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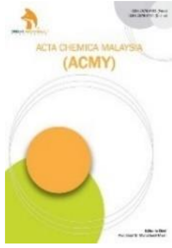

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RESEARCH ARTICLE

1,2-DIMETHYLHYDRAZINE-INDUCED COLON NEOPLASIA: HISTOPATHOLOGICAL AND BIOCHEMICAL ASSESSMENTS FOLLOWING ADMINISTRATION OF BRASSICA JUNCEA EXTRACT

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ABSTRACT

This experimental study investigated the antineoplastic effects of aqueous Brassica juncea extract (AEBJ) on colon neoplasia in Wistar rats. Forty-two rats were randomly divided into six groups of seven rats each. For the initial three months: Group A (control) received standard feeding, Groups B-F received 25 mg/kg body weight of 1, 2 dimethylhydrazine (DMH) solution, in addition to standard feeding. For the subsequent three months: Groups A and B continued with standard feeding only, Group C received the standard anticancer drug Zolon-100, Groups D, E, and F received 150, 300, and 600 mg/kg body weight of AEBJ, respectively. The study revealed that DMH administration induced neoplastic changes in the colon, characterized by: ulcerated epithelial lining, extensive inflammatory cell infiltration in the lamina propria, disorganized pleo-morphic glands, epithelial hyperchromasia and elevated levels of carcinoembryonic antigen (CEA), a tumor marker. Treatment with graded doses of AEBJ significantly mitigated these neo-plastic changes. Notably, the highest dose (600 mg/kg) exhibited the most pronounced ameliora-tive effects, indicating a dose-dependent response. In conclusion, this study demonstrates that AEBJ exhibits potent antineoplastic activity against DMH-induced colon neoplasia in Wistar rats. Notably, AEBJ's efficacy surpasses that of the standard reference drug, Zolon-100. These findings suggest that AEBJ holds promise as a therapeutic agent for the prevention and treatment of colon cancer, warranting further investigation.

KEYWORDS

Brassica juncea, 1, 2 dimethylhydrazine, histopathology, Wistar rats, neoplastic, inflammatory cells.

1. INTRODUCTION

Neoplasia is characterized by the uncontrolled and excessive growth of abnormal tissue, resulting in the formation of a neoplasm. Although often used interchangeably, the terms "neoplasm" and "tumor" have distinct meanings (Ho et al., 2017). A tumor refers specifically to a swelling or lump, typically associated with inflammation. In contrast, a neoplasm encompasses any abnormal new growth, lesion, or ulcer. Both tumors and neoplasms involve abnormal cell growth, which can be either malignant or benign. Cancer, a malignant neoplasm, poses a significant threat worldwide (Ma et al., 2011). Each year, tens of millions of new cancer cases are diagnosed worldwide, with over half resulting in mortality. Existing cancer therapies have limited effective-ness, as evidenced by cancer statistics and are often accompanied by severe side effects (Kazem-ipoor et al, 2012). Colorectal cancer is the third most commonly diagnosed cancer and a leading cause of morbidity and mortality globally (Gellad and Provenzale, 2010). Plant-based remedies have been reported to be free from undesirable side effects (Philomena, 2011). Moreover, ap-proximately 8% of hospital admissions worldwide are attributed to adverse effects of synthetic drugs highlighting the need for safer treatment alternatives. Research suggests that certain phyto-chemicals, such as glucosinolates found in cruciferous vegetables like broccoli, cabbage, Brussels sprouts, and Brassica juncea, may have cancer-preventive properties. B. juncea, derived from mustard plants, are a rich source of these phytochemicals (Philomena, 2011) B. juncea are typically 1-2 millimeters in diameter and range in color from yellowish-white to black (Nasri, 2013). They are a common spice in regional cuisine and come in three main varieties: black mustard (Brassica nigra), brown Indian mustard (B. juncea), and white/yellow mustard (B. hirta/Sinapis alba). When ground and mixed with liquids, the seeds produce the familiar yellow condiment known as prepared mustard. Mustard has been used for centuries in traditional medicine, and its potential anticancer properties are currently being researched. Both table mustard and mustard greens have shown promise as cancer inhibitors. B. juncea contain a range of nutritionally rele-vant compounds, including: 20 different glucosinolates, Vitamins A, B6, and C dietary folate, Omega-3 fatty acids, minerals like magnesium, potassium, selenium, manganese, phosphorus, and copper (Haq, 2004). B. juncea, in particular, has been extensively used in Ayurvedic medicine for its potential to heal the bronchial system, eliminate intestinal parasites, ease sprains and other pains. Physicians in ancient Greek and Roman civilizations also used B. juncea for various health purposes. The plant is said to help alleviate conditions like flatulence, colds, catarrh, chest and bladder ailments. Additionally, it is used in footbaths to combat fatigue and promote circulation (Wen et al.,

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2012).

Colon cancer is a multifactorial disease, influenced by both genetic and environmental factors (Juozas et al., 2014). Exposure to hydrazine derivatives, prevalent in environmental pollution and food contamination, increases the risk of developing colon cancer (Ratasark and Chewonavin, 2014). 1,2-Dimethylhydrazine (DMH) is an environmental pollutant commonly used as an experimental model to study colon cancer pathogenesis (Kanwal and Gupta, 2012). While research suggests that B. juncea inhibits colon cancer formation in normal and obese rats when added to their diet, there is a notable lack of studies investigating the effects of B. juncea extracts on established colon cancer cases and the inhibitory potential of B. juncea against colon cancer.

2. Material And Method

2.1 Collection and Authentication of B. juncea

B. juncea was sourced from the Uselu market in Benin City, Edo State. To confirm its identity, a sample was submitted to the Department of Plant Biology and Biotechnology at the University of Benin. The sample was authenticated and assigned the herbarium number UBH-B539.

2.2 Extraction and Preparation of Aqueous Extract

The fresh B. juncea sample was cleaned with tap water, air-dried, and pulverized. A 250g portion of the powdered sample was soaked in 1000ml of water for 72 hours. The resulting crude aqueous extract was filtered, and the filtrate was freeze-dried using Kumar's method (2019) at the University of Benin's Natural Product Research Laboratory. The freeze-dried extract was stored in a refrigerator at 4°C for future use.

2.3 Animal Selection and Housing

Forty-two Wistar rats, weighing between 180-200g, were obtained from the Animal House, Department of Anatomy, University of Benin. The animals were housed under controlled laboratory conditions, which included temperature of 28 ± 2°C, relative humidity of 50 ± 5% and light-dark cycle of 12 hours. The rats had unrestricted access to food and water, ensuring their optimal comfort and well-being throughout the study.

2.4 Preparation and Administration of AEBJ

A stock solution was prepared by dissolving 10g of AEBJ in 100ml of water. This solution was then orally administered to Wistar rats at doses of 150, 300 and 600 mg/kg body weight. The administration period lasted for three months, following the determination of the median lethal dose (LD50) using Lorke's method (1983).

2.5 Preparation and Administration of DMH

1,2-Dimethylhydrazine (DMH) was dissolved in freshly prepared 1mM EDTA, and the pH was adjusted to 6.5 using 1mM sodium carbonate. To induce neoplasia, rats received a subcutaneous injection of DMH at a dose of 25mg/kg once a week for 15 weeks.

2.6 Experimental Design and Treatment Protocol

Forty-two Wistar rats were randomly assigned to six experimental groups (n=7). The groups were designated as follows: Group A (control): received normal feeding only, Groups B-F: received 25 mg/kg body weight of DMH solution in addition to normal feeding for the initial 3 months. Subsequent treatment protocols for the next 3 months were: Group A: continued normal feeding, Group B: no additional treatment, Group C: received the standard anticancer drug Zolon-100, Groups D, E, and F: received 150, 300, and 600 mg/kg body weight of AEBJ, respectively.

2.7 Tissue Harvesting and Sample Collection

Following the treatment period, all animals were sacrificed under chloroform anesthesia, 24 hours after the final administration. A midline abdomino-thoracic incision was made to expose the abdominal viscera. The colon was carefully harvested, dissected, and immediately fixed in 10% formol saline for histopathological examination. Blood samples were collected by cardiac puncture in sample separation bottles for biochemical analysis.

2.8 Histological Processing and Analysis

The colon tissues, fixed in formol saline, underwent standard histological processing. This in-cluded dehydration in a graded ethanol series (70-100%), xylene clearing and paraffin wax em-bedding. Thin sections were then cut, stained with hematoxylin and eosin (H&E) using the method described by Drury and Wallington (1980). The stained sections were examined under a light microscope to evaluate histopathological changes and alterations in tissue architecture.

2.9 Carcinoembryonic Antigen (CEA) Analysis

Carcinoembryonic antigen (CEA) analysis was performed using the CEA ELISA test. This test is based on the principle of solid-phase enzyme-linked immunosorbent assay (ELISA). The analysis was conducted in accordance with the manufacturer's standard protocols.

2.10 Data Analysis and Statistical Significance

The results obtained were expressed as mean ± standard error of the mean (SEM). Statistical significance was determined using a P-value threshold of 0.05 ($p < 0.05$), where values below this threshold were considered statistically significant.

3. Results

The control group (Group A) showed normal colon cytoarchitectures, with normal epithelial lining, lamina propria, glands lined by normal goblet cells, muscularis mucosa, submucosa and muscularis propria (Plate 1). The colon of the group given 1,2, DHM only (Group B) showed ulcerated epithelial lining, heavy lamina propria infiltrates of inflammatory cells, disorganized pleomorphic glands and epithelial hyperchromasia (Plate 2). The colon of the group given 1,2, DHM plus standard drug (Group C) showed normal glands lined by goblet cells, distorted glands with hyperchromasia nuclei, lamina propria infiltrates of inflammatory cells and vascular congestion (Plate 3). The colon of the group given 1,2, DHM plus 150 mg/kg body weight of AEBJ (Group D) showed normal glands lined by goblet cells, distorted glands lined by hyperchromatic nuclei, lamina propria infiltrates of inflammatory cells and vascular congestion (Plate 4). The colon of the group given 1,2, DHM plus 300 mg/kg body weight of AEBJ (Group E) showed normal glands lined by goblet cells and mild activation of lamina propria lymphoid aggregates (Plate 5). The co-lon of the group given 1,2, DHM plus 600 mg/kg body weight of AEBJ (Group F) showed nor-mal glands lined by goblet cells, lamina propria infiltrates of inflammatory cells and submucosa vascular congestion (Plate 6).

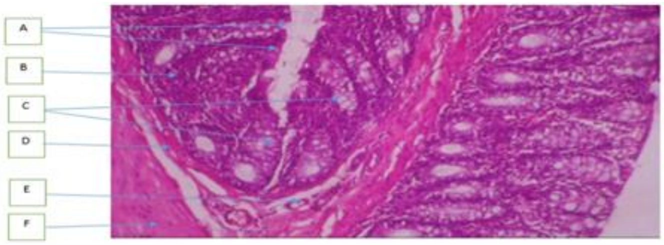

Plate 1: Photomicrograph of the colon of the control group (group A) showing normal colon cytoarchitectures, with normal epithelial lining (A), lamina propria (B), glands lined by normal goblet cells(C), muscularis mucosa (D), submucosa (E) and muscularis propria (F). H and E 100x.

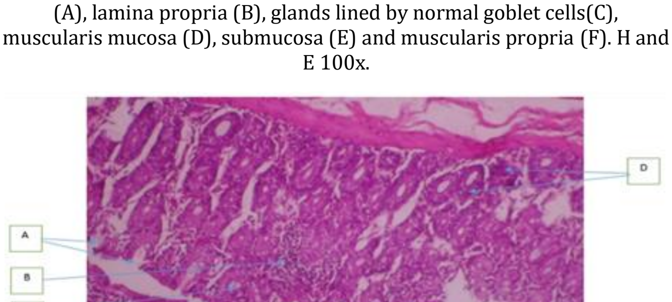

Plate 2: Photomicrograph of the colon of rats given 1,2, DHM only (Group B), showing: ulcerated epithelial lining (A), heavy lamina propria infiltrates of inflammatory cells (B), disorganized pleomorphic glands (C) and epithelial hyperchromasia (D). H and E 100x.

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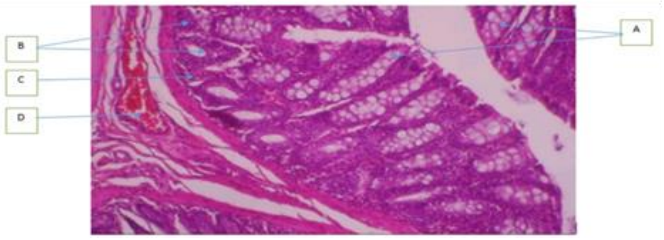

Plate 3: Photomicrograph of the colon of rats given 1,2, DHM plus standard drug (Group C) showing normal glands lined by goblet cells (A), distorted glands with hyperchromasia nuclei (B), lamina propria infiltrates of inflammatory cells (C) and submucosal vascular congestion (D) H and E 100x.

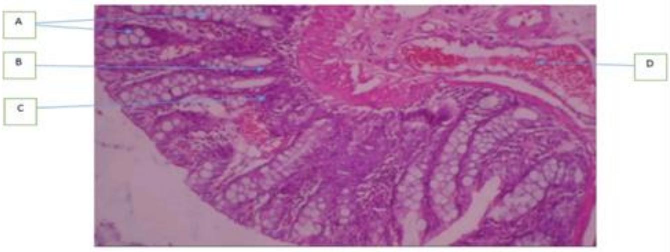

Plate 4: Photomicrograph of the colon of rats given 1,2, DHM plus 150 mg/kg body weight of AEBJ (Group D) showing normal glands lined by goblet cells (A), distorted glands lined by hyperchromatic nuclei (B), lamina propria infiltrates of inflammatory cells (C) and vascular conges-tion (D) H and E 100x.

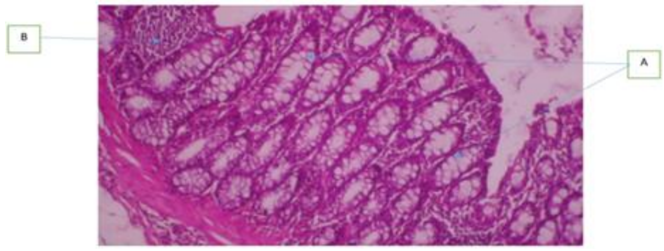

Plate 5: Photomicrograph of the colon of rats given 1,2, DHM plus 300 mg/kg body weight of AEBJ (Group E) showing normal glands lined by goblet cells (A) and mild activation of lamina propria lymphoid aggregates (B) H and E 100x.

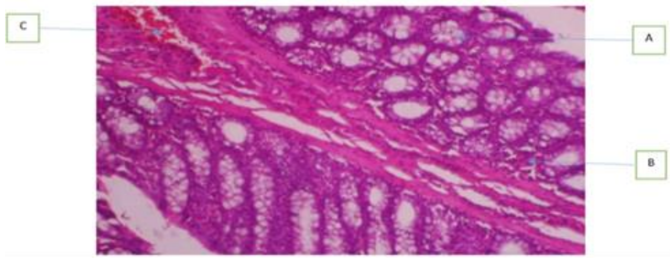

Plate 6: Photomicrograph of the colon of rat given 1,2, DHM plus 600 mg/kg body weight of AEBJ (Group F) showing normal glands lined by goblet cells (A), lamina propria infiltrates of inflammatory cells (B) and submucosa vascular congestion (C). H and E 100x.

Table 1: Effects of administration on the Carcinoembryonic antigen (CEA)

	Control	1,2, DHM only	1,2, DHM + Standard drug	1,2, DHM + 150mg/kg extract	1,2, DHM + 300mg/kg extract	1,2, DHM + 600mg/kg extract	P-value
CEA (ng/ml)	0.08±0.01	0.23±0.08*	0.09±0.00	0.08±0.00	0.09±0.00	0.12±0.00	0.129

**Significantly different from the control group*

4. Discussion

Colon cancer, also referred to as colorectal cancer (CRC), poses a significant threat to global health, ranking as the third most commonly diagnosed cancer and the second leading cause of cancer-related deaths worldwide (Maida et al., 2024). Early detection through regular screening greatly enhances the prospects of successful treatment, underscoring the importance of developing and utilizing effective diagnostic methods. At its core, cancer is primarily triggered by DNA damage (Gotzsche and Jorgensen, 2013). Spontaneous DNA damage occurs at an alarming rate, with over 60,000 new lesions forming daily in each human cell, primarily due to cellular metabolism and the inherent properties of DNA in aqueous environments at body temperature. Additionally, high levels of bile acids, often resulting from a high-fat diet, can cause DNA damage and contribute to the development of colon cancer (Dubas and Ingraffea, 2013). Moreover, re-search suggests that macrophages and neutrophils in inflamed colonic epithelium are sources of reactive oxygen species, which induce DNA damage and initiate colonic tumorigenesis (Klutstein et al., 2016; Cakir et al., 2012). The histopathological examination of colon tissues in this study revealed that neoplasia, characterized by glandular epithelial dysplasia, was induced in rats treat-ed with DMH alone. This was evident from the presence of dysplastic features, including pleomorphic glands and cells, hyperchromatic nuclei, lymphocytic infiltration of the lamina propria and depletion of goblet cells. Goblet cells play a crucial role in maintaining tissue homeostasis through mucin production. Depletion of these cells disrupts tissue homeostasis, promoting cellular proliferation. These findings are consistent with previous studies, who reported the formation of aberrant crypt foci following 1,2-dimethylhydrazine-induced colon carcinogenesis. Similarly, the researcher observed the formation of aberrant crypt foci in rats treated with DMH alone, in a study investigating bone marrow cell therapy on DMH-induced colon cancer in rats (Gellad, 2010; El-Khadragy et al., 2018). Following the induction of neoplasia and subsequent treatment with AEBJ at concentrations of 150, 300, and 600 mg/kg, a noticeable improvement in morpho-logical differentiation of tissue sections was observed compared to the control group and the group treated with 1,2-DMH only. Notably, the group receiving 600 mg/kg AEBJ exhibited the most significant morphological differentiation. This improvement was not attributed to spontaneous resolution, as the recovery group (rats treated with 1,2-DMH and left to recover) still re-tained neoplastic features. The AEBJ intervention appeared to up-regulate mucosal epithelial cell differentiation of dysplastic cells. This finding is consistent with previous who reported that AEBJ prevented tissue growth and induced apoptotic death of SW480 cells (a human colon cell line) in a study on azoxymethane-induced colon carcinogenesis (Half et al., 2009; Clisby et al., 2008). A comparative analysis of the antineoplastic effects of Zolon (a standard anti-colon cancer drug) and AEBJ revealed that AEBJ exhibited greater potency and better ameliorative outcomes. This supports previous research indicating the superiority of phytomedicines in cancer treatment (Philomena, 2011; Nasri, 2013). Given the adverse effects of orthodox drug toxicities phytotherapeutics, such as AEBJ, should be prioritized in cancer treatment (Ali et al., 2015).

Biochemical analysis using carcinoembryonic antigen (CEA) measurements further corroborated the antineoplastic effects of AEBJ. The results, presented in the table, demonstrate CEA levels across various rat groups, consistent with previous findings by Duffy (2001). Notably, rats treat-ed with 1,2-DMH alone exhibited the highest CEA measurements, indicating increased neo-plastic tendencies. Rats administered 150 mg/kg body weight of AEBJ showed the lowest CEA concentrations, suggesting reduced neoplastic tendencies. Interestingly, this outcome diverges slightly from the morphological results, which indicated the best outcome with 600 mg/kg body weight of AEBJ intervention. This discrepancy highlights an area for further research. Nevertheless, this finding underscores the limitations of CEA as a tumor marker, as previously reported. This aspect of the study appears to be novel, as no related studies were found.

5. Conclusion

Despite ongoing efforts, the battle against cancer remains far from won, with rising prevalence and mortality rates for various types of cancer, including colorectal cancer. This study provides compelling evidence of AEBJ's efficacy in combating early pathological changes associated with colon cancer. Leveraging this potential could be a game-changer in the fight against cancer, particularly given AEBJ's ability to reverse

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established neoplastic changes. Furthermore, considering the toxicity risks associated with synthetic drugs, exploring the potential of herbal therapies like AEBJ could yield immense benefits. As the world continues to grapple with the complexities of cancer, embracing natural, effective, and safer alternatives could be a crucial turning point in the quest to conquer this debilitating disease.

DECLARATIONS

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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