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Response of Bovans -Hybrid Chicks to *Allium cepa*, Paracetamol or their mixture

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Summary

The objective of present study was to investigate the effects of *Allium cepa* (Onion), paracetamol or their mixture on the growth, pathological, biochemical and haematological parameters of Bovans-hybrid chicks. *A. cepa* (onion), paracetamol administration or their mixture were fed to Bovans-hybrid chicks at 10% (w/w) *A. cepa*, mixture of 5 % (w/w) *A. cepa* and 250 mg/kg paracetamol and 500mg/kg of paracetamol of normal basal diet. The growth of chicks in tested groups was less than that of untreated / control chicks. Significant decrease in haemoglobin concentration was observed on 10% *A. cepa*. There were no significant changes in the values of red blood cell (RBC), while there was significant increase of packed cell volume (PCV) in *A. cepa* and Paracetamol mixture. 10% (w/w) *A. cepa* group showed significant decrease on serum alanine aminotransferase, (ALT) activity, total protein, and globulin and uric acid concentration. Also this group showed decreased cholesterol level. There were significant differences in the concentration of total protein, albumin and the activity of ALT in the given mixture of 5% (w/w) *A. cepa* and 250 mg/kg paracetamol and those given 500mg/kg of paracetamol. Cholesterol level was significantly decreased in chicks given 500mg/kg of paracetamol. Pathological changes were observed in chicks fed 10% (w/w) A. *cepa*. Histopathological examination showed a damaging effect, necrosis and fibrosis of liver cells in the chicks given 500mg/kg of paracetamol of normal basal diet. There was no evidence of any nephrotoxic activity of the plant used. This study indicated the hepatotoxic effect of 10% (w/w) *A. cepa* and the dose of 500mg/kg of paracetamol on chicks.

Introduction

Paracetamol (acetaminophen, *N*-acetyl- *p*-aminophenol, 4-hydroxy-acetanilide) was introduced into clinical medicine towards the end of the last century **(Smith, 1958).**

There was a resurgence of interest in paracetamol when it was found to be the major metabolite of acetanilide and phenacetin and it was commonly

assumed to be responsible for the therapeutic effects of both of these drugs. Paracetamol has since been used increasingly as a substitute for other analgesics such as aspirin and phenacetin. The first observations a bout the analgesic and anti pyretic properties of

Paracetamol were made back in the nineteen's century and being thought to reduce fever in the treatment of infection **(Brodie and Axelrod, 1948a).**

There is considerable species difference in susceptibility to paracetamol-induced liver damage, which correlates with difference in the activity of the oxidation pathway in these species. The ability of paracetamol to produce acute centrilobular hepatic necrosis in experimental animals has been confirmed repeatedly and there are major species differences in susceptibility. Mice and hamsters are very sensitive while rats are less resistant and this difference has been related to species difference in the extent of the metabolite activation of paracetamol **(Tee *et al.*, 1957; Mitchell, 1977; Prescott, 1983; Elhabib *et al.*, 2007).** Other reported complications of paracetamol poisoning include oliguric renal failure **(Cobden *et al.*, 1982),** pancreatitis and myocarditis **(Wakeel *et al.*, 1987).**

Mitchell *et al.* (1973a; 1973b); Potter *et al.*, (1973) and Jollow *et al.*, (1973) showed that a minor route of paracetamol metabolism involved its corrosion by cytochrome P-450 dependant mixed function oxidase to a reactive arylating metabolite now known to be N. acetyl-p- benzoquinoneimine (NAPQI) which may cause acute hepatic necrosis with toxic doses of paracetamol **(Dahlin *et al.*, 1984; Holme *et al.*, 1984).**

Doses of 750 mg/k (4. 94 μ mol/kg) were sufficient to cause severe hepatic necrosis in mice while doses of 1250-1500 mg/kg (8.3-9.9 μmol/kg caused little hepatic necrosis in rats despite being lethal **(Mitchell *et al.*, 1973).**

Plants are used as sources of pharmaceuticals as they contain chemical constituents and are utilized in traditional medicine for the treatment of many diseases. With modern expensive treatments, herbal medicine has emerged as a safe and cheap alternative to pharmaceuticals.

Allium cepa (onion) is a member of the Liliacae family and is among the oldest of all cultivated plants. It has been traditionally used as an antispasmodic, carminative, diuretic, expectorant, stomachic, anthelmintic and anti-infective agent **(Leung *et al.*, 1980).** Onion has been also used as traditional medicines to treat a variety of diseases including common cold, arthritis, headache and heart disease. Externally, it has been used as a rubefacient and poultice application for relief of pain, skin diseases and insect bites **(Fenwick and Hanley, 1985).**

A. cepa was found to exhibit antibacterial antifungal and anthelmintic activity **(Leung *et al.*, 1980).** Onion and onion extracts have been shown to decrease blood lipid level, increase fibrinolysis, decrease platelets aggregation and lower blood pressure in several clinical studies **(Menon *et al.*, 1969; Louria *et al.*, 1985).** *A. cepa* has been shown to be a significant oral hypoglycaemic agent comparable to that of oral hypoglycaemic agent tolbutamide and phenformin **(Abdel Gadir, 2005; Abdel Gadir *et al.*, 2006).**

Onion extract was found to be cytoxic to tumors in vitro and was found to arrest tumour gross when tumour cells were implanted in rats **(Nepkar *et al.*, 1981)** and used

also for anti lieshmainial lesions (**Saleheen *et al.*, 2004**). Crude extract caused vascular excavation and anti platelet effect by prevention of platelet aggregation and adhesion (**Azuma *et al.*, 1986; Radomeski *et al.*, 1987; Jen *et al.*, 1995**).

Recently it has been reported that Welsh onion extract modulate rat's aortic vascular tone in both endothelium-dependant and endothelium-independent manners (**Chen, *et al.*, 1999**).

Because of the common use of *A. cepa* in tradition medicine, and the wide use of paracetamol as antistress and anti analgesic and the paucity of information and investigation on the toxico-nutritional effect of the *A. cepa* (Onion) and paracetamol on chicks; the present study was carried out to investigate the effects of *A. cepa* (Onion), paracetamol or their mixture on the growth, pathological, biochemical and haematological parameters of Bovans-hybrid chicks.

Materials and Methods

Materials and experimental design:

Chicks and diets: Forty, one-day-old-Bovans cockerels were obtained from Coral Company, Khartoum, Sudan and reared in a poultry house within the premises of the College of Veterinary Medicine and Animal Production, Sudan University of Science and Technology, under illumination at night and early morning with feed (starter ration) and drinking water provided *ad. libitum*. This experiment was carried out in from 1st of May to 28th of 2006. At the age of 14 days, the chicks were divided at random into four groups, each of 10. Chicks in group 1 were the controls and fed commercial starter ration (basic diet) for 2 weeks (treatment period) (Table 1).

Blood samples were collected at slaughter for haematology and serum chemistry analysis. At necropsy, all chicks were examined for gross lesions. The liver, proventriculus, intestines, spleen, heart and kidneys were fixed in 10% neutral buffered formalin.

Average body weights and weight gains were determined weekly for each group.

Plant material: *Allium cepa* (Onion) was bought from a local Market, dried in the shade, cleaned, finely ground and thoroughly mixed with the control diet and fed to chicks at 10% (w/w) of basic diet for 2 weeks (treatment period) (group 2).

Paracetamol: Paracetamol was bought from a local pharmacy (DADVET "DAR ALDAWA Veterinary and Agricultural Industrial Co Ltd, Jordan). Paracetamol was added to feed at 500mg/kg body weight (b. wt.) for 2 weeks (treatment period) (group 4).

Group 3 was given a mixture of onion and paracetamol diet (5% (w/w) onion and 250 mg/kg paracetamol). Average body weight and weight gain and clinical signs and mortality were recorded. Chicks from each group were slaughtered at week 2, for pathological examination. Liver, proventriculus, intestines, heart, kidneys and spleen, were fixed in 10% neutral buffered formalin and processed for histopathology. Blood samples were collected at slaughter for haematology and serum analysis.

Growth changes: Average body weight and weight gains for each group were determined weekly.

Clinical chemistry: Sera were analyzed for the activities of aspartate aminotransferase (AST), and alanine aminotransferase (ALT) and for the concentrations of total protein, albumin, uric acid and cholesterol. by commercial kits (Stanbio Laboratory Inc, San Antonio, Texas, Bio- Analytics, Palm City, Florida, or King Diagnostic Inc, Indianapolis, USA) using spectrophotomer (RA-50 Chemistry Analyzer, Ames, Bayer Diagnostics).

Haematology: Blood samples were estimated by standard methods (Schalm *et al*, 1975) for haemoglobin concentration (Hb), packed cell volume (PCV), red blood cell (RBC) count, mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH) and mean corpuscular haemoglobin concentration (MCHC).

Pathological Examination: Post-mortem findings were recorded for all chicks and specimens of proventriculus, intestines, liver, spleen, kidneys and heart were fixed in 10% neutral buffered formalin, embedded in paraffin wax, sectioned at 5μm and stained with haematoxylin and eosin (H&E).

Statistical Analysis: The significance of differences between means was compared at each time point using Duncan's multiple range tests after ANOVA for one-way classified data **(Snedecor and Cochran, 1989).**

Results

Effect on growth:- The effect of treatment with diets containing paracetamol 500mg/kg b wt, 10% (w/w) *A. cepa* or mixture of paracetamol at 250mg/kg b wt and 5% (w/w) *A. cepa* plant on body weight and weight gain of the chicks are shown in Table 2. The chicks fed a diet consisting of 10% (w/w) *A. cepa* (group2) and paracetamol 500mg (group 4) for 2 weeks showed lower ($P<0.05$) growth rate than the control (group 1) and chicks on mixture of substances (group 3).

None of the chicks died during the course of the experiment.

Pathological changes: No significant lesions were observed in the control chicks. Histopathological examination showed a damaging effect, necrosis, fibrosis and cytoplasmic fatty vacuolation of the centrilobular hepatocytes **(Fig. 1)**, in the chicks given 500mg/kg of paracetamol of normal basal diet.

Haematological changes: Data are presented in Table 3. There was significant decreases ($P<0.05$) in Hb concentration in group 2.

There were no significant changes in the values of RBC. There was significant increase of PCV % in group 4 as shown in Table 3.

Serum chemistry: The effects of paracetamol, onion or their mixture on the concentration of total protein, albumin globulin, cholesterol and uric acid, and the activity of ALT and AST enzymes, in the serum of Bovans- hybrid chicks are given in Table 4.

Group 2 showed significant decrease ($P<0.05$) in serum ALT activity and total protein, albumin in group 3 and 4, globulin in group 2 and uric acid concentration in groups 2 and 3, also decreased cholesterol level in group 2-4 without significant differences in AST activity shown.

Discussion

The results of the present study indicated that the mean body weight gain of the Bovans chicks fed the 10% (w/w) *A. cepa*, paracetamol or their mixture for 2 weeks showed lower weight gain than the control group. The decrease in weight due to feeding10 %(w/w) *A. cepa* alone or in mixture with paracetamol probably is due to the nutritive constituents of the plant and the hepatotoxic effect of paracetamol. Research would be required to verify the role of the *A. cepa* and paracetamol at this concentration or at lower levels e.g. 2% (w/w) because many plants have been investigated for growth promotion effect and paracetamol as antistress drug. For example, the mean body weight of 2% (w/w) *C. italica* seed in the basal diet promoted the growth of elevated due to the presence of high protein content while incorporation of this plant at 10% (w/w) of the basic diet caused hepatonephrotoxicity in rats **(Adam, 1998).** Damage to the liver probably did not contribute to raise serum ALT or AST activity but this might contribute to decreased cholesterol, total protein and albumin concentration particularly in the chicks fed *A. cepa* or mixture of and to the decrease in globulin level in birds fed *A. cepa* singly. The absence of significant renal damage did not cause an increase in serum uric acid concentration. Serum uric acid was found to increase in the chicks fed 10% (w/w) *Ammi visnaga* for 6 weeks **(Ibrahim *et al.*, 2004).** Previous results revealed that onion and garlic were significantly associated with a decreased level of serum cholesterol and HDL cholesterol in goats and also a significant decrease in the level of triglycerides. These results may be associated with suppression in initial mobilization of tissue lipids into circulation **(Lau *et al.*, 1987).** This result agreed with that of **Kumar *et al.* (1998) and Kumar *et al.* (2000),** who found that onion and garlic caused decrease in plasma cholesterol, esterified cholesterol, high density lipoprotein cholesterol, and low density lipoprotein cholesterol content in both sexes of Japanese quails. Decreased total protein and cholesterol concentration in chicks fed paracetamol indicated liver damage. Paracetamol sales in the United Kingdom exceeded those of aspirin for more than a decade. As a consequence of the "back door" introduction of paracetamol, there were no formal preclinical animal toxicity studies such as would be required today, and its potential hepatotoxic effect was not suspected until the first clinical reports of severe and fatal liver damage following over dosage **(Davidson and Eastham, 1966; Thomson and Prescott, 1966).** Severe hepatic necrosis was first observed in cats treated with paracetamol (25 mg/kg and then 50 mg/kg) for 22 weeks **(Eder, 1964),** and it was also described in rats given doses in the range of the acute LD_{50} and the 100-day LD_{50} **(Boyd and Bereczky, 1966; Boyd and Hogan, 1968).** Previous investigations showed macrocytic normochromic anaemia in

chicks fed 10% *R. stricta* **leaf (Al-Homidan *et al.*, 2002)** or 10% (w/w) *Cassia italica* seed **(Bakhiet and Adam, 1996)** and rats fed 10% (w/w) *Citrullus colocynthis* fruit **(Al-Qarawi and Adam, 2003).**

Pharmacological and metabolic studies are required to evaluate paracetamol modes of action, identify its metabolites and possible interactions with other medicinal plants constituents to counteract its toxic effect in chicks.

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Table 1. Composition of starter ration (%)

Ingredients	**%**
Sorghum	58
Soybean	4
Sesame cake	14
Groundnut cake	12
Wheat bran	5
Marble dust	1
Decalcium phosphate	1
Super concentrate	5
Total	100

Source: Commercial ration from local market

Table 2. Weight gain of Bovans chicks fed *A. cepa*, paracetamol or their mixture for 2 weeks.

Groups	Body weight(g) 2nd week	Weight gain (g)3rd week	Weight gain (g) 4th week
Group 1 Control	64.0 ± 1.67	31 ± 1.2	79.0 ± 2.1
Group 2 10% (w/w) *A. cepa*	64.0 ± 4.3 NS	39.0 ± 4.6 NS	48.0 ± 4.0*
Group 3 (Mixture of 5% (w/w) *A. cepa* and 250mg/kg paracetamol	67.0 ± 4.6 NS	48.0 ± 2.9*	83.0 ± 4.0 NS
Group 4 Paracetamol 500mg/kg	61.0 ± 4.6 NS	38.0 ± 5.2 NS	52. ± 4.0*

Table 3. Haematological changes in Bovans chicks fed *A. cepa*, paracetamol or their mixture for 2 weeks.

Parameters	Groups			
	1 (control)	**2 10%(w/w) *A. cepa)***	**3 (Mixture of 5% (w/w) *A. cepa* and 250mg/kg paracetamol**	**4 Paracetamol 500mg/kg**
Hb (g/dl)	6.7 ± 0.35	٥,٧ ± ٤,٢*	٧,٠ ± ٠,٤١ NS	7.٥ ± 0.52 NS
RBC (10^6mm^3)	٢,٥٦ ± ٠,٠٩	٢,١٧ ± 0.٦٧ NS	٢,٩٥ ± ٠,١٦ NS	2.٥٦ ± ٠,٢٢ NS
PCV (%)	19.2 ± 1.٢	±١٦,٢ 1.٢. NS	±٢٠,٢ ١,٢ NS	22.±٠ ١,٧ NS
MCV (m)^3	75 ±1.7	6٠.8 ± ١,١ NS	٦٨,٩ ± ٥,٦ NS	٨٢,٤ ± 2.2 NS
MCH (pg)	26.2 ± 1.8	٢١,٢ ± ٤,٩ NS	٢٢,٨ ± ١,٨ NS	٢٠,٢ ± ٢,٦ NS
MCHC (%)	33.٩ ± ٠,٢٨	35.٢ ± ٠,٢٥ NS	٢٢,٦ ± ١,٠٢ NS	٢٤,٢ ± ٠,٢٢ NS

Values are means = ± SE; * = P<0.05; NS= Not significant

Table 4. Serobiochemical changes in Bovans chicks fed *A. cepa*, paracetamol or their mixture for 2 weeks.

Parameters	Groups			
	1 (control)	2 10%(*w/w) A. cepa)*	3 Mixture of 5% (w/w) *A. cepa* and 250mg/kg paracetamol	4 Paracetamol 500mg/kg
AST (i.u)	18.2 ± 0.6	18.8 ± 2.1 NS	17.6±2.29 NS	18.4 ± 3.28 NS
ALT (i.u)	15.6 ± 0.92	8.6 ± 1.29 *	11.4 ± 1.96*	10.6 ± 2.46*
Total protein (g/dl)	2.59 ± 0.13	1.81±0.25 *	1.71 ± 0.83*	1.82 ± 0.95*
Albumin (g/dl)	1.5 ± 0.08	1.04±0.11 NS	0.68 ± 0.37*	0.84 ± 0.11*
Globulin (g/dl)	1.09 ± 0.2	0.77 ± 0.08*	1.03 ± 0.17 NS	0.98 ± 0.19 NS
Cholesterol mg/dl)	156.5 ± 5.7	168.9± 3.8 *	209.6 ± 2.8 *	145.7 ± 9.2*
Uric acid (mg/dl)	4.64 ± 0.19	2.34 ± 0.57*	2.41 ± 0.46*	4.31 ± 0.56 NS

Values are means= ± SE; * = P<0.05; NS= Not significant

Fig.1. Fatty vacuolation and necrosis of the centrilobular hepatocytes in a chick given paracetamol at 500mg/kg diet for 2 weeks. H & E x 200