Gut and Liver
https://doi.org/10.5009/gnl240380
pISSN 1976-2283 eISSN 2005-1212

Perspective

Gut Microbiota Involved in the Immunopathogenesis of Autoimmune Pancreatitis

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Article Info
Received August 24, 2024
Revised October 8, 2024
Accepted October 14, 2024
Published online March 10, 2025

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Autoimmune pancreatitis (AIP), which is considered the pancreatic expression of a systemic immunoglobulin G4-related disease, is characterized by excessive infiltration of plasmacytes bearing immunoglobulin G4 and a unique form of fibrosis in multiple organs. This relatively new disease entity has garnered great attention from clinicians, but its pathophysiology remains poorly understood. Recent discoveries indicate that plasmacytoid dendritic cell activation followed by robust production of type I interferon and interleukin-33 plays a key role in driving chronic fibro-inflammatory responses in both murine and human AIP. Furthermore, the compositional alterations in the gut microbiota, known as intestinal dysbiosis, triggered plasmacytoid dendritic cell-driven pathogenic type I interferon responses. Intestinal dysbiosis is associated with a breakdown in intestinal barrier function; thus, we examined whether the latter condition affects the development of experimental AIP. Our recent research has revealed that intestinal barrier disruption worsens experimental AIP by facilitating the translocation of pathogenic bacteria, such as *Staphylococcus sciuri*, to the pancreas from the gut. These results indicate the "gut-pancreas axis" underlies the immunopathogenesis of AIP, and the maintenance of intestinal barrier integrity can prevent the worsening of AIP by inhibiting pancreatic colonization by harmful gut bacteria. In this mini review, the interactions between AIP development and gut microbiota are discussed with the aim of providing useful information not only for researchers but also for clinicians. **(Gut Liver 2025;19:171-176)**

Key Words: Autoimmune pancreatitis; Immunoglobulin G4-related disease; Innate immunity; Gastrointestinal microbiota; Dysbiosis

INTRODUCTION

Autoimmune pancreatitis (AIP) is a distinctive form of chronic pancreatitis that was originally discovered in Japan approximately three decades ago.[1] It is now generally accepted that AIP is a pancreatic expression of systemic immunoglobulin G4-related disease (IgG4-RD).[2] AIP and IgG4-RD are marked by significant infiltration of IgG4-positive plasmacyte into the affected organs and chronic fibro-inflammatory conditions across multiple organs.[2,3] The number of patients has increased as disease awareness among clinicians has improved, drawing much attention to AIP and IgG4-RD from various departments of clinicians other than gastroenterologists.[4,5] Despite marked progress in clinicopathological features of AIP and IgG4-RD, the pathophysiology of AIP and IgG4-RD remains unclear.

One clue leading to elucidation of immunopathogenesis of AIP is clarification of immune responses accounting for enhanced IgG4 responses in this disorder. In this regard, Shiokawa *et al.*[6] directly addressed the roles of IgG subtypes in AIP by passively transferring IgG1 and IgG4 from patients with AIP into neonatal mice. Their results indicate that IgG1, rather than IgG4, plays a pathogenic role in AIP because the degree of pancreatitis was much greater in mice received IgG1 antibody (Ab) from patients with AIP than IgG4 Ab from the same patients and that pancreatic injury induced by IgG1 Ab injection was attenuated by IgG4 Ab. These findings provide evidence that excessive production of IgG4 in AIP is an epiphenomenon induced by chronic inflammatory responses and that IgG1, rather

than IgG4, plays a pathogenic role in AIP.[6] In line with this study, recent studies, in which candidate autoantigens were identified, also supported pathogenic roles played by IgG1 Ab, but not IgG4 Ab, in the development of AIP and IgG4-RD.[7,8] Therefore, IgG1 Ab responses driven by type I interferon (IFN) and T helper type 1 (Th1) responses are likely to underlie the immunopathogenesis of AIP and IgG4-RD.[9]

Sensing of microbe-associated molecular patterns (MAMPs) by pattern recognition receptors (PRRs) is a strong inducer of type I IFN responses.[10] In recent years, roles played by innate immune system triggered by PRRs have been increasingly recognized in the immunopathogenesis of autoimmune disorders.[11] Dependence of pathogenicity of IgG1 Ab responses in the development of AIP and IgG4-RD led us to examine involvement of type I IFN responses caused by microbe-mediated PRR activation. Recently, we identified a part of innate immune responses linking type I IFN responses against gut microbiota to the development of AIP and IgG4-RD.[12] We summarize and discuss the recent updates on the interactions between gut microbiota and the development of AIP.

INNATE IMMUNE RESPONSES INVOLVED IN DEVELOPING AIP

Toll-like receptors (TLRs) and nucleotide-binding oligomerization domain-like receptors (NLRs) expressed on macrophages and dendritic cells (DCs) are well-established PRRs for sensing MAMPs derived from microorganisms.[10,13] Upon TLR and NLR activations, type I IFNs as well as proinflammatory cytokines are produced by macrophages and DCs. We previously experienced a case of AIP with numerous IgG4-expressing plasmacyte infiltration in the gastrointestinal mucosa.[14] Stimulation of peripheral blood mononuclear cells isolated from this patient with TLR ligands derived from bacterial cell walls resulted in a marked production of IgG4 Ab as compared with those from healthy controls.[14] Considering that innumerable microorganisms inhabit the gastrointestinal tract, these results indicate that innate immune responses against microorganisms may promote IgG4 production. This experience prompted us to investigate the pathophysiology of AIP through visualization of innate immune responses against bacteria.

We used a well-established mouse model of experimental AIP to explore the innate immune cells involved in the development of IgG4-RD and AIP.[15,16] Repeated intraperitoneal injections of polyinosinic-polycytidylic acid (poly(I:C)), a prototypical TLR3 ligand, to MRL/MpJ mice developed human AIP-like pancreatic lesions, including inflammatory immune cell infiltration, acinar architecture destruction, and fibrosis.[15,16] Additionally, this mouse model exhibited lesions mimicking autoimmune sialadenitis, indicating that this mouse model shares clinical manifestations with human IgG4-RD.[16] Although autoimmunity in MRL/MpJ mice treated with poly(I:C) is well-established, molecular mechanisms accounting for the development of AIP remain poorly understood. Initially, we attempted to determine which innate immune cells have roles in the pathogenesis of AIP. We carried out extensive flow-cytometric analyses of the isolated pancreatic mononuclear cells for identification of types of macrophages and DCs that migrated to the pancreas upon induction of AIP. We found a marked infiltration of plasmacytoid DCs (pDCs), defined as pDC antigen-1^{+}B220^{low} cells by flow-cytometric analysis, in the pancreas of experimental AIP mice.[17] pDCs, known as a specialized DC population, produce large amounts of type I IFNs upon MAMP recognition by endosomal TLRs.[18] Indeed, experimental AIP model mice were characterized by systemic and pancreatic type I IFN responses and that cell-isolation studies identified pDCs as a main producer of type I IFNs.[17] Furthermore, the administration of Abs that deplete pDCs and neutralize type I IFN receptors significantly suppressed the development of AIP.[17] These data employing extensive flow-cytometric analyses indicate that the development of experimental AIP requires pDC activation followed by type I IFN production.[12,17]

A unique form of fibrosis, known as storiform fibrosis, is another pathological feature of human AIP in parallel to prominent infiltration of IgG4-expressing plasmacytes.[2,3,12] Fibrosis akin to human AIP was observed in the pancreas of MRL/MpJ mice treated with poly(I:C). We previously revealed that pancreatic acinar cells that recognize gut bacteria produce interleukin-33 (IL-33) through activation of intracellular nucleotide-binding oligomerization domain 1, thereby inducing chronic fibrotic conditions in the pancreas of the experimental model of chronic pancreatitis.[19] These results obtained from the chronic pancreatitis model prompted us to investigate the role of IL-33 in fibrogenic responses in AIP. High IL-33 expression was noted in the pancreas of the experimental AIP model, and cell-depletion and purification studies using pancreatic mononuclear cells revealed that pDCs produce IL-33 in a type I IFN-dependent manner.[20] The blockade of the IL-33-mediated signaling pathway inhibited both inflammatory and fibrogenic responses in experimental AIP. Confirmation in human samples demonstrated the localization of pDCs that produce both type I IFNs and IL-33 in the pancreas of patients with AIP.[20] Thus, accumulations of pDCs that pro-

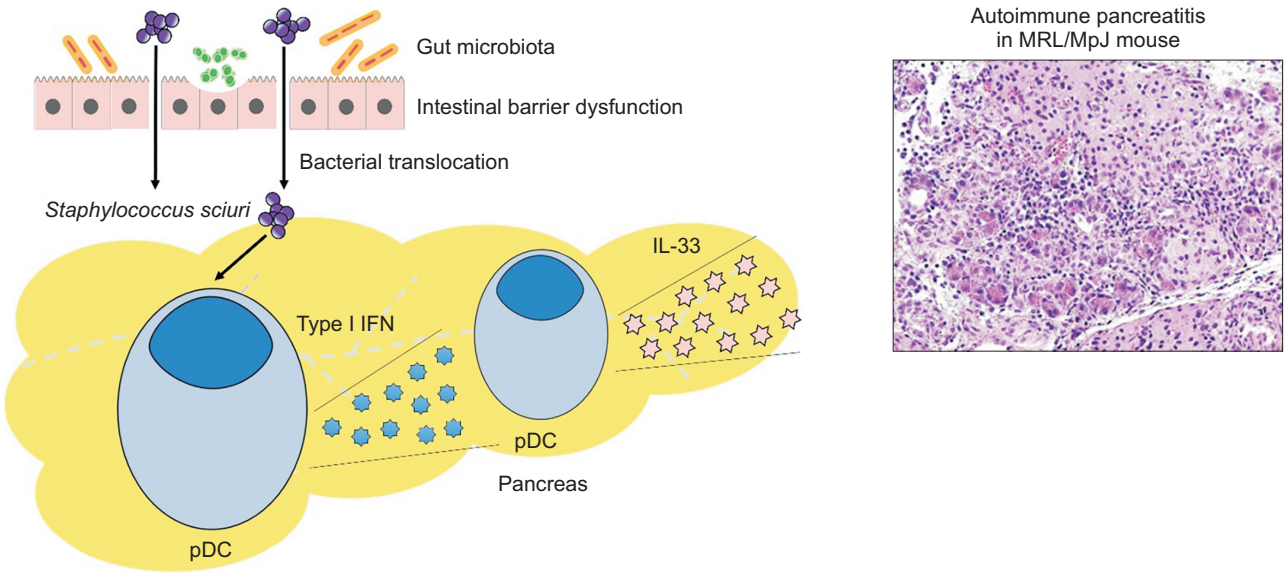

Fig. 1. The gut microbiota and autoimmune pancreatitis "the gut-pancreas axis." Intestinal barrier dysfunction allows pathogenic intestinal bacteria to migrate from the gut to the pancreas. Translocation of pathogenic bacteria, such as *Staphylococcus sciuri* into the pancreas triggers plasmacytoid dendritic cells (pDCs) activation in this organ. Activated pDCs cause chronic fibro-inflammatory changes in the pancreas by producing large amounts of proinflammatory cytokines, including type I interferon (IFN) and interleukin-33 (IL-33). Hematoxylin and eosin stain (×400).

duce both type I IFN and IL-33 underlie the pathogenic innate immune responses in both murine and human AIP (Fig. 1). Our recent identification of serum type I IFN and IL-33 as useful biomarkers for diagnosis and disease activity assessment in human AIP and IgG4-RD fully supported this idea.[21] Additionally, activation of pDCs leading to robust production of type I IFNs fits well with pathogenic roles played by IgG1 Ab rather than IgG4 Ab because the former IgG subclass is under the control of Th1 responses triggered by type I IFNs.

GUT MICROBIOTA AND HUMAN AIP

Recent research has revealed that intestinal dysbiosis plays an important role not only in gastrointestinal disorders but also in pancreatic diseases, including chronic pancreatitis and pancreatic cancer.[22,23] However, our knowledge regarding the interactions between gut microbiota and human AIP and IgG4-RD has been limited. Hamada *et al.*[24] compared the composition of fecal microbiota between patients with AIP and chronic pancreatitis and found significant differences in the gut commensals, despite both diseases causing chronic fibro-inflammatory changes in the pancreas. We investigated the changes in the gut microbiota before and after prednisolone treatment in three patients with AIP.[25] Several bacterial species in the gut microbiota demonstrated alterations before and after prednisolone treatment, the presence of *Klebsiella pneumoniae*, which represented approximately 5% to 10% before prednisolone treatment, were undetectable after prednisolone treatment in the three patients. Gut microbiota composition analyses suggested pathogenicity of *K. pneumoniae* in human AIP. However, oral administration of heat-killed *K. pneumoniae* alone did not cause AIP in MRL/MpJ mice, suggesting that innate immunity against this bacterium itself is not primarily responsible for the development of experimental AIP and thus colonization of this bacterium functions increases the sensitivity to experimental AIP. To explore this possibility, we took advantage of mild experimental AIP induced by low-dose poly(I:C) injection. Combined treatment with administration of heat-killed *K. pneumoniae* and low-dose poly(I:C) resulted in the development of severe AIP.[25] The degree of AIP was more severe in mice treated with both heat-killed *K. pneumoniae* and low-dose poly(I:C) than in those treated with low-dose poly(I:C) alone. In addition, severe AIP caused by combined treatment was accompanied by a significant increase in pDCs producing type I IFNs and IL-33 in the pancreas.[25] Thus, activation of pDCs in response to recognition of *K. pneumoniae* enhances sensitivity to experimental AIP and therefore colonization of this bacterium in human gastrointestinal tract may function as an accelerator rather than a causative factor of chronic fibro-inflammatory responses. Further studies are warranted to evaluate whether this bacterium is a pathobiont, considering the small sample size and the unknown mechanism of disease induction caused by *K. pneumoniae*. However, noteworthily, gut microbiota

is likely to be involved in the pathophysiology of AIP not only in mice but also in humans.

INTESTINAL DYSBIOSIS TRIGGERS EXPERIMENTAL AIP DEVELOPMENT

As noted above, pDCs are pathogenic immune cells involved in the pathogenesis of IgG4-RD and AIP, but the upstream pathways prior to the pDCs-driven pathogenic innate immune responses are unclear. It is known that pDCs are a unique subset of DCs that can produce abundant quantities of type I IFNs upon recognizing MAMPs through TLRs.[12,26] We hypothesized that alterations in the gut microbiota, i.e., intestinal dysbiosis, serve as a trigger for pathogenic pDC activation. A broad range of antibiotics were administered to MRL/MpJ mice treated with repeated injections of poly(I:C) to deplete gut bacteria and to define involvement of innate immune responses against commensal bacteria. Bowel sterilization by antibiotics significantly suppressed AIP development, accompanied by a marked decrease in pancreatic accumulation of pDCs producing type I IFNs and IL-33.[27] In line with our data, Ito *et al.*[28] provided evidence that antimicrobial administration suppressed the onset of pancreatic injury in another experimental AIP model caused by intraperitoneal administration of heat-killed *Escherichia coli.* These results indicate that intestinal dysbiosis is associated with experimental AIP development.

Fecal microbiota transplantation experiments were conducted to dissect molecular mechanisms underlying the association between intestinal dysbiosis and AIP. The severity of AIP in MRL/MpJ mice depends on the injected doses of poly(I:C) in the mouse model of experimental AIP as mentioned above; repeated injections of 100 μg and 10 μg poly(I:C) led to severe and mild AIP as judged by pathological examinations. Gut microbiota transmission from mice treated with 100 μg poly(I:C) to mice treated with 10 μg poly(I:C) transformed mild degree of AIP into severe degree of AIP. In contrast, gut microbiota transmission from healthy mice without poly(I:C) treatment to mice treated with 10 μg poly(I:C) did not alter the severity of AIP.[27] It is worth mentioning that gut microbiota transmission from mice treated with 100 μg poly(I:C) to mice did not cause neither mild nor severe AIP as long as the recipient mice were not treated with poly(I:C).[27] Thus, original mild AIP transformed into severe AIP when gut microbiota from severe AIP mice colonized in the gastrointestinal tract of mild AIP mice. In addition, such transformation of mild AIP into severe AIP was associated with pancreatic accumulation of pDCs producing type I IFNs and IL-33. Altogether, these studies indicate that intestinal dysbiosis is one of the triggers for the initial activation of pathogenic pDCs-driven innate immune pathways. As in the case of *K. pneumoniae*, it would be emphasized that intestinal dysbiosis may function as an accelerator rather than a causative factor of experimental AIP.

PATHOGENIC ROLE OF *Staphylococcus sciuri* IN EXPERIMENTAL AIP

Growing evidence indicates that gut microbiota is crucial for preserving the integrity of the intestinal epithelial barrier and that an imbalance in these microbiota is strongly associated with dysfunction of this barrier.[29,30] Considering AIP and IgG4-RD are frequently detected in older men bearing impaired intestinal barrier integrity, it can be presumed that intestinal barrier dysfunction is involved in the immunopathogenesis of AIP and IgG4-RD. Dextran sodium sulfate (DSS) drinking was used to cause intentional intestinal barrier dysfunction to clarify the association between gut barrier integrity, dysbiosis, and AIP development.[31] Mice cotreated with poly(I:C) and DSS developed more severe AIP than those treated with poly(I:C) alone. Analyses of activation of pDCs revealed increased accumulation of pDCs producing type I IFNs and IL-33 in the pancreas and colon of mice treated with both DSS and poly(I:C) as compared with those treated with DSS or poly(I:C) alone, indicating the presence of crosstalk between the pancreas and colon.[31] Interestingly, intestinal barrier disruption did not exacerbate autoimmune sialadenitis and did not increase pathogenic pDCs or proinflammatory cytokines in the salivary glands.

We hypothesized that the gut-pancreas crosstalk leading to excessive activation of pDCs may exist in AIP and IgG4-RD, considering that the pancreas is more susceptible to the effect of the gut microbiota compared with the salivary glands. Subsequent next-generation sequencing analysis targeting 16S ribosomal RNA revealed the increase of *S. sciuri* in the feces and pancreas of mice displaying severe pancreatitis treated with poly(I:C) and DSS.[31] *S. sciuri* is a Gram-positive, coagulase-negative cocci that inhabits the mucosa and skin of mammals.[32] *S. sciuri* is a popular causative bacterium of wound infection and mastitis in mammals and is sometimes associated with peritonitis or endocarditis in humans.[31,32] Our 16S ribosomal RNA sequence study supported the possibility that migration of gut *S. sciuri* into the pancreas occurred upon disruption of intestinal barrier integrity and then pancreatic colonization of this bacterium activated pDCs.

A mouse model mono-colonized with *S. sciuri* was

developed to investigate the pathogenicity of *S. sciuri* in experimental AIP.[31] Germ-free mice developed mild pancreatitis with repeated injections of low-dose (10 μg) poly(I:C), and mice mono-colonized with *S. sciuri* demonstrated severe pancreatitis treated with the same dose of poly(I:C). Development of severe AIP in *S. sciuri* mono-colonized mice treated with poly(I:C) was accompanied by a marked accumulation of pDCs that produce type I IFN and IL-33 in the pancreas,[31] strongly indicating that *S. sciuri* plays a pathogenic role in exacerbating experimental AIP. In fact, pDCs isolated from the pancreas of mice displaying severe AIP produced large amounts of type I IFN and IL-33 upon *in vitro* exposure to *S. sciuri*. These results indicate the presence of gut-pancreas crosstalk in which mutual communication between intestinal barrier disruption and intestinal dysbiosis allows migration of pathogenic bacterial species, in this instance *S. sciuri*, from the gut to the pancreas and then activates pDCs underlying the immunopathogenesis of AIP (Fig. 1).

CONCLUSION

Intestinal dysbiosis is a significant factor contributing to the immunopathogenesis of AIP and IgG4-RD, emphasizing the potential of maintaining a healthy gut microbiota for disease prevention and treatment. The pathophysiology targeting the "gut-pancreas axis" may help in developing novel treatments for AIP and IgG4-RD.

CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

ACKNOWLEDGEMENTS

This work was supported in part by Grants-in-Aid for Scientific Research (23K07450) from the Japan Society for the Promotion of Science and Kindai University Research Enhancement Grants (IP001).

AUTHOR CONTRIBUTIONS

Study concept and design: K.M., T.W. Drafting of the manuscript: K.M, T.W. Critical revision of the manuscript for important intellectual content: A.H., T.Y., K.K. Obtained funding: K.M. Administrative, technical, or material support; study supervision: M.K. Approval of final manuscript: all authors.

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