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Can Leafy Vegetable Source of Omega-3 fatty Acids Ameliorate Acute Intestinal Inflammation Induced in Mice? A Case study of Purslane (*Portulaca oleracea*)

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Authors' contributions

This work was carried out in collaboration between all authors. Authors HOO and BS designed the study, performed the statistical analysis, and wrote the protocol. Authors HOO and WAP wrote the first draft of the manuscript and made the final corrections. Author BS supervised the experimental studies conducted by author HOO who also managed the literature searches. All authors read and approved the final manuscript.

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ABSTRACT

Aim: To investigate the ameliorative effect of purslane (*Portulaca oleracea)*, an omega-3 rich green leafy vegetable in the treatment therapy of experimental ulcerative colitis in mice.
Study Design: *In vivo* evaluation of the ameliorative effect of purslane on ulcerative colitis.
Place and Duration: Purslane leaves were obtained from the Agricultural Research Institute in Newe Yaár, Israel. Female C57BL/6J mice were purchased from Harlan Animal Research Laboratories Ltd. (Jerusalem, Israel). The actual studies were undertaken at the Department of Nutritional Science, Hebrew University of Jerusalem, Rehovot, Israel, between October 2008 and December 2009.
Methodology: Twenty-five C57BL/6J mice were randomly assigned to dextran sulphate sodium

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(DSS) control group, high-dose (24%) purslane group, medium-dose (8%) purslane group or low-dose (0.8%) purslane group receiving 3.5% dextran sulphate sodium (DSS) in their drinking water for eight days after ten days of receiving the feeding regime as described above. A normal control group was also employed to receive drinking water plus standard diet for the whole duration of the experiment. The animals were sacrificed and subjected to histopathological analysis, as well as myeloperoxide activity, and disease activity index determination.

Results: Animals fed the different concentrations of purslane did not exhibit a statistically significant (P>0.05) trend towards histopathological and clinical improvement after the eighth day of DSS administration. There was no significant difference (P>0.05) observed in fatty acid profile between the intervention and the DSS control groups 8 days post-DSS. However, from the study the higher the consumption of purslane (>6g/d), the higher the concentration of the fatty acid profile in blood plasma. Also, no significant decrease in neutrophil infiltration was observed, as depicted by myelohyperoxidase activity.

Conclusion: Our study concluded that oral administration of purslane at concentrations up to 24% could not suppress ulcerative colitis in mice.

Keywords: Dextran sodium sulphate; ulcerative colitis; omega-3 fatty acids; purslane.

1. INTRODUCTION

Nutritional supplementation with omega-3 fatty acids has been shown to have some level of corrective effect on certain non-communicable diseases including inflammatory bowel disease (IBD) [1]. Both clinical and animal studies show that Omega-3 fatty acid has anti inflammatory properties through its ability to reduce inflammatory eicosanoids, cytokines, adhesion molecule expression (such as intercellular adhesion molecule-1 and vascular cell adhesion molecule -1), nitrogen metabolites and reactive oxygen species from arachidonic acid [2]. Ulcerative colitis (UC), a form of IBD, is a chronic and relapsing condition of which its etiology is poorly understood. It is characterized by colonic and rectal tissue edema, increased colonic epithelial permeability, haemorrhage and extensive infiltration of leukocytes in the mucosa layer of the colon [3]. Several theories in literature have emerged pertaining to the development and pathogenesis of this disease which is currently presumed to result from a complex interplay among genetic, environmental, microbial and immune factors. Immunomodulating agents and corticosteroids have been used to treat UC, although not always with positive outcomes and many side effects [4].

Studies indicate that omega-3 polyunsaturated fatty acids (PUFA) could be efficient in UC management [5,6]. These studies have used fish oil, the main source of these PUFA and also oil extracts of alpha- linolenic acid (ALA) found in vegetable seeds such as flaxseed, soybean oil, canola oil and rapeseed. However, studies on the effect of whole dietary intake of green- leafy vegetable source of Omega-3 fatty acids on UC has not been exploited. As has been reported in many studies, the consumption of purslane in the Mediterranean regions has helped reduce the incidence of cancer and coronary heart diseases [7]. It has also been reported that ALA found in green leafy vegetables desaturates and elongates in human body to eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) and by itself may have beneficial effects in health and in the control of chronic diseases [8].

Animal models of IBD which involve chemically induced colitis show that diet rich in ALA decreases colonic damage and inflammation as compared to omega 6 rich diets [6]. Therefore, there is the need to investigate the role ALA found in leafy vegetables can play in the management of UC. In this study, we aimed to investigate the ameliorative effect of oral consumption of purslane (*Portulaca oleracea)*, an undoubtedly rich source of ALA and other antioxidants including vitamin E [9,10] supplemented diet, on mice model of dextran sulphate sodium-induced (DSS-induced) colitis. The mouse model of DSS-induced colitis is an experimental model that resembles acute human IBD. It shows a preclinical symptom involving the molecular events required for tumour formation in the presence of inflammation and assesses the ability of select agents to inhibit this process [11]. In the present study, it was evaluated by the disease activity index (DAI). This was done by scoring body weight, gross bleeding and stool consistency. Histological injury scores were also taken after the induction of colitis by the administration of DSS orally in drinking water for eight days [3].

2. MATERIALS AND METHODS

2.1 Preparation of Purslane

Purslane leaves were obtained from the Agricultural Research Institute in Newe Yaár, Israel. The leaves were cleaned and oven dried for 24 h at a temperature of 40°C after which they were ground into a fine powder using a kitchen blender and then stored in a covered clean dried container, at-20°C until use. Purslane was mixed with standard diet for the animal experiments. The mixture was blended thoroughly to ensure homogeneity.

2.2 Experimental Design

Twenty five (25) female C57BL/6 J mice weighing approximately 18±1.9 g, a strain known to be susceptible to oral administration of dextran sulphate sodium (DSS), were supplied by Harlan Animal Research Laboratories Ltd. (Jerusalem, Israel) at the age of 7-8 weeks. They were maintained on a standard laboratory diet (control diet) obtained from Harlan laboratories Ltd. (Table 1), and had access to drinking water in bottles *ad libitum* for a period of 1 week before the experimental feeding. They were kept in plastic cages (5 mice /cage) at the animal facility and were exposed to controlled conditions of 50% humidity, light (12/12 hr light /dark cycles) at a temperature of 23±2°C. Animal care and experimental procedures were in accordance with the guidelines of the accredited animal ethics committee of the Hebrew University of Jerusalem (Certificate # AG–10835–3; 7th January 2009).

2.3 Intervention and Induction of Ulcerative Colitis model using Dextran Sulphate Sodium

Twenty five (25) of the female C57BL/6J mice were randomly assigned into five groups, comprising three intervention groups and two control groups (Fig. 1). The three intervention groups received a composite diet of standard diet mixed with dried purslane leaves at replacement levels of 24% (i.e. 6 g in 25 g of total feed), labeled high-dose, 8% (i.e. 2 g in 25 g of total feed), labeled medium-dose, and 0.8% (i.e. 0.2 g in 25 g of total feed), labeled low-dose, on dry weight basis (Fig. 1). This brings the specific purslane dose in the 25 g mixed diet provided for the mice in each cage (i.e. 5 g of mixed diet per animal) to 66.7 g/kg mice body weight, 22.2 g/kg mice body weight and 2.2 g/kg mice body weight for high dose, medium dose, and low dose, respectively. Preliminary screening however showed that 100 g of purslane fresh weight (FW) contained about 300.5±95.3 mg ALA, 2.5 mg/g oxalic acid, 60.4-69.3 ug/g ascorbic acid, 400 ug/g α-tocopherol, and 150 ug/g γ-tocopherol [12]. The purslane leaves used in the preliminary studies are of the same microspecies as used in the present study. It must however be stressed that variations in phytochemical constituents may be brought about by changes in location and field conditions. This fact has been aptly demonstrated by recent work done by Ai, et al. [13] which showed significant variations in eight constituents of *P. oleracea* L. from different locations.

Table 1. Basic nutrient composition of Standard diet (control diet)

Nutrient	Quantity
Crude protein	18.8 g/100 g
Crude fibre	3.8 g/100 g
carbohydrates	50 g/100 g
Vitamin A	15.4 IU/g
Vitamin E(α-tocopherol)	100 mg/kg
Vitamin B1	16.5 mg/kg
Vitamin B6	18.5 mg/kg
β carotene	2.5 mg/kg
sodium	0.23g/100 g
Iron	225 mg/kg
Palmitic acid	7.6 g/kg
Stearic acid	15 g/kg
Oleic acid	12.6 g/kg
Linoleic acid	31.3 g/kg
α-linolenic acid	2.8 g/kg
Total saturated fatty acids	9.6 g/kg
Total monounsaturated fatty acids	12.8 g/kg
Total polyunsaturated fatty acids	34.1 g/kg

Source: Adapted from Harlan Laboratories Ltd (2009)

The animals consumed these different concentrations for 10 days *ad libitum* before colitis was induced and until the end of the experiment. The two control groups (DSS and normal control groups) received standard diet *ad libitum* for the whole period of the experiment. On the 11th day after the start of the experiment the three intervention groups and the DSS-control group were made colitic by replacing their drinking water with distilled water containing 3.5% (w/v) DSS (molecular weight: 36 kDa-50 kDa from MP Biomedicals, Solon, Ohio, USA) for seven days (Fig. 1). The negative-control group was included for reference. Mice were fasted overnight, anaesthetized with isofluorane and

then sacrificed on day 20. Colonic tissues were then removed and cleaned. Colonic length (cecum to rectum) was measured and divided into several sections for organ culture and histology.

2.4 Clinical and laboratory Assessment of Colonic Inflammation

Animals were observed daily for food and water intake, weight changes and relevant symptoms (such as loose stools, diarrhea, hematochezia). Body weight and stool consistency were determined daily for all animals. Disease activity index **(**DAI) was determined by scoring changes in weight, hemoccult results or gross bleeding and stool consistency in all animals. The scoring criteria and methodology used were essentially as shown in Table 2. Furthermore after 8 days of DSS administration, plasma fatty acids were determined by the method described by Ji, et al., [14] with minor modifications. DAI score is the combined value of weight loss, stool consistency and bleeding divided by 3 [15].

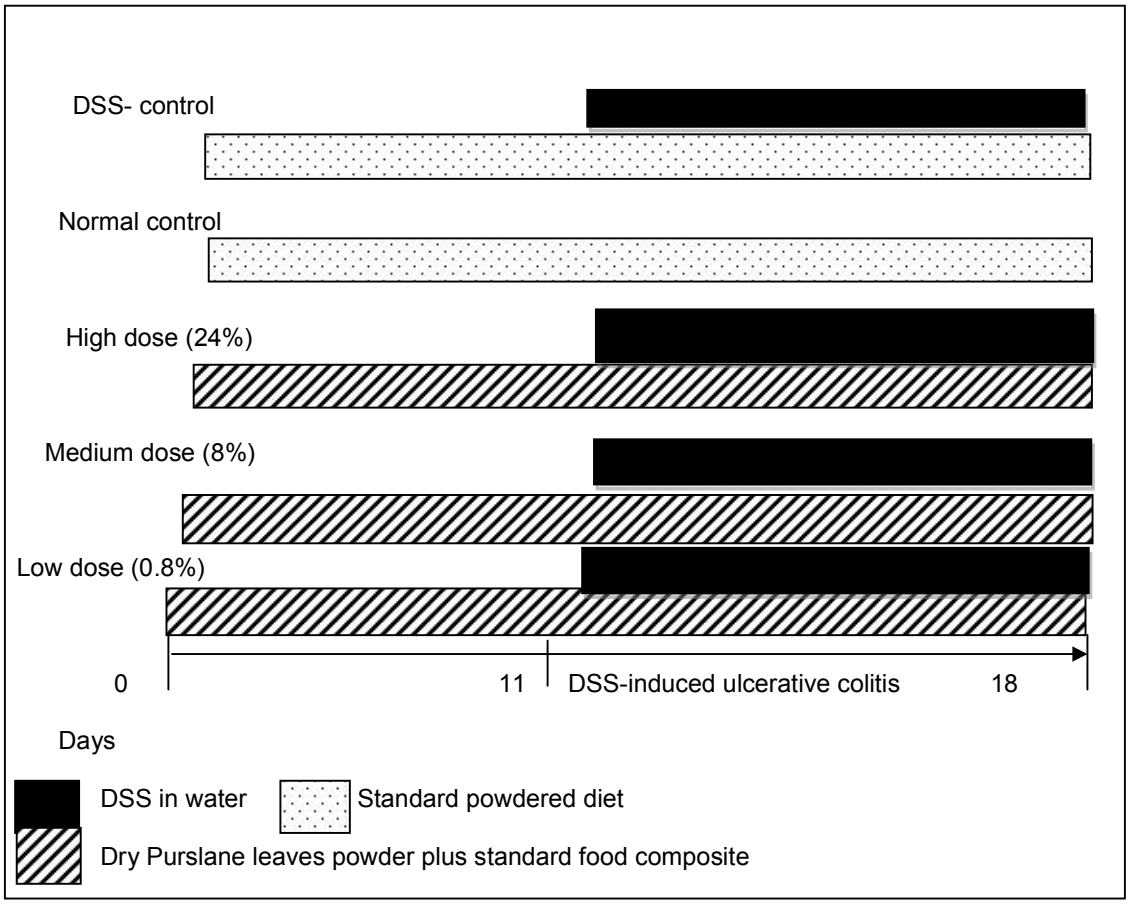

Fig. 1. Experiment *in vivo* (diagram)

Table 2. Scoring of disease activity index (DAI)

Score	Weight loss (%)	Stool consistency	Rectal bleeding
0	None	Normal	No blood
1	1-5	Loose stools	Hemoccult +/-
2	5-10	Loose stools	Hemoccult +
3	10-20	Loose stools	Hemoccult ++
4	> 20	Diarrhea	Gross bleeding

2.5 Histopathological Analysis of Colonic Inflammation Induced by DSS

Ten days after the induction of colitis by DSS (day 20 of the experiment), mice were weighed and sacrificed. Their entire colon was resected from the colo-cecal junction to the anus and rinsed with sterile saline (0.9%) to remove faecal matter, weighed and their lengths (cm) measured. This was divided for histopathological and myeloperoxidase activity determination. The colon segment for histopathological determination was kept in 4% buffered formaldehyde and submitted (transverse sections) for histological processing. Mucosal inflammation in the colon was evaluated in the haematoxylin and eosin (H&E) stained sections as described by Wallace et al., [16]

2.6 Myeloperoxidase (MPO) Determination of Colonic Inflammation Induced by DSS

MPO activity was measured according to the technique described by Bradley et al. [17]. The results were expressed as MPO units per gram of wet tissue; one unit of MPO activity is defined as that degrading one µmol of hydrogen peroxide/min at 25°C.

2.7 Blood Fatty Acid Analysis of Mice

2.7.1 Blood collection

About 1ml of blood was collected into ethylenediaminetetraacetic acid (EDTA) vacuum tubes (BD vacutainer systems, Pre analytical solutions, Belliver Industries Estate, UK) on ice after an overnight fast before centrifugation (3000 x g for 10 mins) at 4°C to separate plasma and Red blood cells (RBC). Plasma was then aliquoted into micro centrifuge tubes and the Buffy layer of the white blood cells (WBC) removed by using a pasture pipette. The RBCs were then aliquoted into separate micro centrifuge tubes which were then washed thoroughly in normal saline (0.9% NaCl). Samples were frozen immediately and stored under nitrogen atmosphere at-80°C until analysis.

2.7.2 Plasma fatty acid analysis

Fatty acids from plasma were isolated and methylated as described by Ji et al. [14], with minor modification. Briefly, 125 µl of plasma was first mixed with 250 µl of ultrapure water and then with 1 ml methanol: dichloromethane (3:1 v/v). After addition of internal standard (50 nmol of heptadecanioc acid), 200 µl acetyl chloride was added, drop by drop, while vortexing in a fume hood using eye protection, laboratory coats and gloves. The samples were then incubated at 75°C for one hour. After removal and cooling to room temperature, the reaction solution was neutralized with 4 ml of 7% potassium carbonate (K_2CO_3) and the lipids were extracted into 2 ml of hexane, mixed vigorously and centrifuged for 10 m at 2,500 x g at room temperature. The hexane fraction was then washed with acetonitrile. The fatty acid methyl ester (FAME) mixture was then re-suspended in 350 µl of hexane and analyzed by gas-liquid chromatography and mass spectrometry (GC-MS).

2.8 Statistical Analysis

The effect of the supplementation was compared to the DSS-control group for all outcomes. All results are expressed as the mean±SEM. Differences between means were tested for statistical significance by using a one-way analysis of variance (ANOVA) and post hoc least significance tests (LSD). All statistical analysis was carried out with JMP 8.0 software package (SAS, USA) with statistical significance set at $P<0.05$ [18].

3. RESULTS

The mice had an average food intake of 16.4±0.3 g/day per cage (five mice per cage) during the ten days before colitis induction, without showing any statistical differences among groups. During the course of the experiment, all purslane-fed colitic mice did not show any reduction and more so any significant ($P>0.05$) reduction in inflammatory response as shown by DAI values. As a matter of fact, DAI from day 16 increased as the concentration of purslane in the feed reduced.

3.1 Macroscopic Findings of Colonic Inflammation Induced by DSS

Macroscopic examination of the colonic specimens revealed that following 8 days of DSS administration, the colon length in all purslane-fed mice groups was not significantly different ($P>0.05$) from the DSS-control group. Treatment with the different concentrations of purslane however did not significantly attenuate DSS-induced shortening of the colonic length (Fig. 2).

After 8 days of DSS induction, the mean body weights from day1 to day 18 for normal control,

DSS-control, 24% group, 8% group and the 0.8% group decreased significantly (P<0.05) from 18.23 g to 17.06 g, 18.32 g to 15.54 g, 18.12 g to 16.24 g, 17.92 g to 15.96 g and 18.0 g to 16.54 g respectively (Fig. 3).

3.2 Histological Findings of Colonic Inflammation Induced by DSS

Eight days after the administration of DSS in all three intervention groups and the DSS-control group, there were significant histopathological changes in the colon in terms of marked destruction of surface epithelium, cell disruption, and severe inflammatory lesions extensively throughout the mucosa. On the contrary, the normal-control mouse colon sections showed intact epithelium, well defined crypt length with no edema, neutrophil infiltration in mucosa and sub mucosa, and no ulcers and erosions. Treatment with the different concentrations of purslane did not show any significant attenuation of DSS-induced histopathological changes in the colon (Fig. 4).

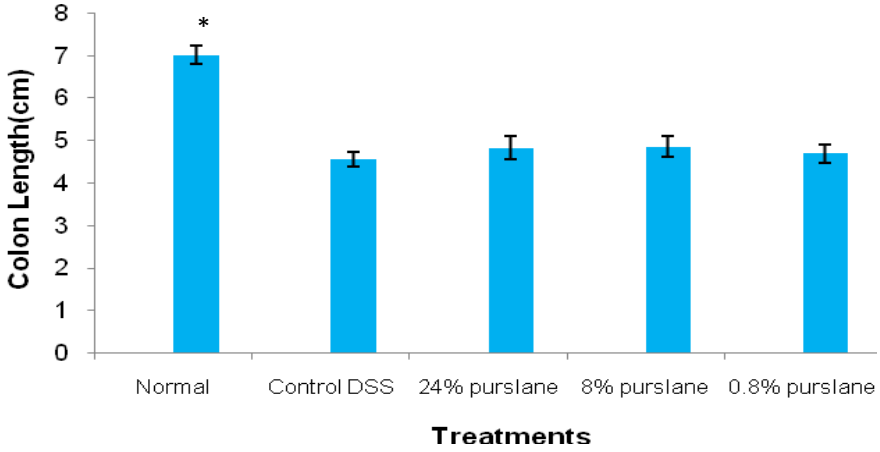

Fig. 2. Mean colon length changes during DSS treatment. Colon length removed on the sacrifice day is depicted as mean±SEM in each group. (*P<0.05vs DSS-control)

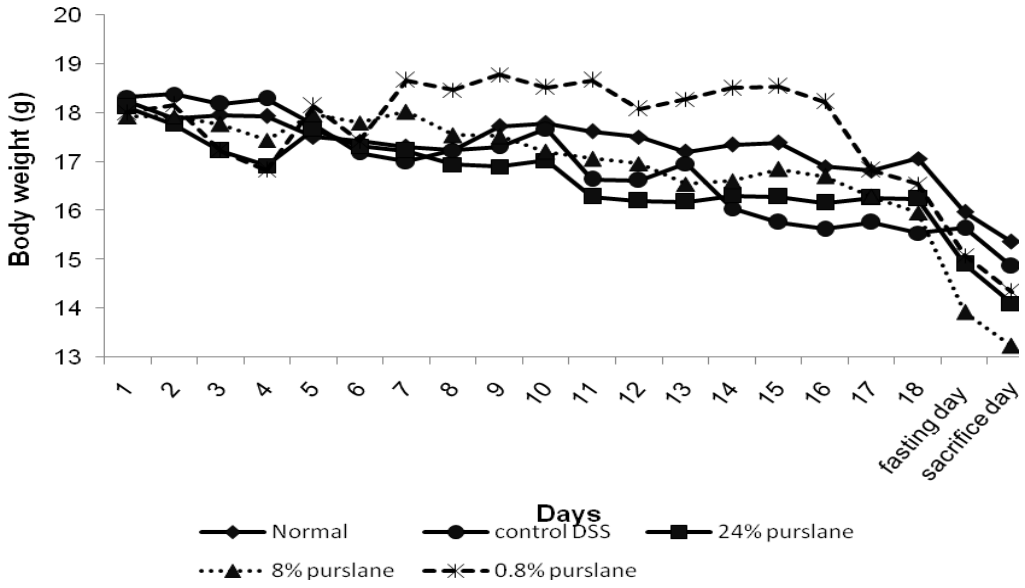

Fig 3. Mean body weight values in all five groups during the 18 day period. Weight changes are shown as an average of 5 observations in each group (n=5)

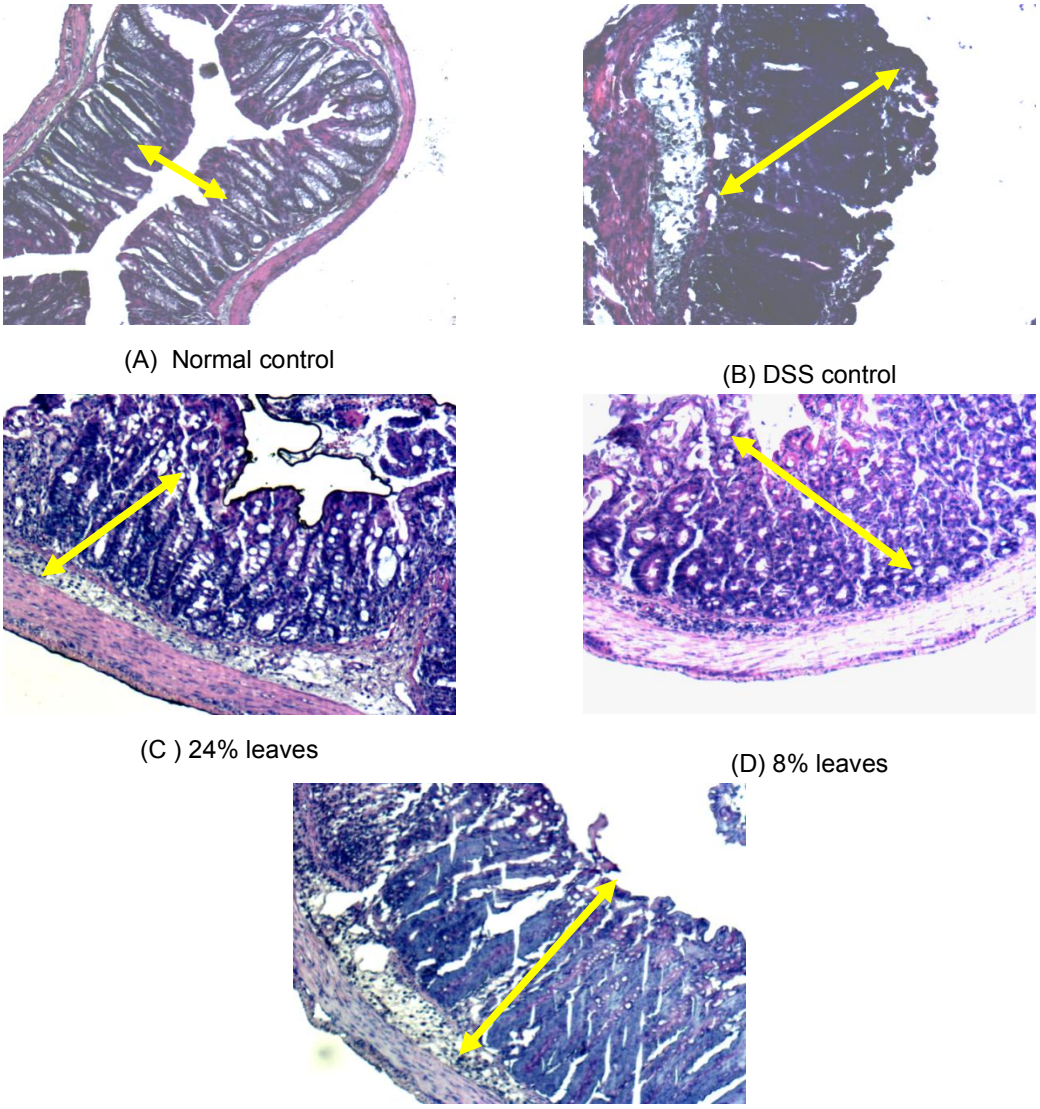

Fig. 4. Hematoxylin and eosin staining of colonic mucosal tissue section from mice receiving drinking water (A, negative-control), dextran sulphate sodium (B, control-DSS), purslane (C, 24% leaves), (D, 8% leaves), (E, 0.8% leaves). Arrowed portions indicate intact (A) and damaged (B, C, D, & E) mucosa and sub mucosa

3.3 Disease Severity (DAI) of Colonic Inflammation Induced by DSS

About 50% of the mice in the 24% purslane, 8% purslane and 0.8% purslane groups developed loose stools after eleven days of DSS administration, which turned to diarrhea in the vast majority of the mice (90–100%) at 14–15 day. After sixteen to seventeen days, gross rectal bleeding was evident in 70% of the 8% (medium dose) and 0.8% (low dose) purslane-fed mice, which persisted until the end of the experiment. The inflammatory process resulted in a progressive loss of weight in all mice administered DSS, which was associated with a reduction in food intake compared with the normal control mice. The weight loss was most evident 11 days after the start of DSS administration. As a consequence of the inflammatory process, DAI was increased in all colitic groups especially in the 0.8% and 8% purslane groups from the fourteenth to the eighteenth day (Fig.5). The different

Concentrations of purslane did not decrease DAI activity. There is an increase in disease activity compared with the control DSS during the DAI evaluation days. The observed lower DAI for higher doses are not significant.

3.4 MPO Activity of Colonic Inflammation Induced by DSS

Colonic MPO activity as an indicator of the extent of neutrophil infiltration into mucosa was measured. Administration of DSS significantly increased MPO activity ($P<0.05$) in colonic tissue compared with the normal control group (Fig. 6). Treatment with the different concentrations of purslane (24%, 8%, and 0.8%) leave powder again did not show a significant ($P>0.05$) attenuation of the DSS-induced rise in colonic MPO activity in colonic tissue. As shown in Fig. 6, the group which received 8% leave concentration did have a slight reduction but was not statistically significant ($P>0.05$) from the DSS- control group.

3.5 Plasma Fatty Acid Concentration

Administration of purslane leaves was initiated 10 days prior to DSS induction and until the end of the experiment. The effect of this was assessed for plasma fatty acid after 8 days post-DSS (Table 3). Compared with the normal control, DSS-control did not significantly change but reduced the concentrations of linoleic acid, α-linolenic, total PUFA, total omega 6, total omega-3, total monounsaturated and the ratio on n-6:n-9 ($P>0.05$) except in arachidonic acid (20:4n6) ($P<0.05$). The different purslane concentrations did not also have any significant effect on the concentrations of the various fatty acids in plasma before and after DSS induction ($P>0.05$). However, the results suggest that the higher the concentration of purslane in the feed, the higher the fatty acid concentration in plasma even though not significant ($P>0.05$). This was especially seen for linoleic acid and α-linolenic acid. The reduction in the linoleic acid content can also be due to the substitution by oleic acid.

4. DISCUSSION

Purslane is a richer source of (n-3) α-linolenic acid than most commercialized vegetables [9,19]. A number of studies have demonstrated that α–linolenic acid (ALA) is able to convert to the longer chain PUFA (EPA and DHA) which are anti-inflammatory agents, through the diet [8,20]. However, this conversion efficiency is small and the effect of ALA itself in inhibiting inflammation is also minimal [21]. Many studies around the use of omega-3 fatty acids in ameliorating inflammation induced by DSS have used either fish or vegetable oils rich in EPA, DHA or ALA [21,22,23]. Recent studies on purslane [24,25,26] also showed high phytochemical and phytosterol constituents which have demonstrated anti-inflammatory activities. These *in-vivo* and *in-vitro* studies however, have used either aqueous, ethanol or oil extracts at high concentrations that produced positive anti-inflammatory effects. No study has been done yet using whole plant tissues and for that matter dry purslane leaves in any such *in-vivo* experiment. In the present study we sought to find out in totality, the potential beneficial effect of consuming whole purslane leaves on ulcerative colitis taking into consideration also its high antioxidant potentials.

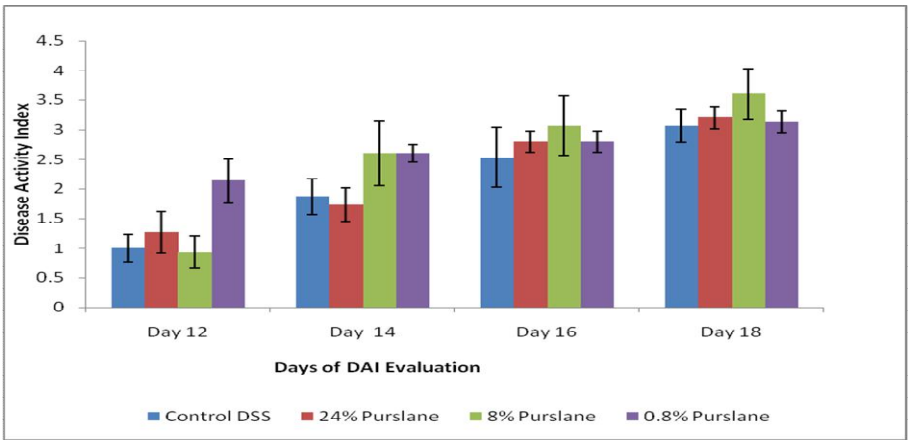

Fig. 5. Severity of disease measured in terms of disease activity index (DAI) in DSS-control, 24% pursalne, 8% purslane and 0.85 purslane based on the criteria shown in table

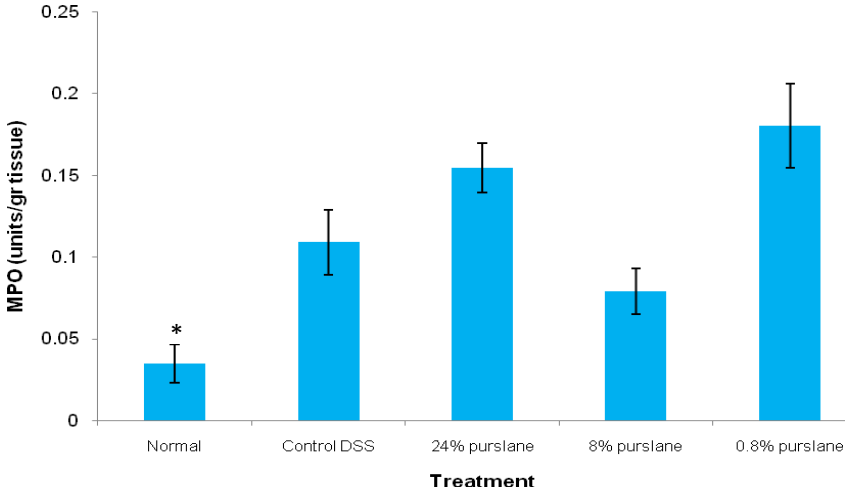

Fig. 6. Effects of different concentrations of *Portulaca* leaves on colonic myeloperoxidase (MPO) levels in dextran sulphate sodium (DSS)-induced colitis in mice. Results are expressed as mean ± SEM of five observations.

Table 3. Fatty acid profile in blood plasma of mice fed three different concentrations of purslane[1]

Fatty acid	Negative-control	DSS-Control	Purslane Supplemented diets		
			24%	8%	0.8%
	µg/ml blood plasma				
C6:0	43.75±21.7	16.46±3.9	5.40±2.5	6.01±0.8	2.62±1.6
C14:0	6.93±6.9	20.78±14.6	33.47±22.2	9.98±8.0	11.54±5.8
C16:0	96.75±11.4	123.14±14.0	123.15±79.7	160.50±46.1	124.20±50.8
C18:0	95.38±11.7	103.92±13.4	132.11±21.7	121.62±30.9	122.28±19.7
C18:1n9	97.97±38.0a	66.22±10.9a	116.10±47.4a	90.67±33.6a	95.09±25.0a
C18:3n6	0	0	2.20±1.3	3.11±2.0	3.14±3.1
C18:2n6	27.39±21.9a	13.28±9.3a	23.81±15.2a	5.13±5.1a	5.49±4.2a
C18:3n3	28.48±28.5	0	7.34±3.9	4.91±2.9	3.79±2.5
C20:4n6	93.97±14.7a	60.48±10.8b	80.16±7.2b	60.39±8.7b	63.81±7.5b
C22:6n3	25.98±6.9a	39.91±7.6a	56.95±14.3a	49.84±10.4a	49.56±4.6a
Total saturated	242.81±35.5a	264.3±23.2a	294.13±116.3a	298.11±72.8a	260.64±61.8a
Total PUFA[2]	175.83±40.7a	113.67±15.3a	170.48±34.5a	123.38±21.1a	125.79±12.4a
Total (n-9)	97.97±38.0a	66.22±10.9a	116.09±47.4a	90.67±33.6a	95.09±25.0a
Total (n-3)	54.47±31.9a	39.91±7.6a	64.31±18.2a	54.75±10.4a	53.35±6.2a
Total (n-6)	121.36±34.5a	73.75±9.5a	106.17±17.1a	68.63±11.4a	72.44±7.1a
(n-6)/(n-9) ratio	1.24±0.91a	1.11±0.87a	0.91±0.36a	0.76±0.34a	0.76±0.28a
(n-6)/(n-3) ratio	2.23±1.08a	1.85±1.25a	1.65±0.94a	1.25±1.10a	1.36±1.15a

[1]Values are means ±SEM, n=5. Means in a row with different letters are significant, p<0.05. Significance was determined by comparing the DSS-control with the different diets including the negative-control diet.
[2]PUFA: Polyunsaturated fatty acid.

In this study, ALA was converted to DHA only and the effect change of the three treatments of purslane was not seen in terms of the various PUFA profiles (*P*>0.05). This is somewhat in contradiction to earlier reports by Gerster [5] and Williams et al., [20] suggesting that conversion of ALA to EPA occurs, but conversion to DHA is severely restricted. This is of great interest in this study because DHA according to the same authors has an autonomous function, e.g. in neurological and spermatozoa development and so consumption of purslane may be beneficial to these functions. Above all, these findings go to confirm the importance of EPA and DHA in the diet.

As shown in (Table 3), the ratio of n-6: n-3 in the present study was found to be lower than the recommended range of about 4:1 [21,27]. Although the concentrations of linoleic acid (LA) are high in purslane, the α-linolenic acid content is also relatively high (Table 3). The low ratios obtained confirm the fact that purslane is a good source of omega-3 fatty acids to contribute effectively in amelioration of inflammation. But this was not the case in the present study; which suggests that other biological components in the purslane may have contributed negatively. This is because it is not the individual amount of n-6 PUFA and n-3 PUFA that is involved in the beneficial effect but their influence on the n-6: n-3 ratio since both PUFA types compete with same enzymes to produce different inflammatory mediators [28].

The aim of this study was to find out the suppressive effect of three different concentration levels of purslane mixed with standard diet on ulcerative colitis. We assumed that though the standard diet also contained some levels of PUFA, minerals and vitamins, this in addition to the purslane did not significantly influence the results positively. However the effect of other biologically active compounds in the matrix of the feed could have affected our results negatively. In this study and for the first time it was shown that consumption of purslane in which about 60% of its total fatty acid in the leaves is ALA converted to DHA and not EPA. Many studies showing anti-inflammatory effect of n-3 fatty acids, especially ALA are very inconclusive [29], and this study has re-emphasized this point that giving purslane at the specified doses in this experiment was not successful in suppressing UC.

Some possible challenges this study could have encountered or overlooked would be the low concentrations of purslane used as well as the high inherent anti nutritive factor (oxalic acid) in purslane [12,30]. In fact this anti nutritive factor in purslane could be one of the reasons for its low adaptation as a domestic vegetable in most regions of the world.

Results from macroscopic, histological, disease activity and MPO findings all prove the inability of the three different concentrations of purslane to ameliorate DSS induced ulcerative colitis. However it can be suggested from the same results that the higher the concentration of purslane, the better the fatty acid concentrations in the blood.

5. CONCLUSION

In this study, we administered supplemented diets containing 6 g, 2 g and 0.2 g of purslane to three groups of mice before and during 7 days of DSS induction. At the end of the experiment, we concluded that the different concentrations of purslane administered were not able to ameliorate ulcerative colitis. However, the higher the consumption of purslane (above the concentrations used in this study), the higher would be the concentration of the essential fatty acid profile in blood plasma. The effect of consuming purslane leaves alone as the sole source of n-3 in suppressing UC is negligible if not impossible. It is suggested that consumption of purslane should be done in combination with ALA-rich vegetable oils/ oily fish or take in fish oil supplements to increase essential fatty acid uptake. Studies to re-engineer purslane can also be looked into to have a lower oxalic acid content to improve the n-6: n-3 ratio and to boost ALA uptake. Finally, this study did not touch on the effect of consumption of purslane leaves on other inflammatory biomarkers and so investigations into them could be done in the future to ascertain its true effect on UC. It must also be stressed that variations in phytochemical and fatty acids constituents may be brought about by changes in location and field conditions.

CONSENT

Not applicable.

ETHICAL APPROVAL

All authors hereby declare that "Principles of laboratory animal care" (NIH publication No. 85-23, revised 1985) were followed, as well as specific national laws where applicable. All experiments have been examined and approved by the appropriate ethics committee (Certificate # AG – 10835 – 3; 7th January 2009).

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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