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Meta-analysis of the Therapeutic Impact of Cannabinoids in Inflammatory Bowel Disease

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Background: With the increasing legalization of medical and recreational cannabis, patients and providers have growing interest in the role of cannabinoids in treating inflammatory bowel disease. Prior meta-analysis has shown inconclusive evidence for efficacy of cannabinoids. We sought to produce an up-to-date meta-analysis that pools new data to evaluate the therapeutic effects of cannabinoids in both Crohn's disease (CD) and ulcerative colitis (UC).

Methods: PubMed, Embase, CENTRAL and CINAHL were queried for randomized-controlled trials evaluating the impact cannabinoids in CD or UC. Random effects modeling was used to compute pooled estimates of risk difference. Heterogeneity was assessed using I^2.

Results: Eight studies, including 4 studies of CD, 3 studies of UC, and 1 study of both diseases met inclusion criteria. Among 5 studies of CD, a statistically significant decrease in clinical disease activity following intervention was observed (risk ratios [RR], −0.91; 95% CI, CI:1.54 to CI:0.28, I^2 = 71.9%). Clinical disease activity in UC was not significantly lower in the pooled analysis (RR, −2.13; 95% CI, −4.80 to 0.55; I^2 = 90.3%). Improvement in quality of life (QoL) was observed in both CD and UC combined (RR, 1.79; 95% CI, 0.92-0.2.66; I^2 = 82.8%), as well as individually. No differences were observed in the analysis on endoscopic disease activity and inflammatory markers.

Conclusions: This meta-analysis of clinical trials suggests that cannabinoids are associated with improved quality of life in both CD and UC, as well as improved disease activity but not inflammation.

Lay Summary

This updated systematic review and meta-analysis suggests that cannabis for inflammatory bowel disease may improve quality of life and disease activity but not inflammation.

Key Words: Crohn's disease, ulcerative colitis, inflammatory bowel disease, meta-analysis, cannabis, marijuana, cannabinoid derivatives

Introduction

The Rohrabacher-Farr amendment of 2014 permitted individual states to allow the use of cannabis for medical purposes in the United States. Since then, 44 states have passed legislation allowing the use of cannabinoids. As of April 2024, recreational marijuana use has been legalized in 25 states, with another 5 states decriminalizing recreational use.[1,2] It is estimated that marijuana use has expanded to up to 19% of Americans.[3] While the exact prevalence of cannabinoid use among inflammatory bowel disease (IBD) patients remains unclear, a growing body of evidence indicates it is sizeable. Inflammatory bowel disease patients are more likely to have ever tried cannabinoids compared with those without IBD, to start using at a younger age (age 15.7 years vs 19.6 years), and to use at least 3 times per day (65% vs 19%).[4] Up to 50% of IBD patients using cannabinoids do so for symptom relief. When used for this purpose, cannabinoids are more often used multiple times a day compared with infrequent use among IBD patients who use recreationally.[5] Marijuana contains over 100 phytocannabinoids that activate endogenous cannabinoid receptors with differing affinity and effect. Preparations containing all phytocannaboids derived from a particular strain of the marijuana plant, including various amounts of the psychogenic compound delta-9-tetrahydrocannabinol (THC), are available for inhalational use as dried leaves or extracts, orally absorbed tinctures, and topical preparations. Purified cannabidiol (CBD) and full spectrum tinctures with minimal or no THC are also available for medicinal use.

Significant interest has been sparked concerning cannabinoids in treating IBD as the endocannabinoid system plays a role in gastrointestinal functions including motility, homeostasis, inflammation, neuroprotection, and neuromodulation.[6,7] Given that the underlying pathophysiology of IBD is characterized by inflammation in combination with abdominal pain and diarrhea, cannabinoids have been postulated to be a promising therapeutic. In line with these theories, surveys among IBD populations

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Key Messages

What is already known? Cannabis has been associated with clinically significant symptom improvement in inflammatory bowel diseases through unknown mechanisms.

What is new here? This updated systematic review and meta-analysis suggests that cannabinoids for inflammatory bowel disease may improve quality of life and disease activity but not inflammation.

How can this study help patient care? Cannabinoids could be considered an adjunct therapy for patients with medically refractory disease or those with persistent symptoms despite resolution of objective measures of inflammation, but the available data do not support use of cannabinoids in place of approved medications that reduce inflammation and improve symptoms.

have observed strong associations between disease severity and cannabinoid usage,[5,8,9] with up to 45% of survey participants endorsing cannabinoid usage to treat symptoms including abdominal pain, diarrhea, nausea, and poor appetite.[10] Accordingly, there is a rising interest in the therapeutic properties of cannabis for the symptom and disease management of IBD.

Over the past decade, multiple clinical trials have studied the therapeutic properties of cannabinoids in CD and UC. Doeve et al published a meta-analysis of studies available prior to 2020 including 15 nonrandomized studies and 5 randomized-controlled trials (RCTs) of cannabinoids in IBD.[11] No difference in rates of clinical remission or improvement of inflammatory biomarkers were seen with cannabinoid use.[11] However, the meta-analysis showed significant differences in clinical symptoms and quality-of-life (QoL) scores.[11] Since then, additional RCTs have been published[12–14] supporting the need for an updated review of the literature.

In this meta-analysis of clinical trials, we sought to systematically examine the current literature to determine the therapeutic effects of cannabinoids in both CD and UC. Our primary aim was to identify whether cannabinoids improve IBD symptom scores or quality of life indices. We secondarily aimed to test whether objective measures of inflammatory disease activity, such as endoscopic scores and laboratory measures, improved among those using cannabinoids.

Materials and Methods

This review was conducted according to Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.[15] A detailed study protocol was registered with The International Prospective Register of Systematic Reviews (PROSPERO; registration number CRD42023436904).

Search Strategy

PubMed, Embase, CENTRAL, and CINAHL were queried for studies evaluating the impact of cannabinoids in CD and/or UC. The search strategy was organized with the following syntax: ((Crohn's disease OR ulcerative colitis OR inflammatory bowel disease) AND (cannabis OR marijuana OR cannabinoid derivative)). Studies were identified from inception until the date of the search in July 2023.

Inclusion and Exclusion Criteria

To address the primary aim of this study, only RCTs comparing the effect of cannabinoids to placebo among human patients with IBD were eligible for inclusion. For inclusion, studies had to report outcomes data, including subjective assessments of symptom burden, and objective evaluation of disease activity such as endoscopic evaluation or pertinent laboratory data. Both peer-reviewed publications and preliminary data presented as a published abstract were allowed. Studies published in abstract form were reviewed by 2 authors (H.K. and C.S.) to ensure the study population was not duplicated in a subsequent peer-reviewed publication. The search was limited to articles written in English. No restrictions on publication date or country of origin were applied.

Data Extraction

Covidence systematic review software (Veritas Health Innovation, Melbourne, Australia) was used to identify studies and abstract data from published studies. Two investigators (H.K. and C.S.) independently screened abstracts of search results and performed full-text reviews of included studies to evaluate eligibility. Discrepancies in voting were resolved by discussion and consensus with a third author (J.L.). Two investigators (H.K. and C.S.) independently extracted pertinent data from included studies. Extracted data included author, publication year, study location, IBD classification, study aim, study design, duration, inclusion and exclusion criteria, primary and secondary outcomes, particular study drug and placebo, age, IBD duration, smoking status, past IBD medications (immunomodulators, biologics), and current IBD medications (immunomodulators, biologics, steroids). We extracted a subjective assessment of IBD-related symptoms (Crohn's Disease Activity Index [CDAI] for CD and the Lichtiger Score for UC), patient-reported outcomes (eg, stool frequency and quality of life), and adverse events were also recorded as available. When reported, most studies used the Simple Endoscopic Score for CD (SES-CD) and Mayo Endoscopic Score for UC to assess endoscopic disease activity. Laboratory-based markers of inflammatory activity including C-reactive protein (CRP) and fecal calprotectin were recorded.

Statistical Analysis

When data were reported separately for intention to treat and "per protocol," only the intention to treat data were incorporated into our analysis. For summary statistics reported as a median, the median was assumed to the equivalent to the mean for analysis purposes. For studies that reported an interquartile range (IQR) or range, the standard deviation was estimated as IQR/1.35 and range/4, respectively.[16] Outcomes of interest for CD and UC were compared using a random-effects model to estimate pooled risk ratios (RRs) with 95% confidence intervals (CIs) for categorical outcomes and standardized mean difference (SMD) with 95% CI for continuous outcomes. Studies were weighted according to the variance estimates to determine their contribution to the final summary statistic. Statistical heterogeneity was assessed using the Inconsistency Index (I^2), with values greater than 50% indicating significant heterogeneity. For all analyses, $P < .05$ was considered statistically significant. Potential bias among included studies were assessed using the Cochrane Risk of Bias 2 tool.[17] The tool grades the risk of bias of cohort studies

across 5 domains using a 3-outcome scale (eg, low risk, some concerns, high risk). Publication bias was assessed using funnel plots and the Egger test as appropriate. Covidence software was used to identify and screen studies for inclusion. Statistical analyses were conducted using Stata Statistical Software, version 17 (College Station, Texas).

Results

Search Results

Our initial screen identified 222 unique studies, with 1 additional study that was included from our literature review as outlined in the PRISMA diagram (Figure 1A). Among these, 18 studies were selected for full-text review. Eight studies[8,12,13,18–22] were ultimately selected for the meta-analysis based on satisfaction of the inclusion criteria.

Study Characteristics

Table 1 summarizes the study characteristics of the 8 RCTs included in the meta-analysis. All included studies were published between 2013 and 2021. Two studies, Naftali 2018 (UEG)[19] and Naftali 2018 (JCC)[18] represented unique data published in abstract form from conference proceedings, while the remaining studies were full-text manuscripts. Seven studies were conducted in Israel, of which 6 were conducted by the same research group. The only multicenter trial was conducted in the United Kingdom. The meta-analysis included 282 participants, of which 141 were in the control group and 142 were in the treatment group. Four studies evaluated patients with CD, 3 studies evaluated patients with UC, and 1 study evaluated both patients with CD and UC. Seven studies had a study duration of 8 weeks, while 1 study had a study duration of 10 weeks, including 2 weeks of dose escalation and 8 weeks of maintenance therapy. Study drug included marijuana cigarette, as well as cannabis oils and capsules containing a range of CBD and THC doses that varied from 20 to 500 mg and 11.5 to 230 mg, respectively. The populations were similar with respect to medications including immunomodulators, biologics, and steroids at baseline (Supplementary Figure 1). Six studies reported either CDAI or Lichtiger score for CD and UC, respectively. Quality of life was evaluated with the 36-item Short Form Survey (SF-36) in 4 studies. Objective assessment of endoscopic activity was reported by 3 studies. Five studies reported CRP levels, and 3 reported fecal calprotectin levels.

Bias Assessment

The risk of inherent bias among included studies is outlined in Figure 1B. Only 1 study, Naftali 2021,[12] was graded as low risk of bias. Three studies had some concerns, while 4 were at high risk for bias.

Symptomatic Improvement

All 5 RCTs among patients with CD reported symptom-based measures of disease activity using CDAI scores. There was no difference in symptom scores between cannabinoids and placebo at baseline (RR, –0.22; 95% CI, –0.74 to 0.30; I^2 = 63.2%; Figure 2A). Following intervention, those using cannabinoids had a significantly lower CDAI score compared with placebo (RR, –0.91; 95% CI, –1.54 to –0.28, I^2 = 71.9%, Figure 2A). However, substantial heterogeneity was observed and funnel plots showed qualitative asymmetry indicating possible publication bias (Supplementary Figure 2A).

Two studies involving UC reported symptom-based outcomes via the Lichtiger Score. There was no statistical

Figure 1. A, PRISMA flow diagram of the literature search and screening process for the meta-analysis of RCTs evaluating cannabinoid therapies in IBD patients. B, Risk of bias for the 8 studies included in the meta-analysis. Assessment utilized 5 domains (D1-5): randomization process, deviations from the intended interventions, missing outcome data, measurement of the outcome, and selection of the reported result.

Table 1. Characteristics of included RCTs evaluating cannabinoid therapies in IBD.

Study	Country	IBD Type	Study Duration (weeks)	Treatment		Study Size (*n*)		Outcomes				
				Formulation	Max Dose	Control	Treatment	Disease Activity Score	Endoscopic Score	CRP	Fecal Calprotectin	QoL
Naftali 2013	Israel	CD	8	2 cigarettes, each with 0.5g dried cannabis flowers	230mg THC	10	11	X		X		
Naftali 2017	Israel	CD	8	2ml of 5mg/ml CHD oil twice daily	20mg CBD	9	10	X		X		
Naftali 2018 (UEG)	Israel	CD	8	cannabis oil with 15% CBD and 4% THC		23	23	X	X	X	X	
Naftali 2021 (JCC)	Israel	CD	8	1-20 drops of Avidekel oil twice daily before meals	320mg CBD + 80mg THC (median dose 80mg CBD and 20mg THC)	26	30	X	X	X	X	X
Matalon 2021	Israel	CD	8	1-20 drops of Avidekel oil twice daily before meals	320mg CBD + 80mg	17	13	X				X
		UC	8	1 cigarette, each with 0.5g dried cannabis flowers	11.5mg THC	10	9	X				X
Naftali 2018 (JCC)	Israel	UC	8	2 cigarettes, each with 0.5g dried cannabis flowers	23mg THC	28[a]				X		
Irving 2018	UK	UC	10	1-5 capsules of 50mg CBD-rich botanical extract twice daily	500mg CBD	31	29		X	X		
Naftali 2021 (PLOS)	Israel	UC	8	1 cigarette, each with 0.5g dried cannabis flowers	80mg THC	15	17	X			X	X

[a]Naftali 2018 (JCC) reported a total number of 28 participants without differentiating control from treatment groups.

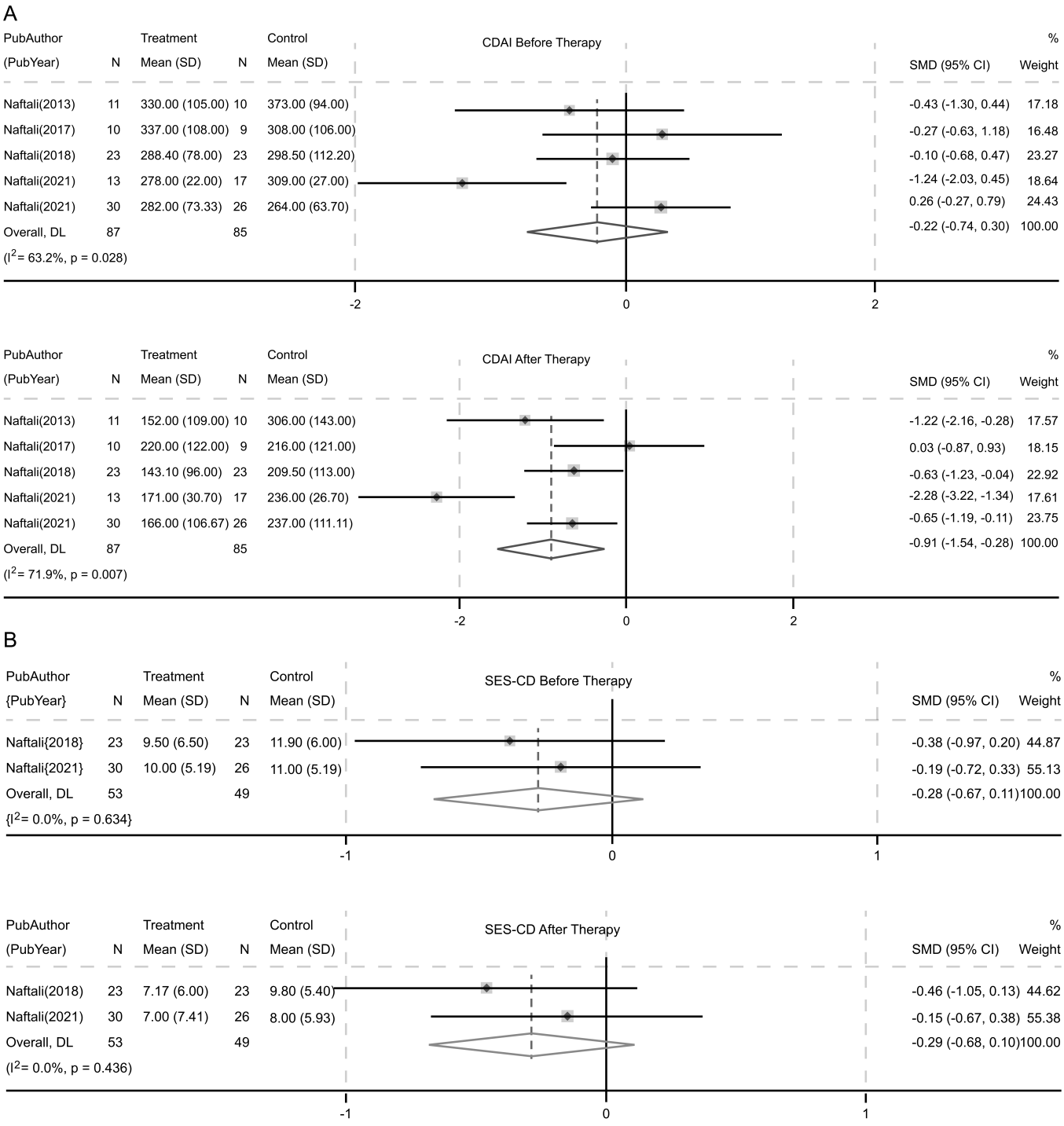

Figure 2. A, Forest plot of 5 studies evaluating CDAI scores before and after cannabinoid therapy in CD patients. B, Forest plot of 2 studies evaluating SES-CD scores before and after cannabinoid therapy in CD patients.

difference in symptom burden between the control and treatment groups before (RR, –0.87; 95% CI, –2.61 to 0.88; I^2 = 86.3%) and after intervention (RR, –2.13; 95% CI, –4.80 to 0.55; I^2 = 90.3%, Figure 3). Substantial heterogeneity was observed between these 2 studies.

Quality of Life

Four studies reported QoL outcomes using the SF-36 metric, including 2 studies in CD, 1 study in UC, and 1 study in both CD and UC cohorts. One study, Naftali 2018,[19] reported QoL scores after intervention with the study drug but not at baseline. No difference in QoL was observed prior to intervention (RR, –0.51; 95% CI, –0.49 to 0.19; I^2 = 0.0%, Figure 4). Following treatment with cannabinoids, there was improvement in QoL among all IBD patients (RR, 1.79; 95% CI, 0.92-0.2.66; I^2 = 82.8%, Figure 4). This was also observed in both UC and CD cohorts individually. Substantial heterogeneity was observed with funnel plots showing qualitative asymmetry, indicating the possibility of publication bias (Supplementary Figure 2B).

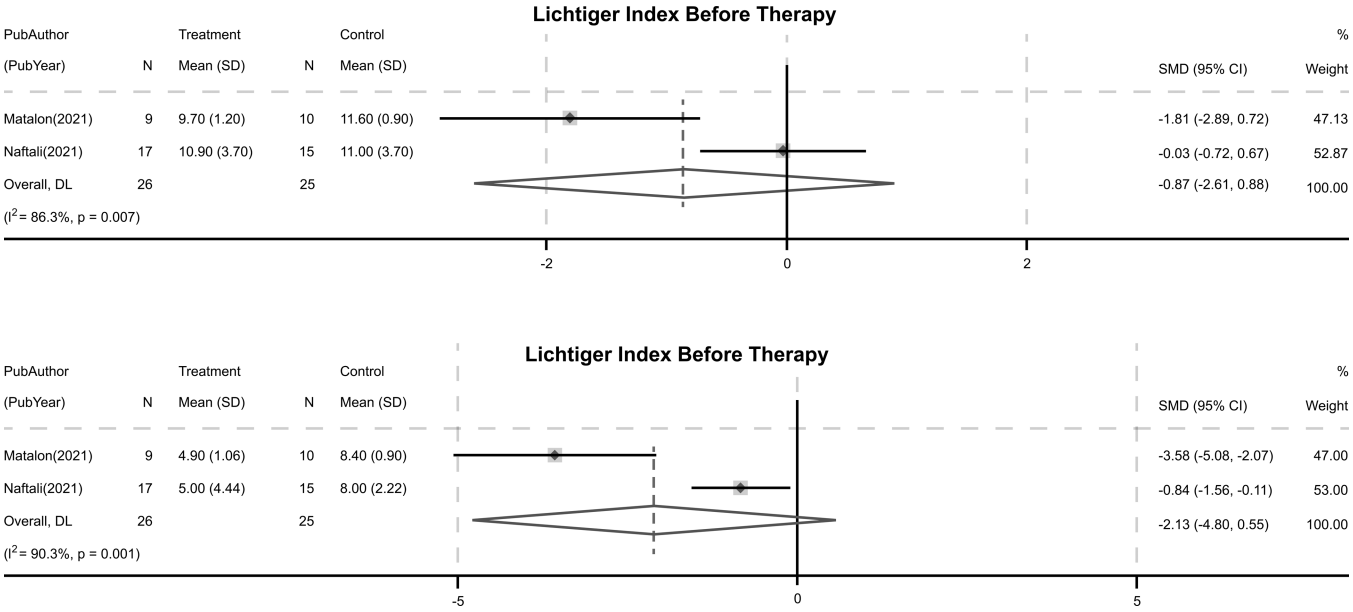

Figure 3. Forest plot of 2 studies evaluating Lichtiger scores before and after cannabinoid therapy in UC patients.

Endoscopic Disease Activity

Two studies reported objective markers of disease activity following cannabinoid treatment for those with CD. There was no statistical difference in SES-CD scores between the control and treatment groups before (RR, –0.28; 95% CI, –0.67 to 0.11; $I^2 = 0.0\%$) or after intervention (RR, –0.29; 95% CI, –0.68 to 0.10; $I^2 = 0.0\%$, Figure 2B). Only 1 study reported endoscopic disease activity in UC. There was no statistical difference in Mayo scores between the control and treatment groups before (RR, –0.02; 95% CI, –0.71 to 0.67) and after intervention with the study drug (RR, –0.27; 95% CI, –0.97 to 0.42).

Inflammatory Markers

Five studies reported CRP levels, including 4 studies in the CD and 1 study in the UC cohort. Among UC patients, there was a lower CRP concentration than in controls both before (RR, 3.23; 95% CI, 2.16-4.30) and after cannabinoid use (RR, 1.21; 95% CI, 0.45-1.97, Figure 5). Conversely, no difference in CRP level was found among those with CD comparing those treated with placebo vs cannabinoid both before (RR, 0.29; 95% CI, –0.84 to 1.42; $I^2 = 91.1\%$) and after intervention (RR, 0.62; 95% CI, –0.14 to 1.39; $I^2 = 81.6\%$, Figure 5). Substantial statistical heterogeneity was observed.

Three studies reported fecal calprotectin levels including 2 studies in CD and one study in UC. Pooling CD and UC, there was no statistical difference before (RR, –0.28; 95% CI, –1.43 to 0.86; $I^2 = 89.9\%$) and after intervention (RR, –0.41; 95% CI, –1.41 to 0.58; $I^2 = 86.7\%$, Figure 6).While there was a statistically significant difference between the control and treatment groups in the UC study, it was present both before (RR, –1.67; 95% CI, –2.49 to –0.86) and after treatment (RR, –1.62; 95% CI, –2.43 to –0.82, Figure 6). A high degree of heterogeneity was observed.

Discussion

While there has been great interest in the impact of cannabinoids in treating inflammation and pain in IBD, prior meta-analyses have been limited by the small number of RCTs with the need for additional and larger RCTs to support more robust conclusions.[11,23] Our study provides an updated meta-analysis that reviews the current literature including several recently completed RCTs.

Consistent with conclusions drawn from the meta-analysis by Doeve et al, our meta-analysis supports the role of cannabinoids in improving patient QoL in both UC and CD.[11] Our analysis also suggests that medical marijuana may significantly decrease disease activity in CD as observed through differences in CDAI scores summarized from 5 studies. However, the completed studies suggest that cannabinoids do not improve inflammatory biomarkers or endoscopic scores in CD. Moreover, the existing evidence does not support a beneficial effect of cannabinoids on inflammatory biomarkers, disease scores, or endoscopic scores in UC.

The beneficial impact of cannabinoids on disease activity in CD but not in UC contrasts with most prescription medications which are effective in both CD and UC and reduce endoscopic and biomarker evidence of inflammation. Our findings of reduction in CDAI scores without statistically significant reductions in inflammatory biomarkers or endoscopic scores in CD lead us to hypothesize that the positive effects of cannabinoids may be related to analgesic and other psychotropic effects given that abdominal pain and other systemic symptoms are typically greater in CD than UC. This hypothesis is consistent with several prior studies where patients most commonly reported using marijuana to manage abdominal pain, poor appetite, and nausea.[8,24] In patients with cancer-related pain, some but not all studies have suggested a beneficial effect of cannabinoids.[25,26] In contrast, there is more consistent evidence that cannabinoids improve noncancer pain.[27] Thus, it is possible that cannabinoids could be used as a means to reduce use of opiate medications for patients with IBD. While opiate medication use in IBD has been linked to increased mortality and complications,[28,29] it is unknown whether this is directly linked to the opiate medication, to masking of other symptoms, or through other mechanisms. Thus, future studies are needed to assess

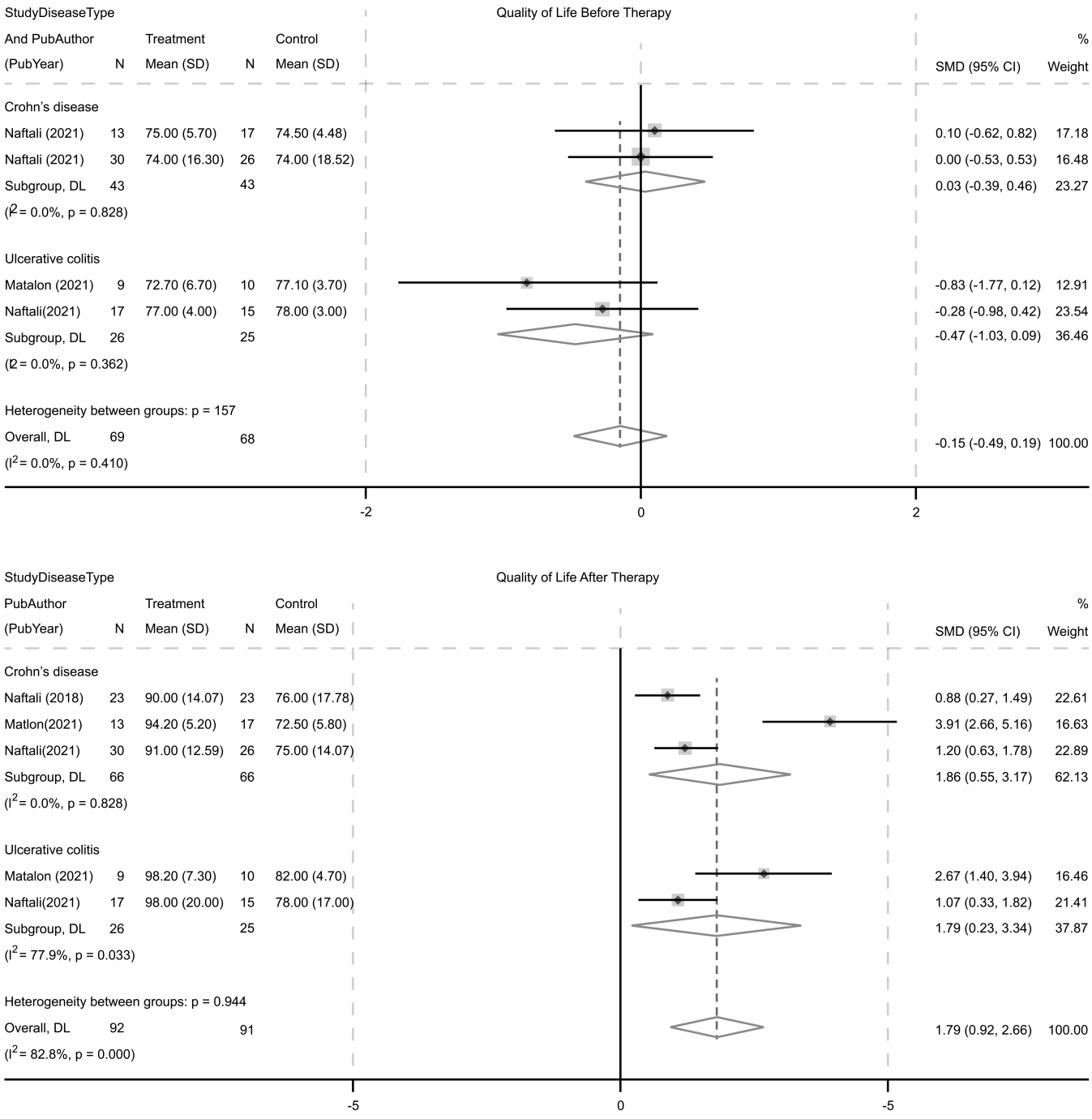

Figure 4. A, Forest plot of four studies evaluating QoL scores before and after cannabinoid therapy in CD and UC patients.

whether cannabinoid use is also associated with worse long-term outcomes.

Whether improvement in quality of life in the cannabinoids treated patients is directly related to improvement in IBD symptoms vs the inherent psychotropic properties of THC is difficult to assess from the completed RCTs. Importantly, there is a high baseline prevalence of anxiety and depression amongst patients with IBD which increases with higher levels of disease activity,[30] but the relationship between cannabis use and depression remains incompletely elucidated. Weiss et al observed that patients who use marijuana had more days with poor mental health but less depression symptoms.[4] Based on the lack of data to support improvement in inflammatory markers with cannabinoid therapies, our review suggests that the psychotropic properties of THC may play a larger role or that there are unidentified mechanisms at play.

Despite the improvement in quality of life and symptom control with cannabis use in IBD, concerns persist regarding potential risks associated with its use. In a retrospective survey exploring adverse events among cannabinoid users with IBD, 19.5% of respondents experienced a side effect. Most common were mild effects including euphoria, drowsiness, xerostomia, anxiety/depression, and headaches. Importantly, the majority of cannabinoid users (95%) remained compliant

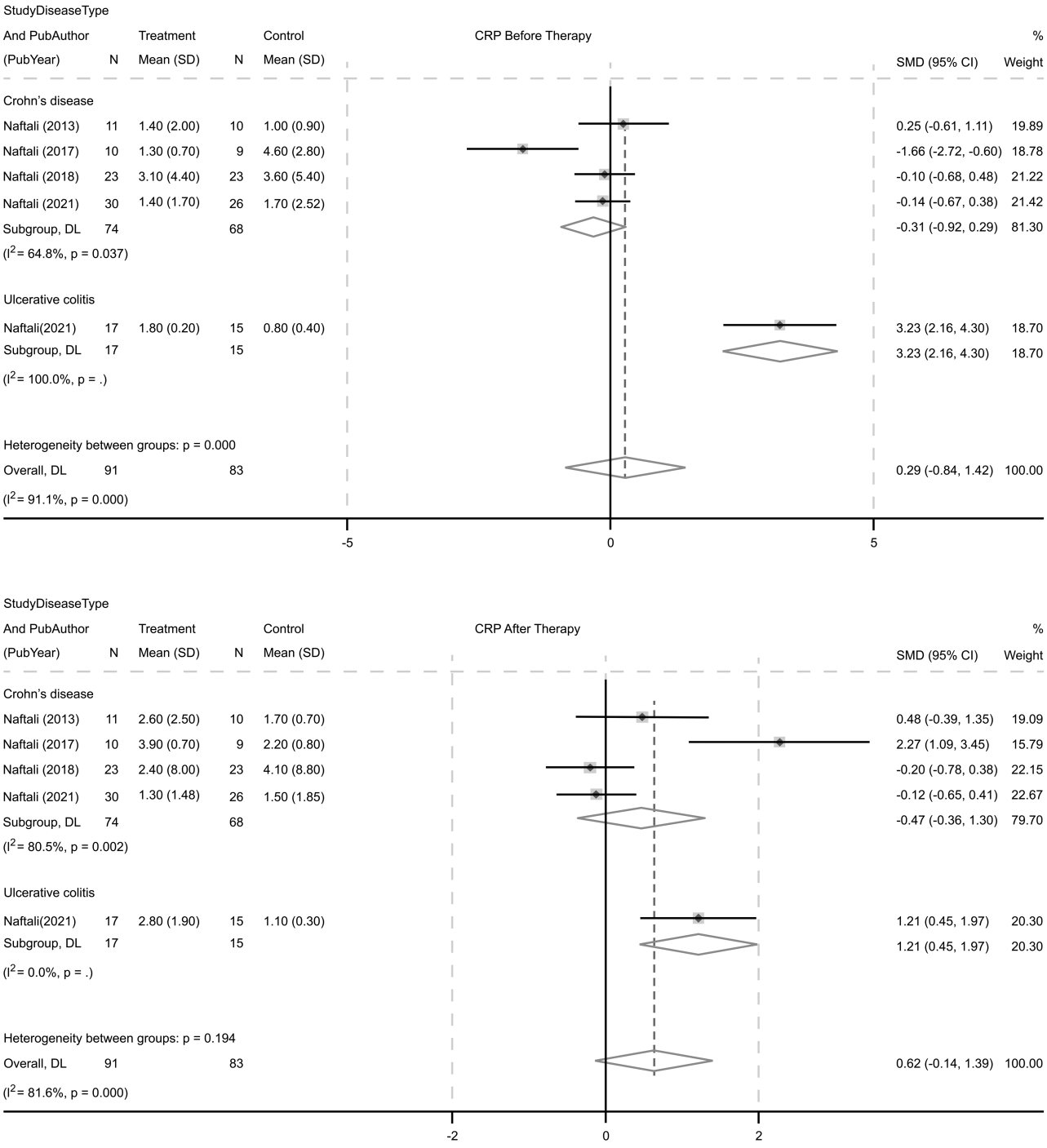

Figure 5. Forest plot of 5 studies evaluating CRP values before and after cannabinoid therapy in CD and UC patients.

with their IBD medication regimen.[31] While the drug was well-tolerated, users of cannabis with IBD tend to have higher rates of anxiety and depression than nonusers.[32] High levels of depression, impulsivity, and use of cannabis as a coping strategy were found on the Substance Use Risk Profiles Scale, indicating a potential risk of cannabis users toward addictive behavior.[33] Current and potential users of cannabinoids should be queried for psychosocial and mental health risk factors that would warrant referral. Cannabis use has also been associated with an increased risk for IBD-related surgery,[34] Although a large population-based analysis did not reproduce that finding, cannabis users with IBD did have increased rates of steroid and opioid use, as well as ED visits and need for hospitalization.[35] Whether these associations are caused by cannabis itself or other factors such as use for symptom control among patients with more refractory disease requires further clarification. Overall, further high-quality and sufficiently powered studies are required to accurately assess the risks of cannabinoid use in IBD.

Our analysis is bound by a few limitations. While this study builds upon prior meta-analyses by doubling the number of participants, the analysis is still limited by a relatively small

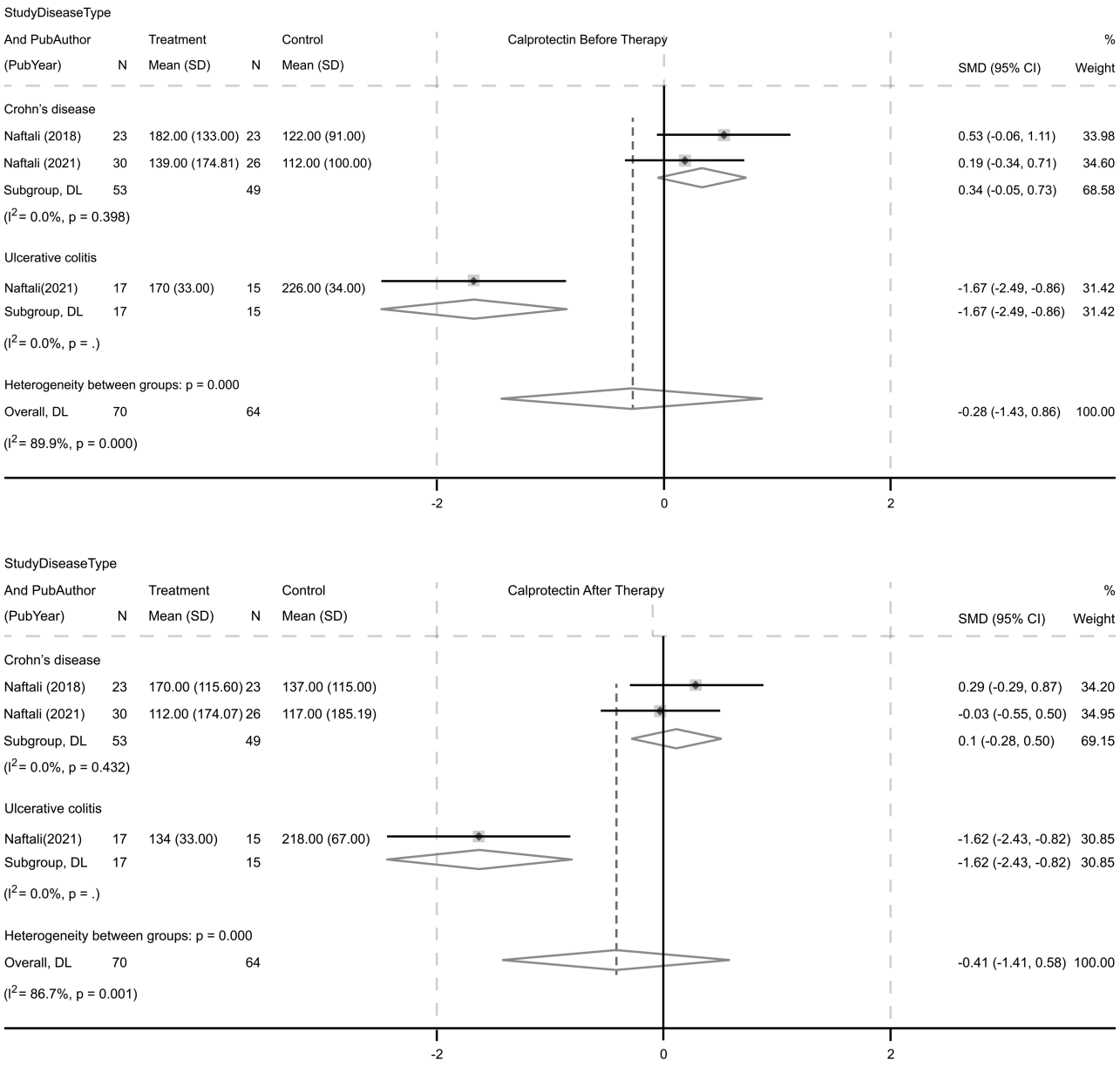

Figure 6. Forest plot of 3 studies evaluating fecal calprotectin values before and after cannabinoid therapy in CD and UC patients.

number of RCTs and participants. We elected not to include observational studies due to concern about incomplete adjustment for confounders in observational studies of cannabinoid use. While we included only RCTs, residual confounding is still a potential problem given the small sample size of most of the trials. Only 2 studies examined disease activity in UC. Both of these showed a lower Lichtiger score in the cannabinoid-treated group at the end of the study even though the pooled analysis was not statistically significant. However, these small studies were subject to imbalance of important confounders. Indeed, 1 study had a significantly lower Lichtiger score in the active treatment group prior to randomization. Thus, the apparent benefits of cannabinoid treatment at the conclusion of that study were more likely due to an imbalance of disease severity between the groups at the time of randomization. Our analysis identified high heterogeneity among the studies and suggested the potential for publication bias. If trials showing a negative effect of cannabinoids on IBD outcomes were less likely to be published, it would bias the results of this meta-analysis toward a favorable effect. Also, the studies are subject to barriers related to blinding, as participants may be able to differentiate placebo from active treatments due to inherent difficulties developing placebo that can mimic the sensory and psychotropic effects of cannabinoid treatments.

A significant limitation to this study and our understanding of cannabinoid effects in IBD is the heterogeneity in dose, route of delivery, and composition of the study drugs used in these trials. There are over 100 types of cannabinoids in marijuana that generate both its psychoactive and medicinal properties. Each strain of the marijuana plant contains varying concentrations of THC, CBD, and other cannabinoids resulting in the unique effects of each strain. Among the studies used in this analysis, the particular strain of marijuana used for the study drug was rarely specified. Also, comparing the smoked leaf or tincture forms of marijuana containing the full spectrum of cannabinoids used in some studies to a purified

CBD preparation used in others adds a potential source of heterogeneity. Inherent differences in pharmacodynamics and time to steady state between the smoked and ingested preparations of marijuana as well as the dose of cannabinoids administered could contribute to the variable degree of benefit observed for symptomatic improvement and quality of life outcomes. The optimal spectrum of cannabinoids, strain of marijuana, and route of delivery for medicinal purposes remains unknown.

To build upon our current knowledge, additional and larger RCTs are needed for a more robust assessment of cannabinoids on inflammatory bowel diseases. As further cannabinoids are developed, it will be helpful to develop a standardized treatment with more effective controls that can be employed across various studies. In addition, future studies should include a more detailed assessment of the components of quality of life and symptoms to help clarify the mechanisms by which cannabinoids appear to improve quality of life and IBD symptoms.

In conclusion, this meta-analysis supports the role of cannabinoid therapies in improving patient's quality of life in both UC and CD as well as symptoms of CD but not inflammation. As such, cannabinoids could be considered as an adjunct therapy for patients with medically refractory disease or those with persistent symptoms despite resolution of objective measures of inflammation. The available data do not support use of cannabinoids in place of approved medications that reduce inflammation and improve symptoms.

Supplementary Data

Supplementary data is available at *Inflammatory Bowel Diseases* online.

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Conflict of Interest

J.D.L. consulted or served on an advisory board for AbbVie, Amgen, Arena Pharmaceuticals, Bridge Biotherapeutics, Bristol-Myers Squibb, Celgene, Eli Lilly and Company, Entasis Therapeutics, Galapagos, Gilead, Janssen Pharmaceuticals, Samsung Bioepis, Merck, Nestle Health Science, UCB, Pfizer, Protagonist Therapeutics, Sanofi, and Scipher Medicine. He has had research funding from Nestle Health Science, Takeda, Janssen Pharmaceuticals, and AbbVie. He has had educational grants from Takeda and Janssen.

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