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REVIEW ARTICLE

Epidemiology and contemporary risk factors of peptic acid disease

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Abstract

Acid-peptic diseases, which include gastroesophageal reflux disease (GERD), gastritis, peptic ulcer disease (PUD), and functional dyspepsia (FD), comprises a group of highly prevalent disorders around the world. The epidemiological burden is particularly significant due to the recurrence of symptoms and their impact on quality of life. This is a narrative review that describes the epidemiological aspects and contemporary risk factors associated with these four groups of diseases. The literature shows that there is data on the prevalence that varies according to each of these entities, and type of population and country that has been studied. In addition, they are more prevalent in women, except for GERD, and there are some risk factors that are common to all of them, such as smoking, alcohol consumption, use of non-steroidal anti-inflammatory drugs, and infection by *Helicobacter pylori*. In contrast, others such as obesity are more related to GERD, and previous gastrointestinal infections with FD. Also, it is important to highlight that in recent years there has been an important decrease in PUD, probably in relation to a lower infection rate of *H. pylori*, and maybe with a higher use of proton pump inhibitors. Accordingly, it is important to ask patients about these factors so they can be modified as part of the symptom treatment. Finally, it is important to note the lack of studies on incidence and of the natural history of GERD, gastritis and FD, which are warranted.

Keywords: Epidemiology. Gastroesophageal reflux disease. Gastritis. Acid peptic disease. Functional dyspepsia.

Epidemiología y factores de riesgo contemporáneos de la enfermedad ácido-péptica

Resumen

La enfermedad acido-peptica, que incluye la enfermedad por reflujo gastroesofagico (ERGE), la gastritis, la ulcera peptic (UP) y la dispepsia funcional (DF), constituye un grupo de trastornos de alta prevalencia en todo el mundo. Su carga epidemiologica es tambien de gran relevancia por la recurrencia de los sintomas y por el impacto en la calidad de vida. La presente es una revision narrativa en la que se describen los aspectos epidemiologicos y los factores de riesgo contemporaneous para estos cuatro tipos de trastornos. La literatura muestra datos sobre la prevalencia que varian de acuerdo con la enfermedad y segun la poblacion y los paises estudiados. Ademas, son mas prevalentes en las mujeres que en los hombres, excepto la ERGE, y hay algunos factores de riesgo que son comunes, tales como el tabaquismo, el consumo de alcohol, el uso de antiinflamatorios no esteroideos y la infeccion por *Helicobacter pylori;* en cambio, otros factores, como la obesidad, estan mas asociados con

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la ERGE, y las infecciones gastrointestinales previas con la DF. Asi mismo, es importante resaltar que en los ultimos anos ha habido una disminucion importante de la UP, en probable relacion con el descenso de las infecciones por *H. pylori* y quiza por el mayor uso de los inhibidores de la bomba de protones. Por lo anterior, es importante interrogar a los pacientes sobre estos factores para asi poder modificarlos como parte del manejo de los sintomas. Por ultimo, cabe senalar que faltan estudios de incidencia y de evolucion natural, especialmente para la ERGE, la gastritis y la DF, los cuales son necesarios.

Palabras clave: Epidemiología. Enfermedad por reflujo gastroesofágico. Gastritis. Enfermedad ácido-péptica. Dispepsia funcional.

Introduction

Acid-peptic disease (APD) refers to a group of conditions that originate from an imbalance between aggressive factors (such as gastric acid, pepsin, *Helicobacter pylori* infection, and harmful drugs) and the defense mechanisms of the gastroduodenal mucosa,[1,2] which leads to structural damage of the mucosa and submucosa of the esophagus, stomach, and duodenum, possibly with esophagogastroduodenal symptoms.

From this perspective, the present is a narrative review that analyzes the epidemiological factors of the three diseases classically recognized within the spectrum of APD: gastroesophageal reflux disease (GERD), gastritis, and peptic ulcer disease (PUD), whose pathogenesis and treatment depend on acid-peptic aggression and its modulation through antisecretory drugs. Additionally, we include functional dyspepsia (FD) since its symptoms originate in the gastroduodenal area and share clinical manifestations, diagnostic approach, and therapeutic strategies with these diseases.

Epidemiology

Gastroesophageal reflux disease

GERD is one of the most frequent digestive disorders, whose typical symptoms are heartburn and regurgitation, which are also common symptoms in the general population. In fact, in a national survey on gastrointestinal symptoms conducted in the United States of America, approximately two out of three people reported at least one gastrointestinal symptom during the week prior to the interview, and reflux/heartburn was the most frequent, present in 30.9% of respondents.[3] On the other hand, the prevalence of gastroesophageal reflux symptoms in the general population, based on pooled data from 108 study populations in a meta-analysis, was 14.8%.[4] Consistently, in Mexico, according to the SIGAME national survey, the frequency of heartburn or regurgitation occurring at least once a week was 12.1%, while almost half of the subjects (49.1%) reported these symptoms at least once a month and 1.2% reported them daily.[5] Together, these data show that heartburn and regurgitation are extremely frequent manifestations in the general population, thus representing a very common reason for consultation in clinical practice.

Regarding GERD, worldwide its prevalence shows significant heterogeneity according to regions, with generally higher figures in North America and Europe (19.55% and 14.12%, respectively) and intermediate in Latin America (12.88%) and some areas of Asia (12.92%).[6] Various studies on the global burden of disease have documented a progressive increase in the frequency of GERD during recent decades, parallel to the increase in obesity, population aging, and the adoption of dietary patterns.[7] These changes reinforce the consideration of GERD as an emerging public health issue in numerous countries.

In Mexico, the available population-based studies also show a high frequency of typical GERD symptoms; however, the prevalence of this disease properly demonstrated has not been documented. Nevertheless, in surveys conducted among general population in Mexico, the prevalence of GERD, defined by the recurrent presence of heartburn or regurgitation (for example, at least once per week), has been estimated in an approximate range of 19.6% to 40%.[5] These findings suggest that about one-fifth of the Mexican adult population meets GERD criteria, which places the country among the regions with the highest disease burden in the world and reinforces its relevance as a public health issue.

Traditionally, GERD has been considered a disease of middle-aged adults and older people, with higher prevalence of symptoms in subjects aged ≥ 50 years old and higher risk of severe erosive esophagitis as age advances.[8] However, recent data suggest a change in this pattern. In an analysis of more than 54 million electronic records in the United States of America (2006-2016), the proportion of patients with a GERD diagnosis increased in all age groups, with the greatest relative increase observed in the 30 to 39 years age

group, indicating that GERD is increasingly affecting young adults.[9] This probably reflects the greater population-wide exposure to lifestyle-related risk factors (see the section "Contemporary risk factors") rather than a change in the clinical definition of the disease.

A slight predominance of symptoms such as heartburn or regurgitation reported in women has been described,[10] but there is a higher prevalence in men of the GERD spectrum, as it has been suggested that esophageal mucosa epithelium is more fragile to gastroduodenal reflux in men than in women, and on the other hand, that women are more susceptible to GERD symptoms than men.[11]

Within the GERD spectrum (non-erosive reflux disease, erosive disease, peptic stricture, and Barrett's esophagus), most patients correspond to forms of non-erosive reflux disease,[12,13] while only a subset presents erosive esophagitis on endoscopy and a minority develops complications such as Barrett›s esophagus.[13]

However, unlike prevalence, data on the incidence of GERD are limited, mainly due to symptom variability and the absence of standardized diagnostic criteria in population studies, which prevents accurate estimation of the occurrence of new cases over time.

Peptic ulcer disease

PUD is defined as a lesion consisting of a solution of continuity of the gastric or proximal duodenal mucosa, in the esophagus, in the gastric or duodenal mucosa, or in the gastric mucosa of Meckel's diverticulum, measuring more than 0.5 cm in diameter.[14] The annual incidence of PUD is variable and is modified by risk factors such as *H. pylori* infection or the use of non-steroidal anti-inflammatory drugs (NSAIDs), as up to 70% of gastric ulcers and 90% of duodenal ulcers are positive for *H. pylori*.[15] *H. pylori* infection and NSAID use increase the risk of ulcer bleeding independently, increasing the risk 1.79 and 4.85 times,[16] respectively. In global terms, in the past the prevalence of PUD was estimated to be 5% to 10% and the incidence 0.1% to 0.3% per year, but there is evidence that both have decreased[17] and that the current prevalence is 0.12% to 1.5% and the annual incidence 0.10% to 0.19%.[14]

One of the consistent findings is that the incidence of PUD has been decreasing over the years in relation to the decrease in *H. pylori* in certain developed countries. For example, a study conducted in Belgium showed that the incidence of PUD from 1994-1995 decreased when compared to that of 2002-2003, from 0.40% to 0.19%, respectively,[18] and another Danish cohort showed a decrease from 0.18% to 0.15% between 1993 and 2002, although accompanied by an increase in the incidence of PUD related to NSAID consumption from 39% to 53% in the same time period.[19] When comparing the average annual percentage change (AAPC) in incidence in different countries between the years 2000 and 2018, it is observed that incidence has decreased in many countries over the years. For example, in Latin American countries such as Costa Rica, Chile, and Mexico, the AAPC was –7.7, –3.9, and –2.5%, respectively, and collectively the three countries had an AAPC of –4.1%, similar to that of Western Asia (–4.0%) and Northern and Eastern Europe (–4.2%), in contrast to a greater decrease in Southern Europe (–5.1%) and a smaller decrease in East Asia (–3.4%), Western Europe (–3.2%), and Oceania (–2.0%).[20] Another factor that may be related to the decrease in PUD is the uncontrolled use of proton pump inhibitors in certain regions, such as Latin America, which should be studied.

On the other hand, the prevalence of PUD appears to be less reported than incidence. In a systematic review, it was found that only three studies reported the prevalence of PUD with a median of 7% and a range of 5.3-15.7%.[14] A Swedish study reported representative epidemiological data of its population of patients with symptomatic and asymptomatic PUD, with the prevalence being 4.1%.[21]

Some authors mention that in Latin American countries the estimated prevalence and incidence may not be so accurate, as the diagnosis of PUD is usually counted with hospitalizations or with the diagnosis of doctors only from public hospitals,[20] and there is Mexican evidence that up to 35% of patients taking proton pump inhibitors chronically did so for an incorrect diagnosis in public hospitals,[22] which may be related to different figures due to incorrect diagnoses and patients not counted in private hospitals.

Gastritis

Gastritis is defined as a spectrum of conditions in which there is histologically documented inflammation by an infiltrate with lymphocytes, plasma cells, and polymorphonuclear cells, and in some cases, glandular atrophy may occur.[23] It is classified as acute or chronic depending on the type of inflammatory infiltrate related to differences in its etiology. Acute gastritis covers a wide spectrum of etiologies, which are divided into transmissible and non-transmissible agents according

to the Kyoto classification. The most common etiological agent of chronic gastritis is *H. pylori*, although other agents and host-related disorders, such as autoimmune gastritis, may also be included.[23]

To discuss the epidemiology of gastritis, it is inevitable to set aside the epidemiological data on *H. pylori* infection. Once a person becomes infected, the infection usually persists throughout life. The estimated global prevalence in adults has decreased from 50-55% to 43% between 2014 and 2020,[24] which is attributed to an improvement in socioeconomic level and housing and hygiene standards. The estimated age of infection is before 10 years of age. An increase in the prevalence of infection has been seen in regions and countries where low and medium socioeconomic levels predominate. Prevalence is also higher in rural than in urban settings. Regarding Mexico, it is estimated that the percentage of the population susceptible to infection is 85.9% and the annual infection rate among the susceptible population is 0.092.[25] According to the latest Mexican consensus on *H. pylori*, there is a decrease in prevalence in the young population, but the results may vary according to geographic region. On the other hand, it is important to mention that *H. pylori* eradication, when timely, decreases the risk of progression to premalignant lesions such as atrophic gastritis.[26]

According to a systematic review,[27] between 1990 and 2020, there has been a decrease in the prevalence of gastritis, exactly 12.91%, as it went from being 521.67 cases (95% confidence interval [95% CI]: 419.28-647.52) per 100,000 subjects to 454.31 cases (95% CI: 372.66, 558.27) per 100,000 subjects. It is more common in women than in men in all age groups, and the highest prevalence occurs in middle age, probably correlated with the average age of *H. pylori* infection. It is estimated that by the year 2050, the prevalence will increase by 33.82%, attributed to aging and therefore population growth.[27] It is also important to mention that this increase is linked to greater use of endoscopy as a primary diagnostic tool for gastritis.[28]

Another type of gastritis, which is not directly related to *H. pylori* infection, is autoimmune gastritis, whose mechanism is regulated by autoreactive $CD4^+$ and Th1 lymphocytes that attack the hydrogen ion pump, and the production of autoantibodies against parietal cells as a consequence of the damage generated by Th1 cells. Currently, the estimated worldwide prevalence of autoimmune gastritis is estimated in a range of 0.1% to 4.5% in the general population, and between 8% and 20% in studies that have used serological diagnosis, with variations among different ethnicities and geographic regions.[29]

Functional dyspepsia

FD is part of the disorders of gut-brain interaction (DGBI) defined by the Rome criteria, of which the most recent are the Rome IV criteria, but in May 2026, the new version (Rome V criteria) will be published. The Rome IV criteria are:

– One or more of the following: bothersome postprandial fullness, bothersome early satiation, bothersome epigastric pain, or bothersome epigastric burning.
– No evidence of structural disease (including abnormalities on upper digestive endoscopy) that can explain the symptoms.

These criteria must have been present during the previous 3 months, with symptom onset at least 6 months before diagnosis.

In addition, FD is classified into postprandial distress syndrome (PDS) and epigastric pain syndrome (EPS). PDS must include one or both of the following at least 3 days per week:

– Bothersome postprandial fullness, that is, severe enough to impact usual activities.
– Bothersome early satiation, that is, severe enough to impact usual activities.

Postprandial epigastric pain or burning, subjective epigastric bloating, excessive belching, and nausea may be present. The presence of vomiting warrants consideration of another disorder, and heartburn is not a dyspeptic symptom, but it can frequently coexist. Symptoms relieved by bowel movements or gas should generally not be considered part of dyspepsia.

EPS must include at least one of the following for at least 1 day per week:

– Bothersome epigastric pain, that is, severe enough to impact usual activities.
– Bothersome epigastric burning, that is, severe enough to impact usual activities.

As supportive criteria, pain can be induced by food intake, or relieved by food intake, or can occur during fasting. Subjective epigastric bloating, belching, and nausea may be present. Persistent vomiting probably suggests another disorder. Heartburn is not a dyspeptic symptom, but it can often coexist. Pain does not meet biliary pain criteria. In general, symptoms relieved by bowel movements or gas should not be considered part of dyspepsia.[30]

The Rome Foundation Global Epidemiology Study (EGFR) was conducted using the Rome IV criteria in the general population via the Internet in 24 countries, by personalized household interviews in seven countries, and by both methods in two countries, including a total of 73,076 subjects. The global prevalence of FD by Internet (n = 54,127 subjects) was 7.2% (95% CI: 7.0, 7.4) and in household surveys was 4.8% (95% CI: 4.5, 5.1). The EGFR included four Latin American countries, which were Argentina, Brazil, Colombia, and Mexico, and found a prevalence of FD/uninvestigated with a variability of 6.6% in Mexico to 10.6% in Brazil.[31] It is important to mention that the EGFR showed that FD has become the second DGBI in order of frequency after functional constipation, which is extremely important.[32] However, since this is a cross-sectional study based on clinical symptoms without upper digestive endoscopy (uninvestigated dyspepsia), this prevalence could be overestimated.

It should be noted that the prevalence of FD can vary according to the diagnostic criteria used, and thus a systematic review and meta-analysis published practically at the time the EGFR was published reported a pooled prevalence of uninvestigated dyspepsia that varied from 6.9% (95% CI: 5.7-8.2) in studies that used Rome IV criteria to 17.6% (95% CI: 9.8-27.1) in those that defined dyspepsia with Rome I criteria.[33]

Regarding FD, a recent published study, derived from the EGFR, has shown that FD/uninvestigated in the general population is more frequent in women than in men, with 8.7% (95% CI: 8.4-9.1) and 5.7% (95% CI: 5.5-6.1), respectively, and decreases with age from 9.5% (95% CI: 9.1-9.9) in the 18 to 34 years age group to 3.9% (95% CI: 3.5-4.3) in those over 65 years. The most frequent subtype is PDS (66.6%).[34] More recently, a clinic study conducted in Mexico City, using the clinical Rome IV criteria, found that 65.5% of patients corresponded to EPS, 27.6% to PDS, and 6.9% to the overlap of both. The above shows a difference between epidemiological studies in the open population and those conducted in the office.[35]

Additionally, the same EGFR showed that among subjects with FD criteria, 26.1% met the Rome IV criteria for irritable bowel syndrome, 9.0% for functional heartburn, and 7.0% for chronic nausea and vomiting disorder (also DGBI). Likewise, when subjects met the Rome IV criteria for FD, they presented a significant association with an increase in the prevalence of anxiety and depression, lower quality of life, and higher frequency of healthcare-seeking behavior.[34]

Contemporary risk factors

The disorders encompassed in APD share a set of well-established risk factors (Table 1). Historically, some factors have been associated to a greater extent with the development of APD, with *H. pylori* infection and the use of acetylsalicylic acid or other NSAIDs being the most prominent.[1]

Drugs classically associated with APD, but especially with PUD, are NSAIDs.[36] However, others have also been considered at risk, such as corticosteroids and bisphosphonates combined with NSAIDs, sirolimus, selective serotonin reuptake inhibitors, and 5-fluorouracil[37] (Table 2).

Smoking constitutes one of the classic and well-documented risk factors in the APD spectrum[1]. In fact, a Mendelian randomization study showed that genetic predisposition to smoking is associated with increased risk of 20 gastrointestinal diseases, including GERD (odds ratio [OR]: 1.65), gastric ulcer (OR: 1.95) and duodenal ulcer (OR: 1.64), and acute gastritis (OR: 1.54) and chronic gastritis (OR: 1.33).[38] These data confirm that smoking is a key and potentially modifiable determinant in the prevention of APD.

Other commonly associated factors include alcohol consumption. In fact, a meta-analysis of observational studies found that people who consume alcohol have a higher risk of GERD compared to non-drinkers or occasional drinkers (OR: 1.48), with a dose-response relationship in which more frequent drinkers have the highest risks.[39] Furthermore, a Mendelian randomization study showed that genetic predisposition to higher alcohol intake is associated with an increased risk of chronic gastritis (OR: 2.9).[38] Likewise, a study in the Korean population documented that, of 1,638 patients with atrophic gastritis, 67.5% consumed alcohol.[40]

Regarding the type of diet, foods that have been identified as triggers of GERD symptoms include high-fat or fried foods, acidic foods, citrus juice, tomato and its derivatives, chocolate, coffee and tea, carbonated beverages and alcohol, as well as irregular eating patterns, large meals, and food intake immediately before bedtime.[41] In particular, alcohol, chocolate, fatty foods, and smoking have been associated with a reduction in lower esophageal sphincter pressure, which facilitates reflux episodes and the occurrence of heartburn and regurgitation.[42]

On the other hand, in recent decades, other risk factors for APD have been identified, such as socioeconomic and demographic factors, in which low socioeconomic level and inadequate hygiene conditions play an important role in the development of GERD and gastritis.[6,28]

Table 1. Risk factors for acid-peptic disease

Demographic	Lifestyle	Diet	Exposure
Low socioeconomic level Low education level Inadequate hygiene conditions Low access to safe drinking water sources	BMI > 30 kg/m^2 Smoking Sedentary lifestyle Alcohol consumption	Coffee Tea Chocolate Mint Carbonated beverages Vitamin A deficiency	Use of NSAIDs Use of acetylsalicylic acid *H. pylori* infection

BMI: body mass index; NSAIDs: non-steroidal anti-inflammatory drugs.

Table 2. Non-steroidal anti-inflammatory drugs associated with acid-peptic disease

Drugs	Relative risk
Aceclofenac, celecoxib, ibuprofen	< 2
Diclofenac, meloxicam, ketoprofen	2.4
Naproxen, indomethacin, diflunisal	4.5
Piroxicam	7.4
Ketorolac	11.5

Another factor that has great relevance is body mass index (BMI), as excess adiposity has been consolidated as one of the most relevant contemporary risk factors for APD.[43] In a two-sample Mendelian randomization analysis, which included 71,522 GERD cases and 261,079 controls, an increase of one standard deviation in BMI was associated with an increased risk of GERD (OR: 1.49), while a greater genetically predisposed waist circumference also showed a suggestive association (OR per standard deviation of 1.14).[44] Likewise, it has been documented that the pooled prevalence of GERD by BMI increased as BMI was higher.[7]

Other contemporary risk factors have been studied more recently. For example, it is known that bacterial interaction together with host factors determines the outcome of *H. pylori* infection, these bacteria having different strains producing different proteins.[17] These proteins are encoded in certain integrative and conjugative elements (ICE, previously known as "genomic islands"). Mucito-Varela et al.[45] have analyzed these ICEs present in *H. pylori* from Mexican patients, which showed associations between isolated *H. pylori* strains and the pathology that the patients were experiencing, and in particular those concerning PUD, gastric ulcer, duodenal ulcer, and gastritis. Furthermore, certain proteins encoded in ICEs, called ICEHptfs3 and ICEHptfs4, are associated with *H. pylori* normally isolated in different regions of the world, especially in Asia, followed by Africa, America, and Europe, as well as some not previously associated with any particular region, which could demonstrate that these ICEs could contribute to some extent to the development of different gastrointestinal pathologies and that the variability of *H. pylori* is not homogeneous even by continents.[45]

Regarding FD, associated risk factors include smoking, with an apparent higher probability of PDS in active smokers, and frequent alcohol consumption, which has been determined in observational studies and Mendelian randomization analyses.[46] The history of infectious gastroenteritis is an important risk factor that has been studied in recent years; it is what is called post-infection (PI) FD. This can occur after infections by pathogens such as *Salmonella* spp., *Escherichia coli* O157, *Campylobacter jejuni*, *Giardia lamblia*, and Norovirus.[47,48] A systematic review with meta-analysis reported a prevalence of PI-FD of 9.55% among 9,517 subjects who had acute gastroenteritis.[47] Also derived from the EGFR, the analysis of PI-DGBI has just been published, showing that 35.8% (95% CI: 34.3-37.3) of subjects reported that their symptoms started after an infection.[34] The highest frequency of these disorders was reported in Asia (7.1%) and Latin America (6.4%), which is important data for our population, and FD was more frequent among subjects with PI-DGBI compared to those with non-PI DGBI (32.2% vs. 18.2%).[49] As with other APDs, *H. pylori* infection has been related to FD, but this is more controversial. A systematic review that included 21 randomized clinical trials showed a relative risk reduction of FD of 10% after *H. pylori* eradication, with a number needed to treat of 14 (95% CI: 10-25).[50,51]

Finally, a bidirectionality has been reported between mood disorders and DGBIs.[52,53]

Conclusions

APD encompasses symptoms that are part of the most common reasons for consultation in medical

practice, such as GERD, PUD, gastritis, and FD. Their epidemiology is variable according to the countries where studies are conducted, and while there are data on their prevalence, there is actually a lack of information on the real incidence of these disorders. It should be noted, however, that they have some common characteristics, such as higher prevalence in women than in men, and all are associated with risk factors such as smoking, alcohol consumption, and *H. pylori* infection. Furthermore, although gastritis may have an infectious etiology, FD as a DGBI can occur after a gastrointestinal infection of any etiology, that is, PI-FD.

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Conflicts of interest

S.A. Zaragoza-Galicia, J.F. Zárate-Villazón, C.L. Cruz-Rico, G. Mendoza-Domínguez and A.S. Morales-Guzmán have nothing to declare. M.J. Schmulson: Advisory Board of Daewoong South Korea, Gemelli Biotech Inc, Moksha 8 Mexico, Pro.Med.CS. Praha a.s.; speaker for Alfa Sigma Mexico, Armstrong Mexico, Carnot, Daewoong South Korea, Ferrer Mexico/Central America, Medix Mexico, Megalabs Ecuador, Tecnofarma Colombia/Bolivia; educational materials for Moksha 8 Mexico.

Ethical considerations

Protection of people and animals. The authors declare that no experiments were performed on human beings or animals for this research.

Confidentiality, informed consent, and ethical approval. The study does not involve personal data, medical records, or human biological samples, so it does not require ethical approval. The SAGER guidelines do not apply.

Statement on the use of artificial intelligence. The authors declare that no type of generative artificial intelligence was used for the writing or creation of content of this manuscript.

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