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Clinicopathological Characterization and Survival Analysis of 1,2-Dimethylhydrazine-Induced Colorectal Cancer in Swiss Albino Mice

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Abstract

Colorectal cancer (CRC) remains a leading cause of global cancer-related mortality, and preclinical models remain the mainstay to evaluate different treatment regimes. This study aims to characterize the clinicopathological progression of 1,2-dimethylhydrazine (DMH)-induced CRC in Swiss albino mice. Twelve male mice were divided into Control and DMH-treated (15 µg/g i.p., weekly) groups, and were monitored for clinical appearance, hematology, serum biochemistry, and survival analysis subsequently for 12 weeks. Post-sacrifice, parameters like gross morphology, tumor burden, and histological changes in the colon, liver, and lungs were assessed. Notably, DMH-treated mice exhibited 100% tumor incidence with significant clinical signs, including poor grooming and stool alterations. Hematological analysis revealed there is a marked decrease in hemoglobin and WBC counts, with significant SGOT elevation, suggesting potential hepatic stress. Further, histological analysis confirmed hallmarks of human sporadic CRC, like mucosal thickening and goblet cell depletion. Kaplan-Meier analysis showed a survival rate of 67% in the diseased group by week 12. Our study suggests that the DMH-induced model successfully replicates the adenoma-carcinoma sequence of human CRC, and the treatment phase for preclinical analysis can be started from week 13. Furthermore, sample sizes should be adjusted in the study groups, keeping in mind the 33% induction related mortality.

Keywords: Colorectal cancer, 1,2-dimethylhydrazine (DMH), Swiss albino mice, hematological analysis, histopathological analysis, survival analysis

Introduction

As per the recent report of the GLOBOCON 2022, globally, colorectal cancer (CRC) is the third most common cancer in terms of incidence (1.9 million, 9.8%) and the second most common cause of cancer-related mortality (0.9 million, 9.4%) (1). Worldwide, it follows the trend of a higher prevalence rate in males than in females and in developed countries than in developing countries. Still, a higher mortality rate is reported from developing countries (2). Based on the anatomical location, CRC is divided into right-sided (proximal) colon cancer (RSCC) and left-sided (distal) colorectal cancer (LSCRC). They apparently have distinct clinicopathological parameters where RSCCs are typically bulky, exophytic, macroscopic lesions that project towards the lumen and causes anemia. LSCRCs are infiltrating in nature, encircle the lumen and cause obstruction. Further, RSCCs are commonly diagnosed in advance stage and are more incident in females, while LSCRCs are more common in males and younger patients (3,4). Earlier, considered a disease of the old, CRC is gaining prevalence in the younger population, with higher worldwide reporting of early-onset CRC (1,5).

The poor prognosis of CRC stems from non-specific symptoms and the late detection of the disease. Five-year survival rates for CRC depend on the stage of disease at the time of initial diagnosis, typically range from 90% for localized disease to 70% for regional and subsequently to 10 % for metastatic cancer (6). Developed countries have reduced mortality rates and disease burden by implementing extensive routine screening programmes which enable early and interval cancer detection (7,8). Interval cancer is described as cancer that is detected within the appropriate surveillance interval despite following guideline recommendations (9). Various effective screening tests are available, including non-invasive stool-based testing (guaiac testing and fecal immunochemical tests (FITs)), FIT-DNA (cologuard), and invasive tests such as computed tomography colonography, flexible sigmoidoscopy, and colonoscopy (7,10)

Various modifiable and non-modifiable factors are associated with CRC. Some commonly reported modifiable risk factors include dietary habits such as high intake of processed foods, red and processed meat, a diet high in fat and carbohydrates and low in fibres, obesity, a sedentary lifestyle, and alcohol abuse. Certain comorbid conditions such as diabetes and psychological stress are also positively correlated with CRC (11,12). A recently studied association is with intestinal microbiota, as many gut microbiomes are positively or negatively associated with the development of CRC. Certain bacterial species such as Streptococcus bovis, Enterotoxigenic Bacteroides, Enterococcus faecalis, Escherichia coli are positively associated with CRC while certain bacterial species such as Lachnospiraceae are negatively associated. Notably, some probiotic species such as Clostridium butyicum and S.thermophilus are hypothesised to have a protective effect against CRC (12,13). Some commonly reported non-modifiable risk factors include Lynch syndrome, Familial adenomatous polyposis (FAP), a germline mutation and chronic inflammatory bowel disease (IBD) (11,12).

Historically, animal and cell-based models have been used to evaluate drug efficacy and the immune response. Mice have been the preferred model for a long time due to several advantages over other animals, including their short lifespan, ease of generation, maintenance, and handling, which reduce costs. The most significant factor is that genetically and physiologically mice are very close to humans, and their genome has been sequenced. This allows for the comparison of the human genome with the mouse genome. Understanding the function of human genes is crucial for applying this knowledge to human disease and developing new strategies and mechanisms to prevent, detect and treat colorectal cancer (CRC) before progressing to clinical trials (14–16).

Notably, advanced CRC presents several complications, including cachexia, anemia, diarrhoea or constipation. The present study was performed to analyze and understand the development of preneoplastic and neoplastic

lesions in the DMH-induced Swiss albino mice model for CRC. Also, this study helps us estimate the time at which we can start the treatment phase for CRC using this mouse model. Further, this study helps us to understand the mortality rate in the diseased group, so that we can plan an appropriate number of mice per group for our CRC preclinical study for a metronomics combination.

Materials and Methods

Chemical

Chemical inducing agent, 1,2-Dimethyl hydrazine (DMH) was procured as a gift from Dr Abhilasha Sharma, Department of Life Sciences, Gujarat University, Ahmedabad.

Animals

Twelve male Swiss albino mice, six in each group, weighing between 25-35 g and aged between 8-10 weeks, were acquired from the VNS Group of Institutions, Faculty of Pharmacy, Vidhya Vihar, Neelbud, Bhopal (M.P.). The animals were kept in regulated, well-ventilated cages at the Central Animal House Facility, Pharmacology Lab, VNS Group of Institutions, Faculty of Pharmacy, Vidhya Vihar, Neelbud, Bhopal (M.P.). A 12-hour light/dark cycle was observed, the standard temperature was kept at 22 ±2 ◦C, and the relative humidity was adjusted at 60–70%. Water and a regular pellet meal were freely available to the mice. The Institutional Animal Ethics Committee (IAEC) accepted the experimental protocol with reference number PH/IAEC/VNS/2K23/47. Experiments were performed following the Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA) guidelines. An attempt was made to lessen the animals' suffering throughout the trials.

Grouping and induction of CRC

Group 1: Normal control (NC, 0.001M EDTA)
Group 2: Disease control (DC, dissolve the DMH in 0.001 M EDTA to 3.7 mg/mL)

Experiments protocol

CRC Induction

After one week of acclimatization of the animals, CRC induction using DMH was performed by intraperitoneal injections (i.p.) (15 µg/g body weight) once a week for 12 weeks. DMH solution was prepared in EDTA immediately before use, and the pH of the solution was adjusted to 6.5 with 8N NaOH (17).

Prior sacrifice

Animals were observed for the following parameters once a week throughout the study period: *General appearance and state of consciousness:* Body weight, food and water intake, hair appearance and grooming, mental condition, and general appearance and level of consciousness. *Behavior:* Reaction to outside stimuli. *Clinical signs:* stool appearance and hydration level. Before sacrifice, animals were kept on 12 hours fasting, followed by 2% isoflurane as anesthesia on the final day of the 4-week induction period to obtain blood samples from the retro-orbital sinus with a capillary tube and collected in two different tubes (with and without EDTA).

Post sacrifice

Following the collection of blood, all animals were sacrificed humanely using the cervical dislocation method, and their colon, liver, kidney, lungs, and spleen were thoroughly removed, weighed, and studied for final body weight (FBW, g), absolute weight (AW, g), and relative weight (RW, %). Further, histological slides of the colon, liver, and lungs were studied under the microscope. Additionally noted were the tumor load, tumor count, and tumor volume.

Survival analysis

Throughout the experiment, the survival of animals from both groups was monitored and documented. Kaplan-Meier curves were employed to conduct the survival analysis.

Hematology

Whole blood analysis

Blood samples collected in EDTA-coated tubes were used to provide valuable information regarding the different types of blood cells present in both treatment groups, using the Dia-count 60 Auto Hematology Analyzer.

Biochemical assay

Blood samples were allowed to clot by leaving the tube undisturbed at room temperature for 2-4 hours. This was followed by centrifugation at 10,000 rpm for about 10 minutes at 4°C. Then the separated serums were carefully collected in a fresh tube and stored at -80°C until further analysis. Vital organ functional assessment includes liver function tests (SGOT, SGPT) and kidney function tests (serum creatinine, blood urea nitrogen (BUN)).

Gross analysis

Post-euthanasia of the animals, the colons were excised through midline abdominal incision, followed by thorough flushing with ice-cold phosphate-buffered saline (PBS, pH 7.4). Subsequently opened longitudinally along the anti-mesenteric border and examined macroscopically for any visible lesions and pathological changes.

Further, tumor count, tumor volume, and tumor burden were performed. For tumor count, the colon was inspected for visible lesions and the number of tumor nodules in both groups. Tumor burden was calculated by counting the number of tumor nodules/animal. Tumor volume was determined using a digital Vernier calliper (Camarillo, USA) and was estimated from two-dimensional tumor measurements using the formula:

Tumor volume (mm^3) =length (mm) x width2(mm^2)
and we classified tumors as smallest (1 mm^3 or less), small (1-10 mm^3), medium (10-20 mm^3) and large (>20 mm^3).

Histological analysis

To prepare histological slides of tissue samples for examination, we first thoroughly wash them with ice-cold saline, followed by overnight fixation in 10% neutral buffered formalin. Furthermore, the fixed tissues were dehydrated with different grades of ethanol (50-90%) for 24 hours and then embedded in paraffin wax to create blocks. Thin sections (4-5µm) from the blocks were then cut using a microtome.

Further, to prepare the histological sections, we deparaffinized the slides by placing them in xylene solution twice for 5 minutes, followed by rehydrating with a sequence of graded ethanol and staining them with Hematoxylin and Eosin (H&E) reagent.

To perform the histological analysis, a light microscope (Magnus MLXi) with a digital camera (Sony) and ImageJ software was used for analysis. We prepared four animals per group and five slides per animal for histological examination. Magnifications of 10X and 40X were used to examine and capture the general morphology, tissue architecture, and cellular organization.

Statistical analysis

All data for weekly report analysis, blood cell count, liver and renal function tests, and histopathological scores were initially assessed for normality of distribution using the Shapiro-Wilk test. Data were considered normally distributed if the p-value was 0.05 or higher. Further, for normally distributed data, comparisons between groups were performed using one-way ANOVA. For ordinal data, the Wilcoxon Signed Rank Sum Test were employed. All statistical analyses were performed using R (version 4.4.2), a freely available software, and results were presented as Mean ± SEM. A p-value less than 0.05 was considered statistically significant in the study.

Results

General findings

Analysis of the weekly report chart shows that, although there is no significant difference in the percentage weight gain between the two groups, there are significant differences in other parameters, such as hair appearance, Stool appearance, Hydration, Response to external stimuli, and Mental status, which clearly indicate signs of progressive disease (Fig. 1).

An independent t-test between the groups revealed no significant difference in the absolute weight (AW, g), relative weight (RW, %) of internal organs and final body weight (FBW, g) of animals. The p-values were not significant (Table 1).

Tumor burden was found to be 14 ± 2 in the DMH-treated group, with all tumors being the smallest category (1 mm^3 or less). Notably, tumor incidence was 100 % in the DMH-treated group.

Table 1: Absolute weight (AW, g) and relative weight (RW, %) of internal organs, and animals' final body weight (FBW, g) in both groups (mean ± SE)

	Untreated (n=6)	**DMH-treated** (n=4)
Heart		
AW	0.20 ± 0.06	0.17 ± 0.04
RW	0.58 ± 0.01	0.50 ± 0.02
Lung		
AW	0.25 ± 0.03	0.21 ± 0.07
RW	0.72 ± 0.05	0.61 ± 0.01
Spleen		
AW	0.18 ± 0.01	0.20 ± 0.02
RW	0.52 ± 0.08	0.58 ± 0.06
Liver		
AW	1.90 ± 0.02	1.70 ± 0.03
RW	5.51 ± 0.06	4.96 ± 0.04
Left kidney		
AW	0.27 ± 0.06	0.24 ± 0.05
RW	0.78 ± 0.02	0.70 ± 0.05
Right Kidney		
AW	0.25 ± 0.01	0.25 ± 0.03
RW	0.72 ± 0.06	0.73 ± 0.05
FBW	34.50 ± 0.12	34.25 ± 0.27

There was no statistically significant difference between the AW & RW of internal organs and the FBW of animals. The p-values were not significant.

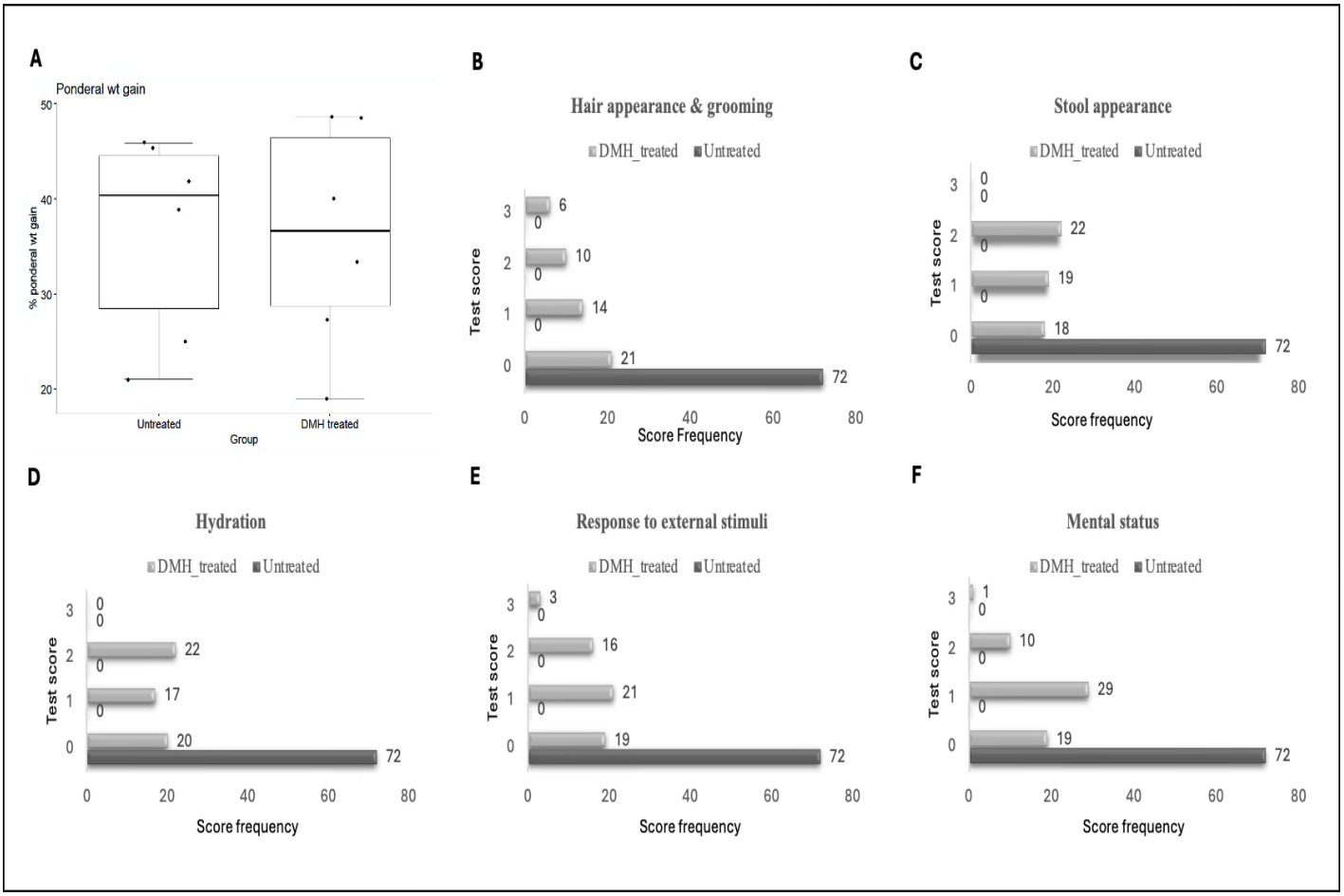

Figure 1: Weekly report of both the group animals for **A.** Ponderal weight gain, **B.** Hair appearance and grooming, **C.** Stool appearance, **D.** Hydration, **E.** Response to external stimuli, **F.** Mental status

Hematology

An independent t-test for the above study shows there is a significant difference between the hematological parameters – RBC, WBC, & Differential counts between the two groups. $p<0.05$ (Table 2).

Table 2: Hematological parameters compared between the groups (mean ± SE)

	RBC parameters			**WBC count**	**Differential count**					
	HB (g/ml)	**RBC count** (mill/cmm)	**PCV** (%)	**Total WBC count** (/cmm)	**Neutrophils** (%)	**Lymphocytes** (%)	**Eosinophils** (%)	**Monocytes** (%)	**Basophils** (%)	**Platelet count** (lac/cmm)
Untreated	14.5 ± 0.7	4.6 ± 0.2	42.9 ± 0.4	4796 ± 0.3	53 ± 0.6	42 ± 0.4	2 ± 0.4	0 ± 0.0	0 ± 0.0	2.6 ± 0.7
DMH-treated	**9.6 ± 0.6**[a]	6.6 ± 0.3	32.5 ± 0.7	**2988 ± 0.5**[b]	**9.3 ± 0.3**[c]	**84.3 ± 0.5**[d]	0 ± 0.0	0 ± 0.0	0 ± 0.0	**1.2 ± 0.3**[e]

[a] Statistically different from the Untreated group ($p<0.05$)
[b] Statistically different from the Untreated group ($p<0.05$)
[c] Statistically different from the Untreated group ($p<0.01$)
[d] Statistically different from the Untreated group ($p<0.05$)
[e] Statistically different from the Untreated group ($p<0.05$)

Renal & Liver function test

An independent t-test for the above study shows there is no significant difference in the Renal functioning of the two groups, p = not significant (Table 3). But, hepatic functioning test between the groups suggests a significant difference between the SGOT levels, with DMH-treated group animals having above normal range.

Table 3: Estimation of Renal and Hepatic Function in both groups

Group	Blood Urea Nitrogen (mg/dL)	Serum Creatinine (mg/dL)	SGOT (IU/L)	SGPT (IU/L)
Untreated	21.5 ± 0.6	0.18 ± 0.2	35.2 ± 0.4	58.2 ± 0.5
DMH treated	26.7 ± 0.4	0.13 ± 0.6	**132.8 ± 0.8**[a]	52.5 ± 0.7

[a] Statistically different from the Untreated group ($p<0.01$)

Gross analysis

Gross anatomy of the gastrointestinal tract of both the untreated and DMH-treated groups is shown in Figure 2. The Untreated groups' animal (Fig. 2a) showed a smooth and continuous small intestine of consistent diameter with pale-pinkish coloration. The large intestine, although smaller in length, is wider in diameter and appears normal.

There is a notable change in the morphology of the small and large intestine of the DMH-treated animals. Both the small and large intestine shows region of inflammation and lesions (Fig. 2b). Various macroscopic lesions or polyps, along with thickening of the intestinal wall, were observed in the colonic region of the DMH-treated animals (Fig. 2c).

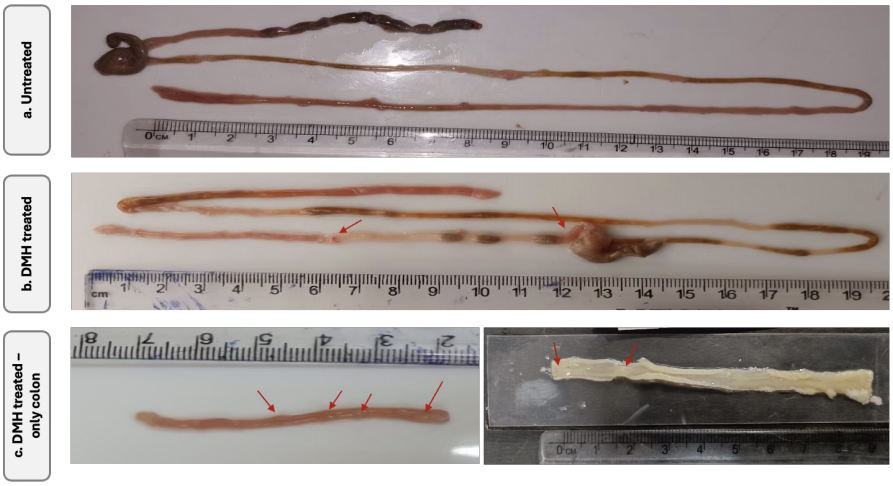

Figure 2: Gross analysis of small and large intestine of a. Untreated group b. DMH-treated group showed inflamed regions in the small intestine and colon region of the mice c. DMH-treated colon showed various polyps and wall thickening.

Histological analysis

The histological slides of H&E-stained slides of colon, liver and lungs of both the treated and untreated groups (Fig. 3). The untreated group's colon shows normal mucosal architecture with regular, parallel crypts lined with a high density of goblet cells. Mucosal lining is also looking well-organised in architecture with optimum thickening. While in the DMH-treated group animals' colon architecture is visibly distorted. There is evidence of mucosal thickening, hypercellularity, and a reduction in the number of mature goblet cells with signs of dysplasia and the presence of an aberrant crypt focus (ACF) or a dense cellular aggregate.

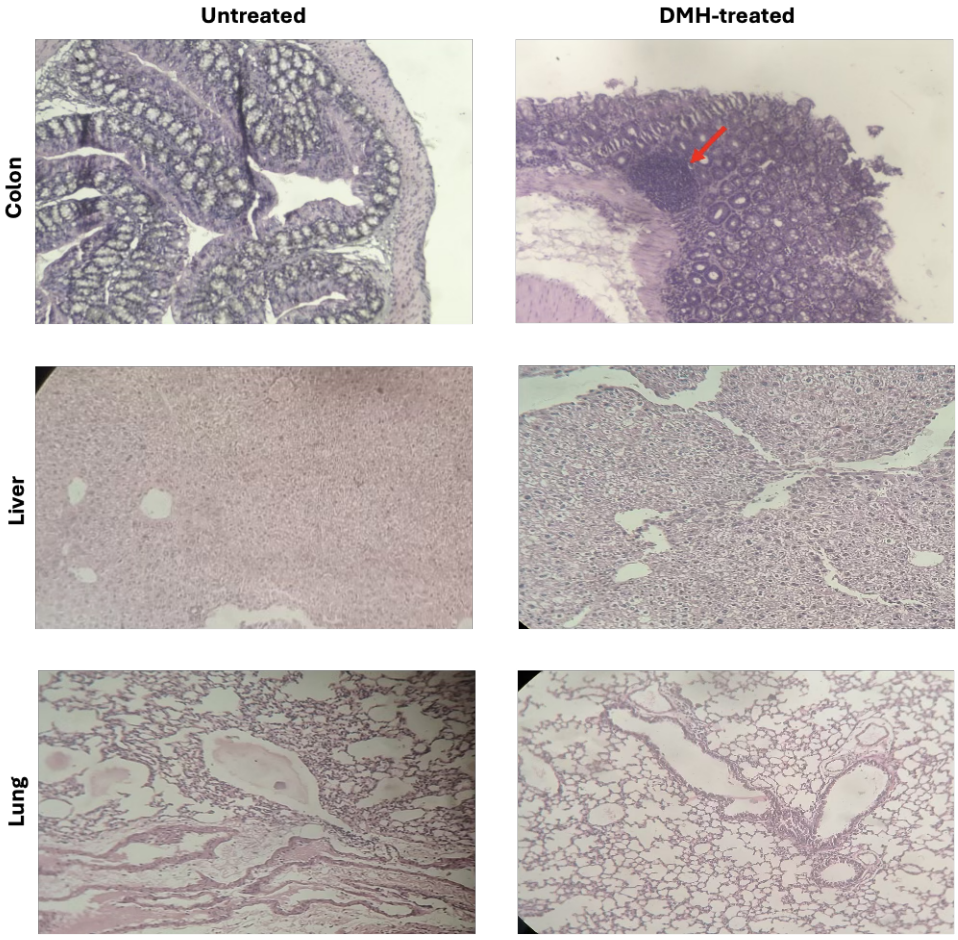

Figure 3: This figure illustrates the Hematoxylin and Eosin (H&E) stained histological slides of colon, liver, and lung of both the untreated group and DMH-treated group. A dense cellular aggregate or aberrant crypt focus (ACF) is visible

The microscopic slide of the liver of the untreated group of animals displays normal hepatic architecture with central veins radiating organized hepatocytes and clear sinusoidal spaces. Also, the nuclei were uniform and centrally located. But the DMH-treated animals' liver showed signs of hepatotoxicity, with increased cytoplasmic vacuolation, sinusoidal irregularity and dilatation, and there was a loss of the uniform radiating organization, suggesting possible metabolic stress and cellular injury.

On examination of the lungs' histological samples from the untreated and DMH-treated groups, the untreated group's histological slide shows intact bronchiolar structure with a normal epithelial lining. While the DMH-treated group showed mild alveolar septal thickening and increased inflammatory cell infiltrates in the interstitial spaces. Some alveolar sacs were also observed to be irregular as compared to the untreated group.

Survival Analysis

The KM survival curve, performed over 12 weeks period, demonstrates 100% survival probabilities for the untreated group, while the DMH-treated groups' survival probabilities notably decline after 6th weeks to 0.85,and then to 0.71 after 7th week, and remain up to 12 weeks.

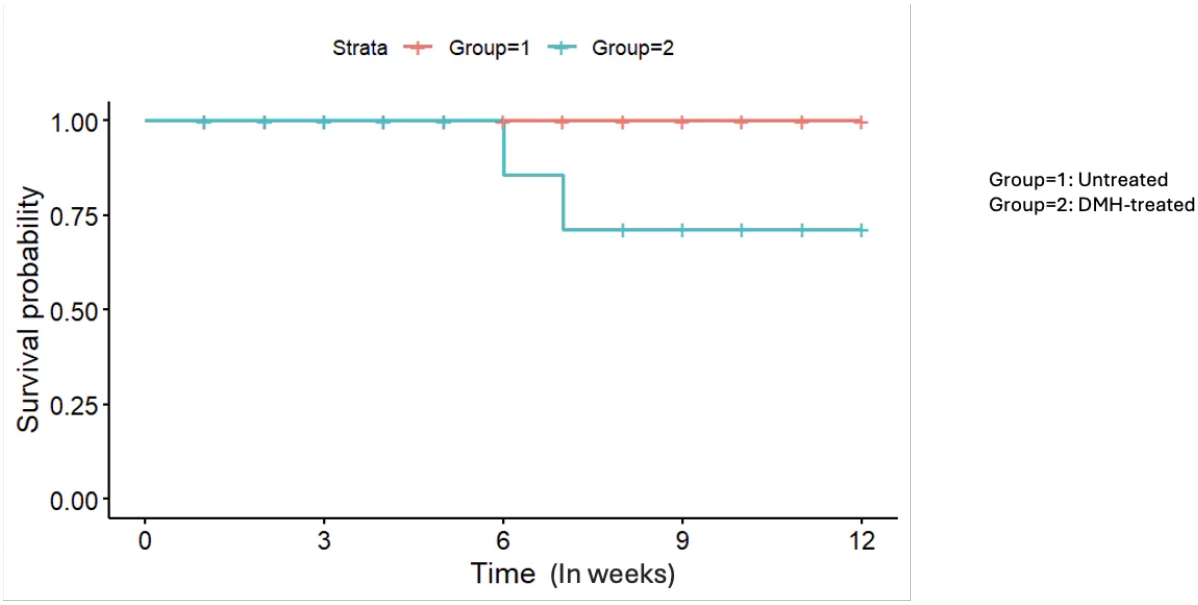

Figure 4: This Kaplan-Meier (KM) survival curve illustrates the survival probabilities of both group animals (Untreated and DMH-treated) over 12 weeks' period. Group 1 (Untreated) showed higher probabilities than group 2 (DMH-treated) and started to decline after the 6th week.

Discussion and Conclusion

DMH, which is considered an effective carcinogen for the induction of tumors of the colon and rectum in mice by i.p. injections, is an alkylating agent that needs liver metabolic activation to become a carcinogen. It has been shown to induce adenoma and carcinoma of both small and large intestine in rats, while showing remarkable specificity in mice to induce colon tumors (18). Our study shows that DMH induces CRC in Swiss albino mice with a 100 % incidence rate, along with characteristic colitis-like features and adenomatous polyps in the distal part of the colon, which have characteristics like sporadic cancer of humans and recapitulate the adenoma-carcinoma sequence. Notably, sporadic CRC is the most common form of CRC (approximately 95%), basically driven by mutation in tumor suppressor genes like APC and TP53 (18–20). Further, a study has used the DMH-induced mice model to study the biological changes of gut microbiota in an 'adenoma-carcinoma sequence", and suggests that microbiota dysbiosis and bacterial metabolites play synergistic actions on related molecular events and contribute to the progression of CRC in mice, similar to humans (21).

Upon analyzing the parameters, including average weight and internal organ weight, between the treatment groups, no significant difference was found. However, daily monitored parameters such as hair grooming, stool appearance, hydration level, and mental states began to differ, indicating signs of progressive disease.

Hematological parameters show there are significant changes in RBC, WBC, & Differential counts. Renal functioning test shows no significant difference between the two groups, however, on analyzing the serum biochemical marker, SGOT shows significant difference, indicative of hepatic inflammation and is also related to liver disorders, especially liver metastasis in CRC (22). However, DMH is also reported to cause hepatic damage (18).

In our study, the mouse model demonstrates mucosal thickening and a reduced number of mucus-producing goblet cells in the colon, which is also observed in human tumors. This decreases the proportion of sulphated mucopolysaccharides in the areas and promotes tumorogenesis (18). Dense cellular aggregates, indicative of ACF, were observed in almost all the DMH-treated animals (22,23).

One of the major weaknesses of this induction method is that a series of injections of DMH is required to induce colon tumors (weekly injection for 12 weeks). Our study has suggested that the Survival rate in this group is about 67 %. So, to achieve a statistically significant 'n', the required number of animals should be more than 8. Also, as adenocarcinoma is well developed after 12th weeks of the induction cycle, we can go for the treatment phase from the 13th week.

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CRediT authorship contribution statement

Anshu Thakur: Conceptualization, Methodology, Literature review, Data collection, Validation, Visualization, Formal analysis, Writing – original draft. **Mehul R. Chorawala:** Conceptualization, Methodology, Resources, Writing – review & editing, Visualization, Supervision, Project administration

Declaration of competing interest

The author declares that they have no known competing financial and/or non-financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Conflict of Interest

The author declares no conflict of interest.

Declaration for Use of Generative AI or AI-assisted technologies

All the authors declare that no generative AI or AI-assisted technologies have been utilized while drafting this manuscript.

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