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The potential role of gut microbiota in pancreatic disease: A systematic review

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Abstract

Background: Several studies have suggested a link between microbiota imbalance and a few gastrointestinal, inflammatory and neoplastic diseases. However, the role in pancreatic diseases remains unclear. to guage the available evidence for pancreatic diseases, we undertook a scientific review. Methods: OVID Medline (1946 to 2017), EMBASE (1980 to 2017) and therefore the Cochrane Central Register of Controlled Trials (CENTRAL Issue 3, 2017) were explore for studies on microbiota in pancreatic disease. We also searched the reference lists of retrieved papers and conference proceedings. We excluded animal studies, reviews, and case reports. Results: a complete of two,833 articles were retrieved. After screening and applying the exclusion criteria, 10 studies were included.

Three studies showed lower levels of Bifidobacterium or Lactobacillus and better levels of Enterobacteriaceae in chronic pancreatitis. Two of those studies were uncontrolled, and also the third (controlled) study which compared patients with endocrine and exocrine insufficiency, reported that Bacteroidetes levels were lower in those patients without diabetes, while Bifidobacteria levels were higher in those without exocrine insufficiency. only 1 study investigated acute pancreatitis, showing higher levels of Enterococcus and lower levels of Bifidobacterium versus healthy participants. There was an overall association between carcinoma and lower levels of Neisseria elongate, Streptococcus mitis and better levels of Porphyromonas gingivalis and Granulicatella adiacens. Conclusions: Current evidence suggests a possible link between microbiota imbalance and carcinoma. Regarding acute and chronic pancreatitis, data are scarce, dysbiosis appears to be present in both conditions. However, further investigation is required to substantiate these findings and to explore therapeutic possibilities.

Despite the colonization of the human digestive tube, only 10 are determined as carcinogenesis by International Agency for Cancer Research which indicates complex relations existing among GM, environmental triggering factors and cancer initiation There is flourishing evidence demonstrating changes in physical characteristics resulting from GM overgrowth GM disorders reduce the thickness of mucus layer, which is usually followed by a decreased antimicrobial defense and fewer gut peptides from L-cells and limited short chain fatty acids .As a consequence, transformations of relative microbial metabolites result in subsequent chain reactions. The reduced gut peptides, like glucagon-like peptide-1 and peptide YY, play regulatory roles in food intake and glucose metabolism. Moreover, PPAR-γ inactivation resulting from lack of SCFAs requires higher oxygen available for the microbiota at the proximal mucosa and promotes Enterobacteriaceae proliferation. Despite with low concentrations, bacterial metabolites, like lipopolysaccharides (LPS), flagellin and SCFAs, can reach the circulation and distant organs via paracellular diffusion or co-transport with chylomicrons.

To be specific, LPS, a crucial cytoderm component of gram-negative bacteria, can interact with several Toll-like receptor (TLR) signaling pathways with a definite structural composition from other bacterial taxa Furthermore, lipoteichoic acid (LTA), a surface component of gram-positive bacteria and a key virulence factor, has been found to trigger the over-secretion of proinflammatory factors by binding to CD14 or TLR2 This activity may lead to the final word development of PC, as illustrated by its involvement in chronic pancreatitis progression on mice with infection of Enterococcus faecalis.Another microbial metabolite generated by 7α-dehydroxylating bacteria and secondary bile acids, deoxycholic acid (DCA), was shown to boost the induction of PC DCA also accelerates the senescence-associated secretory phenotype and also the progression of intestinal cancer through increasing DNA damage and genome instability (Louis et al., 2014). Moreover, DCA-induced activation of the EGFR ligand, amphiregulin was identified as an oncogenic consider both colorectal and PC by EGFR, mitogen activated protein kinase (MAPK) and STAT3 signaling pathways .

Acetate, propionate and butyrate are typical SCFAs. as an example, butyrate can interfere histone modifications and transcriptional regulation (Cani and Jordan, 2018). Additionally, a decrease in propionate lowers the abundance of mucosal-associated invariant T cells and regulatory T cells guarding the intestinal lamina propria. Growing evidence demonstrates that GM affects the outcomes of PC Compared to the short-term survivors, long-term PDAC survivors possessed higher tumor microbial

diversity and more composition, which is presented as a predict on PDAC survival. H. pylori could also be an essential issue for the initiation and progression of PC. The increasing risk of PC referring to peptic ulceration found in an exceedingly prospective cohort study involving 51,529 male subjects may need resulted from the greater endogenous nitrosation and also the inflammatory response because of H. pylori infection. Additionally, given the overlap of oral microbiomes within the intestine, bacterial translocation from mouth or intestinal tract is additionally closely related to PC. as an example, Fusobacterium spp, a gaggle of anaerobic bacterium colonizing the rima, has been demonstrated a potentially prognostic biomarker of PC with the 8.8% presence. Large prospective studies have reported the next risk of PC in patients with higher concentrations of Porphyromonas gingivalis and a reduced risk for increased levels of specific antibodies against commensal oral bacteria. Currently, compared with normal tissue of pancreas, a 1000-fold increase in intrapancreatic bactibilia was observed in pancreatic ductal adenocarcinoma (PDAC) specimens .This finding is widely divergent from the previous assumption of a sterile pancreas .Data from a constituent analysis of microbes harboring in pancreatic cyst fluid characterized this specific microbiota ecosystem and demonstrated microbiota harboring within the pancreas; the results showed that some taxa with possible deleterious effects within this niche may cause the pancreatic neoplastic process.Notably, it's the bacterial composition, rather the bacterial abundance, within the pancreas that correlates with pancreatic carcinogenesis. Therefore, the mechanism by which microbes flow toward and penetrate the pancreas may be a key issue. The identification of Enterococcus and Enterobacter species primarily found in bile indicate a possible pathway where GM is transported toward the pancreatic tissue Additionally,demonstrated the accessibility of gut bacteria to pancreas, suggesting their direct effect on the microenvironment round the pancreas.

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