Na-23 SPIN-LATTICE RELAXATION TIMES OF LIVERS OF NORMAL AND CCl_4 TREATED RATS

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Na-23 NMR relaxation times may provide direct information on the chemical-physical environments of intracellular Na^+ and on changes induced by cellular alterations (1). We studied intracellular Na-23 NMR relaxations of liver tissues from normal and acutely intossicated with CCl_4 rats.

As shown in Table 1 a significative increase of intracellular Na-23 for the rats treated with CCl_4 was observed with respect to the controls. These changes could be related to the damaging effects caused by the CCl_4 treatment such as lipid peroxidation, fatty accumulation and liver necrosis (2).

Table 1. Na-23 T_1 (ms±S.D.) in livers of treated rats (cases analyzed in parenthesis)

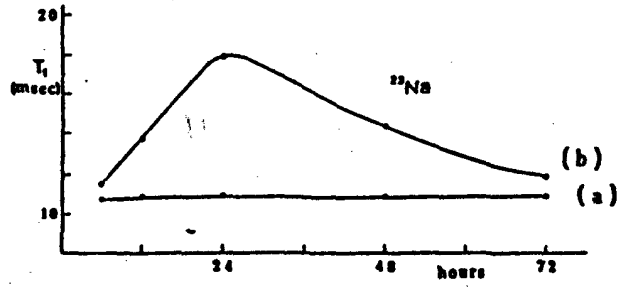
Mineral oil (8)	11.4 ∓ 0.3
CCl_4 (8)	23.3 ∓ 0.7
Bromobenzene (10)	11.3 ∓ 0.5
Vit. E (8)	10.5 ∓ 0.7
Vit. E + CCl_4 (10)	15.9 ∓ 0.8

To elucidate which of these effects is responsible for the observed T_1 lengthenings, T_1 values of livers from Bromobenzene intoxicated rats, were studied, this molecule being known to cause hepatic necrosis but neither lipid peroxidation nor fatty accumulation. The obtained results show that Bromobenzene did not induce T_1 changes in comparison to the control values. Therefore, the liver necrosis was not considered as a factor affecting the T_1 lengthening found in CCl_4 intoxicated rats.

On the other hand a significant attenuation of the lengthening of T_1 was observed in liver rats intoxicated with CCl_4 after pretreatment with vitamin E. This finding was attributed to the antioxidant action exherted by vitamin E, which is known to be efficient in protecting against CCl_4-induced hepatic lipid peroxidation but not in preventing fatty accumulation (2).

In order to obtain further information on the cause of the T_1 lengthening in CCl_4 intoxicated rat livers, T_1 relaxation times of intracellular Na-23 from livers of rats intoxicated with CCl_4 were studied as a function of the time. The results are shown in Fig. 1. It can be observed that T_1 values exhibited a maximum increase 24 hrs after CCl_4 treatment and a decrease towards the control values 72 hrs after the intoxication. Considering that fat accumulation is present in liver cells until 72 hrs after intoxication, the findigs in Fig. 1 strongly suggest that the fatty accumulation in the liver cells is not directly responsible for the observed lengthening of the T_1 values found in CCl_4 intoxicated rats.

Figure 1. Time-dependent Na-23 T_1 values of (a) control and (b) CCl_4-intoxicated rat livers.



These overal findings evidentiate the dependence of T_1 relaxation values of intracellular Na-23 on the cellular damages. Furthermore, the use of particular strategies of investigation as those adopted in this study may give information on the specific cellular alterations responsible of the T_1 changes.

1) Casu, M., Corongiu, F.P., Dessì, M.A., Frau, M. and Lai, A. (1986) Biochem. Biophys. Res. Commun. 134, 1079.

2) Slater, T.F. (1972) Free Radical Mechanisms in Liver Injury, Pyon Ltd, London