Author’s response to reviews

Title: Vitamin D receptor initiation codon polymorphism influences genetic susceptibility to type 1 diabetes mellitus in the Japanese population

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PDF covering letter
June 22th, 2001

BioMed Central Editorial

Re: MS# 53614762221390

Title: “Vitamin D receptor initiation codon polymorphism influences genetic susceptibility to type 1 diabetes mellitus in the Japanese population.”

Authors: Ban et al.

Dear Editors:

Enclosed please find the revised version of our manuscript. Major revisions have been made after carefully considering the issues raised by the referees.

Responses to referee I (Klaus Badenhoop):

Comments:

1. We revised detailed information on Results and Conclusions of the Abstract.

2. We added data on the FokI genotypes of another 50 female controls, and added the comment that the controls used in the present study are identical with the previously published controls (Materials and Methods, Results, Table 1, and Table 2).

3. We discussed the recent finding of Colin et al. about the functional relevance of the VDR FokI polymorphism (Discussion).

Minor points:
1. We revised it (Abstract).

2. We excluded the information on the role of vitamin D for insulin secretion/ type 2 diabetes (Background).

3. We added the information about a Tru9I polymorphism (Ye et al., 2000) (Background).

4. We revised it (Background).

5. We revised it (Materials and Methods).

6. We explained the abbreviation of MS (multiple sclerosis) (Discussion).

7. We revised it (Discussion).

Responses to referee II (Michael McDermott):

**Comments:**

1. We used T1DM as the accepted abbreviation for type 1 diabetes mellitus.

2. We revised the comment that the BsmI, Tru9I, and ApaI restriction fragment length polymorphisms (RFLPs) located between exons 8 and 9, the TaqI RFLP located in exon 9, and that there is apparently no significant linkage disequilibrium between the FokI polymorphism and the BsmI, ApaI, and TaqI polymorphisms (Background).

3. We added the comment that the VDR plays a role in lymphocyte response to microorganisms (tuberculin reactive status in pulmonary tuberculosis, leprosy etc.), so it is conceivable that it may also be involved in immune response to self antigens e.g. GAD65 antibodies (Conclusions).

Additionally, other minor corrections were made. These changes have addressed all of
the critiques of the reviewers. We appreciate the helpful suggestions offered by the two referees, and their comments were very useful for revising this manuscript. I hope you now find this manuscript suitable for publication.

Sincerely yours,

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