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EDITORIAL

Advanced knowledge on long-known diseases—Proceedings of the 4th Eli Lilly International Foundation Workshop

In January 2007, the 4th Eli Lilly International Foundation Workshop took place in Bad Homburg, Germany. At this occasion, more than 25 scientists from all over Europe came together and presented a broad spectrum of research that has been funded by the Lilly International Foundation. The presentations addressed the biochemical and molecular basis of major diseases including diabetes, obesity, Alzheimer’s disease, leukaemia, and depression and schizophrenia. In this special issue of the Archives of Physiology and Biochemistry we present a collection of review and research papers that specifically address novel molecular insights into the basis of major diseases.

Jones and co-workers report on expression and function of the extracellular calcium-sensing receptor in pancreatic beta cells. They conclude that regulation of beta-cell secretory and proliferative responses by calcium-sensing receptor activation may play an important regulatory role in normal beta-cell function. An intriguing study on human islets is presented by Persaud et al., describing important differences between rodent and human islets and showing that the inhibition of both LOX and COX pathways results in increased insulin secretion in human islets. The cell-type specific regulation of the human 11beta-hydroxysteroid dehydrogenase type 1 promoter is reported by Andres et al. This is of great interest since data from knockout animals show improved glycaemic control and insulin sensitivity. The data presented here suggest a cell-type specific regulation of this enzyme in humans. The paper by Marchini and co-workers entitled ‘SHOX at a glance: from gene to protein’ addresses a gene that was identified as a genetic cause of the short stature phenotype in patients with Turner Syndrome. In addition to summarising the involvement of SHOX in several short stature syndromes, this paper also discusses new evidence suggesting involvement of this protein in growth and bone development.

In their paper, Tauber et al. report that infection with Streptococcus pneumoniae worsens experimental autoimmune encephalomyelitis by elevation of pro-inflammatory cytokines and activation of dendritic cells in the systemic circulation. In their work addressing the effects of Alzheimer’s amyloid-beta and tau protein on mitochondrial function, Rhein and Eckert report that Alzheimer-relevant protein alterations seem to have independent actions at the level of mitochondria. This paper also addresses the potential links between glucose utilisation and energy metabolic defects and the functional alterations associated with brain ageing and with Alzheimer’s disease pathogenesis. In their review paper entitled ‘Effects of insulin-like growth factor binding proteins in bone—a matter of cell and site’, Höflich and co-workers attempt to sort the huge number of in part controversial results on IGF-binding protein effects in bone and try to generate a more specific picture of the functions of these proteins in bone. In the final paper of this issue, Franke and co-workers present evidence that advanced glycation endproducts accumulated in bone may alter osteoblasts by activation of the AGE–RAGE pathway. It is therefore concluded that AGEs may play a functional role in the development of bone diseases like osteoporosis.

We hope that this special issue of APB will help to promote our knowledge on the molecular basis of major diseases and may thus contribute to reinforce ongoing research in these fields with the ultimate goal to improve our therapeutic efforts towards combating these diseases.

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Editor-in-Chief, APB