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Contents

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MINIREVIEWS

70 Metabolically healthy obesity: Is it really healthy for type 2 diabetes mellitus?

Wu Q, Xia MF, Gao X

85 Role of dipeptidyl peptidase 4 inhibitors in the new era of antidiabetic treatment

Florentin M, Kostapanos MS, Papazafiropoulou AK

ORIGINAL ARTICLE

Clinical and Translational Research

97 Altered spontaneous brain activity patterns in patients with diabetic retinopathy using amplitude of low-frequency fluctuation

Shi WQ, Zhang MX, Tang LY, Ye L, Zhang YQ, Lin Q, Li B, Shao Y, Yu Y

Retrospective Study

110 Large-scale functional connectivity predicts cognitive impairment related to type 2 diabetes mellitus

Shi AP, Yu Y, Hu B, Li YT, Wang W, Cui GB

LETTER TO THE EDITOR

126 Inflammatory bowel disease and diabetes: Is there a link between them?

Sang MM, Sun ZL, Wu TZ

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Monthly Volume 13 Number 2 February 15, 2022

Contents

ABOUT COVER

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AIMS AND SCOPE

The primary aim of *World Journal of Diabetes* (*WJD*, *World J Diabetes*) is to provide scholars and readers from various fields of diabetes with a platform to publish high-quality basic and clinical research articles and communicate their research findings online.

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LETTER TO THE EDITOR

Inflammatory bowel disease and diabetes: Is there a link between them?

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Abstract

Patients with inflammatory bowel disease (IBD) are reported to have an increased risk of diabetes. IBD therapies may also modulate blood glucose substantially. These observations are indicative of mechanistic connection(s) between IBD and diabetes.

Key Words: Inflammatory bowel disease; Abnormal glucose metabolism

Core Tip: Inflammatory bowel disease is associated with an increased risk of diabetes. Mechanistic insights into their common pathogenesis may render novel therapeutic targets for these major chronic disorders.

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TO THE EDITOR

We read with interest the recent review, entitled "Effect of inflammatory bowel disease treatments on patients with diabetes mellitus", by Bower *et al*[1], which

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provided an excellent summary on the effects of different agents recommended for the treatment of inflammatory bowel disease (IBD) on glucose metabolism. These findings highlight the need for clinicians to consider the impact of IBD-related drugs on blood glucose control among IBD patients with diabetes, and also provide strong impetus to understand the potential mechanistic connection(s) between IBD and the onset of diabetes mellitus.

IBD refers to nonspecific chronic intestinal inflammatory conditions, including Crohn's disease (CD) and ulcerative colitis (UC). In the pursuit of the pathogenesis underlying IBD, 99 susceptibility loci/genes have been found to be related to IBD *via* genome-wide association studies. Interestingly, among those loci/genes, many are also associated with the risk of metabolic diseases, including type 1 and 2 diabetes[2]. A recent nationwide Danish cohort study has reported an increased risk of type 2 diabetes in patients with CD and UC, independent of glucocorticoid use[3]. Similarly, an elevated risk of type 1 diabetes was reported in pediatric patients with UC[4]. More recently, Jasser-Nitsche *et al*[5]. observed that in the German and Austrian population, children and adolescents with type 1 diabetes are at increased risk of IBD. These observations are, therefore, indicative of shared pathway(s) of pathogenesis between IBD and diabetes.

It is now widely appreciated that the gastrointestinal tract plays an important role in glucose homeostasis[6]. In recent years, there is mounting evidence that the gut microbial metabolites and their ensuing effects on the intestinal and systemic inflammation are associated with the occurrence and progression of diabetes; approximately 90% of type 2 diabetes is related to the disrupted gut microbiota, *i.e.* dysbiosis[7], a phenomenon also seen in IBD[8]. In the Danish cohort of IBD, specific abnormal microbial features are linked to the risk of type 2 diabetes[3]. Accordingly, dysbiosis may represent a common pathogenic factor of both IBD and dysglycemia.

Intestinal and metabolic homeostasis is also regulated by a number of gut-derived hormones, as a result of complex interactions between the ingesta and enteroendocrine cells. The incretin hormone glucagon-like peptide (GLP-1) is secreted from L-cells, which predominate in the distal small and large intestine[9]. GLP-1 regulates blood glucose metabolism *via* pleotropic actions, including stimulation of insulin secretion, suppression of glucagon secretion and energy intake, and slowing of gastric emptying [10]. In rodents, GLP-1 was reported to attenuate intestinal mucositis induced by chemotherapy[11]. In both patients with UC and CD and mice with colitis, the expression of GLP-1 receptor of intestinal biopsies was found to be reduced[12]; treatment with the GLP-1 receptor agonist, liraglutide, reduced levels of colonic inflammation in mice with colitis[12]. Accordingly, the reduction in the expression of dipeptidyl peptidase-4 - the enzyme that inactivates endogenously released GLP-1 – in the inflammatory bowel of patients with CD may have reflected a compensatory response of the gut to the development of inflammation[13].

Despite the reported association between the onset of IBD and diabetes, and the potential influence of IBD therapies on glucose metabolism, the common pathogenesis of IBD and diabetes remains elusive. Understanding the latter may provide novel therapeutic opportunities for these major chronic disorders.

REFERENCES

1 **Bower JAJ**, O'Flynn L, Kakad R, Aldulaimi D. Effect of inflammatory bowel disease treatments on patients with diabetes mellitus. *World J Diabetes* 2021; **12**: 1248-1254 [PMID: 34512890 DOI: 10.4239/wjd.v12.i8.1248]
2 **Lees CW**, Barrett JC, Parkes M, Satsangi J. New IBD genetics: common pathways with other diseases. *Gut* 2011; **60**: 1739-1753 [PMID: 21300624 DOI: 10.1136/gut.2009.199679]
3 **Jess T**, Jensen BW, Andersson M, Villumsen M, Allin KH. Inflammatory Bowel Diseases Increase Risk of Type 2 Diabetes in a Nationwide Cohort Study. *Clin Gastroenterol Hepatol* 2020; **18**: 881-888.e1 [PMID: 31394285 DOI: 10.1016/j.cgh.2019.07.052]
4 **Kappelman MD**, Galanko JA, Porter CQ, Sandler RS. Association of paediatric inflammatory bowel disease with other immune-mediated diseases. *Arch Dis Child* 2011; **96**: 1042-1046 [PMID: 21903597 DOI: 10.1136/archdischild-2011-300633]
5 **Jasser-Nitsche H**, Bechtold-Dalla Pozza S, Binder E, Bollow E, Heidtmann B, Lee-Barkley YH, Raile K, de Sousa G, Schramm U, Holl RW. Comorbidity of inflammatory bowel disease in children and adolescents with type 1 diabetes. *Acta Paediatr* 2021; **110**: 1353-1358 [PMID: 33119925 DOI: 10.1111/apa.15643]
6 **Holst JJ**, Gribble F, Horowitz M, Rayner CK. Roles of the Gut in Glucose Homeostasis. *Diabetes Care* 2016; **39**: 884-892 [PMID: 27222546 DOI: 10.2337/dc16-0351]
7 **Arora A**, Behl T, Sehgal A, Singh S, Sharma N, Bhatia S, Sobarzo-Sanchez E, Bungau S.

Unravelling the involvement of gut microbiota in type 2 diabetes mellitus. *Life Sci* 2021; **273**: 119311 [PMID: 33662428 DOI: 10.1016/j.lfs.2021.119311]

8 **Joossens M**, Huys G, Cnockaert M, De Preter V, Verbeke K, Rutgeerts P, Vandamme P, Vermeire S. Dysbiosis of the faecal microbiota in patients with Crohn's disease and their unaffected relatives. *Gut* 2011; **60**: 631-637 [PMID: 21209126 DOI: 10.1136/gut.2010.223263]

9 **Xie C**, Jones KL, Rayner CK, Wu T. Enteroendocrine Hormone Secretion and Metabolic Control: Importance of the Region of the Gut Stimulation. *Pharmaceutics* 2020; **12** [PMID: 32825608 DOI: 10.3390/pharmaceutics12090790]

10 **Meier JJ**. GLP-1 receptor agonists for individualized treatment of type 2 diabetes mellitus. *Nat Rev Endocrinol* 2012; **8**: 728-742 [PMID: 22945360 DOI: 10.1038/nrendo.2012.140]

11 **Kissow H**, Hartmann B, Holst JJ, Poulsen SS. Glucagon-like peptide-1 as a treatment for chemotherapy-induced mucositis. *Gut* 2013; **62**: 1724-1733 [PMID: 23086829 DOI: 10.1136/gutjnl-2012-303280]

12 **Bang-Berthelsen CH**, Holm TL, Pyke C, Simonsen L, Søkilde R, Pociot F, Heller RS, Folkersen L, Kvist PH, Jackerott M, Fleckner J, Vilién M, Knudsen LB, Heding A, Frederiksen KS. GLP-1 Induces Barrier Protective Expression in Brunner's Glands and Regulates Colonic Inflammation. *Inflamm Bowel Dis* 2016; **22**: 2078-2097 [PMID: 27542128 DOI: 10.1097/MIB.0000000000000847]

13 **Moran GW**, O'Neill C, Padfield P, McLaughlin JT. Dipeptidyl peptidase-4 expression is reduced in Crohn's disease. *Regul Pept* 2012; **177**: 40-45 [PMID: 22561447 DOI: 10.1016/j.regpep.2012.04.006]

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