Growth and development of the islets of Langerhans: implications for the treatment of diabetes

A special section consisting of proceedings from the *Journal of Molecular Endocrinology* Symposium at the 190th Meeting of the Society for Endocrinology, London, 8 November 1999

Foreword

This issue of the journal includes three articles based on talks presented at the *Journal of Molecular Endocrinology* symposium held at the 190th Meeting of the Society for Endocrinology in November 1999. The topic of the symposium, *Growth and development of the islets of Langerhans*, was selected as being timely because of recent advances in our understanding of how a breakdown in the balance between β -cell renewal and destruction might contribute to the development of diabetes mellitus. This theme is developed in the article by Susan Bonner-Weir who elegantly describes how the mass of β -cells can change to compensate for factors such as obesity and insulin resistance that contribute to type 2 diabetes. These compensatory mechanisms include changes in β -cell replication and cell death as well as neogenesis or differentiation of new β -cells from precursor cells located within the pancreatic ducts. Chris Rhodes describes how IGF-1 and GH may be exploited to increase the rate of β -cell proliferation. He provides an authoritative overview of cell signalling pathways that are activated by nutrients and hormones in islets of Langerhans, and describes how this knowledge might be used to generate proliferating human islets for replacement therapy for type 1 diabetes. Mark Dunne and colleagues describe a panel of proliferating human islet cell lines derived from patients with persistent hyperinsulinaemic hypoglycaemia of infancy (PHHI). In keeping with the features of isolated islets from PHHI patients these cells lack operational K_{ATP} channel activity and constitutively secrete insulin at elevated levels. Dunne and colleagues were able to repair this defect by transfecting one such cell line, NES2Y, with genes encoding the components of the K_{ATP} channel. The resultant engineered line was shown to exhibit normal K_{ATP} channel activity and to secrete insulin in response to changes in glucose concentrations within the physiological range.

All three articles highlight the progress that has been made in recent years in understanding the mechanisms involved in regulating β -cell replication and differentiation. They also highlight the way in which this knowledge might contribute to the aetiology and treatment of diabetes mellitus.

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