Effect of *Cocos nucifera* and red chilli on intestinal β-glucuronidase and mucinase activity in experimental colon cancer

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**Introduction**

Dietary intervention can protect humans from a variety of diseases and has led to the formulation of a number of explanatory hypotheses, several of which involve the bacteria of the lower intestine\(^2\). These bacteria are capable of a wide variety of metabolic activities including production of toxic metabolites, transformation of bile acids, amines, and hydrolysis of drugs that may positively act as carcinogens and/or co-carcinogens.

β-glucuronidase and mucinase are two important enzymes which reflect the activity of these bacteria. Mucinase is the enzyme which hydrolyses the protective mucin and β-glucuronidase hydrolyses biliary glucuronides. If glucuronide hydrolysis is a rate-limiting step in this process, then the levels of microbial β-glucuronidase in the colon may influence the risk of colon cancer.

Among the spices, red-chilli is consumed in large quantities in different parts of India\(^3\). Coconut kernel is also an important constituent of Indian food. In vitro studies have shown that red chilli and its irritative phenolic compound, capsicum, known to have an established structure of N-(4-hydroxy-3-methoxy benzyl)-3-benzoyl trans-cinnamaldehyde to be a mutagenic, carcinogenic and tumour-promoting agent\(^4\). Since *Cocos nucifera* forms an important constituent of Indian food, we have studied the effect of coconut kernel on 1, 2-dimethylhydrazine (DMH) induced colon carcinogenesis and also its effect, in the presence of red chilli.

**Materials and methods**

Wistar male albino rats bred in the Animal House of Rajah Muthiah Medical College, Annamalai University, weighing 120-150 g were divided into 7 groups of 10 rats each. They were all fed a commercial diet (Upjohn Limited) containing 20% peanut oil. Water was given ad libum.

Group 1 were control rats, group 2 were rats fed fresh coconut kernel (30%), group 3 were rats administered DMH, group 4 were rats fed red chilli powder (8mg/day/100g body weight in water), group 5 chilli + DMH, group 6 fresh coconut kernel + red chilli, group 7 fresh coconut kernel + chilli + DMH.

The fat intake by the animals in groups 2, 6 and 7 was adjusted so that it was similar to the fat intake in groups 3, 4 and 5. The caloric intake of animals in groups 3, 4, and 5 were also similar to that of 2, 6, and 7. p-nitrophenyl β-D-glucuronide, mucin, and 1,2-dimethylhydrazine were purchased from Sigma Chemical Co., St. Louis, MO, USA. All the other chemicals used were of analytical grade and were purchased from SD Fine Chemicals, Bombay, India.

DMH was administered as reported earlier\(^3\). After 15 weeks, the DMH injection was discontinued and the rats were given only commercial diet. The animals were observed daily and weighed every week. At the end of 30 weeks, fresh faecal pellets were collected and the activity of mucinase was estimated by the method of Shab and Chang\(^4\). The rats were then sacrificed and the neoplasms in the intestine and colon were counted after cutting open the tissues taken care not to disturb the tumours. Part of the tissues were sent for histopathological examination. The rest of the tissues and colon contents (bacterial contents) were transferred to ice cold containers, for measuring the activity of β-glucuronidase\(^5\). Protein was estimated by the method of Lowry et al\(^3\).

Results obtained are expressed as mean ± SE from 6 rats in each group. The statistical significance of difference in means was analysed by Student’s t-test. A one way analysis of variance (ANOVA) was also determined\(^6\).

**Results**

Table 1 shows the incidence of colon and intestinal tumours in all the 7 groups. The values expressed are the sum of about 30 surviving rats from different experiments. The incidence and number of tumours decreased when coconut kernel was supplemented in the diet.

The macroscopic and light microscopic observations (histopathological) of the colon of rats in different groups are given in Table 2. The table shows that when kernel was supplemented in the diet the animals showed fewer papillae, lesser infiltration into the submucosa and less changes in the cytoplasm with decreased mitotic figures.

**Table 1. Incidence of colon and intestinal tumours.**

<table>
<thead>
<tr>
<th>Rat group</th>
<th>Rats with tumours/total rats</th>
<th>Incidence of colon tumours (diameter 3 mm)</th>
<th>Tumours in colon</th>
<th>Tumours in intestine</th>
<th>Tumours in tumour-bearing rat</th>
<th>Tumours in tumour-bearing rat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1 Control</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>Group 2 Kernel</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>Group 3 DMH</td>
<td>27/30</td>
<td>90.0</td>
<td>26</td>
<td>12</td>
<td>2</td>
<td>12</td>
</tr>
<tr>
<td>Group 4 Chilli</td>
<td>25/30</td>
<td>83.3</td>
<td>17</td>
<td>8</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>Group 5 Chilli + DMH</td>
<td>28/30</td>
<td>93.3</td>
<td>34</td>
<td>15</td>
<td>2</td>
<td>15</td>
</tr>
<tr>
<td>DMH</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 6 Kernel + chilli</td>
<td>6/30</td>
<td>20.0</td>
<td>2</td>
<td>Nil</td>
<td>2</td>
<td>Nil</td>
</tr>
<tr>
<td>Group 7 Kernel + chilli + DMH</td>
<td>22/30</td>
<td>73.3</td>
<td>12</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
</tbody>
</table>

**Figure 1** gives the average growth rate of the animals in the various groups. It was observed that the weight gained by the control group > kernel group > chilli + DMH > kernel + chilli > DMH > chilli + DMH, even though the average food intake by the animals of the various groups were more or less similar. The energy intake was the same in all the groups. Figure 2 shows the intestine and colon of rat. β-glucuronidase activity showed a significant increase in the DMH, chilli and chilli + DMH groups when compared with the control rats (Table 2).

**Figure 2. Histopathological changes in the colon.**

Macroscope

- Size
- Margin
- Nature

Microscopy

1. Transitional zone with foci of dysplasia
2. Inflammatory cell infiltrate into the mucosa
3. Lymphoid aggregates in the submucosa

4. Papillary pattern
5. Mucin secretion
6. Inflammation in the submucosa

- Large number of papillae
- Few glands dilated, filled with mucin
- Observed

- Not observed
- Not observed
- Observed

- Well defined
- Pedunculated
- Pedunculated

- Defined
- Pedunculated
- Pedunculated

- Mixed population
- Mixed population
- Mixed population

- Focal areas of dysplasia
- Mixed population
- Mixed population

- Mixed population
- Mixed population
- Mixed population

- Occasional lymphoid aggregate
- Occasional lymphoid aggregate
- Occasional lymphoid aggregate

- Small glands filled with secretion
- Small glands filled with secretion
- Small glands filled with secretion

- Present
- Present
- Present

- Occasional focus of infiltration
- Occasional focus of infiltration
- Occasional focus of infiltration

7. Cell morphology

i) Nuclear pleomorphism
ii) Nucleoli
iii) Cytoplasm
iv) Mitotic figures

- Marked
- Moderate
- Numerous

- Prominent
- Less severe
- Scanty

- Less severe
- Moderate
- Numerous

- Prominent
- Moderate
- Present

- Present
- Present
- Present

In the chilli treated animals the β-glucuronidase levels increased significantly in the colon, intestines and liver, and also in the colon contents (bacterial) when compared with kernel + chilli. Similarly, when the chilli + DMH group was compared with the kernel + chilli + DMH group, the β-glucuronidase level was found increased significantly.

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\(^4\) N Nalini, S Chitra, K Sabitha, P Viswanathan, V Menon

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Effect of *Cocos nucifera* and red chilli on an intestinal β-glucuronidase and mucinase mucinase activity, was studied in rats given 1, 2-dimethylhydrazine (DMH). The average weight gain by the animals given coconut kernel was more than the DMH and chilli treated groups. The activity of β-glucuronidase decreased in the kernel groups, in most of the tissues studied, as compared to the DMH and chilli treated groups. A similar pattern was observed in the case of mucinase. Morphological studies showed that the number of visible malignant tumours decreased in the colon and intestine of the animals, when their diet was supplemented with coconut kernel. Histopathological studies also showed that the animals had fewer papillae, lesser infiltration into the submucosa and lesser changes in the cytology with decreased mitotic figures, when kernel was included in the diet. Coconut kernel, thus reduced the mutagenic and carcinogenic effect of chilli and DMH respectively.

Introduction
Dietary intervention can protect humans from a variety of diseases and has led to the formulation of a number of explanatory hypotheses, several of which involve the bacteria of the lower intestine. These bacteria are capable of a wide variety of metabolic activities including production of toxic metabolites, transformation of bile acids, nitrogen and hydrolysis of drugs that may positively act as carcinogens and/or co-carcinogens. β-glucuronidase and mucinase are two important enzymes which reflect the activity of these bacteria. Mucinase is the enzyme which hydrolyzes the protective mucins and β-glucuronidase hydrolyses bile glucuronides. If bile glucuronidase is a rate-limiting step in this process, then the levels of microbial β-glucuronidase in the colon may influence the risk of colon cancer.

Among the spices, red-chilli is consumed in large quantities in different parts of India. Coconut kernel is also an important constituent of Indian food. In vivo studies have shown that red chilli and its irritative phebalance compound, capsaicin, known to have an established structure of N-(4-hydroxy-3-methoxy benzyl)-N-benzoyl trans-6-enamide, be a mutagenic, carcinogenic and tumour promoting agent. Since *Cocos nucifera* forms an important constituent of Indian food, we have studied the effect of coconut kernel on 1, 2-dimethylhydrazine (DMH) induced colon carcinomas and also its effect, in the presence of red chilli.

Materials and methods
Wistar male albino rats bred in the Animal House of Rajah Muthiah Medical College, Annamalai University, weighing 120-150 g were divided into 7 groups of 10 rats each. They were all fed a commercial diet (Upjohn Limited) containing 20% peanut oil. Water was given ad libitum.

Group 1 were control rats, group 2 were rats fed fresh coconut kernel (30%), group 3 were rats administered DMH, group 4 were rats fed red chilli powder (8mg/day/100g body weight in water), group 5 chilli + DMH, group 6 fresh coconut kernel + red chilli, group 7 fresh coconut kernel + chilli + DMH.

The fat intake by the animals in groups 2, 6 and 7 were adjusted, so that it was similar to the fat intake in groups 3, 4 and 5. The caloric intake of animals in groups 3, 4 and 5 were also similar to that of 2, 6 and 7. p-nitrophenol β-glucuronidase, mucin and 1,2-dimethylhydrazine were purchased from Sigma Chemical Co., St. Louis, MO, USA. All the other chemicals used were of analytical grade and were purchased from SD Fine Chemicals, Bombay, India.

DMH was administered as reported earlier. After 15 weeks, the DMH injection was discontinued and the rats were given only commercial diet. The animals were observed daily and weighed every week. At the end of 30 weeks, fresh fecal pellets were collected and the activity of mucinase was estimated by the method of Bai and Chang. The rats were then sacrificed and the neoplasms in the intestine and colon were counted after cutting open the tissues longitudinally taking care not to disturb the tumours. Parts of the tissues were sent for histopathological examination. The rest of the tissues and colon contents (bacterial contents) were transferred to ice cold containers, for measuring the activity of β-glucuronidase. Protein was estimated by the method of Lowry et al.

Results
Table 1 shows the incidence of colon and intestinal tumours in all the 7 groups. The values expressed are the sum of about 30 surviving rats from different experiments. The incidence and

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Table 1. Incidence of colon and intestinal tumours.

<table>
<thead>
<tr>
<th>Rat group</th>
<th>Rats with tumoral rats</th>
<th>Incidence of colon tumours (%)</th>
<th>Tumours in colon/ intestine/ tumour-bearing rat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1 Control</td>
<td>Nil</td>
<td></td>
<td>Nil</td>
</tr>
<tr>
<td>Group 2 Kernel</td>
<td>Nil</td>
<td></td>
<td>Nil</td>
</tr>
<tr>
<td>Group 3 DMH</td>
<td>27/30</td>
<td>90.0</td>
<td>26</td>
</tr>
<tr>
<td>Group 4 Chilli</td>
<td>25/30</td>
<td>83.3</td>
<td>17</td>
</tr>
<tr>
<td>Group 5 Chilli + 28/30</td>
<td>93.3</td>
<td>34</td>
<td>15</td>
</tr>
<tr>
<td>DMH</td>
<td>6300</td>
<td>20.0</td>
<td>2</td>
</tr>
<tr>
<td>Group 7 Kernel + 22/30</td>
<td>73.3</td>
<td>12</td>
<td>3</td>
</tr>
</tbody>
</table>

Figure 1 gives the average growth rate of the animals in the various groups. It was observed that the weight gain by the control group > kernel group > chilli + DMH group > chilli + DMH > chilli > DMH, even though the average food intake by the animals of the various groups were more or less similar. The energy intake was the same in all the groups. Figure 2 shows the intestine and colon of rat. β-glucuronidase activity showed a significant increase in the DMH, chilli and chilli + DMH groups when compared with the control rats (Table 2).

In the chilli treated animals the β-glucuronidase levels increased significantly in the colon, intestines and liver, and also in the colon contents (bacterial) when compared with kernel + chilli. Similarly, when the chilli + DMH group was compared with the kernel + chilli + DMH group, the β-glucuronidase level

Table 2. Histological changes in the colon.

<table>
<thead>
<tr>
<th>Group 2</th>
<th>Group 3</th>
<th>Group 4</th>
<th>Group 5</th>
<th>Group 6</th>
<th>Group 7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Macroscopy</td>
<td>1. Size</td>
<td>-</td>
<td>2 cm</td>
<td>1 cm</td>
<td>2 cm</td>
</tr>
<tr>
<td></td>
<td>2. Margin</td>
<td>-</td>
<td>Pediunculated</td>
<td>Defined</td>
<td>Pediunculated</td>
</tr>
<tr>
<td>Microscopy</td>
<td>1. Transitional zone with foci of dysplasia</td>
<td>-</td>
<td>Not present</td>
<td>Focal areas of dysplasia</td>
<td>Mixed</td>
</tr>
<tr>
<td></td>
<td>2. Inflammatory cell infiltrate into the mucosa</td>
<td>-</td>
<td>Mixed</td>
<td>Mixed</td>
<td>Mixed</td>
</tr>
<tr>
<td></td>
<td>3. Lymphoid aggregations in the submucosa</td>
<td>-</td>
<td>Not observed</td>
<td>Not observed</td>
<td>Not observed</td>
</tr>
<tr>
<td></td>
<td>4. Papillary pattern</td>
<td>-</td>
<td>Large number of papillae</td>
<td>Few papillae</td>
<td>Papillae</td>
</tr>
<tr>
<td></td>
<td>5. Mucin secretion</td>
<td>-</td>
<td>Few glands</td>
<td>-</td>
<td>Filled with mucin</td>
</tr>
<tr>
<td></td>
<td>6. Infiltration in the submucosa</td>
<td>-</td>
<td>Not observed</td>
<td>-</td>
<td>Not observed</td>
</tr>
</tbody>
</table>

7. Cell morphology
- Nuclear pleomorphism
- Nucleoli
- Cytoplasm
- Mitotic figures

8. Other parameters
- Vascular granulation

In conclusion, the treatment with *Cocos nucifera* and chilli significantly decreased the incidence of colon and intestinal tumours, and also the histopathological changes in the colon.
Table 3. β-glucuronidase activity (m of p-nitrophenyl β-glucuronide/min/mg protein).

<table>
<thead>
<tr>
<th>Group</th>
<th>Group2</th>
<th>Group3</th>
<th>Group4</th>
<th>Group5</th>
<th>Group6</th>
<th>Group7</th>
<th>F-ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distant</td>
<td>DS-IB</td>
<td>DS-IB</td>
<td>DS-IB</td>
<td>DS-IB</td>
<td>DS-IB</td>
<td>DS-IB</td>
<td>NS</td>
</tr>
<tr>
<td>Proximal</td>
<td>b</td>
<td>b</td>
<td>b</td>
<td>b</td>
<td>b</td>
<td>b</td>
<td>NS</td>
</tr>
<tr>
<td>Distal intestine</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>NS</td>
</tr>
<tr>
<td>Proximal intestine</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>NS</td>
</tr>
<tr>
<td>Liver</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are mean ± SE from 6 rats in each group. Group 1 has been compared with groups 2-7. 

p < 0.01: b < 0.05: NS: Not significant. *ANOVA: Significant at 1% level.

Table 4. Mucinase activity (in moles of glucose liberated/min/mg protein).

<table>
<thead>
<tr>
<th>Group</th>
<th>Group2</th>
<th>Group3</th>
<th>Group4</th>
<th>Group5</th>
<th>Group6</th>
<th>Group7</th>
<th>F-ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Colon</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>NS</td>
</tr>
<tr>
<td>Faecal</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>a</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are mean ± SE from 6 rats in each group. Group 1 has been compared with groups 2-7. 

p < 0.01: b < 0.05: NS: Not significant. *ANOVA: Significant at 1% level.

felled significantly in the latter group in the distal colon, intestines, liver and also in the colon contents (bacteria).

A similar pattern was noted in the case of mucinase (Table 3). The chilii group showed a significant increase when compared with the kernel group, and kernel + chilli group.

The kernel + chilli + DMH group showed a significant fall in the activity of mucinase both in the colon contents as well as in the faecal contents compared with chilli + DMH group.

The F-value showed that there was a significant difference between and the within the groups at 1% level in all the parameters studied.

Discussion

Treatment with red chilli, DMH and coconut kernel brings about profound alterations in the activity of both β-glucuronidase and mucinase. Chillies treated rats and those given DMH showed increased incidence of tumours both in the colon and intestine. When coconut kernel was included in the diet, the incidence of tumours decreased, the size of the tumours (visible) significantly reduced and were more or less diffused.

Histopathological studies showed a great degree of variation in the different groups. In the untreated control animals there was vascularisation in the colon, but the colon was otherwise normal. The chilli group showed the size of the tumours to be about 1 cm, pedunculated with defined margin. It showed areas of dysplasia which were less severe, the nuclei were less prominent, moderate cytoplasm and with mitotic figures. In the DMH treated group, the size of the tumour was around 2 cm, pedunculated, with well-defined margins, with large number of papillae and an invasive adenocarcinoma, which showed marked pleomorphism. The nuclei were also very prominent, with scanty cytoplasm and numerous mitotic figures. In the chilli + DMH group, the size of the tumour was more than 2 cm. There was a transitional zone with areas of marked dysplasia and infiltrating adenocarcinoma. The nuclei was also prominent. In the kernel + chilli group, the size of the tumour was less than 0.5 cm, with ill defined margin, sessile with occasional dysplasia. Nuclear pleomorphism was less severe, nuclei less prominent with moderate cytoplasm. In kernel + chilli + DMH group, the size of the tumours was 1 cm, sessile with ill defined margin, had a significant number of papillae with few glands filled with mucin and showed occasional infiltration into the submucosa. Nuclear pleomorphism was prominent with marked nuclear scanty, cytoplasm and vascular granulation. The vascular granulation observed in the kernel group may be a protective mechanism, by which the animal tries to resist the invasion of the tumour into the deeper layer.

Glucosylose formation is a major detoxification mechanism in mammals. Many exogenous compounds that are excreted in the bile as glucuronosyl conjugates are deconjugated by bacterial β-glucuronidase and modified further by intestinal bacteria in the large bowel [23]. The activity of this microflora is affected by diet, and they are influenced by the biological activity, toxicity, exercise and reabsorption of many of the exogenous and endogenous compounds which are considered as carcinogens and/or co-carcinogens metabolites. Studies have shown that β-glucuronidase is a key enzyme in the activation of DMH metabolites to carcinogens. These substances can trigger the formation of neoplastic changes in the colon and intestine. The composition of DMH metabolites; fibres 35%, protein 3.6%, fat 38.1%, digestible carbohydrates 9.1% and the rest moisture. The inclusion of coconut kernel in our study significantly decreased the activity of this enzyme in the presence of chilli or DMH, or both, emphasising the protective role of the kernel.

Mucins are glycoproteins consisting of a large number of carbohydrate side chains attached to a protein core. They serve as a source of energy for the intestinal bacteria and are consequently degraded by them [24]. Supplying the microflora with fermentable dietary fibre (i.e., coconut kernel) may permit them to use these substrates preferentially. Thus, the treatment with coconut kernel showed a decrease in the activity of mucinase, while chilli and DMH treatment showed an increase.

Acknowledgment

The authors wish to thank Mr Arumug Perumal and Mr Krishnaswamy, for their technical assistance.

Effect of Cocos nucifera and red chilli on intestinal β-glucuronidase and mucinase activity in experimental colon cancer

The authors found that Cocos nucifera and red chilli significantly decreased the β-glucuronidase and mucinase activity in experimental colon cancer. The mechanism of action involves the inhibition of the activity of these enzymes, leading to a reduction in the formation of neoplastic changes in the colon and intestine.

References


Table 3. B-gluconoridase and B-mucinc activity

<table>
<thead>
<tr>
<th>Group</th>
<th>Group2</th>
<th>Group3</th>
<th>Group4</th>
<th>Group5</th>
<th>Group6</th>
<th>Group7</th>
<th>F-ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distal colon</td>
<td>55.84±6.916</td>
<td>40.37±5.682</td>
<td>108.74±9.275</td>
<td>44.76±4.350</td>
<td>83.40±14.550</td>
<td>42.15±1.101</td>
<td>61.60±2.184</td>
</tr>
<tr>
<td>Proximal colon</td>
<td>47.25±3.783</td>
<td>30.30±2.484</td>
<td>52.46±7.370</td>
<td>50.95±5.449</td>
<td>56.91±7.269</td>
<td>47.95±2.020</td>
<td>59.30±3.301</td>
</tr>
<tr>
<td>Distal intestine</td>
<td>53.66±6.245</td>
<td>42.01±3.537</td>
<td>54.26±5.051</td>
<td>85.76±7.121</td>
<td>80.88±8.648</td>
<td>47.75±3.416</td>
<td>61.52±5.729</td>
</tr>
<tr>
<td>Proximal intestine</td>
<td>53.96±7.638</td>
<td>39.66±5.755</td>
<td>51.76±7.800</td>
<td>59.73±12.591</td>
<td>65.43±9.999</td>
<td>48.83±2.626</td>
<td>51.83±2.847</td>
</tr>
<tr>
<td>Liver</td>
<td>19.95±8.268</td>
<td>85.60±12.371</td>
<td>145.12±12.981</td>
<td>96.08±12.595</td>
<td>171.63±2.970</td>
<td>91.02±1.256</td>
<td>119.43±4.525</td>
</tr>
<tr>
<td>Colon contents</td>
<td>102.1±6.238</td>
<td>71.93±8.093</td>
<td>112.13±13.732</td>
<td>149.00±24.390</td>
<td>173.90±3.530</td>
<td>121.40±4.800</td>
<td>160.20±4.640</td>
</tr>
</tbody>
</table>

Values are mean ± S.E. from 6 rats in each group. *p < 0.01; b p < 0.05; NS = not significant; ANOVA = Significant at 1% level.

Effect of Cocoa nucfera and red chilli on intestinal B-gluconoridase and mucin activity in experimental colon cancer

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References

Discussion
Treatment with red chilli, DMDI and coconut kernel brings about profound alterations in the activity of both B-glucuronidase and mucinase. Chilli treated rats and those given DMDI showed increased incidence of tumours both in the colon and intestine. When coconut kernel was included in the diet, the incidence of tumours decreased. The size of the tumours (visible) were significantly reduced and were more or less diffused.

Histopathological studies showed a great degree of variation in the different groups. A significant degree of treated control animals were vascularisation in the colon, but the colon was otherwise normal. The chilli group showed the size of the tumour significantly decreased the activity of this enzyme in the presence of chilli or DMDI, or both, emphasizing the protective role of the kernel.

Mucins are glycoproteins consisting of a large number of carbohydrate side chains attached to a protein core. They serve as a source of energy for the intestinal bacteria and are consequently degraded by them. Supplying the microflora with fermentable diet fibre (i.e. coconut kernel) may permit them to use these substrates preferentially. Thus, the treatment with coconut kernel showed a decrease in the activity of mucinase, while chilli and DMDI treatment showed an increase.

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