





## **Erratum**

## Erratum to 'Effects of protein kinase A activation on endothelinand ATP-induced signal transduction' <sup>1</sup>

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## Abstract

 $C_6$  glioma cells possess endothelin  $ET_A$  receptor and  $P_2$  purinoceptor coupled to two signaling pathways, i.e. phosphoinositide turnover and inhibition of adenylyl cyclase. In this study, the effects of raising cyclic AMP levels on the inositol phospolipid hydrolysis and adenylyl cyclase inhibition caused by endothelin-1 and ATP in  $C_6$  glioma cells were examined. Pretreatment with cAMP generating agents (forskolin, isoproterenol and cholera toxin) or dibutyryl cAMP for 10 min-3 h did not affect the inositol phosphate accumulation caused by endothelin and ATP. Long-term (8-24 h) pretreatment with isoproterenol, forskolin, cholera toxin or dibutyryl cAMP resulted in a 40-50% inhibition of endothelin- and ATP-stimulated inositol phosphate accumulation, whereas the  $EC_{50}$  values of endothelin and ATP were not affected. Consistent with the effects on endothelin and ATP, NaF-induced inositol phosphate formation was also inhibited by cAMP generating agents to a similar extent. Permeabilized cells from 24 h isoproterenol- or forskolin-pretreated  $C_6$  cells also showed a diminished  $Ca^{2+}$ -sensitivity of phosphoinositide-specific phospholipase C and also attenuated the potentiation response caused by  $GTP\gamma S$ . The inhibitory effects on adenylyl cyclase by endothelin, ATP and 2-methylthio-ATP were unaffected by 24 h pretreatment with isoproterenol or forskolin. Long-term treatment with dibutyryl cGMP did not affect the two signaling pathways caused by ATP and endothelin. It is concluded that the phosphoinositide turnover, but not the adenylyl cyclase inhibition caused by endothelin and ATP in  $C_6$  cells, was inhibited by protein kinase A-dependent pathway. The decreased phospholipase C activity is responsible for the inhibitory effect of protein kinase A-dependent pathway on agonist-induced phosphoinositide turnover in  $C_6$  glioma cells.

Keywords: Phosphoinositide turnover; Protein kinase A; Endothelin; (ATP); C<sub>6</sub> glioma cell

In the above-mentioned paper the errors listed below were not corrected. Our apologies to the author and readers.

The Publishers

p. 3, right, l. 10 should read: 10 µM

p. 4, left, l. 24 should read: endothelin and ATP

p. 4, right, l. 11 should read: As shown in Table 1

p. 5, right, l. 7 should read: endothelin and ATP to inhibit

In the references on p. 7:

right, l. 35 should read: Histamine-induced phospholipid hydrolysis

right, l. 46 should read: inositol phospholipid turnover right, l. 48 should read: 1993b. Endothelin-

left, l. 14 should read: 1990, Phospholipase C

left, l. 24 should read: phosphorylation of phospholipase C.

left, l. 25 should read: 76 kDa protein coprecipitated by anti-phospholipase C

left, l. 53 should read: Proc. Proc. Natl. Acad. Sci. USA

left, l. 55 should read: inositol phospholipid

left, 1. 59 should read: specific phospholipase C

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