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Immuno-pathological effects of experimentally induced sodium selenite toxicity in broiler chickens

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ABSTRACT

Selenium is an essential micronutrient for poultry, but excess intake causes acute and chronic toxicities. The present study was conducted to determine acute oral lethal dose 50 (ALD_{50}) of sodium selenite in broiler chickens and the associated gross, histopathological, and immunological alterations. A total of 300 day-old broiler chicks were used. Acute oral toxicity was assessed in 140 chicks by graded dosing, and the oral ALD_{50} of sodium selenite was calculated as 21 mg/kg body weight (equivalent to 9.59 mg Se/kg), confirming its high toxicity. Chronic exposure was studied by administering sodium selenite at 15 ppm and 30 ppm in drinking water. Birds receiving 30 ppm showed marked anorexia, emaciation, nervous signs, and high mortality, while 15 ppm caused reduced weight gain without mortality. Gross lesions included emaciation, muscular pallor, hepatic haemorrhages, renal congestion, and lymphoid organ atrophy. Histopathology revealed lymphoid depletion, follicular necrosis, and degeneration of reticuloendothelial cells in lymphoid tissues. Immunologically, selenium-fed groups showed significant suppression of cell-mediated immunity, as evidenced by reduced delayed-type hypersensitivity response to phyto-haemagglutinin, and diminished humoral response, reflected by lower antibody titres against Newcastle disease vaccine. These findings established sodium selenite as a potent immuno-toxicant in broiler chickens, with a narrow margin between requirement and toxicity. The study highlighted the risks of excessive selenium exposure in poultry production systems, particularly in selenium-endemic areas, due to its immunosuppressive potential.

Keywords: ALD_{50}, Broiler chickens, Immune-toxicity and sodium selenite

Selenium (Se) is an essential trace element with a paradoxical role in nutrition of both animals and humans. While indispensable in small amounts, it becomes toxic when consumed in large quantity. Selenium is now recognized as a critical micronutrient required for the prevention of deficiency-related disorders such as Keshan disease in humans, white muscle disease in calves, and exudative diathesis in chicks (Chen, 1986, NRC, 1984). The biological functions of selenium are closely linked with those of Vitamin E, both acting as antioxidants that protect cellular membranes from oxidative damage through inhibition of lipid peroxidation. Selenium is incorporated into several selenoproteins, notably glutathione peroxidases, thioredoxin reductases, and selenoprotein P, which play pivotal roles in antioxidant defense, thyroid hormone metabolism, immune regulation, and cellular redox balance (Rayman, 2012, Pillai *et al.* 2014). Despite its critical role, the margin between selenium's essentiality and toxicity is narrow. Dietary concentrations exceeding 5–15 ppm can induce toxicosis in livestock and poultry (Roy, 2001). The toxic effects are influenced by several factors, including the chemical form, dose, route of administration, age, and species sensitivity. Among the different forms, sodium selenite, a highly soluble inorganic compound, is particularly toxic due to its high bioavailability compared to the relatively insoluble selenides or elemental selenium (NRC, 2006). In poultry, acute selenium poisoning is characterized by anorexia, depression, dyspnea, diarrhea, nervous signs, and high mortality. Chronic exposure, on the other hand, results in reduced growth, emaciation, anemia, immune suppression, and degenerative changes in vital organs such as the liver, kidney, and myocardium. Importantly, selenium toxicosis also exerts immune-toxic effects, including suppression of both cell-mediated and humoral immune responses, thereby predisposing birds to increased

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disease susceptibility and poor vaccine responsiveness (Biswas *et al.* 2006; Kieliszek, 2019; Surai *et al.* 2020). Recent advances in poultry nutrition have focused on safer selenium supplementation strategies to minimize toxicity risks, while maximizing biological efficacy. Organic forms such as seleno-methionine and selenium-enriched yeast demonstrate higher bioavailability and reduced pro-oxidant effects compared to inorganic sodium selenite, leading to improved growth, reproductive performance, and immune function (Wang *et al.* 2021, Zhou and Wang, 2023). Additionally, nano-selenium has emerged as a promising alternative due to its enhanced bioactivity, stability, and lower toxicity, making it an attractive candidate for precision supplementation in poultry diets (Zhang *et al.* 2020, Ibrahim *et al.* 2023). Given selenium's dual role as an essential nutrient and a potent toxicant, it is crucial to precisely define the thresholds between its requirement and toxicity. The present study was designed to determine the acute oral LD_{50} of sodium selenite in broiler chicks and to evaluate its immune-pathological effects through gross, histological, and immune-response-based assessments.

MATERIALS AND METHODS

For this study, a total of 300 day-old broiler chicks of either sex from the same hatch were procured from a commercial hatchery and used for acute LD_{50} evaluation, chronic toxicity and immunological assessment. The experimental room and equipment were disinfected with 2.5% phenol and fumigated with formaldehyde prior to placement of chicks, and standard broiler rations containing 22% CP and 2900 kcal/kg ME for the starter phase and 19% CP with 2800 kcal/kg ME for the grower phase, along with clean drinking water, were provided *ad libitum* throughout the trial.

To determine acute oral LD_{50} of sodium selenite, 140 chicks aged 7 days were divided into groups and administered graded doses of 1, 5, 10, 15, 20, 25, and 30 mg/kg body weight, and mortality was recorded within 24 h to calculate the approximate LD_{50} using Karber's method (Ghosh, 1984). For the chronic toxicity study, a total of 120 chicks were randomly divided into three groups of 40 birds each as Group A, Group B, and Group C. Group A received 30 ppm sodium selenite in their drinking water, Group B received 15 ppm, while Group C served as the control group and was given plain water. All birds were monitored daily for clinical signs and mortality along with weekly recording of body weights in representative samples. Selenium concentrations in liver tissue were measured at various intervals. Birds were sacrificed on days 14 and 28 for gross and histopathological examinations.

For immunological assessment, 40 chicks were divided into four groups of 10 each, with two groups receiving 15 ppm sodium selenite for 21 days and the remaining two groups serving as untreated controls. Cell-mediated immunity was evaluated by delayed-type hypersensitivity (DTH) response using phyto-haemagglutinin-purified (PHA-p) injection into the wing web with thickness measured at 9, 18, 27, and 36 h, while humoral immunity was assessed by vaccinating birds with Newcastle disease virus (NDV, Lasota strain) on day 28 and measuring serum antibody titres at weekly intervals post-vaccination using the hemagglutination inhibition test.

The protocol of the study was approved by the Institutional Ethics Committee vide IAEC/BVC/2023/06, dated 28.02.2023. Data generated from the experiment were analysed using analysis of variance (ANOVA), and statistical significance was considered at $P<0.05$ as per Snedecor and Cochran (1967).

RESULTS AND DISCUSSION

Oral ALD_{50} of sodium selenite: Broiler chicks administered sodium selenite at doses ≥10 mg/kg body weight exhibited anorexia, diarrhea, dullness, and depression, with progressive nervous signs and paralysis at higher doses. At 30 mg/kg, all birds died within 9 h. Using Karber's method, the oral ALD_{50} of sodium selenite was calculated as 21 mg/kg body weight, equivalent to 9.59 mg/kg selenium, corroborating earlier reports (Salyi *et al.* 1993) and confirming the high acute toxicity of sodium selenite in broilers (Table 1).

Clinical signs and mortality in chronic toxicity: Broiler chicks in Group A (30 ppm) began showing signs of dullness, depression, weakness, and anorexia from day 3 onward. Between days 8 and 12, neurological symptoms such as fright responses and paralysis became evident. By day 28, mortality in this group had reached 55%, with the majority of deaths occurring within the first three weeks. In contrast, birds in Group B (15 ppm) exhibited only anorexia

Table 1. Acute oral toxicity of sodium selenite in broiler chicks (ALD_{50})

Dose (mg/kg b.wt)	No. of Birds	No. of birds died	Mortality (%) within 24 h	Clinical signs observed
01	20	0	0	No visible signs
05	20	0	0	Mild dullness, anorexia
10	20	2	10	Anorexia, diarrhea
15	20	5	25	Weakness, dullness
20	20	8	40	Ataxia, paralysis
25	20	15	75	Severe nervous signs, paralysis
30	20	20	100	Death within 9 h

Calculated oral LD_{50} = 21 mg/kg b.wt (equivalent to 9.59 mg/kg selenium).

and reduced weight gain, with no mortality observed. The control group remained healthy throughout the trial. These findings supported previous reports of dose-dependent selenosis, which is characterized by anorexia, progressive emaciation, and neurological disorders (Franke and Tully, 1935; Khan *et al.* 1993).

Growth performance: Selenium exposure markedly suppressed body weight gain in both Groups A and B compared with controls, with the effect being more pronounced in Group A. Birds receiving 30 ppm sodium selenite showed negligible weight gain after the first week of treatment. The growth depression appeared to be associated with anorexia, reduced feed intake, and impaired cellular metabolism, as selenium might have substituted for sulphur in proteins and interfered with mitochondrial respiration (Brown and de Wet, 1962).

Selenium concentration in liver: Liver selenium levels increased progressively with both dose and duration of exposure. Mean concentrations ranged from 1.30 to 2.90 mg/g in Group A, 0.80 to 2.43 mg/g in Group B, and 0.30 to 0.66 mg/g in controls. Birds that died naturally during the trial exhibited markedly higher hepatic selenium levels (3–5 mg/g). This dose-dependent accumulation was consistent with earlier reports (Akulov *et al.* 1972; Jensen, 1975).

Gross changes: In Group A, gross lesions included severe emaciation, pale and atrophied muscles, hepatic hemorrhages, congested kidneys, and ecchymotic hemorrhages in skeletal muscles. Birds in Group B exhibited similar but less severe lesions, while no abnormalities were observed in the control group C. These findings supported the role of selenium-induced oxidative cytotoxicity and impaired tissue respiration, as previously reported in both poultry and mammals (Das *et al.* 1990; Salyi *et al.* 1993).

Cell-mediated immunity (CMI): As different T-lymphocyte subpopulations regulate both the effector and regulatory phases of cell-mediated immunity (CMI), and *in-vitro* assessment is often limited, CMI in this study was evaluated *in-vivo* using PHA-p–induced skin sensitivity to determine the effects of chronic selenium toxicity.

Sodium selenite-treated birds showed significant reduction in wing web swelling in the PHA-p test, confirming suppression of T-cell responsiveness. Histological examination of the injection sites revealed decreased lymphocyte infiltration compared with controls.

Phytohaemagglutinin-purified (PHA-p) test: The CMI response was further evaluated by measuring the blastogenic activity of T-lymphocytes stimulated with PHA-p. The results demonstrated that the average increase in skin thickness and duration of reaction at the challenge sites were significantly lower ($P<0.01$) in selenium-treated birds (15 ppm) compared with controls at 9, 18, and 27 h post-injection (Table 2). The immune response peaked at 9 h and progressively declined from 18 h onward. These findings confirmed a dose-related suppression of CMI in selenium-exposed birds. Interestingly, Kegley *et al.* (1996) reported that sodium selenite enhanced the CMI response in Holstein calves, highlighting possible species-specific differences.

Cyto-pathological changes: At 9 hours' post–PHA-p injection, the skin of selenite-treated birds exhibited

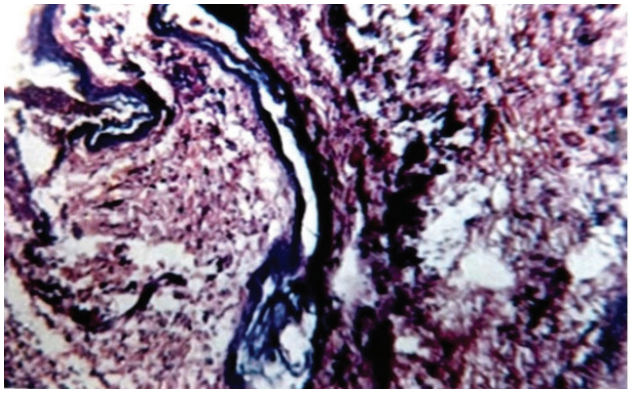

Fig. 1. Section of skin from sodium selenite treated broiler chickens showing haemorrhages, congestion, perivascular cuffing and infiltration of mononuclear cells at 9 hours after PHA-p injection (H and E, 100×)

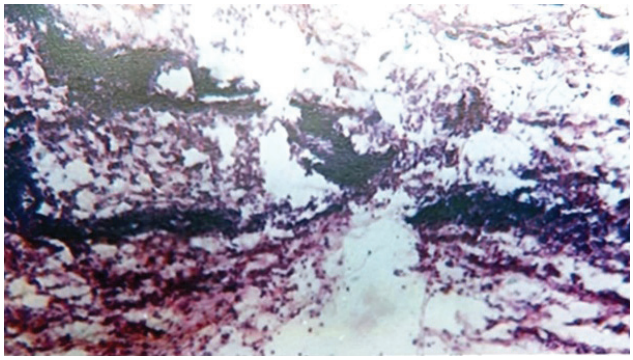

Fig. 2. Section of skin of broiler chickens treated with sodium selenite showing lesser intensity of vascular and cellular changes at 27 hours after injection of PHA-p (H and E, 150×)

Table 2. Effect of sodium selenite on PHA-p induced wing web swelling response in broiler chickens (Mean ± S.E.)

Treatment	Sub- cutaneous injection in wing web	Post-injection hours (Mean ± S.E)			
		9	18	27	36
Control	Left (Saline)	28.6±1.76	20.3±1.34	15.1±0.52	8.2±0.69
	Right (PHA-A)	244.8±4.60	272.8±2.47	223.6±3.16	206.8±2.18
Selenium Treatment	Left (Saline)	23.2±1.16	14.5±0.85	9.2±0.53	4.6±0.70
	Right (PHA-p)	144.9±2.88	205.8±2.70	158.2±3.40	134.7±2.16

haemorrhage, capillary congestion, edema, and lymphocyte infiltration, consistent with a delayed-type hypersensitivity reaction (Fig. 1). These vascular and cellular responses became more pronounced at 18 h but started to decline by 27 h (Fig. 2) and further reduced at 36 h.

Humoral immunity: The humoral immune response was assessed by measuring antibody titres against the RD virus on days 0, 7, 14, 21, and 28 post-vaccinations. In the control group, the mean ± SE antibody titres were 50 ± 5.53, 74 ± 4.34, 148 ± 10.66, 116 ± 12.16, and 106 ± 10.66, respectively. In contrast, the treatment group (selenium-supplemented broilers at 15 ppm) showed titres of 44 ± 4.33, 62 ± 6.29, 92 ± 9.26, 59 ± 7.73, and 56 ± 6.96 on the corresponding days. Selenium-treated chickens exhibited significantly lower antibody titres on days 7, 14, 21, and 28 compared to the control group ($P < 0.05$ or $P < 0.01$). Antibody titres peaked at day 14 in both groups, but the peak response was markedly reduced in selenium-treated birds. This finding indicated selenium-induced suppression of the humoral immune response, which might increase disease susceptibility. Similar immunosuppressive effects of selenium have been documented in fish (O'Neill, 1981) and rats (Glaser *et al.* 1985), further supporting the present results.

These findings demonstrated that selenium induced immunosuppression in experimental birds which might increase susceptibility to infectious diseases and lower the vaccine efficacy. This observation was consistent with earlier reports by Biswas *et al.* (2006) and O'Neill (1982).

The present investigation demonstrated that sodium selenite has a very narrow margin of safety in broiler chickens. The oral median lethal dose (ALD_{50}) was determined to be 9.59 mg/kg body weight of selenium. Chronic dietary exposure at 30 ppm resulted in severe clinical illness, high mortality, growth retardation, and dose-dependent selenium accumulation in the liver. Importantly, immunological assessments revealed significant suppression of both cell-mediated and humoral immune responses, indicating marked immune-toxicity associated with chronic selenium exposure. These findings highlighted that while selenium is essential in trace amounts, its overdose compromises health, immune competence, and survival in broiler chickens. The study emphasized the need for strict regulations of dietary selenium supplementation in poultry production to maximize nutritional benefits, while preventing toxicosis and associated productivity losses.

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