Supplementary Table 1 Key events in the history of gastritis research

Year	Scientist	Discovery	References
1732	*G.E. Stahl*	First mention of inflammation of the GM	[3]
1808	*F.J.V. Broussais*	He described on cadaveric material common inflammations in the stomach, calling them "gastritides", distinguished gastritis (gastritides) as a separate nosologic form	[4]
1838	*R. Carswell*	Refuted the gastric changes described by *F.J.V. Broussais*, attributing them to the result of postmortem self-digestion by gastric juice and putrefaction	[5]
1854	*J. Handfield, W. Fox*	First microscopic description of the inflammation of the gastric mucosa; subdivision of the gastric mucosa lesions into diffuse and segmental forms	[6]
1859	*W. Brinton*	Classified gastritis into acute, subacute, and chronic gastritis; presented their histologic differences and compared them with their clinical manifestations	[6]
1868	*A. Kussmaul*	He proposed the use of a gastric probe, which initiated a period of research on gastric function, including the study of its motility and secretory activity of the glands, which led to the emergence of statements in favor of functional disorders, gastritis began to be identified with dyspepsia	[7]
1870	*S. Fenwick*	Suggested that CG can rightly be considered an organic pathology; suggested a relationship between pernicious anemia and gastric gland atrophy	[8]
1875	G. Bottcher, M. Letulle	first suggested that gastric ulcers were caused by bacteria.	[33]
1881	*C. Klebs*	A report of a bacillus-like organism found in the lumen of gastric glands and in the gastric mucosa of	[34]

		dogs with the formation of a characteristic "inflammatory infiltration" therein	
1889	*W. Jaworski*	examined stomach flushes from humans and discovered a characteristic spiral-shaped bacteria that he named Vibrio Rugula Suggested that Vibrio Rugula may play a possible pathogenic role in the development of gastric diseases	[35]
1893	*G. Bizzozero, C. Golgi*	Described spiral-shaped bacteria in the parietal cells and glands of the gastric mucosa of dogs, later identified as H. canis, H. felis, and H. heilmannii. Noted that these microorganisms can infect both pyloric and fundal gastric mucosa	[36]
1896	*H. Salomon*	reported the presence of spirochetes in the gastric mucosa of dogs, cats, and rats and described a series of experiments where he was able to transfer a spirochete bacterium identified in the stomach of dogs to white mice	[37]
1900	*K. Faber, K.E. Bloch*	Detailed atrophic changes in the inflammation of the gastric mucosa in a patient with pernicious anemia were described	[9]
1906	W. Krienitz	identified spirochetes in the stomach of a carcinoma patient.	[38]
1921	J.S. Edkins	Using Giemsa staining, he identified spiral-shaped bacteria in the floor of a peptic ulcer and in the antral region of the stomach and theorized about the relationship between the development of peptic ulcer disease and the bacterium he discovered, which he named Spirochete regaudi.	[40]
1921	*G.H. Whipple*	Established that raw liver leads to increased blood erythrocyte levels in dogs with posthemorrhagic anemia	[10]

1926	*G.R. Minot, W.P. Murphy*	Used raw liver to treat pernicious anemia in humans	[11]
1927	*K. Faber*	by injecting a 10% formalin solution into the abdominal cavity protected GM from postmortem autolysis and putrefaction.	[4]
1930	*G.E. Konjetzny*	studied resected stomachs from patients with peptic ulcer disease (PUD) and RR, developing a special technique that prevented the possibility of postmortem tissue autolysis.	[4]
1932	*R. Schindler*	Invented the semi-rigid gastroscope.	[16, 17]
1934	*G.H. Whipple, G.R. Minot, W.P. Murphy*	Were awarded the Nobel Prize in Physiology and Medicine	[12]
1940	J.L. Doenges	found spiral-shaped bacteria in the gastric mucosa of the rhesus macaque he studied and in 43% of resected human stomach samples	[41]
1944	*S. Warren, W.A. Meissner*	Described intestinal metaplasia in patients with chronic gastritis	[15]
1947	*R. Schindler*	He classified gastritis into acute and chronic, subdividing the latter into superficial, atrophic and hypertrophic gastritis	[17]
1948	*E.L. Smith, E.L. Rickes*	Vitamin B12 has been isolated from liver	[13]
1949	*I.J. Wood*	Invented a simple biopsy tube.	[18]
1956	*R. Cheli, M. Dobero*	He also classified gastritis into "superficial", "interstitial" and "atrophic" gastritis	[21]
1957	*B.I. Hirschowitz*	Report on the invention of a flexible fiber optic fibrogastroscope	[19]
1958	*I.J. Wood, L.I. Taft*	Outlined possible etiologic factors of chronic gastritis (alcohol, diet, stress, radiation, etc.).	[23]
1962	*W.J. Irvine*	Detected antibodies to gastric parietal cells in the serum of patients with pernicious anemia	[23]

1967	*S. Ito*	published a photograph of a gastric parietal cell showing a bacterium later identified as H. pylori	[42]
1972	*R. Whitehead*	Divided gastritis topographically into antral, fundal, cardiac, and pyloric gastritis; proposed the division of nastritis into "active" and "inactive" gastritis	[24]
1973	*R.G. Strickland, I.R. MacKay*	Suggested using the terms "type A" (autoimmune) gastritis to refer to gastritis of the body of the stomach, and "type B" (non-autoimmune) gastritis to refer to antral gastritis	[25]
1975	*G.BJ. Glass, C.S. Pitchumoni*	Added "type AB gastritis" to the classification to indicate gastritis spreading from the body of the stomach to the prepyloric region	[26]
1975	*P. Correa*	Presented the sequence of pathologic changes of GM in chronic gastritis	[27]
1979	J.R. Warren	noticed a blue line on the surface of the gastric mucosa of a patient with active chronic gastritis. After analyzing a large volume of biopsy material, he suggested that these were bacteria that somehow played a role in gastric disease	[43]
1983	*R. Warren, B. Marshall*	Published findings on the Helicobacter pylori bacterium and its role in shaping GM changes	[29]
1987		The European Helicobacter pylori Study Group (EHSG) was founded to promote interdisciplinary research into the pathogenesis of H. pylori-associated diseases.	[47]
1988	*J.I. Wyatt, M.F. Dixon*	The term "type C gastritis" or "chemical gastritis" has been suggested	[48]
1990	*c. Sydney (Australia)*	The "Sydney System" of gastritis classification has been adopted	[51]
1994	*c. Houston (USA)*	A modification of the Sydney classification, it restores the division of CG into types A, B and C	[50]

2005		A system for determining the stage of chronic gastritis is proposed - OLGA-system	[54]
2005	*B.J. Marshall, J.R. Warren*	The Nobel Assembly of the Karolinska Institute in Stockholm awarded the Prize in Medicine and Physiology to Australian scientists for the "discovery of H. pylori and elucidation of its role in the development of gastritis and peptic ulcer disease"	[44]
2015	*c. Kyoto (Japan)*	An etiologic classification of chronic gastritis has been developed	[62]