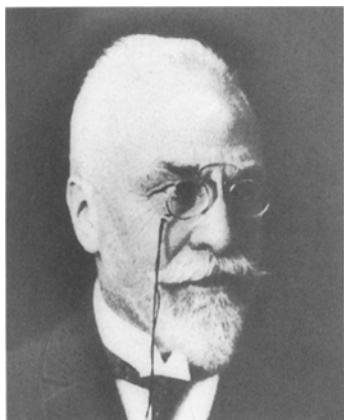


Introduction



Oskar Minkowski
(1858–1931)

Until the mid 19th century the cause of diabetes mellitus was localized in the kidney, the stomach and liver. It was Claude Bernard in 1847 who focused attention on the pancreas while discovering glycogen in the liver. The fact, however, that destruction or disturbance of certain areas in the brain and/or the peripheral nervous system lead to tempo-

rary glycosuria (first described by Claude Bernard in 1849) favoured the hypothesis of the so-called angioneurotic diabetes or *diabète nerveuse*. In 1855 Claude Bernard further explored the function of the pancreas by injecting the pancreatic duct of dogs with paraffin or fat. This led to an almost complete atrophy of the exocrine pancreas of the dogs, however, without inducing diabetes. For the first time in 1886, Joseph von Mering induced an experimental temporary diabetes by injecting phlorizin into dogs; however, he did not recognise islet dysfunction as the cause of the disease.

In studying fat resorption Joseph von Mering ligated the pancreatic duct of dogs, but was unable to achieve complete inhibition of fat digestion and resorption not being aware of the existence of accessory ducts in the dog pancreas. It was Oskar Minkowski who proposed to Mering to remove the whole pancreas to overcome these problems and to study further the role of the pancreas in fat digestion. The surgical procedure performed by Minkowski with the assistance of Mering was a complete success. The animal survived. While von Mering was out of town for a week, Minkowski noted a massive polyuria and measured severe glycosuria. Within a few days several dogs underwent surgical removal of the pancreas by Minkowski. All became severely diabetic. These findings were published in 1889 by von Mering and Minkowski in a short communication. After this publication von Mering had no special interest in further experiments, while Minkowski expanded his studies, since it was not clear what had induced diabetes after pancreatectomy. By feeding pancreas to

pancreatectomized animals he was not able to eliminate diabetes. Subcutaneous implantation of vascularized pieces of pancreatic tissue, however, improved glycosuria, suggesting that the cause of diabetes must lie within the pancreas. From 1909 to 1913 Minkowski also performed parabiosis experiments. Pancreatectomized dogs were connected to healthy dogs by carotid artery anastomosis. These experiments led to a significant reduction of glycosuria. This proved that the pancreas must release a substance into the bloodstream which improves diabetic control. These studies supported the idea of Laguesse and Diamare who in 1893 postulated that the islets of Langerhans, known since 1869 must have inner secretory capacity. In 1895 Sir Edward Sharpey-Schäfer speaking before the British Medical Association assessed the role of islets in diabetes in the following sentence: "The only fact that appears certain in connection with the manner in which the pancreas prevents excessive production of sugar within the body is that this effect must be produced by the formation of some material, secreted internally by the gland and probably by the interstitial vascular islets and that this internally secreted material profoundly modifies the carbohydrate metabolism of the tissues".

Acknowledging the importance of Oskar Minkowski's findings, in particular his persistence in performing research in the field of diabetes, the EASD decided at its second Council Meeting in 1965 in Montecatini to honour young scientists below the age of forty for outstanding diabetes research with a Minkowski prize.

Due to the generous support of Hoechst AG since 1966 the prize has been associated with a considerable amount of money. The EASD has never had difficulty in finding members fulfilling the high standard of this prize. It is more difficult for the Minkowski prize subcommittee to select from numerous excellent applicants the most suitable candidate.

The prize is given for work that has been performed and published up to the age of forty. The idea of this Symposium was to see what had happened to the past prize winners and their scientific performances in the intervening years. Fortunately all the prize winners are still active in science and therefore it was a great opportunity to gather all of them in a scientific meeting in close connection to the annual meeting of the EASD in Istanbul.

On behalf of the organizers and the EASD I would like to thank all those who have been involved in the

organisation of this Symposium and made it possible to publish, in particular Dr. Reden, Dr. Brocks, and Mr. Elmenthaler from Hoechst Company. Unfortunately, two prize winners, namely Dr. Lars Carlson (Louvain, 1968) and Dr. Lelio Orci (Brussels, 1973) were unable to attend the meeting. However, all the other prize winners were present and actively participated in this historical scientific meeting. The content of this Diabetologia Supplement represents the contributions of the prize winners at the meeting. I would like to thank the past Editor-in-Chief, Claes Hellerström, and the present Editor-in-Chief, Ele Ferrannini, for their interest in and support of the publishing plans. All contributions were reviewed by

an editorial board consisting of Dietrich Brocks, Claes Hellerström, Rüdiger Landgraf (chairman), and Åke Lernmark. We hope that the readers will enjoy the following pages as much as did the colleagues who were able to attend the meeting. Those who have been involved in the prize selection committee through the years can be satisfied that they have chosen outstanding scientists still active even 27 years after receiving the Prize. For those who will apply for the Minkowski Prize in the future, this symposium has set the standard.

Rüdiger Landgraf, Munich
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