MEETING ABSTRACT



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Amphetamine actions rely on the availability of phosphatidylinositol-4,5-bisphosphate

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From 17th Scientific Symposium of the Austrian Pharmacological Society (APHAR). Joint meeting with the Hungarian Society of Experimental and Clinical Pharmacology (MFT) Innsbruck, Austria. 29-30 September 2011

Background

Neuronal functions, such as excitability or endo- and exocytosis, require phosphatidylinositol-4,5-bisphosphate (PIP₂) since ion channels and other proteins involved in these processes are regulated by PIP₂. Monoamine transporters control neurotransmission by removing monoamines from the extracellular space. They also display channel properties, but their regulation by PIP₂ has not been reported. The psychostimulant amphetamine acts on monoamine transporters to stimulate transportermediated currents and efflux and thereby increases the levels of extracellular monoamines.

Methods and results

Direct or receptor-mediated activation of phospholipase C (PLC) reduced membrane PIP₂ and amphetamine-evoked currents through recombinant serotonin transporters; extracellular application of a PIP₂-scavenging peptide mimicked this effect. PLC activation also diminished amphetamine-induced reverse transport without altering transmitter uptake. Inhibition of reverse transport by PLC activation was also observed in brain slices and with recombinant dopamine and noradrenaline, but not GABA transporters; rises in intracellular Ca²⁺ or activation of protein kinase C were not involved in these effects.

Conclusions

These data demonstrate for the first time PIP_2 dependence of reverse transport and current in monoamine transporters.

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Acknowledgements

Supported by FWF (P22893-B11, P17611, SFB3502, SFB3506), and a grant from NIH DA13975.

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Published: 5 September 2011

doi:10.1186/1471-2210-11-S2-A19

Cite this article as: Buchmayer *et al*.: **Amphetamine actions rely on the availability of phosphatidylinositol-4,5-bisphosphate**. *BMC Pharmacology* 2011 **11**(Suppl 2):A19.

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