Protective Effects of *Cichoricum intybus* Linn (Kasni) Against Dietary Aflatoxicosis in White Albino Rats

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**Abstract:** In the present study fifty four white albino rats were taken and randomly distributed in three groups C, T₁ and T₂. Further each group divided into 3 subgroup of having six rats in each. C, T₁ and T₂ served as control and two treatment groups supplemented with free diet + 0.00 ppm aflatoxin B₁, Free diet + 7.00 ppm aflatoxin B₁ and free diet + 7.00 ppm aflatoxin B₁ + Kasni (100 g/kg feed) by replacing maize in ration respectively. Each group was provided weighed quantity of respective diet daily up to end of 28 days experiment. Body weight and feed intake were recorded at regular interval. Blood samples were analysed to study the biochemical constituents viz Glutamic-oxaloacetic transaminase (GOT), Glutamic-pyruvic transaminase (GPT), Alkaline Phosphatase activity (ALP), total protein (TP), Albumin, Cholesterol, Glucose and Blood urea nitrogen (BUN) and correlation of aflatoxin B₁ with Kasni. The observations of the present study revealed that the results of the treatment T₁ and T₂ group differ significantly (*P*<0.05 and 0.01) with control. The results showed that GOT, GPT, ALP, Cholesterol, Glucose, BUN level increases more in group T₁ than T₂. Total Protein and Albumin decreased more in T₁ then T₂. Similar results were also obtained with glutathione-S-transferase activity. Kasni treatment was found beneficial as it was observed to attenuate aflatoxicosis in treated rats.

**Key words:** Aflatoxin · Kasni · *Cichoricum intybus* · Rat

**INTRODUCTION**

The historic discovery of aflatoxins was due to the death of turkeys in the United Kingdom as a result of consuming contaminated groundnut meal imported from Brazil. The harmful effects of consuming contaminated groundnut cake have been mainly observed in poultry and milk cattle [1]. Subsequently, aflatoxins have been found in other feeds especially maize and cotton seed meal [2]. A global signification survey carried out in different parts of world revealed that maize and groundnut cake are the commodities most affected by mycotoxins [3,4]. Aflatoxins have been recognized to be more harmful to malnourished than well nourished animals and humans [5]. Aflatoxins *M₁* and *M₂* was frequently detected in breast milk of sudanies [6] and United Arab Emirates women [7]. The studies carried out in various countries have found a positive correlation between hepatocellular carcinoma and aflatoxin ingestion by man. The mycotoxicological etiology has been suggested for several chronic progressively disabling, polyarthritic diseases [8]. The greatest danger to human health from aflatoxins is the consumption of aflatoxins contaminated addible items [9]. Aflatoxin, the secondary fungal metabolite of the fungus *Aspergillus flavus* and *Aspergillus parasiticus* is well known for its rapid death, hepatotoxic, carcinogenic, immunosuppression, growth impairment and adverse effects in laboratory and domestic animals viz. chickens, ducklings, rabbits, guinea fowl, rats as well as in humans [10-12]. Their formation may occur when fungi grow on crops in the field, at harvest, in storage or during the processing of feed when favorable conditions for their formation prevail [13]. *Cichoricum intybus* or Kasni is quite effective in detoxification and prevent liver from the toxic effect of various fungal toxins [14]. The root extract of *Cichoricum intybus* at higher doses (i.e. 1000 mg/kg/p.o) is reported to cytotoxic to certain strains of *Salmonella* [15]. The extract is also found to suitable to treat the fungal hepatic toxicity [16,17], intestinal toxicity [18] and non-ulcer dyspepsia [19]. Considering the deleterious effects of aflatoxin and ameliorative effects of *Cichoricum intybus* (Kasni) in fungal infections, the present study was undertaken to find out the scientific correlation of *Cichoricum Intybus*
Glucose level estimated by the method given by Hultman [29] and Blood Urea Nitrogen (BUN) estimated by the method of Schwartz [30]. Glutathione-s-transferase activity was measured in the cytosol with styrene oxide as substrate as described by Baars et al., [31].

RESULTS AND DISCUSSION

During the experimental period the albino rats did not show any clinical signs of illness and mortality except dullness and reduced feed intake in T1 and T2. The variations of body weight and average feed intake with time of various groups are represented graphically (Fig 1 and 2). The results are expressed as mean±standard error of mean of three replicates of six rats each. The total body weight gain and average feed intake were reduced significantly (p<0.01) in aflatoxin B1 fed group, however, there was significant improvement in the weight gain and average feed intake in the animals of Kasni treated group T1. Frape et al., [33] shown that aflatoxin given at the rate of 0.5,1.0 and 5 ppm diet body weight gain, growth rate, feed intake decreased during the experiment due to consumption of aflatoxin. Result of the present study showed that the average mean values of GOT activity (IU) were 58.07±2.41, 111.20±1.92; 103.00±1.42 I.U. in C, T1 and T2 groups respectively. The average mean values of GPT activity (IU) in rats were 28.26±1.59; 83.13±3.32 and 58.33±2.06 in C, T1 and T2 groups respectively (Table 1). The average mean values of GOT of treatment groups are significantly different (P<0.05 and 0.01) with the values of control group (Table 1). An increase of 0.91 and 0.78 times in GOT activity of rats were found in treatment T1 and T2 with control. The average mean values of GPT activity (IU) in rats were 28.26±1.59; 83.13±3.32 and 58.33±2.06 in C, T1 and T2 groups respectively (Table 1). The average mean values of GPT activity of control rats differed significantly (P<0.05 and 0.01) with treatment T1 and dietary levels. In the present study aflatoxin B1 increases GOT and GPT activity while Kasni suppressed GOT and GPT activity induced by aflatoxin B1. The average mean values of ALP activity (units/100ml) in rats were 57.6±2.45; 100.73±1.65; 85.20±1.28 in control, T1 and T2 groups respectively. The average mean values of ALP activity of control rats differed significantly (P<0.05 and 0.01) with treatment T1 and dietary levels. In the present study aflatoxin B1 increases GOT and GPT activity while Kasni suppressed GOT and GPT activity induced by aflatoxin B1. The average mean values of ALP activity (units/100ml) in rats were 57.6±2.45; 100.73±1.65; 85.20±1.28 in control, T1 and T2 groups respectively. The average mean values of total protein and albumin (g/100ml) were 6.60±0.27; 4.06±0.13; 5.28±0.27 and 4.77±0.14; 3.11±0.07; 3.74±0.13 in C, T1 and T2 respectively (Table 1).
Table 1: Changes in hepatic enzymes and other blood constituents in control, aflatoxin B₁ and aflatoxin B₁+Kasni (Cichorium intybus linn.) treated white albino rats. Mean with different superscripts in a column differ significantly (P<0.05 and P < 0.01). Each group has three subgroups having six rats each and data are the mean of four replicates, S.E.M.

<table>
<thead>
<tr>
<th>Groups</th>
<th>GOT (I.U.)</th>
<th>GPT (I.U.)</th>
<th>ALP (Units/100ml)</th>
<th>TP (g/100ml)</th>
<th>Albumin (g/100ml)</th>
<th>Cholesterol (mg/100ml)</th>
<th>Glucose (mg/100ml)</th>
<th>BUN (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (C)</td>
<td>56.80±3.23</td>
<td>29.00±1.42</td>
<td>57.40±2.36</td>
<td>6.76±0.28</td>
<td>5.08±0.06</td>
<td>49.00±1.82</td>
<td>84.20±1.53</td>
<td>11.00±1.71</td>
</tr>
<tr>
<td></td>
<td>57.40±2.59</td>
<td>27.20±1.36</td>
<td>57.60±1.94</td>
<td>6.14±0.22</td>
<td>4.32±0.14</td>
<td>50.04±2.13</td>
<td>79.80±1.12</td>
<td>12.40±1.72</td>
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<tr>
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<td>60.00±1.41</td>
<td>28.60±2.01</td>
<td>57.80±3.04</td>
<td>6.90±0.31</td>
<td>4.92±0.29</td>
<td>48.60±2.44</td>
<td>77.00±0.89</td>
<td>10.40±1.57</td>
</tr>
<tr>
<td>Mean±S.E.M.</td>
<td>58.07±2.41</td>
<td>28.26±1.59</td>
<td>57.8±2.45</td>
<td>6.60±0.27</td>
<td>4.77±0.14</td>
<td>49.21±2.13</td>
<td>80.33±1.18</td>
<td>11.27±1.67</td>
</tr>
<tr>
<td>Treatment (T₁)</td>
<td>104.00±1.41</td>
<td>80.60±3.81</td>
<td>96.80±0.97</td>
<td>4.28±0.17</td>
<td>3.38±0.08</td>
<td>91.40±1.56</td>
<td>126.60±1.07</td>
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<td>115.80±2.01</td>
<td>84.00±2.82</td>
<td>104.20±1.57</td>
<td>3.86±0.06</td>
<td>3.20±0.05</td>
<td>100.60±1.07</td>
<td>126.20±1.36</td>
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<tr>
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<td>113.80±2.94</td>
<td>84.80±3.31</td>
<td>101.20±2.42</td>
<td>4.30±0.16</td>
<td>2.74±0.08</td>
<td>92.60±1.78</td>
<td>132.20±1.02</td>
<td>36.00±1.52</td>
</tr>
<tr>
<td>Mean±S.E.M.</td>
<td>111.20±1.92</td>
<td>83.13±3.32</td>
<td>100.73±1.65</td>
<td>4.06±0.13</td>
<td>3.11±0.07</td>
<td>94.87±1.47</td>
<td>128.33±1.15</td>
<td>36.20±1.60</td>
</tr>
<tr>
<td>Treatment (T₂)</td>
<td>101.80±1.35</td>
<td>54.80±1.66</td>
<td>83.00±0.95</td>
<td>5.20±0.16</td>
<td>3.80±0.12</td>
<td>64.20±2.29</td>
<td>102.00±2.66</td>
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<td></td>
<td>108.20±1.16</td>
<td>58.60±1.97</td>
<td>83.60±1.72</td>
<td>5.58±0.37</td>
<td>3.80±0.13</td>
<td>75.20±0.96</td>
<td>102.80±2.06</td>
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<td>99.00±1.74</td>
<td>61.60±2.56</td>
<td>89.00±1.18</td>
<td>5.06±0.28</td>
<td>3.62±0.14</td>
<td>71.80±3.08</td>
<td>105.40±3.06</td>
<td>29.80±0.66</td>
</tr>
<tr>
<td>Mean±S.E.M.</td>
<td>103.00±1.42</td>
<td>58.33±2.06</td>
<td>85.20±1.28</td>
<td>5.28±0.27</td>
<td>3.74±0.13</td>
<td>70.40±2.11</td>
<td>103.40±2.59</td>
<td>27.73±1.01</td>
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Fig. 1: Time course of body weight (gm) in rats. Results are expressed as mean±S.E.M. b=p<0.05 Vs a

Fig. 2: Time course of feed intake (gm) in rats. Results are expressed as mean±S.E.M. b=p<0.05 Vs a

The present investigation showed that there was significant (P<0.05and 0.01) reduction in total protein and albumin in T₂ with Control. In Kasni treated group T₂ reduction in total protein and albumin is less than T₁ group. The mean values of serum cholesterol (mg/100 ml) 49.21±2.13; 94.87±1.47; 70.40±2.11 in C, T₁ and T₂ which are significantly (P<0.05and 0.01) different with each other. Aflatoxin B₁ is responsible for the toxicity observed in groups fed on diet T₁ and T₂ as shown by the increase in serum enzymes. These enzymes are known to be good indicators of aflatoxin B₁ toxicity [34]. The average mean value of Glucose (mg/100ml) and Blood urea nitrogen (BUN) (%) were 80.33±1.18; 128.33±1.15; 103.40±2.59 and 11.27±1.67; 36.20±1.60; 27.73±1.01 in C and T₂, and
Fig. 3: C, T1 and T2, represents measurement of glutathione-s-transferase (nMol/mg proteins) in rats. Results are expressed as mean±S.E.M. b=p<0.01 Vs a, c=p<0.01 Vs b

T2 respectively (Table 1). Enhancement in the level of glucose and blood urea nitrogen on feeding diet contains aflatoxin B1 was observed in rats. Kasni in T2 group suppressed the enhanced activity of glucose and BUN. Increasing trend in GPT, GOT, ALP, cholesterol, BUN and decreasing trends in total protein and albumin reported by various workers in albino rats and in other species [11,35]. Moundipa and Domngang [36] reported same type of degradation in aflatoxicosis on feeding aflatoxin B1 with solanum nigrum in female rats. In 1999 Hayashi et al. [37] reported that chlorophyllin also have anticarcinogenic activity against aflatoxicosis. The activity of glutathione-s-transferase (Fig 3) was higher in T1 and T2 groups as compared with control. The activity of this enzyme increased by about 42% in T1 and 48% in the T2 group compared with control group. The observations of the present study revealed that the results of treatment T1 and T2 differ significantly (P<0.05 and 0.01) with control groups. The activity of hepatic UDPGT was depressed in male rats given a low protein, energy restricted diet and cytochrome-P450 was depressed by a low protein ad lib energy diet [38]. The hepatic electron transport component and the glycogen content of female rat liver lowered by restricting food by 50% while restriction food by 25% did not alter the level of electron transport component [39]. Our findings shown that in Kasni treated groups the level of drug metabolizing enzymes were higher. The metabolite aflatoxin B1-dihydrodiol derived from epoxide through the intervention of epoxide hydratase an enzyme whose activity has been shown to increase in female rats after chronic administration of aflatoxin B1[40]. This metabolite has a probable role in the toxicity of aflatoxin B1, since its relative production by the microsomes from various species parallels their in vivo susceptibilities to aflatoxin B1 poisoning [41]. Although epoxide hydratase yields this toxic metabolites, it also prevents the formation of AFB1-DNA adducts, which could induce mutation. However, aflatoxin B1-dihydrodiol could be rapidly conjugated by UDPGT and glutathione-s-transferase, which increased in the groups, fed on Kasni. Conjugation to glucuronic acid and glutathione via UDPGT and glutathione-s-transferase respectively has been established as an important pathway in the detoxification and excretion of toxic metabolites of aflatoxin B1[42]. The level of GOT, GPT and ALP in T2 groups after the treatment, suggest the possible importance of Kasni in inducing excretion of the toxin.

CONCLUSION

On the bases of results it may be concluded that aflatoxin B1, resulted in decrease in total protein and albumin and increase in GOT, GPT, ALP, Glucose, BUN and cholesterol. The improvement in glutathione-s-transferase was observed in Kasni treated rats T2 group, which helps in excretion of aflatoxin from body. However toxicity observed in treated group even with the addition of Kasni could be due to insufficient level of Kasni. These diagnostic tools will be beneficial to control aflatoxicosis. Further studies are needed in this direction.

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