CI ACID RED 114

1. Exposure Data

1.1 Chemical and physical data

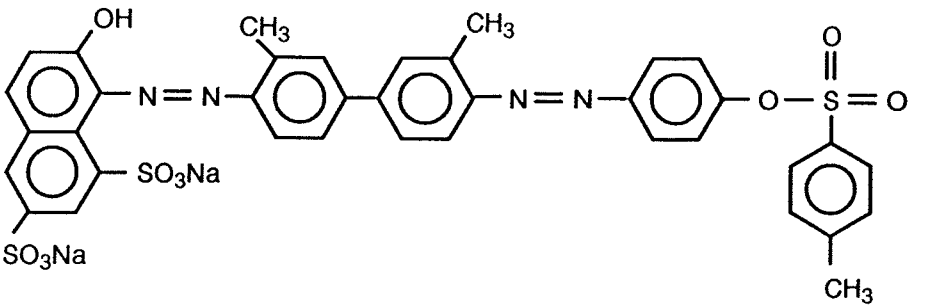
1.1.1 *Synonyms, structural and molecular data*

Chem. Abstr. Serv. Reg. No.: 6459-94-5

Chem. Abstr. Name: 8-[(3,3′-Dimethyl-4′-[(4-[([4-methylphenyl]sulfonyl)oxy]phenyl)-azo][1,1′-biphenyl]-4-yl)azo]-7-hydroxy-1,3-naphthalenedisulfonic acid, disodium salt

Colour Index No.: 23635

Synonyms: Acid Red 114; CI Acid Red 114, disodium salt



$C_{37}H_{28}N_4O_{10}S_3.2Na$ Mol. wt: 830.84

1.1.2 *Chemical and physical properties*

(*a*) *Description*: Deep-maroon powder (Green, 1990); red powder (US National Toxicology Program, 1991)

(*b*) *Melting-point*: 250–300 °C (decomposes) (US National Toxicology Program, 1991)

(*c*) *Spectroscopy data*: Infrared, ultraviolet and nuclear magnetic resonance spectral data have been reported (Pouchert, 1981; Sadtler Research Laboratories, 1988; Green, 1990; US National Toxicology Program, 1991).

(*d*) *Solubility*: Soluble in water (80 g/l at 80 °C) (International Dyestuffs Corp., 1990); very slightly soluble in ethanol (Green, 1990)

1.1.3 *Trade names, technical products and impurities*

Some trade names are: Acid Leather Red BG; Amacid Milling Red PRS; Benzyl Fast Red BG; Benzyl Red BR; Elcacid Milling Fast Red RS; Erionyl Red RS; Fenafor Red PB; Folan Red B; Intrazone Red BR; Kayanol Milling Red RS; Leather Fast Red B; Levanol Red

GG; Midlon Red PRS; Milling Fast Red B; Milling Red B; Milling Red BB; Milling Red SWB; Polar Red RS; Sandolan Red N-RS; Sella Fast Red RS; Sulphonol Red R; Suminol Milling Red RS; Supranol Fast Red 3G; Supranol Fast Red GG; Supranol Red PBX-CF; Supranol Red R; Telon Fast Red GG; Tertracid Milling Red B; Vondamol Fast Red RS.

Technical-grade CI Acid Red 114 is available in commercial mixtures containing 25–85% pure dye. Other typical ingredients include sodium chloride and mineral oil (see IARC, 1984, 1987) (Crompton & Knowles Corp., 1990; International Dyestuffs Corp., 1990; US National Toxicology Program, 1991; Aldrich Chemical Co., 1992).

1.1.4 *Analysis*

No data were available to the Working Group.

1.2 Production and use

1.2.1 *Production*

CI Acid Red 114, a bright red anionic bisazo dye, is manufactured by converting 3,3′-dimethylbenzidine (*ortho*-tolidine; see IARC, 1972) to the tetraazonium salt, which is then coupled successively to G acid (2-naphthol-6,8-disulfonic acid) and phenol. The phenol hydroxy function is then esterified with *para*-tolylsulfonyl chloride (Green, 1990).

CI Acid Red 114 has been produced commercially since the early 1900s. In the USA, there were six manufacturers and two importers of CI Acid Red 114 in 1977 (US Environmental Protection Agency, 1988). Annual production volume by five manufacturers in 1990 was estimated to be 10–100 tonnes, whereas it was 170 tonnes in 1979 (US International Trade Commission, 1980). In 1980, the USA imported about 7 tonnes of CI Acid Red 114 (US International Trade Commission, 1981).

1.2.2 *Use*

CI Acid Red 114 is used to dye wool (from a weak acid bath), silk (from either a neutral or acetic acid bath), jute and leather. Wool and silk are also printed directly (Green, 1990).

1.3 Occurrence

1.3.1 *Natural occurrence*

CI Acid Red 114 is not known to occur as a natural product.

1.3.2 *Occupational exposure*

No data were available to the Working Group.

The US Environmental Protection Agency, the American Textile Manufacturers Institute and the Ecological and Toxicological Association of the Dyestuffs Manufacturing Industry conducted a joint survey in 1986–87 to estimate airborne concentrations of dye dust in dye weighing rooms of plants where powder dyes are used in the dyeing and printing of

textiles. The survey was based on a sample of 24 sites chosen at random from among textile plants where powder dyes are weighed. Although CI Acid Red 114 was not among the dyes included in the survey, the results are considered to be representative of dye dust levels during weighing of this type of powder dye. The mean airborne concentration of total active colourant in the plants monitored was estimated to be 0.085 mg/m^3 (US Environmental Protection Agency, 1990).

On the basis of a survey conducted in the USA between 1981 and 1983, the US National Institute for Occupational Safety and Health estimated that a total of 18 511 workers, including 352 women, may have been exposed to CI Acid Red 114 at 300 textile and leather goods manufacturing plants (US National Library of Medicine, 1992).

1.3.3 *Other*

Anaerobic biodegradation of CI Acid Red 114 gives rise to the amine metabolites, 3,3′-dimethylbenzidine and 4-methylbenzenesulfonic acid (4′-aminophenyl) ester. Following incubation of 100 mg/l of dyestuff at 35 °C in the presence of anaerobic sludge inoculum, primary degradation was complete within seven days (Brown & Hamburger, 1987).

1.4 Regulations and guidelines

In Germany, CI Acid Red 114 must be handled like the corresponding hypothetical reduction amine, 3,3′-dimethylbenzidine, which is classified as an A_2 compound. Such materials are considered to have been proven to be carcinogenic only in animal experimentation but under conditions comparable to those of possible human exposure at the workplace (Deutsche Forschungsgemeinschaft, 1992).

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

Rat

Groups of 50, 35, 65 and 50 male and 50, 35, 65 and 50 female Fischer 344/N rats, five weeks old, were administered 0, 70, 150 or 300 mg/l (ppm) (males) and 0, 150, 300 or 600 ppm (females) CI Acid Red 114 (purity, 85%; with about 15 organic chemicals of similar structure, including approximately 5 ppm 3,3′-dimethylbenzidine and < 1 ppm benzidine in distilled drinking-water for 104 weeks. Survival at 105 weeks was 24/50, 15/35, 26/65 and 1/50 for male rats and 36/50, 13/35, 6/64 and 0/50 for females in the control, low-, mid- and high-dose groups, respectively ($p < 0.001$ for both males and females). All female rats receiving 600 ppm died by week 89. The decreased survival in the treated groups was due to development of treatment-related neoplasms. As shown in Table 1, there were increased

incidences of benign and malignant tumours of the skin, Zymbal gland and liver in male and female rats, and of the clitoral gland, oral cavity, small and large intestine and lung in female rats (US National Toxicology Program, 1991).

Table 1. Survival and tumour incidences in male and female Fischer 344/N rats administered CI Acid Red 114 in the drinking-water for 104 weeks

Survival and tumour types[a]	Dose (mg/l [ppm])				*p* Value[b]
Males	0	70	150	300	
Females	0	150	300	600	
Males					
Survival[c]	24/50	15/35	26/65	1/50	
Skin					
Basal-cell adenoma or carcinoma	1/50	5/35	28/65	32/50	< 0.001
Sebaceous-cell adenoma or carcinoma	1/50	1/35	5/65	6/50	= 0.007
Squamous-cell papilloma or carcinoma	1/50	2/35	11/65	9/50	= 0.001
Keratoacanthoma	1/50	1/35	4/65	7/50	< 0.001
Zymbal gland adenoma or carcinoma	0/50	0/35	8/65	7/50	= 0.005
Liver neoplasms	2/50	2/35	15/65	20/50	< 0.001
Females					
Survival	36/50	13/35	6/64	0/50	
Basal-cell adenoma or carcinoma of the skin	0/50	4/35	7/65	5/50	= 0.012
Zymbal gland adenoma or carcinoma	0/50	3/35	18/65	19/50	< 0.001
Clitoral gland adenoma or carcinoma	11/48	17/32	28/62	23/50	< 0.001
Liver neoplasms	0/50	0/35	19/64	8/50	< 0.001
Lung adenoma or carcinoma	1/50	2/35	9/65	4/50	= 0.007
Oral cavity squamous-cell papilloma or carcinoma	0/50	3/35	9/65	6/50	= 0.017
Small intestine polyps or adenocarcinoma	0/50	0/35	1/63	2/50	> 0.05
Large intestine polyps or adenocarcinoma	0/50	1/35	0/64	3/50	> 0.05

From US National Toxicology Program (1991)
[a]Terms used by authors
[b]Logistic regression test for trend
[c]At 22 months; reduced survival in exposed groups due to tumour development

4. Other Relevant Data

4.1 Absorption, distribution, metabolism and excretion

4.1.1 *Humans*

No data were available to the Working Group.

4.1.2 *Experimental systems*

CI Acid Red 114 (100 mg/kg) containing less than 1% 3,3′-dimethylbenzidine as an impurity was administered once in the diet to two female mongrel dogs weighing 15 kg, and

48-h urine was analysed for 3,3′-dimethylbenzidine, the potential metabolic product (Lynn *et al.*, 1980). Excretion was found to be 0.04% of the dose of dye administered, which is in excess of what would be expected from the level of impurity; *para*-aminophenyl-*para*-toluenesulfonate was also identified as a urinary metabolite. The same dose of CI Acid Red 114 was also administered once to four male Sprague-Dawley rats by intragastric intubation; after 72 h, only 0.01% of the dose could be identified as 3,3′-dimethylbenzidine.

4.2 Toxic effects

4.2.1 *Humans*

No data were available to the Working Group.

4.2.2 *Experimental animals*

CI Acid Red 114 was tested for toxicity in male and female Fischer 344/N rats in a range-finding study for carcinogenicity testing (US National Toxicology Program, 1991). The purity of the test compound was estimated to be 82–85%; impurities consisted of about 15 organic chemicals of similar structure, with benzidine at less than 1 ppm and 3,3′-dimethylbenzidine at about 5 ppm. In a 13-day study, groups of five rats were dosed with 0, 10 000, 20 000 or 30 000 ppm (mg/l) in drinking-water. Except for one accidental death, all rats survived to the end of the study. Final mean body weights were significantly lower for males in the mid- and high-dose groups (83 and 77%, respectively) and for females in all dose groups (92, 88 and 80%, respectively). Hypocellularity of sternal bone marrow was found in three males and in all females given 20 000 ppm. The marrow was depleted of erythroid and myeloid cells. Lymphocytic depletion of the thymus was observed in four males and one female of the same dose group.

In the 13-week study, groups of 10 rats received 0, 600, 1200, 2500, 5000 or 10 000 ppm (mg/l) CI Acid Red 114 in the drinking-water for 94 days (males) or 95 days (females) (US National Toxicology Program, 1991). All rats survived. Body weights were lower than those in controls in all groups that received 1200 ppm and above (94–85%). Relative liver weights were increased in all dosed males and females; absolute and relative kidney weights were increased in females receiving doses of 1200 ppm and above. Haematocrit, haemoglobin and erythrocyte counts were decreased in dosed females, and the erythrocyte count was reduced at 1500 ppm and above in males. Some enzyme levels were elevated, consistent with mild hepatocellular damage, and minimal-to-mild lesions in liver were seen upon histopathological examination. Kupffer cells in the livers of most treated females contained brown pigment. An increased prevalence of reticulum-cell hyperplasia of the mesenteric lymph node was observed in treated males and females. Tubular regeneration and chronic inflammation of the kidneys occurred more frequently in treated females than in controls. Minimal amounts of brownish pigment were also seen in the tubular epithelial cells of the kidneys.

4.3 Reproductive and prenatal 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)

CI Acid Red 114 was mutagenic to *Salmonella typhimurium* strains TA1538 and TA98 under reducing conditions. It did not induce sex-linked recessive lethal mutation in *Drosophila melanogaster* and did not induce unscheduled DNA synthesis in primary cultures of rat hepatocytes (abstract) or sister chromatid exchange or chromosomal aberrations in cultured Chinese hamster ovary cells. It did not induce unscheduled DNA synthesis in the hepatocytes of rats dosed orally (abstract).

Activated *ras* genes were found in 13/16 tumours induced in rats by CI Acid Red 114 (US National Toxicology Program, 1991) and in 1/38 spontaneous tumours tested (a CGA in codon 61 in a clitoral gland adenoma) (Reynolds *et al.*, 1990; Table 3).

5. Summary of Data Reported and Evaluation

5.1 Exposure data

CI Acid Red 114, a bis-azo dye derived from 3,3′-dimethylbenzidine, is used to dye wool, silk, jute and leather.

5.2 Human carcinogenicity data

No data were available to the Working Group.

5.3 Animal carcinogenicity data

CI Acid Red 114 was tested for carcinogenicity in one study in rats by administration in the drinking-water. It increased the incidences of benign and malignant tumours of the skin, Zymbal gland and liver in male and female rats, and of the clitoral gland, lung, oral cavity and small and large intestine in female rats.

5.4 Other relevant data

Reductive cleavage of the azo bonds to yield 3,3′-dimethylbenzidine was demonstrated *in vivo*.

CI Acid Red 114 induced gene mutation in bacteria under reducing conditions. It did not induce gene mutation in insects or sister chromatid exchange or chromosomal aberrations in cultured mammalian cells.

Table 2. Genetic and related effects of CI Acid Red 114

Test system	Result		Dose (LED/HID)[a]	Reference
	Without exogenous metabolic system	With exogenous metabolic system		
SA0, *Salmonella typhimurium* TA100, reverse mutation	–	–	500.0000	Venturini & Tamaro (1979)
SA0, *Salmonella typhimurium* TA100, reverse mutation	0	–[b]	200.0000	Elliot & Gregory (1980)
SA0, *Salmonella typhimurium* TA100, reverse mutation	0	+[c]	62.5000	Elliot & Gregory (1980)
SA0, *Salmonella typhimurium* TA100, reverse mutation	–	–	167.0000	Mortelmans *et al.* (1986)
SA5, *Salmonella typhimurium* TA1535, reverse mutation	–	–	500.0000	Venturini & Tamaro (1979)
SA5, *Salmonella typhimurium* TA1535, reverse mutation	–	–	167.0000	Mortelmans *et al.* (1986)
SA7, *Salmonella typhimurium* TA1537, reverse mutation	–	–	167.0000	Mortelmans *et al.* (1986)
SA8, *Salmonella typhimurium* TA1538, reverse mutation	–	–	500.0000	Venturini & Tamaro (1979)
SA8, *Salmonella typhimurium* TA1538, reverse mutation	–	+[d]	100.0000	Reid *et al.* (1984)
SA9, *Salmonella typhimurium* TA98, reverse mutation	–	–	500.0000	Venturini & Tamaro (1979)
SA9, *Salmonella typhimurium* TA98, reverse mutation	0	–[b,e]	500.0000	Elliot & Gregory (1980)
SA9, *Salmonella typhimurium* TA98, reverse mutation	0	+[c]	62.5000	Elliot & Gregory (1980)
SA9, *Salmonella typhimurium* TA98, reverse mutation	0	+[f]	40.0000	Prival *et al.* (1984)
SA9, *Salmonella typhimurium* TA98, reverse mutation	–	(+)[g]	500.0000	Mortelmans *et al.* (1986)
SA9, *Salmonella typhimurium* TA98, reverse mutation	0	+[e]	125.0000	Dellarco & Prival (1989)
DMX, *Drosophila melanogaster*, sex-linked recessive lethal mutations	–		1500.0000	Woodruff *et al.* (1985)
DMX, *Drosophila melanogaster*, sex-linked recessive lethal mutations	–		50000.0000 feed	Woodruff *et al.* (1985)
URP, Unscheduled DNA synthesis, rat primary hepatocytes *in vitro*	–	0	0.0000	Mirsalis *et al.* (1983); abstr.
SIC, Sister chromatid exchange, Chinese hamster ovary cells *in vitro*	–	–	50.0000	US National Toxicology Program (1991)

Table 2 (contd)

Test system	Result		Dose (LED/HID)[a]	Reference
	Without exogenous metabolic system	With exogenous metabolic system		
CIC, Chromosomal aberrations, Chinese hamster ovary cells *in vitro*	–	–	50.0000	US National Toxicology Program (1991)
UPR, Unscheduled DNA synthesis, rat hepatocytes *in vivo*	–		0.0000 po	Mirsalis *et al.* (1983); abstr.

+, positive; (+), weakly positive; –, negative; 0, not tested
[a]In-vitro tests, μg/ml; in-vivo tests, mg/kg bw
[b]Anaerobic preincubation or with riboflavin supplementation
[c]Plate incorporation, with reduction using sodium dithionate
[d]Bacterial caecal reduction
[e]Plate incorporation with or without riboflavin
[f]Preincubation with hamster or rat liver S9 and flavin mononucleotide supplementation, no aeration
[g]Hamster liver S9

Table 3. Activating *ras* mutations in tumours induced in Fischer 344 rats by CI Acid Red 114 and in untreated animals

Tumour type	Frequency	N-*ras*	H-*ras*							
			Total	Codon 12		Codon 13		Codon 61		
				GAA	AGA	CGC	GTC	AAA	CTA	CGA
Treated										
Clitoral gland adenoma	4/4		4			1		1	1	1
Basal-cell adenoma (skin)	4/5	1	3	2	1					
Basal-cell carcinoma (skin)	1/1		1		1					
Squamous-cell carcinoma (skin)	3/3		3					2	1	
Trichoepithelioma (skin)	1/1		1		1					
Fibrosarcoma	0/1									
Mammary fibroadenoma	0/1									
Untreated										
Clitoral gland adenoma	1/2		1							1
Preputial gland carcinoma	0/1									
Mammary gland fibroadenoma or adenoma	0/11									
Mammary adenocarcinoma	0/2									
Subcutaneous fibroma or fibroadenoma	0/5									
Lipoma	0/1									
Testicular interstitial-cell adenoma	0/5									
Fibrosarcoma	0/2									
Mononuclear-cell leukaemia	0/3									
Adrenal phaeochromocytoma	0/1									
Pancreatic acinar adenoma	0/1									
Pancreatic islet-cell adenoma	0/1									
Pituitary adenoma	0/1									
Splenic haemangiosarcoma	0/1									
Prostatic adenocarcinoma	0/1									

Adapted from Reynolds *et al.* (1990)

5.5 Evaluation[1]

There is *inadequate evidence* in humans for the carcinogenicity of CI Acid Red 114.

There is *sufficient evidence* in experimental animals for the carcinogenicity of CI Acid Red 114.

[1]For definition of the italicized terms, see Preamble, pp. 26–30.

Overall evaluation

CI Acid Red 114 is *possibly carcinogenic to humans (Group 2B).*

6. References

Aldrich Chemical Co. (1992) *Aldrich Catalog/Handbook of Fine Chemicals 1992–1993*, Milwaukee, WI, p. 25

Brown, D. & Hamburger, B. (1987) The degradation of dyestuffs: Part III. Investigations of their ultimate degradability. *Chemosphere*, **16**, 1539–1553

Crompton & Knowles Corp. (1990) *Material Safety Data Sheet: Intrazone Red BR (CI Acid Red 114)*, Reading, PA

Dellarco, V.L. & Prival, M.J. (1989) Mutagenicity of nitro compounds in *Salmonella typhimurium* in the presence of flavin mononucleotide in a preincubation asssay. *Environ. Mutag.*, **13**, 116–127

Deutsche Forschungsgemeinschaft (1992) *MAK- and BAT-Values List 1992. Maximum Concentrations at the Workplace (MAK) and Biological Tolerance Values (BAT) for Working Materials* (Report No. 28), Weinheim, VCH Verlagsgesellschaft, pp. 64, 80–81

Elliot, J. & Gregory, A.R. (1980) Mutagenicity of a series of benzidine congener based dyes. *Vet. hum. Toxicol.*, **22**, 413–417

Green, F.J. (1990) *The Sigma-Aldrich Handbook of Stains, Dyes and Indicators*, Milwaukee, WI, Aldrich Chemical Co., p. 38

IARC (1972) *IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man*, Vol. 1, *Some Inorganic Substances, Chlorinated Hydrocarbons, Aromatic Amines,* N-*Nitroso Compounds, and Natural Products*, Lyon, pp. 87–91

IARC (1984) *IARC Monographs on the Evaluation of the Carcinogenic Risks of Chemicals to Humans*, Vol. 33, *Polynuclear Aromatic Compounds, Part 2: Carbon Blacks, Mineral Oils and Some Nitroarenes*, Lyon, pp. 87–168

IARC (1987) *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*, Suppl. 7, *Overall Evaluations of Carcinogenicity: An Updating of* IARC Monographs *Volumes 1 to 42*, Lyon, pp. 252–254

International Dyestuffs Corp. (1990) *Material Safety Data Sheet: Elcacid Milling Fast Red RS (Acid Red 114)*, Clifton, NJ

Lynn, R.K., Donielson, D.W., Ilias, A.M., Kennish, J.M., Wong, K. & Matthews, H.B. (1980) Metabolism of bisazobiphenyl dyes derived from benzidine, 3,3′-dimethylbenzidine or 3,3′-dimethoxybenzidine to carcinogenic aromatic amines in the dog and rat. *Toxicol. appl. Pharmacol.*, **56**, 248–258

Mirsalis, J., Tyson, K., Beck, J., Loh, F., Steinmetz, K., Contreras, C., Austere, L., Martin, S. & Spalding, J. (1983) Induction of unscheduled DNA synthesis (UDS) in hepatocytes following *in vitro* and *in vivo* treatment (Abstract No. Ef-5). *Environ. Mutag.*, **5**, 482

Mortelmans, K., Haworth, S., Lawlor, T., Speck, W., Tainer, B. & Zeiger, E. (1986) *Salmonella* mutagenicity tests. II. Results from the testing of 270 chemicals. *Environ. Mutag.*, **8** (Suppl. 7), 1–119

Pouchert, C.J., ed. (1981) *The Aldrich Library of Infrared Spectra*, 3rd Ed., Milwaukee, WI, Aldrich Chemical Co., p. 1465

Prival, M.J., Bell, S.J., Mitchell, V.D., Peiperl, M.D. & Vaughan, V.L. (1984) Mutagenicity of benzidine and benzidine-congener dyes and selected monoazo dyes in a modified *Salmonella* assay. *Mutat. Res.*, **136**, 33–47

Reid, T.M., Morton, K.C., Wang, C.Y. & King, C.M. (1984) Mutagenicity of azo dyes following metabolism by different reductive/oxidative systems. *Environ. Mutag.*, **6**, 705–717

Reynolds, S.H., Patterson, R.M., Mennear, J.H., Maronpot, R.R. & Anderson, M.W. (1990) *ras* Gene activation in rat tumors induced by benzidine congeners and derived dyes. *Cancer Res.*, **50**, 266–272

Sadtler Research Laboratories (1988) *Sadtler Standard Spectra*, Philadelphia, PA

US Environmental Protection Agency (1988) *Computer Printout (CIS): 1977 Production Statistics for Chemicals in the Non-confidential Initial TSCA Chemical Substances Inventory*, Washington DC, Office of Pesticides and Toxic Substances

US Environmental Protection Agency (1990) *Textile Dye Weighing Monitoring Study* (EPA Report No. EPA-560/5-90-009, Main Report and Site Visit Reports), Washington DC, Office of Toxic Substances

US International Trade Commission (1980) *Synthetic Organic Chemicals, United States Production and Sales, 1979* (USITC Publication No. 1099), Washington DC, US Government Printing Office, p. 65

US International Trade Commission (1981) *Imports of Benzenoid Chemicals and Products, 1980* (USITC Publication No. 1163), Washington DC, US Government Printing Office, p. 47

US National Library of Medicine (1992) *Registry of Toxic Effects of Chemical Substances* (RTECS No. QJ6475500), Bethesda, MD

US National Toxicology Program (1991) *Toxicology and Carcinogenesis Studies of CI Acid Red 114 (CAS No. 6459-94-5) in F344/N Rats (Drinking Water Studies)* (NTP TR 405; NIH Publ. No. 92-3136), Research Triangle Park, NC, US Department of Health and Human Services

Venturini, S. & Tamaro, M. (1979) Mutagenicity of anthraquinone and azo dyes in Ames' *Salmonella typhimurium* test. *Mutat. Res.*, **68**, 307–312

Woodruff, R.C., Mason, J.M., Valencia, R. & Zimmering, S. (1985) Chemical mutagenesis testing in *Drosophila*. V. Results of 53 coded compounds tested for the National Toxicology Program. *Environ. Mutag.*, **7**, 677–702