Author’s response to reviews

Title: Genetic mutations in SPINK1, CFTR, CTRC in acute pancreatitis

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Version: 5 Date: 29 November 2014

Dear Editor,

We would like to submit our manuscript entitled ‘Genetic mutations in SPINK1, CFTR, CTRC in acute pancreatitis’ by Koziel D et al., for consideration for publication in your BMC Gastroenterology.

The following changes were introduced according to the reviewers’ comments:

1. Title of the manuscript: changed

2. Abstract: the methodological part was corrected, the sentence concerning the results and relationship between SINK1 mutations and etiology of AP was reformatted, and conclusions shortened.

3. Introduction: references were shortened and supplemented [5], the role of CFTR and chronic ER stress in pancreatic acinar cells discussed [15,16], it was explained in what way the mutation of Claudin2 protein leads to pancreatitis [18].

4. Material and methods: The principles of the qualification of patients into the study were explained according to the cause of the disease (alcohol-related, biliary). Principles of selection of the control group were discussed: age, gender. Information concerning the expression of consent by the Bioethical Commission were transferred from the section: Statistical analysis

5. Results: Figures 1-7 were removed, the results repeated in tables were omitted in the text, Table 2 was supplemented by calculations of odds ratio with 95% confidence intervals, table legends were removed from this part of the manuscript.

6. Discussion: abbreviated
7. References: references No. 33, 15 were removed

The language was revised and tables corrected from the editorial aspect.

In reply to the reviewers’ comments we would like to explain the following:

1. In all participants exons 2 and 3 of PRSS1 gene were analysed by HRM and Capillary Sequencing. We did not find any mutations in all but one control subject (pI94L). Analysis of patient samples were always accompanied by the analysis of control DNA (exon 2 [p.N29I, p.A16V] i 3 [p.R116C, p.R122D, p.R122C]. Positive control DNA were kindly provided by dr Katarzyna Wertheim - Tysarowska, Institute of Mother and Child, Department of Medical Genetics, Laboratory of Hereditary Diseases Research Warsaw.

2. In the examined material we did not encounter pancreas divisum, therefore, such a situation was not described.

3. In the manuscript it was previously stated: In one patient with a moderately severe course of AP of alcohol etiology with recurrence of the disease, the presence of p.N34S mutation in SPINK1 and p.V235I mutation in CTRC was observed.

We believe our findings would appeal to the readership of BMC Gastroenterology.

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We look forward to hearing from you at your earliest convenience.

All authors have approved the manuscript and agree with its submission to BMC Gastroenterology.

Yours Faith fully,

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