Preface

Remarkable progress has been made in Alzheimer's research over the last decade. Our understanding of which molecular mechanisms are involved in the cascade of events characteristic of the disease is growing. However, the mechanisms by which they induce deleterious effect are still not clear. Many recent studies have confirmed that the synapse is a key target in the pathogenesis of Alzheimer's disease. Not only does synaptic loss occur at an early stage of the disease, but it seems that β-amyloid peptide deposits or microtubular disorders affect synaptic function. This may explain the memory deficits that are so characteristic of the disease. It is therefore logical to investigate therapeutic approaches targeting the deleterious mechanisms that affect the synapse. This area of research promises to be all the more productive because synaptic dysfunction is involved in various other neurodegenerative diseases or, more generally speaking, central nervous system disorders.

This new awareness has emerged at a time when neuroscientific research is uncovering more about synaptic function and the multiple proteins it involves; hence our decision to invite both fundamental researchers in synaptic biology and specialists in neurogenerative disease to the Colloque Médecine et Recherche organized by the Fondation Ipsen in Paris on April 16, 2007.

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