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2. To unravel genetic abnormalities in relation to development of coronary heart disease.

3. Evalation of "new" cardiovascular risk factors.

4. To follow the patient groups prospectively in order to evaluate the importance of genetic and non-genetic factors for the development and progression of diabetic micro- and macroangiopathy.

Thus, it is evident that the material will be the same in the past and in the publications to come. However, different measurements will be applied to evaluate the different topics referred to above, exactly as was carried out in the publications mentioned in Diabetologia and Diabetes. In the Diabetes paper we were studying a role of the insertion/deletion polymorphism in the angiotensin converting enzyme in relation to diabetic nephropathy and diabetic retinopathy. The paper published in Diabetologia was dealing with the importance of the same polymorphism but in relation to the development of coronary heart disease, of course taking into account numerous well-known cardiovascular risk factors. Finally, we would like to stress that large well-characterized patient materials can be used for the answering of many different questions as attempted in our study. Without comparison, we would like to mention that in addition to the original publication of the Diabetes Control and Complications Trial (DCCT) data in the New England Journal of Medicine an estimated 30–40 additional publications from that study will be available, as well as the numerous important publications in the British Medical Journal by D. J. P. Barker et al. based on the Hertfordshire material.

Yours sincerely,

L. Tarnow H.-H. Parving

# Errata

**Diabetologia** Volume 38 Number 9 pp 1014–1024

C.K.Leow, D.W.R.Gray, P.J.Morris

## The long-term metabolic function of intraportal and renal subcapsular islet isografts and the effect of glomerular basement membrane thickness in rats

Due to an error part of the legend to Figure 1 was transposed, the correct legend is printed here

**Fig.1.** Non-fasting plasma glucose of rats given 1000 or 3000 islets IP or SC and followed for either 6 months or 12 months, compared to normal and diabetic controls.  $(\triangle \dots \triangle)$  1000 islets IP followed 6 months,  $(\bullet \dots \bullet)$  1000 islets SC followed 6 months,  $(\circ \dots \circ)$  3000 islets IP followed 6 months,  $(\bullet \dots \bullet)$  3000 islets SC followed 6 months,  $(\bullet \dots \bullet)$  1000 islets IP followed 12 months,  $(\bullet \dots \bullet)$  1000 islets SC followed 12 months,  $(\bullet \dots \bullet)$  1000 islets SC followed 12 months,  $(\bullet \dots \bullet)$  1000 islets SC followed 12 months,  $(\bullet \dots \bullet)$  1000 islets SC followed 12 months,  $(\bullet \dots \bullet)$  3000 islets SC followed 12

## Diabetologia

Volume 38 Number 10 pp 1249–1250

#### The glycogen synthase gene in NIDDM and hypertension

Y. Hamada, H. Ikegami, Y. Fujioka, E. Yamato, K. Takekawa, T. Fujisawa, Y. Nakagawa, H. Ueda, J. Fu, G.-Q. Shen, T. Miki, T. Ogihara

Due to an unfortunate error Dr. Miki's name was incorrectly printed in the above-mentioned letter to the editor