RESORCINOL

Data were last reviewed in IARC (1977) and the compound was classified in *IARC Monographs* Supplement 7 (1987).

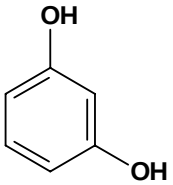
1. Exposure Data

1.1 Chemical and physical data

1.1.1 *Nomenclature*

Chem. Abstr. Serv. Reg. No.: 108-46-3
Chem. Abstr. Name: 1,3-Benzenediol
IUPAC Systematic Name: Resorcinol
Synonyms: *meta*-Benzenediol; resorcin

1.1.2 *Structural and molecular formulae and relative molecular mass*



$C_6H_6O_2$ Relative molecular mass: 110.11

1.1.3 *Chemical and physical properties of the pure substance*

(*a*) *Description*: White, needle-like crystals with a sweetish taste (Budavari, 1996)
(*b*) *Boiling-point*: 280°C (Budavari, 1996)
(*c*) *Melting-point*: 111°C (Lide, 1997)
(*d*) *Solubility*: *S*oluble in water, ethanol, diethyl ether and glycerol; slightly soluble in chloroform (Budavari, 1996)
(*e*) *Vapour pressure*: 665 Pa at 138°C; relative vapour density (air = 1), 3.79 (Verschueren, 1996)
(*f*) *Flash point*: 127°C, closed cup (Lewis, 1993)
(*g*) *Explosive limits*: Lower explosive limit (199°C), 1.4% by volume in air (Schmiedel & Decker, 1993)
(*h*) *Conversion factor*: $mg/m^3 = 4.5 \times ppm$

1.2 Production and use

Worldwide production of resorcinol in 1994 was estimated to be 30 000–35 000 tonnes. Countries producing resorcinol include Germany, Italy, Japan, the United Kingdom and the United States (Krumenacker *et al*., 1995).

Resorcinol is used primarily in the rubber industry for tyres and reinforced rubber products (conveyer belts, driving belts) and in high-quality wood adhesives, which are made from resorcinol, phenol and formaldehyde. It is also used in the preparation of dyes and pharmaceuticals, as a cross-linking agent for neoprene and a rubber tackifier, and in cosmetics (Lewis, 1993; Schmiedel & Decker, 1993; Krumenacker *et al.*, 1995).

1.3 Occurrence

1.3.1 *Occupational exposure*

According to the 1981–83 National Occupational Exposure Survey (NOES, 1997), as many as 100 000 workers in the United States were potentially exposed to resorcinol (see General Remarks). Occupational exposures to resorcinol may occur in its production, in the manufacture of wood adhesives, rubber, wood products, dyes and pharmaceuticals, and in coal processing.

1.3.2 *Environmental occurrence*

Resorcinol may be released to the environment in waste effluents associated with coal gasification and conversion, coal-tar production and shale oil processing and from the combustion of wood and tobacco. It has been detected in low levels in groundwater samples (United States National Library of Medicine, 1997).

1.4 Regulations and guidelines

The American Conference of Governmental Industrial Hygienists (ACGIH) (1997) has recommended 45 mg/m^3 as the 8-h time-weighted average threshold limit value for occupational exposures to resorcinol in workplace air. Similar values have been used as standards or guidelines in many countries (International Labour Office, 1991).

No international guideline for resorcinol in drinking-water has been established (WHO, 1993).

2. Studies of Cancer in Humans

No data were available to the Working Group.

3. Studies of Cancer in Experimental Animals

Resorcinol showed no carcinogenic effect in mice when tested by repeated skin application (IARC, 1977).

3.1 Oral administration

3.1.1 *Mouse*

Groups of 60 male and 60 female B6C3F_1 mice, seven to eight weeks of age, were administered resorcinol (purity, > 99%) by oral gavage in deionized water at dose levels of 0, 112 or 225 mg/kg bw on five days per week for 104 weeks. Groups of 10 animals of each sex were killed after 15 months. Mean body weights of high-dose female mice were 10–15% lower than those of controls from week 85 to the end of the study, whereas those of the remaining exposed groups were similar to those of the controls. Survival of exposed mice was similar to that of controls. All mice were necropsied and tissues examined histologically. There was no treatment-related increase in the incidence of neoplasms or non-neoplastic lesions (United States National Toxicology Program, 1992).

3.1.2 *Rat*

Groups of 60 male and 60 female Fischer 344 rats, six to seven weeks of age, were administered resorcinol (purity, > 99%) by oral gavage in deionized water on five days per week for 104 weeks at dose levels of 0, 112 or 225 mg/kg bw for males and 0, 50, 100 or 150 mg/kg bw for females. Groups of 10 animals of each sex were killed after 15 months. Mean body weights of high-dose male rats were 10–15% lower than those of the controls from week 87 to the end of the study. Mean body weights of high-dose females were 11–14% lower than those of controls from week 95 to the end of the study. Mean body weights of other exposed rats were similar to those of controls. Survival of high-dose males and females was significantly lower than that of controls. Decreased survival in the high-dose groups was attributed to chemical-related toxicity. All rats were necropsied and tissues were examined histologically. There was no treatment-related increase in the incidence of neoplasms or non-neoplastic lesions (United States National Toxicology Program, 1992).

3.2 Administration with known carcinogens

3.2.1 *Rat*

Groups of 15 male Fischer 344 rats, six weeks of age, were given resorcinol [purity unspecified] at concentrations of 0 or 0.2% in the diet for 22 weeks either after ligation of one ureter to enhance bladder carcinogenesis or after exposure to 0.05% *N*-nitrosobutyl-*N*-(4-hydroxybutyl)amine in the drinking-water for two weeks followed by ureteric ligation to initiate bladder carcinogenesis. All animals were killed at 24 weeks. Resorcinol administration did not affect body weight. Resorcinol alone did not induce bladder lesions and did not increase the incidence of any type of tumour when given after the initiator (Miyata *et al*., 1985).

Groups of 16 male Fischer 344 rats, six weeks of age, were given resorcinol (purity, > 99%) at concentrations of 0 or 0.8% in the diet for 51 weeks. Other groups were given 0 or 0.8% resorcinol in the diet for 51 weeks beginning one week after oral gavage of 50 mg/kg bw *N*-methyl-*N*′-nitro-*N*-nitrosoguanidine to initiate gastric carcinogenesis. Resorcinol reduced the body weights of rats given no initiator and of those given initiator.

Resorcinol alone induced a low incidence of mild hyperplasia in the forestomach, but no forestomach tumours. Resorcinol given after the initiator did not increase the incidence of forestomach papillomas or squamous-cell carcinomas induced by the initiator (Hirose *et al*., 1989).

Groups of 9–11 male Sprague-Dawley rats, weighing 200 g, were given 0 or 100 mg/kg resorcinol (purity, > 99%) in the diet for six weeks beginning one week after partial hepatectomy and intraperitoneal injection of 300 mg/kg bw *N*-nitrosodiethylamine to initiate liver carcinogenesis. Resorcinol treatment after initiation did not increase the multiplicity of enzyme-altered (γ-glutamyltranspeptidase) foci (Stenius *et al*., 1989).

Groups of 11–12 male Fischer 344 rats, five weeks of age, were given resorcinol (purity, > 99%) at concentrations of 0 or 0.8% in the diet for 49 weeks either alone or one week after intraperitoneal injection of *N*-nitrosomethyl-*n*-amylamine to initiate upper digestive tract carcinogenesis. Resorcinol given after the initiator reduced body weight gain compared with rats given initiator and increased kidney weights. In the group given resorcinol after initiation, the incidence of papillomas of the tongue was increased to 6/12 ($p < 0.05$) compared with 1/11 in the group given initiator and incidence of oesophageal carcinoma was increased to 7/12 ($p < 0.01$) compared with 0/11 in controls. The incidence of lung alveolar cell hyperplasia was reduced (Yamaguchi *et al*., 1989). [The Working Group noted that no data were reported on the group given resorcinol only.]

Groups of 10 or 20 male Fischer 344 rats, six weeks of age, were given resorcinol (purity, > 99.5%) at concentrations of 0 or 0.8% in the diet for 36 weeks either alone or after exposure to 0.05% *N*-nitrosobutyl-*N*-(4-hydroxybutyl)amine in the drinking-water for four weeks to initiate bladder carcinogenesis. Resorcinol did not affect body or bladder weight either when given alone or after initiator. Resorcinol exposure alone did not induce bladder lesions. Feeding of resorcinol after initiation did not increase the incidence or multiplicity of bladder neoplasms resulting from initiation (Kurata *et al*., 1990).

Groups of 15 or 20 six-week-old male Wistar/Crj rats were given resorcinol [purity unspecified] at concentrations of 0 or 0.8% in the diet for 36 weeks alone or starting one week after exposure to 0.1% *N*-nitrosoethyl-*N*-hydroxyethylamine in the drinking-water for three weeks to initiate liver and kidney carcinogenesis. The final body weights of rats given resorcinol were lower than those of rats given basal diet alone or with initiator. Resorcinol also increased the relative liver and kidney weights. Resorcinol treatment after initiation did not affect the incidence of liver or kidney tumours induced by the initiator, but reduced the multiplicity of liver tumours (Okazaki *et al*., 1993).

3.2.2 *Hamster*

Groups of 10 or 20 female Syrian golden hamsters were given resorcinol (purity, > 99.5%) at concentrations of 0 or 1.5% in the diet for 16 weeks alone or after two subcutaneous injections of 70 mg/kg bw *N*-nitrosobis(2-oxopropyl)amine to initiate pancreatic carcinogenesis. Resorcinol given either alone or after the initiator did not affect body weight gain or pancreas weight; when given after the initiator, it did not affect liver weight, but when resorcinol was given alone, liver weight was reduced. Resorcinol alone

did not induce pancreatic neoplasms. When given after the initiator, it did not affect the incidence of pancreatic adenocarcinomas, but reduced the incidence of atypical ductal hyperplasia. Resorcinol alone did not induce liver lesions. When given after the initiator, it did not affect the incidence of liver carcinomas or cholangiocarcinomas or gall-bladder carcinomas induced by the initiator. In the forestomach and glandular stomach, resorcinol alone or after initiator increased the frequency of epithelial hyperplasia [data not given] (Maruyama *et al*., 1991).

4. Other Data Relevant to an Evaluation of Carcinogenicity and its Mechanisms

4.1 Absorption, distribution, excretion and metabolism

4.1.1 *Humans*

No data were available to the Working Group.

4.1.2 *Experimental systems*

The pharmacokinetics of [^{14}C]resorcinol were studied after subcutaneous injection (single dose of 10–100 mg/kg bw, or repeated dosing at 100 mg/kg bw per day) in Sprague-Dawley rats (Merker *et al*., 1982). Since most measurements were of radioactivity, no distinction was made between resorcinol and its metabolites. The radioactivity was very rapidly cleared, mainly in urine as conjugates. Repeated administration of resorcinol did not change its elimination characteristics as judged by radioactivity.

4.2 Toxic effects

The toxicity of resorcinol has been reviewed (GDCh-Advisory Committee on Existing Chemicals of Environmental Relevance (BUA), 1993).

4.2.1 *Humans*

Concentrated solutions of resorcinol are irritating to the skin and ingestion of large doses may induce hypothermia, hypotension, decreased respiration, tremors, icterus and haemoglobinuria in children (IARC, 1977).

Several cases of serious and even fatal intoxication after dermal application of resorcinol have been described. Methaemoglobinaemia, haemolysis and convulsions have often been noted (Cunningham, 1956; Lundell & Nordman, 1973; Bontemps *et al*., 1995). Resorcinol is a rare cause of allergic contact dermatitis (Vilaplana *et al*., 1991; Pecegueiro, 1992; Serrano *et al*., 1992; Massone *et al*., 1993) and may also induce generalized eczema, urticaria and angioneurotic oedema (Barbaud *et al*., 1996).

4.2.2 *Experimental systems*

In a 17-day gavage study (27.5–450 mg/kg bw per day) in Fischer 344/N rats, hyperexcitability was observed in males at doses of ≥ 225 mg/kg bw and in females at levels

of ≥ 55 mg/kg bw, and tachypnoea at levels of 450 and 110 mg/kg bw, respectively; no gross or microscopic lesions attributable to resorcinol were observed. In a 13-week study at dose levels of 32–520 mg/kg bw per day, no gross or microscopic lesions or haematological or clinical chemistry changes were observed in rats. However, all rats given the highest dose died, and there was mortality also at the dose of 260 mg/kg bw per day (United States National Toxicology Program, 1992).

In the two-year carcinogenicity study (see Section 3.1.2) with dose levels of 112 and 225 mg/kg bw per day, no gross or microscopic lesions were observed in rats, although survival of male and female rats was significantly lower than that of the controls in the high-dose groups (225 and 150 mg/kg bw per day, respectively) (United States National Toxicology Program, 1992).

In mice, no treatment-related gross or microscopic lesions, or haematological or clinical chemistry changes were observed in 17-day (≤ 600 mg/kg bw per day), 13-week (≤ 420 mg/kg bw per day) or two-year (112 or 225 mg/kg bw per day) studies. Survival was decreased at the highest dose in 17-day and 13-week studies, but not in the long-term study (United States National Toxicology Program, 1992).

Administration of resorcinol (0.25% in the diet) for 20 weeks did not induce hyperplasia or papillomatous lesions in the forestomach in Syrian golden hamsters. Labelling index, after an intraperitoneal dose of [*methyl*-^{3}H]thymidine, was not elevated in the pyloric region, forestomach or urinary bladder (Hirose *et al*., 1986).

In male Fischer rats, oral administration of resorcinol for eight weeks (0.8% in the diet) did not induce hyperplasia or DNA synthesis, as measured by a bromodeoxyuridine-labelling index, in the forestomach epithelium. No cell proliferation, increased DNA synthesis or increase in pepsinogen-altered neoplastic foci was observed in the pyloric mucosa (Shibata *et al*., 1990).

Resorcinol (≤ 1.0 μg/mL) had no effect on the elimination by apoptosis of G418-resistant, transformed Swiss 3T3 MxCl1 cells by co-cultured TGF-β-treated C3H 10T½c8 cells (Schaefer *et al*., 1995).

Resorcinol (200 mg/kg bw as a single oral dose) administered to male Sprague-Dawley rats did not affect the urinary excretion of malonaldehyde, did not increase hepatic ornithine decarboxylase activity and at 0.3 mmol/L did not induce depletion of glutathione content in isolated hepatocytes (Stenius *et al*., 1989).

4.3 Reproductive and developmental effects

4.3.1 *Humans*

No data were available to the Working Group.

4.3.2 *Experimental systems*

Resorcinol (125, 250 or 500 mg/kg bw per day) administered to rats by gavage on days 6 through 15 of the gestation induced no embryotoxicity or fetal external, visceral or skeletal anomalies in the fetuses; no toxicity to the dams was observed (DiNardo *et al*., 1985). No embryotoxicity or teratogenic effects were observed in rats (40, 80 or 250

mg/kg bw per day, days 6 through 15 of gestation) or in rabbits (25, 50 or 100 mg/kg bw per day, days 6 through 18 of gestation) in a study reported as an abstract (Spengler *et al*., 1986).

4.4 Genetic and related effects

4.4.1 *Humans*

No data were available to the Working Group.

4.4.2 *Experimental systems* (see Table 1 for references)

Resorcinol did not induce gene mutations in *Salmonella typhimurium* or in *Escherichia coli* strains in either the presence or absence of an exogenous metabolic activation system.

In-vitro exposure to resorcinol did not induce sister chromatid exchanges either in Chinese hamster ovary CHO cells or in human lymphocytes. Chromosomal aberrations were induced *in vitro* in human lymphocytes and amniotic cells but not in CHO cells or human fibroblasts.

In rats *in vivo*, neither sister chromatid exchanges nor micronuclei were induced in bone marrow.

5. Summary of Data Reported and Evaluation

5.1 Exposure data

Exposure to resorcinol may occur in its production, in the manufacture of adhesives, rubber, wood products, dyes and pharmaceuticals. It has been detected at low levels in groundwater and occurs in wood smoke and tobacco smoke.

5.2 Human carcinogenicity data

No data were available to the Working Group.

5.3 Animal carcinogenicity data

Resorcinol was tested for carcinogenicity in one experiment in mice and in one experiment in rats by oral administration. It was also tested in mice by skin application. No carcinogenic effect was observed in these experiments.

In several experiments in rats and hamsters, resorcinol was tested for promoting activity after initiation by known carcinogens. It did not enhance the incidence of tumours of the bladder, forestomach, liver or kidney.

In one study, resorcinol increased the incidence of tongue and oesophageal tumours after initiation with *N*-nitrosomethyl-*n*-amylamine.

Table 1. Genetic and related effects of resorcinol

Test system	Result[a]		Dose (LED or HID)[b]	Reference
	Without exogenous metabolic activation	With exogenous metabolic activation		
SA0, *Salmonella typhimurium* TA100, reverse mutation	–	–	2500	JETOC (1997)
SA2, *Salmonella typhimurium* TA102, reverse mutation	–	–	2500	JETOC (1997)
SA4, *Salmonella typhimurium* TA104, reverse mutation	–	–	2500	JETOC (1997)
SA5, *Salmonella typhimurium* TA1535, reverse mutation	–	–	2500	JETOC (1997)
SA7, *Salmonella typhimurium* TA1537, reverse mutation	–	–	2500	JETOC (1997)
SA9, *Salmonella typhimurium* TA98, reverse mutation	–	–	2500	JETOC (1997)
ECW, *Escherichia coli* WP2 *uvrA*, reverse mutation	–	–	2500	JETOC (1997)
ECW, *Escherichia coli* WP2 *uvrA*/pKM 101, reverse mutation	–	–	2500	JETOC (1997)
DMX, *Drosophila melanogaster*, sex-linked recessive lethal mutations	–[c]		11 000 ppm feed	Foureman *et al*. (1994)
G5T, Gene mutation, mouse lymphoma L5178Y cells, *tk* locus *in vitro*	+	NT	250	McGregor *et al*. (1988)
SIC, Sister chromatid exchange, Chinese hamster ovary CHO cells *in vitro*	–	–	2035	Darroudi & Natarajan (1983)
CIC, Chromosomal aberrations, Chinese hamster ovary CHO cells *in vitro*	–	–	2035	Darroudi & Natarajan (1983)
SHL, Sister chromatid exchange, human lymphocytes *in vitro*	–	NT	125	Darroudi & Natarajan (1983)
CHF, Chromosomal aberrations, human fibroblasts *in vitro*	–	NT	64	Darroudi & Natarajan (1983)
CHL, Chromosomal aberrations, human lymphocytes *in vitro*	+	NT	25	Darroudi & Natarajan (1983)
CHL, Chromosomal aberrations, human lymphocytes *in vitro*	+	NT	80	Schulz *et al*. (1982)
CHL, Chromosomal aberrations, human trisomy 21-lymphocytes *in vitro*	+	NT	80	Schulz *et al*. (1982)
CIH, Chromosomal aberrations, amniotic cells *in vitro*	+	NT	40	Schulz *et al*. (1982)

Table 1 (contd)

Test system	Result[a]		Dose (LED or HID)[b]	Reference
	Without exogenous metabolic activation	With exogenous metabolic activation		
SVA, Sister chromatid exchange, Sprague-Dawley rat bone-marrow cells *in vivo*	–		100 ip × 1	Bracher *et al*. (1981)
SVA, Sister chromatid exchange, Sprague-Dawley rat bone-marrow cells *in vivo*	–		100 po × 1	Bracher *et al*. (1981)
SVA, Sister chromatid exchange, Sprague-Dawley rat bone-marrow cells *in vivo*	–		200 topical × 1	Bracher *et al*. (1981)
MVM, Micronucleus test, mouse bone-marrow cells *in vivo*	–		300 ip × 1	Darroudi & Natarajan (1983)

[a] +, positive; –, negative; NT, not tested
[b] LED, lowest effective dose; HID, highest ineffective dose; in-vitro tests, µg/mL; in-vivo tests, mg/kg bw/day; po, oral; ip, intraperitoneal
[c] Inconclusive results with injection of 12 000 ppm resorcinol

5.4 Other relevant data

Resorcinol is water-soluble and readily conjugated and eliminated. The chemical has no known potential for formation of electrophilic reactive intermediates comparable to those derived from the other dihydroxybenzenes. Resorcinol was tested in various genetic toxicology assays, including in-vitro bacterial and mammalian assays and in-vivo mammalian assays. It gave negative results in all studies, with the exception of a positive response in the two in-vitro studies that assessed chromosomal aberrations in human lymphocytes from whole blood cultures; however, resorcinol did not induce chromosomal aberrations in human fibroblasts.

5.5 Evaluation

No epidemiological data relevant to the carcinogenicity of resorcinol were available.

There is *inadequate evidence* in experimental animals for the carcinogenicity of resorcinol.

Overall evaluation

Resorcinol is *not classifiable as to its carcinogenicity to humans (Group 3)*.

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