International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
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ARTICLE TITLE	FROM INFECTION TO DYSBIOSIS: PATHOGENESIS OF POST-INFECTIOUS IRRITABLE BOWEL SYNDROME - DIAGNOSTIC AND THERAPEUTIC IMPLICATIONS
DOI	https://doi.org/10.31435/ijitss.2(50).2026.6115
RECEIVED	15 March 2026
ACCEPTED	22 June 2026
PUBLISHED	30 June 2026
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FROM INFECTION TO DYSBIOSIS: PATHOGENESIS OF POST-INFECTIOUS IRRITABLE BOWEL SYNDROME - DIAGNOSTIC AND THERAPEUTIC IMPLICATIONS

Aleksander Gajkowski *(Corresponding Author, Email: aleksandergajkowski@gmail.com)*
Samodzielny Publiczny Zespół Zakładów Opieki Zdrowotnej w Wyszkowie, Wyszków, Poland
ORCID ID: 0000-0001-9130-5931

Bartosz Zawadzki
Independent Public Health Care Institution of the Ministry of the Interior and Administration, Krakow, Lesser Poland, Poland
ORCID ID: 0009-0004-9328-175X

Zuzanna Walczak
5th Military Clinical Hospital with Polyclinic SPZOZ, Kraków, Małopolska, Poland
ORCID ID: 0009-0008-8417-5372

Katarzyna Naja
National Medical Institute of the Ministry of the Interior and Administration, Warsaw, Poland
ORCID ID: 0009-0008-6513-6984

Tomasz Adam Szczepanowski
Samodzielny Publiczny Centralny Szpital Kliniczny, Warsaw, Mazovia, Poland
ORCID ID: 0009-0006-2584-5554

Krzysztof Bednarski
Beskid Oncology Center Municipal Hospital of John Paul II, Bielsko-Biała, Poland
ORCID ID: 0009-0000-9801-2832

Jakub Zwardoń
Dr Anna Gostynska Wolski Hospital, Marcina Kasprzaka 17, 01-211 Warsaw, Poland
ORCID ID: 0009-0005-3944-2943

Natalia Kornacka
Beskid Oncology Center Municipal Hospital of John Paul II, Wyzwolenia 18, 43-300 Bielsko-Biała, Poland
ORCID ID: 0009-0003-3657-2027

Anna Świerczek
Beskid Oncology Center Municipal Hospital of John Paul II, Wyzwolenia 18, 43-300 Bielsko-Biała, Poland
ORCID ID: 0009-0006-9612-237X

Miłosz Rewekant
Beskid Oncology Center Municipal Hospital of John Paul II, Wyzwolenia 18, 43-300 Bielsko-Biała, Poland
ORCID ID: 0009-0004-3944-9363

ABSTRACT

Introduction: Post-infectious irritable bowel syndrome (PI-IBS) develops in approximately 14.5% of individuals after acute gastroenteritis and persists long-term, with emerging evidence suggesting an autoimmune component linking infection to chronic gastrointestinal dysfunction. This review aims to synthesize current literature on the proposed pathogenesis of PI-IBS, focusing on molecular mechanisms, the development of small intestinal bacterial overgrowth (SIBO), and resulting diagnostic and therapeutic implications.
Materials and methods: A narrative review of PubMed literature was performed, focusing on autoantibody-mediated disruption of gut motility, candidate diagnostic biomarkers, and emerging therapeutic approaches. Studies were selected based on relevance and methodological quality, without formal inclusion or exclusion criteria.
Results: Infection by cytolethal distending toxin (CDT)-producing bacteria triggers anti-CdtB antibody production, which through molecular mimicry may cross-react with human vinculin in the interstitial cells of Cajal, causing enteric neuropathy and impaired migrating motor complex function. The resulting dysmotility may facilitate SIBO development. Combined anti-CdtB and anti-vinculin serum biomarkers can offer post-test probability exceeding 98% for diarrhea-predominant IBS. Antibody-depleting therapies, such as plasmapheresis and intravenous immunoglobulin, may reduce autoantibody burden and correlate with symptomatic improvement.
Conclusions: PI-IBS may represent an immune-mediated consequence of acute bacterial gastroenteritis, in which CdtB-induced autoimmunity drives dysmotility and microbial dysbiosis. This mechanistic framework offers potential biomarkers and therapeutic targets. Further prospective studies are needed to validate these findings.

KEYWORDS

Biomarkers; Enteric Nervous System; Gastrointestinal Microbiome; Irritable Bowel Syndrome

CITATION

Aleksander Gajkowski, Bartosz Zawadzki, Zuzanna Walczak, Katarzyna Naja, Tomasz Adam Szczepanowski, Krzysztof Bednarski, Jakub Zwardoń, Natalia Kornacka, Anna Świerczek, Miłosz Rewekant. (2026). From Infection to Dysbiosis: Pathogenesis of Post-Infectious Irritable Bowel Syndrome - Diagnostic and Therapeutic Implications. *International Journal of Innovative Technologies in Social Science*. 2(50). doi: 10.31435/ijitss.2(50).2026.6115

1. Introduction

Irritable bowel syndrome (IBS) is one of the most prevalent gastrointestinal disorders worldwide, affecting approximately 14.1% of the global population (Arif et al., 2025). The condition profoundly disrupts daily functioning, with patients frequently reporting debilitating abdominal pain, unpredictable bowel habits, and heightened anxiety that impair social participation, travel, and occupational performance (Ballou & Keefer, 2017; Goodoory et al., 2023). On a systemic level, IBS places a substantial burden on healthcare systems, accounting for a disproportionate number of gastroenterological consultations. Many clinicians treat IBS as a diagnosis of exclusion, so patients typically undergo an extensive clinical workup to rule out other conditions, which significantly drives up healthcare costs (Spiegel et al., 2010). According to historical estimates, the annual economic burden of IBS in the United States ranged from $1.7 billion to $10 billion in direct medical expenses, with up to $20 billion in indirect costs related to lost productivity (Hulisz, 2004). Despite this profound individual and economic impact, IBS lacks widely accepted positive biological markers and mechanistically grounded treatments, leaving clinicians and patients without satisfactory answers (Lacy et al., 2021).

Post-infectious IBS (PI-IBS) is a distinct subtype of IBS triggered by acute enteric infection, developing in approximately 14.5% of individuals following acute gastroenteritis (Porcari et al., 2024) and persisting for months to years thereafter (Thabane & Marshall, 2009). The clear timeline between the initial infection and the onset of chronic symptoms suggests that the acute illness triggers a long-term pathological process—though the exact mechanisms have remained incompletely understood.

Over the past two decades, a compelling body of evidence has emerged implicating autoimmune mechanisms—specifically pathogenic autoantibodies—as central mediators of PI-IBS pathophysiology.

Studies identified circulating autoantibodies against vinculin, a cytoskeletal protein expressed in interstitial cells of Cajal, and against cytolethal distending toxin B (CdtB), a virulence factor produced by enteropathogenic bacteria, in a significant proportion of PI-IBS patients (Pimentel et al., 2015a, 2015b). These findings suggest a mechanism of molecular mimicry, whereby the host immune response to bacterial antigens generates cross-reactive antibodies targeting native intestinal proteins, thereby inducing enteric neuropathy and disordered motility.

The recognition of a potential autoimmune component carries significant clinical implications, offering measurable diagnostic biomarkers and potential therapeutic targets—both of which represent major unmet needs in IBS management. This literature review provides a mechanistic explanation for the development of PI-IBS and its overlap with small intestinal bacterial overgrowth (SIBO). We examine the immunological mechanisms underlying autoantibody generation, characterize the antigenic targets and their functional significance, evaluate the diagnostic utility of available biomarkers, and discuss potential therapeutic strategies.

2. Materials and Methods

A narrative literature review was conducted using the PubMed database to identify studies published between 2000 and 2026. Search terms were used in combination and included: "post-infectious irritable bowel syndrome", "PI-IBS", "cytolethal distending toxin", "CdtB", "anti-CdtB", "vinculin", "anti-vinculin antibodies", "molecular mimicry", "migrating motor complex", "interstitial cells of Cajal", "small intestinal bacterial overgrowth", and "enteric neuropathy". Additional references were identified through manual screening of the bibliographies of retrieved articles.

No language restrictions were applied. No formal inclusion or exclusion criteria were applied. Both human and animal studies were included.

3. Characteristics of PI-IBS

3.1 Prevalence and Chronicity

Published studies have consistently reported the incidence of PI-IBS in a range between 5% and 32%, reflecting variability attributable to differences in study methodology, infecting organism, and follow-up duration (Thabane & Marshall, 2009). The most recent systematic review and meta-analysis of 47 studies and 28,170 subjects established a PI-IBS prevalence of 14.5% — meaning that approximately 1 in 7 individuals may develop Irritable Bowel Syndrome following acute gastroenteritis. Notably, symptoms persisted in 39.8% of affected subjects at long-term follow-up exceeding five years, underscoring the chronicity of the condition (Porcari et al., 2024). From the perspective of international travel medicine, a multicenter prospective study found an overall incidence of PI-IBS of 12.1% following travelers' diarrhea, with the majority (78.7%) retaining PI-IBS symptoms at twelve months post-travel (Chan et al., 2023). These findings collectively underscore the global significance of PI-IBS as a frequent and long-lasting consequence of acute gastrointestinal infection.

3.2 Causative Pathogens

The risk of developing post-infectious irritable bowel syndrome (PI-IBS) varies substantially according to the causative pathogen. The strongest association was observed for parasitic infections, which yielded the highest pooled prevalence (30.1%); however, this estimate is based on only two studies, and the evidence base is therefore limited. Bacterial pathogens, which constitute the majority of the examined literature, showed a pooled prevalence of 18.3%, while viruses demonstrated the lowest prevalence at 10.7%. Among these bacterial species, *Campylobacter* was linked to the greatest PI-IBS prevalence at 20.7% (Porcari et al., 2024).

Campylobacter jejuni alongside *Escherichia coli*, *Salmonella enterica*, and *Shigella dysenteriae*, represents the core group of bacterial pathogens driving both PI-IBS incidence and the global burden of food- and water-borne infectious diarrheal illnesses (Kirk et al., 2015; Newell et al., 2010; Porcari et al., 2024). Most importantly, despite their phylogenetic differences, these four distinct pathogens are unified by a shared virulence factor: cytolethal distending toxin (CDT), a heterotrimeric AB-type genotoxin whose pathological activity is mediated primarily through its catalytic subunit, CdtB (Jinadasa et al., 2011).

3.3 Clinical Subtypes of IBS

According to the Rome IV criteria (Drossman & Hasler, 2016), subtyping for Irritable Bowel Syndrome is primarily determined by the predominant bowel habit as assessed by the Bristol Stool Form Scale (BSFS) (Lacy et al., 2016). This framework categorizes patients into four distinct groups: IBS with constipation (IBS-C), IBS with diarrhea (IBS-D), IBS with mixed bowel habits (IBS-M), and unsubtyped IBS (IBS-U). In the specific context of post-infectious IBS (PI-IBS), recent data identify IBS-D as the most frequent phenotype at 46.1%, closely followed by IBS-M at 38.5%, whereas IBS-C and IBS-U are notably less prevalent at 10.0% and 5.4%, respectively (Porcari et al., 2024).

Table 1. Distribution of Clinical Subtypes in Post-Infectious Irritable Bowel Syndrome (PI-IBS)

IBS Subtype	Clinical Phenotype	Prevalence (%)
IBS-D	Diarrhea-predominant	46.1%
IBS-M	Mixed bowel habits	38.5%
IBS-C	Constipation-predominant	10.0%
IBS-U	Unsubtyped	5.4%

Note: Data derived from a systematic review and meta-analysis of 28,170 subjects (Porcari et al., 2024).

4. Pathogenesis and Supporting Evidence

4.1 Pathogenesis

The pathogenesis of PI-IBS is most rigorously explained by the mechanism of molecular mimicry (Pimentel et al., 2015a), which proposes that structural homology between a pathogen-derived antigen and a host protein leads to the generation of cross-reactive autoantibodies targeting self-tissue (Rojas et al., 2018). The most common causative pathogens in PI-IBS—*Campylobacter jejuni*, *Escherichia coli*, *Salmonella enterica*, and *Shigella dysenteriae*—share a common virulence factor: cytolethal distending toxin (CDT). Its catalytically active subunit, CdtB, triggers a host immune response resulting in the production of anti-CdtB antibodies. Due to structural homology between CdtB and the host cell adhesion protein vinculin, these antibodies are proposed to cross-react with vinculin through molecular mimicry, generating anti-vinculin autoantibodies (Pimentel et al., 2015a).

Vinculin is expressed in the interstitial cells of Cajal (ICCs) and in the myenteric plexus (Pimentel et al., 2015a). ICCs are the primary pacemakers of intestinal motility (Sanders et al., 2014). These structures contribute to generation of the migrating motor complex (MMC), a rhythmic interdigestive activity, which clears the intestinal lumen of undigested debris and excess bacteria during fasting, thereby preventing retrograde bacterial migration from the colon (Deloose et al., 2012). The autoimmune disruption of these structures results in the impairment of MMC function. Consequently, as this MMC-driven clearance fails, microorganisms proliferate within the small intestine or migrate proximally from the colon, establishing the dysbiotic state characteristic of SIBO (Pimentel et al., 2015a).

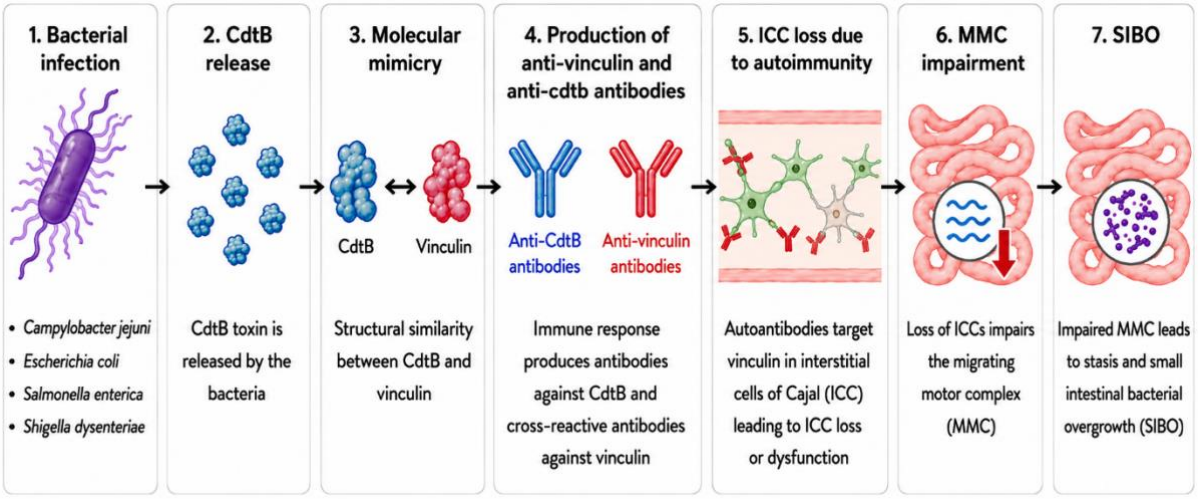

Fig. 1. *Proposed pathophysiological mechanism*

Abbreviations: CdtB — cytolethal distending toxin B, ICC — interstitial cells of Cajal, MMC — migrating motor complex, SIBO — small intestinal bacterial overgrowth

4.2 The Causal Role of CdtB Toxin

Experimental support for the causal role of CdtB toxin in this autoimmune process comes from two complementary rat model studies, which are particularly significant because they isolate the toxin alone—without live bacterial infection—as sufficient to produce disease. Morales et al. (2020) demonstrated that subcutaneous injection of purified CdtB protein in rats, followed by a booster at three weeks, produced markedly elevated anti-CdtB antibodies that in turn precipitated a pronounced rise in anti-vinculin autoantibodies ($p < 0.001$), alongside a measurable reduction in intestinal vinculin expression and quantifiable increases in small bowel bacterial load. This concept was further expanded by Leite et al. (2024), who demonstrated that CdtB inoculation also triggers elevated pro-inflammatory cytokines, marked microbiome dysbiosis, and intestinal barrier disruption. Together, these animal models demonstrate that CdtB exposure alone recapitulates the full immunological, microbiome, motility, and barrier phenotype of human PI-IBS, suggesting that the autoantibody response is the causal mechanism driving chronic gut dysfunction (Leite et al., 2024; Morales et al., 2020).

4.3 Motility issues in IBS patients with SIBO

Direct evidence of this mechanical failure is provided by antroduodenal manometry studies, which serve as the gold standard for assessing interdigestive motility. Specifically, IBS patients with concomitant SIBO display, on average, only one-third as many phase III MMC events during fasting periods compared to healthy controls (Pimentel et al., 2002). Phase III is widely recognized as the most vital component of the MMC cycle, characterized by high-amplitude, propagation-efficient bursts of synchronized contractions that physically sweep the upper gastrointestinal tract (Deloose et al., 2012). In addition to a marked reduction in the absolute frequency of these events, the phase III cycles that do occur in IBS-SIBO patients are significantly shorter in duration and exhibit impaired migratory propagation along the small bowel lumen (Pimentel et al., 2002).

4.4 Anti-vinculin and anti-CdtB antibody positivity in PI-IBS

Anti-CdtB and anti-vinculin antibodies are indeed elevated in a subset of IBS patients, particularly those with diarrhea-predominant disease, compared to healthy controls and patients with inflammatory bowel disease, as demonstrated in a large multicenter validation study (Pimentel et al., 2015b) and subsequently confirmed using an improved assay in a smaller cohort (Morales et al., 2019). Consistent with these findings, a small cohort study in Mexico reported elevated circulating anti-CdtB and anti-vinculin antibodies in 55% of patients with IBS-D (Schmulson et al., 2016).

4.5 Findings in patients with systemic sclerosis

The significance of anti-vinculin antibodies is further enhanced by findings in studies on systemic sclerosis (SSc). Anti-vinculin antibody positivity was significantly associated with slower gastric transit on whole-gut scintigraphy in SSc patients (Herrán et al., 2023), while an earlier study across two independent SSc cohorts demonstrated that anti-vinculin levels correlated with the degree of gastrointestinal system involvement (Suliman et al., 2021).

4.6 Coexistence of SIBO in IBS patient population

Given the direct link between motility failure and bacterial proliferation, the clinical coexistence of IBS and SIBO is substantial. A meta-analysis by Shah et al. (2020) found that up to 62.3% of IBS patients may have concomitant SIBO as diagnosed by lactulose breath test. However, estimates of SIBO prevalence vary substantially depending on the diagnostic method employed: lactulose breath tests yielded higher SIBO positivity rates (62.3%) than xylose (58.2%) or glucose (20.7%) breath tests, highlighting the influence of test selection on prevalence estimates. The authors concluded that SIBO may cause IBS symptoms in a proportion of patients (Shah et al., 2020). While a comprehensive discussion of SIBO diagnostics and treatment falls outside the scope of this review, the high prevalence of SIBO in this population underscores the importance of breath testing and targeted antimicrobial therapy in the management of IBS patients (Lacy et al., 2021; Pimentel et al., 2020).

5. Novel Biomarkers as Diagnostic Tests

The discovery of two circulating blood antibodies, anti-cytolethal distending toxin B (anti-CdtB) and anti-vinculin, has opened the possibility of a simple blood test that can support a positive diagnosis of IBS, particularly its diarrhea-predominant form (IBS-D) (Pimentel et al., 2015b).

In a large multicenter study, Pimentel et al. (2015b) measured plasma anti-CdtB and anti-vinculin antibody levels by ELISA across 2,375 participants, comparing IBS-D against healthy controls, IBD, and celiac disease. Anti-CdtB titers were significantly higher in IBS-D than in all other groups ($p < 0.001$), and anti-vinculin titers were likewise significantly elevated ($p < 0.001$). At predefined cutoffs, anti-CdtB achieved a specificity of 91.6% and a positive likelihood ratio of 5.2, while anti-vinculin reached a specificity of 83.8% and a positive likelihood ratio of 2.0. However, sensitivities were only moderate — 43.7% for anti-CdtB and 32.6% for anti-vinculin — meaning that negative results cannot reliably exclude IBS-D.

In a subsequent smaller study of 100 IBS-D and 31 IBD subjects, Morales et al. (2019) identified that earlier assays suffered from antigen instability. After incorporating epitope stabilization, specificity improved to 93.5% for anti-CdtB and 90.9% for anti-vinculin, with sensitivities of 43.0% and 52.2% and positive likelihood ratios of 6.7 and 5.7, respectively.

Table 2: Diagnostic Accuracy of Second-Generation IBS Biomarkers

Biomarker	Sensitivity	Specificity	LR (+)
Anti-CdtB	43.0%	93.5%	6.7
Anti-Vinculin	52.2%	90.9%	5.7

Note: LR+, positive likelihood ratio. Data adapted from Morales et al. (2019).

Most importantly, from a clinical standpoint, when both antibodies are positive in a patient presenting with abdominal pain and altered bowel habits, the probability of an IBS-D diagnosis exceeds 98% (Morales et al., 2019).

The diagnostic value of these markers differs by IBS subtype. Antibody levels are highest in IBS-D and IBS-M (mixed type), while levels in IBS-C (constipation-predominant) patients are not statistically different from healthy controls (Rezaie et al., 2017).

In summary, second-generation ELISA testing for anti-CdtB and anti-vinculin represents a validated, minimally invasive tool with high specificity for IBS-D. When both markers are positive, the diagnosis can be confirmed with high confidence, potentially reducing the need for invasive investigations.

6. Therapeutic Implications

While standard treatment protocols for IBS remain focused on symptom relief, the recognition of PI-IBS as an autoantibody-mediated condition opens an entirely distinct therapeutic domain— targeting the underlying immune process itself. The most direct clinical evidence for this approach comes from a retrospective chart review by Sharabi et al. (2025) conducted at Cedars-Sinai Medical Center, in which 417 IBS patients previously tested for anti-CdtB and anti-vinculin antibodies were followed over time. Of these, 158 (38.5%) were positive for either antibody. In the subset with serial measurements (n = 38), normalization of anti-vinculin levels correlated with improvements in IBS symptoms ($p = 0.020$), and antibody-depleting therapies—plasmapheresis (PLEX) or intravenous immunoglobulin (IVIG)—were associated with greater antibody normalization than usual management (62.5% vs. 22.2%; $p = 0.046$). The authors acknowledged the limitations of a retrospective design—small treatment numbers, variable intervals between tests, and absence of randomization (Sharabi et al., 2025).

7. Conclusions

This review has traced a pathogenic sequence—from acute enteric infection through CdtB-mediated autoimmunity to chronic gut dysmotility and microbial overgrowth (Pimentel et al., 2015a). The proposed pathogenic mechanism is supported by converging evidence from animal models (Leite et al., 2024; Morales et al., 2020), human biomarker studies (Pimentel et al., 2015b; Rezaie et al., 2017), functional motility studies demonstrating impaired migrating motor complex activity in IBS patients (Pimentel et al., 2002), and epidemiological data highlighting frequent coexistence with small intestinal bacterial overgrowth (Shah et al., 2020), as well as cross-condition observations in systemic sclerosis (Herrán et al., 2023; Suliman et al., 2021). This framework has further generated useful clinical tools in the form of anti-CdtB and anti-vinculin serology (Morales et al., 2019). The recognition of PI-IBS as an autoantibody-mediated condition shifts the focus toward targeting the underlying immune process, with preliminary data suggesting that antibody-depleting strategies represent a promising domain for emerging new therapies (Sharabi et al., 2025).

Several research gaps limit the conclusions that can currently be drawn. Despite compelling data from animal models, the causal chain linking CdtB exposure, anti-vinculin autoimmunity, and subsequent dysmotility remains to be prospectively validated in human cohorts. To date, no longitudinal study has tracked the progression from an initial episode of acute gastroenteritis through the emergence of autoantibodies and the eventual onset of chronic PI-IBS symptoms. Specific intervention trials are scarce, with most therapeutic evidence derived from broadly defined IBS populations (Lacy et al., 2021). The preliminary findings on antibody-depleting therapies are intriguing but rest on a single retrospective study, and prospective randomized trials of immunomodulatory approaches in seropositive PI-IBS patients do not yet exist (Sharabi et al., 2025).

Despite these limitations, the overall trajectory of this field is encouraging. Rather than being viewed merely as a functional disorder, PI-IBS is increasingly recognized as a condition with a plausible biological substrate, measurable biomarkers, and possibly emerging mechanism-directed treatments.

Conflict of Interest Statement: The authors declare no conflicts of interest.

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