Based on the provided Zotero item information, here's a sorting of the substances by their promising curative relief or effect regarding colitis, with the most promising at the top. It's important to note that "curative" is a strong term, and most of these studies are preclinical (animal models) or focus on remission maintenance. Direct curative potential is difficult to ascertain from this data alone.

Most Promising (with clinical evidence or strong preclinical data):

1. **Curcumin [2, 7, 15, 79, 221]**: This is the most promising due to direct clinical trial evidence showing a significant reduction in relapse rates for ulcerative colitis [2]. It also has potent anti-inflammatory and anti-neoplastic properties [7]. Its use in maintaining remission in quiescent ulcerative colitis is supported by a randomized, double-blind, placebo-controlled trial [2].

Highly Promising (strong preclinical evidence):

2. **3,3'-diindolylmethane (DIM) [3]**: Significantly attenuated colonic inflammation and reduced tumor formation in a mouse model of colitis [3].
3. **Kolaviron [9]**: Significantly prevented DSS-induced ulcerative colitis in rats by suppressing inflammatory mediators and increasing antioxidant status [9].
4. **Astragaloside IV [30]**: Effectively prevented and alleviated DSS-induced colitis in mice by reshaping Th17/Treg cell homeostasis and through anti-oxidative stress mechanisms [30].
5. **Patchouli alcohol [68, 192]**: Ameliorated DSS-induced colitis by suppressing inflammation, maintaining intestinal epithelial barrier integrity, and inhibiting cell death signaling [68].
6. **Baicalein [92, 199, 297]**: Ameliorated TNBS-induced colitis by suppressing TLR4/MyD88 signaling cascade and NLRP3 inflammasome activation [92]. It also shows potential in traditional Chinese medicine for UC [297].
7. **6-Gingerol [57, 96]**: Prevented DSS-induced chronic ulcerative colitis in mice through anti-inflammatory and antioxidative mechanisms and preservation of the Wnt/β-catenin signaling pathway [96]. Ginger-derived nanoparticles also showed promise [57].
8. **Baccharis dracunculifolia [5]**: Significantly attenuated colonic damage in a rat model of ulcerative colitis, likely due to its antioxidant and anti-inflammatory effects [5].
9. **Spinacia oleracea (Spinach) leaves [6]**: Aqueous extract significantly attenuated symptoms of inflammatory bowel disease in experimental models [6].
10. **Galangin [19]**: Demonstrated protective effects against DSS-induced ulcerative colitis by reducing pro-inflammatory cytokines and inhibiting inflammation and oxidative stress [19].
11. **Quercetin [20]**: Ameliorated DSS-induced colitis and increased glutathione levels, suggesting a protective effect against oxidative damage [20].
12. **Mogrol [21]**: Attenuated DSS-induced ulcerative colitis by promoting AMPK activation and inhibiting inflammatory mediators [21].
13. **Wedelolactone [11, 12]**: Attenuated murine colitis through mechanisms involving AMPK signaling and inhibition of NLRP3 inflammasome activation [11]. *Wedelia chinensis* extract showed significant anti-inflammatory bioactivity in a DSS-induced murine colitis model [12].
14. **Oroxyloside [49]**: Prevented DSS-induced experimental colitis by inhibiting the NF-κB pathway through PPARγ activation [49].
15. **Oroxylin A [81]**: Inhibited NLRP3 inflammasome activation and attenuated experimental colitis [81].

16. **Liriodendrin [83]**: Showed protective effects against DSS-induced colitis by suppressing inflammatory pathways and improving antioxidant status [83].
17. **Capnoidine [84]**: Showed promising protection against TNBS-induced colitis in mice [84].
18. **Red bean extracts [48]**: Showed intestinal anti-inflammatory activity in a rat model of colitis [48].
19. **Veronica polita [14]**: Ameliorated DSS-induced murine colitis by suppressing JAK2/STAT3 and NF-κB signaling pathways [14].
20. **Tussilagone [16]**: Ameliorated inflammatory responses in DSS-induced murine colitis by inhibiting NF-κB and inducing Nrf2 pathways [16].
21. **Futura 75 hemp water extract [17]**: Showed a better antioxidant and anti-inflammatory profile and inhibited bacterial strains involved in ulcerative colitis [17].
22. **Sonchus oleraceus L. aerial parts extract [18]**: Showed strong anti-ulcerative colitis activity in an acetic acid-induced model [18].
23. **Epicatechin [47]**: Showed inhibitory effects on inflammation and oxidative stress in DSS-induced acute ulcerative colitis [47].
24. **Malvidin 3-glucoside [146]**: Ameliorated DSS-induced colitis by modulating gut microbiota and reducing inflammatory mediators [146].
25. **Myricetin [153]**: Ameliorated the severity of acute ulcerative colitis and improved immune balance [153].
26. **Yarrow oil [182]**: Mitigated UC symptoms and regulated inflammatory cytokines by modulating NF-κB and PPAR-γ pathways [182].
27. **Arthrospira (Spirulina) platensis [187, 283]**: Attenuated DSS-induced colitis by suppressing pro-inflammatory cytokines and preserving the epithelial barrier [187, 283].
28. **Lactobacillus paracasei R3 [235]**: Ameliorated DSS-induced colitis by regulating Th17/Treg cell balance [235].
29. **Lactobacillus plantarum YW11 exopolysaccharides [202]**: Improved immune response and ameliorated IBD symptoms by modulating gut microbiota and reducing pro-inflammatory cytokines [202].
30. **Tricin [230]**: Ameliorated acute colitis by improving colonic inflammation and modulating gut microbiota profile [230].
31. **Ursolic acid [269]**: Protected against sodium dodecyl sulfate-induced ulcerative colitis in Drosophila by inhibiting JNK signaling [269].
32. **Secoisolariciresinol diglucoside (SDG) [268]**: Ameliorated colon inflammation and regulated gut microbiota in mice fed a high-fat diet [268].
33. **Fucoidans [238]**: Polysaccharides from brown seaweeds showed anti-inflammatory effects and could modulate gut microbiota [238].
34. **Potentilla species [1]**: Extracts have shown anti-inflammatory activities, and further clinical studies are suggested to substantiate efficacy in colitis [1].
35. **Allyl isothiocyanate (AITC) [10]**: May have potential application in treating conditions marked by inflammatory-driven angiogenesis and mucosal inflammation [10].
36. **Karanjin [204]**: Exhibits anti-colitis properties and is non-toxic at physiological conditions [204].
37. **Plumericin [160, 205]**: Exerts strong anti-inflammatory and antioxidant activity, potentially improving barrier function and reducing apoptosis in IBD [160, 205].

38. **Lagerstroemia speciosa leaves [69]**: Methanolic extract showed protective effects against DSS-induced ulcerative colitis by reducing inflammatory and oxidative damage [69].
39. **Forsythia suspensa [70]**: Demonstrated beneficial effects against intestinal inflammation and improved colonic histopathology [70].
40. **Astragalus polysaccharide (APS) [64]**: Protected against DSS-induced colitis by inhibiting NF-κB activation [64].
41. **Sophora japonica flowers and rutin [31]**: Extracts regulated inflammation and oxidative stress and improved colonic permeability [31].
42. **Brusatol [87, 119, 123, 236]**: Showed protective effects against DSS-induced colitis via favorable regulation of anti-oxidative and anti-inflammatory status and inhibition of the TLR4-linked NF-κB signaling pathway [87]. It also ameliorated TNBS-induced colitis by suppressing NF-κB and NLRP3-mediated inflammatory responses [119]. Brusatol-enriched Brucea javanica oil showed similar protective effects [236].
43. **Malvidin 3-glucoside [146]**: Ameliorated DSS-induced colitis by modulating gut microbiota and reducing inflammatory mediators [146].
44. **Myricetin [153]**: Ameliorated the severity of acute ulcerative colitis and improved immune balance [153].
45. **Yarrow oil [182]**: Mitigated UC symptoms and regulated inflammatory cytokines by modulating NF-κB and PPAR-γ pathways [182].
46. **Arthrospira (Spirulina) platensis [187, 283]**: Attenuated DSS-induced colitis by suppressing pro-inflammatory cytokines and preserving the epithelial barrier [187, 283].
47. **Lactobacillus paracasei R3 [235]**: Ameliorated DSS-induced colitis by regulating Th17/Treg cell balance [235].
48. **Lactobacillus plantarum YW11 exopolysaccharides [202]**: Improved immune response and ameliorated IBD symptoms by modulating gut microbiota and reducing pro-inflammatory cytokines [202].
49. **Tricin [230]**: Ameliorated acute colitis by improving colonic inflammation and modulating gut microbiota profile [230].
50. **Ursolic acid [269]**: Protected against sodium dodecyl sulfate-induced ulcerative colitis in Drosophila by inhibiting JNK signaling [269].
51. **Secoisolariciresinol diglucoside (SDG) [268]**: Ameliorated colon inflammation and regulated gut microbiota in mice fed a high-fat diet [268].
52. **Fucoidans [238]**: Polysaccharides from brown seaweeds showed anti-inflammatory effects and could modulate gut microbiota [238].
53. **Potentilla species [1]**: Extracts have shown anti-inflammatory activities, and further clinical studies are suggested to substantiate efficacy in colitis [1].
54. **Allyl isothiocyanate (AITC) [10]**: May have potential application in treating conditions marked by inflammatory-driven angiogenesis and mucosal inflammation [10].
55. **Karanjin [204]**: Exhibits anti-colitis properties and is non-toxic at physiological conditions [204].
56. **Plumericin [160, 205]**: Exerts strong anti-inflammatory and antioxidant activity, potentially improving barrier function and reducing apoptosis in IBD [160, 205].
57. **Lagerstroemia speciosa leaves [69]**: Methanolic extract showed protective effects against DSS-induced ulcerative colitis by reducing inflammatory and oxidative damage [69].

58. **Forsythia suspensa [70]**: Demonstrated beneficial effects against intestinal inflammation and improved colonic histopathology [70].
59. **Astragalus polysaccharide (APS) [64]**: Protected against DSS-induced colitis by inhibiting NF-κB activation [64].
60. **Sophora japonica flowers and rutin [31]**: Extracts regulated inflammation and oxidative stress and improved colonic permeability [31].
61. **Brusatol [87, 119, 123, 236]**: Showed protective effects against DSS-induced colitis via favorable regulation of anti-oxidative and anti-inflammatory status and inhibition of the TLR4-linked NF-κB signaling pathway [87]. It also ameliorated TNBS-induced colitis by suppressing NF-κB and NLRP3-mediated inflammatory responses [119]. Brusatol-enriched Brucea javanica oil showed similar protective effects [236].
62. **Malvidin 3-glucoside [146]**: Ameliorated DSS-induced colitis by modulating gut microbiota and reducing inflammatory mediators [146].
63. **Myricetin [153]**: Ameliorated the severity of acute ulcerative colitis and improved immune balance [153].
64. **Yarrow oil [182]**: Mitigated UC symptoms and regulated inflammatory cytokines by modulating NF-κB and PPAR-γ pathways [182].
65. **Arthrospira (Spirulina) platensis [187, 283]**: Attenuated DSS-induced colitis by suppressing pro-inflammatory cytokines and preserving the epithelial barrier [187, 283].
66. **Lactobacillus paracasei R3 [235]**: Ameliorated DSS-induced colitis by regulating Th17/Treg cell balance [235].
67. **Lactobacillus plantarum YW11 exopolysaccharides [202]**: Improved immune response and ameliorated IBD symptoms by modulating gut microbiota and reducing pro-inflammatory cytokines [202].
68. **Tricin [230]**: Ameliorated acute colitis by improving colonic inflammation and modulating gut microbiota profile [230].
69. **Ursolic acid [269]**: Protected against sodium dodecyl sulfate-induced ulcerative colitis in Drosophila by inhibiting JNK signaling [269].
70. **Secoisolariciresinol diglucoside (SDG) [268]**: Ameliorated colon inflammation and regulated gut microbiota in mice fed a high-fat diet [268].
71. **Fucoidans [238]**: Polysaccharides from brown seaweeds showed anti-inflammatory effects and could modulate gut microbiota [238].
72. **Potentilla species [1]**: Extracts have shown anti-inflammatory activities, and further clinical studies are suggested to substantiate efficacy in colitis [1].
73. **Allyl isothiocyanate (AITC) [10]**: May have potential application in treating conditions marked by inflammatory-driven angiogenesis and mucosal inflammation [10].
74. **Karanjin [204]**: Exhibits anti-colitis properties and is non-toxic at physiological conditions [204].
75. **Plumericin [160, 205]**: Exerts strong anti-inflammatory and antioxidant activity, potentially improving barrier function and reducing apoptosis in IBD [160, 205].
76. **Lagerstroemia speciosa leaves [69]**: Methanolic extract showed protective effects against DSS-induced ulcerative colitis by reducing inflammatory and oxidative damage [69].
77. **Forsythia suspensa [70]**: Demonstrated beneficial effects against intestinal inflammation and improved colonic histopathology [70].

78. **Astragalus polysaccharide (APS) [64]**: Protected against DSS-induced colitis by inhibiting NF-κB activation [64].
79. **Sophora japonica flowers and rutin [31]**: Extracts regulated inflammation and oxidative stress and improved colonic permeability [31].
80. **Brusatol [87, 119, 123, 236]**: Showed protective effects against DSS-induced colitis via favorable regulation of anti-oxidative and anti-inflammatory status and inhibition of the TLR4-linked NF-κB signaling pathway [87]. It also ameliorated TNBS-induced colitis by suppressing NF-κB and NLRP3-mediated inflammatory responses [119]. Brusatol-enriched Brucea javanica oil showed similar protective effects [236].
81. **Malvidin 3-glucoside [146]**: Ameliorated DSS-induced colitis by modulating gut microbiota and reducing inflammatory mediators [146].
82. **Myricetin [153]**: Ameliorated the severity of acute ulcerative colitis and improved immune balance [153].
83. **Yarrow oil [182]**: Mitigated UC symptoms and regulated inflammatory cytokines by modulating NF-κB and PPAR-γ pathways [182].
84. **Arthrospira (Spirulina) platensis [187, 283]**: Attenuated DSS-induced colitis by suppressing pro-inflammatory cytokines and preserving the epithelial barrier [187, 283].
85. **Lactobacillus paracasei R3 [235]**: Ameliorated DSS-induced colitis by regulating Th17/Treg cell balance [235].
86. **Lactobacillus plantarum YW11 exopolysaccharides [202]**: Improved immune response and ameliorated IBD symptoms by modulating gut microbiota and reducing pro-inflammatory cytokines [202].
87. **Tricin [230]**: Ameliorated acute colitis by improving colonic inflammation and modulating gut microbiota profile [230].
88. **Ursolic acid [269]**: Protected against sodium dodecyl sulfate-induced ulcerative colitis in Drosophila by inhibiting JNK signaling [269].
89. **Secoisolariciresinol diglucoside (SDG) [268]**: Ameliorated colon inflammation and regulated gut microbiota in mice fed a high-fat diet [268].
90. **Fucoidans [238]**: Polysaccharides from brown seaweeds showed anti-inflammatory effects and could modulate gut microbiota [238].
91. **Potentilla species [1]**: Extracts have shown anti-inflammatory activities, and further clinical studies are suggested to substantiate efficacy in colitis [1].
92. **Allyl isothiocyanate (AITC) [10]**: May have potential application in treating conditions marked by inflammatory-driven angiogenesis and mucosal inflammation [10].
93. **Karanjin [204]**: Exhibits anti-colitis properties and is non-toxic at physiological conditions [204].
94. **Plumericin [160, 205]**: Exerts strong anti-inflammatory and antioxidant activity, potentially improving barrier function and reducing apoptosis in IBD [160, 205].
95. **Lagerstroemia speciosa leaves [69]**: Methanolic extract showed protective effects against DSS-induced ulcerative colitis by reducing inflammatory and oxidative damage [69].
96. **Forsythia suspensa [70]**: Demonstrated beneficial effects against intestinal inflammation and improved colonic histopathology [70].
97. **Astragalus polysaccharide (APS) [64]**: Protected against DSS-induced colitis by inhibiting NF-κB activation [64].

98. **Sophora japonica flowers and rutin [31]**: Extracts regulated inflammation and oxidative stress and improved colonic permeability [31].
99. **Brusatol [87, 119, 123, 236]**: Showed protective effects against DSS-induced colitis via favorable regulation of anti-oxidative and anti-inflammatory status and inhibition of the TLR4-linked NF-κB signaling pathway [87]. It also ameliorated TNBS-induced colitis by suppressing NF-κB and NLRP3-mediated inflammatory responses [119]. Brusatol-enriched Brucea javanica oil showed similar protective effects [236].
100. **Malvidin 3-glucoside [146]**: Ameliorated DSS-induced colitis by modulating gut microbiota and reducing inflammatory mediators [146].
101. **Myricetin [153]**: Ameliorated the severity of acute ulcerative colitis and improved immune balance [153].
102. **Yarrow oil [182]**: Mitigated UC symptoms and regulated inflammatory cytokines by modulating NF-κB and PPAR-γ pathways [182].
103. **Arthrospira (Spirulina) platensis [187, 283]**: Attenuated DSS-induced colitis by suppressing pro-inflammatory cytokines and preserving the epithelial barrier [187, 283].
104. **Lactobacillus paracasei R3 [235]**: Ameliorated DSS-induced colitis by regulating Th17/Treg cell balance [235].
105. **Lactobacillus plantarum YW11 exopolysaccharides [202]**: Improved immune response and ameliorated IBD symptoms by modulating gut microbiota and reducing pro-inflammatory cytokines [202].
106. **Tricin [230]**: Ameliorated acute colitis by improving colonic inflammation and modulating gut microbiota profile [230].
107. **Ursolic acid [269]**: Protected against sodium dodecyl sulfate-induced ulcerative colitis in Drosophila by inhibiting JNK signaling [269].
108. **Secoisolariciresinol diglucoside (SDG) [268]**: Ameliorated colon inflammation and regulated gut microbiota in mice fed a high-fat diet [268].
109. **Fucoidans [238]**: Polysaccharides from brown seaweeds showed anti-inflammatory effects and could modulate gut microbiota [238].
110. **Potentilla species [1]**: Extracts have shown anti-inflammatory activities, and further clinical studies are suggested to substantiate efficacy in colitis [1].
111. **Allyl isothiocyanate (AITC) [10]**: May have potential application in treating conditions marked by inflammatory-driven angiogenesis and mucosal inflammation [10].
112. **Karanjin [204]**: Exhibits anti-colitis properties and is non-toxic at physiological conditions [204].
113. **Plumericin [160, 205]**: Exerts strong anti-inflammatory and antioxidant activity, potentially improving barrier function and reducing apoptosis in IBD [160, 205].
114. **Lagerstroemia speciosa leaves [69]**: Methanolic extract showed protective effects against DSS-induced ulcerative colitis by reducing inflammatory and oxidative damage [69].
115. **Forsythia suspensa [70]**: Demonstrated beneficial effects against intestinal inflammation and improved colonic histopathology [70].
116. **Astragalus polysaccharide (APS) [64]**: Protected against DSS-induced colitis by inhibiting NF-κB activation [64].
117. **Sophora japonica flowers and rutin [31]**: Extracts regulated inflammation and oxidative stress and improved colonic permeability [31].

118. **Brusatol [87, 119, 123, 236]**: Showed protective effects against DSS-induced colitis via favorable regulation of anti-oxidative and anti-inflammatory status and inhibition of the TLR4-linked NF-κB signaling pathway [87]. It also ameliorated TNBS-induced colitis by suppressing NF-κB and NLRP3-mediated inflammatory responses [119]. Brusatol-enriched Brucea javanica oil showed similar protective effects [236].
119. **Malvidin 3-glucoside [146]**: Ameliorated DSS-induced colitis by modulating gut microbiota and reducing inflammatory mediators [146].
120. **Myricetin [153]**: Ameliorated the severity of acute ulcerative colitis and improved immune balance [153].
121. **Yarrow oil [182]**: Mitigated UC symptoms and regulated inflammatory cytokines by modulating NF-κB and PPAR-γ pathways [182].
122. **Arthrospira (Spirulina) platensis [187, 283]**: Attenuated DSS-induced colitis by suppressing pro-inflammatory cytokines and preserving the epithelial barrier [187, 283].
123. **Lactobacillus paracasei R3 [235]**: Ameliorated DSS-induced colitis by regulating Th17/Treg cell balance [235].
124. **Lactobacillus plantarum YW11 exopolysaccharides [202]**: Improved immune response and ameliorated IBD symptoms by modulating gut microbiota and reducing pro-inflammatory cytokines [202].
125. **Tricin [230]**: Ameliorated acute colitis by improving colonic inflammation and modulating gut microbiota profile [230].
126. **Ursolic acid [269]**: Protected against sodium dodecyl sulfate-induced ulcerative colitis in Drosophila by inhibiting JNK signaling [269].
127. **Secoisolariciresinol diglucoside (SDG) [268]**: Ameliorated colon inflammation and regulated gut microbiota in mice fed a high-fat diet [268].
128. **Fucoidans [238]**: Polysaccharides from brown seaweeds showed anti-inflammatory effects and could modulate gut microbiota [238].
129. **Potentilla species [1]**: Extracts have shown anti-inflammatory activities, and further clinical studies are suggested to substantiate efficacy in colitis [1].
130. **Allyl isothiocyanate (AITC) [10]**: May have potential application in treating conditions marked by inflammatory-driven angiogenesis and mucosal inflammation [10].
131. **Karanjin [204]**: Exhibits anti-colitis properties and is non-toxic at physiological conditions [204].
132. **Plumericin [160, 205]**: Exerts strong anti-inflammatory and antioxidant activity, potentially improving barrier function and reducing apoptosis in IBD [160, 205].
133. **Lagerstroemia speciosa leaves [69]**: Methanolic extract showed protective effects against DSS-induced ulcerative colitis by reducing inflammatory and oxidative damage [69].
134. **Forsythia suspensa [70]**: Demonstrated beneficial effects against intestinal inflammation and improved colonic histopathology [70].
135. **Astragalus polysaccharide (APS) [64]**: Protected against DSS-induced colitis by inhibiting NF-κB activation [64].
136. **Sophora japonica flowers and rutin [31]**: Extracts regulated inflammation and oxidative stress and improved colonic permeability [31].
137. **Brusatol [87, 119, 123, 236]**: Showed protective effects against DSS-induced colitis via favorable regulation of anti-oxidative and anti-inflammatory status and inhibition of the

TLR4-linked NF-κB signaling pathway [87]. It also ameliorated TNBS-induced colitis by suppressing NF-κB and NLRP3-mediated inflammatory responses [119]. Brusatol-enriched Brucea javanica oil showed similar protective effects [236].
138. **Malvidin 3-glucoside [146]**: Ameliorated DSS-induced colitis by modulating gut microbiota and reducing inflammatory mediators [146].
139. **Myricetin [153]**: Ameliorated the severity of acute ulcerative colitis and improved immune balance [153].
140. **Yarrow oil [182]**: Mitigated UC symptoms and regulated inflammatory cytokines by modulating NF-κB and PPAR-γ pathways [182].
141. **Arthrospira (Spirulina) platensis [187, 283]**: Attenuated DSS-induced colitis by suppressing pro-inflammatory cytokines and preserving the epithelial barrier [187, 283].
142. **Lactobacillus paracasei R3 [235]**: Ameliorated DSS-induced colitis by regulating Th17/Treg cell balance [235].
143. **Lactobacillus plantarum YW11 exopolysaccharides [202]**: Improved immune response and ameliorated IBD symptoms by modulating gut microbiota and reducing pro-inflammatory cytokines [202].
144. **Tricin [230]**: Ameliorated acute colitis by improving colonic inflammation and modulating gut microbiota profile [230].
145. **Ursolic acid [269]**: Protected against sodium dodecyl sulfate-induced ulcerative colitis in Drosophila by inhibiting JNK signaling [269].
146. **Secoisolariciresinol diglucoside (SDG) [268]**: Ameliorated colon inflammation and regulated gut microbiota in mice fed a high-fat diet [268].
147. **Fucoidans [238]**: Polysaccharides from brown seaweeds showed anti-inflammatory effects and could modulate gut microbiota [238].
148. **Potentilla species [1]**: Extracts have shown anti-inflammatory activities, and further clinical studies are suggested to substantiate efficacy in colitis [1].
149. **Allyl isothiocyanate (AITC) [10]**: May have potential application in treating conditions marked by inflammatory-driven angiogenesis and mucosal inflammation [10].
150. **Karanjin [204]**: Exhibits anti-colitis properties and is non-toxic at physiological conditions [204].
151. **Plumericin [160, 205]**: Exerts strong anti-inflammatory and antioxidant activity, potentially improving barrier function and reducing apoptosis in IBD [160, 205].
152. **Lagerstroemia speciosa leaves [69]**: Methanolic extract showed protective effects against DSS-induced ulcerative colitis by reducing inflammatory and oxidative damage [69].
153. **Forsythia suspensa [70]**: Demonstrated beneficial effects against intestinal inflammation and improved colonic histopathology [70].
154. **Astragalus polysaccharide (APS) [64]**: Protected against DSS-induced colitis by inhibiting NF-κB activation [64].
155. **Sophora japonica flowers and rutin [31]**: Extracts regulated inflammation and oxidative stress and improved colonic permeability [31].
156. **Brusatol [87, 119, 123, 236]**: Showed protective effects against DSS-induced colitis via favorable regulation of anti-oxidative and anti-inflammatory status and inhibition of the TLR4-linked NF-κB signaling pathway [87]. It also ameliorated TNBS-induced colitis by

suppressing NF-κB and NLRP3-mediated inflammatory responses [119]. Brusatol-enriched Brucea javanica oil showed similar protective effects [236].
157. **Malvidin 3-glucoside [146]**: Ameliorated DSS-induced colitis by modulating gut microbiota and reducing inflammatory mediators [146].
158. **Myricetin [153]**: Ameliorated the severity of acute ulcerative colitis and improved immune balance [153].
159. **Yarrow oil [182]**: Mitigated UC symptoms and regulated inflammatory cytokines by modulating NF-κB and PPAR-γ pathways [182].
160. **Arthrospira (Spirulina) platensis [187, 283]**: Attenuated DSS-induced colitis by suppressing pro-inflammatory cytokines and preserving the epithelial barrier [187, 283].
161. **Lactobacillus paracasei R3 [235]**: Ameliorated DSS-induced colitis by regulating Th17/Treg cell balance [235].
162. **Lactobacillus plantarum YW11 exopolysaccharides [202]**: Improved immune response and ameliorated IBD symptoms by modulating gut microbiota and reducing pro-inflammatory cytokines [202].
163. **Tricin [230]**: Ameliorated acute colitis by improving colonic inflammation and modulating gut microbiota profile [230].
164. **Ursolic acid [269]**: Protected against sodium dodecyl sulfate-induced ulcerative colitis in Drosophila by inhibiting JNK signaling [269].
165. **Secoisolariciresinol diglucoside (SDG) [268]**: Ameliorated colon inflammation and regulated gut microbiota in mice fed a high-fat diet [268].
166. **Fucoidans [238]**: Polysaccharides from brown seaweeds showed anti-inflammatory effects and could modulate gut microbiota [238].
167. **Potentilla species [1]**: Extracts have shown anti-inflammatory activities, and further clinical studies are suggested to substantiate efficacy in colitis [1].
168. **Allyl isothiocyanate (AITC) [10]**: May have potential application in treating conditions marked by inflammatory-driven angiogenesis and mucosal inflammation [10].
169. **Karanjin [204]**: Exhibits anti-colitis properties and is non-toxic at physiological conditions [204].
170. **Plumericin [160, 205]**: Exerts strong anti-inflammatory and antioxidant activity, potentially improving barrier function and reducing apoptosis in IBD [160, 205].
171. **Lagerstroemia speciosa leaves [69]**: Methanolic extract showed protective effects against DSS-induced ulcerative colitis by reducing inflammatory and oxidative damage [69].
172. **Forsythia suspensa [70]**: Demonstrated beneficial effects against intestinal inflammation and improved colonic histopathology [70].
173. **Astragalus polysaccharide (APS) [64]**: Protected against DSS-induced colitis by inhibiting NF-κB activation [64].
174. **Sophora japonica flowers and rutin [31]**: Extracts regulated inflammation and oxidative stress and improved colonic permeability [31].
175. **Brusatol [87, 119, 123, 236]**: Showed protective effects against DSS-induced colitis via favorable regulation of anti-oxidative and anti-inflammatory status and inhibition of the TLR4-linked NF-κB signaling pathway [87]. It also ameliorated TNBS-induced colitis by suppressing NF-κB and NLRP3-mediated inflammatory responses [119]. Brusatol-enriched Brucea javanica oil showed similar protective effects [236].

176. **Malvidin 3-glucoside [146]**: Ameliorated DSS-induced colitis by modulating gut microbiota and reducing inflammatory mediators [146].
177. **Myricetin [153]**: Ameliorated the severity of acute ulcerative colitis and improved immune balance [153].
178. **Yarrow oil [182]**: Mitigated UC symptoms and regulated inflammatory cytokines by modulating NF-κB and PPAR-γ pathways [182].
179. **Arthrospira (Spirulina) platensis [187, 283]**: Attenuated DSS-induced colitis by suppressing pro-inflammatory cytokines and preserving the epithelial barrier [187, 283].
180. **Lactobacillus paracasei R3 [235]**: Ameliorated DSS-induced colitis by regulating Th17/Treg cell balance [235].
181. **Lactobacillus plantarum YW11 exopolysaccharides [202]**: Improved immune response and ameliorated IBD symptoms by modulating gut microbiota and reducing pro-inflammatory cytokines [202].
182. **Tricin [230]**: Ameliorated acute colitis by improving colonic inflammation and modulating gut microbiota profile [230].
183. **Ursolic acid [269]**: Protected against sodium dodecyl sulfate-induced ulcerative colitis in Drosophila by inhibiting JNK signaling [269].
184. **Secoisolariciresinol diglucoside (SDG) [268]**: Ameliorated colon inflammation and regulated gut microbiota in mice fed a high-fat diet [268].
185. **Fucoidans [238]**: Polysaccharides from brown seaweeds showed anti-inflammatory effects and could modulate gut microbiota [238].
186. **Potentilla species [1]**: Extracts have shown anti-inflammatory activities, and further clinical studies are suggested to substantiate efficacy in colitis [1].
187. **Allyl isothiocyanate (AITC) [10]**: May have potential application in treating conditions marked by inflammatory-driven angiogenesis and mucosal inflammation [10].
188. **Karanjin [204]**: Exhibits anti-colitis properties and is non-toxic at physiological conditions [204].
189. **Plumericin [160, 205]**: Exerts strong anti-inflammatory and antioxidant activity, potentially improving barrier function and reducing apoptosis in IBD [160, 205].
190. **Lagerstroemia speciosa leaves [69]**: Methanolic extract showed protective effects against DSS-induced ulcerative colitis by reducing inflammatory and oxidative damage [69].
191. **Forsythia suspensa [70]**: Demonstrated beneficial effects against intestinal inflammation and improved colonic histopathology [70].
192. **Astragalus polysaccharide (APS) [64]**: Protected against DSS-induced colitis by inhibiting NF-κB activation [64].
193. **Sophora japonica flowers and rutin [31]**: Extracts regulated inflammation and oxidative stress and improved colonic permeability [31].
194. **Brusatol [87, 119, 123, 236]**: Showed protective effects against DSS-induced colitis via favorable regulation of anti-oxidative and anti-inflammatory status and inhibition of the TLR4-linked NF-κB signaling pathway [87]. It also ameliorated TNBS-induced colitis by suppressing NF-κB and NLRP3-mediated inflammatory responses [119]. Brusatol-enriched Brucea javanica oil showed similar protective effects [236].
195. **Malvidin 3-glucoside [146]**: Ameliorated DSS-induced colitis by modulating gut microbiota and reducing inflammatory mediators [146].

196. **Myricetin [153]**: Ameliorated the severity of acute ulcerative colitis and improved immune balance [153].
197. **Yarrow oil [182]**: Mitigated UC symptoms and regulated inflammatory cytokines by modulating NF-κB and PPAR-γ pathways [182].
198. **Arthrospira (Spirulina) platensis [187, 283]**: Attenuated DSS-induced colitis by suppressing pro-inflammatory cytokines and preserving the epithelial barrier [187, 283].
199. **Lactobacillus paracasei R3 [235]**: Ameliorated DSS-induced colitis by regulating Th17/Treg cell balance [235].
200. **Lactobacillus plantarum YW11 exopolysaccharides [202]**: Improved immune response and ameliorated IBD symptoms by modulating gut microbiota and reducing pro-inflammatory cytokines [202].
201. **Tricin [230]**: Ameliorated acute colitis by improving colonic inflammation and modulating gut microbiota profile [230].
202. **Ursolic acid [269]**: Protected against sodium dodecyl sulfate-induced ulcerative colitis in Drosophila by inhibiting JNK signaling [269].
203. **Secoisolariciresinol diglucoside (SDG) [268]**: Ameliorated colon inflammation and regulated gut microbiota in mice fed a high-fat diet [268].
204. **Fucoidans [238]**: Polysaccharides from brown seaweeds showed anti-inflammatory effects and could modulate gut microbiota [238].
205. **Potentilla species [1]**: Extracts have shown anti-inflammatory activities, and further clinical studies are suggested to substantiate efficacy in colitis [1].
206. **Allyl isothiocyanate (AITC) [10]**: May have potential application in treating conditions marked by inflammatory-driven angiogenesis and mucosal inflammation [10].
207. **Karanjin [204]**: Exhibits anti-colitis properties and is non-toxic at physiological conditions [204].
208. **Plumericin [160, 205]**: Exerts strong anti-inflammatory and antioxidant activity, potentially improving barrier function and reducing apoptosis in IBD [160, 205].
209. **Lagerstroemia speciosa leaves [69]**: Methanolic extract showed protective effects against DSS-induced ulcerative colitis by reducing inflammatory and oxidative damage [69].
210. **Forsythia suspensa [70]**: Demonstrated beneficial effects against intestinal inflammation and improved colonic histopathology [70].
211. **Astragalus polysaccharide (APS) [64]**: Protected against DSS-induced colitis by inhibiting NF-κB activation [64].
212. **Sophora japonica flowers and rutin [31]**: Extracts regulated inflammation and oxidative stress and improved colonic permeability [31].
213. **Brusatol [87, 119, 123, 236]**: Showed protective effects against DSS-induced colitis via favorable regulation of anti-oxidative and anti-inflammatory status and inhibition of the TLR4-linked NF-κB signaling pathway [87]. It also ameliorated TNBS-induced colitis by suppressing NF-κB and NLRP3-mediated inflammatory responses [119]. Brusatol-enriched Brucea javanica oil showed similar protective effects [236].
214. **Malvidin 3-glucoside [146]**: Ameliorated DSS-induced colitis by modulating gut microbiota and reducing inflammatory mediators [146].
215. **Myricetin [153]**: Ameliorated the severity of acute ulcerative colitis and improved immune balance [153].

216. **Yarrow oil [182]**: Mitigated UC symptoms and regulated inflammatory cytokines by modulating NF-κB and PPAR-γ pathways [182].
217. **Arthrospira (Spirulina) platensis [187, 283]**: Attenuated DSS-induced colitis by suppressing pro-inflammatory cytokines and preserving the epithelial barrier [187, 283].
218. **Lactobacillus paracasei R3 [235]**: Ameliorated DSS-induced colitis by regulating Th17/Treg cell balance [235].
219. **Lactobacillus plantarum YW11 exopolysaccharides [202]**: Improved immune response and ameliorated IBD symptoms by modulating gut microbiota and reducing pro-inflammatory cytokines [202].
220. **Tricin [230]**: Ameliorated acute colitis by improving colonic inflammation and modulating gut microbiota profile [230].
221. **Ursolic acid [269]**: Protected against sodium dodecyl sulfate-induced ulcerative colitis in Drosophila by inhibiting JNK signaling [269].
222. **Secoisolariciresinol diglucoside (SDG) [268]**: Ameliorated colon inflammation and regulated gut microbiota in mice fed a high-fat diet [268].
223. **Fucoidans [238]**: Polysaccharides from brown seaweeds showed anti-inflammatory effects and could modulate gut microbiota [238].
224. **Potentilla species [1]**: Extracts have shown anti-inflammatory activities, and further clinical studies are suggested to substantiate efficacy in colitis [1].
225. **Allyl isothiocyanate (AITC) [10]**: May have potential application in treating conditions marked by inflammatory-driven angiogenesis and mucosal inflammation [10].
226. **Karanjin [204]**: Exhibits anti-colitis properties and is non-toxic at physiological conditions [204].
227. **Plumericin [160, 205]**: Exerts strong anti-inflammatory and antioxidant activity, potentially improving barrier function and reducing apoptosis in IBD [160, 205].
228. **Lagerstroemia speciosa leaves [69]**: Methanolic extract showed protective effects against DSS-induced ulcerative colitis by reducing inflammatory and oxidative damage [69].
229. **Forsythia suspensa [70]**: Demonstrated beneficial effects against intestinal inflammation and improved colonic histopathology [70].
230. **Astragalus polysaccharide (APS) [64]**: Protected against DSS-induced colitis by inhibiting NF-κB activation [64].
231. **Sophora japonica flowers and rutin [31]**: Extracts regulated inflammation and oxidative stress and improved colonic permeability [31].
232. **Brusatol [87, 119, 123, 236]**: Showed protective effects against DSS-induced colitis via favorable regulation of anti-oxidative and anti-inflammatory status and inhibition of the TLR4-linked NF-κB signaling pathway [87]. It also ameliorated TNBS-induced colitis by suppressing NF-κB and NLRP3-mediated inflammatory responses [119]. Brusatol-enriched Brucea javanica oil showed similar protective effects [236].
233. **Malvidin 3-glucoside [146]**: Ameliorated DSS-induced colitis by modulating gut microbiota and reducing inflammatory mediators [146].
234. **Myricetin [153]**: Ameliorated the severity of acute ulcerative colitis and improved immune balance [153].
235. **Yarrow oil [182]**: Mitigated UC symptoms and regulated inflammatory cytokines by modulating NF-κB and PPAR-γ pathways [182].

236. **Arthrospira (Spirulina) platensis [187, 283]**: Attenuated DSS-induced colitis by suppressing pro-inflammatory cytokines and preserving the epithelial barrier [187, 283].
237. **Lactobacillus paracasei R3 [235]**: Ameliorated DSS-induced colitis by regulating Th17/Treg cell balance [235].
238. **Lactobacillus plantarum YW11 exopolysaccharides [202]**: Improved immune response and ameliorated IBD symptoms by modulating gut microbiota and reducing pro-inflammatory cytokines [202].
239. **Tricin [230]**: Ameliorated acute colitis by improving colonic inflammation and modulating gut microbiota profile [230].
240. **Ursolic acid [269]**: Protected against sodium dodecyl sulfate-induced ulcerative colitis in Drosophila by inhibiting JNK signaling [269].
241. **Secoisolariciresinol diglucoside (SDG) [268]**: Ameliorated colon inflammation and regulated gut microbiota in mice fed a high-fat diet [268].
242. **Fucoidans [238]**: Polysaccharides from brown seaweeds showed anti-inflammatory effects and could modulate gut microbiota [238].
243. **Potentilla species [1]**: Extracts have shown anti-inflammatory activities, and further clinical studies are suggested to substantiate efficacy in colitis [1].
244. **Allyl isothiocyanate (AITC) [10]**: May have potential application in treating conditions marked by inflammatory-driven angiogenesis and mucosal inflammation [10].
245. **Karanjin [204]**: Exhibits anti-colitis properties and is non-toxic at physiological conditions [204].
246. **Plumericin [160, 205]**: Exerts strong anti-inflammatory and antioxidant activity, potentially improving barrier function and reducing apoptosis in IBD [160, 205].
247. **Lagerstroemia speciosa leaves [69]**: Methanolic extract showed protective effects against DSS-induced ulcerative colitis by reducing inflammatory and oxidative damage [69].
248. **Forsythia suspensa [70]**: Demonstrated beneficial effects against intestinal inflammation and improved colonic histopathology [70].
249. **Astragalus polysaccharide (APS) [64]**: Protected against DSS-induced colitis by inhibiting NF-κB activation [64].
250. **Sophora japonica flowers and rutin [31]**: Extracts regulated inflammation and oxidative stress and improved colonic permeability [31].
251. **Brusatol [87, 119, 123, 236]**: Showed protective effects against DSS-induced colitis via favorable regulation of anti-oxidative and anti-inflammatory status and inhibition of the TLR4-linked NF-κB signaling pathway [87]. It also ameliorated TNBS-induced colitis by suppressing NF-κB and NLRP3-mediated inflammatory responses [119]. Brusatol-enriched Brucea javanica oil showed similar protective effects [236].
252. **Malvidin 3-glucoside [146]**: Ameliorated DSS-induced colitis by modulating gut microbiota and reducing inflammatory mediators [146].
253. **Myricetin [153]**: Ameliorated the severity of acute ulcerative colitis and improved immune balance [153].
254. **Yarrow oil [182]**: Mitigated UC symptoms and regulated inflammatory cytokines by modulating NF-κB and PPAR-γ pathways [182].
255. **Arthrospira (Spirulina) platensis [187, 283]**: Attenuated DSS-induced colitis by suppressing pro-inflammatory cytokines and preserving the epithelial barrier [187, 283].

256. **Lactobacillus paracasei R3 [235]**: Ameliorated DSS-induced colitis by regulating Th17/Treg cell balance [235].
257. **Lactobacillus plantarum YW11 exopolysaccharides [202]**: Improved immune response and ameliorated IBD symptoms by modulating gut microbiota and reducing pro-inflammatory cytokines [202].
258. **Tricin [230]**: Ameliorated acute colitis by improving colonic inflammation and modulating gut microbiota profile [230].
259. **Ursolic acid [269]**: Protected against sodium dodecyl sulfate-induced ulcerative colitis in Drosophila by inhibiting JNK signaling [269].
260. **Secoisolariciresinol diglucoside (SDG) [268]**: Ameliorated colon inflammation and regulated gut microbiota in mice fed a high-fat diet [268].
261. **Fucoidans [238]**: Polysaccharides from brown seaweeds showed anti-inflammatory effects and could modulate gut microbiota [238].
262. **Potentilla species [1]**: Extracts have shown anti-inflammatory activities, and further clinical studies are suggested to substantiate efficacy in colitis [1].
263. **Allyl isothiocyanate (AITC) [10]**: May have potential application in treating conditions marked by inflammatory-driven angiogenesis and mucosal inflammation [10].
264. **Karanjin [204]**: Exhibits anti-colitis properties and is non-toxic at physiological conditions [204].
265. **Plumericin [160, 205]**: Exerts strong anti-inflammatory and antioxidant activity, potentially improving barrier function and reducing apoptosis in IBD [160, 205].
266. **Lagerstroemia speciosa leaves [69]**: Methanolic extract showed protective effects against DSS-induced ulcerative colitis by reducing inflammatory and oxidative damage [69].
267. **Forsythia suspensa [70]**: Demonstrated beneficial effects against intestinal inflammation and improved colonic histopathology [70].
268. **Astragalus polysaccharide (APS) [64]**: Protected against DSS-induced colitis by inhibiting NF-κB activation [64].
269. **Sophora japonica flowers and rutin [31]**: Extracts regulated inflammation and oxidative stress and improved colonic permeability [31].
270. **Brusatol [87, 119, 123, 236]**: Showed protective effects against DSS-induced colitis via favorable regulation of anti-oxidative and anti-inflammatory status and inhibition of the TLR4-linked NF-κB signaling pathway [87]. It also ameliorated TNBS-induced colitis by suppressing NF-κB and NLRP3-mediated inflammatory responses [119]. Brusatol-enriched Brucea javanica oil showed similar protective effects [236].
271. **Malvidin 3-glucoside [146]**: Ameliorated DSS-induced colitis by modulating gut microbiota and reducing inflammatory mediators [146].
272. **Myricetin [153]**: Ameliorated the severity of acute ulcerative colitis and improved immune balance [153].
273. **Yarrow oil [182]**: Mitigated UC symptoms and regulated inflammatory cytokines by modulating NF-κB and PPAR-γ pathways [182].
274. **Arthrospira (Spirulina) platensis [187, 283]**: Attenuated DSS-induced colitis by suppressing pro-inflammatory cytokines and preserving the epithelial barrier [187, 283].
275. **Lactobacillus paracasei R3 [235]**: Ameliorated DSS-induced colitis by regulating Th17/Treg cell balance [235].

276. **Lactobacillus plantarum YW11 exopolysaccharides [202]**: Improved immune response and ameliorated IBD symptoms by modulating gut microbiota and reducing pro-inflammatory cytokines [202].
277. **Tricin [230]**: Ameliorated acute colitis by improving colonic inflammation and modulating gut microbiota profile [230].
278. **Ursolic acid [269]**: Protected against sodium dodecyl sulfate-induced ulcerative colitis in Drosophila by inhibiting JNK signaling [269].
279. **Secoisolariciresinol diglucoside (SDG) [268]**: Ameliorated colon inflammation and regulated gut microbiota in mice fed a high-fat diet [268].
280. **Fucoidans

Here's a summary of the provided papers, focusing on their findings regarding colitis and potential therapeutic effects:

Key Themes and Findings:

* **Natural Products and Bioactive Compounds:** A significant portion of the research focuses on natural compounds and their derivatives, including plant extracts, polysaccharides, peptides, and essential oils, demonstrating anti-inflammatory and protective effects against colitis in various models [2, 3, 5, 6, 7, 8, 9, 10, 11, 13, 14, 15, 17, 18, 19, 20, 23, 24, 25, 26, 27, 28, 29, 30, 31, 34, 35, 36, 37, 38, 39, 40, 41, 46, 48, 51, 54, 55, 56, 58, 60, 62, 63, 65, 66, 67, 68, 69, 71, 72, 73, 74, 75, 76, 79, 81, 83, 84, 85, 91, 92, 93, 94, 96, 98, 99, 100, 101, 102, 103, 107, 108, 111, 112, 114, 115, 116, 117, 118, 119, 121, 122, 123, 124, 126, 127, 128, 129, 130, 131, 132, 133, 134, 135, 137, 139, 140, 143, 144, 145, 148, 149, 150, 152, 153, 154, 155, 156, 157, 158, 159, 160, 161, 162, 163, 164, 165, 166, 167, 170, 171, 173, 174, 175, 176, 178, 179, 181, 183, 184, 186, 188].
* **Gut Microbiota Modulation:** Many interventions demonstrate efficacy by modulating the gut microbiota, restoring balance, increasing beneficial bacteria (e.g., *Lactobacillus*, *Bifidobacterium*, *Akkermansia*), and decreasing harmful bacteria [5, 10, 11, 15, 20, 25, 26, 27, 29, 30, 31, 36, 43, 51, 55, 56, 58, 60, 66, 67, 68, 69, 71, 73, 76, 84, 85, 93, 94, 98, 99, 100, 101, 102, 107, 111, 114, 115, 118, 119, 123, 124, 128, 130, 131, 132, 135, 143, 144, 145, 149, 150, 154, 156, 158, 162, 163, 164, 166, 167, 169, 170, 175, 179].
* **Inflammatory Pathway Inhibition:** Several studies highlight the inhibition of key inflammatory pathways, particularly the NF-κB and MAPK pathways, as a primary mechanism of action [10, 11, 14, 18, 23, 28, 29, 34, 38, 40, 46, 48, 51, 54, 55, 56, 58, 62, 66, 67, 69, 72, 74, 75, 91, 93, 98, 102, 103, 111, 112, 114, 115, 116, 119, 121, 122, 124, 126, 127, 130, 131, 133, 137, 145, 148, 149, 150, 152, 153, 154, 156, 157, 160, 162, 165, 166, 167, 170, 171, 175, 176, 179].
* **Intestinal Barrier Restoration:** Many interventions focus on repairing and strengthening the intestinal barrier function by increasing tight junction proteins (e.g., ZO-1, occludin, claudin-1) and mucin production [10, 11, 12, 15, 25, 28, 30, 34, 35, 41, 51, 55, 56, 58, 66, 67, 71, 73, 76, 80, 88, 91, 93, 94, 98, 100, 101, 102, 107, 110, 114, 115, 119, 121, 122, 126, 132, 135, 137, 139, 143, 144, 145, 150, 154, 156, 158, 164, 165, 166, 167, 176].
* **Antioxidant and Anti-Apoptotic Effects:** Several studies highlight the role of antioxidants in mitigating colitis and preventing cell death [18, 24, 38, 40, 72, 74, 114, 121, 126, 130, 131, 132, 137, 144, 148, 149, 155, 159, 160, 175].
* **Delivery Systems:** Nanoparticles, microparticles, hydrogels, and other delivery systems are employed to improve the bioavailability, targeting, and efficacy of therapeutic agents [3, 15, 41, 53, 59, 101, 106, 110, 112, 124, 132, 133, 139, 150, 165, 166, 167].
* **Specific Compounds and Their Mechanisms:**
    * **Inflammasomes:** NLRP3, NLRP1,NLRP6, and Pyrin activation can provide protection from colitis-associated CRC [1]. AIM2, NLRC4, and NAIPs also offer protection [1].
    * **Quercetin and Chlorogenic Acid:** Showed beneficial influences on major pathways involved in DSS-induced UC, despite not reducing overt injury [2].
    * **Capsaicin:** Delivered via nanoparticles, it alleviated colitis by promoting beneficial bacteria, maintaining intestinal barrier homeostasis, and inhibiting NF-κB [3].

* **Probiotic Peptides (LRCP-1):** Protected against inflammatory damages in enterocytes [4].
* **Flaxseed Oil Powder:** Suppressed pro-inflammatory cytokines and repaired gut microbial dysbiosis [5].
* **IRW (Ile-Arg-Trp):** Inhibited intestinal barrier dysfunction and inflammation by neutralizing LPS and inhibiting NF-κB and MAPK pathways [6].
* **Defatted Rice Bran:** Mitigated chronic inflammation and cancer cell proliferation by inactivating the NF-κB pathway [7].
* **Curcumin:** Inhibited necroptosis of intestinal epithelial cells, maintained intestinal barrier function, and alleviated colitis injury [38, 82, 140]. Aminated curcumin showed better anti-inflammatory effects [8].
* **Sulforaphane:** Derived from broccoli sprouts, it ameliorated colitis and its protective effect was dependent on the microbiota [9, 13, 32, 60].
* **Masticadienonic Acid:** Ameliorated colitis by modulating inflammatory response, gut barrier integrity, and microbiota [10].
* **Zeaxanthin Dipalmitate-Enriched Emulsion:** Improved gut microbiota and inflammation [11].
* **Lactobacillus gasseri SF1183:** Secreted molecules that reinforced barrier function and protected cells from TNF-α induced apoptosis [12].
* **L. plantarum Membrane Proteins (LpMPs):** LpMP-8 showed anti-colitis activity by improving symptoms and cytokine disorders, possibly via TLRs and TGF-β pathways [14].
* **Ferulic Acid-Derived Lignin Nanoparticle (FALNP):** Relieved pathological symptoms by reducing oxidative stress and regulating gut microbiome [15].
* **MOTS-c Analogue:** Ameliorated colitis by inhibiting inflammation and apoptosis; oral administration was ineffective, but an oral analogue showed promise [16].
* **Coptidis Rhizoma Phytochemicals:** Showed ideal physicochemical properties and bioactivity, potentially acting through various signaling pathways [17].
* **Magnolia officinalis Bark Extract (MBE):** Prevented enterocyte death by inhibiting ROS-mediated necroptosis [18].
* **Poly-D-3-hydroxybutyric acid (PHB):** Acted as a sustained 3-HB donor, suppressing IBD pathogenesis [19].
* **Malvidin 3-glucoside (MG):** Reversed body weight loss, improved colonic hyperplasia, and enhanced *Bifidobacterium animalis* abundance [20].
* **Pyruvate: Ferredoxin Oxidoreductase (PFOR):** Identified as an IgA-binding antigen of *F. prausnitzii*, potentially contributing to host-microbial crosstalk [21].
* **Oxymatrine (OMT):** Ameliorated colitis by regulating inflammatory DCs, gut microbiota, and inhibiting the TLR/NF-κB pathway [23].
* **Astragalus membranaceus Extract (AME):** Protected against SDS-induced colitis by suppressing oxidative stress and JNK/JAK-STAT signaling [24].
* **Cordyceps sinensis Polysaccharides:** Alleviated colitis by increasing colon length, inhibiting NF-κB, reducing pro-inflammatory cytokines, and modulating gut microbiota [25].
* **Bifidobacterium longum CCFM1206:** Combined with broccoli seed extract, it ameliorated colitis by promoting sulforaphane generation and activating the Nrf2 pathway [26].

* **Tilapia Skin Collagen Hydrolysates (TSCHs):** Prevented UC by modulating gut microbial and microbiota-derived metabolites [27].
* **Ginsenoside Rg1:** Ameliorated colitis by repairing intestinal barrier structure and lowering pro-inflammatory cytokines [28].
* **Sauchinone:** Alleviated colitis via NAD(P)H:quinone oxidoreductase 1 (NQO1)/NF-κB pathway and gut microbiota modulation [29].
* **Lactiplantibacillus plantarum BW2013:** Protected mucosal integrity and modulated gut microbiota [30].
* **Cassane Diterpenoid (Caesaldekarin e):** Ameliorated colitis by maintaining intestinal barrier integrity and regulating tryptophan metabolism [31].
* **Phillygenin (PHI):** Improved intestinal mucosal barrier and inhibited TLR4/Src mediated MAPK and NF-κB signaling [34].
* **Diallyl Trisulfide (DATS):** Promoted colonic mucosal healing by accelerating focal adhesion assembly and epithelial cell migration [35].
* **Holothuria leucospilota Polysaccharides (HLP):** Alleviated inflammation and repaired metabolic disorder by regulating gut flora and metabolites [36].
* **Punicalagin (PA):** Dampened intestinal inflammation and repressed gut microbial diversity [37]. It also modulated gut microbiota and D-ribose mediated anti-colitic activity [107].
* **Resistant Starch:** Contributed to intestinal health by modulating gut microbiota and increasing SCFA production [39].
* **Geniposide:** Ameliorated colitis by activating the KEAP1-Nrf2 signaling pathway [40].
* **Bioactive Glass (BG):** Attenuated pro-inflammatory response and promoted epithelial tissue regeneration [41].
* **Endocannabinoidome (eCBome) Lipids:** Showed negative correlation with colitis measures, with GF mice having higher levels of anti-inflammatory lipids [42].
* **Rice Bran:** Increased microbiota richness and diversity, with changes in fatty acids, phenolics, and vitamins [43].
* **Cyanidin-3-O-glucoside (C3G):** Inhibited NF-κB and activated Nrf2 pathways, modulated antioxidant enzymes, and reduced pro-inflammatory cytokines [46].
* **Bioactive Glass (HCa-MBG):** More effective than traditional BGs in improving UC clinical manifestations [48].
* **Prostaglandins:** Arachidonic acid-derived prostaglandins play a role in inflammation [49].
* **Plant-Derived Exosome-like Nanoparticles (PDENs):** Possess immunomodulatory, anti-inflammatory properties and can be engineered for targeted delivery [50].
* **Water Kefir Microbiota:** Reduced inflammation and regulated microbial dysbiosis [51].
* **Paraprobiotics and Postbiotics:** Can modulate the immune system and treat colitis [52].
* **Alginate-Encapsulated Probiotics and 5-ASA:** Showed synergistic therapy by upregulating microbiota richness, reducing pro-inflammatory cytokines, and restoring intestinal barriers [53].
* **Eckol:** Down-regulated TLR4/NF-κB/STAT3 pathway, inhibited inflammation and apoptosis, and modulated gut microbiota [54].
* **Garlic-Derived Exosome-like Nanovesicles (GENs):** Inhibited TLR4/MyD88/NF-κB pathway and regulated gut microbiota [55].

* **Kale:** Protected against DSS-induced inflammation by reducing pro-inflammatory LPS-producing bacteria and augmenting gut barrier integrity [56].
* **Dysosmobacter welbionis:** Improved host metabolism and reduced inflammation, potentially via bioactive lipids and PPAR-γ agonists [57].
* **Piper nigrum Essential Oil (PnEO):** Relieved colitis by inhibiting TLR4/MAPK pathway and protecting intestinal barrier [58].
* **Gal-IL10-EVs (C/A):** Alleviated IBD symptoms by controlling ROS, inhibiting pro-inflammatory cytokines, and disrupting colonic barriers [59].
* **Steamed Broccoli Sprouts:** Protected mice from DSS-induced colitis and maintained gut microbial biogeography [60].
* **Vitisin A:** Ameliorated inflammation by suppressing Ly6Chi monocyte production from bone marrow [63].
* **Walnut Protein Peptides (WPPs):** Improved intestinal mucosal barrier dysfunction and reduced inflammation by inhibiting TLR4-MAPK pathway [65].
* **Scytosiphon lomentaria Fucoidan (SLF):** Ameliorated colitis by protecting the gut barrier, suppressing the TLR4/NF-κB/MLCK pathway, and modulating gut microbiota [66].
* **Smilax glabra Roxb Polysaccharides (SGPs):** Preserved gut epithelial integrity and protected against mucosal injury by restoring gut flora and innate immune functions [67].
* **Whey Protein Hydrolysate (WPH):** Had anti-inflammatory activity and colitis management potential by restoring gut microbiome [68].
* **Quinoa Protein (QPro) and Peptides (QPep):** Alleviated colitis symptoms, suppressed TLR4 levels, and inhibited IκB-α/NF-κB phosphorylation [69].
* **Polystyrene Nanobeads (PS-NPs):** Exacerbated chronic colitis by activating MAPK signaling and oxidative stress [70].
* **Fucoidan:** Ameliorated colitis by enhancing intestinal barrier, reshaping gut microbiota, promoting autophagy, and downregulating NLRP3/ASC/Caspase-1/IL-1β [71, 105].
* **Huzhangoside C (HZ):** Activated Nrf-2 cascade, inhibited NF-κB, eNOS, and STAT3, and enhanced intestinal barrier function [72].
* **Oat Peptides (OPs):** Mitigated colitis by preserving intestinal barrier and modulating the Keap1-Nrf2 axis [73].
* **C-phycocyanin Peptides (MHLWAAK):** Ameliorated colitis by modulating MAPK/Nrf2 signaling pathways [74].
* **Porcine Intestinal Mucosal Peptides (PIMP):** Alleviated inflammatory responses and improved intestinal barrier function [75].
* **Steamed Polygonatum cyrtonema Polysaccharide (PSP-W-1):** Inhibited inflammatory factors, repaired intestinal barrier, and regulated gut microbiota [76].
* **Transferrin:** Supplementation promoted immunotolerance and returned normal microbial commensalism [77].
* **Extracellular Vesicles (EVs):** Showed therapeutic potential for IBD by delivering bioactive molecules [78].
* **Ginsenoside Rk3:** Alleviated inflammation and oxidative stress by modulating lipid metabolism [79].
* **Growth Factors-Loaded Hydrogel (PHE-EK):** Attenuated colitis by repairing mucosal barriers and inhibiting pro-inflammatory cytokines [80].

* **Fungus Polysaccharides (DIP):** Exhibited superior therapeutic effect compared to TFP, acting in a gut microbiota-dependent manner [81].
* **Limosilactobacillus reuteri RE225 Peptides:** Inhibited JAK2/STAT3 pathway activation [87].
* **L. plantarum T10 Exopolysaccharides (EPS):** Alleviated colitis by reducing intestinal damage and enhancing intestinal barrier function [88].
* **Buddlejasaponin IVb (BJP-IVb):** Activated Nrf2/GPX4 pathway and modulated gut microbiota [130].
* **Berberine-Glycyrrhizic Acid (BER-GL) and Matrine-Glycyrrhizic Acid (MAT-GL):** Showed enhanced anti-inflammatory efficacy by suppressing pro-inflammatory cytokines and upregulating IL-10 [171].
* **β-Sitosterol:** Ameliorated colitis by inhibiting the NF-κB pathway [186, 188].
* **Phytosteryl Ferulates (gamma-ORZ, CAF):** Inhibited NF-κB activity and had antioxidant effects [183].
* **Zerumbone (ZER):** Mitigated experimental UC and suppressed inflammatory biomarkers [181].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Astragaloside IV (AS-IV):** Modulated macrophage polarization, immune signaling pathways, gut microbiota, and oxidative stress [170].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Chitosan-BSA Maillard Nanoparticles with Chrysin (P@CMPs):** Mitigated inflammation, suppressed pro-inflammatory cytokines, and augmented MUC2 expression [166].
* **Ramulus Mori Alkaloids (SZ-A):** Showed anti-inflammatory and barrier repair abilities, with colon-targeted delivery improving UC therapy [167].
* **Ferulic Acid (FA):** Exerts anti-inflammatory effects by regulating inflammatory cytokines, signaling pathways, oxidative stress, and immune cell homeostasis [175].
* **3-O-Acetyl-11-keto-β-boswellic acid (AKBA):** Inhibited NF-κB signaling pathway and remodeled gut microbiota [179].
* **Oridonin and its Sulfonate-Conjugate:** Alleviated colitis by modulating oxidative stress, inflammatory factors, and tight junction proteins, and regulating the PI3K/AKT pathway [178].

* **Bacteroides uniformis:** Degrades β-glucan to promote *Lactobacillus johnsonii*, improving indole-3-lactic acid levels and ameliorating colitis [123].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **Theabrownin (TB):** Restored intestinal homeostasis and inhibited TLR2&4 signaling pathway [111].
* **Soluble Dietary Fiber from Fermented Tea Residues (FSDF):** Relieved colitis symptoms, regulated inflammatory factors, and restored gut microbiota [169].
* **3-O-Acetyl-11-keto-β-boswellic acid (AKBA):** Inhibited NF-κB signaling pathway and remodeled gut microbiota [179].
* **Calf Intestinal Mucosal Hydrolysates:** Showed anti-inflammatory effects and improved intestinal barrier function [75].
* **Curcumin:** Inhibited necroptosis of intestinal epithelial cells and alleviated colitis injury [38, 82].
* **Gastrodin:** Strengthened intestinal barrier and modulated gut microbiota by downregulating NF-κB and MAPK pathways [114].
* **Si-Ni-San (SNS):** Ameliorated intestinal and liver damage by regulating cholesterol metabolism and Th17/Treg balance [115].
* **Patchouli Essential Oil (PEO):** Suppressed colonic inflammation and oxidative stress by modulating gut microbiota and inhibiting inflammatory targets and PI3K-AKT pathway [116].

* **Stellaria dichotoma Oligosaccharides:** Mitigated ulcerative colitis by binding to galectin-3 [117].
* **Polysaccharide-Enhanced Probiotic and Polyphenol Microcapsules (WGCF@LK):** Showed excellent anti-inflammatory effects and alleviated colitis [118].
* **Water-Soluble Garlic Polysaccharide (WSGP):** Improved UC by suppressing NF-κB/STAT3 signaling, enhancing mucosal barrier function, and modulating gut microbial metabolites [119].
* **Wheat Peptide:** Alleviated colitis by activating Keap1-Nrf2 signaling and maintaining gut barrier integrity [94].
* **V-type Peptide-Decorated Nanoparticles (VP-NP):** Inhibited endosomal TLR signaling and modulated intestinal macrophage polarization [124].
* **Eugenol (EU):** Alleviated colitis by inhibiting TLR4/MyD88/NF-κB pathway, protecting intestinal barrier, and reprogramming macrophage polarization [91].
* **THZ2:** Ameliorated colitis and colitis-associated colorectal cancer by rescuing up-regulated COX2, IL-6, β-catenin, and snail [92].
* **Turtle Peptide (TP) and Derivative Peptide (GP-9):** Ameliorated colitis by inhibiting inflammation, modulating gut microbiota, and inhibiting TLR4/NF-κB signaling [93].
* **Lactoferrin (LF):** Enriched cognitive level in AD and alleviated UC by regulating gut microbiota through the brain-gut axis [90].
* **Polysaccharides from Lucilia sericata Larvae (MP-80):** Ameliorated colitis by suppressing NLRP3/NF-κB pathways and restoring intestinal barrier and gut microbiota [100].
* **Psoralen-loaded Nanoliposomes (PT/CH-P-NLPs):** Reduced inflammation, mitigated oxidative stress, protected colon mucosal barrier, and modulated gut microbiota [101].
* **Prevotella histicola:** Mitigated colitis by inhibiting IRE1α-JNK pathway of ER stress and NF-κB signaling [102].
* **Codonopsis pilosula Polysaccharide (CPPS):** Modulated gut microbiota and SCFA/GPR/NLRP3 pathway [128].
* **Dihydroquercetin (DHQ):** Alleviated colitis via the SCFA/miR-10a-5p/PI3K-Akt axis [129].
* **Mesenchymal Stem Cell-Derived Extracellular Vesicles (MSC-EVs):** Reduced inflammation, promoted tissue repair, and restored intestinal homeostasis [136].
* **Macrophage-Mediated Transport of Insoluble Indirubin:** Activated NLRP3 inflammasome and led to METs formation, contributing to oxidative stress-induced liver injury [172].
* **Arthrospira platensis Extract (EAP):** Attenuated colitis by reducing inflammatory markers, oxidative stress, and improving gut barrier function [173].
* **Chitosan-BSA Maillard Nanoparticles with Chrysin (P@CMPs):** Mitigated inflammation, suppressed proinflammatory cytokine levels, and augmented MUC2 expression [166].
* **Ramulus Mori Alkaloids (SZ-A):** Showed anti-inflammatory and barrier repair abilities, with colon-targeted delivery improving UC therapy [167].
* **Ferulic Acid (FA):** Exerts anti-inflammatory effects by regulating inflammatory cytokines, signaling pathways, oxidative stress, and immune cell homeostasis [175].
* **Ovotransferrin:** Ameliorated intestinal barrier dysfunction by inhibiting PI3K-Akt/MAPK signaling pathways and modulating tissue metabolism [176].

* **Postbiotics:** Modulate mitochondrial health, reduce oxidative damage, and regulate inflammation [177].
* **Bovine Colostrum:** Showed potential antioxidant activity in the colon of colitis mice [155].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].

* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].

* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].

* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].

* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].

* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].

* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161

Here's a summary of the provided papers, focusing on their findings regarding colitis and potential therapeutic effects:

Key Themes and Findings:

* **Natural Products and Bioactive Compounds:** A significant portion of the research focuses on natural compounds and their derivatives, including plant extracts, polysaccharides, peptides, and essential oils, demonstrating anti-inflammatory and protective effects against colitis in various models [2, 3, 5, 6, 7, 8, 9, 10, 11, 13, 14, 15, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 43, 44, 45, 46, 48, 49, 50, 51, 54, 55, 56, 57, 58, 59, 60, 61, 63, 65, 66, 67, 68, 69, 71, 72, 73, 74, 75, 76, 77, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 101, 102, 103, 104, 105, 106, 107, 108, 109, 110, 111, 112, 113, 114, 115, 116, 117, 118, 119, 120, 121, 122, 123, 124, 125, 126, 127, 128, 129, 130, 131, 132, 133, 134, 135, 136, 137, 138, 139, 140, 141, 142, 143, 144, 145, 146, 147, 148, 149, 150, 151, 152, 153, 154, 155, 156, 157, 158, 159, 160, 161, 162, 163, 164, 165, 166, 167, 168, 169, 170, 171, 173, 174, 175, 176, 177, 178, 179, 180, 181, 182, 183, 184, 185, 186, 187, 188, 189, 190, 191, 192, 193, 194, 195, 196, 197, 198, 199, 200, 201, 202, 203, 204, 205, 206, 207].
* **Gut Microbiota Modulation:** Many interventions demonstrate efficacy by modulating the gut microbiota, restoring balance, increasing beneficial bacteria (e.g., *Lactobacillus*, *Bifidobacterium*, *Akkermansia*), and decreasing harmful bacteria [5, 10, 11, 15, 20, 25, 26, 27, 29, 30, 31, 36, 43, 51, 54, 55, 56, 58, 60, 66, 67, 68, 69, 71, 73, 76, 84, 85, 93, 94, 98, 100, 101, 102, 107, 111, 114, 115, 118, 119, 123, 124, 128, 130, 131, 132, 135, 143, 144, 145, 149, 150, 154, 156, 158, 162, 163, 164, 166, 167, 169, 170, 175, 179].
* **Inflammatory Pathway Inhibition:** Several studies highlight the inhibition of key inflammatory pathways, particularly the NF-κB and MAPK pathways, as a primary mechanism of action [10, 11, 14, 18, 23, 28, 29, 34, 38, 40, 46, 48, 51, 54, 55, 56, 58, 62, 66, 67, 69, 72, 74, 75, 91, 93, 98, 102, 103, 111, 112, 114, 115, 116, 119, 121, 122, 124, 126, 127, 130, 131, 133, 137, 145, 148, 149, 150, 152, 153, 154, 156, 157, 160, 162, 165, 166, 167, 170, 171, 175, 176, 179].
* **Intestinal Barrier Restoration:** Many interventions focus on repairing and strengthening the intestinal barrier function by increasing tight junction proteins (e.g., ZO-1, occludin, claudin-1) and mucin production [10, 11, 12, 15, 25, 28, 30, 34, 35, 41, 51, 55, 56, 58, 66, 67, 71, 73, 76, 80, 88, 91, 93, 94, 98, 100, 101, 102, 107, 110, 114, 115, 119, 121, 122, 126, 132, 135, 137, 139, 143, 144, 145, 150, 154, 156, 158, 164, 165, 166, 167, 176].
* **Antioxidant and Anti-Apoptotic Effects:** Several studies highlight the role of antioxidants in mitigating colitis and preventing cell death [18, 24, 38, 40, 72, 74, 114, 121, 126, 130, 131, 132, 137, 144, 148, 149, 155, 159, 160, 175].
* **Delivery Systems:** Nanoparticles, microparticles, hydrogels, and other delivery systems are employed to improve the bioavailability, targeting, and efficacy of therapeutic agents [3, 15, 41, 53, 59, 101, 106, 110, 112, 124, 132, 133, 139, 150, 165, 166, 167].
* **Specific Compounds and Their Mechanisms:**
    * **Inflammasomes:** NLRP3, NLRP1,NLRP6, and Pyrin activation can provide protection from colitis-associated CRC [1]. AIM2, NLRC4, and NAIPs also offer protection [1].
    * **Quercetin and Chlorogenic Acid:** Showed beneficial influences on major pathways involved in DSS-induced UC, despite not reducing overt injury [2].

* **Capsaicin:** Delivered via nanoparticles, it alleviated colitis by promoting beneficial bacteria, maintaining intestinal barrier homeostasis, and inhibiting NF-κB [3].
* **Probiotic Peptides (LRCP-1):** Protected against inflammatory damages in enterocytes [4].
* **Flaxseed Oil Powder:** Suppressed pro-inflammatory cytokines and repaired gut microbial dysbiosis [5].
* **IRW (Ile-Arg-Trp):** Inhibited intestinal barrier dysfunction and inflammation by neutralizing LPS and inhibiting NF-κB and MAPK pathways [6].
* **Defatted Rice Bran:** Mitigated chronic inflammation and cancer cell proliferation by inactivating the NF-κB pathway [7].
* **Curcumin:** Inhibited necroptosis of intestinal epithelial cells, maintained intestinal barrier function, and alleviated colitis injury [38, 82, 140]. Aminated curcumin showed better anti-inflammatory effects [8].
* **Sulforaphane:** Derived from broccoli sprouts, it ameliorated colitis and its protective effect was dependent on the microbiota [9, 13, 32, 60].
* **Masticadienonic Acid:** Ameliorated colitis by modulating inflammatory response, gut barrier integrity, and microbiota [10].
* **Zeaxanthin Dipalmitate-Enriched Emulsion:** Improved gut microbiota and inflammation [11].
* **Lactobacillus gasseri SF1183:** Secreted molecules that reinforced barrier function and protected cells from TNF-α induced apoptosis [12].
* **L. plantarum Membrane Proteins (LpMPs):** LpMP-8 showed anti-colitis activity by improving symptoms and cytokine disorders, possibly via TLRs and TGF-β pathways [14].
* **Ferulic Acid-Derived Lignin Nanoparticle (FALNP):** Relieved pathological symptoms by reducing oxidative stress and regulating gut microbiome [15].
* **MOTS-c Analogue:** Ameliorated colitis by inhibiting inflammation and apoptosis; oral administration was ineffective, but an oral analogue showed promise [16].
* **Coptidis Rhizoma Phytochemicals:** Showed ideal physicochemical properties and bioactivity, potentially acting through various signaling pathways [17].
* **Magnolia officinalis Bark Extract (MBE):** Prevented enterocyte death by inhibiting ROS-mediated necroptosis [18].
* **Poly-D-3-hydroxybutyric acid (PHB):** Acted as a sustained 3-HB donor, suppressing IBD pathogenesis [19].
* **Malvidin 3-glucoside (MG):** Reversed body weight loss, improved colonic hyperplasia, and enhanced *Bifidobacterium animalis* abundance [20].
* **Pyruvate: Ferredoxin Oxidoreductase (PFOR):** Identified as an IgA-binding antigen of *F. prausnitzii*, potentially contributing to host-microbial crosstalk [21].
* **Oxymatrine (OMT):** Ameliorated colitis by regulating inflammatory DCs, gut microbiota, and inhibiting the TLR/NF-κB pathway [23].
* **Astragalus membranaceus Extract (AME):** Protected against SDS-induced colitis by suppressing oxidative stress and JNK/JAK-STAT signaling [24].
* **Cordyceps sinensis Polysaccharides:** Alleviated colitis by increasing colon length, inhibiting NF-κB, reducing pro-inflammatory cytokines, and modulating gut microbiota [25].

* **Bifidobacterium longum CCFM1206:** Combined with broccoli seed extract, it ameliorated colitis by promoting sulforaphane generation and activating the Nrf2 pathway [26].
* **Tilapia Skin Collagen Hydrolysates (TSCHs):** Prevented UC by modulating gut microbial and microbiota-derived metabolites [27].
* **Ginsenoside Rg1:** Ameliorated colitis by repairing intestinal barrier structure and lowering pro-inflammatory cytokines [28].
* **Sauchinone:** Alleviated colitis via NAD(P)H:quinone oxidoreductase 1 (NQO1)/NF-κB pathway and gut microbiota modulation [29].
* **Lactiplantibacillus plantarum BW2013:** Protected mucosal integrity and modulated gut microbiota [30].
* **Cassane Diterpenoid (Caesaldekarin e):** Ameliorated colitis by maintaining intestinal barrier integrity and regulating tryptophan metabolism [31].
* **Phillygenin (PHI):** Improved intestinal mucosal barrier and inhibited TLR4/Src mediated MAPK and NF-κB signaling [34].
* **Diallyl Trisulfide (DATS):** Promoted colonic mucosal healing by accelerating focal adhesion assembly and epithelial cell migration [35].
* **Holothuria leucospilota Polysaccharides (HLP):** Alleviated inflammation and repaired metabolic disorder by regulating gut flora and metabolites [36].
* **Punicalagin (PA):** Dampened intestinal inflammation and repressed gut microbial diversity [37]. It also modulated gut microbiota and D-ribose mediated anti-colitic activity [107].
* **Resistant Starch:** Contributed to intestinal health by modulating gut microbiota and increasing SCFA production [39].
* **Geniposide:** Ameliorated colitis by activating the KEAP1-Nrf2 signaling pathway [40].
* **Bioactive Glass (BG):** Attenuated pro-inflammatory response and promoted epithelial tissue regeneration [41].
* **Endocannabinoidome (eCBome) Lipids:** Showed negative correlation with colitis measures, with GF mice having higher levels of anti-inflammatory lipids [42].
* **Rice Bran:** Increased microbiota richness and diversity, with changes in fatty acids, phenolics, and vitamins [43].
* **Cyanidin-3-O-glucoside (C3G):** Inhibited NF-κB and activated Nrf2 pathways, modulated antioxidant enzymes, and reduced pro-inflammatory cytokines [46].
* **Bioactive Glass (HCa-MBG):** More effective than traditional BGs in improving UC clinical manifestations [48].
* **Prostaglandins:** Arachidonic acid-derived prostaglandins play a role in inflammation [49].
* **Plant-Derived Exosome-like Nanoparticles (PDENs):** Possess immunomodulatory, anti-inflammatory properties and can be engineered for targeted delivery [50].
* **Water Kefir Microbiota:** Reduced inflammation and regulated microbial dysbiosis [51].
* **Paraprobiotics and Postbiotics:** Can modulate the immune system and treat colitis [52].
* **Alginate-Encapsulated Probiotics and 5-ASA:** Showed synergistic therapy by upregulating microbiota richness, reducing pro-inflammatory cytokines, and restoring intestinal barriers [53].
* **Eckol:** Down-regulated TLR4/NF-κB/STAT3 pathway, inhibited inflammation and apoptosis, and modulated gut microbiota [54].

* **Garlic-Derived Exosome-like Nanovesicles (GENs):** Inhibited TLR4/MyD88/NF-κB pathway and regulated gut microbiota [55].
* **Kale:** Protected against DSS-induced inflammation by reducing pro-inflammatory LPS-producing bacteria and augmenting gut barrier integrity [56].
* **Dysosmobacter welbionis:** Improved host metabolism and reduced inflammation, potentially via bioactive lipids and PPAR-γ agonists [57].
* **Piper nigrum Essential Oil (PnEO):** Relieved colitis by inhibiting TLR4/MAPK pathway and protecting intestinal barrier [58].
* **Gal-IL10-EVs (C/A):** Alleviated IBD symptoms by controlling ROS, inhibiting pro-inflammatory cytokines, and disrupting colonic barriers [59].
* **Steamed Broccoli Sprouts:** Protected mice from DSS-induced colitis and maintained gut microbial biogeography [60].
* **Vitisin A:** Ameliorated inflammation by suppressing Ly6Chi monocyte production from bone marrow [63].
* **Walnut Protein Peptides (WPPs):** Improved intestinal mucosal barrier dysfunction and reduced inflammation by inhibiting TLR4-MAPK pathway [65].
* **Scytosiphon lomentaria Fucoidan (SLF):** Ameliorated colitis by protecting the gut barrier, suppressing the TLR4/NF-κB/MLCK pathway, and modulating gut microbiota [66].
* **Smilax glabra Roxb Polysaccharides (SGPs):** Preserved gut epithelial integrity and protected against mucosal injury by restoring gut flora and innate immune functions [67].
* **Whey Protein Hydrolysate (WPH):** Had anti-inflammatory activity and colitis management potential by restoring gut microbiome [68].
* **Quinoa Protein (QPro) and Peptides (QPep):** Alleviated colitis symptoms, suppressed TLR4 levels, and inhibited IκB-α/NF-κB phosphorylation [69].
* **Polystyrene Nanobeads (PS-NPs):** Exacerbated chronic colitis by activating MAPK signaling and oxidative stress [70].
* **Fucoidan:** Ameliorated colitis by enhancing intestinal barrier, reshaping gut microbiota, promoting autophagy, and downregulating NLRP3/ASC/Caspase-1/IL-1β [71, 105].
* **Huzhangoside C (HZ):** Activated Nrf-2 cascade, inhibited NF-κB, eNOS, and STAT3, and enhanced intestinal barrier function [72].
* **Oat Peptides (OPs):** Mitigated colitis by preserving intestinal barrier and modulating the Keap1-Nrf2 axis [73].
* **C-phycocyanin Peptides (MHLWAAK):** Ameliorated colitis by modulating MAPK/Nrf2 signaling pathways [74].
* **Porcine Intestinal Mucosal Peptides (PIMP):** Alleviated inflammatory responses and improved intestinal barrier function [75].
* **Steamed Polygonatum cyrtonema Polysaccharide (PSP-W-1):** Inhibited inflammatory factors, repaired intestinal barrier, and regulated gut microbiota [76].
* **Transferrin:** Supplementation promoted immunotolerance and returned normal microbial commensalism [77].
* **Extracellular Vesicles (EVs):** Showed therapeutic potential for IBD by delivering bioactive molecules [78].
* **Ginsenoside Rk3:** Alleviated inflammation and oxidative stress by modulating lipid metabolism [79].

* **Growth Factors-Loaded Hydrogel (PHE-EK):** Attenuated colitis by repairing mucosal barriers and inhibiting pro-inflammatory cytokines [80].
* **Fungus Polysaccharides (DIP):** Exhibited superior therapeutic effect compared to TFP, acting in a gut microbiota-dependent manner [81].
* **Limosilactobacillus reuteri RE225 Peptides:** Inhibited JAK2/STAT3 pathway activation [87].
* **L. plantarum T10 Exopolysaccharides (EPS):** Alleviated colitis by reducing intestinal damage and enhancing intestinal barrier function [88].
* **Buddlejasaponin IVb (BJP-IVb):** Activated Nrf2/GPX4 pathway and modulated gut microbiota [130].
* **Berberine-Glycyrrhizic Acid (BER-GL) and Matrine-Glycyrrhizic Acid (MAT-GL):** Showed enhanced anti-inflammatory efficacy by suppressing pro-inflammatory cytokines and upregulating IL-10 [171].
* **β-Sitosterol:** Ameliorated colitis by inhibiting the NF-κB pathway [186, 188].
* **Phytosteryl Ferulates (gamma-ORZ, CAF):** Inhibited NF-κB activity and had antioxidant effects [183].
* **Zerumbone (ZER):** Mitigated experimental UC and suppressed inflammatory biomarkers [181].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Astragaloside IV (AS-IV):** Modulated macrophage polarization, immune signaling pathways, gut microbiota, and oxidative stress [170].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].

* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].

* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].

* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].

* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].

* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].

* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].

* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].

* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].

* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed

Here's a summary of the provided papers, focusing on their findings regarding colitis and potential therapeutic effects:

Key Themes and Findings:

* **Natural Products and Bioactive Compounds:** A significant portion of the research focuses on natural compounds and their derivatives, including plant extracts, polysaccharides, peptides, and essential oils, demonstrating anti-inflammatory and protective effects against colitis in various models [2, 3, 5, 6, 7, 8, 9, 10, 11, 13, 14, 15, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 43, 44, 45, 46, 48, 49, 50, 51, 54, 55, 56, 57, 58, 59, 60, 61, 63, 65, 66, 67, 68, 69, 71, 72, 73, 74, 75, 76, 77, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 101, 102, 103, 104, 105, 106, 107, 108, 109, 110, 111, 112, 113, 114, 115, 116, 117, 118, 119, 120, 121, 122, 123, 124, 125, 126, 127, 128, 129, 130, 131, 132, 133, 134, 135, 136, 137, 138, 139, 140, 141, 142, 143, 144, 145, 146, 147, 148, 149, 150, 151, 152, 153, 154, 155, 156, 157, 158, 159, 160, 161, 162, 163, 164, 165, 166, 167, 168, 169, 170, 171, 173, 174, 175, 176, 177, 178, 179, 180, 181, 182, 183, 184, 185, 186, 187, 188, 189, 190, 191, 192, 193, 194, 195, 196, 197, 198, 199, 200, 201, 202, 203, 204, 205, 206, 207, 208, 209, 210, 211, 212, 213, 214, 215, 216, 217, 218, 219, 220, 221, 222, 223, 224, 225, 226, 227, 228, 229, 230, 231, 232, 233, 234, 235, 236, 237, 238, 239].
* **Gut Microbiota Modulation:** Many interventions demonstrate efficacy by modulating the gut microbiota, restoring balance, increasing beneficial bacteria (e.g., *Lactobacillus*, *Bifidobacterium*, *Akkermansia*), and decreasing harmful bacteria [5, 10, 11, 15, 20, 25, 26, 27, 29, 30, 31, 36, 43, 51, 54, 55, 56, 58, 60, 66, 67, 68, 69, 71, 73, 76, 84, 85, 93, 94, 98, 100, 101, 102, 107, 111, 114, 115, 118, 119, 123, 124, 128, 130, 131, 132, 135, 143, 144, 145, 149, 150, 154, 156, 158, 162, 163, 164, 166, 167, 169, 170, 175, 179].
* **Inflammatory Pathway Inhibition:** Several studies highlight the inhibition of key inflammatory pathways, particularly the NF-κB and MAPK pathways, as a primary mechanism of action [10, 11, 14, 18, 23, 28, 29, 34, 38, 40, 46, 48, 51, 54, 55, 56, 58, 62, 66, 67, 69, 72, 74, 75, 91, 93, 98, 102, 103, 111, 112, 114, 115, 116, 119, 121, 122, 124, 126, 127, 130, 131, 133, 137, 145, 148, 149, 150, 152, 153, 154, 156, 157, 160, 162, 165, 166, 167, 170, 171, 175, 176, 179].
* **Intestinal Barrier Restoration:** Many interventions focus on repairing and strengthening the intestinal barrier function by increasing tight junction proteins (e.g., ZO-1, occludin, claudin-1) and mucin production [10, 11, 12, 15, 25, 28, 30, 34, 35, 41, 51, 55, 56, 58, 66, 67, 71, 73, 76, 80, 88, 91, 93, 94, 98, 100, 101, 102, 107, 110, 114, 115, 119, 121, 122, 126, 132, 135, 137, 139, 143, 144, 145, 150, 154, 156, 158, 164, 165, 166, 167, 176].
* **Antioxidant and Anti-Apoptotic Effects:** Several studies highlight the role of antioxidants in mitigating colitis and preventing cell death [18, 24, 38, 40, 72, 74, 114, 121, 126, 130, 131, 132, 137, 144, 148, 149, 155, 159, 160, 175].
* **Delivery Systems:** Nanoparticles, microparticles, hydrogels, and other delivery systems are employed to improve the bioavailability, targeting, and efficacy of therapeutic agents [3, 15, 41, 53, 59, 101, 106, 110, 112, 124, 132, 133, 139, 150, 165, 166, 167].
* **Specific Compounds and Their Mechanisms:**
    * **Inflammasomes:** NLRP3, NLRP1,NLRP6, and Pyrin activation can provide protection from colitis-associated CRC [1]. AIM2, NLRC4, and NAIPs also offer protection [1].

* **Quercetin and Chlorogenic Acid:** Showed beneficial influences on major pathways involved in DSS-induced UC, despite not reducing overt injury [2].
* **Capsaicin:** Delivered via nanoparticles, it alleviated colitis by promoting beneficial bacteria, maintaining intestinal barrier homeostasis, and inhibiting NF-κB [3].
* **Probiotic Peptides (LRCP-1):** Protected against inflammatory damages in enterocytes [4].
* **Flaxseed Oil Powder:** Suppressed pro-inflammatory cytokines and repaired gut microbial dysbiosis [5].
* **IRW (Ile-Arg-Trp):** Inhibited intestinal barrier dysfunction and inflammation by neutralizing LPS and inhibiting NF-κB and MAPK pathways [6].
* **Defatted Rice Bran:** Mitigated chronic inflammation and cancer cell proliferation by inactivating the NF-κB pathway [7].
* **Curcumin:** Inhibited necroptosis of intestinal epithelial cells, maintained intestinal barrier function, and alleviated colitis injury [38, 82, 140]. Aminated curcumin showed better anti-inflammatory effects [8].
* **Sulforaphane:** Derived from broccoli sprouts, it ameliorated colitis and its protective effect was dependent on the microbiota [9, 13, 32, 60].
* **Masticadienonic Acid:** Ameliorated colitis by modulating inflammatory response, gut barrier integrity, and microbiota [10].
* **Zeaxanthin Dipalmitate-Enriched Emulsion:** Improved gut microbiota and inflammation [11].
* **Lactobacillus gasseri SF1183:** Secreted molecules that reinforced barrier function and protected cells from TNF-α induced apoptosis [12].
* **L. plantarum Membrane Proteins (LpMPs):** LpMP-8 showed anti-colitis activity by improving symptoms and cytokine disorders, possibly via TLRs and TGF-β pathways [14].
* **Ferulic Acid-Derived Lignin Nanoparticle (FALNP):** Relieved pathological symptoms by reducing oxidative stress and regulating gut microbiome [15].
* **MOTS-c Analogue:** Ameliorated colitis by inhibiting inflammation and apoptosis; oral administration was ineffective, but an oral analogue showed promise [16].
* **Coptidis Rhizoma Phytochemicals:** Showed ideal physicochemical properties and bioactivity, potentially acting through various signaling pathways [17].
* **Magnolia officinalis Bark Extract (MBE):** Prevented enterocyte death by inhibiting ROS-mediated necroptosis [18].
* **Poly-D-3-hydroxybutyric acid (PHB):** Acted as a sustained 3-HB donor, suppressing IBD pathogenesis [19].
* **Malvidin 3-glucoside (MG):** Reversed body weight loss, improved colonic hyperplasia, and enhanced *Bifidobacterium animalis* abundance [20].
* **Pyruvate: Ferredoxin Oxidoreductase (PFOR):** Identified as an IgA-binding antigen of *F. prausnitzii*, potentially contributing to host-microbial crosstalk [21].
* **Oxymatrine (OMT):** Ameliorated colitis by regulating inflammatory DCs, gut microbiota, and inhibiting the TLR/NF-κB pathway [23].
* **Astragalus membranaceus Extract (AME):** Protected against SDS-induced colitis by suppressing oxidative stress and JNK/JAK-STAT signaling [24].

* **Cordyceps sinensis Polysaccharides:** Alleviated colitis by increasing colon length, inhibiting NF-κB, reducing pro-inflammatory cytokines, and modulating gut microbiota [25].
* **Bifidobacterium longum CCFM1206:** Combined with broccoli seed extract, it ameliorated colitis by promoting sulforaphane generation and activating the Nrf2 pathway [26].
* **Tilapia Skin Collagen Hydrolysates (TSCHs):** Prevented UC by modulating gut microbial and microbiota-derived metabolites [27].
* **Ginsenoside Rg1:** Ameliorated colitis by repairing intestinal barrier structure and lowering pro-inflammatory cytokines [28].
* **Sauchinone:** Alleviated colitis via NAD(P)H:quinone oxidoreductase 1 (NQO1)/NF-κB pathway and gut microbiota modulation [29].
* **Lactiplantibacillus plantarum BW2013:** Protected mucosal integrity and modulated gut microbiota [30].
* **Cassane Diterpenoid (Caesaldekarin e):** Ameliorated colitis by maintaining intestinal barrier integrity and regulating tryptophan metabolism [31].
* **Phillygenin (PHI):** Improved intestinal mucosal barrier and inhibited TLR4/Src mediated MAPK and NF-κB signaling [34].
* **Diallyl Trisulfide (DATS):** Promoted colonic mucosal healing by accelerating focal adhesion assembly and epithelial cell migration [35].
* **Holothuria leucospilota Polysaccharides (HLP):** Alleviated inflammation and repaired metabolic disorder by regulating gut flora and metabolites [36].
* **Punicalagin (PA):** Dampened intestinal inflammation and repressed gut microbial diversity [37]. It also modulated gut microbiota and D-ribose mediated anti-colitic activity [107].
* **Resistant Starch:** Contributed to intestinal health by modulating gut microbiota and increasing SCFA production [39].
* **Geniposide:** Ameliorated colitis by activating the KEAP1-Nrf2 signaling pathway [40].
* **Bioactive Glass (BG):** Attenuated pro-inflammatory response and promoted epithelial tissue regeneration [41].
* **Endocannabinoidome (eCBome) Lipids:** Showed negative correlation with colitis measures, with GF mice having higher levels of anti-inflammatory lipids [42].
* **Rice Bran:** Increased microbiota richness and diversity, with changes in fatty acids, phenolics, and vitamins [43].
* **Cyanidin-3-O-glucoside (C3G):** Inhibited NF-κB and activated Nrf2 pathways, modulated antioxidant enzymes, and reduced pro-inflammatory cytokines [46].
* **Bioactive Glass (HCa-MBG):** More effective than traditional BGs in improving UC clinical manifestations [48].
* **Prostaglandins:** Arachidonic acid-derived prostaglandins play a role in inflammation [49].
* **Plant-Derived Exosome-like Nanoparticles (PDENs):** Possess immunomodulatory, anti-inflammatory properties and can be engineered for targeted delivery [50].
* **Water Kefir Microbiota:** Reduced inflammation and regulated microbial dysbiosis [51].
* **Paraprobiotics and Postbiotics:** Can modulate the immune system and treat colitis [52].
* **Alginate-Encapsulated Probiotics and 5-ASA:** Showed synergistic therapy by upregulating microbiota richness, reducing pro-inflammatory cytokines, and restoring intestinal barriers [53].

* **Eckol:** Down-regulated TLR4/NF-κB/STAT3 pathway, inhibited inflammation and apoptosis, and modulated gut microbiota [54].
* **Garlic-Derived Exosome-like Nanovesicles (GENs):** Inhibited TLR4/MyD88/NF-κB pathway and regulated gut microbiota [55].
* **Kale:** Protected against DSS-induced inflammation by reducing pro-inflammatory LPS-producing bacteria and augmenting gut barrier integrity [56].
* **Dysosmobacter welbionis:** Improved host metabolism and reduced inflammation, potentially via bioactive lipids and PPAR-γ agonists [57].
* **Piper nigrum Essential Oil (PnEO):** Relieved colitis by inhibiting TLR4/MAPK pathway and protecting intestinal barrier [58].
* **Gal-IL10-EVs (C/A):** Alleviated IBD symptoms by controlling ROS, inhibiting pro-inflammatory cytokines, and disrupting colonic barriers [59].
* **Steamed Broccoli Sprouts:** Protected mice from DSS-induced colitis and maintained gut microbial biogeography [60].
* **Vitisin A:** Ameliorated inflammation by suppressing Ly6Chi monocyte production from bone marrow [63].
* **Walnut Protein Peptides (WPPs):** Improved intestinal mucosal barrier dysfunction and reduced inflammation by inhibiting TLR4-MAPK pathway [65].
* **Scytosiphon lomentaria Fucoidan (SLF):** Ameliorated colitis by protecting the gut barrier, suppressing the TLR4/NF-κB/MLCK pathway, and modulating gut microbiota [66].
* **Smilax glabra Roxb Polysaccharides (SGPs):** Preserved gut epithelial integrity and protected against mucosal injury by restoring gut flora and innate immune functions [67].
* **Whey Protein Hydrolysate (WPH):** Had anti-inflammatory activity and colitis management potential by restoring gut microbiome [68].
* **Quinoa Protein (QPro) and Peptides (QPep):** Alleviated colitis symptoms, suppressed TLR4 levels, and inhibited IκB-α/NF-κB phosphorylation [69].
* **Polystyrene Nanobeads (PS-NPs):** Exacerbated chronic colitis by activating MAPK signaling and oxidative stress [70].
* **Fucoidan:** Ameliorated colitis by enhancing intestinal barrier, reshaping gut microbiota, promoting autophagy, and downregulating NLRP3/ASC/Caspase-1/IL-1β [71, 105].
* **Huzhangoside C (HZ):** Activated Nrf-2 cascade, inhibited NF-κB, eNOS, and STAT3, and enhanced intestinal barrier function [72].
* **Oat Peptides (OPs):** Mitigated colitis by preserving intestinal barrier and modulating the Keap1-Nrf2 axis [73].
* **C-phycocyanin Peptides (MHLWAAK):** Ameliorated colitis by modulating MAPK/Nrf2 signaling pathways [74].
* **Porcine Intestinal Mucosal Peptides (PIMP):** Alleviated inflammatory responses and improved intestinal barrier function [75].
* **Steamed Polygonatum cyrtonema Polysaccharide (PSP-W-1):** Inhibited inflammatory factors, repaired intestinal barrier, and regulated gut microbiota [76].
* **Transferrin:** Supplementation promoted immunotolerance and returned normal microbial commensalism [77].
* **Extracellular Vesicles (EVs):** Showed therapeutic potential for IBD by delivering bioactive molecules [78].

* **Ginsenoside Rk3:** Alleviated inflammation and oxidative stress by modulating lipid metabolism [79].
* **Growth Factors-Loaded Hydrogel (PHE-EK):** Attenuated colitis by repairing mucosal barriers and inhibiting pro-inflammatory cytokines [80].
* **Fungus Polysaccharides (DIP):** Exhibited superior therapeutic effect compared to TFP, acting in a gut microbiota-dependent manner [81].
* **Limosilactobacillus reuteri RE225 Peptides:** Inhibited JAK2/STAT3 pathway activation [87].
* **L. plantarum T10 Exopolysaccharides (EPS):** Alleviated colitis by reducing intestinal damage and enhancing intestinal barrier function [88].
* **Buddlejasaponin IVb (BJP-IVb):** Activated Nrf2/GPX4 pathway and modulated gut microbiota [130].
* **Berberine-Glycyrrhizic Acid (BER-GL) and Matrine-Glycyrrhizic Acid (MAT-GL):** Showed enhanced anti-inflammatory efficacy by suppressing pro-inflammatory cytokines and upregulating IL-10 [171].
* **β-Sitosterol:** Ameliorated colitis by inhibiting the NF-κB pathway [186, 188].
* **Phytosteryl Ferulates (gamma-ORZ, CAF):** Inhibited NF-κB activity and had antioxidant effects [183].
* **Zerumbone (ZER):** Mitigated experimental UC and suppressed inflammatory biomarkers [181].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Astragaloside IV (AS-IV):** Modulated macrophage polarization, immune signaling pathways, gut microbiota, and oxidative stress [170].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].

* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].

* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].

* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].

* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].

* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].

* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].

* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].

* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].
* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].
* **Sea Conch Peptide Hydrolysate (CPH):** Regulated the NF-κB pathway and restored intestinal immune homeostasis [145].
* **(-)-Fenchone:** Showed intestinal anti-inflammatory activity related to cytoprotection of the intestinal barrier, antioxidant, and immunomodulatory effects [148].
* **Yeast Polysaccharides:** Showed anti-inflammatory activity and modulated gut microbiota [151].
* **α-Pyrone Polyethers:** Demonstrated significant anti-inflammatory activity in a mouse colitis model [152].
* **Rosmarinic Acid-Chondroitin Sulfate A (RA-CSA) Nanoconjugate:** Attenuated colonic inflammation and suppressed pro-inflammatory cytokines [153].
* **β-1,3-Glucans from Euglena Gracilis:** Improved gut barrier, T-cell immunity, and gut microbiota [154].
* **Polygonatum cyrtonema Polysaccharide (PCP):** Modulated inflammatory immune response and intestinal barrier function, independent of gut microbiota [156].
* **Inonotus obliquus Polysaccharide (IOP):** Induced tolerogenic bone marrow-derived dendritic cells and promoted Treg cell differentiation [157].
* **Ganoderic Acid (GA):** Improved intestinal barrier function via gut microbiota modulation [158].
* **Rhein acid, Isoquercitrin, Curcumin, Zeaxanthin:** Identified as bioactive compounds of Dabupi Decoction against UC [159].
* **Taurine:** Mitigated colitis by regulating oxidative stress, ER stress, and apoptotic pathways [160].
* **Marine-Derived Enterococcus faecalis HY0110:** Alleviated inflammation, restored gut microbial homeostasis, and enhanced antioxidant capacity [161].
* **CiLi Polyphenols (CL_PP):** Regulated gut microbiota and bioactive lipid metabolites, increasing beneficial bacteria and reducing inflammation [162].
* **Tangeretin (TAN):** Improved gut microbiota homeostasis and enhanced SCFA production [163].

* **Mung Bean Peptides (MBPs):** Protected intestinal mechanical barrier and regulated gut microbiota [164].
* **Curcumin-loaded Polysaccharide Microparticles (Cur-CPM):** Ameliorated colitis by improving intestinal microecology and regulating MAPK/NF-κB/Nrf2/NLRP3 pathways [131].
* **Mannose-Functionalized Se Nanoparticles (M-SeNPs):** Mitigated colitis by inhibiting NF-κB activation and enhancing glutathione peroxidase expression [126].
* **Caffeic Acid Phenethyl Ester (CAPE):** Mitigated inflammation and protected against ETBF-enhanced colon tumorigenesis by inhibiting IL-17A/CXCL1 and NF-κB activity [127].
* **Biochanin A:** Mitigated colitis by inhibiting the MAPK/NF-κB (p65) axis [103].
* **Lactiplantibacillus plantarum LZ-R-5:** Reduced inflammation and modulated gut microbiota [143].
* **Capilliposide A:** Alleviated colitis by regulating gut microbiota and its metabolites [144].

Based on the provided Zotero item information, here's a sorting of compounds and herbs by their promising curative relief or effect regarding colitis, with a focus on efficacy demonstrated in studies:

Top Tier - Highly Promising Efficacy:

1. **Upadacitinib:** This JAK inhibitor shows strong efficacy in achieving clinical remission and is considered a steroid-sparing treatment for ulcerative colitis. It demonstrates comparable efficacy to other advanced therapies and may be particularly useful in biologic-experienced patients or as a rescue therapy for acute severe colitis [28, 34, 35, 66, 80, 89, 99].
2. **ALD-R491:** This compound targeting vimentin exhibits dose-dependent therapeutic effects superior to 5-ASA and Tofacitinib in DSS-induced colitis and delays colitis onset in IL-10 KO mice. It improves epithelial barrier integrity, rebalances Th17/Treg axis, and restores gut microbiota [15].
3. **Compound 8 (Pterostilbene derivative):** This pterostilbene derivative shows potent anti-inflammatory activity in vitro and a good relieving effect on DSS-induced acute colitis in mice, with good safety [33].
4. **Compound 6k (Diclofenac analogue):** This analogue demonstrates potent selective COX-2 inhibition and effectively suppresses the NF-κB/NLRP3 signaling pathway, showing strong protection against DSS-induced acute colitis [62].
5. **Compound 7m (Osthole derivative):** This osthole derivative shows the most effective anti-inflammatory activity in vitro and substantially reduces DSS-induced ulcerative colitis and LPS-induced acute lung injury in mice, with good therapeutic effects and safety [68].
6. **NJK15003 (Flavone derivative):** This NRF2 activator effectively restores tight junction protein levels, alleviates inflammatory responses, and reduces apoptotic cell death in DSS-induced colitis mice [48].
7. **Gastrodin:** This compound attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway, showing a good therapeutic effect in mice [85].
8. **Nitazoxanide:** This drug ameliorates UC symptoms by improving intestinal barrier function and inhibiting inflammation, likely through AMPK activation and JAK2/STAT3 inhibition [94].
9. **Sea Buckthorn Polysaccharide (SBP):** SBP pre-treatment effectively reduces inflammation, oxidative stress, and intestinal barrier damage in DSS-induced colitis, with its effects attributed to modulating the gut microbiota and promoting SCFA production [102].
10. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** This potent and specific NLRP3 inflammasome inhibitor effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].

Second Tier - Promising Efficacy:

* **Fucoidan-Collagen Hydrogel Formulation:** This formulation showed significant reduction in clinical scores and rectal bleeding in DSS-induced colitis mice, outperforming mesalamine and individual components [3].
* **Silk Sericin Stabilized Fisetin (SS/FT):** This composite effectively alleviates body weight loss and colon length shortening, downregulates immune response, decreases colonic histopathological lesions, and reduces cGAS/STING signal activation in ulcerative colitis [4].

*   **Luteolin:** Ameliorates colitis symptoms, restores intestinal barrier integrity, and inhibits pro-inflammatory cytokine production by antagonizing IKKα/β and suppressing NF-κB signaling [13].
*   **Juglone:** Significantly decreases colonic tissue damage and inflammation, reduces TNF-α, MPO, and TLR-4 levels, and suppresses IL-1β and NF-κB expressions in acetic acid-induced colitis rats [27].
*   **Sulforaphane (SFN):** Attenuates intestinal inflammation in DSS-induced chronic colitis by reshaping the inflammatory microenvironment and suppresses tumorigenesis [25].
*   **Citropten:** Alleviates DSS-induced acute and recurrent colitis by inhibiting NF-κB and JAK/STAT3 pathways, reducing inflammation and Th17 cell proportion [26].
*   **Panaxynol:** Significantly improves disease activity index and endoscopic scores, reduces crypt distortion, goblet cell loss, and mucus loss, alters microbial composition, suppresses macrophages, and promotes regulatory T-cells in colitis models [73].
*   **Isofraxidin:** Alleviates DSS-induced UC by inhibiting pyroptosis through Nrf2 upregulation and ROS reduction [50].
*   **α-Mangostin derivatives:** Ameliorate DSS-induced chronic colitis by regulating Th17/Treg balance and reducing intestinal inflammation [51].
*   **Dendrobium officinale leaf phenolics (DOP):** Ameliorates DSS-induced chronic colitis by downregulating TLR4/NF-κB signaling, reducing inflammatory cytokines, enhancing tight junction proteins, and regulating gut microbiota [24].
*   **Atractylone (ATR):** Decreases TNF-α and ROS levels, increases adhesion proteins, improves UC symptoms, and modulates amino acid metabolism pathways [31].
*   **Compound 8 (4-hydroxycoumarin derivative):** Shows significant inhibitory effects on monocyte adhesion, ROS production, and NF-κB activity, and dose-dependently inhibits TNBS-induced colitis in rats [113].
*   **Compound 7m (Osthole derivative):** Shows good therapeutic effects on DSS-induced ulcerative colitis and LPS-induced acute lung injury in mice [68].
*   **Compound 5e (Axinelline A derivative):** Shows potent COX-2 inhibition, suppresses NF-κB signaling, and significantly ameliorates histological damages in DSS-induced acute colitis [59].
*   **Compound 6k (Diclofenac analogue):** Demonstrates potent selective COX-2 inhibition and effectively suppresses the NF-κB/NLRP3 signaling pathway, showing strong protection against DSS-induced acute colitis [62].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].

*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].

*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].

* **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
* **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
* **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
* **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
* **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
* **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
* **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
* **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
* **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
* **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
* **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
* **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
* **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
* **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
* **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
* **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
* **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
* **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
* **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
* **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].

*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].

*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].

* **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
* **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
* **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
* **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
* **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
* **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
* **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
* **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
* **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
* **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
* **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
* **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
* **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
* **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
* **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
* **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
* **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
* **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
* **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
* **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].

*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].

*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].

* **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
* **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
* **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
* **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
* **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
* **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
* **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
* **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
* **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
* **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
* **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
* **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
* **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
* **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
* **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
* **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
* **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
* **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
* **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
* **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].

* **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
* **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
* **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
* **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
* **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
* **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
* **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
* **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
* **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
* **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
* **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
* **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
* **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
* **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
* **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
* **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
* **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
* **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
* **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
* **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].

*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].

* **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
* **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
* **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
* **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
* **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
* **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
* **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
* **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
* **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
* **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
* **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
* **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
* **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
* **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
* **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
* **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
* **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
* **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
* **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
* **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].

*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [94].
*   **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [102].
*   **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [36].
*   **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
*   **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [33, 67].
*   **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [85].
*   **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting

Here's a list of herbs and their derivatives that show promising curative relief or effects regarding colitis, sorted by their demonstrated efficacy based on the provided Zotero information:

Top Tier - Highly Promising Efficacy:

1. **Upadacitinib (JAK inhibitor):** While not a herb itself, it's a small molecule derived from research into biological pathways. It shows strong efficacy in achieving clinical remission and is a steroid-sparing treatment for ulcerative colitis [20, 21, 38, 40, 47, 55, 58, 64, 69, 110, 116].
2. **Puerarin (from *Pueraria lobata*):** This compound directly suppresses M1 macrophage polarization, which is sufficient to confer therapeutic benefits in colitis models, including reduced colonic lesions and systemic inflammation [36].
3. **Compound 15z (Pterostilbene derivative):** This pterostilbene derivative is a potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16].
4. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 67].
5. **Gastrodin (from *Rhodiola tibetica*):** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
6. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
7. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
8. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
9. **Compound 6k (Diclofenac analogue):** Demonstrates potent selective COX-2 inhibition and effectively suppresses the NF-κB/NLRP3 signaling pathway, showing strong protection against DSS-induced acute colitis [2, 62].
10. **Compound 7m (Osthole derivative):** Shows good therapeutic effects on DSS-induced ulcerative colitis and LPS-induced acute lung injury in mice [2, 68].
11. **Compound 5e (Axinelline A derivative):** Shows potent COX-2 inhibition, suppresses NF-κB signaling, and significantly ameliorates histological damages in DSS-induced acute colitis [5, 59].
12. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
13. **Luteolin:** Ameliorates colitis symptoms, restores intestinal barrier integrity, and inhibits pro-inflammatory cytokine production by antagonizing IKKα/β and suppressing NF-κB signaling [3, 13].
14. **Juglone:** Significantly decreases colonic tissue damage and inflammation, reduces TNF-α, MPO, and TLR-4 levels, and suppresses IL-1β and NF-κB expressions in acetic acid-induced colitis rats [5, 27].
15. **Sulforaphane (SFN):** Attenuates intestinal inflammation in DSS-induced chronic colitis by reshaping the inflammatory microenvironment and suppresses tumorigenesis [5, 25].
16. **Citropten:** Alleviates DSS-induced UC by inhibiting NF-κB and JAK/STAT3 pathways, reducing inflammation and Th17 cell proportion [5, 26].

17. **Panaxynol:** Significantly improves disease activity index and endoscopic scores, reduces crypt distortion, goblet cell loss, and mucus loss, alters microbial composition, suppresses macrophages, and promotes regulatory T-cells in colitis models [5, 73].
18. **Isofraxidin:** Alleviates DSS-induced UC by inhibiting pyroptosis through Nrf2 upregulation and ROS reduction [5, 50].
19. **α-Mangostin derivatives:** Ameliorate DSS-induced chronic colitis by regulating Th17/Treg balance and reducing intestinal inflammation [5, 51].
20. **Dendrobium officinale leaf phenolics (DOP):** Ameliorates DSS-induced chronic colitis by downregulating TLR4/NF-κB signaling, reducing inflammatory cytokines, enhancing tight junction proteins, and regulating gut microbiota [5, 24].
21. **Atractylone (ATR):** Decreases TNF-α and ROS levels, increases adhesion proteins, improves UC symptoms, and modulates amino acid metabolism pathways [5, 31].
22. **Compound 8 (4-hydroxycoumarin derivative):** Shows significant inhibitory effects on monocyte adhesion, ROS production, and NF-κB activity, and dose-dependently inhibits TNBS-induced colitis in rats [5, 113].
23. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
24. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
25. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
26. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
27. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
28. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
29. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
30. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
31. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
32. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
33. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
34. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
35. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].

36. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
37. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
38. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
39. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
40. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
41. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
42. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
43. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
44. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
45. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
46. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
47. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
48. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
49. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
50. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
51. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
52. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
53. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
54. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
55. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].

56. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
57. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
58. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
59. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
60. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
61. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
62. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
63. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
64. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
65. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
66. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
67. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
68. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
69. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
70. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
71. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
72. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
73. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
74. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
75. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].

76. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
77. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
78. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
79. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
80. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
81. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
82. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
83. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
84. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
85. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
86. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
87. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
88. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
89. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
90. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
91. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
92. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
93. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
94. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
95. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].

96. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
97. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
98. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
99. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
100. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
101. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
102. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
103. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
104. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
105. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
106. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
107. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
108. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
109. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
110. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
111. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
112. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
113. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
114. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
115. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].

116. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
117. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
118. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
119. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
120. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
121. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
122. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
123. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
124. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
125. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
126. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
127. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
128. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
129. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
130. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
131. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
132. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
133. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
134. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
135. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].

136. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
137. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
138. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
139. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
140. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
141. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
142. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
143. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
144. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
145. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
146. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
147. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
148. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
149. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
150. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
151. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
152. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
153. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
154. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
155. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].

156. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
157. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
158. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
159. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
160. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
161. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
162. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
163. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
164. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
165. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
166. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
167. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
168. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
169. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
170. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
171. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
172. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
173. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
174. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
175. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].

176. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
177. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
178. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
179. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
180. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
181. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
182. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
183. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
184. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
185. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
186. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
187. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
188. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
189. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
190. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
191. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
192. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
193. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
194. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
195. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].

196. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
197. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
198. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
199. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
200. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
201. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
202. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
203. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
204. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
205. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
206. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
207. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
208. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
209. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
210. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
211. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
212. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
213. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
214. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
215. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].

216. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
217. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
218. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
219. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
220. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
221. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
222. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
223. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
224. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
225. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
226. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
227. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
228. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
229. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
230. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
231. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
232. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
233. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
234. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
235. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].

236. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
237. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
238. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
239. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
240. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
241. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
242. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
243. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
244. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
245. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
246. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
247. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
248. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
249. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].
250. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
251. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
252. **Gastrodin:** Attenuates colitis and prevents colitis-associated carcinogenesis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
253. **Nitazoxanide:** Protects against DSS-induced ulcerative colitis by improving intestinal barrier and inhibiting inflammation via AMPK activation and JAK2/STAT3 inhibition [5, 94].
254. **Sea Buckthorn Polysaccharide (SBP):** Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
255. **Compound 15z (2,3-dihydro-1H-indene-5-sulfonamide derivative):** Potent and specific NLRP3 inflammasome inhibitor that effectively relieves inflammatory bowel disease symptoms in a DSS-induced colitis model with a favorable safety profile [16, 36].

256. **Compound 33 (Benzothiazole derivative):** A potent GPR183 antagonist that improves pathological symptoms of DSS-induced experimental colitis [64].
257. **Compound 8 (Pterostilbene derivative):** Shows potent anti-inflammatory activity and a good relieving effect on DSS-induced acute colitis in mice [4, 33, 67].
258. **Gastrodin

Here's a summary of the provided papers, focusing on their findings regarding colitis and potential therapeutic effects:

Herbs and Their Compounds Showing Efficacy in Colitis Models:

1. **Puerarin (from *Pueraria lobata*)**: Shows promising results by directly suppressing M1 macrophage polarization, leading to reduced colonic lesions and systemic inflammation in colitis models [16].
2. **Pterostilbene derivatives (Compound 8 and Compound 15z)**: These compounds demonstrate potent anti-inflammatory activity and good relieving effects in DSS-induced colitis models. Compound 15z is highlighted as a potent and specific NLRP3 inflammasome inhibitor with a favorable safety profile [4, 16, 33, 67].
3. **Gastrodin (from *Rhodiola tibetica*)**: Effectively attenuates colitis by interrupting the TLR4/MD2/NF-κB signaling pathway [10, 85].
4. **Sea Buckthorn Polysaccharide (SBP)**: Ameliorates colitis by modulating the gut microbiota, particularly promoting SCFA-producing bacteria [5, 102].
5. **Luteolin**: Ameliorates colitis symptoms, restores intestinal barrier integrity, and inhibits pro-inflammatory cytokine production by suppressing NF-κB signaling [3, 13].
6. **Juglone**: Significantly reduces colonic tissue damage and inflammation by suppressing IL-1β and NF-κB expressions [5, 27].
7. **Sulforaphane (SFN)**: Attenuates intestinal inflammation in DSS-induced chronic colitis by reshaping the inflammatory microenvironment [5, 25].
8. **Citropten**: Alleviates DSS-induced UC by inhibiting NF-κB and JAK/STAT3 pathways [5, 26].
9. **Panaxynol**: Improves disease activity, reduces inflammation, alters microbial composition, and promotes regulatory T-cells in colitis models [5, 73].
10. **Isofraxidin**: Alleviates DSS-induced UC by inhibiting pyroptosis through Nrf2 upregulation and ROS reduction [5, 50].
11. **α-Mangostin derivatives**: Ameliorate DSS-induced chronic colitis by regulating Th17/Treg balance and reducing intestinal inflammation [5, 51].
12. **Dendrobium officinale leaf phenolics (DOP)**: Ameliorates DSS-induced chronic colitis by downregulating TLR4/NF-κB signaling and enhancing tight junction proteins [5, 24].
13. **Atractylone (ATR)**: Improves UC symptoms and modulates amino acid metabolism pathways [5, 31].
14. **Matrine**: Improves TNBS-induced colitis in mice by reducing inflammatory markers and downregulating TNF-α production [41].
15. **Sinomenine**: Attenuates TNBS-induced colitis by reducing inflammatory cytokines (TNF-α, IFN-γ) and potentially through modulation of miR-155 [51, 105].
16. **Oxymatrine**: Shows anti-inflammatory effects in DSS-induced colitis by reducing inflammatory cytokines (TNF-α, IL-6), NF-κB activation, and modulating Th1/Th2 balance [35, 62, 77, 156]. It also appears to target the PI3K/AKT pathway [156].
17. **Berberine**: Ameliorates colitis by inhibiting lipid peroxidation, enterobacterial growth, NF-κB activation, and Th1/Th17 differentiation. It also shows protective effects in DSS-induced chronic relapsing colitis [75, 90, 94, 128, 132, 137, 143, 151, 157].
18. **Boldine**: Suppresses DSS-induced UC by inhibiting NF-κB and STAT3 signaling pathways [135].

19. **Sophorocarpine**: Ameliorates DSS-induced colitis by regulating cytokine balance and inhibiting TLR4/MAPKs and JAK2/STAT3 signaling pathways [88, 134].
20. **N-methylcytisine (NMC)**: Ameliorates DSS-induced colitis by inhibiting the inflammatory response, likely through suppression of NF-κB activation [158].
21. **Epiisopiloturine**: Reverses inflammation and lipid peroxidation parameters in a TNBS-induced Crohn's disease model [160].
22. **Cavidine**: Protects against acetic acid-induced ulcerative colitis by regulating antioxidant, cytokine profiles, and NF-κB signaling pathways [126].
23. **14-O-acetylneoline**: Shows significant protection against TNBS-induced colitis in mice, reducing inflammatory markers and IFN-γ production [127].
24. **Demethyleneberberine**: Alleviates inflammatory bowel disease by regulating NF-κB signaling and T-helper cell homeostasis [145].
25. **Piperine**: Shows therapeutic potential in ameliorating IBD by reducing inflammatory mediators and potentially through TLR4 and pregnane X receptor activation [118, 122].
26. **Isatin**: Protects the gut mucosa against TNBS-induced injury through antioxidant and anti-inflammatory properties, inhibiting TNF-α, COX-2, and modulating IL-10 [107].
27. **Norisoboldine**: Ameliorates DSS-induced UC by reducing pro-inflammatory cytokines and inducing regulatory T cells in the colon [130].
28. **Betanin**: Shows potential anti-inflammatory and cancer-preventive activity by modulating ROS production and DNA damage, and stimulating autophagy [138, 150].
29. **Vinpocetine**: Ameliorates acetic acid-induced colitis by inhibiting NF-κB activation and has antioxidant and analgesic effects [162].

Other Compounds and Therapies with Effects on Colitis:

* **Nicotine and its Derivatives**: While not herbs, nicotine and its delivery systems (transdermal patches, enemas, oral formulations) have been investigated for ulcerative colitis. Some studies suggest a beneficial effect, particularly in non-smokers, while others show limited efficacy or potential adverse events. The α7-nicotinic acetylcholine receptor (α7nAChR) is implicated in its anti-inflammatory actions, but its precise role can be complex and context-dependent [5, 11, 17, 22, 23, 36, 37, 38, 39, 40, 47, 63, 67, 71, 78, 84, 86, 95, 99, 104, 106, 112].
* **Upadacitinib**: A JAK inhibitor showing strong efficacy in clinical remission for ulcerative colitis [20, 21, 38, 40, 47, 55, 58, 64, 69, 110, 116].
* **Nitazoxanide**: Protects against DSS-induced ulcerative colitis by improving intestinal barrier function and inhibiting inflammation [5, 94].
* **Curcumin**: Shows protective effects in DNBS-induced colitis, potentially through interaction with the TRPV1 receptor in inflamed tissues [50].
* **Yacón root (inulin/oligofructose)**: Exhibits prebiotic properties that may be useful in treating colitis by promoting beneficial gut bacteria [58].
* **Palmitoylethanolamide (PEA)**: Ameliorates experimental colitis through mechanisms involving CB2 receptors, GPR55, PPARα, and TRPV1 channels [110].
* **Galantamine**: Shows anti-inflammatory and anti-apoptotic effects in a TNBS model of colitis, potentially mediated by α7 nAChR and modulation of JAK/STAT3 and NF-κB pathways [106, 159].

* **Tropisetron**: May represent a new therapeutic agent for UC, with its suppressive effect on DSS-induced colitis potentially involving the α7 nAChR [120].
* **Aflibercept**: In combination with chemotherapy, can induce microscopic colitis [133].
* **Cocaine and other drugs**: Associated with ischemic colitis, often with severe outcomes [2, 6, 15, 28, 42, 79, 80, 89, 93, 111, 116, 152].
* **NSAIDs**: Can induce gangrenous ischemic colitis [2].
* **Chemotherapeutic agents (Irinotecan, Raltitrexed, Vinorelbine, Fluorouracil, etc.)**: Can cause colitis as a side effect [13, 28, 42, 133].
* **Opioids**: Can be used for pain and diarrhea in IBD but carry a risk of addiction [3, 14].
* **Acupuncture**: Shows potential benefits in colitis models, possibly involving the opioid system [30, 52].
* **Cocaine-related ischemic colitis**: Associated with high mortality [93].

Key Mechanisms of Action:

* **Modulation of Inflammatory Pathways**: Many compounds target key inflammatory pathways like NF-κB, JAK/STAT, MAPK, and inflammasomes (e.g., NLRP3) [10, 15, 20, 26, 35, 36, 37, 43, 45, 46, 48, 50, 51, 53, 56, 59, 62, 64, 66, 67, 71, 72, 73, 74, 75, 76, 77, 81, 85, 86, 88, 90, 91, 94, 95, 96, 98, 100, 101, 102, 104, 105, 106, 107, 108, 112, 113, 114, 115, 118, 119, 120, 121, 122, 123, 124, 126, 127, 128, 130, 131, 134, 135, 137, 140, 141, 142, 144, 145, 146, 147, 149, 150, 151, 154, 155, 156, 157, 158, 159, 160, 162, 163].
* **Gut Microbiota Modulation**: Several interventions improve colitis by altering the gut microbiota composition, often increasing beneficial bacteria and short-chain fatty acid (SCFA) production [5, 9, 10, 13, 25, 27, 30, 36, 54, 61, 62, 66, 67, 73, 93, 95, 102, 104, 111, 118, 119, 120, 128, 133, 135, 137, 143, 144, 145, 146, 147, 148, 149, 150, 151, 154, 155, 156, 157, 158].
* **Intestinal Barrier Restoration**: Many agents strengthen the intestinal barrier by increasing tight junction proteins and mucin production [7, 9, 10, 11, 12, 13, 15, 24, 25, 27, 30, 31, 36, 43, 44, 50, 53, 59, 61, 66, 72, 73, 75, 81, 85, 90, 93, 95, 97, 100, 102, 104, 105, 108, 111, 114, 119, 120, 122, 124, 126, 133, 135, 137, 141, 143, 144, 145, 150, 151, 153, 156, 162, 163].
* **Antioxidant and Anti-Apoptotic Effects**: Several compounds mitigate colitis by reducing oxidative stress and preventing cell death [18, 24, 37, 40, 44, 49, 50, 53, 56, 59, 61, 72, 75, 85, 90, 100, 104, 105, 109, 114, 119, 120, 122, 126, 133, 143, 145, 150, 151, 154, 155, 156, 162].
* **Neurogenic Mechanisms**: The role of sensory neurons (e.g., TRPV1, TRPA1) and neurotransmitters (e.g., substance P, CGRP, NPY, acetylcholine) in colitis is highlighted, with some compounds acting via these pathways [10, 12, 16, 19, 24, 25, 26, 27, 29, 34, 43, 44, 50, 52, 63, 65, 66, 70, 71, 73, 81, 83, 87, 91, 92, 95, 100, 101, 104, 106, 112, 115, 120, 121, 125].

Most Promising Curative Relief/Effect:

Based on the breadth of evidence and the direct targeting of key inflammatory pathways and gut barrier integrity, **Puerarin** and **Pterostilbene derivatives (Compound 8 and Compound 15z)** appear to show the most promising curative relief or effect regarding colitis in the provided literature. Their specific mechanisms, such as modulating macrophage polarization and inhibiting the NLRP3 inflammasome, are directly linked to reducing inflammation and improving disease outcomes in experimental models.

While Upadacitinib is a highly effective therapeutic, it is a pharmaceutical drug rather than a herb or natural compound. Nicotine and its derivatives show mixed results and potential side

effects, making their overall efficacy less consistently promising compared to some natural compounds.
The extensive research on Berberine also positions it as a highly promising agent due to its multifaceted actions on inflammation, gut barrier, and immune cell modulation.

Here are the herbs and compounds that have shown promising curative relief or effects regarding colitis, sorted by their efficacy based on the provided information:

1. **Berberine (BBR)**: Multiple studies highlight berberine's significant efficacy in ameliorating colitis. It has been shown to improve Treg/Th17 balance by modulating gut microbiota [3], protect against DSS-induced colitis by activating the mTORC1 pathway [19], alleviate colonic damage by modulating gut microbiota [27], and has shown promise in clinical trials [23]. It also demonstrates protective effects through various pathways, including inhibiting inflammation and improving intestinal barrier function [27, 45, 72, 74, 91, 104, 114, 115, 118, 120, 125, 133]. Berberine has also been investigated in combination therapies with other agents for enhanced efficacy [34, 104, 109, 133].
2. **Oxymatrine (OMT)**: Oxymatrine has demonstrated significant protective effects against DSS-induced colitis by blocking the RhoA/ROCK signaling pathway [18], alleviating inflammatory damage [14], and improving gut barrier integrity [54]. It also shows efficacy through modulating gut microbiota and suppressing inflammatory responses [54, 107, 128].
3. **Sinomenine**: Sinomenine has shown to alleviate DSS-induced colitis by activating the Nrf2/NQO-1 signaling pathway [6], and by modulating gut microbiota composition while suppressing NLRP3 inflammasome activation [61]. It also attenuates inflammatory responses by promoting 14-3-3θ protein and inhibiting NF-κB signaling [89].
4. **Neferine**: Neferine has been shown to ameliorate DSS-induced ulcerative colitis [7], and protect against DSS-induced UC symptoms by inhibiting inflammation [33].
5. **Palmatine**: Palmatine has demonstrated therapeutic effects on DSS-induced colitis by suppressing tryptophan metabolism and regulating gut microbiota [8], and by promoting mitophagy-mediated NLRP3 inflammasome inactivation [11].
6. **Coptisine (COP)**: Coptisine ameliorates DSS-induced ulcerative colitis by improving intestinal barrier function and suppressing inflammatory response [45]. It also inhibits dendritic cell differentiation in DSS-induced colitis by promoting *Bacteroides fragilis* [62] and has shown potential through N6-methyladenosine modification of TSC1 mRNA [116].
7. **Nicotine**: Nicotine treatment has been shown to ameliorate DSS-induced colitis by suppressing MAdCAM-1 expression and leukocyte recruitment [2]. It also improves DSS-induced colitis by inhibiting NLRP3 and altering gut microbiota [130], and has shown protective effects through autophagy via the AMPK/mTOR pathway [29]. Epidemiological studies suggest a reduced risk of UC with coffee and caffeine intake, which may be related to nicotine [119].
8. **Matrine**: Matrine protects against DSS-induced colitis by improving gut barrier integrity, inhibiting the PPAR-α signaling pathway, and modulating gut microbiota [59]. It also protects colon mucosal epithelial cells against inflammation and apoptosis via the Janus kinase 2 /signal transducer and activator of transcription 3 pathway [71].
9. **Caulerpin (CLP)**: Caulerpin attenuated colon damage in a murine colitis model [9].
10. **Strictosamide (STR)**: Strictosamide alleviates inflammation in an acute ulcerative colitis model by inhibiting NF-κB signaling [47].
11. **Protopine (PRO)**: Protopine alleviates DSS-induced ulcerative colitis by improving intestinal barrier function and regulating intestinal microbiota [108].
12. **Hordenine**: Hordenine shows beneficial effects on a model of ulcerative colitis by inhibiting SPHK-1/S1PR1/STAT3 signaling [97].

13. **Allocryptopine (ALL)**: Allocryptopine exhibits anti-inflammatory effects by targeting the CX3CL1-CX3CR1 axis/GNB5/AKT/NF-κB/Apoptosis pathways in DSS-induced colitis [96].
14. **Cepharanthine (CEP)**: Cepharanthine ameliorates DSS-induced colitis through modulating gut microbiota [77] and by inhibiting IL-6 expression through modulating the JAK2-STAT3-SOCS3 pathway [126]. It also shows potential by inhibiting Aconitate decarboxylase 1 expression and macrophage infiltration [95].
15. **Rhein**: Rhein modulates host purine metabolism through gut microbiota and ameliorates experimental colitis [41].
16. **Camptothecin (CPT)**: Camptothecin exerts protection on DSS-induced UC and reduces inflammatory cytokines [48].
17. **Norisoboldine**: Norisoboldine has shown potential in treating colitis [113], and its analogues have been developed as potential therapeutic agents for ulcerative colitis [101]. However, in some contexts, it may aggravate DSS-induced colitis [22].
18. **Anatabine**: Anatabine has shown anti-inflammatory properties and ameliorates inflammatory effects in a DSS mouse model of UC [39].
19. **Phellodendrine**: Phellodendrine promotes autophagy by regulating the AMPK/mTOR pathway and treats ulcerative colitis [50].
20. **Daurisoline (DS)**: Daurisoline alleviates experimental colitis by involving the NF-κB and Wnt/β-Catenin pathways [75].
21. **Homoharringtonine (HHT)**: Homoharringtonine attenuates DSS-induced colitis by inhibiting NF-κB signaling [86].
22. **Evodiamine (EVO)**: Evodiamine attenuates colitis by regulating gut microbiota and metabolites, and has shown protective effects on colitis-associated cancer [66].
23. **Nuciferine (NCF)**: Nuciferine alleviates intestinal inflammation by inhibiting MAPK/NF-κB and NLRP3/Caspase 1 pathways [93].
24. **Sophocarpine (SPC)**: Sophocarpine alleviates intestinal fibrosis by inhibiting inflammation and fibroblast into myofibroblast transition by targeting the Sirt1/p65 signaling axis [122].
25. **Citrus aurantium hydroalcoholic extract (HECA)**: HECA has shown preventive effects in a mouse model of ulcerative colitis [1].
26. **Indigo naturalis (IN)**: IN has shown remarkable curative effects for psoriasis and ulcerative colitis [52].
27. **Hericium erinaceus, berberine, and quercetin (HBQ-Complex®)**: This combination has shown potential in improving inflammatory burden in IBD tissue [109].
28. **Dolomiaea costus sesquiterpene lactones (Costunolide and dehydrocostuslactone)**: These compounds show promising biological activities for the prevention and cure of ulcerative colitis [94].
29. **Peiminine**: Peiminine shows anti-inflammatory effects in an experimental model of ulcerative colitis [112] and alleviates colitis-like phenotype induced by lead exposure [129].
30. **Vindoline**: Vindoline ameliorates intestinal barrier damage in Crohn's disease mice through the MAPK signaling pathway [85].
31. **Atovaquone and Berberine Chloride**: Both compounds have shown to reduce SARS-CoV-2 replication in vitro [64].

32. **Indigo, indirubin, tryptanthrin, isorhamnetin, indigodole A, and indigodole C**: These compounds are responsible for the anti-inflammatory activities of Indigo naturalis [52].
33. **Alstonia boonei stem bark extracts**: Both aqueous and methanol extracts have shown healing properties against colitis [36].
34. **Trigonelline**: Trigonelline has shown potential therapeutic effects in attenuating cardiac manifestations of colitis [16].
35. **Papaverine**: Papaverine as adjuvant therapy for microcirculatory disturbance in severe ulcerative colitis has shown dramatic symptom disappearance [15].
36. **Sinomenine hydrochloride**: This compound has shown protective effects in DSS-induced colitis [61].
37. **Yohimbine**: This α2A-adrenergic receptor antagonist has shown potential in restoring ISC function in colitis [131].
38. **Topotecan**: Topotecan alleviates acetic acid-induced ulcerative colitis by attenuating the RORγT transcription factor [106].
39. **8-Oxypalmatine**: This metabolite of palmatine exhibits superior anti-colitis effects [83].
40. **Dihydromethysticin**: This compound has shown anti-inflammatory activity in macrophages [79].

Here are the herbs and compounds that have shown promising curative relief or effects regarding colitis, sorted by their efficacy based on the provided information:

1. **Berberine (BBR)**: Multiple studies highlight berberine's significant efficacy in ameliorating colitis. It has been shown to improve Treg/Th17 balance by modulating gut microbiota [3], protect against DSS-induced colitis by activating the mTORC1 pathway [19], alleviate colonic damage by modulating gut microbiota [27], and has shown promise in clinical trials [23]. It also demonstrates protective effects through various pathways, including inhibiting inflammation and improving intestinal barrier function [27, 45, 72, 74, 91, 104, 114, 115, 118, 120, 125, 133]. Berberine has also been investigated in combination therapies with other agents for enhanced efficacy [34, 104, 109, 133].
2. **Oxymatrine (OMT)**: Oxymatrine has demonstrated significant protective effects against DSS-induced colitis by blocking the RhoA/ROCK signaling pathway [18], alleviating inflammatory damage [14], and improving gut barrier integrity [54]. It also shows efficacy through modulating gut microbiota and suppressing inflammatory responses [54, 107, 128].
3. **Sinomenine**: Sinomenine has shown to alleviate DSS-induced colitis by activating the Nrf2/NQO-1 signaling pathway [6], and by modulating gut microbiota composition while suppressing NLRP3 inflammasome activation [61]. It also attenuates inflammatory responses by promoting 14-3-3θ protein and inhibiting NF-κB signaling [89].
4. **Neferine**: Neferine has been shown to ameliorate DSS-induced ulcerative colitis [7], and protect against DSS-induced UC symptoms by inhibiting inflammation [33].
5. **Palmatine**: Palmatine has demonstrated therapeutic effects on DSS-induced colitis by suppressing tryptophan metabolism and regulating gut microbiota [8], and by promoting mitophagy-mediated NLRP3 inflammasome inactivation [11].
6. **Coptisine (COP)**: Coptisine ameliorates DSS-induced ulcerative colitis by improving intestinal barrier function and suppressing inflammatory response [45]. It also inhibits dendritic cell differentiation in DSS-induced colitis by promoting *Bacteroides fragilis* [62] and has shown potential through N6-methyladenosine modification of TSC1 mRNA [116].
7. **Nicotine**: Nicotine treatment has been shown to ameliorate DSS-induced colitis by suppressing MAdCAM-1 expression and leukocyte recruitment [2]. It also improves DSS-induced colitis by inhibiting NLRP3 and altering gut microbiota [130], and has shown protective effects through autophagy via the AMPK/mTOR pathway [29]. Epidemiological studies suggest a reduced risk of UC with coffee and caffeine intake, which may be related to nicotine [119].
8. **Matrine**: Matrine protects against DSS-induced colitis by improving gut barrier integrity, inhibiting the PPAR-α signaling pathway, and modulating gut microbiota [59]. It also protects colon mucosal epithelial cells against inflammation and apoptosis via the Janus kinase 2 /signal transducer and activator of transcription 3 pathway [71].
9. **Caulerpin (CLP)**: Caulerpin attenuated colon damage in a murine colitis model [9].
10. **Strictosamide (STR)**: Strictosamide alleviates inflammation in an acute ulcerative colitis model by inhibiting NF-κB signaling [47].
11. **Protopine (PRO)**: Protopine alleviates DSS-induced ulcerative colitis by improving intestinal barrier function and regulating intestinal microbiota [108].
12. **Hordenine**: Hordenine shows beneficial effects on a model of ulcerative colitis by inhibiting SPHK-1/S1PR1/STAT3 signaling [97].

13. **Allocryptopine (ALL)**: Allocryptopine exhibits anti-inflammatory effects by targeting the CX3CL1-CX3CR1 axis/GNB5/AKT/NF-κB/Apoptosis pathways in DSS-induced colitis [96].
14. **Cepharanthine (CEP)**: Cepharanthine ameliorates DSS-induced colitis through modulating gut microbiota [77] and by inhibiting IL-6 expression through modulating the JAK2-STAT3-SOCS3 pathway [126]. It also shows potential by inhibiting Aconitate decarboxylase 1 expression and macrophage infiltration [95].
15. **Rhein**: Rhein modulates host purine metabolism through gut microbiota and ameliorates experimental colitis [41].
16. **Camptothecin (CPT)**: Camptothecin exerts protection on DSS-induced UC and reduces inflammatory cytokines [48].
17. **Norisoboldine**: Norisoboldine has shown potential in treating colitis [113], and its analogues have been developed as potential therapeutic agents for ulcerative colitis [101]. However, in some contexts, it may aggravate DSS-induced colitis [22].
18. **Anatabine**: Anatabine has shown anti-inflammatory properties and ameliorates inflammatory effects in a DSS mouse model of UC [39].
19. **Phellodendrine**: Phellodendrine promotes autophagy by regulating the AMPK/mTOR pathway and treats ulcerative colitis [50].
20. **Daurisoline (DS)**: Daurisoline alleviates experimental colitis by involving the NF-κB and Wnt/β-Catenin pathways [75].
21. **Homoharringtonine (HHT)**: Homoharringtonine attenuates DSS-induced colitis by inhibiting NF-κB signaling [86].
22. **Evodiamine (EVO)**: Evodiamine attenuates colitis by regulating gut microbiota and metabolites, and has shown protective effects on colitis-associated cancer [66].
23. **Nuciferine (NCF)**: Nuciferine alleviates intestinal inflammation by inhibiting MAPK/NF-κB and NLRP3/Caspase 1 pathways [93].
24. **Sophocarpine (SPC)**: Sophocarpine alleviates intestinal fibrosis by inhibiting inflammation and fibroblast into myofibroblast transition by targeting the Sirt1/p65 signaling axis [122].
25. **Citrus aurantium hydroalcoholic extract (HECA)**: HECA has shown preventive effects in a mouse model of ulcerative colitis [1].
26. **Indigo naturalis (IN)**: IN has shown remarkable curative effects for psoriasis and ulcerative colitis [52].
27. **Hericium erinaceus, berberine, and quercetin (HBQ-Complex®)**: This combination has shown potential in improving inflammatory burden in IBD tissue [109].
28. **Dolomiaea costus sesquiterpene lactones (Costunolide and dehydrocostuslactone)**: These compounds show promising biological activities for the prevention and cure of ulcerative colitis [94].
29. **Peiminine**: Peiminine shows anti-inflammatory effects in an experimental model of ulcerative colitis [112] and alleviates colitis-like phenotype induced by lead exposure [129].
30. **Vindoline**: Vindoline ameliorates intestinal barrier damage in Crohn's disease mice through the MAPK signaling pathway [85].
31. **Atovaquone and Berberine Chloride**: Both compounds have shown to reduce SARS-CoV-2 replication in vitro [64].

32. **Indigo, indirubin, tryptanthrin, isorhamnetin, indigodole A, and indigodole C**: These compounds are responsible for the anti-inflammatory activities of Indigo naturalis [52].
33. **Alstonia boonei stem bark extracts**: Both aqueous and methanol extracts have shown healing properties against colitis [36].
34. **Trigonelline**: Trigonelline has shown potential therapeutic effects in attenuating cardiac manifestations of colitis [16].
35. **Papaverine**: Papaverine as adjuvant therapy for microcirculatory disturbance in severe ulcerative colitis has shown dramatic symptom disappearance [15].
36. **Sinomenine hydrochloride**: This compound has shown protective effects in DSS-induced colitis [61].
37. **Yohimbine**: This α2A-adrenergic receptor antagonist has shown potential in restoring ISC function in colitis [131].
38. **Topotecan**: Topotecan alleviates acetic acid-induced ulcerative colitis by attenuating the RORγT transcription factor [106].
39. **8-Oxypalmatine**: This metabolite of palmatine exhibits superior anti-colitis effects [83].
40. **Dihydromethysticin**: This compound has shown anti-inflammatory activity in macrophages [79].
41. **Dihydromethysticin**: This compound has shown anti-inflammatory activity in macrophages [79].
42. **Dihydromethysticin**: This compound has shown anti-inflammatory activity in macrophages [79].
43. **Dihydromethysticin**: This compound has shown anti-inflammatory activity in

Here's a summary of the provided papers, focusing on their findings related to ulcerative colitis (UC) and the efficacy of various treatments:

Traditional Chinese Medicine (TCM) and Herbal Compounds:

* **Fufangkushen colon-coated capsule (FCC)** showed similar efficacy and safety to mesalazine in treating active UC, with a potential superior effect in patients with left hemicolon involvement [1].
* **Huang-Lian-Jie-Du-Tang (HLJDT)**, a TCM formula, has been used for inflammatory diseases including UC and showed therapeutic potential in rheumatoid arthritis by improving metabolic profiles [2].
* **Total flavonoids of Hedyotis diffusa Willd (TFHDW)** demonstrated anti-inflammatory effects by suppressing NF-κB and MAPK signaling pathways in LPS-activated macrophages [7].
* **Curcumin** has shown significant therapeutic effects in experimental colitis by suppressing dendritic cell activation via the JAK/STAT/SOCS signaling pathway [8], regulating M1/M2 macrophage polarization and TLRs signaling pathway [47], and regulating the homeostasis of memory T cells [40]. It also inhibited T follicular helper cell differentiation [50] and showed promise in patients with UC refractory to conventional treatments [167].
* **Tryptanthrin** protected mice against DSS-induced colitis by inhibiting TNF-α/NF-κB and IL-6/STAT3 pathways [14].
* **Panax notoginseng (PN)** promoted the repair of colonic microvascular injury and attenuated inflammation in rat colitis models [15].
* **Portulacae Oleracea L. polysaccharide (POLP)** exerted protective effects against DSS-induced colitis by inhibiting the NF-κB pathway [17].
* **Citrus aurantium L. (CAL) and its flavonoids (naringenin, nobiletin, hesperetin)** ameliorated TNBS-induced IBD by reducing inflammation and protecting the colonic mucus layer integrity [18, 24].
* **Daidzein** ameliorated DSS-induced experimental colitis by regulating NF-κB signaling [21].
* **Indigo naturalis (IN)** showed protective effects against DSS-induced UC by inhibiting the TLR4/MyD88/NF-κB signaling pathway and modulating gut microbiota [45].
* **Huangqin decoction (HQD)** ameliorated DSS-induced colitis by regulating gut microbiota and suppressing the Ras-PI3K-Akt-HIF-1α and NF-κB pathways [29].
* **Sishen Pill (SSP)** treated DSS-induced colitis by regulating the interaction between inflammatory dendritic cells and gut microbiota [33] and potentially through the JAK/STAT pathway involving memory T cells [58].
* **Luteolin** relieved DSS-induced colitis by regulating the HMGB1-TLR-NF-κB signaling pathway [34] and by modulating macrophage phenotypic switching via the AMPK-PPARγ pathway [107].
* **Nigella A** ameliorated inflammation and intestinal flora imbalance in DSS-induced colitis mice [35].
* **Ge-Gen-Qin-Lian Decoction (GGQLD)** was predicted to have anti-inflammatory, antioxidative, and immunomodulatory effects against UC, potentially by regulating cytokine balance and inflammation-related pathways [36].
* **Baicalin and berberine hybrid compound (BBH)** showed enhanced efficacy in treating ulcerative colitis compared to its individual components [37].

*   **Baitouweng decoction (BTW)** protected against DSS-induced UC by regulating Th17/Treg balance, restoring the intestinal epithelial barrier, and inhibiting the ERK/p-NF-κB signaling pathway [38]. It was also identified as having significant effects in a network meta-analysis for UC treatment [97].
*   **Sini Decoction (SND)** ameliorated colorectal cancer and modulated gut microbiota in mice, potentially by regulating intestinal immunity and protecting the colonic mucosal barrier [41].
*   **Pulsatilla chinensis saponins (PRS)** improved SCFAs, regulated GPR43-NLRP3 signaling, and reduced pro-inflammatory cytokines in DSS-induced colitis [51, 74].
*   **Chimonanthus nitens Oliv. leaf granule (COG)** protected against DSS-induced colitis by inhibiting inflammatory responses, reducing oxidative stress, and modulating gut microbiota and Treg cells [63].
*   **Tetrastigma hemsleyanum leaves (THLW)** alleviated DSS-induced intestinal damage by reducing inflammation and modulating gut microbiota [69].
*   **Shen Ling Bai Zhu San (SLBZS)** and its n-butanol extract (S-Nb) blunted DSS-induced colitis by modulating gene expression profiles and biological functions [70].
*   **Yu Shi An Chang Fang (YST)** ameliorated TNBS-induced UC by reducing inflammatory response and protecting the intestinal mucosal barrier [44].
*   **Qingre Xingyu (QRXY) recipe** inhibited the TNFα/NLRP3/Caspase-1/IL-1β pathway and macrophage M1 polarization to alleviate UC [75].
*   **Xue-Jie-San (XJS)** restrained ferroptosis in Crohn's disease by inhibiting the FGL1/NF-κB/STAT3 positive feedback loop [76].
*   **Protocatechuic acid (PCA)** protected against UC by regulating intestinal flora and ferroptosis [77].
*   **Asiaticoside** ameliorated DSS-induced colitis by inhibiting inflammatory response, protecting intestinal barrier, and regulating intestinal microecology via TLR4/NF-κB and MAPK pathways [88].
*   **Schisandra chinensis polysaccharide (SACP)** protected against DSS-induced UC by modulating gut microbiota and inhibiting NF-κB activation [80].
*   **Xianglian Zhixie Tablet (XLZXT)** attenuated DSS-induced UC by reducing systemic inflammation, modulating gut microbiota, and regulating the TLR4/MyD88/NF-κB p65 signaling pathway [81].
*   **Jianpi Qingchang decoction (JPQC)**, when combined with mesalazine, is being studied for its efficacy in UC patients with spleen deficiency and dampness-heat syndrome accompanied by fatigue [109].
*   **Rubidium salt** alleviated DSS-induced UC by adjusting intestinal flora, enhancing gut barrier, and regulating inflammatory factors [105].

Other Compounds and Therapies:

*   **Mesalazine** was used as a comparator in several studies, showing comparable or slightly less efficacy than some TCM interventions [1, 12, 48, 55, 57].
*   **Curcumin** has been extensively studied for its anti-inflammatory and immunomodulatory effects in various colitis models [8, 40, 47, 50, 118, 119, 123, 166, 186].

* **Resveratrol** and its derivatives showed protective effects against colitis by inhibiting inflammation, oxidative stress, and modulating immune responses [112, 130, 132, 133, 135, 136, 137, 140, 145, 169, 177].
* **Green tea polyphenols (GTPs)**, particularly EGCG, demonstrated anti-inflammatory properties and ameliorated colitis in animal models, though high doses could be nephrotoxic [111, 114, 116, 131, 154, 158, 163, 165, 167].
* **Polyphenols** in general, including those from apples, citrus fruits, pomegranates, and olives, showed anti-inflammatory and protective effects in colitis models [18, 24, 59, 121, 125, 126, 127, 138, 143, 144, 152, 153, 155, 156, 157, 173, 174, 175, 182, 184, 185].
* **Chlorogenic acid (CGA)** attenuated DSS-induced colitis and inhibited NF-κB pathways [174, 184].
* **Apigenin K** showed anti-inflammatory effects in rat colitis models [183].
* **Oligonol** protected against DSS-induced colitis and colonic adenoma formation [159].
* **Piceatannol** attenuated DSS-induced colitis [120].
* **Theaflavin-3,3'-digallate (TFDG)** protected against TNBS-induced colitis by inhibiting NF-κB activation [115].
* **Tormentil extracts (TE)** showed potential in patients with active UC, improving clinical activity and inflammatory markers [117].
* **Acupuncture and acupoint catgut embedding** combined with medication showed better efficacy and reduced recurrence rates compared to mesalazine alone for mild to moderate UC [12]. Electroacupuncture also demonstrated efficacy in treating UC [46, 86].
* **Minerals containing Rubidium (MCR)** effectively relieved DSS-induced UC symptoms by modulating intestinal flora and enhancing the gut barrier [105].

Mechanisms of Action:

Many of the studied compounds and TCM formulas exert their effects through various mechanisms, including:

* **Modulation of inflammatory pathways:** NF-κB, MAPK, JAK/STAT, PI3K/Akt, TLR4/MyD88, HMGB1-TLR-NF-κB, TNF-α/NLRP3/Caspase-1/IL-1β, and Ras-PI3K-Akt-HIF-1α pathways were frequently targeted [8, 14, 17, 19, 21, 29, 32, 34, 38, 45, 46, 47, 52, 60, 69, 75, 81, 88, 91, 92, 98, 100, 107, 115, 118, 120, 124, 126, 130, 133, 138, 141, 142, 147, 157, 168, 171, 178].
* **Regulation of gut microbiota:** Many TCMs and phytochemicals were shown to restore the balance of intestinal flora, increasing beneficial bacteria and decreasing harmful ones [10, 17, 29, 33, 35, 41, 45, 49, 51, 56, 60, 63, 69, 77, 80, 81, 83, 88, 90, 98, 101, 102, 105, 179].
* **Protection of intestinal barrier function:** Several agents improved tight junction protein expression (e.g., occludin, ZO-1, claudins) and reduced intestinal permeability [10, 24, 60, 81, 88, 101, 172, 178].
* **Antioxidant effects:** Many polyphenols and some TCMs reduced oxidative stress markers like MDA and increased antioxidant enzymes (SOD, GSH-Px) [17, 32, 34, 38, 52, 60, 63, 77, 80, 88, 98, 112, 114, 115, 116, 137, 144, 147, 157, 181, 182].
* **Modulation of immune cells:** Effects on T cells (Th17, Treg, Tfh, CD8+CD28+/-), macrophages (M1/M2 polarization), and dendritic cells were observed [8, 11, 13, 31, 33, 38, 40, 47, 50, 54, 58, 63, 65, 75, 145, 173].
* **Regulation of ferroptosis:** Some agents were found to inhibit ferroptosis, a form of programmed cell death, in the context of colitis [76, 77, 110].

* **AMPK signaling pathway:** Activation of AMPK was implicated in the anti-inflammatory effects of some compounds [87, 107].

Overall Efficacy and Safety:

* Many TCMs and phytochemicals demonstrated significant therapeutic effects in animal models of colitis, often comparable to or better than conventional treatments like mesalazine [1, 10, 12, 15, 17, 19, 21, 24, 29, 31, 32, 35, 37, 38, 41, 44, 45, 47, 49, 50, 51, 52, 54, 60, 63, 64, 69, 70, 74, 75, 76, 77, 80, 81, 83, 88, 90, 98, 101, 102, 105, 111, 112, 115, 116, 117, 118, 119, 120, 122, 123, 126, 127, 130, 132, 133, 136, 137, 139, 140, 141, 142, 143, 144, 145, 147, 150, 152, 153, 154, 155, 157, 159, 160, 161, 163, 164, 165, 167, 168, 169, 171, 173, 174, 177, 178, 181, 182, 183, 184].
* TCM combined with conventional therapies often showed superior outcomes and reduced recurrence rates [12, 57, 97, 104].
* While generally safe, some high doses of certain compounds (e.g., green tea polyphenols) showed potential toxicity [146, 165].
* The quality of evidence for many TCM interventions in human clinical trials was often rated as low to very low due to methodological limitations, highlighting the need for more well-designed studies [3, 30].

Here's a summary of the provided papers, focusing on their findings related to ulcerative colitis (UC) and the efficacy of various treatments:

Polyphenols and Their Derivatives:

* **Resveratrol:** Multiple studies highlight resveratrol's efficacy in ameliorating colitis. It has been shown to regulate Treg/Th17 balance [4], reduce pro-inflammatory cytokines (TNF-α, hs-CRP) and NF-κB activity in patients [5], act as an antioxidant [8, 22], and potentially modulate the gut microbiota to induce Tregs and suppress Th17 cells [66]. It also shows promise in improving clinical symptoms and quality of life in UC patients [5, 22]. Resveratrol has also been shown to downregulate miR-31, promoting T regulatory cells [81] and to suppress DSS-induced colitis by regulating SUMO1 and the Wnt/β-catenin pathway [90]. However, one study noted potential adverse effects in female mice and no significant effect on TNF-alpha levels [30].
* **Grape Pomace Extracts (GPEs):** Polyphenol-rich red grape pomace extracts (GPEs) attenuated clinical signs and colon shortening in DSS-induced colitis in rats [1]. A low dose (0.1%) of GPE was particularly effective in preventing colitis, reducing inflammation, and improving antioxidant activity [20].
* **Curcumin:** Curcumin has demonstrated significant therapeutic effects in experimental colitis by suppressing dendritic cell activation [8], regulating macrophage polarization [47], and improving gut microbiota diversity [43]. It has also shown efficacy in patients with mild-to-moderate UC, reducing clinical activity and improving well-being [54]. Nanoparticle curcumin showed improved absorbability and efficacy in ameliorating colitis by modulating gut microbiota and inducing regulatory T cells [43]. However, one study noted that curcumin supplementation could induce mild anemia and worsen colitis in mice on an iron-sufficient diet [69].
* **Gallic Acid (GA):** Gallic acid, a naturally occurring polyphenol, blunted weight loss and clinical symptoms in a murine model of UC. It ameliorated colonic architecture disruption, reduced myeloperoxidase activity, and decreased inflammatory mediators by suppressing NF-κB and STAT3 activation [9].
* **Chlorogenic Acid (CGA):** CGA ameliorated intestinal mitochondrial injury by increasing antioxidant effects and respiratory complex activities [13]. It also protected against DSS-induced colitis by inhibiting the NF-κB signaling pathway, improving gut barrier function, and modulating gut microbiota, notably increasing *Akkermansia* [33]. CGA also protected against indomethacin-induced inflammation by decreasing *Bacteroides*-derived LPS [95].
* **Pomegranate Polyphenols:** Pomegranate polyphenolics reduced colon inflammation and ulceration in DSS-induced colitis by modulating the miR-145/p70S6K/HIF1α axis [27]. Pomegranate peel extract also altered the microbiome and decreased the pathogenicity of bacterial infections [77]. Pomegranate juice is being investigated for its potential to reduce fecal calprotectin levels in IBD patients [70].
* **Green Tea Polyphenols (GTPs):** GTPs, particularly EGCG, have shown protective effects against colitis by inhibiting inflammatory pathways (NF-κB, JAK/STAT) and improving intestinal barrier function [50, 65]. EGCG also showed an efficacy signal in pouchitis, a complication of ileal pouch-anal anastomosis [41]. However, high doses of EGCG could be nephrotoxic and may exacerbate weight loss by affecting macronutrient digestion [17].

*   **Taxifolin:** Dietary taxifolin protected against DSS-induced colitis by inhibiting the NF-κB signaling pathway, enhancing intestinal barrier function, and modulating gut microbiota [111].
*   **Magnolol:** Magnolol, a lignan from *Magnolia officinalis*, attenuated DSS-induced colitis by decreasing pro-inflammatory cytokines and restoring tryptophan metabolites [35].
*   **Hydroxytyrosol Acetate (HTy-Ac):** This extra virgin olive oil polyphenol demonstrated anti-inflammatory effects and protected against DSS-induced colitis by modulating inflammatory pathways [12].
*   **Arion Berry:** Aronia berry extracts improved clinical signs of DSS-induced UC, inhibited prostaglandin E2 production, and decreased nitric oxide, IL-6, and TNF-α levels [34]. Dietary aronia berry also prevented colitis by increasing Th17 and Treg cells [57].
*   **Mango Polyphenols:** Mango polyphenols reduced plasma levels of pro-inflammatory cytokines (IL-8, GRO, GM-CSF) and increased *Lactobacillus* species in patients with mild to moderate IBD [91].
*   **Pterostilbene:** Pterostilbene, a resveratrol derivative, reduced colonic inflammation by suppressing dendritic cell activation and promoting regulatory T cell development [101].
*   **Rosmarinic Acid (RA):** RA-loaded nanovesicles alleviated inflammation and oxidative stress in acute colitis by modulating the NLRP3 inflammasome and enhancing the intestinal barrier [108].
*   **Dioscorea alata L. Anthocyanins:** Anthocyanins from purple yam demonstrated potent anti-inflammatory effects in a TNBS-induced colitis model [25].
*   **Hylocereus polyrhizus Ethanolic Extract:** This extract exerted anti-inflammatory effects and prevented murine colitis by inhibiting NF-κB signaling [14].
*   **Terminalia catappa L. Ethanolic Extract:** This extract showed anti-inflammatory and immunomodulatory properties, improving intestinal barrier integrity and reducing pro-inflammatory mediators [21].
*   **Olea europaea Leaf Extract:** Olive leaf extract demonstrated anti-inflammatory and immunomodulatory effects, reducing pro-inflammatory mediators and restoring intestinal epithelial barrier integrity [37].
*   **Quercus ilex Extract:** This polyphenol extract improved TNBS-induced colitis by inhibiting inflammatory pathways and activating Nrf2/heme oxygenase-1 signaling [68].
*   **Citrus limon Peel Powder:** Lemon peel powder reduced intestinal barrier defects and inflammation in colitic mice, likely through the production of acetate and n-butyrate from dietary fibers [105].
*   **Moringa oleifera Leaf Extract:** This extract alleviated colonic inflammation by inhibiting the NF-κB signaling pathway and reducing pro-inflammatory cytokines [103].
*   **Taxifolin:** Dietary taxifolin protected against DSS-induced colitis by inhibiting the NF-κB signaling pathway, enhancing intestinal barrier function, and modulating gut microbiota [111].
Other Natural Compounds and Therapies:
*   **Fucoidans:** Orally administered fucoidan preparations significantly reduced the inflammatory pathology associated with DSS-induced colitis [6].
*   **Curcuminoids (Nanoformulation):** Curcuminoids nanomicelles, when added to mesalamine treatment, significantly improved symptoms and clinical activity in patients with mild to moderate UC [54].

*   **Propolis:** Propolis extracts from different origins significantly reduced colitis activity, protected against colonic tissue damage, and reduced oxidative stress [53].
*   **Honey and Spirulina platensis:** Honey and Spirulina platensis showed antioxidant and anti-inflammatory effects comparable to or better than sulfasalazine and mesalazine in an acetic acid-induced colitis model [72].
*   **Rape Bee Pollen Extract:** This extract ameliorated DSS-induced colitis by neutralizing IL-1β and regulating the gut microbiota [73].
*   **Jasonian glutinosa (L.) DC. (Rock Tea):** This extract showed therapeutic properties in a DSS-induced UC model, likely due to its anti-inflammatory and antioxidant phenolic and flavonoid constituents [74].
*   **Amyloid-Polyphenol Hybrid Nanofilaments:** These structures significantly ameliorated colitis in mice, promoted intestinal barrier function, suppressed pro-inflammatory gene expression, and regulated gut microbial dysbiosis [87].
*   **Maize Near-Isogenic Lines (NILs):** Maize lines enriched in anthocyanins and phlobaphenes showed antioxidant capacity and pilot studies indicated they could alleviate experimental colitis in mice [107].
*   **Bee Pollen (BP) Extract:** BP extract showed protective effects against DSS-induced intestinal barrier dysfunction and inflammation in Caco-2 cells [75].
Traditional Chinese Medicine (TCM) Formulas:
*   **Fufangkushen colon-coated capsule (FCC):** Showed similar efficacy and safety to mesalazine in treating active UC [1].
*   **Huang-Lian-Jie-Du-Tang (HLJDT):** A TCM formula with potential therapeutic benefits in inflammatory diseases [2].
*   **Sishen Pill (SSP):** Treated DSS-induced colitis by regulating the interaction between inflammatory dendritic cells and gut microbiota [33].
*   **Baitouweng decoction (BTW):** Protected against DSS-induced UC by regulating Th17/Treg balance and intestinal epithelial barrier [38].
*   **Xipayi Kui Jie'an (KJA) enema:** Showed promising therapeutic effects in UC by regulating gut microbiota [29].
*   **Turkish Galls:** Alleviated DSS-induced UC by modulating gut microbiota, including increasing SCFA-producing bacteria and anti-inflammatory bacteria [64].
Other Findings:
*   **Dietary Polyphenols in General:** Many dietary polyphenols have shown anti-inflammatory, antioxidant, and immunomodulatory properties relevant to IBD [2, 10, 23, 24, 38, 47, 49, 71, 98, 106, 109]. However, some polyphenols, like high doses of EGCG and isoflavones, have exacerbated colitis [2].
*   **Gut Microbiota Modulation:** Several studies emphasize the role of gut microbiota in colitis and how various natural compounds can modulate it to exert therapeutic effects [10, 17, 29, 33, 35, 43, 45, 53, 57, 60, 64, 66, 73, 76, 77, 81, 83, 87, 92, 111].
*   **Mechanisms of Action:** Common mechanisms include inhibition of inflammatory pathways (NF-κB, MAPK, JAK/STAT), reduction of pro-inflammatory cytokines, antioxidant effects, protection of intestinal barrier integrity, and modulation of immune cell populations (Tregs, Th17, macrophages) [8, 14, 17, 18, 21, 23, 29, 33, 34, 38, 45, 47, 50, 52, 60, 63, 65, 66, 68, 75, 80, 81, 85, 90, 97, 101, 103, 108, 111].

*   **Clinical Trials:** While many studies are preclinical, some human studies suggest the potential benefits of resveratrol [5, 22] and mango polyphenols [91] in UC patients. Further well-designed clinical trials are needed to confirm the efficacy and optimal dosages of many of these natural compounds [2, 5, 22, 40, 41].

Here's a summary of the provided papers, focusing on their findings related to ulcerative colitis (UC) and the efficacy of various treatments:

Polyphenols and Their Derivatives:

* **Resveratrol:** Multiple studies highlight resveratrol's efficacy in ameliorating colitis. It has been shown to regulate Treg/Th17 balance [4], reduce pro-inflammatory cytokines (TNF-α, hs-CRP) and NF-κB activity in patients [5], act as an antioxidant [8, 22], and potentially modulate the gut microbiota to induce Tregs and suppress Th17 cells [66]. It also shows promise in improving clinical symptoms and quality of life in UC patients [5, 22]. Resveratrol has also been shown to downregulate miR-31, promoting T regulatory cells [81] and to suppress DSS-induced colitis by regulating SUMO1 and the Wnt/β-catenin pathway [90]. However, one study noted potential adverse effects in female mice and no significant effect on TNF-alpha levels [30].
* **Grape Pomace Extracts (GPEs):** Polyphenol-rich red grape pomace extracts (GPEs) attenuated clinical signs and colon shortening in DSS-induced colitis in rats [1]. A low dose (0.1%) of GPE was particularly effective in preventing colitis, reducing inflammation, and improving antioxidant activity [20].
* **Curcumin:** Curcumin has demonstrated significant therapeutic effects in experimental colitis by suppressing dendritic cell activation [8], regulating macrophage polarization [47], and improving gut microbiota diversity [43]. It has also shown efficacy in patients with mild-to-moderate UC, reducing clinical activity and improving well-being [54]. Nanoparticle curcumin showed improved absorbability and efficacy in ameliorating colitis by modulating gut microbiota and inducing regulatory T cells [43]. However, one study noted that curcumin supplementation could induce mild anemia and worsen colitis in mice on an iron-sufficient diet [69].
* **Gallic Acid (GA):** Gallic acid, a naturally occurring polyphenol, blunted weight loss and clinical symptoms in a murine model of UC. It ameliorated colonic architecture disruption, reduced myeloperoxidase activity, and decreased inflammatory mediators by suppressing NF-κB and STAT3 activation [9]. It also showed protective effects by trapping ammonia and improving gut microbiota dysbiosis [78].
* **Chlorogenic Acid (CGA):** CGA ameliorated intestinal mitochondrial injury by increasing antioxidant effects and respiratory complex activities [13]. It also protected against DSS-induced colitis by inhibiting the NF-κB signaling pathway, improving gut barrier function, and modulating gut microbiota, notably increasing *Akkermansia* [33]. CGA also protected against indomethacin-induced inflammation by decreasing *Bacteroides*-derived LPS [95].
* **Pomegranate Polyphenols:** Pomegranate polyphenolics reduced colon inflammation and ulceration in DSS-induced colitis by modulating the miR-145/p70S6K/HIF1α axis [27]. Pomegranate peel extract also altered the microbiome and decreased the pathogenicity of bacterial infections [77]. Pomegranate juice is being investigated for its potential to reduce fecal calprotectin levels in IBD patients [70]. Pomegranate seed oil and avocado seed oil also showed anti-inflammatory effects in UC models [100].
* **Green Tea Polyphenols (GTPs):** GTPs, particularly EGCG, have shown protective effects against colitis by inhibiting inflammatory pathways (NF-κB, JAK/STAT) and improving intestinal barrier function [50, 65]. EGCG also showed an efficacy signal in pouchitis, a complication of

ileal pouch-anal anastomosis [41]. However, high doses of EGCG could be nephrotoxic and may exacerbate weight loss by affecting macronutrient digestion [17].

* **Taxifolin:** Dietary taxifolin protected against DSS-induced colitis by inhibiting the NF-κB signaling pathway, enhancing intestinal barrier function, and modulating gut microbiota [111].
* **Magnolol:** Magnolol, a lignan from *Magnolia officinalis*, attenuated DSS-induced colitis by decreasing pro-inflammatory cytokines and restoring tryptophan metabolites [35].
* **Hydroxytyrosol Acetate (HTy-Ac):** This extra virgin olive oil polyphenol demonstrated anti-inflammatory effects and protected against DSS-induced colitis by modulating inflammatory pathways [12]. Hydroxytyrosol itself also showed similar benefits [20].
* **Aronia Berry:** Aronia berry extracts improved clinical signs of DSS-induced UC, inhibited prostaglandin E2 production, and decreased nitric oxide, IL-6, and TNF-α levels [34]. Dietary aronia berry also prevented colitis by increasing Th17 and Treg cells [57].
* **Mango Polyphenols:** Mango polyphenols reduced plasma levels of pro-inflammatory cytokines (IL-8, GRO, GM-CSF) and increased *Lactobacillus* species in patients with mild to moderate IBD [91].
* **Pterostilbene:** Pterostilbene, a resveratrol derivative, reduced colonic inflammation by suppressing dendritic cell activation and promoting regulatory T cell development [101]. Pterostilbene also showed protective effects against colitis by intervening in colonic innate and adaptive immune responses [58].
* **Rosmarinic Acid (RA):** RA-loaded nanovesicles alleviated inflammation and oxidative stress in acute colitis by modulating the NLRP3 inflammasome and enhancing the intestinal barrier [108]. RA also restored colonic mucus secretion by regulating gut microbiota-derived metabolites and inflammasome activation [59].
* **Dioscorea alata L. Anthocyanins:** Anthocyanins from purple yam demonstrated potent anti-inflammatory effects in a TNBS-induced colitis model [25].
* **Hylocereus polyrhizus Ethanolic Extract:** This extract exerted anti-inflammatory effects and prevented murine colitis by inhibiting NF-κB signaling [14].
* **Terminalia catappa L. Ethanolic Extract:** This extract showed anti-inflammatory and immunomodulatory properties, improving intestinal barrier integrity and reducing pro-inflammatory mediators [21].
* **Olea europaea Leaf Extract:** Olive leaf extract demonstrated anti-inflammatory and immunomodulatory effects, reducing pro-inflammatory mediators and restoring intestinal epithelial barrier integrity [37].
* **Quercus ilex Extract:** This polyphenol extract improved TNBS-induced colitis by inhibiting inflammatory pathways and activating Nrf2/heme oxygenase-1 signaling [68].
* **Citrus limon Peel Powder:** Lemon peel powder reduced intestinal barrier defects and inflammation in colitic mice, likely through the production of acetate and n-butyrate from dietary fibers [105].
* **Moringa oleifera Leaf Extract:** This extract alleviated colonic inflammation by inhibiting the NF-κB signaling pathway and reducing pro-inflammatory cytokines [103].
* **Wheat Bran Polyphenols (WBP):** WBP exerted remarkable protective effects against experimental colitis by reducing inflammation, improving colon morphology, suppressing the MAPK/NF-κB pathway, and modulating gut microbiota [80].

* **Chicoric Acid (CA):** CA alleviated the symptoms of colitis and maintained intestinal barrier integrity. It downregulated inflammatory factors and modulated gut microbiota, with fecal microbiota transplantation from CA-treated mice verifying the key role of gut microbiota [88].
* **Tannic Acid (TA):** TA protected against colitis by regulating the IL-17-NF-κB pathway and modulating gut microbiota and methylation pathways [103]. TA also showed protective effects by trapping ammonia and improving gut microbiota dysbiosis [78].
* **Ligustroside:** Ligustroside was effective in preventing colitis by reducing inflammation, enhancing the intestinal barrier, and altering gut microbiota and lipid composition [86].
* **Asiatic acid:** Asiatic acid attenuated molecular, biochemical, and histological alterations in acetic acid-induced UC models by improving antioxidative status and attenuating inflammatory and apoptotic challenges [97].
* **Geraniin:** Geraniin exhibited anti-IL-1β activity and anticolitis effects by hindering the IL-1β and IL-1R interaction and suppressing IL-1β-induced tight junction damage [92].
* **Hippuric acid (HA):** HA alleviated DSS-induced colitis by improving the mucosal barrier in a gut microbiota-dependent manner [49].
* **Ellagic Acid and Gallic Acid:** These polyphenols showed beneficial effects in animal models of colitis and exhibited antibacterial potential against IBD bacterial isolates [38].
* **Urolithin A:** This metabolite of ellagic acid attenuated pro-inflammatory factors and showed anti-cancer and anti-inflammatory effects in various in vivo studies, including colitis models [29].
* **Oxyresveratrol (OXY):** OXY ameliorated DSS-induced colitis by reducing inflammation, restoring the intestinal mucus layer, and downregulating pro-inflammatory cytokine genes [203].
* **Nobiletin (NOB):** NOB, delivered via yeast microcapsules, alleviated inflammatory reaction and oxidative stress in UC models by inhibiting NLRP3 inflammasome and balancing macrophage polarization [101].
* **Zanthoxylum bungeanum Maxim. polyphenols:** These polyphenols showed therapeutic potential in preclinical studies for inflammatory diseases, including ulcerative colitis [84].
* **Theabrownin from Fu Brick Tea:** This biomacromolecule alleviated UC by shaping the gut microbiota and modulating tryptophan metabolism, activating the AhR pathway [52]. Fu brick tea polyphenols also showed similar benefits [48].
* **Peanut Skin Procyanidins:** These polyphenols attenuated DSS-induced UC by increasing goblet cell numbers, tight junction protein expression, reducing inflammation and oxidative stress, and elevating SCFAs production [42].
* **Foxtail Millet Bran Polyphenols:** These polyphenols effectively relieved colitis by preventing intestinal barrier damage and promoting gut microbiota community [44].
* **Artichoke Pectin:** Artichoke pectin and its modified fractions showed potential in ameliorating inflammatory bowel disease in a mice model of colitis [131].
* **Black Lentil Water Extract:** This extract showed anti-inflammatory and proimmune response effects and prevented the growth of neoplasia in a colitis-associated colon cancer model [137].
* **Maqui Berry:** Maqui berry water extract alleviated colitis by reducing inflammatory markers, immune stress, and regulating gut microbiota [112].
* **Taraxacum officinale Extract:** This extract ameliorated DSS-induced colitis by regulating fatty acid degradation and microbial dysbiosis [113].

*   **Cyrtocarpa procera Bark Extract:** This extract showed anti-inflammatory and antioxidant activities, reducing UC symptoms and inflammatory markers [150].
*   **Gmelina arborea:** Extracts from this plant showed antioxidant, anti-diabetic, anti-inflammatory, antiulcer, and analgesic activities, with potential for colitis treatment [184].
*   **Canavalia gladiata Extract:** This extract showed anti-inflammatory effects by inhibiting NF-κB signaling and reducing pro-inflammatory mediators in colitis models [123].
*   **Paeonia lactiflora:** This herb showed multi-component and multi-target pharmacological effects on UC through various signaling pathways [117].
*   **Achillea spp.:** Extracts from this genus have shown promising antibacterial, antioxidant, anti-proliferative, and anti-inflammatory properties, with potential for UC treatment [165].
*   **Physalis pubescens L. Polysaccharides:** These polysaccharides ameliorated colitis by preventing oxidative damage, aberrant immune responses, and gut dysbiosis [166].
*   **Bruguiera gymnorrhiza Leaves Extract:** This extract ameliorated DSS-induced UC by suppressing NF-κB activation and modulating intestinal microbiota [136].
*   **Hyptis suaveolens Extract:** This extract showed intestinal anti-inflammatory activity through antioxidant, immunomodulatory, and anti-proliferative mechanisms [168].
*   **Ajuga chamaepitys subsp. chia Extract:** This extract and its iridoids showed beneficial effects in colitis models, with potential for IBD treatment [169].
*   **Fumaria capreolata Alkaloid Fraction:** This fraction showed intestinal anti-inflammatory effects by modulating immune responses and enhancing intestinal barrier markers [170].
*   **Grape Seed Proanthocyanidin Extract (GSPE):** GSPE ameliorated DSS-induced colitis by improving the intestinal barrier, reducing oxidative stress, and modulating inflammatory cytokines and gut microbiota [171].
*   **Maytenus robusta Extract:** This extract attenuated macro and microscopic alterations in the colon of mice with colitis, restoring antioxidant homeostasis [172].
*   **Artemisia gmelinii Extract:** This extract attenuated IBD symptoms and improved immune signaling by suppressing NF-κB signaling [200].
*   **Molokhia Leaf Extract:** This extract inhibited lipid accumulation, reduced gut permeability, and attenuated colonic inflammation by altering gut microbiota [144].
*   **Rosemary Extract (RE):** RE significantly improved the disease activity index in DSS-induced colitis and showed improvement in intestinal barrier integrity [195].
*   **Rhodiola crenulata Extract (RCE):** RCE alleviated DSS-induced colitis by anti-inflammation, mediating gut barrier integrity, and reshaping the gut microbiome [196].
*   **Artocarpus heterophyllus Extract:** This extract showed chemopreventive, cytotoxic, anticancer, and anti-inflammatory responses, validating its potential as a therapeutic agent [197].
*   **Glochidion ellipticum Wight Extracts:** These extracts ameliorated DSS-induced colitis by blocking NF-κB signaling pathway [198].
*   **Picrorhiza kurroa:** This herb and its active metabolites exhibit hepatoprotective, antioxidant, anti-inflammatory, anticancer, immunomodulator, and anti-ulcerative colitis activities [199].
*   **Phytoncide Extracts:** These extracts showed anti-inflammatory activity in digestive organs, with potential for functional agent development [201].

* **N-benzyl docosahexaenamide (NB-DHA):** This macamide alleviated DSS-induced colitis by reducing inflammation, restoring intestinal mucus layer, and upregulating tight junction proteins [202].
* **Total Flavonoids of Glycyrrhiza uralensis:** These flavonoids alleviated irinotecan-induced colitis by modifying gut microbiota and fecal metabolism, and inhibiting NLRP3 inflammasome activation [204].
* **Croton crassifolius Geisel Extract:** This extract alleviated UC symptoms by reducing inflammation and rectifying metabolic disorders [205].
* **Phaseolin:** This protein from *Phaseolus vulgaris* showed antioxidant, antigenotoxic, and chemopreventive potential [206].
* **Bilobalide (BI):** BI treatment reduced disease severity, increased colon length, and normalized colon histological characteristics by suppressing MAPK and AKT/NF-κB p65 signaling pathways and remodeling intestinal microbial communities [207].
* **Ginkgo biloba (GB):** GB extract has antioxidant and anti-inflammatory properties and has shown positive effects in animal models of colitis and in humans with ischemic colitis [141].
* **Cicer arietinum L. Extract:** This extract showed protective effects in DSS-induced colitis by suppressing pro-inflammatory mediators and inactivating NF-κB and STAT3 signaling [139].
* **Opuntia ficus indica Fruit Peel Extract:** This extract showed prophylactic effects against irradiation-induced colitis by its antioxidant and anti-inflammatory properties [121].
* **Cornus mas L. Extract:** This extract showed healing effects in experimentally induced ulcerative colitis in rats, verifying its ethnomedical use [122].
* **Momordica charantia L. Extract:** This extract showed protective anti-inflammatory effects on TNBS-induced colitis [125].
* **Ziziphus jujuba Mill. Fruit:** This fruit has been used traditionally for gastrointestinal problems, including colitis, and exhibits various pharmacological activities [126].
* **Pu-erh tea:** Bioactive components of Pu-erh tea interact with gut microbiomes and have potential health benefits [1]. Ripened Pu-erh tea extract attenuated DSS-induced colitis by modulating the NF-κB and HIF-1α signaling pathways [145] and promoted gut microbiota resilience [191].
* **Tea Polysaccharides (TPS) and Tea Polyphenols (TPP):** The combination of TPS and TPP showed greater effects on alleviating colitis and promoting intestinal barrier function than either component alone [28]. TPP showed superior anticolitis activity compared to TPS and theabrownin [74].
* **Theanine (TA):** TA did not show obvious alleviative effects on colitis [74].
* **Propolis:** Propolis extracts showed significant reduction in colitis activity, protection against colonic tissue damage, and reduced oxidative stress [53].
* **Honey and Spirulina platensis:** Both showed antioxidant and anti-inflammatory effects comparable to or better than sulfasalazine and mesalazine in an acetic acid-induced colitis model [72].
* **Rape Bee Pollen Extract:** This extract ameliorated DSS-induced colitis by modulating gut microbiota and increasing SCFAs [73].
* **Jasonian glutinosa (L.) DC. (Rock Tea):** This extract showed therapeutic properties in a DSS-induced UC model due to its anti-inflammatory and antioxidant phenolic and flavonoid constituents [74].

* **Amyloid-Polyphenol Hybrid Nanofilaments:** These structures significantly ameliorated colitis by promoting intestinal barrier function, suppressing pro-inflammatory gene expression, and regulating gut microbial dysbiosis [87].
* **Maize Near-Isogenic Lines (NILs):** Maize lines enriched in anthocyanins and phlobaphenes showed antioxidant capacity and pilot studies indicated they could alleviate experimental colitis [107].
* **Lonicera caerulea Pomace:** This pomace attenuated DSS-induced colitis by improving the intestinal barrier and modulating gut microbiota [68].
* **Spirulina platensis Extract:** This extract showed protective effects against DSS-induced UC by mitigating inflammation and restoring gut barrier function [114].
* **Balanophora polyandra Griff. Extract:** This extract ameliorated DSS-induced colitis by regulating NF-κB and NLRP3 inflammasome [163].
* **American Ginseng (AG):** AG and its components activate the Nrf2 pathway, decreasing oxidative stress and suppressing colitis [164].
* **Flaxseed Oligosaccharides (FOSs):** FOSs attenuated DSS-induced UC by modulating gut microbiota and repairing the intestinal barrier [175].
* **Flaxseed Linusorbs (LOMIX):** LOMIX exerted anti-inflammatory effects by targeting Src and Syk in the NF-κB pathway and ameliorated symptoms of colitis [158].
* **Silver Nanoparticles from Blackcurrant Extract:** These nanoparticles showed potent anti-inflammatory effects in vitro and in DSS-induced colitis [159].
* **Pomegranate Mesocarp:** Decoctions and fractions of pomegranate mesocarp reduced visceral hypersensitivity and colonic damage in colitis models [160].
* **Soy Isoflavones (SIFs):** SIFs alleviated DSS-induced colitis by targeting ERα/NLRP3 inflammasome pathways [161].
* **Total Alkaloids of Sophora alopecuroides L.:** These alkaloids ameliorated murine colitis by regulating bile acid metabolism and gut microbiota [142].
* **Pectin:** Pectin with different esterification degrees showed preventive effects on DSS-induced colitis, with low esterified pectin displaying better protective effects [143]. Pectic substances in general can inhibit gut inflammation and relieve IBD symptoms [192].
* **Seaweed Extracts and Polysaccharides:** These compounds have shown potential for the treatment and prevention of IBD by targeting various pathogenic mechanisms [157]. Cymopol from *Cymopolia barbata* activated Nrf2-mediated antioxidant response and reduced inflammation [109].
* **Garlic Extracts:** Hydroalcoholic and water extracts from Nubia red garlic showed protective effects in an ex vivo model of UC by counteracting LPS-induced inflammation and oxidative stress [45]. Jinxiang garlic polysaccharides also showed anti-inflammatory effects and modulated gut microbiota [181].
* **Carnosic Acid and Carnosol:** These rosemary diterpenes improved the disease activity index in DSS-induced colitis and showed improvement in intestinal barrier integrity [195].
* **Eleutherine palmifolia Extract:** This extract showed anticancer and anti-inflammatory properties, with potential for colitis treatment [189].
* **Croton crassifolius Geisel Extract:** This extract showed anti-inflammatory and antioxidant activities, reducing UC symptoms and inflammatory markers [205].

*   **Periplaneta americana Extract:** This extract promoted intestinal mucosal repair in colitis models, possibly by inhibiting IL-13 secretion and promoting EGF formation [186, 193].
*   **Aphanizomenon flos-aquae Extract (AphaMax®):** This extract attenuated the severity of colitis by decreasing NF-κB activation, iNOS and COX-2 expression, and inhibiting oxidative stress [187].
*   **Xique-xique Juice:** This cactus juice showed intestinal anti-inflammatory effects by downregulating IL-17, NF-κB, and iNOS, and upregulating ZO-1 and MUC-2 [120].
*   **Miracle Fruit (Synsepalum dulcificum) Extract:** While not directly studied for colitis, its potential anti-inflammatory properties are noted in a broader context of natural products [102].
*   **Miracle Fruit (Synsepalum dulcificum) Extract:** While not directly studied for colitis, its potential anti-inflammatory properties are noted in a broader context of natural products [102].

1-2000 above

Here's a summary of the provided papers, focusing on their findings related to ulcerative colitis (UC) and the efficacy of various treatments:

Natural Products and Their Extracts:

* **Lonicera japonica extract (LJE):** Showed dose-dependent inhibitory effects against colon shortening, weight loss, and histological damage in DSS-induced colitis. It down-regulated pro-inflammatory cytokines like IL-1β, TNF-α, IFN-γ, IL-6, IL-12, and IL-17, but did not significantly affect IL-10 or Treg cells. The protective effects were attributed to the Th1/Th17 pathway [1].
* **Ginger and Zingerone:** Ameliorated TNBS-induced colitis in mice by modulating NF-κB activity and IL-1β signaling. Ginger and zingerone improved colonic injury in a dose-dependent manner and regulated cytokine-related pathways [2]. Ginger volatile oil also reduced colon weight/length ratio, ulcer severity, and inflammation in acetic acid-induced colitis in rats, with effects comparable to prednisolone [19].
* **Ursodeoxycholic acid (UDCA):** Ameliorated experimental colitis induced by TNBS in rats at a high dose (50 mg/kg/day), showing improved body weight recovery and reduced alkaline phosphatase activity. Lower doses were ineffective [3].
* **Aegle marmelos unripe fruit extract (AMFE):** Demonstrated dose-dependent anti-inflammatory, antioxidant, and mast cell stabilizing effects in acetic acid-induced ulcerative colitis and indomethacin-induced enterocolitis in rats [4].
* **Rhodomyrtus tomentosa methanol extract (Rt-ME):** Inhibited the production of nitric oxide (NO) and prostaglandin E2 (PGE2) in LPS-activated macrophages and ameliorated gastritis and colitis in mice. It suppressed NF-κB and AP-1 pathways by targeting Syk/Src and IRAK1/IRAK4 [5].
* **Gallic Acid (GA):** Blunted weight loss and clinical symptoms in a murine model of UC, ameliorated colonic architecture disruption, and reduced inflammatory mediators by suppressing NF-κB and STAT3 activation. It also showed protective effects by trapping ammonia and improving gut microbiota dysbiosis [9, 78].
* **Chlorogenic Acid (ChA):** Ameliorated DSS-induced colitis by inhibiting NF-κB signaling, improving gut barrier function, and modulating gut microbiota. It also suppressed pro-inflammatory and apoptotic signaling pathways, including ERK1/2, JNK1/2, Akt, STAT3, and NF-κB p65 [33, 62].
* **Pomegranate Polyphenols:** Reduced colon inflammation and ulceration in DSS-induced colitis by modulating the miR-145/p70S6K/HIF1α axis. Pomegranate peel extract altered the microbiome, and pomegranate juice is being investigated for its potential to reduce fecal calprotectin in IBD patients [27, 70].
* **Green Tea Polyphenols (GTPs):** Showed protective effects against colitis by inhibiting inflammatory pathways and improving intestinal barrier function. However, high doses could be nephrotoxic and exacerbate weight loss [20].
* **Taxifolin:** Dietary taxifolin protected against DSS-induced colitis by inhibiting the NF-κB signaling pathway, enhancing intestinal barrier function, and modulating gut microbiota [111].
* **Magnolol:** Attenuated DSS-induced colitis by decreasing pro-inflammatory cytokines and restoring tryptophan metabolites [35].
* **Hydroxytyrosol Acetate (HTy-Ac):** Demonstrated anti-inflammatory effects and protected against DSS-induced colitis by modulating inflammatory pathways [12].

* **Aronia Berry:** Extracts improved clinical signs of DSS-induced UC, inhibited prostaglandin E2 production, and decreased nitric oxide, IL-6, and TNF-α levels [34].
* **Mango Polyphenols:** Reduced plasma levels of pro-inflammatory cytokines and increased *Lactobacillus* species in patients with mild to moderate IBD [91].
* **Pterostilbene:** Reduced colonic inflammation by suppressing dendritic cell activation and promoting regulatory T cell development [101].
* **Rosmarinic Acid (RA):** RA-loaded nanovesicles alleviated inflammation and oxidative stress in acute colitis by modulating the NLRP3 inflammasome and enhancing the intestinal barrier. RA also restored colonic mucus secretion [59, 108].
* **Dioscorea alata L. Anthocyanins:** Demonstrated potent anti-inflammatory effects in a TNBS-induced colitis model [25].
* **Hylocereus polyrhizus Ethanolic Extract:** Exerted anti-inflammatory effects and prevented murine colitis by inhibiting NF-κB signaling [14].
* **Terminalia catappa L. Ethanolic Extract:** Showed anti-inflammatory and immunomodulatory properties, improving intestinal barrier integrity and reducing pro-inflammatory mediators [21].
* **Olea europaea Leaf Extract:** Demonstrated anti-inflammatory and immunomodulatory effects, reducing pro-inflammatory mediators and restoring intestinal epithelial barrier integrity [37].
* **Quercus ilex Extract:** Improved TNBS-induced colitis by inhibiting inflammatory pathways and activating Nrf2/heme oxygenase-1 signaling [68].
* **Citrus limon Peel Powder:** Reduced intestinal barrier defects and inflammation in colitic mice, likely through the production of acetate and n-butyrate [105].
* **Moringa oleifera Leaf Extract:** Alleviated colonic inflammation by inhibiting the NF-κB signaling pathway and reducing pro-inflammatory cytokines [103].
* **Wheat Bran Polyphenols (WBP):** Exerted protective effects against experimental colitis by reducing inflammation, improving colon morphology, suppressing the MAPK/NF-κB pathway, and modulating gut microbiota [80].
* **Chicoric Acid (CA):** Alleviated colitis symptoms and maintained intestinal barrier integrity by downregulating inflammatory factors and modulating gut microbiota [88].
* **Tannic Acid (TA):** Protected against colitis by regulating the IL-17-NF-κB pathway and modulating gut microbiota and methylation pathways [103].
* **Ligustroside:** Prevented colitis by reducing inflammation, enhancing the intestinal barrier, and altering gut microbiota and lipid composition [86].
* **Asiatic acid:** Attenuated molecular, biochemical, and histological alterations in acetic acid-induced UC models by improving antioxidative status and attenuating inflammatory and apoptotic challenges [97].
* **Geraniin:** Exhibited anti-IL-1β activity and anticolitis effects by hindering the IL-1β and IL-1R interaction and suppressing IL-1β-induced tight junction damage [92].
* **Hippuric acid (HA):** Alleviated DSS-induced colitis by improving the mucosal barrier in a gut microbiota-dependent manner [49].
* **Urolithin A:** Attenuated pro-inflammatory factors and showed anti-cancer and anti-inflammatory effects in colitis models [29].

*   **Oxyresveratrol (OXY):** Ameliorated DSS-induced colitis by reducing inflammation, restoring the intestinal mucus layer, and downregulating pro-inflammatory cytokine genes [203].
*   **Nobiletin (NOB):** Alleviated inflammatory reaction and oxidative stress in UC models by inhibiting NLRP3 inflammasome and balancing macrophage polarization [101].
*   **Zanthoxylum bungeanum Maxim. polyphenols:** Showed therapeutic potential in preclinical studies for inflammatory diseases, including ulcerative colitis [84].
*   **Theabrownin from Fu Brick Tea:** Alleviated UC by shaping the gut microbiota and modulating tryptophan metabolism, activating the AhR pathway [52].
*   **Peanut Skin Procyanidins:** Attenuated DSS-induced UC by increasing goblet cell numbers, tight junction protein expression, reducing inflammation and oxidative stress, and elevating SCFAs production [42].
*   **Foxtail Millet Bran Polyphenols:** Effectively relieved colitis by preventing intestinal barrier damage and promoting gut microbiota community [44].
*   **Artichoke Pectin:** Showed potential in ameliorating inflammatory bowel disease in a mice model of colitis [131].
*   **Black Lentil Water Extract:** Showed anti-inflammatory and proimmune response effects and prevented the growth of neoplasia in a colitis-associated colon cancer model [137].
*   **Maqui Berry:** Water extract alleviated colitis by reducing inflammatory markers, immune stress, and regulating gut microbiota [112].
*   **Taraxacum officinale Extract:** Ameliorated DSS-induced colitis by regulating fatty acid degradation and microbial dysbiosis [113].
*   **Cyrtocarpa procera Bark Extract:** Showed anti-inflammatory and antioxidant activities, reducing UC symptoms and inflammatory markers [150].
*   **Gmelina arborea:** Extracts showed antioxidant, anti-diabetic, anti-inflammatory, antiulcer, and analgesic activities, with potential for colitis treatment [184].
*   **Canavalia gladiata Extract:** Showed anti-inflammatory effects by inhibiting NF-κB signaling and reducing pro-inflammatory mediators in colitis models [123].
*   **Paeonia lactiflora:** Showed multi-component and multi-target pharmacological effects on UC through various signaling pathways [117].
*   **Achillea spp.:** Extracts have shown promising antibacterial, antioxidant, anti-proliferative, and anti-inflammatory properties, with potential for UC treatment [165].
*   **Physalis pubescens L. Polysaccharides:** Ameliorated colitis by preventing oxidative damage, aberrant immune responses, and gut dysbiosis [166].
*   **Bruguiera gymnorrhiza Leaves Extract:** Ameliorated DSS-induced UC by suppressing NF-κB activation and modulating intestinal microbiota [136].
*   **Hyptis suaveolens Extract:** Showed intestinal anti-inflammatory activity through antioxidant, immunomodulatory, and anti-proliferative mechanisms [168].
*   **Ajuga chamaepitys subsp. chia Extract:** Showed beneficial effects in colitis models, with potential for IBD treatment [169].
*   **Fumaria capreolata Alkaloid Fraction:** Showed intestinal anti-inflammatory effects by modulating immune responses and enhancing intestinal barrier markers [170].
*   **Grape Seed Proanthocyanidin Extract (GSPE):** Ameliorated DSS-induced colitis by improving the intestinal barrier, reducing oxidative stress, and modulating inflammatory cytokines and gut microbiota [171].

*   **Maytenus robusta Extract:** Attenuated macro and microscopic alterations in the colon of mice with colitis, restoring antioxidant homeostasis [172].
*   **Artemisia gmelinii Extract:** Attenuated IBD symptoms and improved immune signaling by suppressing NF-κB signaling [200].
*   **Molokhia Leaf Extract:** Inhibited lipid accumulation, reduced gut permeability, and attenuated colonic inflammation by altering gut microbiota [144].
*   **Rosemary Extract (RE):** Significantly improved the disease activity index in DSS-induced colitis and showed improvement in intestinal barrier integrity [195].
*   **Rhodiola crenulata Extract (RCE):** Alleviated DSS-induced colitis by anti-inflammation, mediating gut barrier integrity, and reshaping the gut microbiome [196].
*   **Artocarpus heterophyllus Extract:** Showed chemopreventive, cytotoxic, anticancer, and anti-inflammatory responses, validating its potential as a therapeutic agent [197].
*   **Glochidion ellipticum Wight Extracts:** Ameliorated DSS-induced colitis by blocking NF-κB signaling pathway [198].
*   **Picrorhiza kurroa:** Exhibits hepatoprotective, antioxidant, anti-inflammatory, anticancer, immunomodulator, and anti-ulcerative colitis activities [199].
*   **Phytoncide Extracts:** Showed anti-inflammatory activity in digestive organs, with potential for functional agent development [201].
*   **N-benzyl docosahexaenamide (NB-DHA):** Alleviated DSS-induced colitis by reducing inflammation, restoring intestinal mucus layer, and upregulating tight junction proteins [202].
*   **Total Flavonoids of Glycyrrhiza uralensis:** Alleviated irinotecan-induced colitis by modifying gut microbiota and fecal metabolism, and inhibiting NLRP3 inflammasome activation [204].
*   **Croton crassifolius Geisel Extract:** Alleviated UC symptoms by reducing inflammation and rectifying metabolic disorders [205].
*   **Phaseolin:** Showed antioxidant, antigenotoxic, and chemopreventive potential [206].
*   **Bilobalide (BI):** Reduced disease severity, increased colon length, and normalized colon histological characteristics by suppressing MAPK and AKT/NF-κB p65 signaling pathways and remodeling intestinal microbial communities [207].
*   **Ginkgo biloba (GB):** Has antioxidant and anti-inflammatory properties and has shown positive effects in animal models of colitis and in humans with ischemic colitis [141].
*   **Cicer arietinum L. Extract:** Showed protective effects in DSS-induced colitis by suppressing pro-inflammatory mediators and inactivating NF-κB and STAT3 signaling [139].
*   **Opuntia ficus indica Fruit Peel Extract:** Showed prophylactic effects against irradiation-induced colitis by its antioxidant and anti-inflammatory properties [121].
*   **Cornus mas L. Extract:** Showed healing effects in experimentally induced ulcerative colitis in rats [122].
*   **Momordica charantia L. Extract:** Showed protective anti-inflammatory effects on TNBS-induced colitis [125].
*   **Ziziphus jujuba Mill. Fruit:** Exhibits various pharmacological activities, including those relevant to gastrointestinal problems [126].
*   **Pu-erh tea:** Bioactive components interact with gut microbiomes and have potential health benefits. Ripened Pu-erh tea extract attenuated DSS-induced colitis by modulating the NF-κB and HIF-1α signaling pathways and promoted gut microbiota resilience [145, 191].

* **Tea Polysaccharides (TPS) and Tea Polyphenols (TPP):** The combination of TPS and TPP showed greater effects on alleviating colitis and promoting intestinal barrier function than either component alone [28]. TPP showed superior anticolitis activity compared to TPS and theabrownin [74].
* **Theanine (TA):** Did not show obvious alleviative effects on colitis [74].
* **Propolis:** Extracts showed significant reduction in colitis activity, protection against colonic tissue damage, and reduced oxidative stress [53].
* **Honey and Spirulina platensis:** Both showed antioxidant and anti-inflammatory effects comparable to or better than sulfasalazine and mesalazine in an acetic acid-induced colitis model [72].
* **Rape Bee Pollen Extract:** Ameliorated DSS-induced colitis by modulating gut microbiota and increasing SCFAs [73].
* **Jasonian glutinosa (L.) DC. (Rock Tea):** Showed therapeutic properties in a DSS-induced UC model due to its anti-inflammatory and antioxidant phenolic and flavonoid constituents [74].
* **Amyloid-Polyphenol Hybrid Nanofilaments:** Significantly ameliorated colitis by promoting intestinal barrier function, suppressing pro-inflammatory gene expression, and regulating gut microbial dysbiosis [87].
* **Maize Near-Isogenic Lines (NILs):** Maize lines enriched in anthocyanins and phlobaphenes showed antioxidant capacity and pilot studies indicated they could alleviate experimental colitis [107].
* **Lonicera caerulea Pomace:** Attenuated DSS-induced colitis by improving the intestinal barrier and modulating gut microbiota [68].
* **Spirulina platensis Extract:** Showed protective effects against DSS-induced UC by mitigating inflammation and restoring gut barrier function [114].
* **Balanophora polyandra Griff. Extract:** Ameliorated DSS-induced colitis by regulating NF-κB and NLRP3 inflammasome [163].
* **American Ginseng (AG):** Activates the Nrf2 pathway, decreasing oxidative stress and suppressing colitis [164].
* **Flaxseed Oligosaccharides (FOSs):** Attenuated DSS-induced UC by modulating gut microbiota and repairing the intestinal barrier [175].
* **Flaxseed Linusorbs (LOMIX):** Exerted anti-inflammatory effects by targeting Src and Syk in the NF-κB pathway and ameliorated symptoms of colitis [158].
* **Silver Nanoparticles from Blackcurrant Extract:** Showed potent anti-inflammatory effects in vitro and in DSS-induced colitis [159].
* **Pomegranate Mesocarp:** Decoctions and fractions reduced visceral hypersensitivity and colonic damage in colitis models [160].
* **Soy Isoflavones (SIFs):** Alleviated DSS-induced colitis by targeting ERα/NLRP3 inflammasome pathways [161].
* **Total Alkaloids of Sophora alopecuroides L.:** Ameliorated murine colitis by regulating bile acid metabolism and gut microbiota [142].
* **Pectin:** Showed preventive effects on DSS-induced colitis, with low esterified pectin displaying better protective effects. Pectic substances in general can inhibit gut inflammation and relieve IBD symptoms [143, 192].

*   **Seaweed Extracts and Polysaccharides:** Showed potential for the treatment and prevention of IBD by targeting various pathogenic mechanisms [157].
*   **Garlic Extracts:** Showed protective effects in an ex vivo model of UC by counteracting LPS-induced inflammation and oxidative stress. Jinxiang garlic polysaccharides also showed anti-inflammatory effects and modulated gut microbiota [45, 181].
*   **Carnosic Acid and Carnosol:** Improved the disease activity index in DSS-induced colitis and showed improvement in intestinal barrier integrity [195].
*   **Eleutherine palmifolia Extract:** Showed anticancer and anti-inflammatory properties, with potential for colitis treatment [189].
*   **Periplaneta americana Extract:** Promoted intestinal mucosal repair in colitis models, possibly by inhibiting IL-13 secretion and promoting EGF formation [186, 193].
*   **Aphanizomenon flos-aquae Extract (AphaMax®):** Attenuated the severity of colitis by decreasing NF-κB activation, iNOS and COX-2 expression, and inhibiting oxidative stress [187].
*   **Xique-xique Juice:** Showed intestinal anti-inflammatory effects by downregulating IL-17, NF-κB, and iNOS, and upregulating ZO-1 and MUC-2 [120].
*   **Miracle Fruit (Synsepalum dulcificum) Extract:** While not directly studied for colitis, its potential anti-inflammatory properties are noted [102].

Pharmaceuticals and Other Treatments:

*   **Mesalamine:** Used as a comparator, showing comparable efficacy to some natural products. Higher doses (4.8 g/day) showed greater effectiveness in certain patient populations compared to lower doses (3.6 g/day) [33].
*   **Azathioprine (AZA):** Side effects were reported in 32.4% of IBD patients, with dose-dependent effects occurring mostly between 12-18 months of treatment [17].
*   **Tofacitinib:** An orally available JAK inhibitor that demonstrated efficacy in UC, but systemic immunosuppression can be a concern. Intracolonic delivery showed similar efficacy with lower plasma levels and no impact on splenic NK cells [53]. A macromolecular prodrug of tofacitinib (P-Tofa) showed superior therapeutic efficacy compared to daily oral tofacitinib due to passive targeting and retention in the inflamed colon [66].
*   **SRT2104 (SIRT1 activator):** Did not demonstrate significant clinical activity in mild to moderately active UC [29].
*   **VX765 (caspase-1 inhibitor):** Attenuated DSS-induced colitis in mice by suppressing the Caspase-1/GSDMD pathway and downstream inflammatory cytokines IL-1β and IL-18 in a dose-dependent manner [87].
*   **OPS-2071 (quinolone antibacterial agent):** Showed both immunosuppressive and antibacterial effects, making it a promising candidate for IBD [86].
*   **Phenytoin:** Topical administration accelerated the healing of acetic acid-induced colitis in rats by increasing TGFβ, PDGF, and VEGF in a dose- and time-dependent manner [92].
*   **Camelina sativa Defatted Seed Meal (DSM):** Showed a dose-dependent pain-relieving effect in DNBS-treated rats and counteracted visceral hyperalgesia by reducing intestinal inflammatory damage and preventing enteric neuron damage, with efficacy linked to PPAR α receptor activation [95].
*   **Anemoside B4 (AB4):** Showed dose-dependent therapeutic effects against chronic relapsing colitis by modulating inflammatory response, colonic gene expression, and intestinal

microbiota. Its effects were comparable to tofacitinib and berberine, but without the adverse effects of tofacitinib on splenic swelling and anemia [96].
* **Prednisolone:** Low-dose perioperative prednisolone treatment showed beneficial effects on postoperative recovery and anastomotic healing in a murine colitis model, limiting disease activity without increasing anastomotic leakage [97].
* **Dietary Proteins:** Casein and red meat exacerbated colitis, while whey protein mitigated it most effectively [98].
* **Aflatoxin B1 (AFB1):** Induced colitis in mice, with macrophage AHR/TLR4/STAT3 signaling implicated in the process [99].
* **Immune Checkpoint Inhibitors (ICIs):** CTLA-4 inhibitors are associated with a higher risk of colitis compared to PD-1/PD-L1 inhibitors. Combination therapy with PD-1/PD-L1 and CTLA-4 inhibitors significantly increased the risk of colitis [76].

Mechanisms of Action:

Many natural products and pharmaceuticals exert their effects through various mechanisms, including:

* **Modulation of inflammatory pathways:** NF-κB, MAPK, STAT3, NLRP3 inflammasome, TLR4, AhR, Nrf2, PPARα, and caspase-1 pathways were frequently targeted [1, 2, 8, 9, 13, 14, 15, 17, 21, 23, 24, 27, 29, 33, 34, 35, 38, 39, 40, 45, 46, 47, 49, 50, 53, 57, 60, 62, 63, 65, 66, 67, 68, 69, 73, 74, 75, 80, 81, 82, 83, 85, 86, 87, 91, 92, 93, 95, 96, 99].
* **Regulation of gut microbiota:** Several compounds modulated gut microbiota composition, increasing beneficial bacteria and decreasing harmful ones [10, 17, 29, 33, 35, 43, 45, 53, 57, 60, 64, 66, 73, 76, 77, 81, 83, 87, 91, 96].
* **Protection of intestinal barrier function:** Compounds improved tight junction protein expression (e.g., ZO-1, occludin, claudins) and reduced intestinal permeability [10, 24, 60, 81, 83, 88, 91].
* **Antioxidant effects:** Many compounds reduced oxidative stress markers (e.g., MDA) and increased antioxidant enzymes (e.g., SOD) [4, 9, 13, 17, 34, 40, 44, 47, 61, 62, 77, 79, 83, 91].
* **Modulation of immune cells:** Effects on T cells (Th1, Th17, Treg, CD4+ T cells), macrophages, and NKT cells were observed [1, 8, 15, 30, 38, 40, 46, 50, 53, 65, 66, 75, 80, 81, 86, 93, 99].
* **Inhibition of pyroptosis:** VX765 suppressed caspase-1-mediated pyroptosis [87].
* **Regulation of ferroptosis:** Some agents were found to inhibit ferroptosis [76, 77].
* **AMPK signaling pathway:** Activation of AMPK was implicated in the anti-inflammatory effects of some compounds [87, 107].

Overall Efficacy and Safety:

* Many natural products and their extracts demonstrated significant therapeutic effects in preclinical models of colitis, often showing dose-dependent improvements.
* Some studies suggest that natural products may offer comparable or even superior efficacy to conventional treatments like mesalamine, prednisolone, and sulfasalazine [12, 19, 55, 72, 94].
* Combinations of natural products (e.g., BRAE and RA) sometimes showed additive or synergistic effects [56].
* While generally safe, some compounds showed potential toxicity at high doses (e.g., GTPs) [17, 20].

* Further clinical trials are needed to confirm the efficacy and safety of many of these agents in human UC patients.

Here's a summary of the provided papers, focusing on their findings related to ulcerative colitis (UC) and the efficacy of various treatments:

Natural Products and Their Extracts:

* **Garlicnin B1:** A cyclic sulfide from garlic, showed strong anti-tumor activity (IC50 ~20 μM) and ameliorated DSS-induced colitis in mice at a low dose (5 mg/kg), suggesting potential for treating cancer and inflammatory diseases [2].
* **Barley Leaf (BL):** Supplementation with BL significantly enriched arginine, which in turn ameliorated *Citrobacter-rodentium*-induced colitis symptoms in mice. Arginine intervention also improved gut microbiota, with dose-dependent effects [4].
* **Icariin-1 (GH01):** This flavonoid from *Epimedium* effectively ameliorated DSS-induced colitis symptoms in mice by restoring intestinal barrier function and microbial community balance. It enhanced macrophage phagocytic ability and promoted M1 to M2 polarization, inhibiting TLR4 and NF-κB pathways [5].
* **Ganoderic Acid A (GAA):** Derived from *Ganoderma lucidum*, GAA effectively prevented DSS-induced colitis in mice by preserving epithelial and mucus layer integrity, modulating gut microbiota, promoting tryptophan metabolism (3-IAld generation), activating the aryl hydrocarbon receptor (AhR), and inducing IL-22 production [6].
* **Chinese Water-Soluble Propolis (WSP):** Showed dose-dependent protective effects against DSS-induced ulcerative colitis in rats by reducing pro-inflammatory cytokines (TNF-α, IL-6, IL-9) and oxidative stress (enhancing SOD and GSH-Px activity) [7].
* **Highland Barley β-Glucan (HBG):** Alleviated DSS-induced colitis symptoms and promoted intestinal stem cell proliferation by increasing docosahexaenoic acid (DHA), which activates the peroxisome proliferator-activated receptor α (PPARα) [9].
* **Limosilactobacillus reuteri RE225:** This probiotic strain attenuated DSS-induced colitis in mice by inhibiting the TLR4/MyD88/NF-κB inflammatory pathway and the Nrf2/HO-1 oxidative stress pathway, and by regulating gut microbiota and short-chain fatty acid (SCFA) production, which in turn inhibited M1 macrophage polarization [49, 93].
* **Rutin:** Demonstrated coloprotective effects against acetic acid-induced UC in rats by ameliorating histopathological changes and suppressing the HMGB1/TLR4/MYD88/NF-κB signaling pathway [22].
* **Xylan Acetate Ester (XylA):** Significantly alleviated DSS-induced UC in mice by repairing the colon structure and intestinal barrier function (increasing ZO-1 and occludin expression), inhibiting LPS-TLR4 pathway, downregulating M1 macrophage polarization, and reducing pro-inflammatory cytokines [47].
* **Helix pomatia mucin:** Showed potential efficacy against DSS-induced UC in mice by reducing disease activity index, increasing fecal Firmicutes, enhancing colonic CCR7, CXCL9, and CXCL10 expression, and alleviating oxidative stress and inflammation [48].
* **Pu-erh tea:** Ripened Pu-erh tea extract attenuated DSS-induced colitis by modulating the NF-κB and HIF-1α signaling pathways and promoting gut microbiota resilience [145].
* **Tea Polyphenols (TPP):** Showed superior anticolitis activity compared to tea polysaccharides and theabrownin [74].
* **Myricetin:** Protected mice against DSS-induced colitis by activating the aryl hydrocarbon receptor (AhR) signaling pathway, which was linked to increased expressions of CYP1A1 and CYP1B1, and improved Treg/Th17 balance [151].

* **Formononetin (FMN):** Ameliorated DSS-induced colitis in mice by inhibiting the MAPK/PPAR-γ/NF-κB/ROS signaling pathways, reducing NLRP3 inflammasome expression, and preventing mitochondrial damage [134].
* **Lactiplantibacillus plantarum SFFI23:** Alleviated DSS-induced colitis by activating AhR downstream signaling, including CYP1A1 and IL-22, and repairing intestinal barrier function, which was linked to high indole-3-lactic acid (ILA) production [161].
* **Coscinium fenestratum alkaloids (CFAs):** Showed therapeutic potential in alleviating UC through interactions with key target genes involved in immunological response and inflammatory pathways [118].
* **Eurotium cristatum-fermented black tea (FBT):** Protected against DSS-induced UC by activating PPARγ and inhibiting the NF-κB signaling pathway, thereby enhancing intestinal barrier integrity and promoting mucosal repair [125].
* **Clonorchis sinensis infection:** Ameliorated DSS-induced colitis in mice in a gut microbiota-dependent manner, increasing secondary bile acids (SBAs) and TGR5 expression, which benefited anti-inflammation [157].
* **Glycyrrhiza uralensis Fisch. aqueous extract (GE):** Exerted therapeutic effects on TNBS-induced UC in rats by alleviating cell injury and inflammatory responses, potentially through the NOD2/RIP2/NF-κB signaling pathway [145].

Pharmaceuticals and Other Treatments:

* **Antibiotics:** Broad-spectrum antibiotic use was associated with a dose-dependent increased risk of IBD, with effects observed up to 9 years before diagnosis. Early-life antibiotic exposure also increased the risk of childhood-onset IBD [3].
* **Tofacitinib and Etrasimod:** These small molecule therapies, targeting JAK and S1P receptors respectively, are emerging as treatment options for moderate to severe UC, offering novel mechanisms to attenuate inflammation [24, 78, 81].
* **VX765 (caspase-1 inhibitor):** Attenuated DSS-induced colitis in mice by suppressing the Caspase-1/GSDMD pathway and downstream inflammatory cytokines [87].
* **SRT2104 (SIRT1 activator):** Did not demonstrate significant clinical activity in mild to moderately active UC [29].
* **Fenofibrate:** As a PPARα agonist, it showed potential as an adjunct therapy for mild to moderate UC, improving clinical outcomes and quality of life by reducing NLRP3 and fecal calprotectin, and increasing SIRT1 and AMPK [88].
* **Cannabidiol (CBD) and HU308:** Both showed promise in DSS-induced colitis models, with HU308 demonstrating enhanced therapeutic potential at a lower dose and modulating GLP-1 levels [62, 107].
* **GLP-1 receptor agonists:** Found to be effective for weight loss in IBD patients and did not increase rates of IBD exacerbation [68].
* **Pravastatin:** Showed potential in preventing colitis-associated carcinogenesis by reducing CX3CR1high M2-like fibrocytes [59].
* **Lacidipine:** An anti-hypertension drug, effectively reduced colon lesions and inflammatory markers in DSS-induced colitis by inhibiting NF-κB and Notch signaling pathways [39].
* **TRPM7 knockdown:** Showed therapeutic potential in UC by inactivating the NLRP3-dependent pyroptosis pathway [40].

* **PBT002 (dual GPBAR1 agonist and RORγt inverse agonist):** Reduced inflammation and disease severity in IBD models by modulating macrophage polarization and T cell responses [41].
* **TL1A inhibitors:** Show promising efficacy in clinical trials for moderate to severe IBD, targeting pathways of inflammation and fibrosis [42, 122, 143].
* **Anti-FcεRIα therapy:** Ameliorated DSS-induced colitis in mice in a gut microbiota-dependent manner, potentially linked to increased Lactobacillus abundance and NLRP6 inflammasome activation [43].
* **Kynurenine (KYN):** Ameliorated UC by improving intestinal barrier function, activating AhR, and inhibiting NF-κB and NLRP3 inflammasome pathways [44].
* **Vitexin:** Targeted the Vitamin D receptor (VDR) and modulated macrophage polarization through the VDR/PBLD pathway, mitigating colitis-associated colorectal cancer [45].
* **XAV939 (Wnt/β-catenin inhibitor):** Did not alleviate inflammation in a DSS-induced UC model, suggesting the Wnt/β-catenin pathway may not be the primary driver in this model [100].
* **CB2 receptor agonists (e.g., compound 33):** Showed potent and selective activity, attenuating colon inflammation in a DSS-induced colitis model [101].
* **PGLYRP-1:** Functions as an intracellular peptidoglycan receptor, and its activation by GMTriP-K protected mice from TNBS-induced colitis [75, 154].
* **Luteolin:** Ameliorated DSS-induced colitis by activating the Notch signaling pathway, promoting NCR+ILC3 differentiation, and improving intestinal barrier function [76].
* **CCL5/CCR5 axis:** Plays a role in UC pathogenesis, and CCR5 antagonists are being explored for UC treatment [90].
* **PTPN22:** Its tyrosine phosphatase activity influences T cell differentiation and JAK/STAT signaling, contributing to UC pathogenesis [97].
* **Fructo-oligosaccharide (FOS):** Alleviated DSS-induced colitis by modulating gut microbiota and tryptophan metabolism, activating the AhR/IL-22 axis [98].
* **Cannabis:** Shows potential as a complementary therapy for IBD, acting on CB1 and CB2 receptors to reduce inflammation, but requires more large-scale clinical trials [95, 114].
* **Metformin:** Ameliorated DSS-induced colitis by suppressing γδT17 cell differentiation, potentially through inhibiting mTOR/RORγt activity [146].
* **Anti-TNF monoclonal antibody (ADA):** Prevented DSS-induced colitis pathology and protected enteric neurons [147].
* **Bicalutamide (nano-formulated):** Targeted macrophages to degrade NLRP3 via MAP3K1, showing potential for treating colitis [137].
* **Phillygenin (PHI):** Alleviated chronic colitis by activating TGR5 and inhibiting the PERK-eIF2α-Ca2+ pathway, improving intestinal barrier function [141].
* **Buspirone (5-HT1A agonist):** Post-colitis alterations in mice showed dose-dependent effects on RMg and DR neurons, weakening its antinociceptive action [130].
* **NPS 2143 (CaSR inhibitor):** Showed an anti-inflammatory effect on medium-grade colitis, reducing colon ulceration and IL-22 expression [120].
* **Pellino1:** An ubiquitin ligase that targets STAT3, its inhibition may be a potential therapeutic strategy for chronic inflammation and cancer [140].
* **Cart-T cell therapy:** Can induce severe colitis as an immune-related adverse event, requiring careful management [115, 158].

*   **EP4 receptor agonists:** Show promise for UC treatment, but systemic side effects have been a challenge; prodrugs are being developed for GI-specific delivery [128].
*   **S1P Lyase Inhibition:** Increased intestinal S1P, disrupted the intestinal barrier, and aggravated DSS-induced colitis [149].
*   **ADCY7:** Variants in this gene are associated with UC risk, and enhancing cyclic AMP signaling may be beneficial [150].

Immune System and Signaling Pathways:

*   **Membrane receptor blockade:** Strategies targeting membrane receptors like Toll-like receptors, IL-1 receptor, and others are proposed as early-stage inflammation modulators with immunomodulatory capabilities [8].
*   **CD8+ regulatory T cells (Treg-of-B cells):** A novel subpopulation identified with suppressive effects, potentially offering therapeutic potential for IBD [12].
*   **Muscularis macrophages (MMφs):** During colitis, Lyve1+ MMφs are lost, while resident Cx3cr1+ MMφs show inflammatory plasticity, highlighting potential therapeutic targets [13].
*   **Gut microbiota and Tregs:** Microbiota influences colonic Treg functionality, leading to distinct phenotypic subsets with enhanced suppressive capabilities that regulate enteric inflammation [14].
*   **TLR2:** Deficiency in TLR2 exacerbates DSS-induced colitis by attenuating cell cycle signaling, potentially mediated by Marinifilaceae [58].
*   **PD-1/PD-L1 pathway:** Upregulated in UC patients and modulated by tonsil-derived mesenchymal stem cells (TMSCs), suggesting a role in UC pathogenesis and treatment [83].
*   **Syk:** Gain-of-function variants in Syk may contribute to pediatric IBD by promoting macrophage migration, ROS production, and NLRP3 inflammasome activation [84].
*   **Vitamin D receptor (VDR):** Plays a role in protecting the intestinal barrier against colitis by regulating the Notch pathway [23] and microfold cells [37]. Its absence accelerates colitis-associated colorectal cancer [45]. VDR expression did not correlate with disease activity in UC patients [131].
*   **Aryl hydrocarbon receptor (AhR):** Activated by nutritional ligands, it plays a crucial role in gut immunity and inflammation [121]. AhR activation by myricetin ameliorated DSS-induced colitis [151], while its deficiency in myeloid cells exacerbated colitis [18]. AhR activation by indole-3-carbinol (I3C) reduced colitis severity in a sex-dependent manner [32].
*   **NLRP3 inflammasome:** Implicated in the intersection of diabetes mellitus and IBD, regulating pro-inflammatory cytokines [51]. Its inhibition is a target for UC treatment [40, 53].
*   **TLR4:** Its signaling pathway is targeted by several natural compounds for their anti-inflammatory effects in colitis [21, 28, 49, 92].
*   **Toll-like receptor 3 (TLR3):** Its signaling attenuates colitis-associated cancer development [96].
*   **TL1A and DR3:** These molecules are key players in IBD pathogenesis, and TL1A inhibition is a promising therapeutic strategy [42, 122, 143].
*   **P2X7 receptor:** Persistent activation is linked to chronic inflammation and cancer in the intestine, and its blockade may have therapeutic effects in IBD [70].
*   **Dectin-1:** A pattern recognition receptor with potential as a therapeutic target for IBD, influencing immune responses and gut microbiota [80].

*   **Chemokines:** Their expression profiles are altered in IBD and colorectal cancer, offering insights into disease mechanisms and potential drug targets [63, 91].
*   **GILZ/c-Rel/RACK1 signaling:** Alterations in this pathway disrupt intestinal epithelial barrier integrity in IBD [112].
*   **Pellino1:** Targets STAT3 to regulate macrophage-mediated inflammation and tumor development, suggesting it as a potential therapeutic target [140].
*   **Mincle (Clec4e):** Essential for innate immune responses to *Enterococcus faecalis*, influencing colitis severity [133].
*   **Clec12a:** Controls colitis by tempering inflammation and restricting the expansion of specific commensals like *Faecalibaculum rodentium* [126].
*   **GPR4 and OGR1:** Simultaneous loss of these pH-sensing receptors ameliorates colitis with additive effects on inflammation [156].
*   **IL-10 producing ILCs:** Crucial for intestinal immune homeostasis and preventing colitis [155].
*   **Tissue factor (TF):** TF-dependent thrombogenicity of colitogenic CD4+ T cells is regulated by activated protein C signaling and is implicated in IBD [148].
*   **ADCY7:** Variants in this gene are associated with UC risk, and enhancing cyclic AMP signaling may be beneficial [150].

Other Findings:

*   **Gut Microbiota:** Plays a crucial role in IBD pathogenesis, with various compounds modulating its composition to exert therapeutic effects [6, 10, 14, 21, 25, 29, 43, 45, 49, 53, 64, 73, 93, 98, 103, 105, 129, 157].
*   **Nanomedicine:** Nanostructured drug-delivery systems offer promising approaches for colon-targeted drug delivery in IBD [15].
*   **Biomarkers:** Coagulation and fibrinolytic-related genes, as well as chemokine families, are being investigated as diagnostic biomarkers for UC [36, 63].
*   **Sex Differences:** Biological sex influences IBD epidemiology, pathophysiology, and treatment response, highlighting the need for sex-specific research and therapies [32, 109, 152].
*   **Mucosal Healing:** Targeting pathways involved in intestinal epithelial barrier repair and mucosal healing is crucial for effective IBD treatment [87].
*   **Tryptophan Metabolism:** Modulating this pathway via nutritional interventions and microbial metabolites shows promise for mitigating colitis [6, 25, 98, 103, 105, 132, 139, 161].
*   **Bile Acids:** Their altered metabolism and interaction with receptors like GPBAR1 and S1PR2 are implicated in IBD pathogenesis and offer therapeutic targets [64, 104, 119, 129, 157].
*   **Colitis-Associated Cancer (CAC):** Chronic inflammation in IBD increases CAC risk, and targeting pathways like VDR, AhR, and MGL1 may help prevent or treat it [45, 56, 59, 96, 124, 162, 163].
*   **Visceral Hypersensitivity:** Spinal astrocyte-derived M-CSF and reactive microglia contribute to this symptom in IBD, identifying spinal M-CSF as a potential therapeutic target [153].
*   **T-cell repertoire:** Longitudinal analysis reveals dynamic clonal T cell populations in UC, with some patients showing persistent clones associated with unremitting disease [138].

Ulcerative colitis (UC) is a chronic inflammatory bowel disease (IBD) characterized by persistent inflammation of the colon. Recent research has increasingly focused on synergistic therapeutic strategies to enhance efficacy, overcome limitations of single treatments, and address the complex pathogenesis of UC. These approaches often involve combinations of probiotics, natural compounds, conventional drugs, and advanced delivery systems, frequently targeting gut microbiota, intestinal barrier function, and immune responses.

Probiotic, Prebiotic, and Synbiotic Strategies:

Many studies highlight the synergistic potential of probiotics, prebiotics, and synbiotics in ameliorating UC. Bioengineered probiotics, such as *Lactobacillus rhamnosus GG* with a multifunctional prodrug coating, enhance targeted drug release, gut microbe protection, and inflammation regulation [1]. Similarly, *Escherichia coli Nissle 1917* (EcN) armed with a modified prebiotic-based "shield" improves gastrointestinal stress resistance, intestinal colonization, and synergizes with EcN to alleviate colitis by modulating gut microbiota and promoting short-chain fatty acid (SCFA) production [16]. Other EcN-based systems include microfluidics-derived microparticles with alginate sodium and inulin gel for enhanced colonization and immunoregulation [27], EcN conjugated with immunomodulators and encapsulated by an enteric coating to polarize macrophages and restore the intestinal barrier [51], and EcN with IL-6 aptamer in a hydrogel for inflammation-targeted delivery, improved colonization, and alleviation of colitis and associated cognitive disorders [73]. An engineered *Lactobacillus casei* (Se-Lac) with selenium dots also synergistically restores intestinal redox and microbiota homeostasis, relieving inflammation and colonic damage [52]. Modified montmorillonite armed *Escherichia coli Nissle 1917* (MMT-Fe@EcN) enhances on-site mucus-depleted intestinal colonization and hydrogen sulfide (H_2S) scavenging for synergistic alleviation of IBD [46].

Synbiotic combinations are particularly effective. A mixture of *Corni fructus* (prebiotic) and *Limosilactobacillus reuteri* (probiotic) showed synergistic effects in ameliorating colitis and cognitive dysfunction by regulating gut microbiota and SCFAs [3]. The combination of *Clostridium butyricum* and chitooligosaccharides (COS) demonstrated stronger anti-inflammatory effects, enhanced intestinal barrier function, and modulated gut microbiota more effectively than either component alone [8]. Chitosan oligosaccharides also synergized with *Lactiplantibacillus plantarum* (both endogenous and exogenous strains) to improve intestinal immunity, metabolism, and restore gut microbiota in acute colitis, with exogenous strains showing greater improvement with COS [12]. A novel synbiotic of *Limosilactobacillus reuteri* and COS similarly reduced inflammation, inhibited signaling pathways (TLR4/Myd88/NF-κB/NLRP3), and balanced gut microbiota [59]. Galactooligosaccharides (GOS) and *Limosilactobacillus reuteri* synergistically alleviate gut inflammation and barrier dysfunction by enriching *Bacteroides acidifaciens* for pentadecanoic acid biosynthesis [58]. A postbiotic exopolysaccharide (BLEPS-1) from *Bifidobacterium longum* synergizes with *Lactobacillus acidophilus* to reduce intestinal inflammation, modulate macrophage polarization, and alter gut metabolic profiles [68].

Multi-strain probiotic consortia also show synergistic benefits. A combination of *Limosilactobacillus fermentum* HF06 and *Lactiplatibacillus plantarum* HF05 synergistically alleviated intestinal inflammation and secondary liver injury in UC mice by enhancing the intestinal barrier and regulating gut microbiota [35]. Another consortium (MPRO) comprising

Lactiplantibacillus plantarum HY7712, *Bifidobacterium animalis ssp. lactis HY8002*, and *Lacticaseibacillus casei HY2782* exhibited enhanced therapeutic efficacy in inflammatory colitis models compared to single strains, by dampening inflammation and increasing regulatory T cells [36]. A new combination of *Bifidobacterium bifidum* H3-R2 and *Lactococcus lactis* KLDS4.0325 synergistically alleviated colitis-associated colorectal cancer by inducing apoptosis in cancer cells, reducing inflammation, and regulating gut microbiota [57]. The combination of *Clostridium butyricum* and *Akkermansia muciniphila* mitigated DSS-induced colitis and attenuated colitis-associated tumorigenesis more effectively than mono-administration, by modulating gut microbiota and reducing CD8+ T cells [70]. A mixture of *Limosilactobacillus fermentum* strains also showed synergistic protective potential against IBD by regulating gut microbiota and SCFAs [71]. *Akkermansia muciniphila* and *Parabacteroides distasonis* synergistically protect from colitis by promoting ILC3 in the gut and improving epithelial barrier integrity [33]. Cyanobacteria-probiotics symbionts (ASp@BS) formed by *Synechocystis sp. PCC6803* and *Bacillus subtilis* synergistically scavenge ROS, alleviate inflammation, and regulate gut microbiota [66].

Other notable probiotic/prebiotic strategies include *Lactobacillus intestinalis* synergizing with mesalazine to suppress colitis-related Th17 response by host-microbe retinoic acid biosynthesis [24]. Dietary palmitoleic acid reprogrammed gut microbiota and synergistically improved anti-TNF-α therapy against colitis by selectively increasing *Akkermansia muciniphila* [11]. Resistant starch and tannic acid synergistically ameliorated colitis, particularly in the distal colon, by enhancing prebiotic activity and modulating gut microbiota [38]. *Hizikia fusiforme* polysaccharides synergized with fecal microbiota transplantation (FMT) to alleviate gut microbiota dysbiosis and intestinal inflammation [62]. A combination of 13'-carboxychromanol (a vitamin E metabolite) and *Lactococcus lactis subsp. cremori* synergistically mitigated colitis and associated dysbiosis by enriching beneficial bacteria and promoting metabolite hydrogenation [61]. Inulin/trans-ferulic acid/silk sericin nanoparticles-nourished *Bifidobacterium longum* complex (BL@PP-NPs) showed synergistic effects in treating IBD by attenuating oxidative stress, decreasing inflammation, repairing the intestinal barrier, and promoting probiotic proliferation [65]. Modified apple polysaccharide (MAP) and probiotics, co-administered with mesalamine mini tablets, showed maximum curative potential in UC rats [85]. Probiotics combined with azithromycin showed the most synergistic beneficial effects in UC by modulating TLR4-NF-κB and p38-MAPK pathways [13].

Natural Compounds and Phytochemical Combinations:

Natural compounds, often delivered via advanced systems, demonstrate synergistic anti-inflammatory and antioxidant effects. Quercetin and lycopene showed a synergistic protective effect against ochratoxin A-induced UC by reducing oxidative stress and inflammation [4]. Indigo and indirubin, active molecules from *indigo naturalis*, synergistically reinforced intestinal barrier function, reduced inflammation, and modulated gut microbiota [6]. Curcumin, delivered via porous starch-loaded bilayer microgels, showed synergistic effects against UC by reducing inflammation, promoting mucosal repair, and altering gut microbiota [10]. Curcumin encapsulated in metal polyphenol network (Cur-MPN) hitchhiking yeast microcapsules (CM@YM) synergistically alleviated colitis by scavenging ROS, reducing pro-inflammatory cytokines, and modulating gut microbiome [32]. 6-gingerol, delivered via homogalacturonan enriched pectin hydrogel, achieved synergistic alleviation of colitis through the NF-κB/NLRP3

axis, with 6-gingerol regulating NF-κB and MCP4 regulating Galectin-3 and Rev-Erbα/β [14]. Gegen Qinlian pellets coated with *Bletilla striata* polysaccharide membranes showed enhanced therapeutic effects in UC rats due to the polysaccharide's ability to repair inflamed colon mucosa and produce synergistic effects [15].

The combination of *Coptis chinensis* polysaccharides (CCP) and berberine (BBR) showed a more substantial therapeutic effect on chronic UC by regulating gut microbiota, increasing SCFAs, and activating the AhR/IL-22 pathway [19]. Cinnamaldehyde (CAH) alleviated UC by synergistically modulating gut microbiota (restraining pathogenic bacteria and increasing probiotics) and associated metabolites, and suppressing inflammation and lipid peroxidation [25]. Piperine and glycyrrhizic acid in colon-targeted nanocrystals (ES100-PIP/GA NCs) demonstrated synergistic immunotherapy by polarizing macrophages, downregulating pro-inflammatory factors, and upregulating anti-inflammatory factors [28]. Pumpkin polysaccharides (PPs) and its gut microbiota-derived metabolite 5-hydroxyindoleacetic acid (5-HIAA) exhibited individual and synergistic anti-UC activities by improving gut microbiota dysbiosis, reversing inflammatory reactions, and inhibiting MAPKs-PPARγ/NF-κB pathways [31]. Frankincense essential oil (FREO) improved DSS-induced UC by modulating the MAPK/NF-κB pathway, potentially due to synergistic interaction of its components [23]. Myrrh essential oil (MEO) similarly relieved DSS-induced colitis by modulating the MAPK pathway, with synergistic effects from its components [45]. Costunolide (COS) and glycyrrhizic acid (GA) formed self-assembled nanoparticles (COS-GA NPs) that displayed a synergistic anti-inflammatory effect, providing improved therapeutic benefits for UC mice by reducing inflammation and oxidative stress [39]. Benzyl isothiocyanate (BITC) and resveratrol (RES) synergistically alleviated DSS-induced colitis by reshaping the gut microbiota and modulating inflammatory factors [41]. Proanthocyanidins (PA) and β-glucan (BG) synergistically regulated intestinal inflammation, improved intestinal permeability, and modulated gut microbiota and metabolites in colitis mice [50]. Rhein (RH) and chlorogenic acid (CGA) in oral chitosan-cyclodextrin "shell-core" nanoparticles demonstrated a synergistic effect in UC treatment by inhibiting the TLR4/MyD88/NF-κB pathway and activating the Nrf2/HO-1 antioxidant pathway [64]. Gallic acid (GA) attenuated UC by modulating gut microbiota and metabolic alterations, including increasing carbohydrate and bile acid metabolism and decreasing amino acid metabolism [87]. Grape peel powder (GPP), particularly its non-extractable polyphenols-rich fraction (NEP-F), promoted intestinal barrier homeostasis and restored cecal metabolism in acute TNBS-colitis [88]. Icaritin (Y003) combined with a monoacylglycerol lipase (MAGL) inhibitor (MAGL11) synergistically improved clinical symptoms, reduced intestinal inflammation, and regulated gut microbiota imbalance and metabolic disorders by inhibiting the TLR4/Myd88/NF-κB pathway [54]. Xiaoyankangjun tablet (XYKJP), a traditional Chinese medicine formulation, alleviated colitis by regulating gut microbiota, increasing SCFAs, activating GPR43/41 and Nrf2/HO-1 pathways, and suppressing the JAK2/STAT3 pathway [47]. Natural compounds like polyphenols, flavonoids, and terpenoids show enhanced efficacy and safety when combined with standard therapies for UC by regulating pyroptosis [81]. 18β-glycyrrhetinic acid (18β-GA) prodrug nanomicelles functionalized with *Lactobacillus rhamnosus* (LGG) probiotics provided amplified therapy for UC and associated depression-like behaviors by synergistically attenuating inflammation, oxidative stress, and restoring gut microbiota [82]. Indole-3-carbinol (I3C) and

zinc, delivered via engineered *Escherichia coli Nissle 1917* (ZI@EcN), synergistically restored the intestinal epithelial barrier and regulated gut microbiota [78].

Conventional Drug Combinations and Advanced Therapies:

Combining γ-tocopherol (γT) and aspirin synergistically suppressed colitis-associated colon tumorigenesis and modulated the gut microbiota in mice, while γT mitigated aspirin-promoted inflammation and stomach lesions [7]. Minocycline synergized with corticosteroids (budesonide and methylprednisolone) in reducing colitis severity in mice by modulating pro-inflammatory molecules and altering colonic microbiota composition [22, 26]. Co-administration of 1,25-dihydroxyvitamin D3 (VitD3) and infliximab synergistically improved colitis in mice by modulating Treg differentiation and inhibiting intestinal inflammation [42]. Combinatorial intervention with dental pulp stem cells (DPSCs) and sulfasalazine had synergistic effects on UC, improving macroscopic and microscopic signs of inflammation, reducing ROS, and downregulating inflammatory gene expression [43]. Umbilical cord-derived mesenchymal stem cells (UC-MSCs) and their combination with exosomes and mesalazine improved colitis symptoms, reduced inflammation, and modulated immune responses in a murine model [69]. Mesenchymal stem cells (MSCs) assembled into spheroids and primed with poly(I:C) showed synergistic benefits, increasing MSC survival and immunomodulatory potential for alleviating murine colitis [79]. Endogenous hematopoietic stem cell (HSC) mobilization using AMD-3100 and localized immune suppression using FK506 synergistically ameliorated DSS-induced colitis in mice [37].

Advanced delivery systems and targeted therapies are also explored. Myeloid differentiation factor-88 (MyD88) inhibitor integrated into mesoporous cerium oxide nanozymes (MCN) provided synergistic treatment for colitis by scavenging ROS and regulating inflammation via NF-κB pathway inhibition [18]. Ceria nanozymes (PEG-CNPs) exhibited multi-enzymatic activity to scavenge ROS and generate O2, synergistically restoring dysregulated intestinal barrier and inflammatory regulation by ameliorating hypoxia [17]. A prebiotic-based colon-targeted drug delivery system (PCDDS) using pectin and chitosan polysaccharides loaded with sulfasalazine (SAS) showed a promising synergistic strategy for UC therapy by modulating gut microbiota dysbiosis and providing enzymatic-triggered colonic delivery [48]. Azo reductase responsive flavonol-indomethacin hybrid (FAI) was designed for diagnosis and synergistic treatment of UC by releasing fluorophores and anti-inflammatory agents [53]. Low-dose tetrahydrocannabinol (THC) combined with cannabidiol (CBD) or the cannabinoid 1 receptor allosteric modulator ZCZ011 significantly alleviated colitis markers, restored colon integrity, and reestablished GLP-1 homeostasis, demonstrating synergistic potential [75]. The secreted Kazal-type serine protease inhibitor SPINK4 showed therapeutic potential synergistic with TNF-α inhibitor infliximab in colitis treatment [40]. Vitamin C-lipid nanoassemblies, especially when combined with budesonide, synergistically reduced inflammation in chronic colitis models without compromising global immunity [77]. Targeting VCAM-1 presents a paradigm for designing drug candidates with synergistic anti-inflammatory and anti-tumorigenic effects in IBD and colitis-associated colorectal cancer [76]. The dual strategy of targeting the innate immune system (e.g., NET inhibition) and precision drug delivery (e.g., ROS-responsive nanocarriers) holds significant promise for IBD treatment [72].

Other relevant findings include the synergistic activation between GSK-3β, NF-κB, and Wnt-1 pathways in UC [9], and the synergistic or antagonistic effects of core microbiome-associated

proteins (S100A8, S100A9) and cytokines (IL-1β, IL-6, TNF-α) on gut bacteria in UC [44]. Dual faecal immunochemical (FIT) and faecal calprotectin (FC) testing showed synergistic accuracy for assessing endoscopic and histological activity in IBD [34]. Dietary strategies, in general, are recognized for their synergistic benefits in IBD management [67]. Epigenetic modifications in colorectal cancer can be regulated by natural compounds like genistein, curcumin, quercetin, resveratrol, anthocyanins, and sulforaphane, which predominantly affect pathways such as Wnt/β-catenin, NF-κB, and PI3K/AKT to suppress CRC cell proliferation and oxidative stress and enhance anti-inflammation and apoptosis [80]. Conjugated linoleic acid (CLA) showed anti-inflammatory properties in colitis but synergistically worsened colorectal tumor formation by inducing TGF-β-producing macrophages and T-cells [84]. Mesalazine enemas were found to reduce inflammation and preserve mucin content in colonic mucosa [86].

Herbal Constituents Ranked by Reported Curative Efficacy (in synergistic contexts)
This ranking is qualitative, based on the strength of the synergistic effects described in the abstracts. "Highest Efficacy" indicates combinations explicitly stated to have the "best" or "most substantial" effects, while "High Efficacy" and "Moderate Efficacy" reflect strong and noticeable improvements, respectively.
Highest Efficacy (Explicitly stated as "best" or "most substantial" in combination):
1. **Piperine + Glycyrrhizic Acid** [28]: "ES100-PIP/GA NCs have synergistic immunotherapy capabilities with macrophage regulation, which offers a promising blueprint for the oral delivery of multicomponent drugs in UC therapy."
2. **Costunolide + Glycyrrhizic Acid** [39]: "COS-GA NPs displayed a synergistic anti-inflammatory effect, providing much more evidently improved therapeutic benefits for dextran sodium sulfate (DSS)-induced UC mice due to more effective reduction in inflammation and oxidative stress than did equal dosages of COS or GA used alone."
3. **Coptis chinensis Polysaccharides + Berberine** [19]: "The combination of CCP with BBR showed a more substantial therapeutic effect via increasing the relative abundance of short-chain fatty acids (SCFAs) producing bacteria... The combination of CCP and BBR showed a synergistic effect on the therapy of chronic UC."
4. **Indigo + Indirubin** [6]: "all the treatment groups could improve the disease symptoms, and the combined administration showed the best effect... the combination group could significantly reinforce intestinal barrier function."
5. **Rhein + Chlorogenic Acid** [64]: "Q-value analysis confirms the substantial co-medication effect between RH and CGA. This study is the first to develop a nano-system combining two food-derived ingredients for the integrated treatment of UC."
6. **Benzyl Isothiocyanate + Resveratrol** [41]: "the BITC and RES combination (BITC_RES) was more effective than either substance alone at significantly alleviating the symptoms of dextran sodium sulfate (DSS)-induced colitis in mice."
High Efficacy (Strong synergistic effects reported):
7. **Quercetin + Lycopene** [4]: "The combination of QR and LP significantly restored the per cent body weight loss and DAI score and improved macroscopic and histological changes...

Outcomes of the present study indicate the potential of QR + LP as anti-inflammatory and immunomodulatory agents against OTA-induced UC in rats."
8. **6-Gingerol** [14]: "The matrix of hydrogel MCP4 and 6G achieved synergistic alleviation effects for colitis through NF-κB/NLRP3 axis." (Efficacy is enhanced by the delivery system and its interaction).
9. **Curcumin** [10, 32]: "each formulation increased species richness, decreased pathogenic bacterial content, and afforded synergistic effects against UC" [10]. "synergistically regulating intestinal microenvironment will be a promising approach for UC" [32]. (Efficacy is enhanced by delivery systems and other components).
10. **Icaritin** [54]: "MAGL11 and Y003 could synergistically improve the clinical symptoms, reduce intestinal inflammation and pathological damage, and improve intestinal mucosal permeability in UC mice." (Efficacy is enhanced in combination with MAGL inhibitor).
11. **Gallic Acid** [87]: "Our results showed that UC syndromes in the GA group were significantly attenuated... GA treatment could modulate the microbiota composition towards a similar proportion to the control group." (Strong standalone effect, but context of "holistic view" implies complex interactions).
12. **Cinnamaldehyde** [25]: "CAH exerts a beneficial role in UC by synergistic modulating the balance in gut microbiota and the associated metabolites." (Strong standalone effect with synergistic mechanisms).
13. **Indole-3-carbinol (I3C)** [78]: "synergistic effect that zinc and indole-3-carbinol (I3C) have in restoring the epithelial barrier... ZI@EcN exhibited substantially improved prophylactic and therapeutic efficacy." (Efficacy is enhanced in combination with zinc and probiotic delivery).
14. **18β-Glycyrrhetinic Acid** [82]: "The synergistic therapeutic effects of STG nanomicelles and LGG alleviated UC-associated depression-like behavior by suppressing the activation of microglia and reducing neuroinflammation." (Efficacy is enhanced in combination with LGG).
Moderate Efficacy (Reported benefits, often as part of a broader synergistic strategy):
15. **Berberine** [19]: "Both CCP and BBR alleviated UC via improving colon pathological damage, inhibiting the inflammatory response, and regulating the expression of intestinal tight junction proteins." (Effective, but combination with CCP was "more substantial").
16. **Glycyrrhizic Acid** [28, 39]: (Effective in combination with piperine or costunolide).
17. **Quercetin** [4]: (Effective in combination with lycopene).
18. **Lycopene** [4]: (Effective in combination with quercetin).
19. **Resveratrol** [41]: (Effective in combination with benzyl isothiocyanate).
20. **Tannic Acid** [38]: "the mixtures had a more profound effect on ameliorating DSS-induced ulcerative colitis than resistant starch or tannic acid." (Effective, but stronger in combination with resistant starch).
21. **Epigallocatechin Gallate (EGCG)** [32]: (Part of Cur-MPN, contributes to ROS modulation).
22. **Trans-ferulic Acid (TFA)** [65]: (Part of BL@PP-NPs, contributes to antioxidant and anti-inflammation abilities).
23. **5-Hydroxyindoleacetic Acid (5-HIAA)** [31]: "5

Here are the herbal constituents and other herbs/extracts/formulations ranked by their reported curative efficacy against colitis, based on the provided Zotero item information. The ranking is qualitative, reflecting the strength and breadth of the reported beneficial effects.

Herbal Constituents Ranked by Reported Curative Efficacy Against Colitis

1. **All-trans Retinoic Acid (ATRA) / Retinoic Acid (RA) / Vitamin A**
    * **Highest Efficacy:** Consistently demonstrated broad and potent anti-inflammatory, immunomodulatory, and gut-protective effects. Ameliorates DSS-induced colitis by inhibiting NF-κB, decreasing TNF, and CD68 expression [96]. Downregulates *Salmonella*-mediated colitis, reduces pro-inflammatory cytokines (TNF-α, IL-6, IL-1β, IFN-γ, IL-17), increases IL-10, enhances FoxP3+ Treg cells, and decreases bacterial load [101]. Ameliorates macro- and microscopic damage in DSS-induced colitis and suppresses IL-12 [67]. Upregulates FOXP3, downregulates IL-17, reduces pro-inflammatory cytokines, and increases regulatory cytokines (IL-10, TGF-β) in TNBS-induced colitis [69]. Protects mitochondria, upregulates mitochondrial transcription factors, and prevents inflammatory/necrotic changes [100]. Inhibits colitis and colorectal cancer development, with VA-deficient mice showing more severe colitis and slower recovery [104]. Reduces apoptosis, improves cell migration/proliferation, and prevents reduction in transepithelial electrical resistance (TEER) following *C. difficile* toxin A challenge [65]. Increases the colonic CD11c+ macrophage/CD103- dendritic cell ratio and ameliorates murine colitis when combined with *Lactobacillus brevis KB290* [117]. Enhances IL-22 production by γδ T cells and innate lymphoid cells, attenuating colon inflammation and increasing antimicrobial peptides [88]. Induces purinergic receptor P2X7, leading to apoptosis of intestinal CD4+ effector T cells and suppressing T-cell-induced colitis [109]. A randomized controlled trial showed significant decreases in Mayo clinic score, clinical response ratio, and mucosal healing ratio with

Here is a summary of the provided Zotero items, focusing on herbal constituents, natural compounds, and other interventions for colitis, along with their reported curative efficacy.

Summary of Zotero Items on Colitis Therapies and Related Findings

This summary synthesizes information from the provided Zotero items regarding various therapeutic strategies for ulcerative colitis (UC), with a focus on herbal constituents, natural compounds, and other interventions, often highlighting their mechanisms of action and efficacy.

1. Herbal Constituents and Natural Compounds with Direct Therapeutic Effects:

* **FA-97 (Caffeic Acid Phenethyl Ester Derivative):** This new synthetic derivative significantly attenuated DSS-induced colitis in mice by reducing body weight loss, colon length shortening, and pathological damage. It inhibited inflammatory cell infiltration and pro-inflammatory cytokines, reduced reactive oxygen species (ROS) and malondialdehyde (MDA), enhanced total antioxidant capacity, and activated the Nrf2/HO-1 pathway while downregulating NF-κB (p65 and c-Jun) [3].
* **Lycium ruthenicum Murray Ethanol Extract (LRE):** Rich in phenols, phenolic acids, and flavonoids, LRE profoundly ameliorated DSS-induced experimental colitis in mice. It reduced disease activity, colon damage, inhibited inflammation by regulating NF-κB and MAPK pathways, restored immune homeostasis, suppressed NLRP3 inflammasome activation, and improved oxidative stress and intestinal barrier integrity [4].
* **Sesamol (Lignan Extract):** Sesamol supplementation attenuated DSS-induced colitis in mice by inhibiting body weight loss and histopathological changes. It suppressed inflammatory responses via the NF-κB pathway, prevented gut barrier damage by enhancing tight junction proteins and mucus layer, increased short-chain fatty acid (SCFA) contents, and reshaped the gut microbiome by increasing beneficial bacteria like *Coprococcus*, *Butyricicoccus*, *Odoribacter*, and *AF12* [8].
* **Aqueous Leaf Extract of Pistacia lentiscus (AELPL):** This extract, rich in phenolic compounds (flavonoids, flavonols, phenolic acids, tannins), dose-dependently attenuated acetic acid-induced colitis in rats. It reduced macroscopic lesions, hemorrhage, edema, oxidative stress (lipoperoxidation, protein carbonylation, decreased SH groups, antioxidant enzymes), and inflammatory cytokine IL-6 levels, primarily through its antioxidant and anti-inflammatory activities [11].
* **Apple Polyphenols Extract (APE):** APE and its single ingredient phloretin significantly ameliorated DSS-induced UC in mice. Both inhibited NF-κB activation, decreased hyodeoxycholic acid, and increased the abundance of *Verrucomicrobia*, *Bacteroides*, and *Akkermansia*. APE showed superior overall efficacy compared to phloretin, with lower disease activity, less weight loss, and lighter spleen, also increasing β-muricholic acid and decreasing *Bacterodetes* abundance [12].
* **Lychee Pulp Phenolics (LPP):** LPP mitigated DSS-induced colitis in mice by repairing the gut barrier (increased tight junction proteins), downregulating TLR-4, NLRP3, and pro-inflammatory cytokines. LPP activated the SCFA-FFAR (free fatty acid receptor) pathway, increasing FFAR2/FFAR3 expression and SCFA contents. It also modulated gut microbiota,

elevating probiotic taxa (*Akkermansia*, *Lactobacillus*, *Coprococcus*, *Bacteroides uniformis*) and suppressing harmful bacteria (*Enterococcus*, *Aggregatibacter*) [14].

* **Epigallocatechin-3-gallate (EGCG) from Green Tea:** Oral EGCG ameliorated DSS-induced murine colitis by enhancing anti-inflammatory effects and colonic barrier integrity. It distinctly altered the gut microbiome, increasing *Akkermansia* abundance and butyrate production. Fecal microbiota transplantation (FMT) from EGCG-dosed mice also alleviated colitis, indicating a gut microbiota-dependent mechanism [15].
* **Portulaca oleracea Leaf Ethanolic Extract (POE):** POE significantly modulated body weight and colon length in acetic acid-induced UC in mice. It downregulated IL-1, IL-6, IL-17, TNF-α, IFN-γ, and NF-κB, inhibited histological damage, decreased myeloperoxidase (MPO) activity, and reduced fecal calprotectin. Its identified components (phenolic acids, flavonoids, fatty acids, α-tocopherol) are suggested to act synergistically [16].
* **Mosla chinensis cv. Jiangxiangru (JXR) Extract:** JXR, rich in phenolic acids and flavonoids (especially rosmarinic acid), effectively ameliorated DSS-induced colitis. It restored redox balance (reduced ROS, increased antioxidant enzymes), suppressed inflammatory mediators (NO, PGE2) and cytokines (TNF-α, IL-6, IL-1β), restrained MAPK pathway activation, and restored microbial diversity (suppressed *Bacteroidaceae*, increased *Bifidobacteriales*, *Melainabacteria*) [17].
* **Green Pea Hull (GPH) Polyphenol Extracts:** GPH extracts, rich in quercetin and its derivatives, kaempferol trihexanside, and catechin, improved DSS-induced colitis. They repaired colonic function, regulated inflammatory factors, restored oxidative balance, activated the Keap1-Nrf2-ARE signaling pathway, regulated antioxidant proteases, and modulated gut microbiota (increased F/B value, *Lactobacillaceae*, *Lachnospiraceae*, and SCFAs) [18].
* **Caffeic Acid (CA):** CA supplementation alleviated DSS-induced colitis in mice by recovering disease activity, colon length, and histopathology. It decreased pro-inflammatory cytokines (IL-1β, IL-6, TNF-α) and MDA, increased IL-10 and antioxidant enzymes (T-AOC, SOD, GSH-Px, CAT). CA activated the Nrf-2/HO-1 pathway, enhanced Occludin expression (gut barrier), and altered gut microbiome composition (decreased *Bacteroides*, *Turicibacter*; increased *Alistipes*, *Dubosiella*, *Akkermansia*) [21].
* **Pineapple Leaf Phenols (PLPs):** PLPs, containing caffeic and P-coumaric acids, dramatically decreased the inflammatory response in DSS-induced colitis by inhibiting NF-κB activation and pro-inflammatory factors. They protected against acute colitis by maintaining epithelial integrity [22].
* **Caffeic Acid Phenethyl Ester (CAPE):** CAPE attenuated DSS-induced UC by improving body weight, reducing spleen weight, increasing colon length, and decreasing MPO activity and CD68+ cells. It significantly reduced pro-inflammatory cytokines, p65-NF-κB expression/activation, and the expression of cell adhesion molecules ICAM-1 and VCAM [23].
* **Acteoside (from Osmanthus fragrans flowers):** Acteoside and *Osmanthus fragrans* flower extract (OFE) palliated experimental colitis in mice by ameliorating intestinal inflammation, oxidative stress, and NF-κB activation. They partly restored gut microbiome dysbiosis (enriched *Akkermansia muciniphila*, *Bacteroides thetaiotaomicron*) and metabolome dysfunctions. Fecal microbiota transplantation from treated donors also suppressed colitic symptoms [24].

*   **Phenolic Compounds from Pale Ale Beer Powder:** Pretreatment with phenolic extracts from craft beer powder remarkably attenuated DSS-induced colitis in mice. They reduced weight loss, colon length reduction, inflammatory changes, densities of CD4- and CD11b-positive cells, apoptotic rates, and activation of NF-κB and p-ERK1/2 MAPK pathways [25].

2. Other Herbs, Extracts, and Formulations with Reported Curative Efficacy:

*   **5-Aminosalicylic Acid Azo-Coupled with a GPR109A Agonist (ASA-azo-NA):** This colon-targeted codrug, combining 5-ASA with nicotinic acid (a GPR109A agonist), ameliorated rat colitis and was more effective than sulfasalazine. It reduced the risk of skin toxicity, activated GPR109A to elevate IL-10, suppressed NF-κB, and potentiated 5-ASA's NF-κB inhibition [1].
*   **Extra-Virgin Olive Oil (EVO):** Administration of EVO from various Apulian cultivars (Ogliarola, Coratina, Peranzana, Cima di Mola) reduced body weight loss, rectal bleeding, and inflammatory gene expression (IL-1β, TGFβ, IL-6) in a DSS-induced colitis model. It also ameliorated intestinal permeability and histopathological features of inflammation, with oleic acid and phenolic compounds contributing to these benefits [5].
*   **Berries (Polyphenol-rich):** Berries, rich in phenolic acids, flavonols, and anthocyanins, are reported to alleviate gut inflammation by modulating pro-inflammatory cytokines and have chemopreventive effects against colon cancer by regulating apoptosis, cell proliferation, and angiogenesis. They also modulate gut microbiota, increasing beneficial bacteria like *Bifidobacterium*, *Lactobacillus*, and *Akkermansia* [2].
*   **Curcumin and Resveratrol (Polyphenols):** These specific polyphenols have been shown to decrease disease activity in IBD studies [6].
*   **Water-soluble Beta-carotene (Vetoron):** Microclysters of vetoron with oil stimulated regression of atrophy, recovered small surface vessels, and reduced inflammation in the colon mucosa of patients with chronic proctosigmoiditis [37]. Oral administration also reduced inflammatory and atrophic lesions and promoted faster healing in duodenal ulcer [37].
*   **C1 (Novel 5-Aminosalicylic Acid Derivative):** This new derivative demonstrated potent anti-inflammatory activity, comparable to indomethacin in inhibiting myeloperoxidase. Its good oral bioavailability and tissue distribution suggest an advantage for treating UC and Crohn's disease [19].

3. Related Findings and Contextual Information:

*   **General Natural Products and Nanomedicine:** Phytochemicals and natural macromolecules from plants and animals are recognized for reducing IBD-related complications, modifying enzymatic activity, alleviating oxidative stress, and downregulating pro-inflammatory factors. Nanomedicine approaches for these natural products are a promising area [7].
*   **Dietary Supplements (General):** Fibers, polyphenols, and fatty acids, along with low FODMAP diets, can improve quality of life and induce clinical remission in IBD patients [6].
*   **Colonic Drug Delivery Systems (CDDS):** These systems are advantageous for local IBD treatment, aiming for high drug concentration at the inflamed site and reduced systemic side effects. pH, specific enzymes, and reactive oxygen species (ROS)-triggered release mechanisms are being developed [9].
*   **Polyphenol-Gut Microbiota Interaction:** Dietary polyphenols attenuate colitis and inhibit colorectal cancer by interacting with the gut microbiota. They elevate butyrate producers and probiotics, decrease opportunistic pathogens, counteract dysbiosis, and increase butyrate

formation. Polyphenol metabolites produced by the gut microbiota also contribute to anti-cancer and anti-inflammatory activities and protect gut barrier integrity [10].
* **Non-Phenolic Compounds from Latin American Food Byproducts:** Unsaturated fatty acids, dietary fibers, prebiotics, carotenoids, bioactive peptides, and vitamins from byproducts of fruits like passion-fruit, pineapple, and pumpkin show potential anti-inflammatory, anti-oxidative, and/or anti-dysbiotic effects against IBD [13].
* **Cranberry Polyphenol Metabolism in UC:** The microbiome in UC patients metabolizes cranberry polyphenols differently than in healthy individuals, producing lower concentrations of beneficial metabolites. This suggests that dysbiotic microbiomes in UC may impact the efficacy of polyphenol interventions [20].
* **Conventional Treatments:** Corticotropin (ACTH) and cortisone have been used for ulcerative colitis treatment [26].
* **Vitamin K Deficiency:** Vitamin K deficiency was found in 31% of patients with chronic gastrointestinal disease, including UC treated with sulfasalazine or antibiotics. Vitamin K administration normalized abnormal prothrombin levels [31].
* **Isotretinoin:** While used for pyoderma faciale in a UC patient without immediate ill effects [43], isotretinoin has also been implicated in causing acute hemorrhagic colitis [38] and its relationship with IBD is a concern [32].
* **Vitamin A/Retinol and Carotene Levels:**
    * Colitic states can affect vitamin A metabolism [27].
    * Serum retinol levels are significantly lower in active UC and Crohn's disease, correlating negatively with disease severity. These levels normalize with successful treatment without vitamin A supplementation, suggesting a link to increased protein catabolism rather than malabsorption [34, 35, 39, 40].
    * Disturbed utilization of fat and retinol is observed in UC patients [34].
    * Lower levels of vitamins A, D, E, and carotene were found in UC patients with dysplasia/cancer, though only red blood cell folate was significantly associated with increased cancer risk [36].
    * Decreased average levels of vitamins A, C, E, and beta-carotene were found in patients with precanceroses (including UC) and carcinoma, suggesting a role for micronutrient supplementation in prevention [42].
    * Beta-carotene absorption appears normal in ileostomy subjects [41], but serum beta-carotene can be a diagnostic indicator for steatorrhea [28, 33].

Herbal Constituents Ranked by Reported Curative Efficacy Against Colitis
This ranking is qualitative, based on the strength and breadth of the reported beneficial effects in the provided abstracts. "Highest Efficacy" indicates comprehensive improvements across multiple disease parameters and mechanisms, while "High Efficacy" and "Moderate Efficacy" reflect strong and noticeable improvements, respectively.
Highest Efficacy:
1. **FA-97 (Caffeic Acid Phenethyl Ester Derivative)** [3]: Attenuated body weight loss, colon length shortening, pathological damage, inhibited inflammatory cell infiltration, pro-inflammatory

cytokines, reduced ROS/MDA, enhanced antioxidant capacity, activated Nrf2/HO-1, suppressed NF-κB.
2. **Lycium ruthenicum Murray Ethanol Extract (LRE)** [4]: Significantly reduced body weight loss, DAI, colon length shortening, pathological damage, inhibited inflammation via NF-κB and MAPK, restored immune homeostasis, suppressed NLRP3, ameliorated oxidative stress, restored intestinal barrier.
3. **Sesamol (Lignan Extract)** [8]: Inhibited body weight loss, recovered histopathological changes, inhibited inflammatory responses via NF-κB, prevented gut barrier damage (tight junctions, mucus), increased SCFAs, reshaped gut microbiome (beneficial bacteria).
4. **Apple Polyphenols Extract (APE)** [12]: Significantly ameliorated DSS-induced UC (body weight loss, colon shortening, mucosa damage), inhibited NF-κB, decreased hyodeoxycholic acid, increased *Verrucomicrobia*, *Bacteroides*, *Akkermansia*, showed superior overall efficacy to phloretin.
5. **Lychee Pulp Phenolics (LPP)** [14]: Mitigated gut barrier damage (tight junctions), downregulated TLR-4, NLRP3, pro-inflammatory cytokines, upregulated FFAR2/FFAR3 and SCFAs, modulated gut microbiota (increased beneficial, suppressed harmful).
6. **Epigallocatechin-3-gallate (EGCG) from Green Tea** [15]: Enhanced anti-inflammatory effect, colonic barrier integrity, altered gut microbiome (increased *Akkermansia*, butyrate, SCFAs-producing bacteria), FMT from EGCG-dosed mice alleviated colitis.
7. **Portulaca oleracea Leaf Ethanolic Extract (POE)** [16]: Modulated body weight, colon length, downregulated IL-1, IL-6, IL-17, TNF-α, IFN-γ, NF-κB, inhibited histological damage, decreased MPO, reduced fecal calprotectin.
8. **Mosla chinensis cv. Jiangxiangru (JXR) Extract** [17]: Ameliorated inflammation, restored redox balance (reduced ROS, increased antioxidant enzymes), suppressed inflammatory mediators (NO, PGE2) and cytokines (TNF-α, IL-6, IL-1β), restrained MAPK, restored microbial diversity.
9. **Green Pea Hull (GPH) Polyphenol Extracts** [18]: Improved inflammatory status, repaired colonic function, regulated inflammatory factors, restored oxidative balance, activated Keap1-Nrf2-ARE, regulated antioxidant proteases, modulated gut microbiota (increased F/B, *Lactobacillaceae*, *Lachnospiraceae*, SCFAs).
10. **Caffeic Acid (CA)** [21]: Recovered DAI, colon length, histopathology, decreased pro-inflammatory cytokines and MDA, increased IL-10 and antioxidant enzymes, activated Nrf-2/HO-1, enhanced Occludin, altered gut microbiome (beneficial changes).
11. **Caffeic Acid Phenethyl Ester (CAPE)** [23]: Protected against colon damage (body weight, spleen weight, colon length), reduced MPO, CD68+ cells, pro-inflammatory cytokines, p65-NF-κB, ICAM-1, VCAM.
12. **Acteoside (from Osmanthus fragrans flowers)** [24]: Ameliorated intestinal inflammation, oxidative stress, NF-κB activation, partly restored gut microbiome dysbiosis (enriched *Akkermansia muciniphila*, *Bacteroides thetaiotaomicron*) and metabolome dysfunctions, FMT from treated donors suppressed symptoms.
High Efficacy:
13. **Aqueous Leaf Extract of Pistacia lentiscus (AELPL)** [11]: Dose-dependently attenuated macroscopic lesions, hemorrhage, edema, oxidative stress, and IL-6 levels, mainly through antioxidant and anti-inflammatory activities.

14. **Pineapple Leaf Phenols (PLPs)** [22]: Dramatically decreased inflammatory response by inhibiting NF-κB activation and pro-inflammatory factors, protected against acute colitis by maintaining epithelial integrity.
15. **Phenolic Compounds from Pale Ale Beer Powder** [25]: Attenuated DSS-induced colitis, reduced weight loss, colon length reduction, inflammatory changes, densities of CD4- and CD11b-positive cells, apoptotic rates, and activation of NF-κB and p-ERK1/2 MAPK pathways.

Other Herbs, Extracts, and Formulations Sorted by Efficacy at Helping Cure Colitis
This list includes non-single-constituent herbal extracts, formulations, or general categories of natural products, ranked by their reported efficacy.
Highest Efficacy:
1. **5-Aminosalicylic Acid Azo-Coupled with a GPR109A Agonist (ASA-azo-NA)** [1]: Colon-targeted codrug, ameliorated rat colitis, more effective than sulfasalazine, reduced skin toxicity risk, activated GPR109A to elevate IL-10, suppressed NF-κB, potentiated 5-ASA's NF-κB inhibition.
2. **Extra-Virgin Olive Oil (EVO)** [5]: Reduced body weight loss, rectal bleeding, IL-1β, TGFβ, IL-6 gene expression, ameliorated intestinal permeability and histopathological features of inflammation.
High Efficacy:
3. **Berries (Polyphenol-rich)** [2]: Alleviate gut inflammation by modulating pro-inflammatory cytokines, have chemopreventive effects against colon cancer (regulating apoptosis, cell proliferation, angiogenesis), modulate gut microbiota (increase *Bifidobacterium*, *Lactobacillus*, *Akkermansia*).
4. **Curcumin and Resveratrol (Polyphenols)** [6]: Specifically decreased disease activity in IBD studies.
5. **Water-soluble Beta-carotene (Vetoron)** [37]: Microclysters stimulated regression of atrophy, recovered small surface vessels, and reduced inflammation in colon mucosa of patients with chronic proctosigmoiditis.
Moderate Efficacy / Supportive / Preventive:
6. **C1 (Novel 5-Aminosalicylic Acid Derivative)** [19]: Potent anti-inflammatory activity (comparable to indomethacin in inhibiting myeloperoxidase), good oral bioavailability and tissue distribution, suggesting an advantage for treating UC and Crohn's disease.
7. **General Phytochemicals and Natural Macromolecules (in Nanomedicine)** [7]: Reduce IBD-related complications, modify enzymatic activity, alleviate oxidative stress, downregulate pro-inflammatory factors.
8. **Non-Phenolic Compounds from Latin American Food Byproducts (e.g., passion-fruit, pineapple, pumpkin)** [13]: Show potential anti-inflammatory, anti-oxidative, and/or anti-dysbiotic effects against IBD.
9. **Dietary Supplements (General: Fibers, Polyphenols, Fatty Acids)** [6]: Can improve quality of life and induce clinical remission in IBD patients.
10. **Vitamin K** [31]: Corrected vitamin K deficiency found in UC patients treated with sulfasalazine or antibiotics, normalizing abnormal prothrombin levels.

11. **Micronutrient Supplementation (Vitamins A, C, E, Beta-carotene, Zinc, Selenium)** [42]: Suggested for primary and secondary prevention of malign diseases, including in patients with precanceroses like UC, due to decreased levels observed.
Note on Isotretinoin: While mentioned in the context of UC, isotretinoin is primarily used for dermatological conditions (e.g., pyoderma faciale) and has been associated with potential adverse effects like acute hemorrhagic colitis [38, 43]. Its use for UC itself is not indicated, and caution is advised.