

“NUCLEAR LIPID SIGNALLING SYMPOSIUM”

FOREWORD

Nuclear lipid signalling

Since 46 years ago, when Hokin and Hokin presented the first evidence of an enhanced incorporation of [^{32}P] orthophosphate only into phosphatidylinositol (PI) after stimulation of tissue slices with acetylcholine, the biological role of phospholipids has been confined to the structural organisation of cellular membranes. Stemming from this pionieristic observation an increasing body of evidence pointed out the essential role of inositol phospholipids in cellular signalling and more recently become clear that also other phospholipids contribute to the complex machinery of intracellular signalling, namely phosphatidylcholine (PC) and sphingomyelin (SM). The outcome of this lipid signalling, mediated by the so-called second messengers, such as diacylglycerol and inositol-trisphosphate, is the activation of both transcriptional and replicational events at the cell nucleus. In this context, late in the eighties, the very first evidence came out of a new localisation of the inositol lipid cycle, i.e. not only at the

plasma membrane, but also in the nucleus. During the last decade increasing attention has been devoted to this new field of signal transduction, strengthening the concept of a localisation-specific function. Indeed the regulation of the nuclear inositol lipid cycle is temporally and operationally separated from that which takes place at the plasma membrane. It was tempting to believe that inositol phospholipids and other phospholipids capable of generating signals had already revealed their most important secrets, but the discovery of an “autonomous” nuclear lipid signalling system could really pave the way for more and unexpected features. The reviews collected in this issue will certainly clarify a number of aspects, even though it could happen that sometimes more questions than answers will be provided and this is, in a scientific field in continuous progress, the most exciting new.

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