# Effects of Honey Consumption on Acetic Acid-induced Colitis in Albino Rats

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### ABSTRACT

This study investigates the effects of honey consumption on colitis healing in albino rats. 45 albino rats (divided into 3 groups - control, low and high dose honey groups) of average weight of 200g were used for this study. Colitis was induced in all groups using 6 % acetic acid. The low and high doses were administered with 0.25ml and 0.5ml of honey respectively for 20 days. The control animals ate normal rat chow. The stools of all animals were scored according to the scale of Masonobu et al (2002) for 20 days. On days 7, 14 and 20, three animals were sacrificed from each group and 8cm of the colon was cut out for gross morphological scoring. The results showed that on day 16 colitis scores were  $0.90 \pm 0.05$ ,  $0.30 \pm 0.02$ ,  $0.50 \pm 0.05$ , on day 20,  $0.80 \pm 0.03$ ,  $0.33 \pm 0.03$ ,  $0.50 \pm 0.05$  for control, low and high doses respectively. Honey consumption increased the colitis healing rate compared to control. It can be concluded from this study that honey consumption has a positive effect on colitis healing and may be useful in patients with inflammation bowel diseases. **Keywords:** Honey, Colitis, Albino rats

# **INTRODUCTION**

Honey is a natural sweetener that is widely consumed especially in the rural areas of southern Nigeria. It has antibacterial properties (Al-Waili, 2004). Colitis is an inflammation of the colon. There are many types of colitis viz:ulcerative, ischemic, crohn's disease, collagenous, chemical colitis etc (Romano et al, 2008). Honey is widely consumed in Nigeria. It serves as nutritional supplement, traditional remedy, and wound dressing (Ajibola 1995). It is a sweet food made by bees using nectar from flowers. The variety produced by honey bees (the genus Apis) is the one most commonly referred to and is the type of honey collected by beekeepers and consumed by humans. Humans began hunting for honey at least 10,000 years ago (EvaCrane, 1983).

Honey gets its sweetness from the monosaccharides fructose and glucose, and has approximately the same relative sweetness as that of granulated sugar (Oregon State University, 2010). It contains tiny amounts of several compounds thought to function as antioxidants including chrysin, pinobanksin, vitamin C, catalase and pinocembrin (Martos, Ferreres and Tomas-Barberan, 2000; Gheldof, Wang and Engeseth, 2002). Antibacterial properties of honey are the result of the low water activity causing osmosis, hydrogen

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peroxide effect (Waikato Honey Research Unit, 2006) high acidity and the antibacterial activity of methylglyoxal (Science Daily, 2008). Colitis is an inflammation of the colon and is often used to describe an inflammation of the large intestine (colon, caecum and rectum). There are many types of colitis viz: auto immune, idiopathic, iatrogenic, vascular and infectious (Beutin, 2006). Signs seen on colonoscopy include: colonic mucosal erythema, ulcers, bleeding. How a given colitis is treated is dependent on it's etiology e.g infectious colitis is usually treated with antimicrobial agents (antibiotics), autoimmune mediated colitis is treated with immune modulators or immune suppressants. This study was undertaken to investigate the effects of honey consumption on acetic acid- induced colitis in albino rats.

## **MATERIALS AND METHOD**

This study was carried out during the period October and November 2011 in the department of Physiology, Faculty of Basic Medical Sciences, Anambra State University, Uli, Anambra State. Forty-five healthy adult albino rats of wistar strain weighing between 180-220g were used in the study. The animals were housed under standard conditions of temperature  $(23 \pm 2^{\circ}C)$  and humidity and 12h light (7.00am - 7.00pm). They were kept in wire meshed cages and fed with commercial rat pellets (Greg feeds, Uli centre, Anambra state) and allowed water ad libitum.

The animals were divided into three groups of 15 rats each. Colitis was induced in all the groups. Group 1 served as control and received normal rat chow and water. Group 2 received low dose of honey (0.25ml), while group 3 received high dose of honey (0.5ml). Male albino rats weighing between 180 - 220g each were used for the Experimental Colitis. Colitis was induced according to the previously described method (Jagtap, Shirke and Phadke, 2004). Animals were deprived of food for 24hrs before the induction of colitis but allowed free access to water. Rats were anaesthetized with thiopental and a flexible catheter (diameter 2mm) was inserted into the anus and the tip was advanced to 8cm proximal to the anus. 1ml of acetic acid 6% was instilled into the colon through cannula for 30s after which fluid was withdrawn. To prevent spillage of solution from rectum, animals were allowed to hang in air by holding their tails for 45-60 seconds.

Honey was administered to the rats by oral cannula. Group 1 (control) did not receive honey. Group 2 (low dose) received 0.25ml while 0.5ml was administered to the high dose group (group 3) daily for 20 days. Every morning all rats from the three groups were scored (Stool Scoring). Each of the rats from each group was brought out kept on a white paper, the tail of the rat was held upwards and then the rat passed stool from its anus. The stool was examined physically and then scored. The following scoring pattern of Masonobu et al (2002) was used

0	-	Normal stool		
1	-	Soft, stool but still formed		
2	-	Soft/wet stool/unformed stool		
3	-	Soft/wet stool + blood		
4	-	Bloody diarrhea		
After scoring the mean, standard deviation, and standard error of mean were calculated.				

Every one week, 3 animals from each group were sacrificed. The rats were dissected open using a scapel. The colon was traced to the anus and was cut open and the inside washed with clean water from a running tap. A hand lens of X3 magnification was used to examine and score the internal structure of the colon. The following scoring pattern of gross morphological damage was used.

0	-	No damage
1	-	Localized hyperemia with no ulcers
2	-	Linear ulcers with no significant inflammation.
3	-	Linear ulcer with inflammation at one side.
4	-	More site of ulcers and inflammation, the size of ulcer < 1cm.
5	-	Multiple inflammation and ulcers, the size of ulcer > 1cm.

After scoring, the mean, standard error of mean were calculated. The colons from each were weighed on an electronic weighing balance and values taken. The data obtained were expressed as mean  $\pm$  SEM (standard error of mean). The student's t-test was applied and p - values were determined. Differences were considered significant at p < 0.05.

# **RESULTS AND DISCUSSION**

Table 1 presents the Stool score. It indicates that on day 16 the scores were  $0.90 \pm 0.05$  (control)  $0.30 \pm 0.02$  (low dose).  $0.50 \pm 0.05$  (high dose). On day 20,  $0.80 \pm 0.03$  (control)  $0.33 \pm 0.03$  (low dose),  $0.5 \pm 0.05$  (high dose). The acetic acid induced colitis is one of the commonly used experimental models while screening natural products and drugs active against inflammatory bowel disease. Consistent with previous reports in this study, intrarectal administration of acetic acid caused diffused inflammation leucocyte infiltration ulcerated mucosa and necrosis (Hager, Medany, Eter and Arafa, 2007). Table 2 shows that honey consumption significantly reduced tissue damage in rat model of colitis induced by acetic acid as evidenced by macroscopic changes and variations in tissue (colon) weight. Using the gross morphological damage result where the scores were  $4 \pm 0.10$ ,  $2.5 \pm 0.05$  and  $2.5 \pm 0.05$  for control, low and high dose respectively at day 20. From the results, there was a significant reduction in tissue damage in the experimental group when compared to the control group (p<0.05). However there was no significant difference between the low and high dose groups.

Table 3 shows the weight of damaged colon tissue which is considered an indicator of the severity and extent of inflammatory response. The weight score from the table shows  $1.72 \pm 0.28$ ,  $1.02 \pm 0.40$  and  $1.29 \pm 0.44$  for control, low and high dose respectively at 20 days. Honey administered groups showed a decrease in colon weight and macroscopic scores for the inflammation. Based on this study, it can be stated that honey consumption significantly improved the healing rate of acetic acid induced colitis in albino rats. Extrapolating this to man, honey consumption may be beneficial in people with inflammatory bowel diseases. The aim or treatment of ulcerative colitis is the induction and maintenance of remission of symptoms and mucosal inflammation. Hence, treatment of ulcerative colitis

is difficult because of its complex etiology. Although drugs like 5-aminosalicylic acid sulfapyridine and glucocorticoids could inhibit the inflammatory mediators through different mechanisms locally or systemic, these agents are limited in their use due to the involvement in the down-regulation of the immune and inflammatory responses of ulcerative colitis including adverse reactions during prolonged treatment and high relapse rate. Recent studies suggest that honey has ameliorative properties in diabetes, skin ulcers and infections (Al-Waili, 2004 and Molan, 1992).

### Table 1: Stool Scoring

	Day 16	Day 20		
	Mean $\pm$ SEM	Mean $\pm$ SEM		
Control	$0.9\pm0.05$	$0.80\pm0.03$		
Low Dose	$0.30 \pm 0.02$	$0.33\pm0.03$		
High Dose	$0.05 \pm 0.05$	$0.5\pm0.05$		
Source: Experimentation, 2011				

### Table 2: Gross Morphological Damage

	1 0	0				
	Control	Low Dose	High Dose			
Days	Mean $\pm$ SEM	Mean $\pm$ SEM	Mean $\pm$ SEM			
7	$2.5\pm0.05$	$2.5\pm0.05$	$2.5\pm0.05$			
14	$0.66 \pm 0.02$	$1.0 \pm 0.10$	$0.66 \pm 0.02$			
20	$4 \pm 0.10$	$2.5 \pm 0.05$	$2.5\pm0.05$			
Source: Experimentation 2011						

Source: Experimentation, 2011

### Table 3: Tissue Weight Scoring Pattern

	0 0				
	Control	Low Dose	High Dose		
Days	Mean $\pm$ SEM	Mean $\pm$ SEM	Mean $\pm$ SEM		
7	$0.89 \pm 0.16$	$1.15\pm0.17$	$1.18\pm0.23$		
14	$1.32 \pm 0.14$	$1.09 \pm 0.30$	$1.24 \pm 0.33$		
20	$1.72 \pm 0.28$	$1.02\pm0.40$	$1.29\pm0.44$		
Source: Experimentation, 2011					

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