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Insertion/deletion polymorphism in the angiotensin-I-converting enzyme gene

Dear Sir,
We read with great interest the recent paper published in Diabetologia by Tarnow et al. [1].

A considerable part of the data in this paper is also contained in a paper recently published in Diabetes [2] by the same group. These areas include the 'Subjects and methods' section which is very similar between the two papers. The data in Table 1 of both papers is more than 75 % identical.

The beginning of the 'Results' section contains identical data and the text is the same. Both papers present the results of angiotensin converting enzyme (ACE) genotyping in patients with nephropathy compared to those with normoalbuminuria. The Diabetes paper also presents these data in a Table. Both papers have presented the same plasma ACE levels with respect to ACE genotypes as well as nephropathy and normoalbuminuria. The Diabetes paper has also presented the data in a Table.

It is of particular interest that both papers were received on the same day (10 October 1994) in the editorial offices of Diabetologia and Diabetes. The Diabetologia paper makes only a brief reference to the Diabetes paper [ref. 36]. The revised form of the Diabetologia paper was received on 20 December 1994 whilst the revised Diabetes paper was received on 19 January 1995. With respect to the Diabetes paper the authors must have known that the Diabetologia paper was likely to be accepted yet no mention of its existence or the data regarding coronary heart disease (CHD) has been made. Consequently, the discussion in the Diabetes paper has been written with no mention of the role of CHD in nephropathy despite the authors' having a paper which looks at CHD in exactly the same group of patients. This is very misleading for the reader and the discussion in the Diabetes paper has been written without disclosing the full facts.

Finally, the 'Statement to be signed by all authors' for submission of manuscripts to Diabetologia states 'We confirm that neither the manuscript submitted nor any part of it has been published or is being considered for publication elsewhere . . . ' clearly this is not the case. Likewise, Diabetes also requires a statement where 'authors must state in their transmittal letter that the material has not been published or submitted simultaneously to another journal'.

We believe that it is very poor scientific practice for a group to attempt to publish data twice.

Yours sincerely,
A. Demaine, M. Hibberd, A. Millward

References

1. Tarnow L, Cambien F, Rossing P et al.: (1995) Insertion/deletion polymorphism in the angiotensin-I-converting enzyme gene is associated with coronary heart disease in IDDM patients with diabetic nephropathy. Diabetologia 38: 798–803
2. Tarnow L, Cambien F, Rossing P et al.: (1995) Lack of relationship between an insertion/deletion polymorphism in the angiotensin I-converting enzyme gene and diabetic nephropathy and proliferative retinopathy in IDDM patients. Diabetes 44: 489–494

Response from the authors

Dear Sir,
Drs. A. Demaine, M. Hibberd, and A. Millward believe 'that it is very poor scientific practice for a group to attempt to publish data twice'. We completely agree and consequently we have never done so and do not intend to in the future. Actually, Diabetologia received a full copy of the paper submitted to Diabetes before acceptance. However, we are puzzled and astonished over the accusation since we are planning to publish many more papers (an additional one has already been accepted by Diabetes and another two have been submitted to Diabetes/Diabetologia) based on our large case/control study consisting of 198 insulin-dependent diabetic (IDDM) patients with diabetic nephropathy and a matched group of 190 IDDM patients with normoalbuminuria. The two groups have been carefully evaluated in relation to demographic data, microangiopathy, macroangiopathy, autonomic neuropathy, cardiovascular risk factors, endothelial dysfunction etc. DNA was isolated from all patients. The reason for conducting such a large case/control study was to answer questions within the following topics:
1. Importance of genetic abnormalities in relation to initiation and progression of diabetic nephropathy and diabetic retinopathy.

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. (△---△) 1000 islets IP followed 6 months, (●---●) 1000 islets SC followed 6 months, (○---○) 3000 islets IP followed 6 months, (▲---▲) 3000 islets SC followed 6 months, (■—■) 1000 islets IP followed 12 months, (●—●) 1000 islets SC followed 12 months, (▲– – –▲) 3000 islets IP followed 12 months, (△– – –△) 3000 islets SC followed 12 months, (□—□) normal controls from 6 month follow up groups, (■---■) normal controls from 12 month study groups, (□---□) diabetic controls form 6 month study groups, (▲—▲) diabetic controls from 12 month study groups. Error bars indicate SEM

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