values are negative in both cases (excepting for IO_3^- at 45 °C) which indicates that the transfer of salts from water to dioxane + water is favoured as far as chemical interactions are concerned. The $\Delta G^0_{t(el)}$ is positive in both cases and is of the order $BrO_3^- > IO_3^-$ and hence the ionic solvation is of the reverse order. The $\Delta H^0_{t(el)}$ is negative whereas $\Delta H^0_{t(ch)}$ is positive (excepting in 2 cases) and both increases with the increase in dioxane content. $\Delta S^0_{t(el)}$ is negative in all cases and becomes more and more negative with increase in dioxane content, indicating that the orderedness in the solvent structure $\Delta S^0_{t(ch)}$ is positive in case of $NaBrO_3$ and decreases with increase in temperature, but in case of $NaIO_3$ it is positive at 30 and 35 °C and negative at 40 and 45 °C, which indicates the chemical interaction.

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Effect of dietary cholic acid and cholesterol on liver and kidney cystathionase and cysteine sulfinate decarboxylase activities and taurine concentrations in the rat[1]

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Summary. Hepatic cystathionase and cysteine sulfinate decarboxylase activities are drastically affected by cholic acid added to the diet without cholesterol. When cholic acid and cholesterol are given together, only cysteine sulfinate decarboxylase activity is changed. Neither kidney enzyme activity nor taurine concentrations in the liver and kidney are noticeably modified, whatever the diet.

It is known that cholesterol is involved in atherosclerosis and there are indications that sulfur amino acids can also be involved in this disease[2]. This suggests that cholesterol and sulfur amino acid metabolisms are not independent and we have been interested in studying the effect of cholesterol on the enzymes of sulfur amino acid metabolism and on taurine concentrations in the liver and kidney. A series of rats was given a cholesterol-rich diet in which cholic acid was added to promote a good absorption of cholesterol. Another series of rats received a control diet containing cholic acid but no cholesterol. 2 enzyme activities, cystathionase (CNase EC 4.4.1.1) and cysteine sulfinate decarboxylase (CSD EC 4.1.1.29), and taurine concentrations were measured. Surprising results obtained even in the control rats made us wonder if they were due to cholic acid and not the other components of the diet. Therefore we also used rats fed with a control diet without cholic acid.

Materials and methods. Several interrelated adult male albino rats from our breeding unit were divided into 2 groups. They were fed (15 g/day) purified diets (formulations are shown in table 1) with free access to water. In experiment 1, 6 rats were given the control A diet and 6 rats were fed a cholesterol-rich diet (chol. group). These 12 rats received cholic acid. In experiment 2, 3 rats were given either the control A or the control B diet (without cholic acid). After 3 or 4 weeks on the diets, rats were killed by decapitation. Immediately upon removal, a fragment of liver and a kidney were frozen for later determination of taurine concentrations according to Anzano et al.[3]. Another sample of liver and the second kidney were homogenized in cold 0.25 M sucrose, 2 mM dithiothreitol (4 ml/g tissue) in a glass homogenizer with a Teflon pestle. The 105000 × g supernatant was used for enzymatic analysis, since these 2 enzymes are present in the cytosol.

These assays were performed as already described[4,5]. The results are expressed as µmoles of H_2S produced per h per g of wet tissue for CNase activity and as µmoles of CO_2

Table 1. Composition of diets (% by wt)

	Control A	High cholesterol diet (chol.)	Control B
Sucrose	64	62	64
Cholic acid	0.4	0.4	
Cholesterol		2	

In addition each diet contained 18% USBC vitamin-free casein, 10% hydrogenated coprah purchased from ITERG (Paris), 2% USBC total vitamin supplement, 4% USBC salt mixture (Wesson modification) and 0.7% sunflower oil for essential linoleic acid supply.

Table 2. Summary of experiment 1

	Control A	Chol.
Initial body wt (g)	423 ± 18	423 ± 12
Final body wt (g)	399 ± 14	392 ± 13
Liver wt (g)	13.8 ± 0.5	18.2 ± 0.9 $p < 0.01$*
Kidneys wt (g)	2.51 ± 0.07	2.53 ± 0.04
Liver enzyme activity		
CNase µmoles H_2S per h per g	103.9 ± 7.8	59.3 ± 3.4 $p < 0.01$*
CSD µmoles CO_2 per h per g	12.26 ± 1.38	11.88 ± 0.82
Kidney enzyme activity		
CNase	18.7 ± 0.2	17.3 ± 0.4
CSD	8.44 ± 0.44	9.10 ± 1.00
Taurine concentration µmole/g		
Liver	1.30 ± 0.07	1.22 ± 0.08
Kidney	9.76 ± 0.45	8.31 ± 0.27 $p = 0.02$*

Dietary treatment for 4 weeks. Values are the mean ± SEM for 6 rats, except CNase activity (4 rats). * Values different from control A.

produced per h per g of wet tissue for CSD activity. They are presented as the mean ± SEM. Statistical significance is estimated with the Student's t-test.

Results and discussion. The data of experiments 1 and 2 are presented in tables 2 and 3. They show that 3 or 4 weeks feeding (15 g/day) with these diets depressed body weight, and cholesterol plus cholic acid supplementation increased the liver weight as was shown recently[6].

In the liver, the CNase activity was significantly higher in the control A group than in rats fed the control B diet. Therefore dietary cholic acid enhanced CNase activity. In contrast, when cholic acid and cholesterol were associated in the diet, CNase activity was not increased. The diets containing cholic acid led to a drastic decrease of CSD activity (about 15 µmoles of CO_2 produced per h per g instead of 44 µmoles in rats fed the control B diet). When expressed as µmoles of CO_2 produced per h per liver, CSD activity was higher in chol. rats than in control A rats but was still lower than in control B rats. These results clearly indicate that cholic acid was responsible for lowering CSD activity. This striking alteration in CSD activity was not associated with an important change in taurine levels although CSD is involved in the major synthetic pathway of taurine in mammals. However it has been pointed out that in rat liver CSD activity does not reflect the taurine concentration[7,8]. Furthermore, as Awapara has already shown[9], we observed that dietary cholic acid did not induce change in the liver taurine concentration.

In the kidney, unlike in the liver, cholic acid, whether associated or not with cholesterol, seemed to be without effect on CNase and CSD activities and on the taurine concentration although the taurine concentration was slightly lower in the chol. group than in control A rats.

In this stage of our research, it is impossible to explain the opposite effects of cholic acid on liver CNase and CSD activities and also the effect of cholesterol associated with cholic acid which brought liver CNase activity to its normal level. The effect of cholic acid on biological membranes is known, whereas very little information is available about the action of cholic acid feeding on enzyme activities. However, Tsai and Dyer[6] reported a depressant effect of cholic acid, or cholic acid plus cholesterol, on some liver enzymes whose activities are correlated with the rate of lipogenesis. From these preliminary results it appears that the use of cholic acid complicates the examination of the effect of cholesterol on CNase and CSD.

Table 3. Summary of experiment 2

	Control A	Control B
Initial body wt (g)	327 ± 2	327 ± 2
Final body wt (g)	315 ± 11	326 ± 13
Liver wt (g)	12.6 ± 1.2	12.3 ± 0.8
Kidneys wt (g)	1.25 ± 0.09	1.18 ± 0.11
Liver enzyme activity		
CNase µmoles H_2S per h per g	87.5 ± 3.6 $p < 0.01$*	58.4 ± 2.6
CSD µmoles CO_2 per h per g	14.7 ± 1.8 $p < 0.01$*	44.0 ± 9.6
Kidney enzyme activity		
CNase	18.8 ± 0.6	18.3 ± 0.6
CSD	7.12 ± 2.06	7.26 ± 1.26
Taurine concentration µmole/g		
Liver	1.42 ± 0.07	2.02 ± 0.27
Kidney	8.47 ± 0.32	8.68 ± 0.05

Dietary treatment for 3 weeks. Values are the mean ± SEM for 3 rats. * Values different from control B.

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Effets du sevrage alcoolique sur les activités alcool- et aldehyde-déshydrogénasiques extra-hépatiques chez le rat[1]

Effect of withdrawal from alcohol on extra-hepatic and alcohol- and aldehyde-dehydrogenasic activities in the rat

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Summary. Alcohol- and aldehyde-dehydrogenasic activities have been measured in different tissues; these activities are modified after chronic alcoholic intoxication and/or withdrawal in digestive tract, spleen, kidney and lung. The results underline the possible relationship between extra-hepatic ethyl-oxidation and withdrawal syndrome.

Parmi les différentes voies du catabolisme de l'alcool, tous les auteurs s'accordent pour estimer que la voie des déshydrogénases est prépondérante et que le foie à lui seul aurait une capacité métabolique suffisante pour dégrader la totalité de l'alcool ingéré[2]. Cependant, la répartition des enzymes de l'éthyl-oxydation dans différents tissus est intéressante à étudier: en effet, ces enzymes permettraient, même faiblement, l'éthyl-oxydation, in situ, dans le cerveau, dans des organes d'absorption, d'excrétion ou de stockage[3-5] et interviendraient dans le métabolisme de composés endogènes comme les catécholamines ou la sérotonine[6].

Le présent travail a donc pour objet la mesure des activités alcool-déshydrogénasiques et aldéhyde déshydrogénasiques dans le tractus digestif, la rate, le rein, le tissu adipeux, le poumon et le cerveau, comparativement au foie; cette mesure a été réalisée chez des animaux témoins, chez des