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Alcohol Intoxication and Sialic Acid in Erythrocyte Membrane and in Serum Transferrin

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SCHELLENBERG, F., F. BEAUGÉ, C. BOURDIN, J. M. BOURRE AND J. WEILL. *Alcohol intoxication and sialic acid in erythrocyte membrane and in serum transferrin.* PHARMACOL BIOCHEM BEHAV 39(2) 443–447, 1991.—Microheterogeneity of serum transferrin as well as erythrocyte membrane sialic acid content were examined in alcoholic patients and healthy controls. Both the sialic acid content of erythrocyte membranes and of the circulating transferrin were significantly lower in alcoholic patients than in controls. A moderate daily ethanol intake (less than 80 g) allowed to observe a proportional relationship between alcohol intake and the carbohydrate deficient forms of transferrin, and also a correlation between alcohol intake and the membrane sialic acid content. This supports the hypothesis of ubiquitary alterations of glycosylations in connection to ethanol intoxication. Additional disturbances could explain the absence of correlations between membrane sialic acid, pattern of abnormal forms of serum transferrin, and alcohol intake in heavy alcoholic patients.

Alcohol Erythrocyte membrane Serum transferrin Sialic acid

SOME years ago, abnormal forms of serum transferrin were found present in the spinal fluid as well as in the serum of alcoholic patients when analyzed by isoelectric focusing (23). These abnormal forms were characterized by their higher isoelectric point (5.7 and 5.9 versus 5.4 for the usual form). This microheterogeneity of the serum transferrin disappeared after alcohol withdrawal (17). Transferrin is a sialoglycoprotein containing 4 to 5 sialic acid residues responsible for the electrical charge of the molecule. Neuraminidase-treated serum samples exhibited the same microheterogeneity than samples from alcoholics (29), that suggested a reduced sialic acid content of the abnormal forms of transferrin molecule. This was later confirmed (25) by the determination of the sialic acid content of the transferrin forms. In order to use this microheterogeneity as a potential indicator of alcohol intoxication, different quantification methods of the abnormal fractions of transferrin were developed using either isoelectric focusing (21, 22, 33) or a radioimmunoassay (31).

Furthermore, a deficiency in sialic acid was also observed in the proteins of erythrocyte membranes of alcoholic patients (26). As other studies (13) have demonstrated that inhibition of glycosyltransferases by ethanol was involved in these two phenomena, alcohol consumption and sialic acid deficiency were possibly correlated. The purpose of the present investigation was, therefore, first to ascertain the decrease in the erythrocyte membrane sialic acid, as well as the microheterogeneity of transferrin in alcoholic patients and second to study a possible relation of these sialic acid alterations to the alcohol consumption.

POPULATION

Blood samples were collected from 27 alcoholic patients aged 23–64 years (median 36 years) who had been admitted for detoxication and rehabilitation to an alcohol treatment center, and from 17 control subjects aged 22–62 years (median 33 years) without any previous alcohol problem. Daily ethanol consumption was evaluated through a questionnaire. Control subjects were asked to complete this questionnaire at the time of the blood collection. Alcoholic patients were asked to complete it twice, first a week after they were admitted, and again two months later. All patients whose answers were divergent were excluded from the study. Those who had stopped drinking a few days before their admittance and those who had reduced their alcohol intake during the last two weeks were also excluded. According to these criteria, the daily ethanol intake was 0–60 g in the control group (mean 10 g) and 80–360 g in the alcoholic group (mean 196 g). All controls were in good health at the time of the blood collection. Alcoholic patients were supposed not to have severe liver injury (ascites or cirrhosis). For ethical reasons, it was not possible to submit alcoholic patients to liver bi-

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