Dear Sir:

Ahmed and Sackner [1985] have recently reported increased serum copper in primary pulmonary hypertension and suggested involvement of the α-adrenergic system, especially as dopamine-β-hydroxylase, is copper dependent. I would like to suggest that thromboxane could be involved and is regulated by copper levels.

Thromboxane is involved in pulmonary hypertension [Frolich et al., 1980]. The distribution of copper is unbalanced in the brain and is more concentrated in the left hemisphere [Delva, 1970]. Since copper acts as a coenzyme of dopamine-β-hydroxylase, which converts dopamine into noradrenaline, there is a higher level of noradrenaline in the left hemisphere [Myslobodsky and Weiner, 1978; Oke et al., 1978]. In the pineal, neural stimulation causes the release of noradrenaline which elevates pineal-N-acetyltransferase which controls melatonin production [Namboodiri et al., 1981] and this formation of melatonin is markedly stimulated by noradrenaline [Brownstein and Axelrod, 1974]. Since melatonin is necessary for the thromboxane (TXA2) receptor to be in the active state, increased melatonin can raise TXA2 levels [Horrobin et al., 1980].

Thus, the two concepts of increased serum copper and increased thromboxane can be linked via effects on melatonin production.


