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Enhancing Peptic Ulcer Therapy: Innovations in Mucoadhesive Drug Delivery

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Abstract

Peptic ulcer disease remains a sincere gastrointestinal demand that makes medication administration difficult because of the harsh and changing stomach environment. This study investigated the potential of mucoadhesive drug delivery devices to improve the accuracy and effectiveness of peptic ulcer therapies. Owing to the inherent characteristics of mucoadhesive drug delivery systems, localized and sustained drug release is made possible by their extended contact and adherence to the mucosal surfaces of the gastrointestinal tract. These formulations can overcome the drawbacks of traditional administration techniques, such as quick evacuation and short residence times at the ulcer site, by exploiting their sticky qualities. This study investigates the different mucoadhesive polymers used in peptic ulcer medication distribution, clarifying their adherence, biocompatibility, and formulation methods. Among other polymers, chitosan, alginate, and carbopol have exceptional mucoadhesive properties that allow them to adhere to the ulcerated mucosa and improve medication retention. This paper also covers the design concepts and developments in mucoadhesive formulations, such as site-specific delivery strategies, nanotechnology-based systems, and innovative drug carriers. These advancements aim to reduce systemic adverse effects, enhance bioavailability, and optimize medication release kinetics. We examined published studies and commercial formulations of mucoadhesive drug delivery devices for the treatment of peptic ulcers. This article emphasizes the revolutionary significance of mucoadhesive drug delivery methods in the treatment of peptic ulcers. This highlights their ability to get around delivery issues, improve therapeutic results, and perhaps transform treatment modalities for this common gastrointestinal illness.

Keywords: Peptic ulcer, mucoadhesion, mucoadhesive polymers, design, application

INTRODUCTION

Peptic Ulcer

A chronic illness known as peptic ulcer arises from a mismatch between the natural defensive mechanisms of the gastrointestinal mucosa (fluids acids and pepsin) and proactive mechanisms (fluids of mucin and bicarbonate or sufficient prostaglandin E2, nitric oxide, sulfhydryl compounds, blood flow, and antimicrobial agents). The etiology of stomach ulcers has also been linked to behavioral and environmental factors, including smoking, eating poorly, drinking using non-steroidal anti-inflammatory drugs (NSAIDs) medicines drugs, and infection caused by *Helicobacter pylori* [1]. Ulcers are characterized by the shedding of inflamed, dead layers of tissue, resulting in exposed wounds on the layer of the skin and mucosa of the mouth. Cover ulcers of the outer layer of the skin or mucous membranes that display superficial tissue loss are known as ulcers. Although ulcers can occur anywhere, they tend to

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arise in the digestive system of the skin of the legs. There are multiple types of ulcers, including genital, peptic, intestinal, and oral ulcers [2]. Causative factors include either Helicobacter bacterial infestation or adverse pharmacological responses to specific medications such as non-steroidal anti-allergic medicines. Peptic ulcers can cause weight loss, bloating, vomiting, nausea, and dark stools, which are signs of gastrointestinal bleeding [3].

Peptic ulcer illness is often defined as a noticeable mucous breach farther over three to five millimeters in the gastrointestinal and duodenal regions. Consequently, unlike medical criteria, indigestion in an endoscope is based only on scientific issues [4]. Peptic ulcer is a common condition affecting 0.1–0.3% of the world's population annually, or between 5 and 10% of people at some point in their lives [5].

Anatomy of Peptic Ulcer

The gastrointestinal tract is situated in the upper abdomen directly below the diaphragm [6] (Figure 1).

Types of Peptic Ulcer

"Peptic ulcer" refers to any type of gastrointestinal scar, including duodenum or gastric scars. Gastrointestinal tract ulcers caused by acids and peptic ulcers are often observed in the duodenum or stomach. It was once thought that eating spicy food and stress caused this type of ulcer. Recent studies have revealed that these are exacerbating variables. The infection causes either an infection with *H. pylori* or an unfavorable response to medication, such as non-steroidal anti-inflammatory medicines [6].

Esophageal Ulcer

Similar to heartburn symptoms, esophageal ulcers develop in the esophagus (Figure 1) [7]. They often originate near the end of the food pipe and cause pain directly below the breast. Acid reflux, GERD, and prolonged NSAIDS are linked to esophageal ulcers [8].

Apthous Ulcer

Oral mucosal ulceration is a pathological disorder known as recurrent aphthous stomatitis (RAS) [9]. These ulcers often have round or ovoid lesions, as well as grayish or yellow flooring [10]. RAS is the most prevalent ulcerative disease infecting the mucous membrane of the mouth in the US, claims Shulman [11]. RAS has been linked to several predisposing variables including immunity, systemic illness, and local causes. However, the precise cause of RAS is still a mystery [12]. The literature describes three primary forms of RAS: minor, major, and herpetiform.

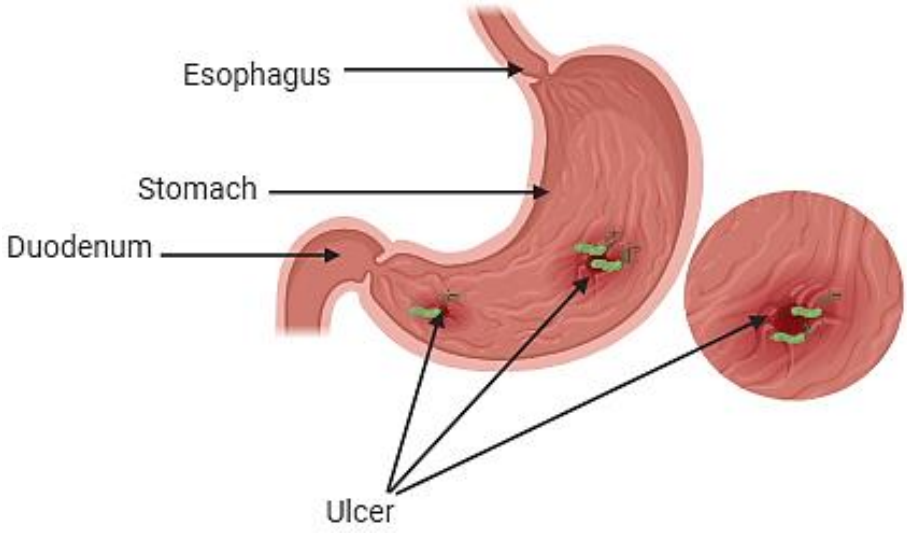

Figure 1. Peptic ulcer disease.

Etiology

Peptic ulcer disorder (PUD), or gastric ulcer disease, can have several causes, but the two most prevalent are PUD linked to NSAIDs and PUD caused by *Helicobacter pylori* [13].

The reasons for this condition are.

- *H. pylori* disease
- Use of steroids
- Medication

Rarely

- Disease of Zollinger-Ellison
- Misalignment
- Tension
- Viral infection
- Lack of the veins
- Laser treatment

PUD Connected to Helicobacter pylori

The gram-negative bacillus *Helicobacter pylori*, commonly known as Helicobacter, causes 70–90% of stomach ulcerations along with approximately 90% of ulcers in the duodenum and is present within the gastrointestinal epidermal cells. The illness is usually detected in children and is more prevalent in affluent neighborhoods. Living organisms can adhere to and irritate the mucosa of the gastrointestinal tract (GI) tract owing to their broad range of variables associated with virulence, which can lead to hypochlorhydria or achlorhydria, ultimately resulting in gastric ulcers.

Characteristics of Helicobacter pylori Virulence

1. *Urease:* By neutralizing the acidic gastric environment, urease's release protects the organism by breaking down urea into ammonia.
2. *Toxins:* CagA/VacA has been related to damage to host tissue and inflammation of the stomach mucosa.
3. *Flagella:* Enables movement in the direction of the stomach epithelium and provides motility.

PUD Associated with NSAIDs

After *Helicobacter pylori* inflammation, NSAID use is the 2nd most prevalent cause of PUD [14, 15]. Normally, prostaglandin production protects stomach mucosa. NSAIDs suppress COX-1, which prevents prostaglandin formation. This reduces the mucosal plasma supply or the formation of bicarbonate and digestive mucosa.

Medication

In addition to NSAIDs, the etiology of PUD is associated with fluorescence, potassium chloride, bisphosphonates, and medication.

There seems to be a nonlinear relationship between smoking and duodenal ulcers. Alcohol can cause acidity and irritation to the stomach mucosa.

Epidemiology

It is projected that 5–10% of individuals may have peptic ulcer (PU) at a given time in their lives. Its annual incidence ranges from 0.3 to 1.9. The incidence of PU complications is now lower in accordance with the majority of epidemiological analyses conducted over the past few years [16]. This is especially true in developed countries. Over the last 20 years, hospital admissions and PU mortality have both decreased significantly in Europe [16, 17]. Such a decline is accompanied by a decrease in pyloric diseases as well as the widespread use of antisecretory drugs with a decrease in the prescription of non-steroidal anti-inflammatory drugs, medicines (NSAIDs), and smoke [18, 19].

Pathophysiology

A balance between the variables that maintain and destroy the stomach mucosa is the cause of PUD. Risk factors that make PUD more likely to develop [20].

The use of Aspirin and the presence of *Helicobacter pylori* are typical risks that precede PUD and the stomach. Less common risk factors include Crohn's syndrome, radiation therapy, severe sickness, autoimmune issues, alcoholism, cigarettes, and heroin [21].

Infection with *Helicobacter pylori*

- Bacteria can invade the stomach lining and cause persistent inflammation.
- This makes the duodenum and stomach more vulnerable to damage from stomach acid by weakening the protective mucous membrane.

Use of NSAIDs

- Aspirin and ibuprofen are examples of NSAIDs that can directly irritate the stomach lining.
- They prevent the production of prostaglandins, which are necessary for preserving the integrity of the stomach lining.

Acid Generation

- The development of ulcers may be facilitated by excessive gastric acid (stomach acid) output.
- Several substances, notably the hormone gastrin, which increases acid secretion, control the amount of acid produced.

Reduced Mucosal Defense Systems

- Mucus secretion and bicarbonate generation are two defense mechanisms of the stomach and duodenum that aid in maintaining a barrier [22].

Diagnosis

An ongoing investigation of patients in Taiwan found that approximately two-thirds of those suffering from digestive illness who have an upper endoscopy as part of normal medical keeping remain symptomatic [23]. Among individuals with PU disease who show symptoms, the most typical symptom is stomach discomfort. This has been linked to early satiety, bloating, nausea, and abdominal fullness [21]. In many cases, sporadic symptoms may occur.

Distinctive symptoms play a significant role in making a diagnosis. The first indication of PU is stomach pain. Sometimes, medical professionals will treat ulcers without performing certain tests to diagnose them, and then watch to see whether the symptoms go away to confirm that their initial diagnosis was correct. The presence of *H. pylori* colonization can be demonstrated noninvasively using fecal antigen detection, breath testing, serologic analysis, or invasively using endoscopy and biopsy [24].

Precision of Helicobacter pylori Infections Testing

Urea Breath Tests

The nonradioactive isotopes carbon 13 or carbon 14 must be consumed to pass the urea breath tests. Four to six weeks following the conclusion of eradication therapy, breathing tests for ammonia are one possible remedy. One drawback of this test is that inhibition of proton pumps (PPIs) must be discontinued for a minimum of 14 days prior to it. It is also expensive and inconvenient [25].

Testing for Stools Single-Antigens

Stool antigenic single-antibody testing is similarly dependable for urine breathing testing when conducted using approved laboratory experiment antibody testing [26, 27]. PPI usage should be avoided for some time before the test; nevertheless, using a PPI has a greater impact on urea breathing tests than on stool antibodies for antigens.

Serologic Tests

Serologic antigen tests detect *H. pylori*-specific antigen-G H in the serum; however, this test cannot distinguish between a current virus and a prior illness. Serologic evaluations may be particularly beneficial to clients who are undergoing extensive demographic inspections and are unable to stop using PPIs (such as those with some bleeding from the gut or continued NSAID usage), as they are unchanged by PPI or the use of antibiotics [28].

Endoscopy with Biopsy

For subjects aged 55 years, endoscopic biopsies are recommended for those who have several alert signs to rule out cancer and other severe reasons. Fast urine tissue tests are performed whenever a person is missing a bismuth or antibiotic for a period of four weeks, or a PPI for one to two weeks following an endoscope offers a reliable and affordable method for identifying *H. pylori* infection [28]. Histology is necessary for patients taking these drugs, either with or without fast urease testing.

MUCOADHESIVE DRUG DELIVERY

The idea of skin adhesion garnered significant interest in pharmaceutical technology until the beginning of the 1980s. Prof. Joseph. Robinson is the father of mucoadhesion [29]. The United Material and Tests Society, as the doses the form's resident duration at the application or absorption site is prolonged by sticky delivery methods. They enhance the therapeutic efficacy of the drug by allowing the dose form to make direct contact with the absorbent surface below. Recently, a variety of mucoadhesive drug delivery methods have been developed to produce effects that are simultaneously regional and systemic in the urinary, nasal, oral, chewing, and genital pathways [30].

Loading capacity: The most desirable Eigenschaften einer sticky dosage form is its smoothness of texture, tastelessness, simplicity of administration, excellent mucoadhesive capabilities, and controlled drug flow—ideally directed release.

Since erodible formulations do not need to be retrieved from the system after the dosing interval, they may be beneficial for use. An appropriate adhesion dose is created for a range of medicines. Insulin, leuprolide, octreotide, the hormone that releases thyrotropin (TRH), and ocin are among the peptides administered through the mucosal route; however, their bioavailability is relatively low. that go from 0.1 to 5%) [31].

MUCUS MEMBRANES

Wet surfaces that border the exterior walls of many bodily spaces, such as those used for respiration and digestion, are known as mucosae or their membranes. They are composed of an epithelial layer that sits on top of the lamina propria, a film of connecting tissues whose surfaces are usually wet in the presence of a mucus film [32]. In organs involving the alimentary tract, genitals, and cornea, the epithelium can be multiple or stratified; in organs such as the stomach, small and large intestine, and lungs, the epithelial cells can be coated (Figure 2). The latter have specialized glands, such as salivary glands that release mucus into the outside of the epithelium, or they are then transferred to cells that have such glandular glands. These cells produce a mucous straight on the surface of the epithelium. There are two different types of mucus: a gel coat that sticks to the mucosal surface, and a suspended or luminally soluble form. Mucin glycoproteins, lipids, inorganic salts, and water make up almost every mucous jelly; water makes up more than ninety-five of the system volume, maintaining its hydration [33].

The main structure component of mucus gel that gives it one-of-a-kind cohesive, adhesive, and gel-like qualities is the mucin glycoproteins. The thickness of the mucus layer depends on the gastrointestinal tract; on the mucosal surface, it ranges from 50 to 450 Am. [34]. Lower in the oral cavity to less than 1 Am. Mucus primarily supports lubrication and acts as a barrier against infection [35].

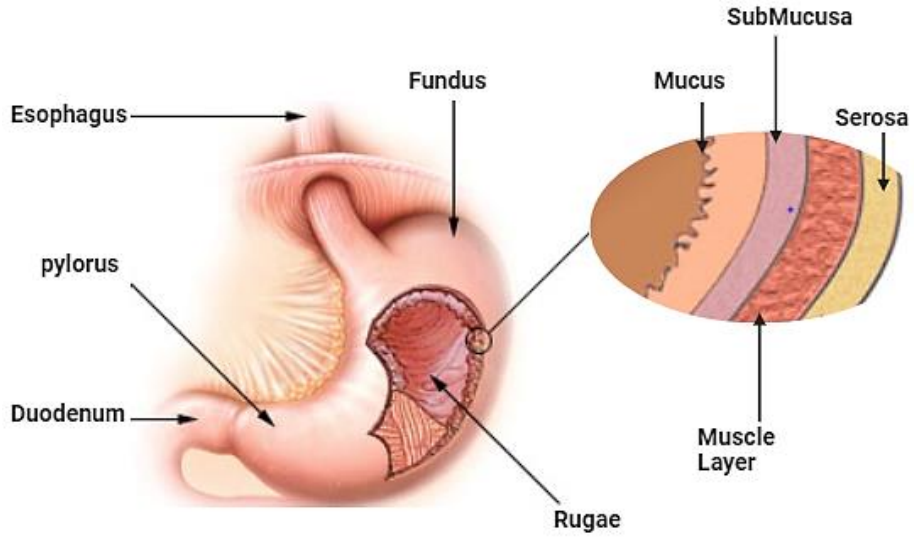

Figure 2. The mucus membrane of the stomach.

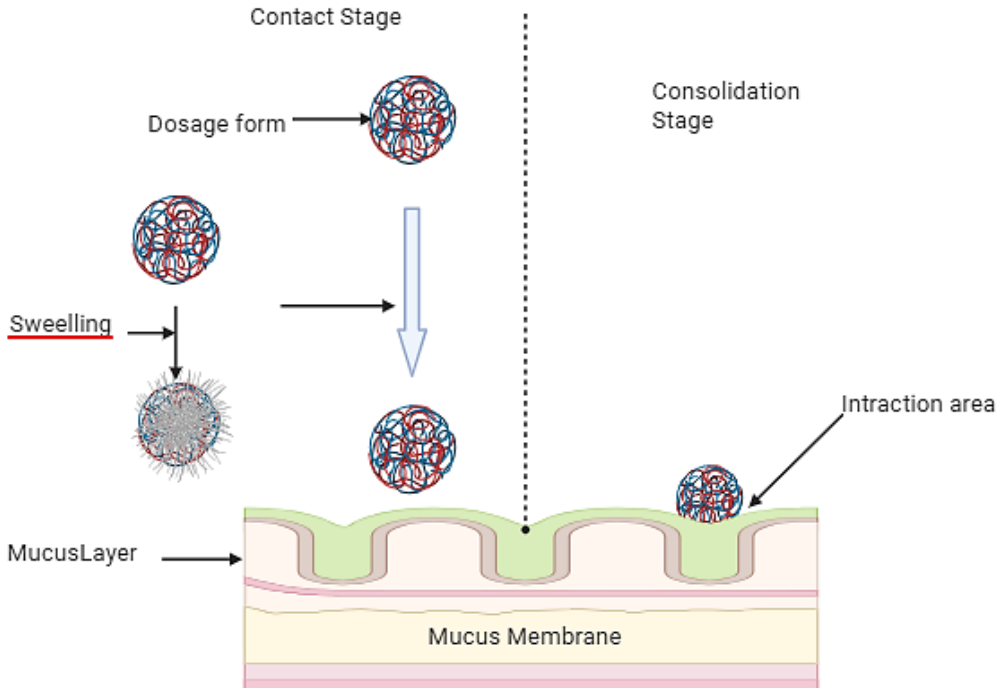

Figure 3. Mechanisms of mucoadhesion.

MECHANISMS OF MUCOADHESION

The process by which certain polysaccharides adhere to the outer layer of the mucus tissue remains largely unclear. For the mucoadhesive to creates intimate contacts and improve surface interactions; it must expand over the substrates to aid in the diffusion of its chains inside the mucous membrane. Condensation and interaction are the two primary stages of the mucoadhesion process (Figure 3). The method of spread characterizes the initial level of swelling and begins to create a profound connection using the musculature through interaction with the mucous layer and adhesion [36]. Occasionally, the distribution mechanism is mechanically connected over the membrane, similar to ocular or vaginal formulations. In other situations, such as when using the nasal route, the aerodynamics of the organ systems are connected to supply help to facilitate the deposition. The particles encounter both repelling (osmosis stress, electric repelling) and attraction (van der Waals forces and electrostatic attraction), etc.) forces as they approach the mucosal surface. Thus, the particle must pass through this repellent obstacle

[37]. The presence of moisture activates the mucoadhesive components in the second stage of consolidation. Weak van der Waals hydrogen bonds enable mucoadhesive molecules to bind together when moisture plasticizes the system. Several main theories describing coagulation processes are the diffusion theory and dehydration. The diffusion hypothesis states that glycoproteins in mucus and mucoadhesive molecules interact with one another through chain penetration and the formation of second-tier ties [37]. This is made possible by the properties of the adhesive device, which encourage both chemical and mechanical contact.

FACTORS AFFECTING MUCOADHESION

- *Molecular weight:* When a polymer's molecular weight surpasses 100,000, its mucoadhesive strength increases. The mucoadhesive strength of polyoxyethylene polymers is directly correlated with their molecular weight, with the two variables falling between 200,000 and 7,000,000 [38].
- *Adaptability distribution:* Intestinal adhesion begins with the formation of polymer chains at the interfacial region. To achieve appropriate attachment to the mucous, polymer chain structures must possess a critical degree of elasticity. After adding polyethylene glycol, the structural flexibility of the polymer improved, which was linked to enhanced chain interpenetration. In general, the viscosity and diffusion coefficient of a polymer can be linked to its mobility and flexibility because a polymer with higher flexibility is more inside the structure of the mucous [39].
- *Crosslinking concentration:* The cross-density volume linkage, the quantity, and mean that some significant and pertinent structural characteristics of a polymeric network are the average holes of voids, volume in molecules of the cross-linked polymers, and both. Thus, the aqueous solution should logically infiltrate the polymer network more slowly as the binding ratio increases. Because of this, the polymer swells insufficiently, and the rate of polymer-to-mucin interpenetration slows [40].
- *Capacity of hydrogen bonds:* Hydrogen bonds are an essential part of the mucoadhesion of a polymer. Bonds involving hydrogen potential are enhanced by the flexibility of the polymer, which is a necessary feature of the desired polymers. Good hydrogen bonding capability is achieved by polymers, such as hydrogen bonding, which is a crucial component of a polymer's mucoadhesion and hydrogen-containing bonds [41].
- *Concentration:* A balance needs to be achieved, and an optimal polymer concentration is essential for mucoadhesion. Excessively high concentrations can prevent adhesion due to a considerably coiled shape, restricting polymer penetration into the mucus, whereas too low a concentration leads to unstable contact. Thus, optimizing mucoadhesive qualities requires determining the ideal concentration [42].

THEORIES OF MUCOADHESION

Wetting Theory

It is among the most venerable materials and creates adhesive hypotheses. Fluids or bioadhesives with reduced solubility to a natural layer are best explained by this idea. Surface and interfacial tension can be used to describe adhesion [43]. The power produced per centimeter whenever an interaction occurs is known as the adhesive work. The thermodynamic work of adhesion and contact angle are topics covered by hypothesis A of moisture. The work relates to the determination of the adhesive and substrate surface tensions using Dupre's formula:

$$w_{A=}\ y_b + y.yb_t$$

where yb, yt, and ybt represent the coating forces of the substrate, and the particular thermodynamic action of adherence, denoted as wA, strain on contact as a result of biological adhesive monomers. Exterior tensions of the two adhering phases added together, minus the visible interface stresses among them, is how the adhesives work [44].

Diffusion Theory

A semi-permanent adhesive connection is formed, according to the hypothesis, when the polymer linkages attach to the epithelial and mucous tissues to a suitable depth [45]. Bioadhesive substances and glycoproteins found in the mucus membrane are tightly connected. The duration of the interaction, coefficients of diffusion, and other experimental factors determine how deeply the polymer chains must enter the mucus to obtain adequate bioadhesion. The molecular weight determines the diffusion coefficient, which rapidly decreases with increasing crosslinking density [46]. This implies that crucial factors influencing interdiffusion control are the adaptability and elasticity of the linkage segments of the mucus glycoprotein molecules and the bioadhesive polymer. A concentration gradient was created during chain interpenetration. Chemical energy variations and small-molecule diffusion efficiencies control the penetration of the bioadhesive polymer chain. In the case of cross-linked plastics, lengthy sequences are extremely difficult to interpenetrate [47].

Electronic Theory

It states that if the mucus glycoprotein network and sticky polymers come into contact, a transfer of electrons takes place because of their different electrical topologies. This caused an electrical double layer to develop at the contact point. Similar to a capacitor, which discharges one time, two sides Discard from contact and fill when they connect, is how this kind of structure behaves [48]. This notion is reinforced by the fact that the organic substrates and mucous adhesive material have specific electrical charges that are in opposition to one side. As a result, particles are exchanged when two of these substances come into contact, forming a second layer of electricity. Freshly generated electronic dual-layer mucous potency is determined by the attractive force inside it [49].

Adsorption Theory

Mucous material will absorb on living membranes as a result of pressures on both various after its initial connection, according to this theory, which holds that the substances will stick jointly after the initial site meeting because of the contact with each of their atomic units. Weak power, such as a van der Waals connection, has significant nearby contact points in absorption [50]. In addition to chemical bonding, the main bonds are secondary. Primary chemical bonds are detrimental to bioadhesion because they are powerful and result in sustainability. By definition, primary chemical bonds are cohesive. Secondary chemical connections include attraction-causing factors, such as hydrophilicity, hydrogen, van der Waals, and electrical bonds [48].

Fracture Theory

After adhesion, the two surfaces separate, which is related to the fracture hypothesis of adhesion [51]. The following equation is necessary. The adhesion resistance equals the fracturing resistance.

$$\sigma = (E \times €/L)1/2$$

where the crucial crack length (L), break power, Young's modulus of elasticity (E), and breaking capacity are described. The adhesion and breaking intensities (σ) were closely correlated. This hypothesis is perhaps the most often applied in research on the mechanistic assessment of mucoadhesion. It looks at how much force is needed to split the two different surfaces following adherence [52].

Mechanical Theory

Takes into account the impact of surface roughness increases the interaction area and promotes stickiness [52].

MUCOADHESIVE POLYMER

Powerful and greatly increasing oral medication delivery, monomers adhere to the mucin/epithelial interface, overcome the comparatively shorter stomach period, and enhance localization for mouth-controlled or sustained release drug delivery systems. Gains at other mucus-covered delivery locations

were also anticipated. Bioadhesive monomers are utilized in the nasal area, eyesight, and sexual cavity, in addition to the gastrointestinal system, which comprises the oral cavity and the rectum [53]. Mucoadhesive polymers and their types are shown in Figure 4.

Hydrophilic Polymers

This category of polymers dissolves in water [54]. When matrices made with these polymers are placed in an aqueous medium, they expand and eventually dissolve [55]. The mucoadhesive components of polyelectrolytes are higher than those of neutral polymers [56]. Anionic polyelectrolytes, such as powerful hydrogen bonds, may be formed between carboxymethyl cellulose, poly(acrylic acid), and mucin present in the mucosal membrane, which can be used to create methods for mucous adhesive delivery [57]. As a cationic polyelectrolyte with outstanding biocompatibility and biodegradability, chitosan has been exploited for the development of mucoadhesive polymers [58]. Chitosan, as a bioadhesive, exhibits adhesive attributes through a study that discovered that levobetaxolol hydrochloride, a potent cardiac β-blocker, produced a mostly eliminated poly(acrylic acid) complex. The ejection of the absorbed medicine over time made the delivery method fragile to deterioration. Electrical contact with conductive mucus groups [56]. A drug delivery matrix with mucoadhesive properties can be created by combining polyamide and counter-ionic medicinal compounds to form charged complexes [59, 60].

A wide range of polysaccharides and their derivatives, including carrageenan, hydroxypropyl methylcellulose, xanthan gum, gellan gum, guar gum, methylcellulose, hyaluronic acid, and chitin, have been developed for use in adhesive delivery methods. Surface activity has also been observed in cellulose and its derivatives [61] In addition to their film-forming ability, reduced ocular irritation is a common reason for using cellulose derivatives with low surface activity in ocular administration systems. Among the many cellulose derivatives, sodium carboxymethyl cellulose (CMC) has been discovered to have the best ocular mucoadhesive characteristics. Sustained delivery systems can be created by combining different anionic polymers with variants of cationic cellulose, including cationic hydroxyethyl cellulose [62].

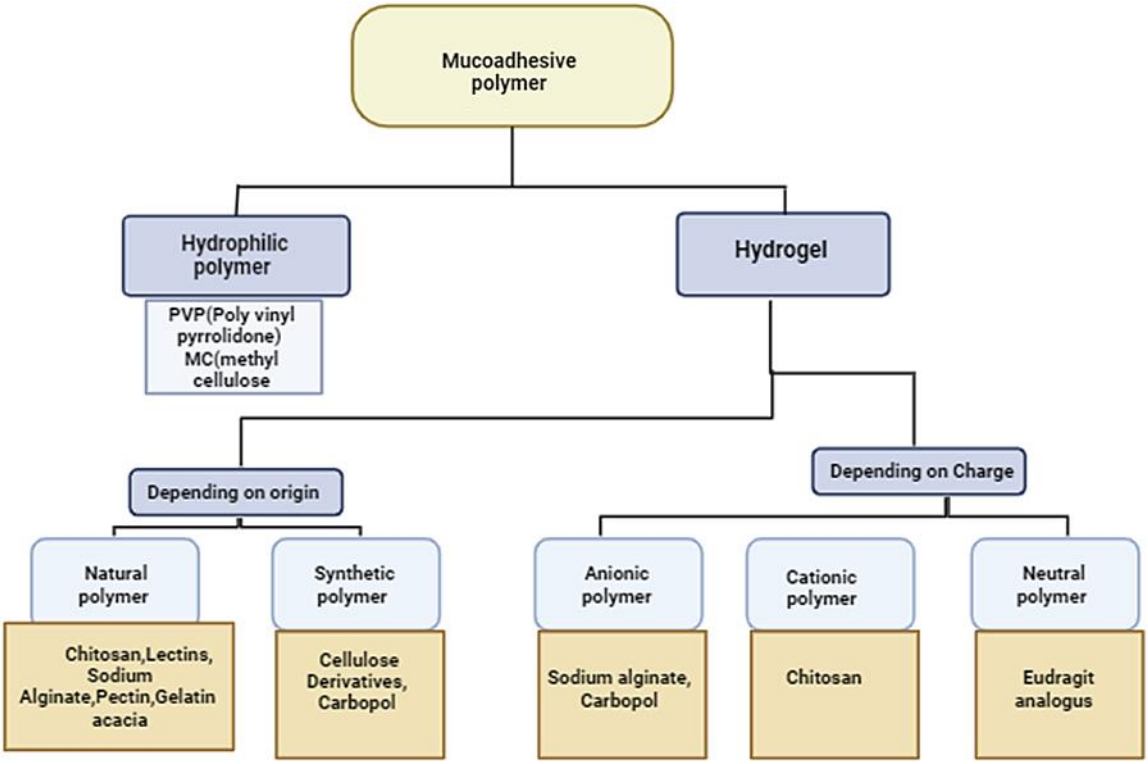

Figure 4. Schematic representation of types of mucoadhesive polymers.

Hydrogels

Hydrogels are polymers of polyester connected in three dimensions that have capacity retention in the liquid inside their transparent framework. Hydrophilic functions, such as carboxyl, amino, and hydroxyl groups, are primarily responsible for the capacity of hydrogels to retain liquids. In general, when the crosslinking ratio increases, mucoadhesion decreases in proportion [63]. Thielmann et al. described the thermal crosslinking of methylcellulose and poly(acrylic acid). They observed a rise in cross-linkage strength and a decrease in swelling and solubility parameters, which resulted in a reduction in membrane adhesion [64]. The mucoadhesive property of hydrogels made from the condensation rising reactivity among poly(acrylic acid) was ascribed to an increase in the chain density of poly(acrylic acid) per unit size and sucrose as the cross-linkage density increased [65]. Mucoadhesive delivery systems that carry biologically active peptides to the top part of the small intestinal tract while preserving the biological absorption of the peptide has been developed using acrylates. In a typical test, Woods and Peppas set up a structure where ethyl acetate chains grafted onto wheat germ agglutinin were used to integrate acrylamide hydrogels. Wheat grain agglutinin increased the gastrointestinal resident duration of the delivery mechanism by reacting with certain carbohydrates present in the gastrointestinal mucosa [66]. Formulations based on mucoadhesive hydrogels are used in addition to drug targeting to improve the permeability of less permeable drugs. Mueller and Jacob produced small suspensions of non-water-soluble materials using hydrogels based on carbopol and chitin. Compared with nanosuspensions, mucoadhesive delivery methods have demonstrated enhanced drug bioavailability. The longer delivery method retention duration in the gastrointestinal tract has been suggested as the cause [67].

Thiolated Polymers (Thiomer)

Molecules containing open thiol groups added to the monomer framework prolong the monomer's adherence to the mucosal membrane and improve mucoadhesion even further by promoting the creation of robust covalent connections among the cysteine-rich mucin domains of the mucosal surfaces with transformed polymers. Regarding intestinal adhesion and medication delivery methods, sulfur content values of less than 300 μmol/g in polymers are considered optimal. However, thiolation at higher levels may enhance mucoadhesion [68]. Since the 1980s, when the concept of mucoadhesion was originally proposed, multiple measures have been taken to improve the sticky properties of polymeric materials. The use of linear poly(ethylene glycol) as a hydrogel adhesion enhancer is just one of those endeavors [69]. The creation of polymer–adhesion conjugates provide particular binding to epithelia [70]. The process of neutralizing ion monomers [71], and mucoadhesion over an extended hydrated period [72]. Nonetheless, the foundation of these systems is the creation of noncovalent connections, including ionic interactions [73], hydrogen bonds, and van der Waals forces [74]. Consequently, they only offer comparatively modest mucoadhesion, which is sometimes insufficient to ensure that a drug delivery system is localized to a specific target region. Thus, mucoadhesive polymers have frequently failed to demonstrate efficacy as medicinal glues [75]. Iminothiolane-chitosan, poly(acrylic acid), alginate-cysteine, chitosan-thioglycolic acid, chitosan-thioethylamidine, homocysteine-poly(acrylic acid), and poly(methacrylic acid) are a few examples of thiolated polymers sodium carboxymethylcellulose-cysteine and cysteine [76].

Lectin-based Polymers

In addition to being present in the kingdoms of plants and animals, lectins can engage inversely with specific sugar or carbohydrate residues [77]. It has been demonstrated that some lectins are immunologic and poisonous, and in those who are susceptible, frequent contact may result in systemic anaphylaxis [78]. Leptins have a unique cytoadhesive feature that is being investigated to create tailored delivery systems because of their affinity for sugar or carbohydrate residues. Lectins derived from legumes have been extensively studied as targeted delivery methods. Lens culinarius, Ulex europaeus I, soybean, and peanut lectins are among several lectins that show particular adherence to the mucosa [79]. Because Agglutinin from wheat germ attaches itself to the intestinal and alveolar epithelial cells and has the least immunogenic responses of all the lectins now on the market, it is becoming increasingly popular as a potential ingredient in oral and aerosol delivery systems [80].

CONCLUSION

A multifactorial gastrointestinal tract condition called PU is characterized by lesions or open wounds in the GI tract and duodenal mucus liners. Globally, most of the population has experienced PU. To obtain a sufficient amount of medication to its site of absorption—the stomach—and cure ulcers, the innovative oral mucoadhesive drug delivery system may stay in the stomach for extended periods of time and gradually discharge its medication substance, PUs, mucoadhesion mechanisms, mucosal polymers, benefits and drawbacks of the mucoadhesion drug delivery system, prospects for the future, and delivery issues were all successfully covered in the presentation. Ultimately, it may be claimed that the mucoadhesive medication delivery method is a novel and effective treatment for PUs with a wide range of applications.

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