

PLATELET MONOAMINE OXIDASE-B ACTIVITY IN ALCOHOLICS WITH LOWERED DOPAMINE D2 RECEPTOR ACTIVITY

CONOR K. FARREN* and TIMOTHY G. DINAN¹

Yale University School of Medicine, Substance Abuse Treatment Unit, 1 Long Wharf, New Haven, CT 06511, USA and
¹St Bartholomew's Hospital, West Smithfield, London EC1A 7BE, UK

(Received 30 October 1995; in revised form 28 December 1995; accepted 4 January 1996)

This letter is a report on a comparison between two methods of assessment of dopamine function in a group of abstinent alcoholics, one a neuroendocrine challenge test using a dopaminergic probe, and the other a measurement of platelet monoamine oxidase (MAO) enzyme activity. This enzyme is on the dopamine degradation pathway and has been reported as having diminished activity in alcoholics (Pandey *et al.*, 1988; Faraj *et al.*, 1994). Balldin *et al.* (1994) reported in this journal evidence that a group of alcoholics with a reduced dopamine D2 receptor function, as explored using a neuroendocrine challenge, relative to healthy controls, possessed normal platelet monoamine oxidase activity. These authors, however, failed to compare the level of platelet MAO activity between alcoholics with and without reduced dopamine function.

In a previous report (Farren *et al.*, 1995), we found evidence for a diminished dopamine D2 receptor activity in abstinent alcoholics by measuring the growth hormone (GH) response to the dopamine agonist bromocriptine. We measured the GH response over a 3 h period to 1.25 mg of the dopamine D2 agonist bromocriptine administered orally to eight alcoholics and eight healthy controls. We found evidence for reduced dopamine activity in the alcoholics with a reduced delta GH response to bromocriptine, a reduced peak GH response, and an overall reduced GH response over the challenge time points (Farren *et al.*, 1995). Four of the eight alcoholics had no rise in GH above baseline, compared with the control group in which all eight subjects had a rise in GH in response to bromocriptine.

We also measured the platelet MAO activity in the alcoholics, who were abstinent from alcohol for 2.5 weeks or more. The mean age of the alcoholics was 45 years (range 26–54). The mean (\pm SEM) duration of abstinence was 3.6 ± 1.2 weeks. The mean length of drinking history was 25.8 ± 2.9 years, and the mean duration of dependence was 9.1 ± 2.4 years. The platelet MAO activity in the alcoholics with no rise in GH above baseline ($n = 4$) was 0.85 ± 0.025 nmol for MAO-B/min, whereas that for alcoholics with a significant rise in GH above baseline ($n = 4$) was 0.46 ± 0.01 nmol for MAO-B/min; this difference did not reach statistical significance. Thus, we found no evidence for a reduced platelet MAO activity in the subgroup of alcoholics who showed evidence of a significantly diminished dopamine D2 receptor function.

Balldin *et al.* (1994) reported that 11 out of 14 alcoholics measured had a blunted GH response to apomorphine relative to healthy controls, showing evidence for reduced dopamine function in the alcoholics. Apomorphine is a dopamine agonist with primarily dopamine D2 receptor activity, but with some D1 activity as well. They did not report the platelet MAO activity in the group of alcoholics with blunted response, compared with the normally responding alcoholics, although it seems probable that there was no significant difference between those groups, as the number of alcoholics with a normal response ($n = 3$) was so small.

These two reports taken together would indicate that there is normal platelet MAO activity in alcoholics who appear to have reduced dopamine function in two similar neuroendocrine challenge tests. It thus appears that despite neuroendocrine evidence for reduced dopamine activity in some alcoholics and despite evidence for reduced

*Author to whom correspondence should be addressed.

platelet MAO activity in alcoholics, these disturbances appear to be unrelated to each other. The possibility remains that it would take a much larger number of subjects to detect any significant difference in platelet MAO activity between alcoholics grouped by reduced dopamine function, but there is no evidence from these data to support such a hypothesis.

REFERENCES

- Balldin, J., Berggren, U., Lindstedt, G. and Oreland, L. (1994) Alcoholics with reduced dopamine D2 receptor function possess normal platelet monoamine oxidase activity. *Alcohol and Alcoholism* **29**, 659–661.
- Faraj, B. A., Davis, D. C., Camp, V. M., Mooney, A. J. 3rd, Holloway, T. and Barika, G. (1994) Platelet monoamine oxidase activity in alcoholics, alcoholics with drug dependence, and cocaine addicts. *Alcoholism: Clinical and Experimental Research* **18**, 1114–1120.
- Farren, C. K., Clare, A. W., Ziedonis, D., Hameedi, F. A. and Dinan, T. G. (1995) Evidence for reduced dopamine D2 receptor sensitivity in post withdrawal alcoholics. *Alcoholism: Clinical and Experimental Research* **19**, 1520–1524.
- Pandey, G. N., Fawcett, J., Gibbons R., Clarke, C. D. and Davis, J. M. (1988) Platelet monoamine oxidase activity in alcoholism. *Biological Psychiatry* **24**, 15–24.