 2B: possibly carcinogenic to humans
Para- dichlorobenzene (CAS RN 106-46- 7)	Eye Irritation 2: causes eye irritation Carcinogen 2: suspected of causing cancer	Eye Irritation 2: causes eye irritation Skin Irritation 2: causes skin irritation Carcinogen 2: suspected of causing cancer STOT Rep. Exp. 2: may cause damage to organs	Eye Irritation 2: causes eye irritation Carcinogen 2: suspected of causing cancer	Group 2B: possibly carcinogenic to humans
Reference/Source	ECHA	NZ EPA CCID	Safe Work Australia	(IARC 1999; 2002)

EU: European Union, NZ EPA: New Zealand's Environmental Protection Authority, IARC: International Agency for Research on Cancer, STOT SE: Specific target organ toxicity – single exposure, STOT RE: Specific target organ toxicity – repeated exposure; ECHA: European Chemicals Agency; CCID: Chemical Classification and Information Database

^aIn GHS 7.0, this category is only for narcotic effects. So, it is not applicable to naphthalene.

The hazard classifications of naphthalene and PDCB are largely aligned in Australia and the EU with the exception of naphthalene being classified for specific target organ toxicity– single exposure (STOT SE) in Australia and it is possible that the classification has not been updated by Safe Work Australia. However, an assessment done by the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) in 2015 did not classify naphthalene for STOT SE (NICNAS 2015). Similarly, the chemical classification and information database (CCID)

maintained by the NZ EPA has not been updated with the human health hazard classifications for naphthalene and PDCB.

Although none of the regulatory agencies have classified naphthalene as flammable, study data and recent safety data sheets show that naphthalene has a burning time of ≤ 45 sec with the rate of ≥ 2.2 mm/sec which classifies it as a flammable solid category 2 (ECHA 2010).

3. HAZARD IDENTIFICATION

3.1 PREVIOUS ASSESSMENTS

No previous health impact assessments that characterised the acute risks of accidental ingestion of mothballs in children were found for New Zealand.

3.2 HEALTH EFFECTS – INCIDENT SURVEILLANCE AND CASE REPORTS

3.2.1 Incident Surveillance – New Zealand

The National Poisons Centre (NPC) provided a summary of mothball exposure incidents between 2017 to 2025. There were 109 incidents (including exposure to camphor mothballs), of which 29% involved possible/probable/witnessed ingestion of mothball material and 71% were reportedly non-ingestions involving mucosal, topical, inhalational, or other exposures.

Yearly ingestion exposures to mothballs containing naphthalene and PDCB are summarised in Table 3. Note that the NPC also provided data on camphor mothballs which is not included in the table.

Table 3: Exposure cases to mothball products of interest in contacts with National Poisons Centre, 2017–2025

Exposure type	2017	2018	2019	2020	2021	2022	2023	2024	2025	Total
Apparent exposure incidents										
Naphthalene mothball	6	6	8	7	7	8	3	2	6	53
Ingestion	1	3	1	1	4	3	2	0	3	18
No ingestion	5	3	7	6	3	5	1	2	3	35
para-Dichlorobenzene mothball	5	1	1	0	0	0	0	0	1	8
Ingestion	1	0	0	0	0	0	0	0	0	1
No ingestion	4	1	1	0	0	0	0	0	1	7
Unknown mothball	1	0	5	2	2	0	2	1	2	15
Ingestion	0	0	4	1	1	0	0	0	0	6
No ingestion	1	0	1	1	1	0	2	1	2	9
Smell of mothball*	5	2	4	1	2	1	3	0	1	19
No known exposure										
Unknown if exposure to a mothball	1	0	0	0	0	0	0	0	0	1

No exposure, query about mothballs	1	0	3	3	1	0	1	0	0	9
Total contacts	19	9	21	13	12	9	9	3	10	105

*Source of smell believed to be from mothballs but not observed by reporter to be a mothball

Overall, about half of the exposures (50%, n=53) reportedly involved naphthalene mothballs and 8% (n=8) of exposure incidents involved PDCB mothballs.

The distribution of exposure incidents involving mothballs among children up to 9 years of age is presented in Table 4.

Table 4: Patient age groups in exposures to mothballs; National Poisons Centre, 2017-2025

Exposure type	0 to 2 years	3 to 9 years
Naphthalene mothball		
Ingestion	9	3
No ingestion	8	4
para-Dichlorobenzene mothball		
Ingestion	1	0
No ingestion	7	0
Unknown mothball		
Ingestion	3	1
No ingestion	5	1

Naphthalene mothballs accounted for the majority of ingestion incidents, with the highest number occurring in children aged 0–2 years (n = 9), followed by those aged 3–9 years (n = 3). A similar trend was observed for non-ingestion exposures to naphthalene mothballs, with 8 and 4 cases reported in the 0–2 and 3–9 year age groups, respectively. Only one ingestion involving PDCB mothballs was reported, occurring in the 0–2 year age group. Unknown mothball exposures also followed a similar pattern, with more incidents reported in the younger age group than in older children. Overall, exposure incidents declined with increasing age, with relatively few cases reported beyond childhood (data not shown). However, this was not the case in other countries like USA where incidences of naphthalene poisonings/exposure events were similar between children and adults (Comstock et al 2026). The predominance of naphthalene-related exposures compared with PDCB in New Zealand may reflect the greater availability or more widespread use of naphthalene mothballs among the general public.

Some reports also documented adverse health effects (haemolytic anaemia) in newborns and infants (3 to 7 years old) after dermal exposure to diapers and clothes heavily impregnated with naphthalene (ATSDR 2025; Schafer 1951; Sohn et al 1967). These incidents were reported in the 1950s and 1960s. It is possible that impregnating clothes and diapers with naphthalene was a widespread, normal household practice at that time due to the lack of alternatives and knowledge about the health risks associated with naphthalene. There is no intoxication cases reported recently due to dermal exposure to mothballs in infants. Therefore, skin contact represents a minor route of acute exposure to mothballs.

3.2.2 Case reports – poisoning due to mothballs

Accidental ingestion of mothballs containing naphthalene or PDCB can have detrimental adverse health effects in toddlers and children. There are many clinical reports of poisoning due to consumption of naphthalene mothballs. However, the data on PDCB poisoning in children are scarce. The case reports involve haematological investigations and it should be noted that these can be age dependent. Some recent case reports are summarised below.

1. A case of accidental ingestion of a naphthalene mothball was reported in a 15-year-old boy in India (Deo et al 2016). Following the ingestion, the boy had episodes of vomiting, and his urine developed a blackish-brown discolouration. His urine volume (<100 mL/day) also decreased in the next 24 hours. On hospital admission, he was in an irritable and confused state with tachycardia (heart rate: 118/min), tachypnoea (respiratory rate: 34/min), oedema and deep central cyanosis (bluish-purple discolouration of the skin and mucous membranes). Further investigations revealed that the boy suffered massive intravascular haemolysis and acute kidney injury. Arterial blood gas (ABG) analysis showed methaemoglobin (MetHb) of 25.3% (normal 0 to 2%) on day 1 of hospital admission. After receiving appropriate treatment (oxygen inhalation, forced alkaline diuresis and oral ascorbic acid, N-acetyl cysteine), MetHb decreased to 1% (day 8) and other parameters (Hb, creatinine, urine output) also improved by day 12. The high MetHb (25%) on day 1 from ingestion of only one mothball was considered unusual. Later, he was found to be glucose-6-phosphate dehydrogenase (G6PD)-deficient. In G6PD-deficient individuals, naphthalene poisoning causes MetHb levels to rise rapidly. Because these individuals lack sufficient NADPH to protect their red blood cells (RBCs) from oxidative stress, naphthalene metabolites (eg, naphthoquinones) quickly overwhelm the body's natural reduction pathways, leading to severe methaemoglobinaemia, tissue hypoxia, and acute intravascular haemolysis (Ata et al 2020; Deo et al 2016; Shah et al 2024).
2. A 2-year-old boy with the history of pica in India accidentally consumed an unknown number of naphthalene mothballs (Annamalai et al 2012). After 3 days, the boy was noticed to have discolouration of the eyes, altered senses and three episodes of generalised seizures. Haematological analysis revealed a low haemoglobin (Hb) level of 5 g/L (normal: 110 to 135 g/L), and an elevated white blood cell (WBC) count of 20,100 cells/mm³ (normal: 6200 to 17,000 cells/mm³). Blood smear analysis showed the presence of RBCs, spherocytes and fragmented RBCs, confirming haemolysis. Liver and renal functions were normal. His MetHb level was 7.5% and ABG analysis revealed metabolic acidosis. After receiving appropriate treatment (oxygen, IV fluids, KCO₃ infusion and methylene blue), his condition gradually improved. Haemoglobin level increased to 75 g/dL on day 2 and on the day of discharge it was 87 g/L. ABG analysis was normal. There were no further seizures and urine colour became lighter and was normal by day 5 (Annamalai et al 2012).
3. Kapoor et al (2014) also reported acute intravascular haemolysis and methemoglobinemia in an autistic 14-year-old boy in India following ingestion of an unknown number of naphthalene mothballs. After the ingestion of the mothballs, the boy had 3-4 episodes of vomiting containing pieces of naphthalene mothball. His urine developed a dark brown colour and he was admitted to the hospital. He was pale, icteric (yellow skin) and had central cyanosis with low oxygen saturation (SpO₂) of 86% (normal: 95 to 100%). Blood tests revealed low Hb (91 g/L; normal range: 120 to 160 g/L) and platelet count (195,000 cells/mm³) but with elevated total WBC count (16,000 cells/mm³; normal: 4,800 to 10,800/mm³). Peripheral blood smear analysis showed haemolysis as evidenced by the detection of nucleated RBCs, spherocytes and fragmented RBCs with reticulocyte count of 2.4%. MetHb level was found to be 12% (normal 0 to 2%) and ABG analysis revealed metabolic acidosis. His G6PD levels were found to be within the normal limits. After receiving the required treatment (oxygen inhalation, forced alkaline diuresis and

intravenous methylene blue, N-acetyl cysteine), gradual improvement was seen in oxygen saturation and normalisation of Hb and other biochemical parameters. His urine colour also became lighter and was normal by the end of his stay. He was discharged on day 7 of admission (Kapoor et al 2014).

4. A 3-year-old boy in Riyadh, Saudi Arabia who had accidentally ingested a naphthalene mothball was brought to the emergency department (ED) by his mother, after noting decreased activity and poor diet intake in the last three days (Eskandarani and Alghamdi 2020). The boy was of Mediterranean descent (high prevalence of G6PD deficiency) with asthma, autism, and attention deficit hyperactivity disorder. Family history revealed that one of his sibling had G6PD deficiency and his father was a sickle cell trait carrier. The boy was never tested for G6PD deficiency before admission to the ED. He had yellow sclera (white part of the eye), mild tachypnoea (respiratory rate: 28 breaths/min) and an oxygen saturation reaching 85% while receiving 15 L of supplemental O₂. He was presumptively diagnosed with acute haemolytic anaemia and emergency transfusion of RBCs was performed after blood tests revealed low Hb (67 g/L; normal range: 110 to 135 g/L) on day 1. Blood tests also revealed high WBC count and absolute neutrophil count but the platelet count and reticulocytes were within normal range. After 2 hours of admission to the ED, MetHb level was 6.1%. After undergoing appropriate treatment (oxygen inhalation, oral ascorbic acid, N-acetyl cysteine), slight improvement was observed on the second day with less laboured breathing despite requiring supplemental O₂ to achieve 95% saturation. After 3 days, the Hb level increased to 73 g/dL while MetHb fell to 1.3%. Other blood parameters also normalised during his hospital stay. Just before his discharge from the hospital he was found to be G6PD deficient (Eskandarani and Alghamdi 2020).
5. Nongrum et al (2021) reported a rare case of naphthalene poisoning with hypoxic brain injury in a 1-year-old male child in India. He was admitted to the hospital after several episodes of vomiting, passing red coloured urine and altered sensorium for the last two days. Investigation with the family revealed that the child had potentially ingested two mothballs after the mother noticed mothballs in the stool. At the time of hospital admission, he exhibited elevated tachycardia and tachypnoea with severely reduced blood oxygen saturation (64% on room air). Abdominal examination showed hepatomegaly and mild splenomegaly. Blood tests revealed low Hb (45 g/L), normal platelet count but high total WBC count, reticulocyte count and total bilirubin. Levels of liver enzymes and creatinine were also elevated. A peripheral blood smear showed anisopoikilocytosis (different sizes and shapes of RBCs), microcytic hypochromic RBCs (pale coloured) along with fragmented cells, polychromatic and occasional nucleated cells, indicating haemolysis. The patient's chest radiograph showed no abnormality. He was found to not be G6PD deficient. MetHb levels were within the normal range (1.7%). On the following day, the child developed generalised tonic-clonic convulsions which were managed by anticonvulsant drugs. Magnetic resonance imaging of the brain revealed acute lacunar infarcts in the caudate and lentiform nuclei along with hypoxic damage in the bilateral parietooccipital cortex. After appropriate management (oral ascorbic acid, blood transfusion) and requisite supportive treatments, his laboratory parameters gradually improved. After day 5, repeat blood tests revealed increases in Hb (84 g/L), normal total WBC and reticulocyte count. His MetHb levels also decreased to 1.3%. Other parameters (total bilirubin, liver enzymes) were also normal.
6. Naphthalene poisoning from chewing a mothball was reported in a 13-month-old male who was presented to the ED in Honolulu, Hawai'i, USA after developing a cough, runny nose, and fever which progressed to lethargy, pallor, jaundice, vomiting, and diarrhea after four days (Kuwada et al 2022). Initial examination showed tachypnoea, decreased urine output and severely reduced blood oxygen saturation (80% on room air). At the time of admission to the hospital, tachycardia (170/min, normal: 60 to 100/min),

tachypnoea (70/min; normal 12 to 16/min) and a slight fever (37.8°C) were also noted. Blood tests revealed low Hb level (27 g/L) and reticulocyte count (1.2%). Blood smear work showed haemolysis. ABG analysis revealed metabolic acidosis (pH 6.75, PCO₂ of 26.5 mm Hg, bicarbonate 3.6 mmol/L, lactate 15.47 mmol/L) and elevated MetHb (6.4%). During this time, the patient also developed rarely reported multiple organ system dysfunction including haemolytic anaemia with peak values of haptoglobin, lactate dehydrogenase (LDH), creatine kinase (CK) (indicating myolysis), creatinine, BUN, alanine aminotransferase (ALT) and respiratory failure. He required aggressive fluid resuscitation, blood transfusions, and mechanical ventilation. After receiving N-acetyl cysteine, his condition gradually improved and he made a full recovery after 2 weeks. Follow-up testing after full recovery showed G6PD deficiency with levels at 3 U/g Hb (normal value 7.0 to 20.5) (Kuwada et al 2022).

7. Acute intravascular haemolysis was reported after accidental ingestion of a naphthalene mothball by 18-month-old female child in India (Sarangi et al 2016). The girl did not show common symptoms of naphthalene poisoning, such as headache, altered mental status, abnormal movements, cyanosis, shortness of breath, abdominal pain, vomiting, tachycardia, tachypnoea and reduced blood oxygen saturation. However, she appeared severely pale and icteric. Blood tests revealed low Hb (49 g/L), normal platelet count but high total WBC count and reticulocyte count. Peripheral blood smear analysis revealed haemolysis in the form nucleated RBC, fragmented RBC and schistocytes. ABG analysis did not show any evidence of metabolic acidosis and methaemoglobinaemia. Her renal function, coagulation profile, electrolytes, and G6PD activity were normal. After receiving appropriate treatment (rapid infusion of normal saline, packed RBCs), her Hb level improved to 102 g/L and bilirubin declined. Her overall condition improved and she was discharged after 5 days of hospitalisation. A similar case of accidental naphthalene poisoning was also reported by Sravanthi et al (2024).
8. A 5-year-old girl in Riyadh, Saudi Arabia did not exhibit any complications such as haemolysis or methaemoglobinaemia after allegedly ingesting four naphthalene mothballs and was discharged safely from the hospital after 3 days (Alqahtani et al 2024). Other symptoms such as headache, altered mental status, abnormal movements, cyanosis, shortness of breath, abdominal pain, vomiting, diarrhoea, fever, or changes in urine or stool colour were also not observed. There was no family history of haematological diseases or G6PD deficiency. It should be noted that the naphthalene mothball ingestion was not witnessed by anyone and the presumption of ingestion was based on mothball smell from the girl's mouth along with remnants between her teeth. It is possible that the girl may have ingested less than one mothball and the quantity was not sufficient to induce adverse health effects.
9. As mentioned before, there is very limited data on PDCB poisoning in children due to accidental ingestion of mothballs. Hallowell (1959) reported acute haemolytic anaemia in a 3-year-old boy in Birmingham, United Kingdom after ingesting an unknown quantity of PDCB crystals. The boy was irritable, had yellow sclera and his urine developed a blackish discolouration before hospitalisation. On hospital admission, his body temperature, pulse rate and blood pressure was normal. Blood tests revealed low Hb (82 g/L) but high reticulocytes and total WBC count. Serum bilirubin and blood urea was also elevated. Urine analysis showed the presence of MetHb. After receiving blood transfusion his Hb level was 110 g/L but gradually declined over the next five days to 78 g/L. Follow up showed that the boy was clinically well and his Hb level was 116 g/L.
10. A 19-month-old boy in Baltimore, USA ingested an unknown number of PDCB mothballs and was brought to the ED after his aunt found a white material around his mouth with a characteristic odour (Sillery et al 2009). The boy was asymptomatic and his vital signs

(pulse rate, respiratory rate, body temperature) were normal. Blood tests also showed no abnormality. He was given activated charcoal and was discharged. However, he was again back to the ED after the family noticed a yellow hue in the skin. This time he was found to be icteric and fussy. His vital signs were elevated. Blood tests showed low Hb (64 g/L), high WBC count, MetHb (2.5%) and high total bilirubin concentration. After receiving blood transfusion, his condition improved quickly without any complications and his Hb level rose to 84 g/L.

11. In a rare case of a PDCB mothball ingestion in Florida, USA, Laughrey et al (2020) reported solid food dysphagia in a 3-year-old boy. After ingesting mothball, the boy was in respiratory distress, coughing, foamy sputum, drooling, and vomiting. He was airlifted to hospital and during transportation he developed hypoxia. ABG analysis revealed metabolic acidosis. Endoscopy showed that the mothball (1.7 cm in diameter) was adhered in the upper oesophagus and was exerting a compressive effect on the trachea. After many unsuccessful attempts due to the slippery nature of the mothball, the PDCB was finally removed using specialised equipment. Another endoscopy showed minimal mucosal erosions and no perforations. After receiving supportive care, the boy was discharged after 4 days. Two weeks after the discharge he returned to the clinic with further solid food dysphagia. Endoscopic examinations found a stricture (abnormal narrowing) in the proximal oesophagus at the location where the mothball had come into contact with the mucosa. With the suitable treatment his condition improved and was able to tolerate solid food. Further endoscopy confirmed the resolution of stricture (Laughrey et al 2020).

3.2.3 Summary

The cases summarised above suggest that reported incidents of poisoning are more common in countries like India where naphthalene mothballs are easily available and are sold without any regulatory oversight. There were no mothball poisoning case reports from New Zealand. The clinical data suggests that accidental ingestion of at least one naphthalene mothball can cause haemolytic anaemia and methaemoglobinaemia leading to cyanosis, reduced oxygen saturation, tissue hypoxia and metabolic acidosis. Nonspecific symptoms that generally appear before acute haemolytic anaemia include discoloured urine, headache, anorexia, vomiting, diarrhoea and other gastrointestinal disturbances. The intoxication can have more pronounced effects (MetHb up to 25%) in children with G6PD deficiency. This is due to the inability of RBCs to defend against the oxidative stress caused by naphthalene metabolites. PDCB poisoning data are scarce but have been observed to present with mechanical/obstructive complications. However, haemolytic anaemia and methaemoglobinuria in children are milder than naphthalene poisoning .

3.3 TOXICOLOGY

The toxicology of naphthalene and PDCB has been extensively reviewed by several authorities, such as the International Agency for Research on Cancer (IARC), Agency for Toxic Substances and Disease Registry (ATSDR), United States Environmental Protection Agency (US EPA), NICNAS and European Chemicals Agency (ECHA). Hence, the toxicology of naphthalene and PDCB is only briefly summarised below based on information provided in reports.

3.3.1 Naphthalene

Naphthalene is readily absorbed via the inhalation and oral route in humans (NICNAS 2015). In experimental animals, the absorption is rapid but concentration dependent. Following absorption, naphthalene is widely distributed in humans (ATSDR 2025). It has been detected in adipose tissues and breast milk of humans and is known to cross the placenta at sufficient doses to cause toxicity in newborns.

The metabolism of naphthalene has been extensively reported in experimental animals and is presented in Figure 2 (ATSDR 2025).

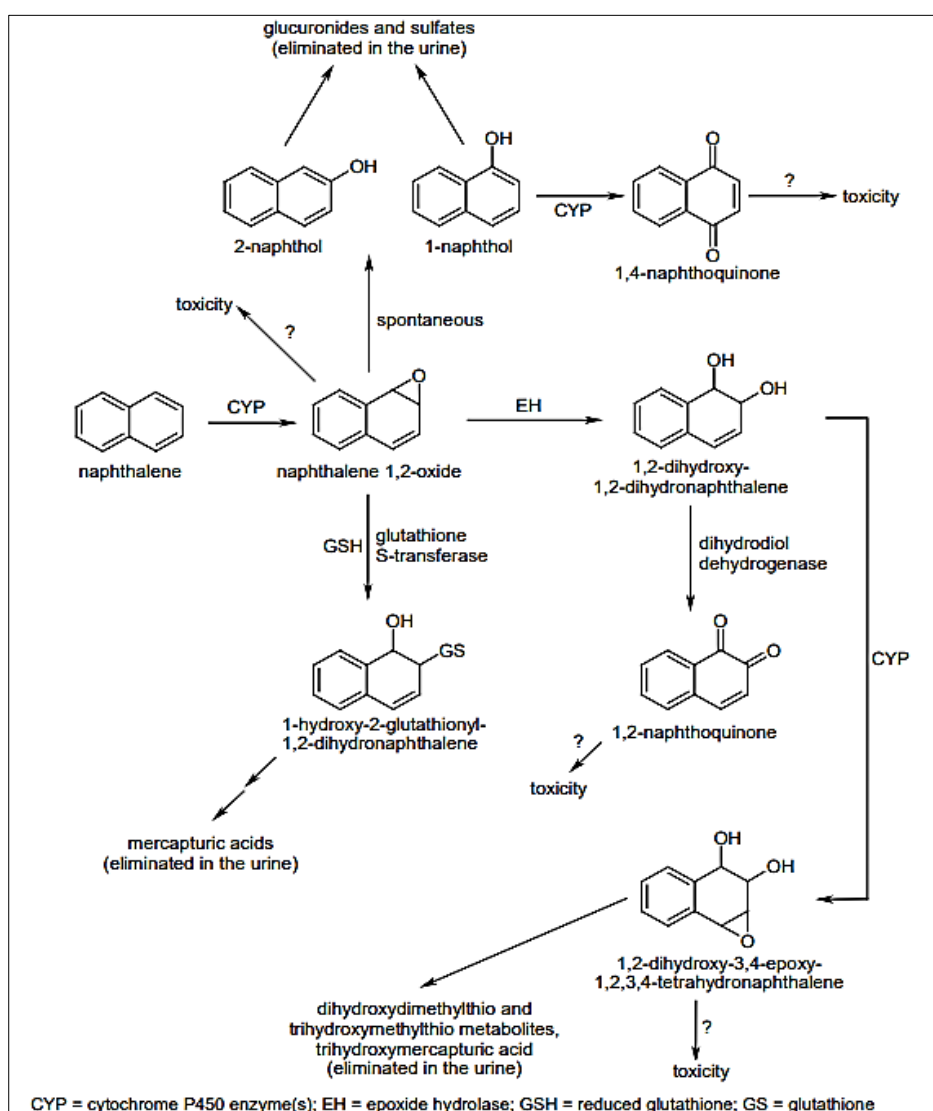


Figure 2: Metabolism of naphthalene; adapted from ATSDR (2025)

The metabolism of naphthalene generates a number of reactive metabolites by cytochrome P450 (CYP)-mediated epoxidation (ATSDR 2025; INCHEM 1992). These metabolites are thought to be responsible for toxicity in animals and potentially in humans.

Naphthalene-1,2-oxide, 1-naphthol and other metabolites are strong oxidising agents. The combined oxidative effects of these reactive metabolites are likely to be responsible for haemolysis in humans (INCHEM 1992). They deplete glutathione (GSH) and generate reactive oxygen species within RBCs. Oxidative damage denatures Hb, forming Heinz bodies, and damages the RBC membrane, leading to intravascular haemolysis. This is especially of concern in G6PD deficient individuals, since their RBCs cannot regenerate reduced GSH fast enough to neutralise the oxidative stress (ATSDR 2025; INCHEM 1992).

Acute Toxicity

The probable oral lethal dose of naphthalene is reported to be 5 to 15 grams (71.4–214.2 mg/kg, assuming a 70 kg body weight) in non-sensitive human adults and 2 to 3 grams for children (Clayton 1981; INCHEM 1992; Uthuman et al 2019). Only one case of paediatric mortality is reported in a child and none in adults. It should be noted that the lethal dose range for adults is extrapolated from just one paediatric death that occurred after ingesting 2 g of naphthalene over two days. The original study and subsequent reference do not provide supporting evidence (dose response, individual susceptibility) beyond a single anecdotal case.

Haemolytic anaemia is the major toxic effect observed after naphthalene poisoning in humans. The typical symptoms of naphthalene intoxication in humans are dark urine, pallor, abdominal pain, fever, nausea, diarrhoea and vomiting (INCHEM 1992; NICNAS 2015).

In contrast to the haemolytic effects observed in humans such effects were not seen in mice and rats after oral exposure. However, acute haemolytic anaemia has been observed in dogs at an oral dose of >411 mg/kg bw naphthalene (INCHEM 1992; NICNAS 2015). There are numerous well conducted acute oral toxicity studies in mice and rats. Naphthalene is of low acute oral toxicity in rats and moderately toxic in mice. The median oral lethal dose (LD₅₀) in Sprague Dawley rats was >2000 mg/kg bw and in mice the LD₅₀ in males and females was 533 and 710 mg/kg bw, respectively (ATSDR 2025; ECHA 2010; NICNAS 2015). Signs of toxicity included ataxia and depressed breathing, but not haemolytic anaemia in mice (NICNAS 2015).

Naphthalene was found to be a mild skin and eye irritant in rabbits (NICNAS 2015).

Repeated Dose Toxicity

In a 13-week oral toxicity study in rats, mortality was observed in two rats and transient signs of toxicity were reported including lethargy, hunched posture, rough hair coats and decreased body weight in the highest dose group (400 mg/kg bw/day) (ATSDR 2025; ECHA 2010; NICNAS 2015). These effects at 400 mg/kg bw/day (LOAEL) were considered for the derivation of aRfD by the US EPA. Decrease in body weight (12%) was also observed at 200 mg/kg bw/day. Haematological changes were observed in the highest dose group but were not considered biologically significant. A no observed adverse effect level (NOAEL) of 100 mg/kg bw/day was established based on decreased body weight (ECHA 2010; NICNAS 2015; US EPA 1998). Similarly in mice, transient signs of toxicity (lethargy, rough hair coats, and decreased food consumption) occurred between weeks 3 and 5 in the highest dose group (200 mg/kg bw/day).

Similar effects were seen in a sub-chronic oral toxicity in mice (NICNAS 2015). A NOAEL of 100 mg/kg bw/day was established based on increased body weight in male mice (US EPA 1998). Additionally, some studies indicated that the repeated administration of the chemical at very high doses (1000 mg/kg bw/day) can cause cataracts in rats and rabbits (NICNAS 2015) but this effect was not seen in mice (Poulsen et al 2015).

Genotoxicity

Naphthalene is not considered to be genotoxic based on a battery of *in-vitro* and *in-vivo* studies (ATSDR 2025; ECHA 2010; IARC 1999; NICNAS 2015).

Carcinogenicity

IARC has classified naphthalene as “possibly carcinogenic to humans” (Group 2B) on the basis of “inadequate evidence in humans” and “sufficient evidence in experimental animals” for determination of carcinogenicity (IARC 2002). Similarly, ECHA also classified it as Carc. 2, “Suspected of causing cancer” by the inhalation route (ECHA 2010). The classification was based on increased incidence of tumours in the respiratory tract of male rats even at the lowest administered concentration of 50 mg/m³ (ATSDR 2025; ECHA 2010). The exact mechanism of carcinogenicity is unknown. However, strong correlation has been observed between the rates of formation of the stereoisomer (1R,2S)-naphthalene oxide in various tissues and the selective toxicity of naphthalene to these tissues, suggesting that this metabolite may play a role in naphthalene’s toxicity to the lung and other tissues

3.3.2 para-Dichlorobenzene

PDCB is substantially absorbed following oral and inhalation exposure in rats and mice (RAR 2004). Oral absorption was similar in mice and rats (approximately 70–72%), whereas absorption following inhalation exposure was approximately 60% in mice and 33% in rats (RAR 2004). Thus, absorption was somewhat lower following inhalation than oral exposure, particularly in rats. No quantitative data on human absorption are available.

The tissue distribution of PDCB in rats was similar irrespective of the exposure route as well as after single or repeated modes of administration (NICNAS 2000; RAR 2004). PDCB is distributed primarily in fatty tissues followed by kidney and liver, with lower concentrations in the lungs, muscles, and in plasma (NICNAS 2000; RAR 2004).

The metabolism of PDCB has been extensively reported in experimental animals and in hepatic microsomes derived from humans. The proposed metabolic pathway is presented in Figure 3 (NICNAS 2000).

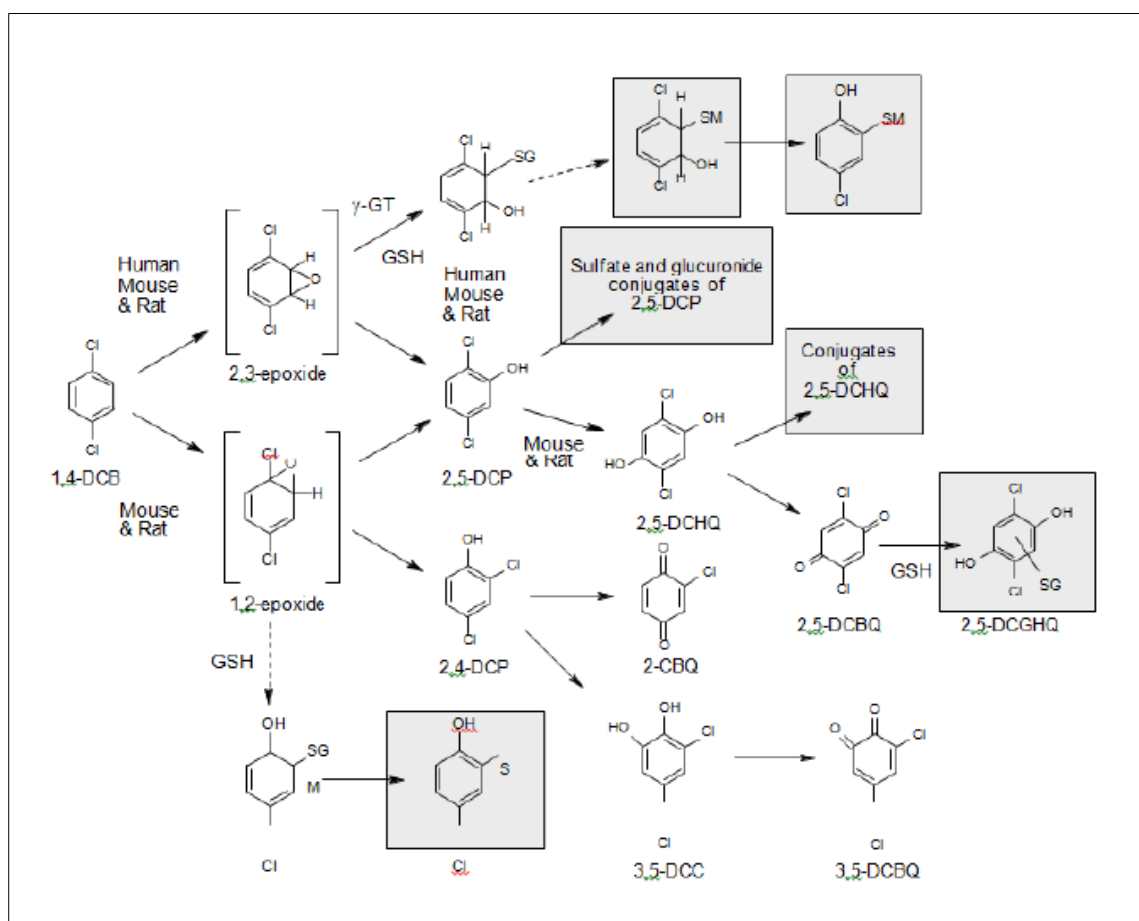


Figure 3: Metabolism of PDCB in humans and experimental animals; adapted from NICNAS (2000)

The metabolism of PDCB occurs through CYP-450 mediated epoxidation followed by formation of corresponding dichlorophenols- mainly 2,5-dichlorophenol (2,5-DCP) (ATSDR 2006; ECHA 2023; NICNAS 2000). 2,5-DCP is then conjugated with glucuronic and sulphuric acid and finally excreted through urine. The dichlorophenol may undergo oxidation to form dichlorocatechols, or possibly a dichlorohydroquinone, or may be conjugated by several phase II metabolism pathways (ATSDR 2006). Minor metabolites include 2,5-

dichlorohydroquinone (2,5-DCHQ), dichlorocatechol, 2,5-dichlorobenzene mercapturic acids, 2,5-dichlorophenyl-methylsulphoxide and -methylsulphone (NICNAS 2000).

Acute Toxicity

PDCB is of low acute toxicity by the oral, dermal and inhalation route of exposure. The LD₅₀ for oral and dermal routes of exposure in rats is >2000 mg/kg (ECHA 2023; RAR 2004; US EPA 2018). The LC₅₀ in rats after inhalation exposure was > 5.07 mg/L air (ECHA 2023; RAR 2004; US EPA 2018). No signs of toxicity were reported regardless of the exposure route.

PDCB was found to be a mild skin and eye irritant in rabbits but the irritation effects were fully reversible within the observation period (NICNAS 2000; RAR 2004; US EPA 2018). However, a classification of eye and skin irritant is warranted based on the limited data from workplace exposure reporting irritating effects on the mucous membranes on eyes and nose (ECHA 2023; RAR 2004).

Repeated Dose Toxicity

Repeated exposure to PDCB in experimental animals has shown effects on the respiratory system, liver, and kidneys from inhalation exposure while oral studies have reported effects on the blood, liver, and kidneys (ECHA 2023; NICNAS 2000).

In sub-chronic oral toxicity studies in rats, histopathological changes were seen at all doses (300-1500 mg/kg bw/day) in the kidneys of all male rats characterised by renal tubular cell alterations with hyaline droplet formation (ECHA 2023; NICNAS 2000; US EPA 2018). A statistically significant increase in liver weights was observed at doses of 900 mg/kg bw/day for both sexes. At the two high doses (1200 and 1500 mg/kg bw/day), pathological conditions were seen in both sexes which included degeneration and necrosis of hepatocytes, bone marrow hypoplasia, lymphoid depletion of the thymus and spleen and epithelial necrosis of the nasal turbinates. Slight but significant haematological changes were observed in male rats (300-1200 mg/kg bw/day) at the highest dose but not in female rats. The NOAEL for females was 600 mg/kg bw/day while a NOAEL for males could not be identified (RAR 2004). In another study in rats, a NOAEL of 300 mg/kg bw/day for males was established where renal cortical degeneration was observed at 600 mg/kg bw/day (NICNAS 2000; RAR 2004). Other studies in rats also reported similar effects and established NOAELs of similar order (RAR 2004).

In mice, reduced body weight gain and hepatocellular degeneration was observed in both sexes at all doses (600-1800 mg/kg bw/day) (ECHA 2023; NICNAS 2000). Serum cholesterol levels were increased in male mice at the highest dose. Leukopenia was also observed in male (at 600 mg/kg bw/day or more) and female mice (at 1000 mg/kg bw/day or more) (NICNAS 2000). At high doses (1500 and 1800 mg/kg/day), a hypoplasia of the bone marrow and spleen resulting in decreased haematopoiesis, lymphoid depletion of the spleen, and lymphoid necrosis of the thymus were observed (RAR 2004). In contrast to rats, no renal pathologies were observed in any treatment group. A LOAEL of 600 mg/kg bw/day for both sexes of mice was established (NICNAS 2000; RAR 2004).

Following long-term inhalation exposure (up to 2 years) to PDCB, an increase in kidney weight with mineralisation of the papilla collecting tubule and urothelial hyperplasia was seen at the highest dose (300 ppm) in male rats (NICNAS 2000). Respiratory metaplasia in the nasal cavity gland and eosinophilic changes in the respiratory epithelium were observed in females in the highest dose group. Based on renal pathology alone a NOAEL of 75 ppm was established. In another long term study in mice, liver weights, kidney weights as well as

hepatic enzyme levels were increased in both the sexes in highest dose group (300 ppm). Histopathological changes in the livers of male mice was also observed (NICNAS 2000).

Genotoxicity

PDCB is not considered to be genotoxic based on a battery of *in-vitro* and *in-vivo* studies (ECHA 2023; IARC 1999).

Carcinogenicity

The IARC has classified PDCB as “possibly carcinogenic to humans” (Group 2B) on the basis of “inadequate evidence in humans” and “sufficient evidence in experimental animals” for determination of carcinogenicity (IARC 1999). The classification was based on increased incidences of liver tumours in exposed mice, and a suggested mechanism of carcinogenicity that could plausibly occur in humans.

3.3.3 Summary

The adverse health effects differ substantially between humans and experimental animals after acute oral exposure to naphthalene and PDCB mothballs. The critical effects in humans are on the haemopoietic system, whereas in experimental animals, neurotoxic effects were predominantly observed.

4. DOSE–RESPONSE INFORMATION

A point of departure (POD) is defined as the point on a toxicological dose–response curve established from experimental data or observational data generally corresponding to an estimated low effect level or no effect level. It marks the beginning of extrapolation to health-based guidance values (HBGVs) such as the reference dose (RfD) or reference concentration (RfC), tolerable daily intake (TDI), tolerable weekly intake (TWI), provisional tolerable monthly intake (PTMI) or provisional tolerable weekly intake (PTWI). The most common PODs used are the NOAEL, the lowest-observed-adverse-effect level (LOAEL), or the statistical benchmark dose (BMD).

In the current context, concerns associated with mothballs exposure relate to acute exposure. The toxicology assessments summarised in this report have used HBGVs for naphthalene and PDCB derived by the US EPA that are summarised below.

4.1 NAPHTHALENE

The US EPA has derived acute and chronic RfDs for naphthalene that are presented in Table 5.

Table 5: Reference dose for naphthalene

Key Study	POD (mg/kg bw/day)	UF	Critical effect	HBGV (mg/kg bw/day)	Reference
Sub chronic oral toxicity study - rats	LOAEL: 400	1000	Hunched posture in females, head shaking in males and females, and reduced motor activity in males and females.	Acute RfD: 0.4	US EPA (2008)
	NOAEL: 71	3000	Decreased mean terminal body weight in males.	Chronic RfD: 0.02	US EPA (1998)

POD: Point of departure; UF: Uncertainty factor; HBGV: Health-based guidance values; RfD: Reference dose

4.1.1 Para-dichlorobenzene

The US EPA has derived an inhalation chronic reference concentration (RfC) for PDCB that is presented in Table 6. Although the RfC was derived from an oral toxicity study, the US EPA did not derive an oral RfD. The NOAEL from multigeneration reproductive study in rats was converted to human equivalent concentration (HEC) by route-to-route extrapolation followed by a dosimetry adjustment.

Table 6: Reference dose for paradichlorobenzene

Key Study	POD (mg/m ³)	UF	Critical effect	HBGV (mg/m ³)	Reference
Multigeneration reproductive study - rats	NOAEL (HEC): 75	1000	Increased liver weights in P1 males	RfC: 0.8 mg/m ³	IRIS (1994)

POD: Point of departure; HEC: Human equivalent concentration; UF: Uncertainty factor; HBGV: Health-based guidance values; RfC: Reference concentration

4.1.2 Summary

The US EPA has derived an acute oral RfD for naphthalene based on neurotoxic effects observed in a sub-chronic oral toxicity study in rats. In humans, the nervous system has not been shown to be a target organ for toxicity after acute ingestion of naphthalene. The critical

effects in humans are on haemopoietic system. Therefore, the acute RfD established by the US EPA is of questionable relevance for the current risk assessment of naphthalene after acute exposures. There were no HBGV derived from human data due the lack of any dose-response data for naphthalene-induced haemolytic anaemia. Similarly, no acute oral RfDs for PDCB were found in the literature.

5. EXPOSURE ASSESSMENT

Possible routes for exposure to mothballs in children are through oral, inhalation and dermal pathways which are discussed below. Naphthalene and PDCB readily sublime, meaning that inhalation represents a likely route of exposure. However, this exposure is typically chronic in nature, which does not fall within the scope of this report. Since, only accidental ingestion of mothballs is within the scope of this report, an oral exposure dose was estimated for children (body weight: 16 kg [75th percentile, 2 – < 3 year age) and 21 kg (75th percentile, 3 – <6 year age)] after accidentally ingesting one 5 g mothball (Cressey 2016).

5.1 ORAL EXPOSURE

The incidents reported in the literature as well as exposure cases in New Zealand show that accidental ingestion of mothballs is the main route of exposure in toddlers and children. This is due to the mouthing and exploratory behaviour in children.

Mouthing behaviour is a normal part of infant/toddler development — putting objects in the mouth to explore texture, taste, and shape is developmentally typical, especially in children under the age of 3 years. This is exactly the age bracket the NPC data flagged as highest-risk: 9 of 18 naphthalene ingestion cases were in children aged 0–2 years.

Curious or exploratory behaviour in slightly older toddlers can attract them to mothballs due to the round, shiny and white or coloured appearance often mistaken for candy.

The exposure after accidental ingestion of one mothball containing naphthalene or PDCB in children was estimated using the below equation:

$$\text{Exposure dose } \left(\frac{\text{mg}}{\text{kg}} \right) = \frac{C \times W}{BW}$$

Where, C is the concentration of naphthalene or PDCB (99% or 0.99), W is the weight of one mothball (5 g) and BW is the body weight of child (16 or 21 kg).

The external exposure dose after ingesting one mothball was 309 and 235 mg/kg for a 16 and 21 kg child, respectively.

6. RISK CHARACTERISATION

Risk characterisation is the final step of the risk assessment process that integrates hazard identification, hazard characterisation and exposure assessment to estimate the likelihood of adverse effects in a given population. The lack of dose-response information in humans for naphthalene and PDCB makes definitive characterisation of risks problematic.

As mentioned in section 4.1, there are no HBGVs that could be used for the risk assessment for naphthalene and PDCB after acute ingestion of mothballs by toddlers and children.

As a result, a qualitative risk evaluation is carried out using the available data on intoxication following mothball ingestion. The standard acute toxicity tests in experimental animals suggest that naphthalene and PDCB are of low acute toxicity by the oral route. However, based on the clinical literature, it can be established that ingestion of even a single naphthalene mothball (up to 5 g, 99% purity) by children and toddlers causes haemolytic anaemia and methaemoglobinaemia, progressing to cyanosis, tissue hypoxia, and metabolic acidosis. Individuals with G6PD deficiency face substantially elevated risk.

PDCB poisoning data remains comparatively scarce, but data suggests milder haemolytic effects than for naphthalene with a distinct risk of mechanical/obstructive complications (eg, oesophageal impaction and stricture) due to the slippery nature of PDCB mothballs.

At this point, it is not possible to establish a dose or exposure that may not cause adverse health effects in children after accidental ingestion of naphthalene or PDCB containing mothballs.

7. CONCLUSIONS

The present qualitative risk assessment was conducted to evaluate health risks associated with accidental ingestion of mothballs in toddlers and children.

The acute oral toxicity studies in experimental animals show that naphthalene and PDCB are of low acute toxicity. However, reported poisoning incidents involving the accidental ingestion of just one naphthalene mothball (up to 5 g, 99% purity) by children and toddlers demonstrate that even one mothball can result in adverse health effects. Acute haemolytic anaemia is the most important adverse health effect reported after accidental consumption of mothballs. Individuals with a G6PD deficiency are more prone to haemolysis after exposure to naphthalene mothballs. Non-specific symptoms that generally appear before acute haemolytic anaemia include discoloured urine, headache, anorexia, vomiting, diarrhoea and other gastrointestinal disturbances.

PDCB poisoning data is scarce or under-reported and can also present with mechanical/obstructive complications. However, haemolytic anaemia and methaemoglobinuria in children are milder than naphthalene poisoning.

Despite the 2014 ban on naphthalene and PDCB mothballs in New Zealand, NPC data confirm these products remain available and are being accidentally ingested by young children, predominantly those aged 0 – 2 years. While the exposure incidents due to mothballs are low in New Zealand, their availability in the market remains a concern for public health, especially for children and toddlers.

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