ETHANOL RAISES PROSTACYCLIN IN VIVO AND IN VITRO

To the Editor:

Landolfi and Steiner’s elegant study1 shows that ethanol increases prostacyclin (PGI2) concentration, both in vitro and in vivo. In the light of recent findings, we would like to add a few comments which may be of relevance.

We have also shown that ethanol enhances the in vitro conversion by rat aortic rings of arachidonic acid to PGI2.2 However, the concentrations of ethanol required (0.5 g/dL)1 were considerably greater than those shown to be stimulatory by Landolfi and Steiner (0.08 g/dL). Furthermore, two other in vitro models (including human umbilical vessel rings) showed that ethanol had no effect on PGI2 synthesis.2 The in vivo 6-oxo-PGF1, measurements should be interpreted with some caution, since others reported median plasma levels of only <1 pg/mL in normal subjects when using gas chromatography-mass spectrometry, which is probably the most sensitive and specific method for plasma 6-oxo-PGF1, assays.3 This value contrasts with the 20.6 pg/mL reported by Landolfi and Steiner.4 The methodological discrepancies2,5 of plasma prostacyclin measurements may induce investigators to assess such data with adequate caution.

Landolfi and Steiner correctly point out that relatively high blood ethanol concentrations are needed to demonstrate significant inhibition of platelet aggregation. However, we have recently shown, using sensitive techniques, that median blood ethanol levels of 0.05 to 0.07 g/dL were considerably greater than those shown to be stimulatory by Landolfi and Steiner (0.08 g/dL). Furthermore, two other in vitro models (including human umbilical vessel rings) showed that ethanol had no effect on PGI2 synthesis.2 The in vivo 6-oxo-PGF1, measurements should be interpreted with some caution, since others reported median plasma levels of only <1 pg/mL in normal subjects when using gas chromatography-mass spectrometry, which is probably the most sensitive and specific method for plasma 6-oxo-PGF1, assays.2 This value contrasts with the 20.6 pg/mL reported by Landolfi and Steiner.4 The methodological discrepancies2,5 of plasma prostacyclin measurements may induce investigators to assess such data with adequate caution.

Landolfi and Steiner correctly point out that relatively high blood ethanol concentrations are needed to demonstrate significant inhibition of platelet aggregation. However, we have recently shown, using sensitive techniques, that median blood ethanol levels of 0.05 to 0.07 g/dL significantly inhibit thromboxane A2 (TXA2, a vasoconstrictor and stimulator of platelet aggregation) release from human platelets, both in vivo and ex vivo. Therefore, when considering the findings of both studies2,1 in conjunction, it would appear that moderate blood ethanol levels alter the PGI2/TXA2; balance in favor of PGI2. These findings lend further support to the view that ethanol-mediated modulation of prostacyclin synthesis contributes to the protection from ischemic heart disease observed with moderate drinking.

References

Ethanol raises prostacyclin in vivo and in vitro [letter]

DP Mikhailidis, JY Jeremy, MA Barradas and P Dandona