

Erratum

Erratum to ‘Time course of phorbol ester-induced contraction and protein kinase C activation in rat aorta’ [Eur. J. Pharmacol. Mol. Pharmacol. Sect. 290 (1995) 253–257]¹

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Abstract

This study investigates the relationship between the rate of phorbol ester-induced contraction of intact rat aorta and protein kinase C activation, as assessed by the translocation of protein kinase C from the cytosolic to the particulate fraction. Aorta was exposed to Ca^{2+} -free physiological salt solution prior to phorbol ester to prevent Ca^{2+} -induced protein kinase C translocation during tissue homogenization. Phorbol myristate acetate, as well as phorbol dibutyrate, decreased cytosolic and/or increased particulate protein kinase C activity as early as 5 s following ester addition, which was prior to, or coincident with, the onset of contraction. These results suggest that phorbol ester-induced contraction of intact vascular smooth muscle is associated in a time-dependent manner with protein kinase C activation.

Keywords: Smooth muscle, vascular; Ca^{2+} ; Protein kinase C, cytosolic; Protein kinase C, particulate

In the above-mentioned paper, part of Fig. 2 was inadvertently omitted. On the next page, please find the complete Fig. 2.

Our apologies to the authors and readers for this omission.

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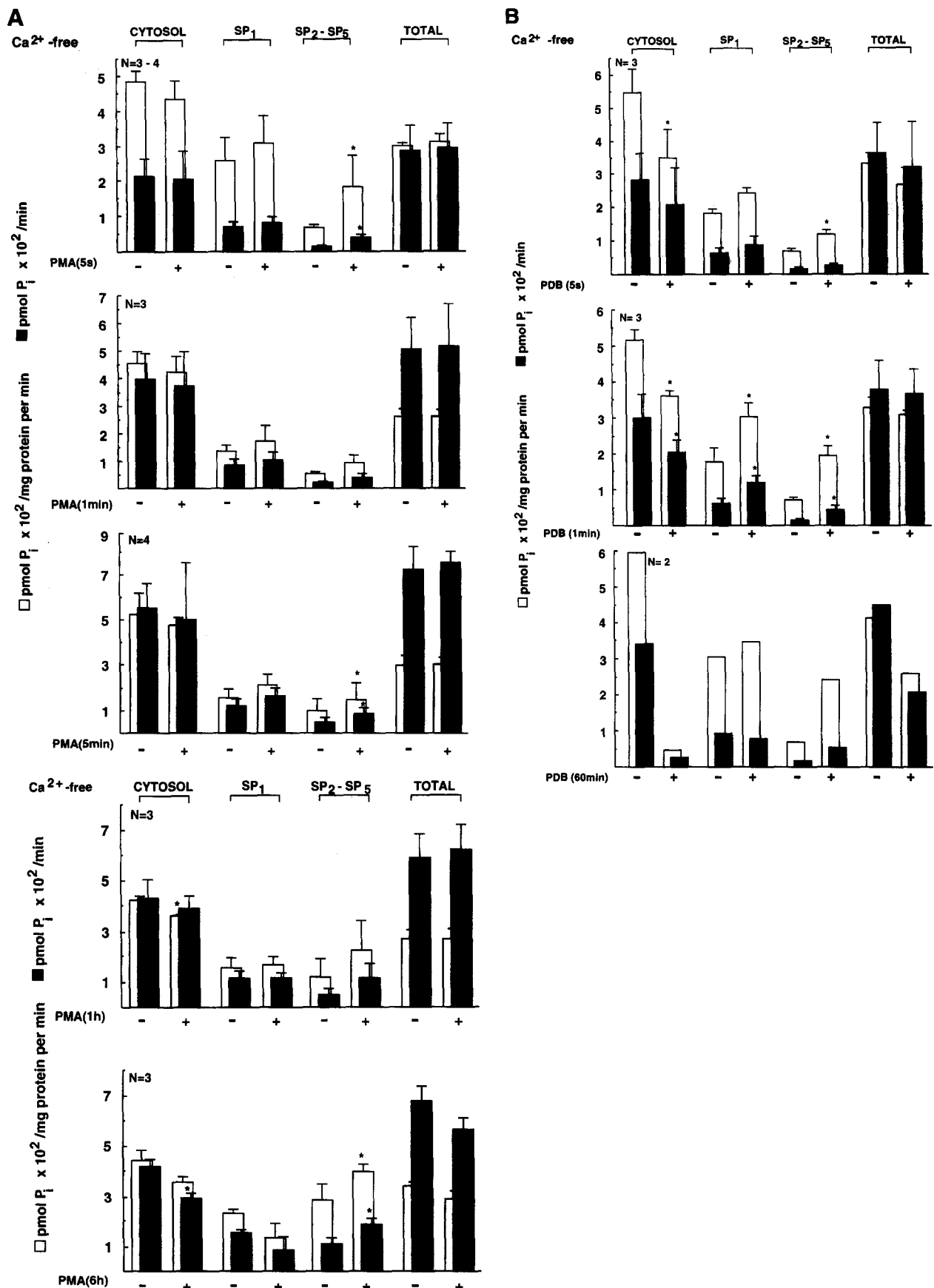


Fig. 2. Effects of time of exposure to phorbol ester on protein kinase C activity in rat aorta exposed to Ca²⁺-free KRB solution containing 2 mM EGTA, followed by (A) 1 μ M PMA or (B) 10 μ M PDB, for periods between 5 s and 6 h. Tissues were then frozen and protein kinase C activity [pmol P_i/mg protein per min (□), pmol P_i/min (■)] measured as described in Materials and methods. Shown are means \pm S.E. and *N* values. * Significantly different from tissues exposed to phorbol ester.