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# Comparative Study Between Virgin Coconut Oil and Omeprazole Drug on Ulcerative Colitis in Experimental Rats

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## ABSTRACT

This study aims to identify the curative effect of virgin coconut oil on ulcerative colitis(UC),the present study was carried (24) adult albino male rats Sprague –Dawley strain weighting 180 ±10g were classified into four groups,(6 rats each) the first group rats were fed on basal diet only control negative(-ve) group and three rat groups were injured of ulcerative colitis by intra colonic administration of acetic acid 4% given rectally by single dose ( 1 ml/ rat ) and reclassified into control positive (+ve) group (fed on basal diet only), group treated with (20 mg/kg b.w) of omeprazole drug and group treated with (2 ml per/ rat) daily of virgin coconut oil . The results revealed that, the control (+ve) group showed a significant decrease in final weight(FW) ,weight gain(WG) feed efficiency ratio (FER), organs weight of heart, kidney, and liver , catalase (CAT), superoxide dismutase (SOD), glutathione reduced (GSH), Glutathione reductase (GSR),Glutathione peroxidase (GPx), glutathione-S-transferees (GST) and significant increase in malondialdehyde (MDA) , hydrogen peroxide ($H_2O_2$), C-reactive protein **(**CRP), interleukin-6 (IL-K) and prostaglandin (PG2) compared with control (- ve) group. The virgin coconut oil group showed a significant increase in (FW),(WG),(FI),(FER),organ stomach,(CAT),(SOD),(GSH),(GSR (GPx), (GST) but significant decrease in (MDA),($H_2O_2$),(CRP),(ILK), (PG2) compared with control (+ve) group . the study clearly showed that consumption virgin coconut oil has a role in treating rats with acetic acid-induced ulcerative colitis. It can be recommended that virgin coconut oil should be consumed daily to reduce the incidence of ulcerative colitis.

***Keywords**:* virgin coconut oil, acetic acid, ulcerative colitis, experimental rat.

## INTRODUCTION

Virgin Coconut Oil (VCO) is considered a medicinal functional food in the many popular countries that cultivate it. It is an unrefined oil extracted from the fresh and ripe coconut fruit (*Cocos nucifera L)* and is extracted manually or mechanically using special oil extraction machines, with Or without the use of heat and without chemical bleaching and deodorization (Marina *et al.* 2009).Coconut contains a high percentage of dietary fiber, which studies indicate absolute importance for the colon. Reports confirm that coconut oil is not considered one of the causes of obesity, as it does not accumulate in fatty tissues and is therefore It is not considered a reason for weight gain. It tends to increase HDL (Assuncao *et al*., 2009). Recent studies have shown the importance of coconut oil that it has phenol-dependent antioxidant properties for VCO in animal models (Seneviratne *et al*., 2009). Coconut oil contains a high percentage of saturated fats, and thus, this may lead to an increase in the level of cholesterol in the blood, but studies have indicated its importance and its confrontation with cardiac risks. thus, a systematic study was conducted to clarify the effect of coconut oil intake on cardiovascular attacks and increased blood lipids by comparing it with other cooking oils using data from clinical trials (Neelakantan *et al*., 2020). As a result of the increase in coconut oil and the popularity of it in the past decades, its importance in improving the metabolism process. Anti-oxidant, anti-inflammatory and anti-diabetic. (Malaeb and Spoke *et al.*, 2020) Coconut oil has a significant lowering effect by stimulation in cholesterol. The pathophysiology of ulcerative colitis entails, along with a limited set of therapeutic qualities that distinguish it and make it a natural product alternative to treatments. It is suggested that coconut oil contains medium-chain fatty acids because it has an effect on the symptoms of colic and ulcerative colitis of patients by modifying inflammatory responses in diseased mice (Chandra 2013). Coconut oil (CO) has garnered interest in Western medicine due to its promising therapeutic effect in treating Alzheimer's disease as well as combating neurodegeneration caused by induced oxidative chains. (Odubanjo *et al.*, 2020).

Ulcerative colitis (UC) is a chronic disease that affects the large intestine or colon and causes ulceration and inflammation of the mucous membrane lining the large intestine and rectum (Xavier and Podolsky, 2007). It can occur in any area of the large intestine, although complementary colitis may affect certain parts of some patients with UC, the immune system sends false signals and colon gases appear (Itzkowitz and Yio 2004). Several types of medications are available as amino salicylates. It is a common disease that affects millions of individuals all over the world and can be considered one of the most common diseases in the world. (Awaad *et al.*, 2013).

Omeprazole is widely used to treat disorders of the digestive system However, in the long run use of OME increases the risk of stomach cancer. We aimed to characterize the pharmacological effects of OME and

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correlate its adverse effects and toxicological risks with mechanisms of genetic instability and cancer based on the database reports (Marcia *et al*., 2020).

The laboratory results revealed that omeprazole has antioxidant and anti-inflammatory properties. There was also a significant amelioration in body weight percentage, colonic, ulcer degree, and biochemical analyzes in the omeprazole treated group comparison to the colitis group, which had prevenient anti-inflammatory and antioxidant efficacy Ittiyavirah and Shenika (2014).

This work is designed to use virgin coconut oil in diets to treat the side effects of acetic acid-induced ulcerative colitis in rats.

## MATERIALS AND METHODS

**A – Materials**

1-Acetic acid and distilled water were purchased from El-Gomhouria company for trading chemicals and medical appliance, Dakahlia branches, El Mansoura , Egypt**.**

2-Omeprazol was obtained from Egyphar Company, Obour City. Each tablets contains 20 mg of Omeprazol .The animal dose (20 mg/kg) adjustable to anterior of paper as listed by (Raeesi *et al* .,2019).

3-Coconut fruit were purchased from the local market in Mansoura city, Egypt. Virgin Coconut oil is given (2 ml per rat). (Meng *et al*., 2019).

**4-Experimental animals:**

This work was carried on 24 healthy adult albino male rats of Sprague–Dawley strain, purchased from the Agricultural Research Center, Giza, Egypt weighted180±10 g.

**B- Methods:**

**1`-Preparation of virgin coconut oil (VCO):**

The VCO had been prepared using **Divina and Keith (2006)** method as follows: Freshly ripened coconuts were peeled by hand and coconut pulp using a coconut slicing machine. then the milk was extracted from the hand grated coconut pulp. The milk extracted from the pulp is set aside, and then the coconut residue sepal is taken for the second extraction process. The sepal was mixed with distilled water in a ratio of 2 sepal: 1 water. The mixture was pressed to obtain a second milk extract. The milk obtained from the first and second extractions was mixed by stirring vigorously for 10 minutes. Then it was cooled in the refrigerator for 3 hours and left to stand, then separate the coconut cream (the fatty part) from the skimmed coconut milk (the water part) by stripping the cream on top. Put coconut cream in a beaker and put it in a water bath at 50°C for 2.5 hours, to isolate coconut protein (latex) from coconut oil. drain the oil from the latex so that it is separated from it using a muslin cloth. Coconut oil is incubated at 50°C for 12 hours to remove all remaining moisture. The extracted oil is stored in a dark place at room temperature. The final result oil was term VCO.

**2-Grouping of experimental rats**

After the adaptation were rats were classified into four groups (6 rats each). Rat were kept under surveillance for seven days for adaptation and fed on basal diet and water was given ad libitum access. The basal diet was prepared according to NRC (1992). The experiment lasted for 60 days, and the rats were randomly classified into control (-ve) group fed basal diet only and three ulcerative colitis groups by intra colonic administration of acetic acid 4% given rectally by single dose (1 ml/ rat ) using 8-cm- soft 6F pediatric catheter and reclassified into control (+ve), and treated group by Omeprazole drug (20 mg/kg b.w) and treated group by coconut oil (2 ml per rat) by stomach tube.

**3-Induction of ulcerative colitis by acetic acid**

Before infection with ulcerative colitis, rats are fasted for 24 hours. Ulcerative colitis was induced in rats by intra-rectal (IR) administration of 1ml, of acetic acid 4% in normal saline solution under ether anesthesia. A 1 mL 4% AA solution was instilled into the rectum into the colon slowly by a soft, 8 cm diameter polypropylene 6F catheter lubricated with gel 2 mm in diameter through the anal canal into the colon. Rats are placed in a horizontal position to prevent the solution from escaping for 30 seconds (Elife *et al.*, 2010).

**4-Calculation of some Nutritional Parameters:**

Nutrition performance and physical recorded and tracked by recording daily food intake, body weight gain and feeding efficiency ratio (FER) as average daily body weight gain divided growth were by average daily food intake. according to (Chapman *et al*.,1959) Rats were sacrificed after 60 days. Relative organs weight = organ weight (g) / Final body weight (g) × 100 . Blood and livers were collected for biochemical analysis

**Biochemical analysis**

When experimentation period was over, animals in all groups were anesthetized by diethyl ether. Blood samples were taken from the eye of rats using heparin capillary tubes, then the serum were obtained after centrifugation at 3000 rpm for 10 minutes in a centrifugal machine(Xiang tian SH-120B). Samples were preserved in a deep freezer at -20 ˚C until used for various biochemical analyzes. Serum (CRP, IL-6, PG2) levels were tested, using laboratory kits from El Gomhouria company in Mansoura city, Dakahlia Governorate, Egypt; as the following:

**5-Anti-inflammation (CRP, IL-6 and PG2) C- reactive protein level (CRP):** was measured depending on the method of Vaishnavi (1993). While interleukin-6 was assessed and quantified according to the method of (Calabrese and John 2014), PG2 prostaglandins was measured depending on the method of (Robert .,1979)

After the completion of the experiment period, the rats were slaughtered; the abdomen was cut longitudinally and cleaned with saline solution to be prepared for the evaluation of antioxidants and free radical in the abdominal tissues.

**6-Serum Antioxidant enzymes:** Confirmation of glutathione peroxides (GPx ) activity according to (Paglia and Valentine 1967),Confirmation of glutathione reduced (GSH) activity according to (Beutler *et al* .,1963) ,Confirmation of glutathione reductase (GSR) activity according to (Goldberg and Spooner 1983),Confirmation of glutathione-S- transferees (GST) activity according to (Habig and Jakoby 1974 ). While Confirmation of superoxide dismutase SOD activity according to (Nishikimi *et al*., 1972). Enzymatic CAT activity was measured according to ( Aebi 1984).

**7- Blood:** Malondialdehyde (MDA) measured calorimetrically according to the method of (Satoh 1978)while $H_2O_2$ hydrogen peroxide was measured depending on the method according to ( Aebi 1984).)

**8-Statistical data analysis:**

All tests were accomplish using computer package of the statistical analysis program (SPSS, version 24, 2016), the

collected data were presented as means ± standard deviations (means ± S.D), statistically analyzed using one way analysis of Variance (ANOVA), and the means between groups were compared by least significant difference (LSD) statistic test, according to (Artmitage and Berry 1987).

## RESULTS AND DISSCUSION

Data in Table (1) showed that final weight, body weight gain, body weight gain% and food efficiency ratio (FER) were significantly lower in ulcerative colitis rat than in the negative group and showed non- significant difference in food intake. Virgin Coconut oil treated group (VCO) showed significant increase in final weight and feed efficiency ratio (FER), however explained non- significant difference in body weight gain % and food intake compared with control (+ve) group. while showed significant decrease in body weight gain and feed efficiency ratio (FER) whoever appeared non- significant difference in final weight, and food intake in compared with control omeprazole drug group. These results in agreement, with (St-Onge *et al*., 2003) who showed that the coconut oil enhances metabolism because it contains of medium-chain triglycerides (MCT). thus the VCO role in weight loss is also consistent with (Famurewa *et al.*, 2018) who said that VCO addition for 14 days induced insignificant body weight change in rats. And leads a weight-loss intervention.

**Table 1. Nutritional indicators of negative control (-ve) group, untreated ulcerative colitis rat (+ve) group and treated by Omeprazole drug or Virgin Coconut oil.**

| Variable / Groups | Initial weight (gm) | Final weight (gm) | Weight gain (gm) | Weight gain% | Food intake (gm) | FER |
|---|---|---|---|---|---|---|
| Control ( -ve) | a<br>187.00±9.63 | a<br>314.50± 11.26 | a<br>127.50±17.65 | a<br>68.67±12.60 | a<br>17.49±0.04 | a<br>0.122±0.02 |
| Control(+ve) | a<br>184.00± 9.00 | c<br>253.83±20.54 | d<br>69.83±19.32 | b<br>38.06±11.04 | a<br>18.48±0.07 | d<br>0.063±0.02 |
| Omeprazole drug | ab<br>178.00±7.18 | b<br>281.67 ±16.84 | b<br>103.67±21.75 | a<br>58.63±14.14 | a<br>17.88±0.01 | a<br>0.097±0.02 |
| Virgin Coconut oil | a<br>184.50±9.42 | b<br>273.17± 14.26 | c<br>88.67±19.35 | b<br>48.48±12.60 | a<br>17.88±0.03 | b<br>0.083±0.02 |

**Mean values in each column having different subscript (a, b, c and d) are significantly at (P<0.05).**

Data in Table (2) showed significant increase in heart, kidney, and liver in ulcerative colitis rat(v+) group, but appeared non- significant difference in stomach weight compared with negative control. Virgin Coconut oil treated group (VCO) showed that significant decrease in organs weight stomach, heart, kidney, and liver in compared with control (+ve) group, while showed non-significant deference in these organs compared with omeprazole drug group.

**Table 2. Some organs body weight of negative control (-ve) group, untreated ulcerative colitis rat (+ve) group and treated by Omeprazole drug or Virgin Coconut oil.**

| Variables / Group | Stomach (gm) | Heart (gm) | Kidney (gm) | Liver (gm) |
|---|---|---|---|---|
| Control ( -ve) | a<br>0.91<br>±0.02 | b<br>0.34<br>±0.03 | b<br>0.87<br>±0.04 | b<br>3.23<br>±0.20 |
| Control(+ve) | a<br>0.93<br>±0.07 | a<br>0.47<br>±0.02 | a<br>1.34<br>±0.14 | a<br>5.01<br>±0.10 |
| Omeprazole drug | b<br>0.66<br>±0.14 | b<br>0.33<br>±0.07 | b<br>0.83<br>±0.08 | b<br>3.27<br>±0.64 |
| Virgin Coconut oil | b<br>0.68<br>±0.07 | b<br>0.36<br>±0.05 | b<br>0.90<br>±0.10 | b<br>3.33<br>±0.49 |

**Mean values in each column having different subscript (a, b, c and d) are significantly at (P<0.05).**

These results in agreement with Asagba *et al.*, (2008) who said that virgin coconut oil supplementation gained a general increase in organ weight in mice but this was only significant ($p<0.05$) increase in liver weight when compared to the healthy group. The significant increase in liver weight may be attributed to the liver's ability to rapidly form cholesterol. (Garg and Blake 1997).

Data in Table (3) showed significant increase in malondialdehyde (MDA) level and, hydrogen peroxide ($H_2O_2$) in ulcerative colitis rat in comparison with the negative control group. Virgin Coconut oil treated group (VCO) showed significant lowering in malondialdehyde (MDA) level and, hydrogen peroxide ($H_2O_2$) in comparison with positive control (+ve) group, while showed non-significant difference in (MDA) and ($H_2O_2$) compared with omeprazole drug group.

**Table 3. (MDA) and ($H_2O_2$) in tissue of negative control (-ve) group, untreated ulcerative colitis rat (+ve) group and treated by Omeprazole drug or Virgin Coconut oil.**

| Groups / variable | Control (-ve) | Control (+ve) | Omeprazole drug | Virgin Coconut oil |
|---|---|---|---|---|
| MDA nmol/g tissue | c<br>1.29<br>±0.13 | a<br>15.27<br>±0.80 | b<br>6.31<br>±1.10 | b<br>7.22<br>±1.01 |
| $H_2O_2$ mM/g tissue | c<br>0.06<br>±0.01 | a<br>2.85<br>±0.51 | b<br>1.25<br>±0.45 | b<br>1.39<br>±0.18 |

**Mean values in each column having different subscript (a, b, c and d) are significantly at (P<0.05): Malondialdehyde (MDA) hydrogen peroxide ($H_2O_2$).**

These results were in agreement with(Zakaria *et al.*, 2011) who reported that the virgin coconut oil is rich in polyphenols and these antioxidants may contribute to the increased levels of antioxidant enzymes, which subsequently reduce lipid peroxidation and inflammation (MDA and $H_2O_2$) in VCO-treated rat.(Arunima and Rajamohan 2013) who said that virgin coconut oil administration controlled oxidative stress, which shows by reducing the formation of lipid peroxidation and protein oxidation products such as malondialdehyde, hydroperoxides, denise syndrome and carbonyl protein in serum and tissues comparing to other oil-fed mice ($P<0.05$),

this interesting excitement returns to the presence of large quantities of phenolic compounds in the VCO.It is distinguished by antioxidant property, which was affected by the deficiency of free radicals with ferulic and b-coumaric acids and other flavonoids that contribute to the antioxidant and pragmatic effects of the kidneys with the VCO polyphenol fraction in this study (Srivastava *et al.*, 2016). Also (Famurewa *et al.*, 2018) reported that the level of malondialdehyde (MDA), showed a lipid peroxidation marker, remarkably reduced activities of hepatic antioxidant enzymes-superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) were markedly increased in VCO diet-fed rats.

Data in Table (4) showed significant decrease in CAT, SOD, GSH ,GSR, GPx and GST levels in ulcerative colitis rat comparing with the negative control group. Virgin Coconut oil treated group (VCO) appeared increased in CAT, SOD, GSH ,GSR, GPx and GST levels comparing with positive group, while showed non significant difference in SOD and GSH whoever found slightly increase in CAT and GST but significant decrease in GSR and GPx levels in compared with omeprazole drug group. Studies that are consistent indicates that the VCO exhibits a protective property against ethanol. The results also indicate a similar trend in cold stress ulcers; it also leads to an increase in the levels of GSH and a decrease in the levels of nitrite MDA, CAT, SOD and GP which showed that VCO demonstrated with its protective action in the ethanol-induction in rats (Meng *et al.*, 2019). There is an raise in the levels of GSH and nitrite corresponding to the reduced in MDA, CAT, SOD and GP appearing by VCO Proposed that there is a strong link of its antiulcer activity with the free radical scavenging activity, similar to omeprazole drug (Biswas *et al.*, 2003). VCO significantly increased superoxide dismutase activity compared to NS-treated mice. Moreover, epithelial tissue restoration and inflammatory cell reduction were greater in VCO treatment compared to omeprazole drug treated animals and control animals.VCO contains high unsaponifiable lipid components like vitamin E and polyphenols, tocopherols, β carotene and phytosterol in stabilizing cell membranes by preventing alterations in membrane lipid polarity and fluidity (Jaarin *et al.*, 2014).these organic acids are thought to have a synergistic effect on phenolic antioxidants so that they can increase their antioxidant activity (Septiana and Hidayah 2010). the ferulic acid found in the VCO was more potent as an antioxidant against LDL oxidation than ascorbic acid. It seems that VCO derives most of its effects from the free-radical scavenging and antioxidant properties of ferulic acid (Gastelluccio *et al.*, 1996).

**Table 4. Some antioxidant enzymes in tissue of negative control (-ve)group , untreated ulcerative colitis rat(+ve) group and treated by Omeprazole drug or Virgin Coconut oil.**

| Variable<br>Groups | CAT<br>(u/g) tissue | SOD<br>(u/g) tissue | GSH<br>(mmol/g) tissue | GSR<br>(u/g) tissue | GPx<br>(u/g) tissue | GST<br>(u/g) tissue |
|---|---|---|---|---|---|---|
| Control ( -ve) | a<br>6.58±0.81 | a<br>632.28±4.20 | a<br>341.67±14.59 | a<br>24.33±2.60 | a<br>66.95±7.24 | a<br>312.67±7.55 |
| Control(+ve) | c<br>1.34±0.16 | c<br>227.83±7.70 | d<br>156.00±12.24 | d<br>6.26±1.50 | c<br>29.56±2.68 | c<br>73.15±2.56 |
| Omeprazole drug | b<br>4.51±0.55 | b<br>403.00±83.01 | b<br>262.50±42.34 | b<br>15.96±1.90 | a<br>52.60±7.83 | b<br>242.83±14.80 |
| Virgin Coconut oil | a<br>5.93±0.38 | b<br>401.83±62.01 | b<br>227.17±21.08 | c<br>10.61±1.27 | b<br>38.60±6.43 | a<br>268.83±59.92 |

**Mean values in each column having different subscript ( a, b, c, d,.)are significantly at( P<0.05).**
**Catalase (CAT) , Superoxide dismutase (SOD), Glutathione reduced (GSH) glutathione peroxidase (GPX) , glutathione reductase (GSR) and glutathione-S-transferase (GST).**

Table (5) showed significant increase in IL-6 and PG2 in ulcerative colitis rats group comparing with the negative control group, while CRP showed slightly increase. Virgin Coconut oil treated group (VCO) showed that significant lowering in CRP,IL-6 and PG2 levels compared with control (+ve) group. However appeared non-significant difference in CRP, IL-6 and PG2 levels in VCO group compared with omeprazole drug group. these studies indicate that the VCO group has a significant increase in anti-inflammatories.(Meng *et al.*,2019) reported that the Virgin coconut oil has antioxidant and anti-inflammatory properties that help control the regulation of prostaglandin synthesis and protect against damage by reactive oxygen species. The effect of VCO polyphenols on blood C-reactive protein (CRP) levels, interleukin-6 (IL-6), and nitric oxide (NO) has been demonstrated (Famurewaa *et al.*, 2018). this is due mechanism behind the anti-inflammatory effect may be because of the ability of the VCO to reverse MTX-induced oxidative stress As indicated by the increase in activities of SOD, CAT, GPx, and GSH as well as an apparent decrease in MDA level and the ability of food polyphenols Modifiers of inflammatory cascades to avoid cause Pathogenesis and evidence of beneficial health effects.

**Table 5. Some serum Anti-inflammation indicators of negative control (-ve) group, untreated ulcerative colitis rat (+ve) group and treated by Omeprazole drug or Virgin Coconut oil.**

| Variable<br>Groups | CRP<br>(Mg/l) serum | IL-6<br>(Pg/ml) serum | PG 2<br>(Mg/l) serum |
|---|---|---|---|
| Control ( -ve) | b<br>0.67±0.15 | c<br>16.32±3.02 | c<br>14.34±1.06 |
| Control(+ve) | a<br>2.13±0.39 | a<br>81.22±6.93 | a<br>38.53±1.38 |
| Omeprazole drug | b<br>0.99±0.37 | b<br>55.93±3.35 | b<br>25.28±1.48 |
| Virgin Coconut oil | b<br>0.83±0.97 | b<br>55.76±7.12 | b<br>24.46±1.59 |

**Mean values in each column having different subscript ( a, b, c, and d)are significantly at (P<0.05).**
**C-reactive protein (CRP) , interleulin-6 (IL-6) and prostaglandin (PG2).**

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## دراسه مقارنة بين زيت جوز الهند البكر ودواء الأوميبرازول على التهاب القولون التقرحي

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تهدف هذه الدراسة الي المقارنة بين تأثير زيت جوز الهند البكر ودواء الأوميبرازول المعالج لألتهاب القولون التقرحي الذي تم احداثة بواسطه حمض الخليك في فئران التجارب، حيث أجريت الدراسة علي ٢٤ من ذكور الفئران البيضاء البالغه التي بتراوح اوزنهم بين ١٨٠± ١٠جم وتم تقسيم الفئران الي أربع مجموعات كل مجموعه تحتوى علي ستة فئران، المجموعة الاولي هي الضابطة السالبة التي تغذت علي الغذاء القياسي فقط خلال فترة التجربة، وباقي المجموعات الثلاثة تم أصابتها بالتهاب القولون التقرحي باعطاء واحد مل لكل فأر من حمض خليك تركيز ٤% بعد صيام ليله واعادة تقسيمهم الي المجموعه الضابطة الموجبة (المريضة، دون علاج) والتي تغذت علي الغذاء القياسي فقط . ومجموعة تم علاجها بالأوميبرازول (٢٠ملجم/ كجم من وزن الجسم يوميا)، ومجموعه تم علاجها بزيت جوز الهند البكر(٢مل لكل فار يومياً) واستمرت الدراسة لمدة ٦٠ يوم. واظهرت نتائج الدراسه عن انخفاض معنوي في الوزن النهائي للفئران والوزن المكتسب ومعدل كفاءه الطعام ووزن اعضاء القلب والكبد والكلي ومستوي الكتاليز وسوبر اكسيد ديسميوتاز ومستويات اكسيد الجلوتاثيون وزياده معنويه في مستوي المالوندالدهيد والبيروكسيد هيدروجين في المجموعه الضابطة الموجبة (المريضه) وذلك بمقارنتها بالمجموعة الضابطة (السليمة).كما اظهرت نتائج المجموعه المعالجه بزيت جوز الهند البكر ودواء الأوميبرازول زياده معنويه في (WG), (FW), (FER) ووزن المعدة ,(GSH) (SOD) (CAT), ونقص معنوي في مستوي(MDA),(PG2),(IL-6),($H_2O_2$) وذلك بمقارنتها بالمجموعة الضابطة الموجبة المريضة كما اظهرت النتائج بوضوح علي عدم وجود فروق معنوية عند العلاج بزيت جوز الهند البكر والأوميبرازول، وأظهرت الدراسة بوضوح أن إعطاء زيت جوز الهند البكر له دور في علاج الفئران المصابين بالتهاب القولون التقرحي المحدث بحمض الخليك.وتوصي الدراسة بضرورة تناول زيت جوز الهند البكر يوميا لتقليل الإصابة بالتهاب القولون التقرحي.

***الكلمات الدالة:*** زيت جوز الهند البكر – التهاب القولون التقرحي – فئران التجارب – حمض الخليك.