Review of: "Exploring the Autoimmune Hypothesis of Type 1 Diabetes: Investigating the Potential Role of Peritoneal Membrane Defects in the Pancreatic Tail and Revisiting Alternative Theories of Disease Etiology"

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Potential competing interests: No potential competing interests to declare.

It is an interesting and useful article on the approach to type I diabetes. The information is not complete; more studies are needed. There are also aspects that should be revised or reformulated.

Clarity

3/5

News

3/5

Impact

2/5

Merits

3/5

,,The pancreatic tail is a critical site for endocrine function, housing approximately 25-30% of the pancreas's beta cells, which are responsible for insulin secretion [8]. “

I did not find this statement in the reference you mentioned.

,,This region also has a relatively higher density of GLP-1 receptors, which play a significant role in enhancing insulin secretion and beta-cell proliferation [9].

,,Glucagon-like peptide-1 (GLP-1) and glucagon-like peptide-1 receptor (GLP-1 R)

A promising candidate is the incretin glucagon-like peptide-1 (GLP-1) and its respective receptor, the glucagon-like peptide 1 receptor (GLP-1R). This receptor was cloned approximately 25 years ago (Thorens et al. 1993). Similar to the SSTR, it is a member of the class 2 G-protein-coupled receptor family. Only a single GLP-1R is known so far, which is

structurally identical in all tissues (Thorens et al. 1993).

The GLP-1R is of clinical interest not only due to its physiological expression and functions in pancreatic islet cells and its established role in the therapy of type 2 diabetes using GLP-1 analogs (Nauck 2016), but also because of its possible role in cancer."

This is a fragment from the paper mentioned in reference no. 9.

In this text, at reference no. 9, I did not find data related to the proliferation of beta cells and the increase in insulin secretion. Data on GLP-1 R are mentioned in the respective study.

,,A potential defect in the peritoneal membrane or its associated immune-regulatory[10] mechanisms could allow autoreactive immune cells or antibodies to access and destroy beta cells specifically in this region, leading to the onset of T1DM [11]."

I read the article from reference 11, and the connection between the peritoneal membrane and the occurrence of T1DM is not presented.

,, An extensive literature search was conducted across multiple databases, including PubMed, Scopus, and Web of Science, to gather relevant studies on the autoimmune hypothesis of T1DM, pancreatic anatomy, peritoneal membrane defects, and related immunological processes."

During what period did you conduct the research for the specialized literature?

,,In light of the findings, specific research directions were proposed to test the validity of the new hypothesis, including experimental designs to verify the impact of peritoneal defects on beta-cell autoimmunity and clinical studies aimed at identifying potential biomarkers related to peritoneal integrity in T1DM patients."

Are you referring to research directions specific to humans or animal research?

,, This location allows the tail to be more accessible to potential peritoneal defects, making it a key focus in understanding the etiological factors contributing to diseases like Type 1 Diabetes Mellitus (T1DM)."

What is the bibliographic reference for this statement?

The tail of the pancreas is particularly significant from an endocrine perspective, as it contains a higher concentration of islets of Langerhans compared to other regions of the pancreas. It is estimated that approximately 25-30% of the pancreas's beta cells reside in the tail region, which is responsible for insulin production [16]"

The phrase is repeated above, except that it has reference number 8.

I congratulate you on your effort and the data provided.

My comments are only intended to make the paper better. Good luck!