Biol. Chem. Hoppe-Seyler
Vol. 374, pp. 665–670, August 1993

Lysosomal Enzyme Activities in Liver and Sera from Guinea Pigs Fed Oil Related to the Toxic Oil Syndrome

M. Mario SÁNCHEZ-MARTÍN, Carmen SÁNCHEZ-BERNAL, Antonia SÁNCHEZ-LLORENTE, José A. CABEZAS and Nieves PÉREZ-GONZÁLEZ

Departamento de Bioquímica y Biología Molecular, Facultad de Biología, Universidad de Salamanca, Spain

(Received 2 April / 3 June 1993)

Summary: β-*N*-Acetylglucosaminidase and β-galactosidase activities were determined in serum and liver from guinea pigs fed "toxic oil" (related to cases of TOS) under different experimental conditions. The results obtained were compared with those of guinea pigs fed non "toxic oil" (case-unrelated oil; controls 1) and animals fed no oil (controls 2). In serum, both activities were significantly increased after all treatments with case-related oil as compared with controls 1 and 2. In the liver, β-galactosidase activity did not show significant differences in any of the treatments when compared with controls 2. However, NAG activity decreased significantly after 7 days of treatment with non-heated oil – either case-related or not – when compared with controls 2; it also decreased significantly after 28 days of treatment with heated case-unrelated oil, both with respect to controls 2 and the animals fed case-related oil. Liver weights tended to increase in the animals fed oil – toxic or not – with respect to those of the livers from untreated animals. Morphologically, a slight vacuolization of the hepatocytes was observed in some of the samples from the treated animals.

Key terms: Toxic Oil Syndrome, lysosomal enzymes, serum, liver, β-*N*-acetylglucosaminidase, β-galactosidase.

The Toxic Oil Syndrome (TOS) is a previously undescribed disease that began in Spain, in the spring of 1981, as a massive epidemic causing a high number of deaths. The symptoms corresponded to those of a multisystemic disease and the clinical features varied considerably[1]. The disease has been associated with the ingestion of denatured rapeseed oil, considered to be the vehicle of the etiologic agent[2,3]. Although several etiologies have been advanced, so far the causes of the epidemic remain essentially unexplained. Preliminary data suggested that fatty acid anilides were involved[4]. More recently, Kilbourne et al.[5] have also reported evidence pointing to the existence, in humans, of an association between increased risk of illness and the concentrations of aniline and anilide contaminants. Other industrial chemicals, probably arising from poorly cleaned tankers used to transport the oil, have now been suggested as contaminants. Nevertheless, the precise etiological agent has yet to be identified[3]. The evaluation of different etiologic hypotheses based on toxicological studies has not been successful owing to the lack of an animal model for studying the disease and the additional difficulty derived from either the use of oil specimens that are not always illness case-related or insufficient administration of the TOS agent[5]. However, the need to develop animal experimentation with a view to finding a species susceptible to the etiologic agent, thus making it possible to assess the effects of the disease, has been stressed[6].

Enzymes:
β-*N*-Acetylglucosaminidase, NAG, (EC 3.2.1.52);
β-Galactosidase (EC 3.2.1.23).

Abbreviation:
TOS, Toxic oil syndrome.