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Implication of ceramide kinase in the regulation of pancreatic cancer cell migration

Pancreatic cancer is the fourth leading cause of cancer mortality with a 5-year survival rate of only 6%. This aggressive disease is characterized by invasiveness, rapid progression and profound resistance to treatment. Using an *in vitro* cell migration assay we have found that ceramide 1-phosphate (C1P) enhances human pancreatic cancer cell migration and invasion potently and that this effect is completely abolished by Pertussis Toxin (PTX), suggesting the participation of a Gi protein-coupled receptor in this process. Activation of the C1P receptor caused phosphorylation and activation of ERK1-2 and Akt and inhibition of these kinases abolished C1P-stimulated cell migration/invasion. We have also observed that human pancreatic cancer PANC-1 and Mia PaCa-2 cells migrate spontaneously. Contrary to the effect of C1P, spontaneous cell migration was insensitive to treatment with PTX. Investigation into the mechanisms responsible for spontaneous migration of the pancreatic cancer cells revealed that Ceramide Kinase (CerK) is a key enzyme in the regulation of this process. In fact, inhibition of CerK activity with the selective inhibitor NVP-231 or treatment with specific CerK siRNA to silence the gene encoding this kinase, potently reduced migration of the pancreatic cancer cells. By contrast, overexpression of CerK stimulated cell migration, an action that was concomitant with prolonged phosphorylation of ERK1-2 and Akt, which are kinases involved in C1P-stimulated cell migration/invasion in a PTX independent manner. It can be concluded that the axis CerK/C1P plays a critical role in pancreatic cancer cell migration/invasion and that targeting CerK expression or activity may be a relevant factor for controlling pancreatic cancer cell dissemination.

Biography

Antonio Gómez-Muñoz received his Ph.D in Biochemistry from the University of the Basque Country, Bilbao (Spain) in 1988. He achieved postdoctoral training at the University of Alberta in Edmonton (Canada) from 1988 to 1994. He then accepted a Research position at the Spanish Research Council (CSIC) from 1995 to 1996. From 1997 to 1998, he worked as Research Associate at the University of British Columbia (Vancouver, Canada). Since then, he has been working at the University of the Basque Country in Bilbao (Spain), where he is currently Professor of Biochemistry and Molecular Biology. He belongs to the Editorial Advisory Board of various scientific journals. Since 2001, he is a member of the International Advisory Committee of the Charleston Ceramide Conference, and since 2005, he is permanent Co-chair of the European Sphingolipid Club. His research interest is on the regulation of lipid metabolism and signalling, and in recent years he focused on the targeting of sphingolipid metabolites and enzymes with the aim of developing new strategies for prevention of disease.

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