

Lentils (*Lens culinaris*, L.) Attenuate Colonic Lesions and Neoplasms in Fischer 344 Rats

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Abstract

Objective: Lentils (*Lens culinaris*, L.) contain several bioactive compounds that have been linked to the prevention of cancer. However, the *in vivo* chemopreventive ability of lentils against chemically-induced colonic lesions has not been previously examined. Our present study examined the hypothesis that lentils could suppress neoplastic growth *in vivo* by virtue of their bioactive micro- and macro-constituents, and that culinary thermal treatment could affect the chemopreventive potential.

Methods: To accomplish this goal, raw whole lentils (RWL), raw split lentils (RSL), cooked whole lentils (CWL) and cooked split lentils (CSL) were used. Pluronic F-68 (PF68), which is a well-studied chemopreventive agent, was used also for the purpose of comparison. Sixty weanling Fisher 344 male rats, 4-5 weeks of age, were randomly assigned to six groups (10 rats/group): the control group (C) received AIN-93G diet; treatment lentil groups of RWL, CWL, RSL and CSL received the treatment diets containing AIN-93G+5% of lentils, while PF68 group received C+1% PF68 diet. After acclimatization for 1 week, all animals were put on the control and treatment diets separately for 5 weeks. At the end of the fifth week of feeding, all rats received two s.c. injections of azoxymethane (AOM) carcinogen at 15 mg/kg rat body weight/dose once a week for two consecutive weeks. After 17 weeks of the last AOM injection, all rats were euthanized.

Results: Total colonic lesions and neoplasms (mean \pm SEM) ranged from 6 to 8 for lentil groups, with a reduction value of 43 to 57% from the control (C) group (14 lesions). Incidence of severe dysplasia was reduced significantly ($P=0.0022$) in the colons of rats fed on lentils (0%-10%), except RWL (20%) when compared with the control (40%) whilst incidence of adenocarcinomas was reduced significantly ($P=0.0430$) in lentil groups (0-10%), except RSL (20%), when compared with the control (40%).

Conclusion: Our findings indicate that the consumption of lentils may attenuate colon carcinogenesis in animal models, and that hydrothermal treatment resulted in an improvement in the chemopreventive potential.

Keywords: Lentils [*lens culinaris*, L.], Colon Cancer, Fischer 344 Rats, Azoxymethane.

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Introduction

Colorectal cancer (CRC) is the fourth leading cause of cancer-related deaths throughout the world. More than 85% of persons with CRC have sporadic type; this emphasizes the role of environmental factors, including eating practices, on the development of CRC¹, which has been described as a diet-related cancer. Because advanced metastasized cancers are almost impossible to treat, prevention is looked at as the "front line in the complex battle against cancer".²

Of the Leguminosae family, lentils (*Lens culinaris*, L.) are considered nowadays among the most important legumes for human nutrition³. Lentils are commonly mixed with cereals or consumed as dehulled split lentil soup; both are the most customarily consumed forms of lentils in Mediterranean countries.

Lentil seeds are rich sources of protein (25.8%), carbohydrates (60.08%), soluble and insoluble dietary fibers (30.5%)⁴, as well as prebiotic oligosaccharides⁵ and resistant starches⁶. A cancer-preventive effect for the latter two functional components has been reported^{7, 8}, and good quantities of fermentable carbohydrates in lentils contribute to the bifidogenic effect of lentils in the gut⁹. Lentils have been shown to exhibit low glycemic index (GI) value in humans.¹⁰ The importance of low glycemic index foods arises from the fact that hyperglycemia and hyperinsulinemia, associated with eating foods of high glycemic load, have been shown to be correlated with an increased incidence of CRC.¹¹

Lentils are an excellent source of many water-soluble vitamins, particularly folate (479µg/100g).⁴ In addition, lentils have non-nutrient functional phytochemicals such as phytic acid and tannins¹², of the latter proanthocyanidins constitute a major class.¹³ Lentils are also good sources for the antioxidants, catechins and procyanidins.¹⁴ Glutathione, soluble proteins and tocopherols are second to polyphenolics in contributing to the total antioxidant capacity of lentils.¹⁵ As a consequence of their presence in lentils, a wide spectrum of phenolic and non-phenolic antioxidants have shown the highest

antioxidant and oxygen radical absorbing capacities among different legumes.¹⁶ Among the nutraceutical fractions of lentils are lentil proteins and bioactive peptides, which include lectins, "defensin" protein, and Bowman-Birk trypsin inhibitors (BBI). These substances aid in halting neoplasm growth and have gained a special attention for their inhibitory effect against CRC.¹⁷

Because lentils constitute variable types of potentially bioactive micro- and macro-constituents and chemopreventive agents, we hypothesized that lentils may suppress early colon carcinogenesis in an animal model. To examine this hypothesis, we designed a study to examine the chemopreventive effect of lentils against early colon carcinogenesis induced by azoxymethane (AOM) in Fischer 344 (F344) rat models by using histopathological examination of the colonic tissue. In our laboratory, pre-carcinous lesions called aberrant crypt foci (ACF), and glutathione *-S-* transferases activity were used as indicative surrogate biomarkers. Significant results were obtained confirming our hypothesis regarding the chemopreventive ability of lentils against early carcinogenesis.¹⁸

Due to the relatively long period (23 weeks) of the aforementioned study, which was longer than optimum period for evaluating ACF, clear dysplastic lesions and neoplasms were macroscopically noticed in the different colons of the treatment and control groups. This observation encouraged us to evaluate the effect of those different lentil treatments on the development of colonic lesions and neoplasms in experimental rats. Therefore, this continuum part was published separately in order to uncover the effect of lentils in preventing intermediate stages of colon carcinogenesis, namely colonic lesions and neoplasms that precede the frank malignant tumors.

Materials and Methods

Animals and Housing

As the current study is a continuum for our previously mentioned study, animals and housing, preparation of experimental diets, study

design and carcinogen administration, experiment termination and sample collection were as reported in the aforementioned study.¹⁸

Histopathological Examination

For neoplasm evaluation, the collected colons were dipped in 10% buffered formalin solution for 24 hours. Tissues showing a deviation from normal morphology and suspicious areas of neoplasms in the colons were excised, incubated sequentially in formalin, ethanol and wax by automatic rotary incubator (Citadel, 2000, ThermoShandon) for 16 hours, and then embedded in paraffin wax blocks (Lipshaw Cryo-Therm, USA). Serial sections of 5 µm thick were obtained (LEICA Rotary Microtome, RM 2125) and stained with standard hematoxylin (GCC, Germany) and eosin stains (BDH, UK) (H & E staining method). The stained sections were evaluated by a histopathologist. The pathologist examined slides of 180 sites of macroscopically abnormal colonic tissue. Specimens were assessed on the basis of histological abnormalities, grading and pattern of the neoplastic growth according to the criteria described by Kumar et al.¹⁹ If the lesion was neoplastic, it was divided into benign lesions called adenoma or malignant lesions called adenocarcinoma. Adenocarcinomas were distinguished by the invasion of the stroma of the stalk by neoplastic glands and by the invasion across the line of the muscularis mucosa. Either benign neoplastic lesions (adenomas) or malignant ones (adenocarcinomas) were scored, and the dysplastic lesions were graded according to the degree of dysplasia into mild or severe grades.

Statistical Analysis

All data of colonic lesions, including neoplasms and internal organ weights were analyzed by the one-way analysis of variance (ANOVA) (SAS, 1998, Version 7 for Windows analysis package).²⁰ Comparison of a single treatment group to the control group was analyzed for statistical significance. Means were separated using Fischer's-protected Least Significant Difference (PLSD) test, and differences were

considered significant at $P < 0.05$.

Results

Rat Body and Internal Organ Weights

Figure 1 shows both the pattern of growth and weight gain by different rat groups throughout the study period. It can be noticed from the growth curves of experimental animals that a rapid weight gain and growth occurred during the first five weeks of the experiment, the period prior to AOM injection. However, at the start of week 7 (next to the second AOM injection), growth rates of all rats injected with AOM declined substantially until they reached a steady state (plateau) and remained at that state until the end of the experiment (week 23). After the carcinogen administration, it can be noticed that animals in all of the treatment groups grew and gained body weights at faster rates as compared with those of the control (C) group.

As shown in Table 2, no significant differences were found in the initial body weights, final body weights, and body weight gains for the animals fed different lentils and PF68 diets throughout the 23-week study when compared with animals of the control diet (Table 2). Similarly, no significant differences were found in liver, spleen, and kidney weights for rats fed different treatments in comparison with the rats of the control diet.

Histopathology of Colonic Lesions for Experimental Animals

As shown in Table 3, the majority of rats in the Pluronic F-68 group showed no evidence of lesions or neoplasms (7/10; 70%), whereas half or less (1-5; 10-50%) of the animals in the legume-based diet groups showed such absence of lesions or neoplasms, whilst no animal in the control group showed such a result. It can be noticed also that rats fed the raw split lentils, followed by those fed the PF68, showed the lowest number of dysplastic lesions (2 and 3, respectively), whilst those rats fed the raw whole lentils showed the highest number of dysplastic lesions (8 lesions), with the latter even having a

higher number than the control diet group (5 lesions). Table 3 shows that in all of the experimental diet groups, the majority of neoplasms induced by exposure to AOM were adenocarcinomas (8/11), with only a few noninvasive adenomas being observed (3/11). Considerable colonic lesions [dysplasia, adenoma and adenocarcinoma] were observed more in the control diet (C) group than in the treatment diets (14 vs. 2-8), and the control group consistently had more instances of severe dysplasia (4 vs. 0-2) and adenocarcinomas (4 vs. 0-2). More dysplastic lesions were observed in the colons of rats fed the control diet as compared with the rats fed the treatment diets. From Table 3, it can be clearly shown that the development of neoplastic growths was reduced significantly ($P<0.05$) by the different treatment diets in comparison with the control (C) diet. Colon neoplasm incidence (percentage of animals that

had ≥ 1 neoplasm) was significantly ($P<0.05$) lower in rats fed the diets containing different forms of lentils, except the raw split lentils (C+5%RSL) when compared with the control (C) group. There were no significant differences in the incidence of colon adenomas between rats fed diets containing raw whole, cooked whole, cooked split lentils as well as PF68 diets on the one side, and rats fed the control (C) diet on the other side. Zero to thirty % of rats fed legume and non-legume diets, and 50% of rats fed the control diet had one or more neoplasms in the colons. Macroscopically, the majority of the colonic neoplasms were mainly located in the distal, and to less extent, in the middle colon, rather than the proximal one. Colonic neoplasms were macroscopically sessile or pedunculated. Histologically, the neoplastic lesions were adenoma or adenocarcinoma including signet-cell carcinoma.

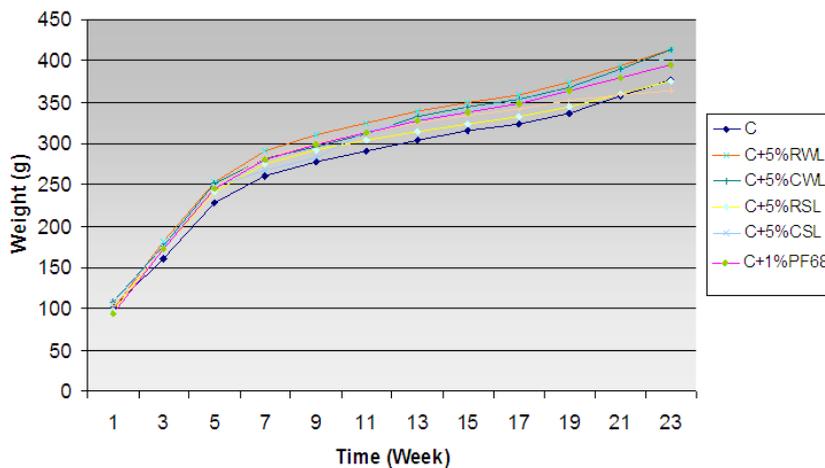


Figure (1): Body weight changes during the 23-week study period.

Table (1): Composition of experimental diets¹ (g component/kg diet).

<u>Ingredients</u>	<u>C</u>	<u>C+5%RWL</u>	<u>C+5%CWL</u>	<u>C+5%RSL</u>	<u>C+5%CSL</u>	<u>C+1%PF68</u>
Lentils**	0	52	50	52	50	0
Pluronic F-68 ²	0	0	0	0	0	10
Cornstarch	397.5	380.5	380.0	365.5	365.5	387.5
Sucrose	100	99.0	97.0	99.0	99.0	100.0
Fiber ³ (α -cellulose)	50	34.6	37.6	49.6	49.6	50
Soybean oil ⁴	70	69.4	69.4	69.5	69.5	70
Casein ⁵	200	184.0	183.5	183.9	183.9	200
Other common ingredients ⁶	182.5	182.5	182.5	182.5	182.5	182.5
Total	1000	1000	1000	1000	1000	1000

* Control diet is based on the American Institute of Nutrition (AIN) diet for experimental animals (Reeves, 1997): RWL, raw whole lentils; CWL, cooked whole lentils; RSL, raw split lentils; CSL, cooked split lentils; PF68, Pluronic F-68.

** Additional two grams (total of 52 g) of raw lentils were added to correct for differences in moisture content between raw and cooked legumes.

1. Dietary modifications for experimental diets of lentils were made according to United States Department of Agriculture Nutrient Database (SR21): RWL (25.8% protein, 1.06% total lipids, 60.08% total carbohydrates, 30.5% total dietary fibers, 2.03% total sugars); raw pink (split) lentils (24.95% protein, 2.17% total lipids, 59.15% total carbohydrates, 10.8% total dietary fibers) ⁵.
2. Pluronic F-68, a block-polymer similar to polyethylene-glycol from Sigma Chemical Co. (St. Louis, MO).
3. α -cellulose from Sigma Chemical Co. (St. Louis, MO).
4. Crude refined soybean oil without added vitamins.
5. Casein from Sigma Chemical Co. (St. Louis, MO). Assuming that casein is $\geq 85\%$ protein (200 g casein provides approximately 170 g protein).
6. Dextrinized cornstarch from Sigma Chemical Co. (St. Louis, MO), 132g; Mineral mixture (AIN-93G-Mix), 35g; Vitamin mixture (AIN-93G-Mix), 10g; DL-Methionine from Sigma Chemical Co. (St. Louis, MO), 3.0 gm; Choline bitartrate from Sigma Chemical Co. (St. Louis, MO), 2.5 g; tertiary butyl hydroquinone (TBHQ), 0.014g.

Table (2). Body and internal organ weights for experimental animals during the 23-week experimental period*.

Diet Group (n)	Initial body weight (g)	Final body weight (g)	Weight gain (g)	Liver weight (g)(% of body weight)	Spleen weight (g) (% of body weight)	Right kidney weight (g)(% of body weight)	Left kidney weight (g) (% of body weight)
C (10)	101.14±17.39 ^a	387.34±10.42 ^a	286.19±27.16 ^a	11.6 ±0.49 ^a (2.99%)	0.62 ± 0.02 ^a (0.16%)	1.10 ± 0.06 ^a (0.28%)	1.07 ± 0.05 ^a (0.28%)
C+5%RWL (10)	99.54±11.38 ^a	414.92±12.99 ^a	299.97±18.46 ^a	10.6 ± 0.50 ^a (2.56%)	0.61± 0.04 ^a (0.15%)	1.12 ± 0.06 ^a (0.27%)	1.12 ± 0.06 ^a (0.27%)
C+5%CWL (10)	107.98±9.32 ^a	413.55±19.05 ^a	288.82±24.91 ^a	9.45 ±0.67 ^a (2.27%)	0.58 ± 0.04 ^a (0.14%)	1.11 ± 0.06 ^a (0.27%)	1.08 ± 0.06 ^a (0.26%)
C+5%RSL (10)	97.81±7.98 ^a	375.71±14.14 ^a	275.72±13.94 ^a	9.46 ± 0.37 ^a (2.52%)	0.57 ± 0.02 ^a (0.15%)	1.13 ± 0.04 ^a (0.30%)	1.11 ± 0.03 ^a (0.30%)
C+5%CSL (9)	106.44±6.87 ^a	402.94±14.66 ^a	296.5±17.50 ^a	9.23 ± 0.37 ^a (2.29%)	0.60±0.04 ^a (0.15%)	1.01 ± 0.04 ^a (0.25%)	1.00 ± 0.03 ^a (0.25%)
C+1%PF68 (10)	94.42±5.68 ^a	386.14±12.62 ^a	311.29±10.53 ^a	8.83 ± 0.33 ^a (2.29%)	0.58 ± 0.04 ^a (0.15%)	1.10 ± 0.05 ^a (0.28%)	1.13 ± 0.06 ^a (0.29%)

Abbreviations: C, control AIN-93G diet; RWL, raw whole lentils; CWL, cooked whole lentils; RSL, raw split lentils; CSL, cooked split lentils; PF68, Pluronic F-68.

n = number of rats involved in the study group.

* Values are means ± SEM. Within a column, values without a common letter differ significantly at (P < 0.05) using Fischer's Protected Least Significant Differences Test (PLSD).

Table (3): Incidence and number of colonic lesions in Azoxymethane-treated rats fed control, lentils and Pluronic F-68 diets.

Diet Group (n)	Type of Colonic Lesion or neoplasm*				Neoplasm-bearing rats (%)
	Dysplasia		Number and type of tumor (%)*		
	Mild	Severe	Adenoma	Adenocarcinoma	
C (10)	5/10 ^a *	4/10 ^{a***}	1/10 ^a (10%)	4/10 ^{a****} (40%)	5/10 (50%)
C+5%RWL (10)	5/10 ^a	2/10 ^{ab}	0/10 ^a (0%)	1/10 ^b (10%)	1/10 (10%)
C+5%CWL (10)	6/10 ^a	0/10 ^b	0/10 ^a (0%)	1/10 ^b (10%)	1/10 (10%)
C+5%RSL (10)	4/10 ^a	0/10 ^b	1/10 ^a (10%)	2/10 ^{ab} (20%)	3/10 (30%)
C+5%CSL (9)	5/9 ^a	0/9 ^b	1/9 ^a (11%)	0/9 ^b (0%)	1/9 (11%)
C+1%PF68 (10)	2/10 ^a	1/10 ^b	0/10 ^a (0%)	0/10 ^b (0%)	0/10 (0%)

Abbreviations: C, control AIN-93G diet; RWL, raw whole lentils; CWL, cooked whole lentils; RSL, raw split lentils; CSL, cooked split lentils; PF68, Pluronic F-68.

n = number of rats involved in the study group.

Lesions include dysplasia, adenoma and adenocarcinoma, and neoplasms = adenoma + adenocarcinoma.

* *Values are means ± SEM. Within a column, values without a common letter differ significantly at (P< 0.05) using Fischer's Protected Least Significant Differences Test (FPLSD).*

** *Values are means ± SEM. Within a column, values without a common letter differ significantly at (P=0.0022) using Fischer's Protected Least Significant Differences Test (FPLSD).*

*** *Values are means ± SEM. Within a column, values without a common letter differ significantly at (P=0.0430) using Fischer's Protected Least Significant Differences Test (FPLSD).*

Discussion

Body and Internal Organ Weights of Experimental Animals

As depicted in Figure 1, lentil- and PF68-fed rats gained slightly better weight than those fed the control diet. As anticipated, and due to the lack of significant difference in food intake¹⁸, final body weights and gained weights of rats fed lentils and Pluronic F-68 diets were not significantly different from those of the control group (Table 2). Internal organs did not differ significantly in their weights between control and treatment groups. This could be ascribed to the fact that for internal organs to be affected by the carcinogenesis process, a longer time period (>30 weeks) is required to allow for tumors to metastasize, and thus to impinge on these organs.²¹

Histopathological Examination (neoplasm incidence and malignancy)

The results of this study demonstrate that lentils are capable of inhibiting AOM-induced colonic lesions in rats. Rats consuming lentils and PF68 diets had significantly ($P<0.05$) lower incidence of adenocarcinomas and severe dysplasia in the colon, except for raw lentils (split and whole) for the two types of lesions, respectively. The numbers of rats with neoplasms in the control group of the current study is 5 out of 10. This low number of neoplasms could be ascribed to the short duration of our current study period (17 weeks after last carcinogen injection), since neoplasms need considerable duration of time to allow for the accumulation of genetic abnormalities needed for full development of frank tumors.²¹ Total colonic lesions were also reduced by 82% (from 14 to 2) by PF68 to about 43% by RWL. The Pluronic F-68 diet had a striking inhibition for the colonic lesions and

neoplasms as indicated by the histopathological examination, a finding that goes in line with that of Parnaud et al²² (Table 3).

This inhibitory effect of lentils could be ascribed to their bioactive microconstituents. Lentils are tannin-rich leguminous seeds (915 mg/100g)¹² and are characterized by the presence of different tannin-related phenolic compounds such as phenolic acids, quercetin, kaempferol, delphinidin, and cyanidins.²³ Tannins and their related compounds are located mainly in the testa. This could explain the further cancer-preventive ability of whole lentil seeds over the split lentil in the current study. Different mechanisms have been proposed for the cancer preventive activity of phenolic acids, including inhibition of carcinogen uptake, inhibition of the formation or activation of carcinogen, deactivation or detoxification of carcinogen, prevention of the carcinogen binding to DNA, and enhancement of the level or fidelity of DNA repair.²³

The reduction in late colon cancer by feeding lentils could also be ascribed to the low glycemic index, which has been shown to lower both plasma insulin and glucose levels. It is proposed that high insulin levels promote colon carcinogenesis.¹¹

Legumes, including lentils, are significant sources of resistant starches⁶, which have been reported to decrease colonic pH, induce apoptosis of colorectal neoplasm producing cells, induce cancer-preventive enzymes, enhance cholic acid secretion and influence colonocyte metabolism.⁷ In addition, lentils contain a considerably high amount of pivotal folic acid, which is expected to be involved in the cancer-preventive ability of lentils. Different molecular and epigenetic effects have been proven for folate in its chemopreventive effect.²⁴

The difference in a lesion-inhibitory effect between whole and split lentils may be ascribed principally to the difference in the polyphenolic content and fibers, which are aggregated mainly in the seed coats of whole lentils.¹³ However, the evidenced inhibitory effect of split lentils indicates that antioxidant polyphenolics of lentil seed coats are not the only responsible factors for the inhibitory effect against early carcinogenesis.

One of the proposed mechanisms for which the cancer preventive ability could be ascribed to is the induction of the xenobiotic detoxifying enzymes. It was found in our laboratories¹⁸ that lentils significantly triggered the induction of the hepatic glutathione-S-transferases activity, which was increased in the livers of rats fed lentil diets when compared with those fed a control diet.

Cooking was found to impose a better inhibitory effect against neoplasm formation than in raw lentils. This is consistent with our finding that cooking whole lentils improved the chemopreventive ability and exhibited further prevention against ACF, particularly the large multicrypt ones that may develop in malignant lesions.¹⁸ The desirable effect of cooking could be explained by the following three factors: (1) phenolics can still be present in cooked seeds in a level comparable to that of raw lentils; (2) a considerable amount of total phenolic compounds might be bound to insoluble fractions of other compounds formed during the cooking of seeds; and (3) the lipophilic phenolic compounds, which may be bound to cell walls, are released during cooking.¹⁵

It is concluded from the current study that lentils, along with Pluronic F-68, are capable of significantly [$P < 0.05$] inhibiting AOM-induced colonic lesions in F344 rats, and they could suppress progression of preneoplasia (dysplastic lesions and adenomas) to malignant neoplasia (adenocarcinomas). Further research is required to elucidate the genetic and molecular mechanisms underlying such an inhibitory effect. Furthermore, longer duration for the experiment (30 weeks) could be more helpful in exhibiting the cancer-protective effect of lentils against

frank malignant tumors.

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العدس في التخفيف من آفات القولون والأورام لدى الفئران من نوع الفيشر 344

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1- كلية الطب، الجامعة الأردنية، عمان، الأردن؛ 2- كلية الزراعة، الجامعة الأردنية، عمان، الأردن؛ 3- كلية الصيدلة والعلوم الطبية، جامعة البتراء، عمان، الأردن

الملخص

هدف الدراسة: يحتوي العدس (*Lens culinaris, L.*) على العديد من المركبات الحيوية التي أثبتت الدراسات العلمية قدرتها على منع السرطان. وعلى الرغم من إثبات هذا التأثير الوقائي لتلك المركبات النباتية منفردة، إلا أن تأثير العدس كغذاء في منع السرطان لم تتم دراسته من قبل. لذا فقد تم تصميم هذه الدراسة بغرض اختبار الفرضية القائلة بقدرة العدس على منع التسرطن عن طريق استخدام نموذج سرطان القولون المحرض بالمسرطن الكيماوي في الحيوانات. كما هدفت الدراسة إلى معرفة أثر المعاملات الحرارية المطبقة على العدس أثناء عمليات التحضير على تلك القدرة المانعة للتسرطن.

طرق البحث: تم في هذه الدراسة استعمال أربعة أشكال من العدس وهي: العدس الكامل النيء والعدس الكامل والمطبوخ، والعدس المقشور النيء والعدس المقشور المطبوخ. ولأغراض المقارنة، استعملت المادة Pluronic F-68 (PF68) وهي مادة كيماوية أثبتت الدراسات مقدرتها على منع الإصابة بالسرطان. تم توزيع 60 جرذا ذكرا فطيما من نوع Fischer 344 بعمر 4-5 أسابيع على ست مجموعات بمعدل 10 جرذان لكل مجموعة. كانت مجموعات التجارب على النحو التالي: مجموعة المراقبة (C) واحتوت على الوجبة المعيارية AIN93-G، ومجموعات العدس الكامل المطبوخ والنيء والعدس المقشور المطبوخ والنيء، وقد أضيفت هذه الوجبات إلى الوجبة العيارية بنسبة 5%، في حين احتوت السادسة على الوجبة المعيارية مضافاً إليها المادة الكيماوية PF68 بنسبة 1%. بعد أقلمة الجرذان لمدة أسبوع في غرفة التجارب، تم إعطاؤها الوجبات الست لمدة خمسة أسابيع، ثم بعدها حقن الجرذان بجرعتين من المسرطن الكيماوي (AOM) تحت الجلد بجرعة مقدارها 15 ملغم/كغم من وزن الجسم بينهما أسبوع واحد. بعد مرور 17 أسبوعاً من الحقنة الثانية، تم قتل الحيوانات وأخذ القولون لتحديد عدد النموات السرطانية والبقع المرضية فيه.

النتائج: تراوح عدد الأورام السرطانية الحميدة والخبيثة والبقع المرضية (المتوسط ± الخطأ المعياري للمتوسط) بين 6-8 لمجموعات العدس الأريع، وكان الانخفاض عن مجموعة المراقبة التي احتوت 14 بقعة مرضية بمعدل 43-57%. انخفض عدد الاختلالات التنسجية المفرطة بشكل معنوي ($P=0.0022$) في قولونات الجرذان التي أعطيت وجبات العدس (معدل 0-10%)، باستثناء مجموعة العدس الكامل النيء، بالمقارنة مع مجموعة المراقبة (40%). في حين كان معدل حصول النموات السرطانية الخبيثة منخفضاً انخفاضاً معنوياً ($P=0.0430$) في مجموعات العدس (0-10%)، باستثناء مجموعة العدس المقشور النيء، بالمقارنة مع مجموعة المراقبة (40%).

الاستنتاجات: خلصت الدراسة إلى أن تناول العدس يسهم في الحد من عملية التسرطن في قولونات الجرذان، وأن المعاملة الحرارية الرطبة (الطبخ) أدت إلى تحسين القدرة المانعة للتسرطن للعدس.

الكلمات الدالة: سرطان القولون، العدس، جرذان فيشر 344، الأزروكسيميثان.