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Growth Factor-Induced Amino Acid Uptake by Vascular Smooth Muscle Cells

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Abstract

Although accumulating evidence suggests that phosphatidylinositol 3-kinase (PI3K) is a common signaling molecule for growth factor-induced amino acid uptake by the cell, the role of PI3K in the uptake of di erent amino acids was not tested under the same conditions. In this study, we asked whether PI3K mediates platelet-derived growth factor (PDGF)- stimulated uptake of di erent amino acids that are taken up through 3 major amino acid transporters expressed in rat vascular smooth muscle cells and other cell types and whether PI3K mediates amino acid uptake stimulated with di erent growth factors and vasoactive substances. PDGF increased the uptake of [3H] leucine, [3H] proline, and [3H] arginine in a dose- and time-dependent fashion. Two di erent PI3K inhibitors, wortmannin (100 nmol/L) and LY294002 (10 Õmol/L), completely inhibited the amino acid uptake stimulated by PDGF. Chinese hamster ovary cells expressing both PDGF receptor-ɓ and a dominant-negative PI3K did not increase their leucine uptake when stimulated with PDGF, whereas [illegible] both PDG [illegible] and a GF [illegible] PDG [illegible] their [illegible] PDG [illegible] on amino acid uptake was observed in Swiss 3T3 cells. We conclude that PI3K mediates the uptake of di erent amino acids by vascular smooth muscle cells and other cell types stimulated with a variety of growth factors, including transforming growth factor-ɓ. Our ýndings suggest that PI3K may play an important role in vascular pathophysiology by regulating amino acid uptake.

Keywords: [illegible]

Introduction

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