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Effect of Vinyl Chloride on Testis in Rats

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A total of 300 male adult Wistar rats (180–220 g) were used. Animals were divided into four groups: control, vinyl chloride (VC) 10-, 100-, and 3000-ppm exposed groups. Seventy-five rats were used for each group. Inhalation exposure to VC was conducted 6 hr per day and 6 days per week. In the third, sixth, and twelfth month following exposure 8–30 rats of each group were killed. The others were under observation and sacrificed after 18 months. After exposure to VC, the organ and body weight ratio, such as the kidney, liver, spleen, and heart were increased, but those of the testis were decreased in the experimental groups in the sixth month. The differences between experimental groups and the control group were statistically significant ($P < 0.05$ or $P < 0.01$). The weights of testis were decreased and damage of testicular seminiferous tubules were found in rats by histopathological examination. Incidence of damage of testicular seminiferous tubules in the control, 10-, 100-, and 3000-ppm groups were 18.9, 29.7, 36.5, and 56.0%, respectively. There was an obvious dose–response relation between the concentration of VC and the incidence of testis damage, with a correlation coefficient of 0.993 ($P < 0.01$). Statistically, the incidence of damage of testicular seminiferous tubules in rats in the 3000-ppm group and in 100-ppm group were significantly higher than that of the control group, respectively ($P < 0.001$ and $P < 0.05$).

It is known that vinyl chloride (VC) is carcinogenic to animals. However, we have found that VC may induce testicular damage in rats in our exposure experiment. This finding has not been reported in the literature. The effect of VC on testicular lesion in rats is described in detail in this paper.

MATERIAL AND METHODS

Material. VC was provided by the Beijing Second Chemical Factory. The purity was 99.99% (the 0.01% impurities mainly consisted of the following chemicals: chloromethane, 1,3-butyldiene, propylene, monoethylene acetylene, water, and others.)

Animals. A total of 300 adult Wistar rats weighting 180–220 g were used. They were randomly divided into four groups and received the following treatments, respectively: control, vinyl chloride 10, 100, and 3000 ppm by inhalation. Rats per group (8, 30, 6, and 10) were killed at the third, sixth, ninth, and twelfth month, respectively, after the initial exposure. Surviving rats were killed at the 18th month (i.e., 6 months after discontinuing the exposure). Animals were weighed once per month before and after exposure and observed for symptoms twice a day. The diet and water was *ad libitum*. The diet was supplied by the Centre for Experimental Animals, Chinese Academy of Medical Sciences.

The animals were kept in cages made of steel nets.

Inhalation exposure. Customized six-angle cubic shaped glass dynamic chambers were employed for this study. These chambers permitted continuous observation during

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exposure and were designed to ensure uniform spatial distributions of gas mixtures introduced through the inlet port. Animals that inhaled VC 10, 100, and 3000 ppm were put into the chambers of 2918, 774, and 388 liters, respectively. Dynamic inhalation of VC was conducted for a 6-hr period per day and 6 consecutive days per week during the 12 months exposure period. The concentration of VC was determined by Sp-E2305-type gas chromatography (made in China, Beijing Analytic Instrument Factory). The conditions of the analysis are summarized in Table 1.

The effluent value of entrance of VC into the three chambers (10, 100, and 3000 ppm) were 5.3, 20, and 330 ml/min, and for fresh air were 624, 132, and 87.2 ml/min, respectively. According to calculation, fresh air entering the chambers would meet the need for the animals of each group. Vinyl chloride concentration of the respiratory region of the animals in each chamber was evenly distributed. The range of change of VC concentration was within 5%. Thirty min after the beginning of the inhalation exposure, VC concentration in each chamber reached to 94% of the predicted value. For about 1 hr, VC concentration reached the predicted value. VC was provided for exactly 6 hr, followed with a supply of fresh air for another hour, then the animals were removed from the chambers. During the 12 months of inhalation, 30 air samples were taken randomly from each chamber (containing 10, 100, or 3000 ppm VC). During exposure, temperature and relative humidity inside and outside the chambers before and after exposure were recorded every day. The rats were deprived of food and water during exposure.

Autopsies of dead or sacrificed rats were carried out. Testes, lungs, liver, heart, kidneys, spleen, and brain were examined visually for focal or generalized lesions and hemorrhage. The above-mentioned organs and tissues were subjected to microscopic examination. Tissue samples were fixed with 10% Formalin solution and embedded in paraffin. Sections were cut at 5 μm and stained with H & E.

The testes were cut transversely and the pathological changes were graded according to the number of damaged testicular seminiferous tubules counted under the microscope as follows (see Table 5): (1) 10–50 seminiferous tubules damaged (disappearance of spermatids, spermatocytes) was shown as "+"; (2) 50–100 seminiferous tubules damaged (disappearance of spermatids, spermatocytes) was shown as "++"; (3) more than 100 seminiferous tubules damaged (disappearance of spermatids, spermatocytes) was shown as "+++."

TABLE 1

GAS CHROMATOGRAPH CONDITIONS

Instrument	Model SP-E 2305	
Column	2 m × 4 mm i.d. SS 15% Apiezon M, coated onto 40- to 60-mesh 6201	
Temperature	Column	80°C
	Injector	room temperature
	Detector	150°C
Carrier gas	Type	N_2
	Flow rate	36 ml/min
Detector	FID	
Accuracy	5×10^{-3} μg	

RESULTS

Concentration

The range of change of VC concentration is shown in Table 2. As seen from Table 2, concentration of VC in the chamber was evenly distributed.

Mean (±SD) of VC the concentration of 30 air samples from each chamber (containing 10, 100, or 3000 ppm VC) were 11.1 ± 1.1, 105.6 ± 13.9, and 2918 ± 190 ppm, respectively.

The concentration of VC was constant during 6 hr of exposure. The range of change of the concentration in the chamber was within 3.3% (Fig. 1).

The average temperature in the chambers of 10, 100, and 3000 ppm VC were 1.43, 2.07, and 3.01°C higher than that outside the chambers and the average relative humidity was 4.95, 4.64, and 0.18%.

Body Weight

The body weight attained by the animals of all experimental groups and the control group are shown in Fig. 2.

Body weight attained by the animals of the two higher dose groups (100 and 3000 ppm) fluctuated during the 18-month period, but the values were less than that of the control group ($P < 0.05$ and $P < 0.01$).

After 3, 6, 12, and 18 months since the start of exposure to VC, the organ and body weight ratio for kidney, liver, spleen, and heart in rats increased but those of the testes decreased in the 100- and 3000-ppm groups at the sixth month (Table 3).

Tumors Induced by VC

Various tumors in rats induced by inhalation of VC for 18 months are shown in Table 4. Comparison of the incidence of tumors in the 3000- or 100-ppm group with

TABLE 2

VC CONCENTRATION MEASURED AT THE RESPIRATORY REGIONS OF EIGHT ANIMALS IN THE 10-ppm CHAMBER

VC distribution at different levels in the 10-ppm chamber				VC distribution in different directions in the 10-ppm chamber			
Level	Points of samples	VC concn (ppm)	Avg concn (ppm)	Direction	Points of samples	VC concn (ppm)	Avg concn (ppm)
Upper respiratory region	A	9.9	9.75	Northeast	A	9.9	9.80
	B	9.9			A′	9.7	
	C	9.6		Northwest	B	9.9	9.95
	D	9.6			B′	10.0	
Lower respiratory region	A′	9.7	9.72	Southeast	C	9.6	9.55
	B′	10.0			C′	9.5	
	C′	9.5		Southwest	D	9.6	9.65
	D′	9.7			D′	9.5	

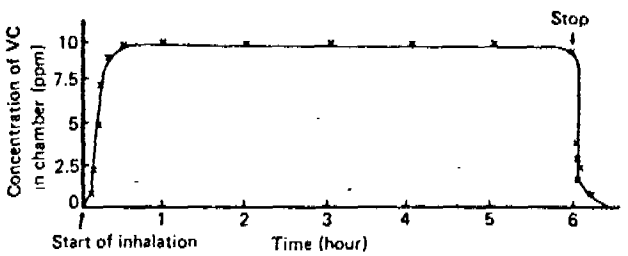

FIG. 1. Range of change of VC concentration in the chamber during 6 hr of exposure.

that of the control showed a statistically significant difference ($P < 0.001$). The difference in the incidence of angiosarcoma between the 3000-ppm group and the control group was also statistically significant ($P < 0.001$).

Damage of Testes

Damage of testicular seminiferous tubules has been found in rats. The main changes of seminiferous tubules in the early stage consisted of irregular vacuolation of cytoplasm, swelling of cells, condensing of nuclei into minute hyperchromatic masses, and sloughing of the spermatids into the lumen. Fusion of several spermatids into multinucleated forms may occur (Fig. 3). Spermatocytes can also fuse into giant cells. At first, spermatids in seminiferous epithelial cells disappeared. After the disappearance of spermatids, the secondary, primary spermatocytes were sloughed into the lumen of the tubules, and in general, regardless of the degree of damage, a few spermatogonia and the Sertoli cells also remained. Some parts of seminiferous epithelial cells disappeared, but the rest remained (Fig. 4). Degeneration and necrosis were found in some parts of seminiferous epithelial cells (Fig. 5). The distribution of microscopic change occurred haphazardly: some tubules had severe degenerative changes while in the nearby tubules the changes were minimal (Fig. 6). This distribution appeared to be random and was neither consistent nor obviously related to the distribution of vascular blood. Damage of testicular seminiferous tubules occurred in the center as well as the

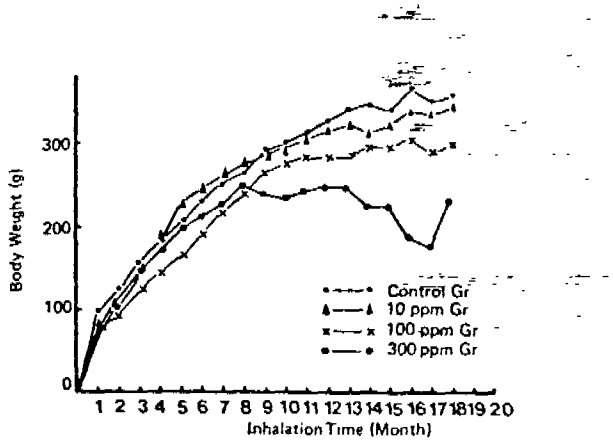

FIG. 2. Effect of long inhalation exposure to VC on body weight of rats.

TABLE 3

EFFECT OF VC ON THE ORGAN AND BODY WEIGHT RATIO IN RATS

Time of exposure (month)	Group	Organ and body wt ratio				
		Testes	Kidney	Liver	Spleen	Heart
3	Control	7.13	5.79	29.61	1.47	3.02
	Expt		6.49[c,**]		1.68[c,*]	3.26[b,*]
6	Control	6.41	5.81	27.96	1.39	2.58
	Expt	5.73[b] **		31.93[a] 34.51[b] **		2.89[a] *
		5.91[c]		47.08[c]	1.51[a,*]	2.79[c]
12	Control	5.49	5.83	30.97	1.34	2.57
	Expt		6.86[c,**]	37.09[c,*]		
18	Control	5.77	5.09	31.68	1.34	2.66
	Expt		5.94[b,*]			

[a] Rats exposed to 10 ppm.
[b] Rats exposed to 100 ppm.
[c] Rats exposed to 3000 ppm.
* $P < 0.05$.
** $P < 0.01$.

peripheral part of testes. In the case of severe damage, normal seminiferous tubules were scarcely found (Fig. 7). A dose–response relation has been shown between the severity of the testicular seminiferous tubules and the exposure concentration of VC. The correlation coefficient is 0.993, $P < 0.001$ (Table 5).

The incidence of damage of testicular tubules in the control, 10-, 100-, and 3000-ppm groups were 18.9, 29.7, 36.5, and 56.0%, respectively. And the incidence of damage of testicular seminiferous tubules in rats exposed to 100 and 3000 ppm of VC were significantly ($P < 0.05$ and $P < 0.001$) higher than that of the control animals.

TABLE 4

CARCINOGENESIS OF VC ON WISTAR RATS UNDER INHALATION EXPOSURE FOR 18 MONTHS

Group	No. animals	No. animals bearing tumors	Incidence of tumor (%)	Target organ					
				Angiosarcoma					
				Liver	Lung	Lung	Nose	Skin	Other
Control	19	1	1/19 (5.2)	0	0	1	1	0	0
10 ppm	20	1	1/20 (5.0)	0	0	1	1	0	0
100 ppm	19	11	11/19 (57.9)	7	2	3	2	2	3
3000 ppm	20	19	17/19 (95.0)	17	9	4	1	1	1

* $\chi^2 = 10.962$, $P < 0.001$.
** $\chi^2 = 31.405$, $P < 0.001$.

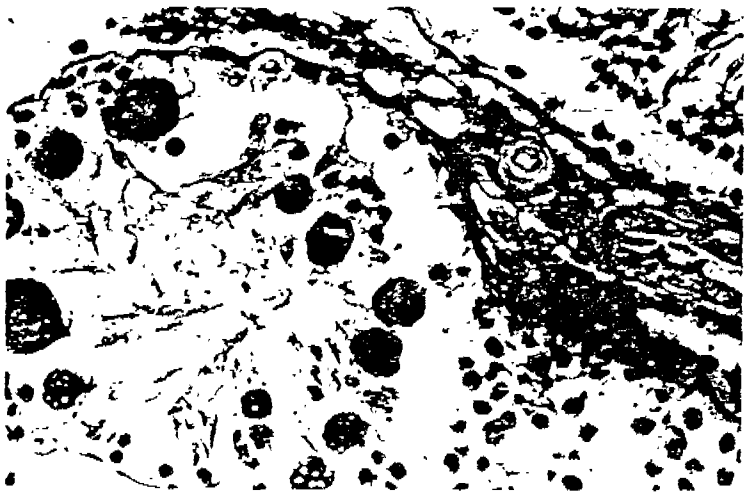

FIG. 3. Fusion of several spermatids into multinucleated forms in a rat exposed to 10 ppm VC for 6 months. H & E, ×200.

DISCUSSION

Ribelin *et al.* (1963) has studied the effect of cadmium, phytic acid, acetoglycerides, jojoba bean meal, naphthalene, *trans*-aconitic acid, and dialdehyde starch on the testes of animals and found that the pathological changes of the testes were of the same type regardless of the compounds exposed. The differences between animals fed different compounds have been no greater than the differences occurring in animals fed any one of these compounds at various levels or various lengths of feeding time. The findings in the present study show that the pathological changes found in the VC-exposed rats were very similar to that described by Ribelin.

FIG. 4. Disappearance of some parts of the seminiferous epithelial cells. Some spermatids sloughed into the lumen. The rat was exposed to 3000 ppm for 6 months. H & E, ×200.

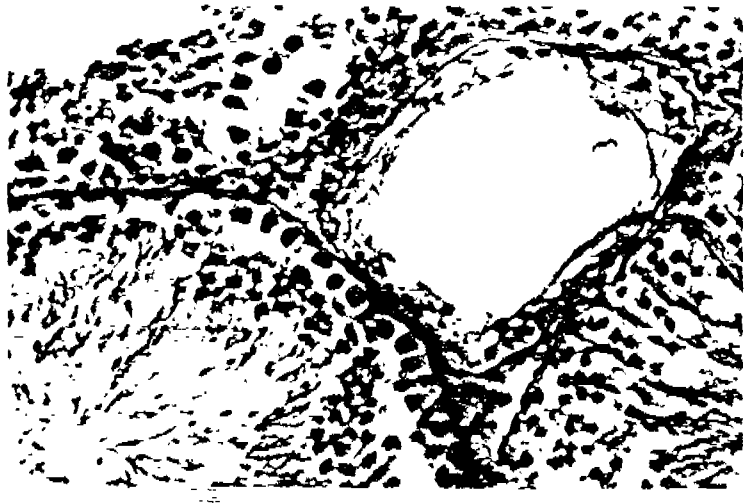

FIG. 5. Degeneration and necrosis in some parts of the seminiferous epithelial cells in the rat exposed to 10 ppm VC for 6 months. H & E, ×400.

According to the processes of damage to the seminiferous epithelial cells, chemicals are divided into the following two categories: (1) Compounds which may cause damage to seminiferous epithelial cells in the order of the spermatids, secondary spermatocytes, primary spermatocytes, and spermatogonia include dichromochloropane, cadmium, naphthalene, acetoglycerides, *trans*-aconitic acid, phenacitine, analgen, fluoroacetamhexachlorophene, DDT, bis(dichloroacetyl)diamines, thiouracil, and cyclohex-

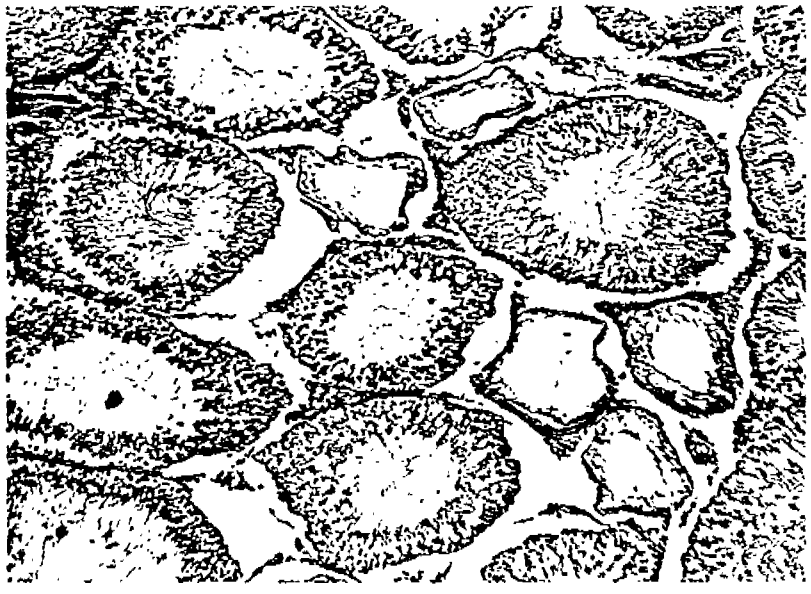

FIG. 6. Severe degenerative changes in some tubules in rats exposed to 100 ppm VC for 12 months. H & E, ×100.

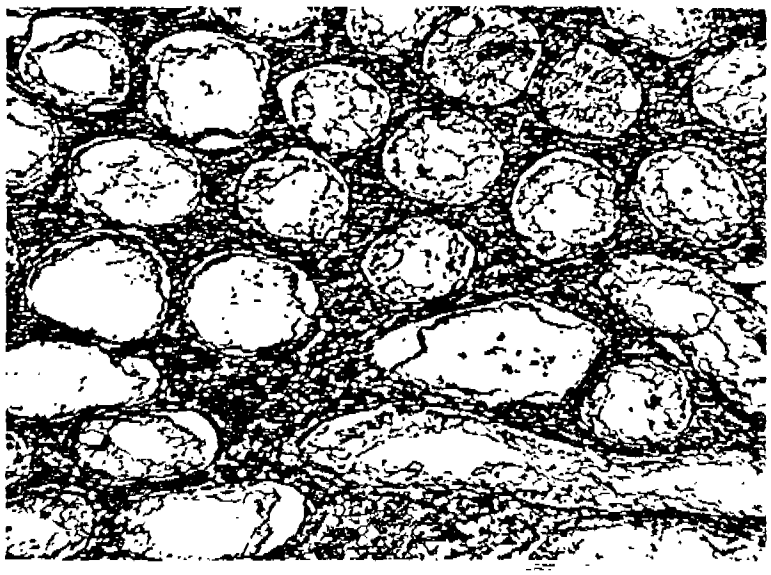

FIG. 7. In the case of severe damage, normal seminiferous tubules were scarcely found. H & E, ×100.

ylamine (Ribelin *et al.*, 1963; Heywood *et al.*, 1978; Biava *et al.*, 1978; Drobeck and Coulston 1962). The lesions produced by VC were similar to those produced by these compounds. (2) Compounds that affect the spermatogonia at first and the spermatids last, include some antitumor drugs and alkyl agents which interfere with DNA synthesis (Heywood *et al.*, 1978; Cheng Yunsu *et al.*, 1965).

In 1981, Hatch reviewed the effect of VC on reproductive systems, and his conclusion was that "At present, there is no data which point unambiguously to a relation between VC and reproductive outcome."

Sokal *et al.* (1980) studied the chronic toxic effect of VC on rats exposed to 50, 500, and 20000 ppm VC in the air for 10 months. Local degeneration, necrosis, and

TABLE 5

DAMAGE OF TESTES INDUCED BY DIFFERENT CONCENTRATIONS OF VINYL CHLORIDE

Group	No. animals	Damage of testis			Total no.	%
		+	++	+++		
Control	74	9	3	2	14	18.9
10 ppm	74	14	5	3	22	29.7
100 ppm	74	19	5	3	27	36.5*
3000 ppm	75	29	8	5	42	56.0**

Note + = 10–50 depleted seminiferous tubules were found in a section. ++ = 50–100 depleted seminiferous tubules were found in a section. +++ = More than 100 depleted seminiferous tubules were found in a section. Correlation coefficient $r = 0.993$, $P < 0.01$.

* $P < 0.05$.

** $P < 0.001$.

disorders of spermatogenesis in the seminiferous epithelial cells were found. However, a dose–response relationship between the changes in testes and concentration of VC was not found.

Heywood *et al.* (1978) pointed out that testicular atrophy may be observed in about 20% of the control rats that had been fed for 2 years under normal conditions. He concluded that the effect of chemicals on testes can be demonstrated by the comparison of experimental results with that of the control and by the dose–response relationship. Our study of the effect of VC on testicular damage in rats is consistent with the conclusions of Heywood *et al.* We have found that 18.9% of the rats in the control group showed atrophy of testicular seminiferous tubules. Damage of testes was increased as the concentration of VC was increased. Statistical tests of the correlation coefficient showed a positive correlation between the incidence of damage of testicular tubules and the concentration of VC. Damage of testes in rats was proved to be related to inhalation of VC.

We believe that the effects of VC on the reproductive system deserve further study.

ACKNOWLEDGMENTS

We thank Professor Li Yurui for critical review of this manuscript. Ms. Luo Yushu and Geng Taibao carried out the inhalation exposure of rats. Ms. Meng Huilin prepared the histological sections.

REFERENCES

BIAVA, C. G. *et al.* (1978). The testicular morphology of individuals exposed to dibromochloropropane. *Exp. Mol. Pathol* **29**, 448–458.

CHENG YUNSU *et al.* (1965). A comparison of the destructive effects of five antitumor agents on various normal tissues in mice. *Acta Pharmacol. Sin.* **12**, 606.

COULSTON, F. *et al.* (1960). The biologic actions of a new series of bis(dichloroacetyl)diamines. *Toxicol. Appl. Pharmacol* **2**, 715.

DROBECK, H. P., AND COULSTON, F. (1962). Inhibition and recovery of spermatogenesis in rats, monkeys, and dogs medicated with bis(dichloroacetyl)diamines. *Exp. Mol. Pathol.* **1**, 251.

HATCH, M. (1981). Power considerations in studies of reproductive effects of vinyl chloride and some structural analogs. *Environ. Health Perspect.* **41**, 195.

HELLER, C. G. *et al.* (1961). Suppression of spermatogenesis and chronic toxicity in men by a new series of bis(dichloroacetyl)diamines. *Toxicol. Appl. Pharmacol.* **3**, 1.

HEYWOOD, R. *et al.* (1978). Assessment of testicular toxicity in laboratory animals. *Environ. Health Perspect.* **24**, 73.

RIBELIN, W. E. *et al.* (1963). Atrophy of rat testis as index of chemical toxicity. *Arch. Pathol.* **75**, 229.

SOKAL, J. A. *et al* (1980). Experimental studies on the chronic toxic effects of vinyl chloride in rats. *J. Hyg. Epidemiol. Microbiol. Immunol.* **24**, 285.

whole blood (AlB). An investigation was made into the distribution of Al between the plasma and the blood-cell compartment and on the extent of binding of Al to the blood cells in rats and dialyzed patients. Al was distributed between plasma and blood cells with only very small quant. differences; binding of Al to blood cells was very weak and the AlP and AlB detns. had similar prognostic value for toxicity.

104: 63731a **Effect of ammonia on some enzymes of the urea cycle and ornithine aminotransferase in the brain.** Swamy, M.; Prasad, M. S. K.; Sadasivudu, B. (Dep. Biochem., Osmania Med. Coll., India). *Neirokhimiya* 1985, 4(3), 296–300 (Russ). The activities of *argininosuccinate synthetase* (ASS) [9023-58-9], *argininosuccinase* (AS) [9027-34-3], *arginase* [9000-96-8], and *ornithine aminotransferase* (OAT) [9030-42-6] were investigated in different sections of the rat brain following i.p. administration of 0.8 mmol NH_4OAc [631-61-8]/100 g (acute) and 0.6 mmol NH_4OAc/100 g administration (6 times with 1-interval; chronic). No significant changes in the arginase and ASS activities were obsd. in all the 3 sections of brain examd. (brain stem, cerebral cortex and cerebellum). The AS activity substantially decreased in the cerebellum both under acute and chronic NH_3 intoxication. OAT markedly increased in the cerebellum during chronic and in the cerebral cortex during acute intoxication from NH_3.

104: 63732b **Effect of chromium on growth and utilization of iron and manganese by oat.** Singh, Vinay; Parmar, Ram Kumar (Dep. Agric. Chem. Soil Sci., R.B.S. Coll., Uttar Pradesh, 283105 India). *J. Indian Soc. Soil Sci.* 1985, 33(2), 450–1 (Eng). A pot expts. on alk. sandy loam soil, oat plants treated with Cr (5–25 ppm) developed toxicity symptoms similar to those reported by S. C. Agarwala and C. P. Sharma (1976) and had decreases in dry-matter yield of 68.8% (5 ppm Cr) to 97.1% (25 ppm Cr) with respect to the control. Thus, Cr addn. resulted in decreases in concn. and uptake of Fe and Mn by oat plants, although the decrease in Mn content was not significant at 5 ppm Cr. The magnitude of the decrease due to Cr addn. was much greater for content and uptake of Fe than of Mn.

104: 63733c **Ozone inhibits prostacyclin synthesis in pulmonary endothelium.** Friedman, Mitchell; Madden, Michael C.; Saunders, D. Stephen; Gammon, Kenneth; White, Gilbert C., II; Kwock, Lester (Sch. Med., Univ. North Carolina, Chapel Hill, NC 27514 USA). *Prostaglandins* 1985, 30(6), 1069–83 (Eng). The effects of O_3 on lung *arachidonate* [506-32-1] metab. in vitro were studied in cultured bovine pulmonary endothelial cells exposed for 2 h to O_3 in concns. ≤1.0 ppm. A concn.-dependent decrease in *prostacyclin* [35121-78-9] synthesis was found (90% decrease at the highest O_3 level of 1.0 ppm). The inhibition of prostacyclin synthesis was not due to a decreased release of arachidonic acid from membrane lipids. The hypoxic pulmonary vasoconstrictive response to 10% O inhalation in anesthetized dogs was studied in vivo after exposure to 1.0 ppm O_3 for 1 h. Pulmonary vascular resistance was increased after O_3 exposure, similar to the findings in dogs given indomethacin (15 mg/kg). The percentage change in the hypoxic pulmonary pressor response was similar between the O_3 exposure and indomethacin-treated groups, although due to the variance of the pulmonary vascular resistance values during hypoxia the results did not reach statistical significance. Evidently, O_3 inhalation affects pulmonary endothelial arachidonate metab. in vivo as well as in vitro.

104: 63734d **Effect of some heavy metals (copper, lead, zinc, cadmium) on bacteria, protozoa, and algae.** Terzieva, S.; Danon, S.; Decheva, R.; Toncheva-Panova, T. (Inst Khim. Prof. Zabol., MA, 1431 Sofia, Bulg.). *Khidrobiologiya* 1985, 25, 30–41 (Bulg). The toxicity of Cu, Zn, Pb, and Cd (alone or in combination) to bacteria (e.g., *Escherichia coli, Streptococcus faecalis, Clostridium perfringens*, or *Salmonella typhimurium*), algae (e.g., *Scenedesmus acutus*), and protozoa (e.g., *Glaucoma chattoni, Uronema marinum, Tetrahymena pyriformis, Colpidium campylum*, or *Chilomonas paramecium*) was studied. The effects of heavy metal pollution on aquatic microorganism diversity and the biodegrdn. of org pollutants is discussed.

104: 63735e **Lead and arsenic effects on pyruvate dehydrogenase activity in rat brain.** Anca, Zoe; Gabor, Silvia (Inst. Ig Sanat Publica, Cluj-Napoca, Rom.). *Stud. Cercet. Biochim* 1985, 28(2), 125–9 (Rom). Pb and As at 0.2, 0.5, and 1.0 μM/50 mg wet tissue inhibited *pyruvate dehydrogenase* (I) [9014-20-4] activity in rat brain in vitro. The inhibition was dose dependent. No additive effects on I activity were noted when Pb and As were administered simultaneously. The I activity may reflect the interaction of Pb and As with the cholinergic system and with the energizing activity of the brain.

104: 63736f **Effect of vinyl chloride on testis in rats.** Bi, Wenfang; Wang, Youshen; Huang, Meiyuang; Meng, Deshan (Inst. Health, China Natl. Cent. Prevent. Med., Beijing, Peop. Rep. China). *Ecotoxicol. Environ. Saf.* 1985, 10(3), 281–9 (Eng). Male rats (75) inhaled 10, 100, or 3000 ppm *vinyl chloride* (VC) [75-01-4] 6 h/day and 6 days/wk. In the 3rd, 6th, and 12th month following exposure 8–30 rats of each group were killed. The others were under observation and sacrificed after 18 mo. After exposure to VC, the organ and body wt. ratio, such as the kidney, liver, spleen, and heart were increased, but those of the testis were decreased in the exptl. groups in the 6th month. The differences between exptl. groups and the control group were statistically significant. The wts. of testis were decreased and damage of testicular seminiferous tubules was found in rats by histopathol. examn. Incidence of damage of testicular seminiferous tubules in the control, 10-, 100-, and 3000-ppm groups was 18.9, 29.7, 36.5, and 56.0%, resp. There was an obvious dose-response relation between the concn. of VC and the incidence of testis damage, with a correlation coeff. of 0.993. Statistically, the incidence of damage of testicular seminiferous tubules in rats in the 3000-ppm group and in the 100-ppm group was significantly higher than that of the control group.

104: 63737g **Subacute toxicity of 1,1,1-trichloroethane, noise, and their combination in rats.** Blohm, Miranda; Braun, Horst; Kaschny, Peter; Schill, Walter; Jastorff, Bernd; Diehl, Horst (Fachber. 1 Phys./Elektrotech., Univ. Bremen, D-2800 Bremen, 33 Fed. Rep. Ger.). *Ecotoxicol. Environ. Saf.* 1985, 10(3), 295–301 (Eng). Parallel groups of rats were simultaneously exposed to defined conditions (*i*) *1,1,1-trichloroethane* (TCE) [71-55-6] vapor; (*ii*) noise pollution of 90 decibels; (*iii*) their combination; and (*iv*) a control group without any exposure. The vapor of TCE was applied at a concn. of 200 ppm/8 h or of 2000 ppm/12 h for 84 days each. The expts. were performed with TCE from 2 different com. sources. One of those TCE preps. caused effects at the high dosage level in terms of enhanced levels of (*i*) the relative of liver to body wt.; (*ii*) liver microsomal protein content; (*iii*) liver microsomal *monooxygenase* [9038-14-6] activity; and (*iv*) *3,4-dihydroxyphenylglycol* [3343-19-9] excretion in urine. Eight other physiol. and biochem. parameters were not changed. The other TCE prepn. at the high dose, both TCE preps. at the low dose, and noise did not affect the investigated parameters.

104: 63738h **Pathomorphological changes in gills of fish fingerlings (Cirrhina mrigala) by linear alkyl benzenesulfonate.** Misra, Virendra; Lal, Hazari; Chawla, Geeta; Viswanathan, P. N. (Ind. Toxicol. Res. Cent., Lucknow, 226 001 India). *Ecotoxicol. Environ. Saf.* 1985, 10(3), 302–8 (Eng). Fish (*Cirrhina mrigala*) fingerlings exposed to a 0.005 ppm (25% of median lethal concn.) concn. of detergents (linear alkyl benzenesulfonate) showed marked behavioral changes and distorted appearance of primary and secondary lamellae along with damage to gill epithelium under SEM at various magnifications. Mucosal cells of gills secreted mucus showing primary reactions for membrane damage leading to dysfunction in respiration and osmoregulation.

104: 63739j **Toxicology of selenium in a freshwater reservoir: implications for environmental hazard evaluation and safety.** Lemly, A Dennis (Dep. Biol., Wake Forest Univ., Winston-Salem, NC 27109 USA). *Ecotoxicol. Environ. Saf.* 1985, 10(3), 314–38 (Eng). A study was conducted to document patterns of accumulation of toxicity of Se to organisms in a power plant cooling reservoir in North Carolina. Se entered the reservoir by way of effluent from the coal ash disposal basin, which contained 100–200 μg Se/L. Concns. of Se in the lake water avs 10 μg/L, but were accumulated from 519 times (periphyton) to 3975 times (visceral tissue, largemouth bass) in the biota. The pattern and degree of accumulation was essentially complete within 2 yr after the initial operation of the power plant, and persisted throughout the remainder of the study: fishes > insects > annelids > mollusks > crustaceans > plankton > periphyton. The planktonic and detrital food pathways exposed fishes to potential dietary concns. of Se that were some 770 and 519–1395 times the waterborne exposure, resp. Of the 20 species of fish originally present in the reservoir, 16 were entirely eliminated, 2 were rendered sterile but persisted as adults, 1 was eliminated but managed to recolonize from a relatively uncontaminated headwater area as sterile adults, and 1 was unaffected. Two nonnative fish species were accidentally introduced and established reproducing populations. Abundance and diversity of biota other than fishes was not affected. Relative to control habitats, the contaminated reservoir had concns. of waterborne Se that were 20–30 times background levels; the flora and fauna contained about 10–15 times background. Thus, Se can accumulate and be biol. magnified to toxic levels in a reservoir even though waterborne concns. are in the low microgram/L range. This study also provides data which indicate that current toxicol. information is neither accurate when used to predict the relative sensitivity of individual fish species to Se, nor is it sufficient to predict community responses in a natural setting. It is very likely that long-term elevation of waterborne Se to 8–10 μg/L in warm-water lakes and reservoirs would result in biotic responses similar to those documented in this report.

104: 63740c **Adenylate cyclase and alkaline phosphatase activity in the cerebral blood vessels of the rat in manganese chloride poisoning.** Szumanska, Grazyna; Mossakowski, Miroslaw J. (Cent. Med. Dos. Klin., PAN, Warsaw, Pol.). *Neuropatol. Pol.* 1985, 23(3), 297–314 (Pol). In rats with Mn encephalopathy [induced by i.v. injection of 0.25 g Mn/kg (as $MnCl_2$) during 4 wk in 7 doses], the activities of *adenylate cyclase* [9012-42-4] and *alk. phosphatase* [9001-78-9] of cerebral capillary blood vessels were below normal. The activity of alk. phosphatase remained low throughout the expt. whereas the activity of adenylate cyclase returned to near normal at the end of the expt.

104: 63741d **Mechanisms of aerosol deposition in a nasal model.** Itoh, H.; Smaldone, G. C.; Swift, D. L.; Wagner, H. N., Jr. (Dep. Nucl. Med., Kyoto Univ. Hosp., Kyoto, Japan 606) *J. Aerosol Sci* 1985, 16(6), 529–34 (Eng). Total and regional aerosol deposition were investigated in a model of a normal human nasal airway. Contributions of fluid turbulence and particle inertia were evaluated using monodisperse aerosols. At fixed turbulent flow conditions, the deposition percentage increased with particle size >1 μm, suggesting that turbulent inertial deposition is a primary mechanism. With the same size aerosol, deposition increased with increasing fluid turbulence but its contribution was less with a larger size aerosol. Turbulent diffusion was the dominant transport mechanism for particles <1 μm, where deposition decreased with particle size. Two major deposition sites were visualized with radioaerosol in the anterior region of the nasal airway. One is close