2-NITROANISOLE

1. Exposure Data

1.1 Chemical and physical data

1.1.1 *Nomenclature*

Chem. Abstr. Serv. Reg. No.: 91-23-6

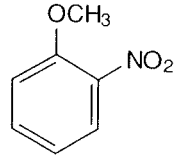
Deleted CAS Reg. No.: 35973-13-8

Chem. Abstr. Name: 1-Methoxy-2-nitrobenzene

IUPAC Systematic Name: ortho-Nitroanisole

Synonyms: 2-Methoxynitrobenzene; 2-methoxy-1-nitrobenzene; *ortho*-nitroanisole; *ortho*-nitrobenzene methyl ether; 2-nitromethoxybenzene; *ortho*-nitromethoxybenzene; 1-nitro-2-methoxybenzene; *ortho*-nitrophenyl methyl ether

1.1.2 *Structural and molecular formulae and relative molecular mass*



$C_7H_7NO_3$ Relative molecular mass: 153.13

1.1.3 *Chemical and physical properties of the pure substance*

(*a*) *Description*: Colourless to yellowish liquid (Budavari, 1989)

(*b*) *Boiling-point*: 277 °C (Budavari, 1989)

(*c*) *Melting-point*: 10.5 °C (Lide, 1993)

(*d*) *Density*: 1.254 at 20 °C/4 °C (Lide, 1993)

(*e*) *Spectroscopy data*: Infrared (prism [5864], grating [24020]), ultraviolet [1643], nuclear magnetic resonance (proton [1887], C-13 [931]) and mass spectral data have been reported (Sadtler Research Laboratories, 1980).

(*f*) *Solubility*: Moderately soluble in warm water (1.69 g/L at 30 °C; BUA, 1987); soluble in ethanol and diethyl ether (Budavari, 1989)

(*g*) *Volatility*: Vapour pressure, 4 Pa at 30 °C (BUA, 1987)

(*h*) *Stability*: Explosive reaction with sodium hydroxide and zinc (Sax & Lewis, 1989)

(*i*) *Octanol/water partition coefficient (P)*: log P, 1.73 (Hansch *et al.*, 1995)

(*j*) *Conversion factor*: $mg/m^3 = 6.26 \times ppm$[1]

1.1.4 *Technical products and impurities*

2-Nitroanisole is commercially available with a purity ranging from 98% to 99% (Aldrich Chemical Co., 1994; Fluka Chemical Corp., 1995).

1.1.5 *Analysis*

2-Nitroanisole in environmental samples can be analysed by gas chromatography with electron capture detection (Mitchell & Deveraux, 1978).

1.2 Production and use

1.2.1 *Production*

2-Nitroanisole is prepared by slowly adding methanolic sodium hydroxide to a solution of 2-chloronitrobenzene in methanol at 70 °C and then, to complete the reaction, by gradually heating the mixture under pressure to 95 °C. After dilution with water, the product is separated as an oil, at a 90% yield; methanol can be recovered from the aqueous layer (Booth, 1991; Lewis, 1993).

In western Europe in 1983, production of 2-nitroanisole was approximately 7200 tonnes per year, including approximately 4000 tonnes per year in Germany (BUA, 1987).

2-Nitroanisole is known to be produced by five companies in Japan, four in China and three in India and by one company each in Brazil, Germany and the Ukraine (Chemical Information Services, 1994).

1.2.2 *Use*

2-Nitroanisole is reduced (using a H_2-catalyst or iron-formic acid) to *ortho*-anisidine (see IARC, 1982, 1987a) or to *ortho*-dianisidine (see IARC, 1974, 1987b), both of which are important as dye intermediates. 2-Nitroanisole has also been used as an intermediate for various pharmaceuticals (Budavari, 1989; Booth, 1991; Lewis, 1993).

1.3 Occurrence

1.3.1 *Natural occurrence*

2-Nitroanisole is not known to occur as a natural product.

1.3.2 *Occupational exposure*

No information was available to the Working Group.

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[1]Calculated from: mg/m^3 = (relative molecular mass/24.45) × ppm, assuming temperature (25 °C) and pressure (101 kPa)

1.3.3 *Environmental occurrence*

(*a*) *Air*

On 22 February 1993, approximately 10 tonnes of vapour containing 2-nitroanisole and other halogenated aromatics were accidentally released from a chemical plant in Grieshem, Germany (Anon., 1993). No concentration of 2-nitroanisole in ambient air has been reported.

(*b*) *Water*

2-Nitroanisole has been detected in water samples in Japan (0.7 μg/L) and the Netherlands (0.3–1.0 μg/L). Also, nitroanisole of unspecified isomerism has been detected in the Netherlands (0.3–1.0 μg/L) and Germany (0.1–0.9 μg/L) (BUA, 1987).

(*c*) *Soil and sediment*

2-Nitroanisole has been detected in sediment samples taken in Japan (0.01 μg/L) (BUA, 1987).

1.4 Regulations and guidelines

The former USSR has set a short-term exposure limit for 2-nitroanisole of 1 mg/m^3, with skin absorption noted as a potentially significant route of exposure (effective date, 1989) (International Labour Office, 1991; United Nations Environmental Programme, 1995).

2. Studies of Cancer in Humans

No data were available to the Working Group.

3. Studies of Cancer in Experimental Animals

3.1 Oral administration

3.1.1 *Mouse*

Groups of 60 male and 60 female B6C3F1 mice, approximately 40 days of age, were administered 0, 666, 2000 or 6000 mg/kg diet (ppm) 2-nitroanisole (purity > 99%) for 103 weeks. Groups of nine or 10 mice of each sex and from each dose group were killed at 15 months for an interim evaluation. Mean body weights of high-dose male and female mice were 33 and 43% lower than those of controls, respectively, at the end of the study; mean body weights of mid-dose male and female mice were 11 and 18% lower than those of controls, respectively, at the end of the study. Survival at 103 weeks did not differ among treated males (35/50 in controls, 43/50 at the low dose, 39/50 at the mid dose, 40/50 at the high dose) but was increased among treated females (38/50 in controls, 26/50 at the low dose, 33/50 at the mid dose, 45/50 at the high dose) when compared with controls. In treated mice, the incidence of hepatocellular adenoma was increased in

both males and females (in males: 14/50 in controls, 26/50 at the low dose, 41/50 at the mid dose, 29/50 at the high dose, $p = 0.012$, logistic regression analysis for trend; in females: 14/50 in controls, 20/50 at the low dose, 36/50 at the mid dose, 18/50 at the high dose, $p < 0.001$ for the mid dose versus controls). The combined incidences of hepatocellular adenomas and carcinomas in males were 21/50 in controls, 32/50 at the low dose, 45/50 at the mid dose and 32/50 at the high dose ($p < 0.001$ for the mid dose versus controls); those in females were 17/50 in controls, 21/50 at the low dose, 37/50 at the mid dose and 20/50 at the high dose ($p < 0.001$ for the mid dose versus controls). The incidence of hepatoblastomas was also increased in male mice (0/50 in controls, 3/50 at the low dose, 17/50 at the mid dose, 9/50 at the high dose; $p < 0.001$). Other hepatic lesions of increased incidence in some or all dose groups included haemorrhage, Kupffer-cell pigmentation, eosinophilic focus, focal necrosis and cytological alteration (hepatocytic hypertrophy, nuclear enlargement and eosinophilic staining of cytoplasm) (United States National Toxicology Program, 1993).

3.1.2 *Rat*

Groups of 60 male and 60 female Fischer 344/N rats, approximately 40 days of age, were administered 0, 222, 666 or 2000 mg/kg diet (ppm) 2-nitroanisole (purity > 99%) for 103 weeks. Groups of nine or 10 rats of each sex and from each dose group were killed at 15 months for an interim evaluation. Body weights of treated rats were similar to those of controls. When compared with controls, survival at 103 weeks was decreased in treated males (32/50 in controls, 34/50 at the low dose, 24/50 at the mid dose, 9/50 at the high dose) but was unchanged in treated females (33/50 in controls, 41/50 at the low dose, 26/50 at the mid dose, 33/50 at the high dose). In treated rats, the incidence of mononuclear-cell leukaemia was increased in both males and females (males: 26/50 in controls, 25/50 at the low dose, 42/50 at the mid dose, 34/50 at the high dose, $p < 0.001$ life table trend test; females: 14/50 in controls, 11/50 at the low dose, 14/50 at the mid dose, 26/50 at the high dose, $p = 0.001$, life table trend test) (United States National Toxicology Program, 1993).

Groups of 60 male and 60 female Fischer 344/N rats, approximately 40 days of age, were administered 0, 6000 or 18 000 mg/kg diet (ppm) 2-nitroanisole (purity > 99%) for 27 weeks (see Table 1). Groups of 10 animals of each sex and from each dose group were killed at three, six, nine or 15 months for interim evaluation. The remaining animals were killed 77 weeks after cessation of treatment (after 104 weeks of study). The body weights of the treated rats were markedly lower than those of controls during the 27-week treatment period and did not recover after cessation of treatment. Survival at 104 weeks was decreased in treated males and females, with the respective Kaplan Meier probabilities of survival at the end of the study being 63% and 68% for control males and females, 4% and 23% for low-dose males and females, and 0% for both high-dose males and high-dose females. Increased incidences of tumours of the urinary bladder, the large intestine and the kidney occurred in treated males and females. The incidences of selected tumours at the interim kills are given in Table 1. The overall incidences of

Table 1. Incidence of selected tumours in rats fed 2-nitroanisole in the diet for 27 weeks

	Males			Females		
	0 ppm	6000 ppm	18 000 ppm	0 ppm	6000 ppm	18 000 ppm
Urinary bladder						
Transitional-cell carcinoma						
3-month interim	0/9	0/9	1/10	0/10	0/10	0/10
6-month interim	0/10	0/10	10/10*	0/10	0/10	10/10*
9-month interim	0/10	3/10	6/6*	0/10	1/9	6/6*
15-month interim	0/9	1/3	–	0/8	9/10*	–
Large intestine						
Carcinoma						
3-month interim	0/10	0/10	0/10	0/10	0/10	0/10
6-month interim	0/10	0/10	0/10	0/10	0/10	0/10
9-month interim	0/10	0/10	1/6	0/10	0/10	0/6
15-month interim	0/9	0/3	–	0/8	0/10	–
Kidney						
Transitional-cell carcinoma						
3-month interim	0/10	0/10	0/10	0/10	0/10	0/10
6-month interim	0/10	0/10	0/10	0/10	0/10	0/10
9-month interim	0/10	0/10	2/6	0/10	0/10	0/6
15-month interim	0/9	0/3	–	0/8	0/10	–

From United States National Toxicology Program (1993)
*, $p < 0.01$, Fisher's exact test

urinary bladder neoplasms were as follows: transitional-cell papilloma — males: 0/59 in controls, 9/59 at the low dose ($p < 0.01$, Fisher's exact test) and 1/60 at the high dose; females: 0/58 in controls, 2/59 at the low dose and 1/60 at the high dose; transitional-cell carcinoma — males: 0/59 in controls, 27/59 at the low dose ($p < 0.01$) and 50/60 at the high dose ($p < 0.01$); females: 0/58 in controls, 28/59 at the low dose ($p < 0.01$) and 48/60 at the high dose ($p < 0.01$). A few squamous-cell papillomas and carcinomas of the urinary bladder also occurred in high-dose males and females, and urinary bladder sarcomas occurred with overall incidences of 0/59, 2/59 and 9/59 ($p < 0.01$) in control, low-dose and high-dose males and 0/58, 2/59 and 14/60 ($p < 0.01$) in control, low-dose and high-dose females, respectively. Squamous metaplasias and connective tissue proliferation also occurred with increased frequency in the urinary bladders of treated males and females. The overall incidences of adenomatous polyps of the large intestine were 0/60, 26/60 ($p < 0.01$) and 30/60 ($p < 0.01$) in control, low-dose and high-dose males and 0/60, 8/60 ($p < 0.01$) and 18/60 ($p < 0.01$) in control, low-dose and high-dose females, respectively. Carcinomas of the large intestine occurred in 5/60 ($p < 0.05$) high-dose males and 2/60 high-dose females. The overall incidence of transitional-cell tumours of the kidney

was also increased in treated males and females compared to controls. No tumour of this type was found in male or female controls; transitional-cell carcinomas were found in 1/60 low-dose males and 8/60 ($p < 0.01$) high-dose males; 4/60 high-dose males had a transitional-cell papilloma. Of high-dose female rats, 1/60 had a transitional-cell papilloma and 1/60 had a transitional-cell carcinoma. The incidence of hyperplasia of the transitional epithelium was significantly increased in all treated males and females. The severity of nephropathy was increased in treated males at three and six months (United States National Toxicology Program, 1993).

3.2 Carcinogenicity of metabolites

One of the metabolites of 2-nitroanisole, *ortho*-anisidine, when tested as the hydrochloride by oral administration in the diet, was found to produce transitional-cell tumours of the urinary bladder in mice and rats and transitional-cell carcinomas of the renal pelvis in rats (IARC, 1982).

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

4.1 Absorption, distribution, metabolism and excretion

4.1.1 *Humans*

No data were available to the Working Group.

4.1.2 *Experimental systems*

The pharmacokinetics and metabolism of 2-nitroanisole were studied in male Fischer 344 rats by Miller *et al.* (1985). Three dose levels of [^{14}C]2-nitroanisole (5, 50 or 500 mg/kg bw) were administered orally to rats, and daily excreta were analysed for ^{14}C. 2-Nitroanisole was readily absorbed from the stomach at the 50-mg/kg bw dose, as less than 10% of the initial dose remained in the stomach at 6 h. At the dose of 500 mg/kg bw, 36% of the initial dose remained in the stomach at 6 h. Peak blood levels reflect the dose-dependence of absorption, with parent 2-nitroanisole reaching maximal concentrations at 3 h (0.9% of dose) after a 50-mg/kg bw dose and at 6 h (0.9% of dose) after a 500-mg/kg bw dose. Within seven days, 7% of the dose had been excreted in the faeces for all dose levels and about 70% of the dose had been eliminated in the urine. For doses of 50 and 500 mg/kg bw, the urinary metabolite profiles at 8 h were not substantially different. The predominant route of elimination was through metabolism to 2-nitrophenol (5–8% of urinary radioactivity), subsequent sulfation to 2-nitrophenyl sulfate (64–68% of urinary radioactivity) and glucuronidation to 2-nitrophenyl glucuronide (13–15% of urinary radioactivity). Seven days after oral administration of 2-nitroanisole, less than 0.5% of the administered dose remained in the carcass.

Since the two highest oral doses saturated the urinary excretion rate of 2-nitroanisole, an intravenous dose of 25 mg/kg bw was used for pharmacokinetic studies. Following a 25 mg/kg bw intravenous injection of [^{14}C]2-nitroanisole, blood, tissues and excreta were collected at times ranging from 15 min to seven days. The distribution of 2-nitroanisole-derived ^{14}C to tissues (muscle, 20%; skin, 10%; fat, 6.8%; blood, 6.5%; liver, 4.8%; plasma, 3.1%; kidney, 2.8%; and small intestine, 1.9%) occurred rapidly following administration. Peak tissue concentrations were reached in all tissues within 15 min. Urinary and faecal elimination were similar to that found after oral administration (urine, 86% by seven days; faeces, 9% by seven days). The subsequent elimination of ^{14}C was rapid and biphasic. The initial elimination phase in all tissues had a half-life of 1–2 h, and the terminal phase half-lives for all tissues ranged from 2.5 to 6.2 days. Elimination of parent 2-nitroanisole from the blood was biphasic with initial and terminal half-lives of 30 min and 2.2 h, respectively. Monophasic elimination of 2-nitroanisole from the liver, kidneys and small intestine occurred with half-lives of 0.35, 0.55, and 0.68 h, respectively. Biliary excretion was similar to faecal elimination, indicating a lack of enterohepatic recirculation. Urine collected for 24 h after intraperitoneal administration of 25 mg/kg bw 2-nitroanisole had a profile similar to that observed after oral administration (63% 2-nitrophenyl sulfate, 11% 2-nitrophenyl glucuronide, 1.5% 2-nitrophenol and 0.6% *ortho*-anisidine) (Miller *et al.*, 1985).

In a study conducted by Yuan *et al.* (1991), male Fischer 344 rats were fed freshly prepared NIH-07 feed or NIOH-07 feed stored for 30 days containing 0.25 mg/g feed 2-nitroanisole. The animals were dosed daily for 3 h for seven consecutive days; on day 7, they were placed in metabolism cages and their urine was collected for 18 h. After the urine collection, the rats were fed control diet for three days. The treated groups then exchanged regimens and were treated for an additional seven days following the same dosing schedule. Again, an 18-h urine sample was collected from each rat and analysed for total 2-nitrophenol. Extraction and analysis indicated that stored feed bound 2-nitroanisole more tightly than freshly prepared feed. Eighteen-hour urine samples collected on day 7 indicated that this binding did not affect systemic bioavailability. Approximately 2.5 mg 2-nitrophenol were excreted in the urine during the 18-h collection period. Total 2-nitrophenol (2-nitrophenol and its conjugates) found in urine decreased in the second week of 2-nitroanisole feeding despite increased feed consumption. Thus, the metabolism of 2-nitroanisole may be affected by continuous exposure.

4.2 Toxic effects

4.2.1 *Humans*

No data were available to the Working Group.

4.2.2 *Experimental systems*

(*a*) *Single-dose studies*

The oral LD_{50} of 2-nitroanisole is 740 mg/kg bw in rats and 1300 mg/kg bw in mice (United States National Institute for Occupational Safety and Health, 1994).

(b) Repeated-dose studies

The United States National Toxicology Program (1993) reported toxic effects after dietary administration of 2-nitroanisole to male and female Fischer 344 rats (583, 1166, 2332, 4665 or 9330 ppm) and B6C3F1 mice (250, 500, 1000, 2000 or 4000 ppm) for 14 days. Mean body-weight gains were depressed in male rats fed 4665 or 9330 ppm, in male mice fed 250 ppm or more and in female mice fed 4000 ppm. Absolute liver weight was increased in male rats fed 1166 ppm and in female rats fed 583 ppm or more. Erythrocyte counts, haematocrit values and haemoglobin concentrations in all exposed male rats were significantly lower than those in controls. Methaemoglobin concentrations were significantly increased in male rats fed 1166 ppm or more. With the exception of depressed body-weight gain, no treatment-related effect was observed in mice.

In 13-week studies, male and female B6C3F1 mice and Fischer 344 rats were fed with diets including 60 (mice only), 200, 600, 2000, 6000 or 18 000 (rats only) ppm 2-nitroanisole (United States National Toxicology Program, 1993). Male and female rats receiving diets containing 6000 or 18 000 ppm and mice receiving diets containing 6000 ppm 2-nitroanisole exhibited lower mean body weights than the controls. Lower haemoglobin and haematocrit values were observed in male and female rats receiving 2000, 6000 or 18 000 ppm and male and female mice receiving 2000 or 6000 ppm 2-nitroanisole. Methaemoglobin increases were observed in male and female rats (6000 and 18 000 ppm) and male mice (6000 ppm). In rats, the principal lesions observed were in the urinary bladder (hyperplasia, 6000 and 18 000 ppm), spleen (congestion, 6000 and 18 000 ppm), kidney (renal tubule necrosis, 600–6000 ppm) and liver (hepatocytic hypertrophy, 18 000 ppm). In male mice, hepatocytic hypertrophy was observed at 200 ppm and above.

4.3 Reproductive and developmental effects

No data were available to the Working Group.

4.4 Genetic and related effects

4.4.1 *Humans*

No data were available to the Working Group.

4.4.2 *Experimental systems* (see also Table 2 and Appendices 1 and 2)

2-Nitroanisole was positive in the *rec* assay in *Bacillus subtilis* strains H17 and M45.

2-Nitroanisole was tested in several laboratories for the induction of gene mutations in *Salmonella typhimurium*. Positive responses were obtained consistently with the strain TA100. Variable responses were obtained with some other strains.

In single studies with cultured mammalian cells, 2-nitroanisole induced sister chromatid exchange and chromosomal aberrations in Chinese hamster ovary (CHO) cells and mutation at the *tk* locus of mouse lymphoma L5178Y cells. The clastogenic activity was weak and observed only in the presence of S9, whereas sister chromatid exchange and *tk* mutations were induced in the absence of S9.

Table 2. Genetic and related effects of 2-nitroanisole

Test system	Result[a]		Dose[b] (LED/HID)	Reference
	Without exogenous metabolic system	With exogenous metabolic system		
BSD, *Bacillus subtilis rec* H97 and M45 strains, differential toxicity	+	0	625	Shimizu & Yano (1986)
SA0, *Salmonella typhimurium* TA100, reverse mutation	(+)	0	1530	Chiu *et al.* (1978)
SA0, *Salmonella typhimurium* TA100, reverse mutation	+	+	256	Haworth *et al.* (1983)
SA0, *Salmonella typhimurium* TA100, reverse mutation	+	0	480	Shimizu & Yano (1986)
SA0, *Salmonella typhimurium* TA100, reverse mutation	0	+	580	Dellarco & Prival (1989)
SA0, *Salmonella typhimurium* TA100, reverse mutation	+	+	128	US National Toxicology Program (1993)
SA5, *Salmonella typhimurium* TA1535, reverse mutation	–	–	385	Haworth *et al.* (1983)
SA5, *Salmonella typhimurium* TA1535, reverse mutation	–	0	2400	Shimizu & Yano (1986)
SA5, *Salmonella typhimurium* TA1535, reverse mutation	(+)	–	1280	US National Toxicology Program (1993)
SA7, *Salmonella typhimurium* TA1537, reverse mutation	–	–	385	Haworth *et al.* (1983)
SA7, *Salmonella typhimurium* TA1537, reverse mutation	–	0	2400	Shimizu & Yano (1986)
SA8, *Salmonella typhimurium* TA1538, reverse mutation	+	0	480	Shimizu & Yano (1986)
SA9, *Salmonella typhimurium* TA98, reverse mutation	(+)	0	765	Chiu *et al.* (1978)
SA9, *Salmonella typhimurium* TA98, reverse mutation	–	–	385	Haworth *et al.* (1983)
SA9, *Salmonella typhimurium* TA98, reverse mutation	+	0	480	Shimizu & Yano (1986)
SA9, *Salmonella typhimurium* TA98, reverse mutation	–	–	385	US National Toxicology Program (1993)
SAS, *Salmonella typhimurium* TA97, reverse mutation	–	–	1280	US National Toxicology Program (1993)
G5T, Gene mutation, mouse lymphoma L5178Y cells *in vitro*	+	0	250	US National Toxicology Program (1993)
SIC, Sister chromatid exchange, Chinese hamster cells *in vitro*	+	+	123	Galloway *et al.* (1987)
CIC, Chromosomal aberrations, Chinese hamster cells *in vitro*	–	(+)	1060	Galloway *et al.* (1987)

[a] +, positive; (+), weak positive; –, negative; 0, not tested; ?, inconclusive

[b] LED, lowest effective dose; HID, highest ineffective dose. In-vitro tests, µg/mL; in-vivo tests, mg/kg bw

5. Summary of Data Reported and Evaluation

5.1 Exposure data

2-Nitroanisole is produced by the reaction of methanolic sodium hydroxide with 2-chloronitrobenzene. It is mainly used in the production of the dye intermediates *ortho*-anisidine and *ortho*-dianisidine. Human exposure may occur during its production and use.

5.2 Human carcinogenicity data

No data on the carcinogenicity of 2-nitrosanisole in humans were available to the Working Group.

5.3 Animal carcinogenicity studies

2-Nitroanisole was tested for carcinogenicity by oral administration in one study in mice and in two studies in rats. In mice, the incidence of hepatocellular adenomas was increased in males and females, and that of hepatoblastomas was increased in males. In one study in rats, the incidence of mononuclear-cell leukaemia was increased in males and females. In the second study, which used a shorter duration of treatment but higher doses, increases were seen in the incidences of tumours of the urinary bladder, the large intestine and the kidney.

5.4 Other relevant data

No human data were available on the metabolism of 2-nitroanisole.

In rats, 2-nitroanisole is absorbed after oral administration, and the major route of its rapid elimination is the urine. The predominant metabolic pathway involves the formation of 2-nitrophenol, with its subsequent conjugation with sulfate and glucuronic acid. 2-Nitroanisole causes methaemoglobinaemia following dietary administration of high doses to rats and mice. Pathological lesions observed in rats occurred in the urinary bladder, spleen, kidney and liver. In mice, 2-nitroanisole causes hypertrophy in the liver.

2-Nitroanisole is mutagenic in bacteria. In single studies, it induced mutations, sister chromatid exchange and a low frequency of chromosomal aberrations in cultured mammalian cells.

5.5 Evaluation[1]

There is *inadequate evidence* in humans for the carcinogenicity of 2-nitroanisole.

There is *sufficient evidence* in experimental animals for the carcinogenicity of 2-nitroanisole.

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[1]For definition of the italicized terms, see Preamble, pp. 24–27.

Overall evaluation

2-Nitroanisole is *possibly carcinogenic to humans (Group 2B).*

6. References

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