



Corrigendum

Br. J. Pharmacol. (1994), **111**, 652–654.

I. Pörsti, A.T. Bara, R. Busse & M. Hecker. Release of nitric oxide by angiotensin-(1-7) from porcine coronary endothelium: implications for a novel angiotensin receptor.

We have recently reported in this journal that angiotensin-(1-7) is a potent vasodilator substance in isolated porcine coronary arteries by stimulating the release of nitric oxide from the endothelium of these arterial segments through activation of an as yet unidentified angiotensin receptor. Similar findings with this peptide were subsequently made in isolated bovine coronary, rabbit carotid and human mammary arteries as well as in the isolated perfused rat heart. In the course of these investigations, the manufacturer of angiotensin-(1-7), Bachem (Bubendorf, Switzerland), produced a new batch of the peptide (lot no. 506147) which did not exert the specific vasorelaxant effect seen with the previous batch (lot no. 124231). At this time, the relaxation elicited by lot no. 124231 in the isolated porcine coronary artery was confirmed by H. Heyne and W. Osswald (Department of Pharmacology, University of Tübingen, Wilhelmstrasse 56, D-72074 Tübingen, Germany) in an independent series of experiments (unpublished observation). They also confirmed that other batches of angiotensin-(1-7), including lot no. 506147 from Bachem, do not exhibit the endothelium-dependent vasodilator effect seen with lot no. 124231 (Heyne *et al.*, 1995). Both h.p.l.c. and FAB mass spectrometry analysis, independently performed by Bachem and Hoechst (Frankfurt/M., Germany), did not reveal any differences between these two batches of angiotensin-(1-7). It is thus unclear what type of contamination in lot no. 124231 may have caused the reported vasorelaxant effect, since lack of sufficient material of this batch precluded any further chemical analysis. One possibility for its biological activity may be the presence of small amounts of angiotensin-(1-7) with (a) retroinverted peptide bond(s).

There is rising interest in the rather unusual biological actions of angiotensin-(1-7) and we regret any potential confusion regarding its vasorelaxant properties that our report may have caused. However, at the time of publication of this article, this matter was beyond our control.

Reference

HEYNE, N., BEER, W. & OSSWALD, H. (1995). Is the renal response to angiotensin-(1-7) mediated via converting enzyme inhibition? *Naunyn-Schmiedeberg's Arch. Pharmacol.*, **351**, (suppl.), R119 (abstract).