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Review Article

A Review on SEDDS for Ulcerative Colitis

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ABSTRACT

Ulcerative colitis (UC) is a chronic idiopathic inflammatory bowel disease (IBD) characterised by continuous mucosal inflammation extending proximally from the rectum, affecting approximately 5 million people worldwide as of 2023, with rising incidence in developing regions including India. First described by Samuel Wilks in 1859, UC typically presents in the second or third decade of life with cramping abdominal pain and bloody diarrhoea, following a relapsing–remitting course. Its multifactorial pathogenesis involves genetic susceptibility (NOD2/CARD15, IL23R, IL10 variants), immune dysregulation through NF-κB, JAK/STAT, NLRP3, and IL-23/IL-17 signalling pathways, gut microbiome dysbiosis, and epithelial barrier dysfunction. A major pharmaceutical challenge is that over 40% of anti-inflammatory drug candidates exhibit poor aqueous solubility (BCS Class II/IV), severely limiting oral bioavailability. Self-Emulsifying Drug Delivery Systems (SEDDS) — isotropic mixtures of oils, surfactants, and co-solvents — overcome this by spontaneously forming fine oil-in-water emulsions in gastrointestinal fluids; advanced variants SMEDDS (100–250 nm) and SNEDDS (<100 nm) offer nanoscale droplets with superior absorption and thermodynamic stability. This review integrates current knowledge of UC pathogenesis with the therapeutic potential of SEDDS-based colon-targeted delivery as a strategy to improve drug bioavailability and clinical outcomes.

INTRODUCTION

Ulcerative colitis (UC) is an idiopathic inflammatory bowel disease (IBD), first described by *Samuel Wilks* in *1859* [1]. It is characterized by continuous inflammation of the colonic mucosa extending proximally from the rectum. UC is a chronic illness that typically manifests in the second or third decade of life with cramping abdominal pain and bloody diarrhoea [2]. Clinical manifestations during flares include bloody diarrhoea, disordered gut motility, systemic effects, and in severe cases, surgical complications [3].

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UC is classified by the extent of inflammation — limited to the rectum (proctitis), one-sided (left-sided colitis), or widespread (pancolitis) — and by symptom severity. Treatment decisions are guided by these parameters. The condition has no definitive cure short of colectomy, and most patients require ongoing long-term management [4,5].

The pathogenesis of UC involves a complex interplay of genetic susceptibility, environmental factors, immune dysregulation, and microbial imbalance [6]. Disruption of the intestinal barrier — including reduced mucus-producing cells and increased epithelial permeability — along with dysregulated T-cell responses, elevated chemokines and cytokines, and reduced microbial diversity are key pathogenic mechanisms [7-8].

Extra-intestinal manifestations are common, particularly affecting joints (arthritis, spondylitis, enthesopathy) in 10–25% of IBD patients, with activity often correlating with gut disease [9].

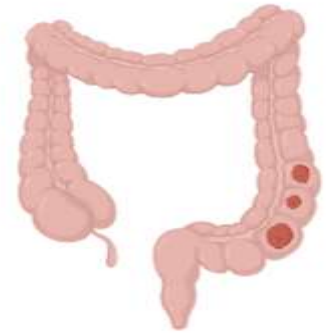


Figure 1. Colon-targeted drug delivery to the UC area.

2. SIGNS AND SYMPTOMS OF ULCERATIVE COLITIS

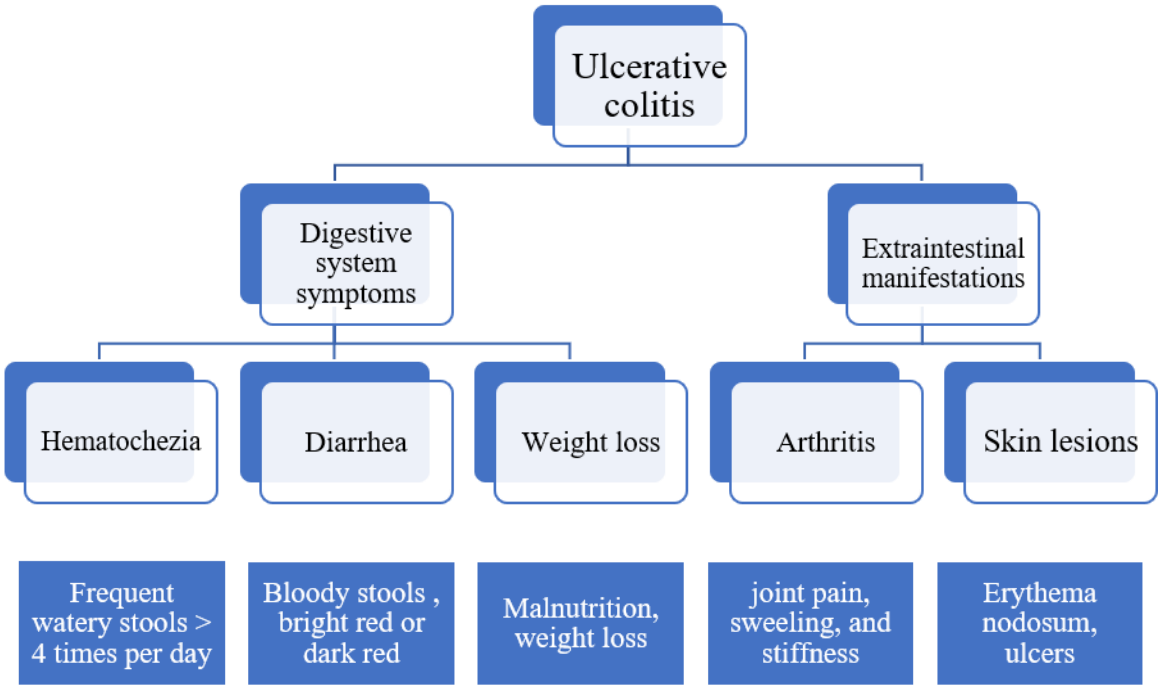
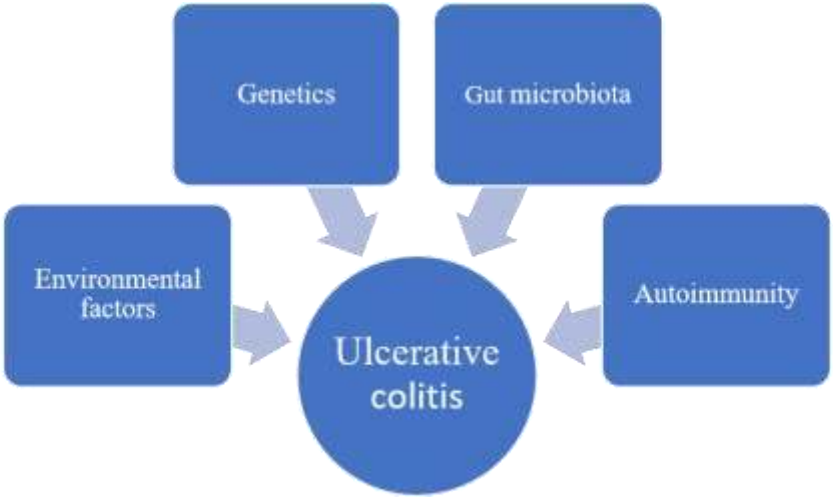


Figure 2: Clinical manifestations of UC

3. RISK FACTORS FOR UC:



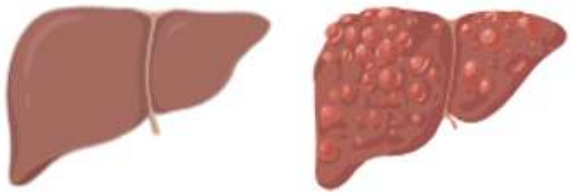
4. ULCERATIVE COLITIS:

Ulcerative Colitis is a kind of IBD (Inflammatory Bowel Disease). Immune systems reacting oddly to bacteria in the gut, again and again. These flare-ups strike the digestive tract without warning. Though both are long-term gut disorders, their patterns within the bowel lining aren't identical. One spreads continuously. The other might skip sections.

1. Crohn's disease

2. Ulcerative Colitis

The lining of the colon and the rectum become inflamed when someone has ulcerative colitis. Ulcerative colitis is an autoimmune disease in which the large intestine's inner lining becomes inflamed and ulcerated. Even though ulcerative colitis may appear at any age, it often shows up between young adulthood and middle years. The gut tissue swells up, sometimes forming raw sores where healing fails.



Normal liver **ulcerative liver**

Figure 3: Normal liver and ulcerative liver

5. TYPES OF ULCERATIVE COLITIS

There are different types or classifications of ulcerative colitis dependent on the extent and location of the inflammation within the colon. The types of ulcerative colitis include:

1. Ulcerative proctitis

2. Proctosigmoiditis

3. Left-sided Colitis

4. Acute Severe Ulcerative Colitis

1. **Ulcerative proctitis:** One out of three people with UC deal with ulcerative proctitis. Inflammation shows up just in the rectum - the last part of the big intestine. Usually, it sticks to about 15 centimetres or less. This form doesn't raise cancer chances.

- **Symptoms**: It includes pain in your rectum, bleeding in your rectum, and a sudden need to go to poop, rectal bleeding, urgency.

2. **Proctosigmoiditis**: In this type, inflammation spreads beyond the rectum and involves the sigmoid colon, which is the part of the colon right above the rectum.

- **Symptoms:** are abdominal cramps, bloody diarrhoea, and increased urgency to have bowel movements.

3. **Left-sided colitis:** Starting at the rectum and moving upward toward the splenic flexure - near the spleen - this form of UC may cause swelling. Inflammation happens here, especially in cases like proctosigmoiditis. That version targets the rectum along with the sigmoid colon, just above it.

- **Symptoms:** Loss of appetite, weight loss, pain in belly or bloody diarrhoea

4. **Extensive colitis (Pancolitis):** This type of UC affects entire colon. Inflammation starts at rectum and goes beyond splenic flexure.

- **Symptoms:**

1. Diarrhoea (may or may not be bloody)

2. Blood, mucus or pus in your stool

3. Severe belly cramping

4. Fatigue (extreme tiredness)

5. Sudden weight loss

5. **Acute Severe Ulcerative Colitis:** This is a severe and potentially life-threatening form of UC that affects the entire colon and develops rapidly. It results in dehydration, severe pain, and a significant risk of complications.

- **Symptoms:**

1. Diarrhoea (may or may not be bloody)

2. Increased bowel movements or episodes of diarrhoea (four or fewer episodes daily)

3. Urgent bowel movements (sudden need to poop)

4. Tenesmus (feeling like you have to poop but being unable to)

5. Mild abdominal (belly) cramping or tenderness

6. Nausea [11]

6. PATHOPHYSIOLOGY OF ULCERATIVE COLITIS

There are both genetic and environmental factors that contribute to IBD, although there is still much to learn about the pathophysiology of ulcerative colitis. Some factors have been repeatedly assessed and shown to be crucial to the development and course of the condition. The primary contributing factors include: a dysregulated immunological response, a compromised epithelial barrier, alterations in the gut microbiome, genetic vulnerability, and the exposome.

6.1 Genetic Factors

There is a significant genetic component to UC. Individuals with a family history of IBD, particularly first-degree relatives with UC, exhibit an increased risk of developing the condition. Multiple genes have been implicated in the susceptibility to UC, including the NOD2/CARD15 gene and the interleukin genes (IL23R and IL10). Although a family history of IBD is present in approximately 25% of UC patients, most cases are sporadic [12]. Identified genetic associations include HLA-DQA1 variants,

and genes involved in epithelial barrier integrity (CDH1, LAMB1), immune signalling (TNFRSF15, TNFRSF9, IL1R2, IL8RA/RB, IL7R), and others [13].

6.2 Dysregulation of the Immune System

One of the key elements in the pathogenesis of UC is an abnormal immune response. Normal gut bacteria are mistakenly perceived as foreign invaders, causing the intestinal lining to become chronically inflamed. Immune cells such as T-cells, B-cells, and macrophages infiltrate the gut lining and release pro-inflammatory cytokines. These cytokines are involved in the inflammatory process and further damage the intestinal tissue.

6.3 Intestinal Barrier Dysfunction

When ulcerative colitis is present, the gut wall weakens. Bacteria slip through cracks they normally cannot enter. Once inside, they trigger the immune system. Immune cells jump into action as a result, perpetuating the inflammatory cycle.

6.4 Gut Microbiome Dysbiosis

In healthy individuals, the gut microbiota is predominantly made up of four bacterial phyla — Firmicutes, Bacteroidetes, Proteobacteria, and Actinobacteria — accounting for roughly 97% of the total. Firmicutes and Bacteroidetes alone represent about 90% of the entire microbial community. These dominant phyla produce short-chain fatty acids (SCFAs), especially butyrate and propionate, through the fermentation of dietary components like non-digestible fibres. SCFAs are vital for preserving intestinal balance and have been shown to regulate immune homeostasis [14]. When ulcerative colitis shows up, that balance shifts — harmful microbes grow more while helpful kinds fade, causing further inflammation. Probiotics appear to benefit individuals with UC mainly by influencing key signalling pathways — such as NF-κB, MAPK, TLR, JAK/STAT, Wnt/β-catenin, and TGF-β — alongside their well-known effects on gut microbiota composition [15].

6.5 Key Inflammatory Signalling Pathways

NF-κB Pathway: The NF-κB transcription factor plays a central role in driving inflammation and immune responses. Evidence indicates that probiotics help dampen intestinal inflammation in UC by suppressing NF-κB pathway activity [16,17].

NLRP3 Inflammasome: Pathogen-associated molecular patterns (PAMPs) and danger-associated molecular patterns (DAMPs) activate the NLRP3 inflammasome, leading to Caspase-1 activation and subsequent release of pro-inflammatory cytokines IL-1β and IL-18, contributing to colonic mucosal inflammation [18].

PI3K/Akt Pathway: In addition to controlling and releasing inflammatory factors, the PI3K/Akt signalling pathway can also indirectly activate the transcription factor NF-κB via phosphorylated IKK. TLR4 signalling triggers the PI3K/Akt signalling pathway and induces downstream mTOR activation [19-20].

The overview of inflammatory signalling pathways involved in UC

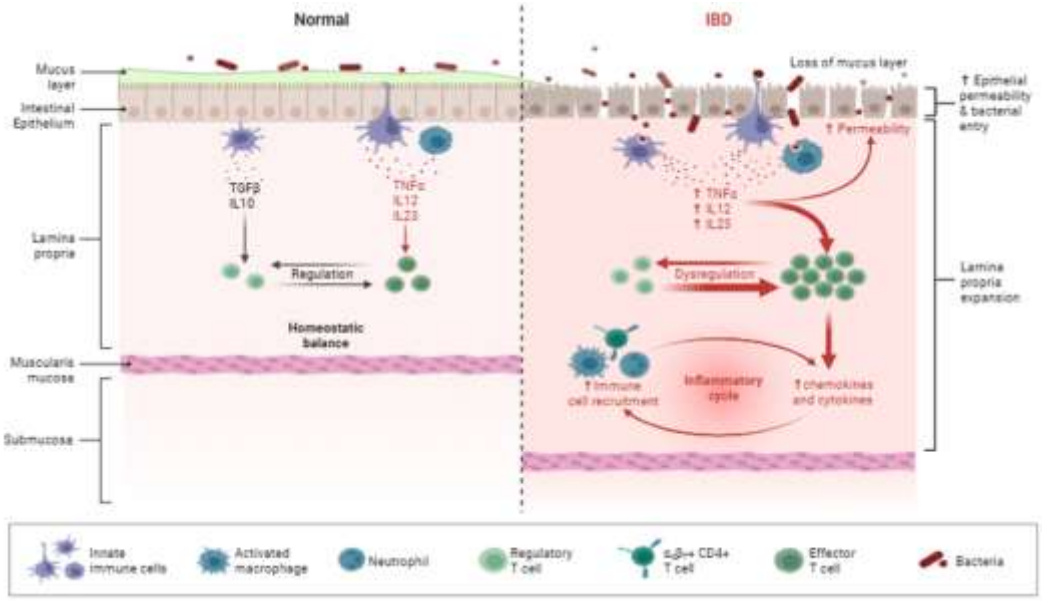


Figure 3: Pathophysiological mechanism of ulcerative colitis (UC).

a. The role of NF-κB pathway in Ulcerative Colitis

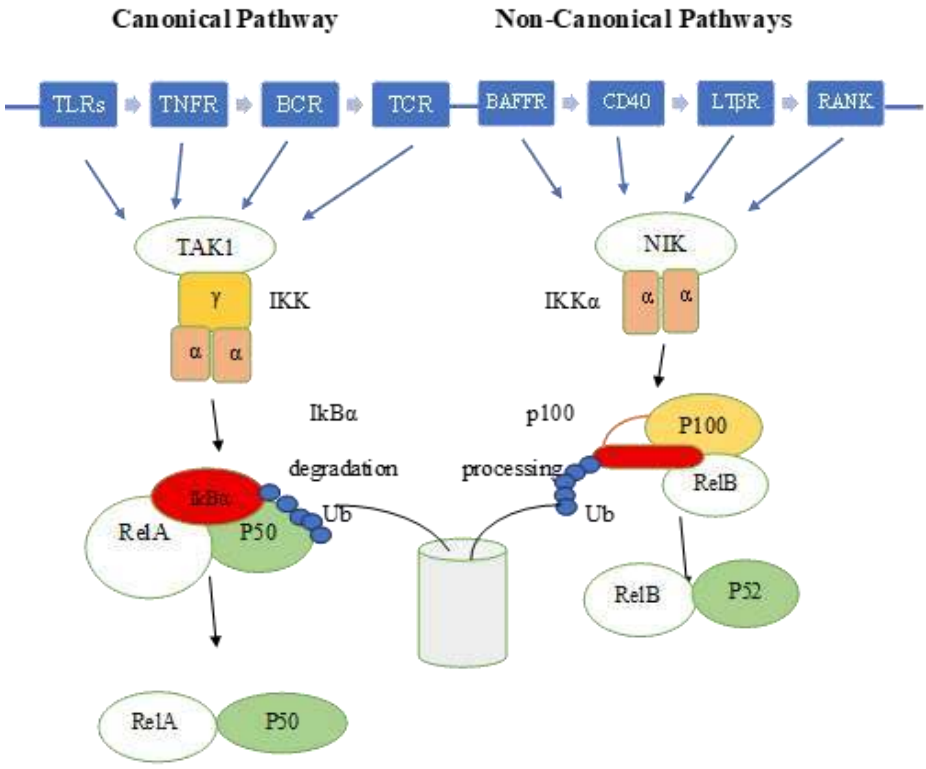
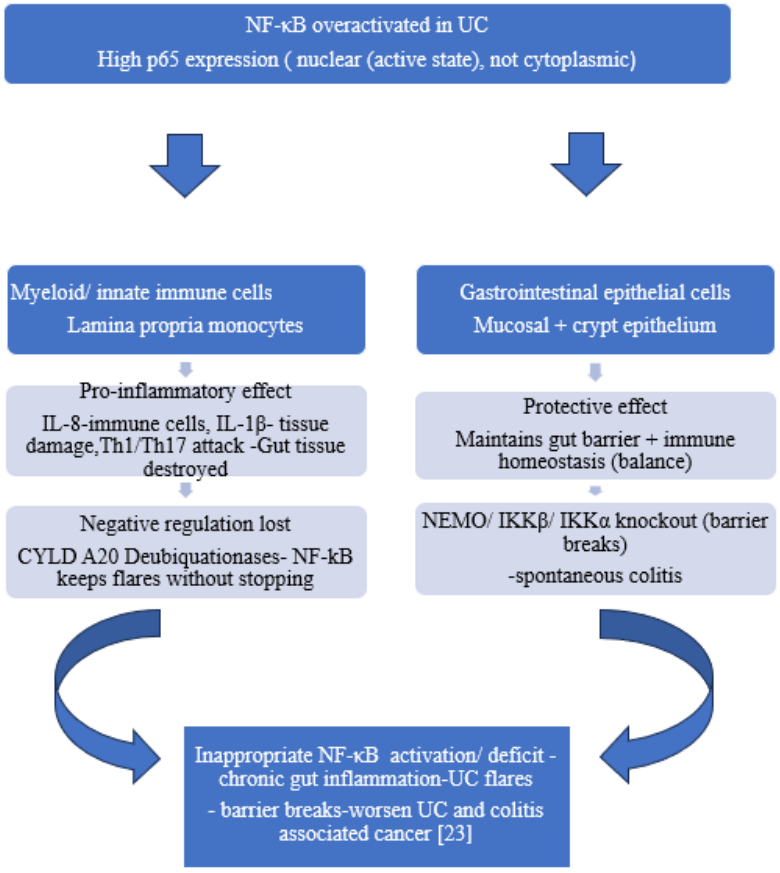


Figure 4: Canonical and non-canonical NF-κB signaling pathways [21]

b. NF-κB in Ulcerative colitis:



Curcumin- SEDDS – blocks IKKβ & p65 nuclear translocation entry

Budesonide-SEDDS- Induces IKBα-locks NF-kB

Quercetin-SEDDS- Blocks IKK+ IkB Stabilisation

Thymoquinone-SEDDS- Inhibits IkB Degradation & NF-kB DNA binding. SEDDS enhances its colonic retention and anti-inflammation potency. Blocks IkB Degradation

7. SELF EMULSIFYING AND SELF MICRO EMULSIFYING DRUG DELIVERY SYSTEM

7.1. Introduction

Oral administration remains the most widely preferred and convenient route of drug delivery. However, more than 40% of new drug candidates exhibit poor water solubility, which presents a significant challenge in oral drug delivery due to inadequate absorption, high intra- and inter-subject variability, and poor dose proportionality [24]. Various formulation strategies have been employed to overcome these limitations, including the use of surfactants, lipids, permeation enhancers, micronization, salt formation, cyclodextrins, nanoparticles, and solid dispersions [25].

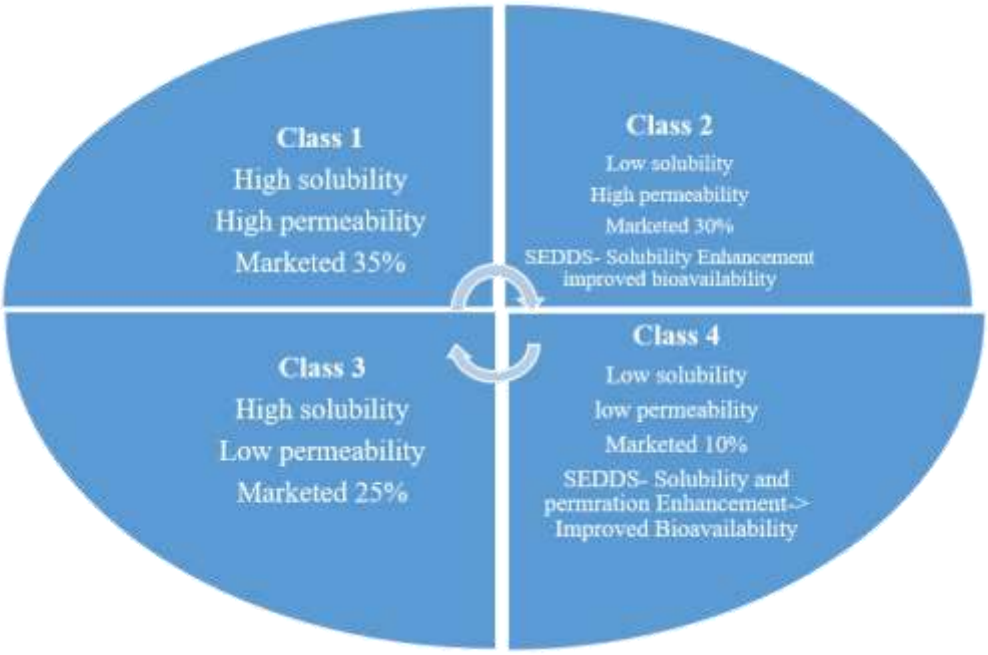


Figure 5. Biopharmaceutics classification system

Solubility and permeability are the two fundamental parameters governing drug absorption following oral administration. The Biopharmaceutics Classification System (BCS), developed by Amidon *et al.* in 1995, classifies drugs into four categories based on these two parameters, as illustrated in Figure 1 [26,27]. Among the four BCS classes, Class II and Class IV drugs are particularly problematic due to their poor water solubility and low oral bioavailability. Consequently, enhancing the dissolution profile of BCS Class II and IV drugs remains one of the most critical challenges in pharmaceutical research.

Since dissolution is considered the rate-limiting step in the absorption of poorly water-soluble drugs [28], achieving efficient self-emulsification through ultra-low oil-water interfacial tension can significantly enhance drug bioavailability and ensure a more consistent and predictable absorption profile from the gastrointestinal tract.

7.1.1. Self-emulsifying drug delivery systems

SEDDS are defined as isotropic mixtures of solid or liquid surfactants, natural or synthetic oils, and/or one or more hydrophilic solvents and co surfactants/solvents [29]. Once more, as previously mentioned, they dilute the drug in the system (aqueous media), such as GI fluids, with mild agitation, forming fine oil-in-water (o/w) emulsions (0.1-100µm) or microemulsions (10-300 nm), which spread easily into the GIT where the movement of the stomach and intestinal fluid provides agitation, creating a self-emulsified system [30].

7.1.2. Properties of SEDDS

- They have the ability to quickly self-emulsify in gastrointestinal fluids and produce a fine o/w emulsion when gently agitated by peristalsis and other gastrointestinal tract movements.
- They are able to successfully add drugs, whether hydrophilic or hydrophobic, to the combination of oil surfactants.
- They are applicable to both liquid and solid dose forms.

- Compared to standard dosage forms, they require a lesser dose of medication.

7.1.3. Advantages associated with SEDDS

- Drug protection from the GIT environment.
- Drug targeting that is specific to a certain GIT absorption window.
- Increased oral bioavailability.
- Consistent medication absorption profile.
- More effective management of drug delivery characteristics.
- The dosage form's versatility allows it to be utilized with both liquids and solids.
- Predictable treatment because food effects are less variable.
- Drug payloads are substantial.
- Sensitive pharmacological substances are protected.

Table: 1: It shows lipid formulation classification

Type	Composition	SEEDS Category	Behaviour in Aqueous media	Why it's used
Type I	Oil + drug only (no surfactant)	Conventional SEDDS	Reduced drug dissolution; increased drug precipitation post-digestion; slower lipolysis via bile salts & phospholipids	Simple formulation for lipophilic drugs; low cost
Type II	Oil + water-insoluble surfactant (HLB < 12)	Conventional SEDDS	Enhances dissolution & emulsification, but inadequate solubilization in vivo	Improves dissolution of poorly water-soluble drugs vs Type I
Type IIIa	Oil + co-surfactant + surfactant (HLB > 12)	SMEDDS	Forms stable o/w emulsion with minimal agitation; transitions readily to aqueous phase after steatolysis	Forms stable O/W emulsion; enhances oral absorption significantly
Type IIIb	Oil + co-surfactant + surfactant (HLB > 12)	SNEDDS	Similar to IIIa; high emulsification efficiency	Forms nanoemulsion; best for maximum drug absorption
Type IV	Surfactant ± co-surfactant (no oil)	—	Excellent dissolution properties; unstable in GI tract upon dilution	Excellent dissolution; used when oil-free formulation is needed

8. EXCIPIENTS USED IN SEDDS FORMULATION

Self-emulsifying drug delivery systems are made using a lot of excipients. Although co-surfactants can also be utilized, oil and surfactant are essential ingredients. The kind of dose form controls the use of various excipients in SEDDS. SEDDS of different medications have been formulated using a variety of oils, including natural, synthetic, and semi-synthetic. Table 1 lists a few instances of the oils utilized in the commercial products. Table 2 lists the oils utilized in the formulation of SEDDS with various medications.

I. Oils:

Table 2: Type of oils used in marketed SEDDS

Type of oil	Marketed Product	Drug
Corn oil	Depakene capsule	Valproic acid
Olive oil	Sandimmune oral solution	Cyclosporine
Sesame oil	Marinol soft gelatin capsule	Dronabinol
Soya bean oil	Accutane soft gelatin capsule	Isotretinoin

Peanut oil	Prometrium soft gelatin capsule	Progesterone
Bees wax	Vesanoid soft gelatin capsule	Tretinoin
Hydrogenated soya bean oil	Accutane soft gelatin capsule	Isotretinoin

Table 3: Type of oils used with different drugs in SEDDS

Oil	Drug
Soya bean oil	Probucol, Ibuprofen
Ethyl oleate	Vinpocetine
Oleic Acid	Puerarin
Maisine oil	Lercanidipine
Polyoxy castor oil	Simvastatin
Peanut oil	Griseofulvin

II. Surfactants

Table 4: Type of surfactants used in marketed SEDDS

Surfactant	Marketed Product	Drug
Span 80, Tween 80	Gengraf soft gelatin capsule	Cyclosporine
Tween 20	Targretin Hard gelatin Capsule	Bexarotene
Cremophor RH 40	BCNU self-emulsifying implant	Carmustine
D-alpha Tocopheryl Poly ethylene Glycol Carmustine Amprenavir 1000 Succinate (TPGS)	Agenerase Soft Gelatin capsule, Agenerase oral solution	Amprenavir
Labrafil M 1944 CS	Sandimmune oral solution.	Cyclosporine

Table 5: Type of surfactants used with different drugs in SEDDS

Surfactant	Drug
Tween 80	Ketoprofen, Carvedilol
TPGS	Tacrolimus
Labrafil M 1944 CS	Probucol
Tween 85	Indomethacin
Cremophor EL	Loratadine [32]

III. Co solvents/Co-surfactants:

Table 6: Type of Co surfactants used in marketed SEDDS

Co surfactants	Marketed preparation
Poly Ethylene Glycol	Targretin soft gelatin Capsule, Gengraf hard gelatin capsule, Agenerase soft gelatin
Glycerine	Sandimmune soft gelatin capsule
Propylene glycol	Neoral soft gelatin, Neoral oral solution, Gengraf hard gelatin, Lamprene soft gelatin capsule.
Ethanol	Neoral Soft gelatin & Neoral oral, sandimmune soft gelatin & oral sol, gengraf hard gelatin capsule

IV. Drugs

Examples: Naproxen, Carbamazepine, Danazol, Ketoconazole, Nifedipine, vitamin E, simvastatin, and mefanimic acid Finasteride

9. SOLID SELF EMULSIFYING DRUG DELIVERY SYSTEM (S--SEDDS)

Although SEDDS are available in both liquid and solid dose forms, solid SEDDS are typically favoured over liquid SEDDS due to their superior stability and simplicity of handling and transportation. A variety of different solid SEDDS, including pellets, microspheres, tablets, beads, implants, and suppositories, have recently been developed.

Table 7. Comparison of SEDDS, SMEDDS, and SNEDDS [33,34].

Characteristics	SEDDS	SMEDDS	SNEDDS
Mean droplet size	250 nm–5 µm	100–250 nm	<100 nm
Appearance	Turbid/Cloudy	Clear to translucent	Optically clear
Solubilizing capacity	High	High	High
Stability	Thermodynamically unstable	Thermodynamically stable	Kinetically stable
Bioavailability	Moderate	Enhanced	Superior

Oil Types	Long-chai–n triglycerides (e.g., soybean oil, olive oil)	Medium-chain triglycerides (e.g., Labrafac®, Captex® 355) (MC triglycerides)	Medium- and short-chain triglycerides (e.g., Capmul®, Miglyol®) (caprylic and capric triglycerides)
HLB of surfactants	<10	10–12	>12
Co-surfactants	Not essential	Short-chain alcohols (e.g., propylene glycol)	Polyethylene glycol (PEG), Transcutol®

Table 8: Marketed Preparations of SEDDS.

Brand Name	Generic Drugs	Dosage Forms	Manufacturer
Convulex	Valproic acid	Soft gelatin capsule	Gerot Pharmazeutika
Norvir	Ritonavir	Soft gelatin capsule	Abbvie, Abbot
Fortovase	Saquinavir	Soft gelatin capsule	Hoffman- Roche
Neoral	Cyclosporine	Soft gelatin capsule	Novartis
Agenerase	Amprenavir	Soft gelatin capsule	GSK
Lipirex	Fenofibrate	Hard gelatin capsule	Sanofi- Aventis
Solufen	Ibuprofen	Hard gelatin capsule	Sanofi- Aventis
Depakene	Valproic acid	Soft gelatin capsule	Abbvie
Accutane	Isotretinoin	Soft gelatin capsule	Hoffman Le Roche
Prometruim	Progesterone	Soft gelatin capsule	Virtus

CONCLUSION

Ulcerative colitis remains one of the most challenging chronic inflammatory diseases of the modern era. With a global prevalence of approximately 5 million cases and a steadily rising incidence in previously low-burden regions — particularly in South Asia, Africa, and South America — UC represents a growing public health concern. In India, rapid urbanisation, dietary westernisation, and changing hygiene standards are contributing to an accelerating disease burden, especially in northern regions such as Punjab, where community-based studies have documented prevalence rates comparable to some Western nations. The relapsing-remitting nature of the disease, combined with its long-term risk of colorectal cancer and need for colectomy in a subset of patients, underscores the urgent need for effective and patient-friendly therapeutic strategies.

The pathogenesis of UC is complex and multidimensional, involving a dynamic interplay between genetic predisposition, dysregulated immune responses, gut microbiome imbalance, and compromised intestinal barrier function. Key inflammatory signalling pathways — including NF-κB, NLRP3 inflammasome, PI3K/Akt, JAK/STAT, and the IL-23/IL-17 axis — drive chronic mucosal inflammation and tissue damage. Dysbiosis, characterised by loss of short-chain fatty acid-producing bacteria such as Firmicutes and Bacteroidetes, further amplifies the inflammatory cascade. Understanding these mechanisms has not only expanded our knowledge of disease biology but has also identified novel therapeutic targets. Despite advances in biologics and small molecules, a significant number of patients remain refractory to current treatments, highlighting the continued need for innovative drug delivery approaches that can enhance therapeutic efficacy at the site of colonic inflammation.

A central pharmaceutical barrier in UC therapy is the poor aqueous solubility of many candidate drugs. Over 40% of new anti-inflammatory

compounds fall into BCS Class II or IV, meaning their therapeutic potential is undermined by inadequate dissolution and absorption in the gastrointestinal tract. SEDDS directly address this limitation by forming isotropic, thermodynamically stable mixtures of oils, surfactants, and co-solvents that spontaneously self-emulsify upon contact with gastrointestinal fluids under gentle agitation. The resulting fine oil-in-water emulsions present drug molecules in a pre-dissolved or nano-dispersed state, dramatically increasing the surface area available for absorption and bypassing the rate-limiting dissolution step. The enhanced wettability, improved membrane permeation, and protection from enzymatic degradation collectively contribute to a significantly elevated oral bioavailability compared to conventional solid dosage forms. Drugs with a log P above 5 show the greatest benefit within SEDDS formulations, as they partition effectively into the lipid phase and are subsequently incorporated into chylomicrons for lymphatic transport, further circumventing hepatic first-pass metabolism.

The successful commercialisation of SEDDS-based products including Neoral (cyclosporine), Norvir (ritonavir), and Accutane (isotretinoin) provides clinical proof of concept and a strong foundation for developing SEDDS formulations tailored specifically for UC-targeted therapy.

Future research should focus on comprehensive in vivo evaluation of SEDDS-loaded colon-targeted formulations for key UC drugs such as mesalazine, budesonide, and curcumin; rigorous stability testing under simulated gastrointestinal conditions; and scale-up feasibility for industrial manufacturing. Advances in nanotechnology, surface functionalisation, and computational formulation design will further accelerate the development of next-generation SEDDS. In summary, SEDDS-based colon-targeted delivery systems hold substantial promise as a transformative therapeutic strategy for UC, and sustained investment in this field is essential to translate laboratory advances into improved patient outcomes.

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