Iodine as an alleviator of bromine toxicity in thyroid, liver and kidney of broiler chickens

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Abstract

The study pursued the hypothesis that bromine (Br) in drinking water at levels > 0.01 mg Br/L may have detrimental effects on the liver, kidneys and thyroid and the thyroid hormones T_3 and T_4 and that iodine (I) may alleviate the potential hazardous effect of Br. The research was done with mixed Ross broiler chickens over a 42-day post-hatch growth period. The trial design was six treatments, T1: 0 mg Br/L and 0 mg I/L; T2: 1 mg Br/L and 0 mg I/L; T3: 3 mg Br/L and 0 mg I/L; T4: 0 mg Br/L and 0.7 mg I/L; T5: 1 mg Br/L and 0.7 mg I/L; and T6: 3 mg Br/L and 0.7 mg I/L delivered via drinking water and three replicates per treatment with 30 birds per replicate. The effect of Br on T_3 and T_4 levels overall was non-significant, but T_3 and T_4 levels decreased between Weeks 4 and 6 with a significant effect at Week 6 on T_3. Br had an overall effect on the thyroid gland (P = 0.0457), liver (P = 0.0025) and kidney (P = 0.0032), and had accumulated in these three organs. Histopathological assessment showed explicit damage to the livers that received the Br treatments. Iodine (0.07 mg/L) ameliorated the negative effects of high Br (3 mg/L Br) concentration and ingestion.

Keywords: Bromide, Iodide, Water, Toxicity, Thyroid hormones

1. Introduction

Groundwater in South Africa contains a high range of natural bromide (Br) with recorded values from 0 to 18.4 mg/L (Casey and Meyer, 2001, 2006). At 18.4 mg/L Br the concentration is eight times the level of 2.3 mg/L recommended by The Safe Drinking Water Committee (1988) for a 70 kg person drinking 2 L/day and 92 times the 0.2 mg/L noted as a maximum Br level in drinking water (El'piner et al., 1972). The current South African Water Quality Guidelines (Second Edition), 1996, set 0.01 mg/L Br as the reference value for livestock (Casey and Meyer, 1996). A concentration of a water quality constituent (WQC) that exceeds the reference value is considered to be a potentially hazardous chemical constituent (PHCC). In the instances where Br exceeds 0.01 mg/L, Br is considered to be a PHCC.

The potential toxicity of Br is well documented. Vaiseman et al. (1986) reported that 96% of ingested Br can accumulate in the human body as in livestock, especially within the thyroid gland, liver and kidneys (Jolles, 1966). The accumulated concentrations of Br in livestock can

lead to excessive intakes of the mineral by humans through consumable products from livestock as via milk noted by Vreman et al. (1985). Mamabolo et al. (2009) found that broilers over a 42-day growth period exposed to 0.1 mg/L Br as NaBr with Total Dissolved Solid (TDS) < 500 mg/L, accumulated 14.89 mg/kg DM (dry matter) in the liver, 12.22 mg/kg DM in the kidney, 20.52 mg/kg DM in heart muscle, 13.96 mg/kg DM in thigh muscle and 7.70 mg/kg DM in breast muscle. The Br accumulation was significantly higher than arsenic (As administered as As_2O_3) or lead (Pb administered as $Pb(NO_3)_2$) in the same tissues under the same conditions. Mamabolo et al. (2009) further reported that TDS of 1500 mg/L increased water intake, but had a positive ameliorating effect on the accumulation of the elements in the tissues. Br concentrations were still greater than the As or Pb.

The risk of exposure to Br levels > 0.01 mg/L on livestock was reaffirmed (Du Toit and Casey, 2010). Broilers were exposed to balanced combinations of 1 and 3 mg Br/L and 0.0 and 0.7 iodine (I) mg/L through drinking water (Days 1–42). Treatments administered at 1 and 3 mg Br/L or at ingestion rates of 1.59 and 4.44 mg Br/day affected production parameters significantly. I had an effective ameliorating effect on Br. The negative effect on production parameters may reflect sub-clinical pathological conditions.

Physiological manifestations may occur following the ingestion of Br over different time periods. The biggest influence is on the production of thyroid gland hormones; the thyroid glands of rats that received high dietary Br increased significantly (Rauws and Van Logten, 1975). The goitrogenic effect of Br is further supported by Velický et al. (2004) who recorded marked changes in the morphology of the thyroid gland and reduced iodide accumulation in the thyroid gland, as well as the skin in rats fed a diet containing a high concentration of Br.

Hellerstein et al. (1960) reported that species differences of Br concentrations in tissues were small and that Br does not accumulate in a particular organ or tissue. Cole and Patrick (1958) and Jolles (1966) on the other hand did report relative proportions of Br in various organs 2 h after intraperitoneal administration of 50 microcuries (μc) ^{82}Br as KBr. The relative proportions were 1.000; 0.536; 0.320; 0.394; 0.131; and 0.602 for the thyroid gland; kidney; adrenals; liver; brain and blood (Jolles, 1966). Langley (1958) reported that the biological half-life of Br could be decreased by the administration of surplus I ions.

The study pursued the hypothesis that Br in drinking water at concentrations exceeding the guideline value (0.01 mg Br/L) may have a detrimental effect on the liver, kidneys and thyroid and the thyroid hormones T_3 and T_4 in broilers over a 42-day growth period, and that I in the drinking water may alleviate the potential hazardous effect of Br.

2. Materials and methods

2.1. Ethics approval

Procedures for this trial were approved by the University of Pretoria Animal Use and Care Committee (Reference EC080805-032).

2.2. Trial design

The statistical design was a 3 × 2 factorial design with three levels of Br (0, 1 and 3 mg/L) and two levels of iodine (I) (0 and 0.7 mg/L) with three replicates per treatment and thirty Ross broilers of mixed sex per replicate. The duration was 42 days post-hatch. Water intake (WI), weight gain and feed intake (FI) were recorded weekly for each replicate. The treatments were designated

> T1: 0 mg Br/L and 0 mg I/L; T2: 1 mg Br/L and 0 mg I/L; T3: 3 mg Br/L and 0 mg I/L; T4: 0 mg Br/L and 0.7 mg I/L; T5: 1 mg Br/L and 0.7 mg I/L; and T6: 3 mg Br/L and 0.7 mg I/L.

2.3. Animal husbandry

The housing, management and nutrient composition of the starter (ME/kg DM 11.2), finisher (ME/kg DM 11.4) and post-finisher (11.6 ME/kg DM) broiler diets were as described by du Toit and Casey (2010). Water was delivered from graduated cylinders via bell drinkers for accurate measuring of water intake. The trace element premix contributed 0.001 g/kg I to the diets, and 0.0 g/kg Br.

2.4. Treatments

Selection of 1 and 3 mg Br/L was by considering these concentrations of PHCC against the recommended level 0.01 mg/L (Casey and Meyer, 1996). Since in many rural settings people use the same water source as the livestock the corollary to the opinion of McKee and Wolf (1963) that water safe for human consumption may presumably be used safely by livestock should be considered. The physiological responses of livestock to PHCC may be indicators of the risk to people.

The administration of 0, 1 and 3 mg Br/L was to verify the recommended levels of Br at 2.3 mg/L (The Safe Drinking Water Committee, 1988), 0.2 mg/L noted as a maximum Br level in drinking water (El'piner et al., 1972) and 0.01 mg/L (Casey and Meyer, 1996). The possible alleviator effect of I for these high Br levels was tested with 0.7 mg I/L since this concentration is within the Target Water Quality Range for I (0–1 mg/L). Broiler chickens were used as the biological model.

Water from the Pretoria Municipal Source was used. Water samples of the treatments and the control were analysed every week to monitor Br and I levels by ICP-AES techniques. Separate sample bottles were used for macro- and trace elements during these procedures. The analyses were done against standards at the accredited Institute for Soil, Climate and Water.

The treatments delivered final concentrations of 1 mg Br/L and 3 mg Br/L as sodium bromide (NaBr), and 0.7 mg I/L as potassium iodide (KI). The final concentrations were confirmed by testing the samples at the point of use.

2.5. Physiological parameters

At Days 16 and 42 blood samples were taken from five birds of each replicate before the birds were slaughtered in the conventional way by experienced personnel at the abattoir on the Hatfield Research Farm. The thyroid, liver and kidneys were collected for analysis.

Tissue samples of two chickens from each replicate were taken and stored in polyethylene bags at − 20 °C. These were analysed by standard inductively coupled mass spectrometry (ICP-MS) techniques to determine trace element concentrations.

The thyroid gland, liver and kidney samples were collected from two chickens of each replicate of treatments T3: 3 mg Br/L and 0 mg I/L and T6: 3 mg Br/L and 0.7 mg I/L for histopathological assessments by a clinical pathologist. The samples were placed in buffered formalin. The tissues were block selected and processed in an automated histological tissue processor before specific wax blocks were produced. Sections of 5 µm were cut on a microtome and the tissue slides were stained with Haematoxylin and Eosin staining in an automated histological stainer. The histopathological assessments were graded as no observable organ damage (−), mild organ damage (1 +), moderate organ damage (2 +) and severe organ damage (3 +), where damage is a deviation from normal morphology.

The serum of blood samples from two chickens of each replicate was analysed for free T_3 and T_4 concentration by Chemiluminescence Enzyme Immunoassay (CLIA) (Kricka et al., 1987).

In the T_3 CLIA, as explained by Rongen et al. (1994), a certain amount of anti-T_3 antibody was coated on micro titer wells. A measured amount of chicken serum and a constant amount of T_3 conjugated with horseradish peroxidase were added to the micro titer wells. During incubation, T_3 in the samples and conjugated T_3 competed for the limited binding sites on the anti-T_3 antibody of the wells. After 60 min of incubation at room temperature, the wells were washed 5 times by wash solution to remove unbound T_3 conjugate. A solution of chemiluminescent substrate was then added and relative light units in a Luminometer were read. The intensity of the emitting light was proportional to the amount of enzyme present and was inversely related to the amount of unlabelled T_3 in the sample. By reference to a series of T_3 standards assayed in the same way, the concentration of T_3 in the unknown sample was quantified (Rongen et al., 1994). The precise method for T_3 was repeated for T_4.

2.6. Statistical evaluation

The GLM procedure of the SAS (Statistical Analyses System®) software system was used in the statistical analysis. The significance of differences between treatments and exposure periods was established by means of the Fisher's test at $P < 0.05$.

3. Results

T_4 hormone levels between treatments within Week 4 and Week 6 did not differ significantly, although the 3 mg Br/L inclusion levels (Treatments 2 and 5) showed slightly lower T_4 hormone levels (Table 1).

Table 1. Average T_4 hormone levels (pmol/L) (means and SD) of broilers with the different bromine (Br), iodine (I) levels (mg/L) in treatments T1 to T6 compared within weeks 4 and 6.

Week	Treatments						Means	P-value
	T1 0 Br + 0 I	T2 1 Br + 0 I	T3 3 Br + 0 I	T4 0 Br + 0 .7 I	T5 1 Br + 0.7 I	T6 3 Br + 0.7 I		
4	6.27 (0.89)	6.38 (1.54)	5.42 (0.28)	5.52 (0.40)	5.77 (0.73)	6.50 (0.60)	5.98 (0.91)	0.7412
6	6.00 (1.09)	5.59 (0.62)	5.29 (0.33)	5.89 (0.75)	5.80 (0.24)	5.10 (0.00)	5.61 (0.63)	0.5360

Differences determined at $P < 0.05$ according to the Fischer's Test.

The treatments had no effect on T_3 hormone at Week 4 ($P = 0.2503$), but the effect became significant at Week 6 ($P = 0.0010$). Within week 6, the increasing level of Br (T2 to T3) significantly reduced T_3 hormone levels. I ($P = 0.0273$), irrespective of Br, as well as the interaction between I and Br ($P = 0.0019$) had an ameliorating effect on the T_3 hormone levels (T6 compared with T3) (Table 2).

Table 2. Average T_3 hormone levels (pmol/L) (means and SD) of broilers with the different bromine (Br) and iodine (I) levels (mg/L) in treatments T1 to T6 compared within weeks 4 and 6.

Week	Treatments						Mean	P-value
	T1 0 Br + 0 I	T2 1 Br + 0 I	T3 3 Br + 0 I	T4 0 Br + 0.7 I	T5 1 Br + 0.7 I	T6 3 Br + 0.7 I		
4	3.33 (0.94)	3.55 (0.74)	4.60 (1.53)	3.27 (0.32)	4.10 (0.30)	3.37 (1.46)	3.70 (0.89)	0.2503
6	2.20^{b} (0.30)	2.78^{a} (0.50)	2.17^{b} (0.16)	2.07^{b} (0.35)	2.55^{a} (0.28)	2.53^{a} (0.33)	2.38 (0.18)	0.0010

^{ab} Row means with different superscripts within weeks differ ($P < 0.05$) according to the Fischer's Test.

Br treatments affected the Br content of the thyroid gland, ($P = 0.0457$), liver ($P = 0.0025$) and kidney ($P = 0.0032$). Br treatments affected the I content of the liver ($P = 0.0235$) and kidney ($P = 0.0141$), but not the I content of the thyroid gland ($P = 0.3950$). T4 (0.0 mg/L Br + 0.07 mg/L I) recorded the lowest Br levels in the thyroid gland ($P = 0.0467$), liver ($P = 0.0025$) and kidney ($P = 0.0032$) and T3 (3 mg/L Br + 0 mg/L I) recorded the highest levels of Br within the three organs (Tables 3, 4 and 5). The Br accumulated within T6 (3 mg Br/L and 0.7 mg I/L) was less than the Br content accumulated within T3 (3 mg/L Br + 0 mg/L I). Although the Br treated groups reported the lowest I accumulation within the thyroid gland, the differences were not significant (Tables 3, 4 and 5). A comparison between the organs shows that the kidney accumulated more Br than the thyroid gland or liver.

Table 3. Average bromine (Br) and iodine (I) (mg/kg DM) (means and SD) in the thyroid gland of broilers exposed to different bromine (Br) and iodine (I) levels (mg/L) in treatments T1 to T6 over a 42-day growth period.

Element	Treatments						Mean	P-value
	T1 0 Br + 0 I	T2 1 Br + 0 I	T3 3 Br + 0 I	T4 0 Br + 0.7 I	T5 1 Br + 0.7 I	T6 3 Br + 0.7 I		
Br	12.52^{ab} (11.25)	28.87^{ab} (33.48)	54.16^{b} (90.43)	8.46^{a} (7.50)	24.67^{ab} (18.85)	39.32^{ab} (37.65)	27.80 (38.04)	0.0457
I	2.70 (6.37)	0.18 (0.10)	0.17 (0.12)	0.63 (1.20)	0.26 (0.13)	0.24 (0.14)	0.70 (2.60)	0.3950

^{ab} Row means with different superscripts differ (P < 0.05) according to the Fischer's Test.

Table 4. Average bromine (Br) and iodine (I) (mg/kg DM) (means and SD) in the liver of broilers exposed to different bromine (Br) and iodine (I) levels (mg/L) in treatments T1 to T6 over a 42-day growth period.

Element	Treatments						Mean	P-value
	T1 0 Br + 0 I	T2 1 Br + 0 I	T3 3 Br + 0 I	T4 0 Br + 0.7 I	T5 1 Br + 0.7 I	T6 3 Br + 0.7 I		
Br	12.04^{ab} (11.92)	32.11^{abc} (27.56)	95.59^{c} (128.41)	6.18^{b} (5.36)	17.75^{ab} (20.68)	77.58^{ac} (104.19)	40.21 (56.57)	0.0025
I	0.27 (0.15)	0.35 (0.18)	0.28 (0.14)	0.26 (0.16)	0.33 (0.14)	0.21 (0.12)	0.28 (0.13)	0.0235

^{abc} Row means with different superscripts differ (P < 0.05) according to the Fischer's Test.

Table 5. Average bromine (Br) and iodine (I) (mg/kg DM) (means and SD) in the kidneys of broilers exposed to different bromine (Br) and iodine (I) levels (mg/L) in treatments T1 to T6 over a 42-day growth period.

Element	Treatments						Means	P-value
	T1 Br + 0 I	T2 1 Br + 0 I	T3 3 Br + 0 I	T4 0 Br + 0 .7 I	T5 1 Br + 0.7 I	T6 3 Br + 0.7 I		
Br	32.08^{a} (40.69)	102.86^{ab} (108.87)	210.05^{b} (320.61)	22.46^{a} (22.37)	71.85^{ab} (65.25)	161.02^{ab} (196.65)	100.05 (130.12)	0.0032
I	0.30^{ab} (0.19)	0.42^{ab} (0.22)	0.26^{b} (0.16)	0.28^{b} (0.13)	0.45^{a} (0.10)	0.29^{ab} (0.16)	0.33 (0.14)	0.0141

^{ab} Row means with different superscripts differ (P < 0.05) according to the Fischer's Test.

The accumulated I levels were the highest in the thyroid gland and less in the liver and kidney.

The histopathology assessments (Tables 6 and 7) indicated a difference between the liver morphology of T1 compared to the livers from both T6 and T3. The hepatocellular hypertrophy appeared to be most severe in T3 and less prominent among T6. Vacuolar degeneration was due to swelling of the intracytoplasmic endoplasmic reticulum and may have followed on different forms of damage to the hepatocytes. It tended to show the same pattern as found in hepatocellular hypertrophy recorded in the liver sections. Fatty change, characterized by round fat droplets within the cytoplasm, did not show any specific pattern among the treatment groups or the control birds. The kidney and thyroid gland appeared

similar without any specific pathological changes among the treatment groups or in the control birds.

Table 6. Histopathological lesions in liver sections from broilers exposed (mg/L) to T1 (0 Br + 0 I), T3 (3 Br + 0 I) and T6 (3 Br + 0.7 I) at Week 4 of the growth period.

Hepatic lesion	T1 0 Br + 0 I			T3 3 Br + 0 I			T6 3 Br + 0.7 I		
	Sample 1	Sample 2	Sample 3	Sample 1	Sample 2	Sample 3	Sample 1	Sample 2	Sample 3
Hepatocellular hypertrophy	–	–	–	2 +	3 +	1 +	1 +	–	1 +
Vacuolar degeneration	–	–	–	3 +	3 +	2 +	1 +	1 +	1 +
Fatty change intracytoplasmic	–	–	–	–	–	–	–	–	1 +

– No observable organ damage; 1 + mild organ damage; 2 + moderate organ damage; 3 + severe organ damage.

Table 7. Histopathological lesions in liver sections from broilers exposed (mg/L) to T1 (0 Br + 0 I), T3 (3 Br + 0 I) and T6 (3 Br + 0.07 I) at Week 6 of the growth period.

Hepatic lesion	T1			T3			T6		
	Sample 1	Sample 2	Sample 3	Sample 1	Sample 2	Sample 3	Sample 1	Sample 2	Sample 3
Hepatocellular hypertrophy	–	–	–	2 +	3 +	2 +	–	1 +	–
Vacuolar degeneration	–	–	–	2 +	3 +	2 +	–	1 +	–
Fatty change intracytoplasmic	–	–	–	3 +	1 +	1 +	1 +	1 +	1 +

– No observable organ damage; 1 + mild organ damage; 2 + moderate organ damage; 3 + severe organ damage.

A comparison of the results of Weeks 4 and 6 (Table 6) shows that the damage due to T6 is similar at both Week 4 and Week 6 (Table 7). T6 had no observable organ damage at week 4 for intracytoplasmic fatty change, but at Week 6 severe to mild organ damage was recorded.

4. Discussion

Ingestion rates need to be calculated when water with a WQC that is a PHCC such as Br is encountered. This is important as it is the actual amount of PHCC ingested that is relevant, and not the concentration in the water alone. The cumulative intake of Br (mg/bird/day) over the 42-day growth period was 1.591 (T2), 4.442 (T3), 0.093 (T4), 1.567 (T5) and 4.618 (T6) as published by du Toit and Casey (2010).

The results reiterate the accumulation of levels of Br in tissues (heart, thyroid gland, liver, thigh and breast muscle) of poultry exposed to drinking water with Br content > 0.01 mg/L that Casey and Meyer (2006) recorded in chickens raised under extensive, free-range

production conditions where the chickens had access to groundwater with high Br content (> 0.01 mg/L). However, they had not investigated the specific effect of Br on the thyroid hormones T_3 and T_4. The results imply that breast and thigh muscle cannot be excluded as a risk of transmitting Br to consumers. Concerns are raised in particular where in rural production systems through slower growth of chickens and longer exposure and high total ingestion of Br (including free-ranging layers) and subsequent accumulation in tissues, Br may be carried over to people via the meat and organs. The risk is increased further when people and their chickens drink water from the same groundwater source that contains high levels of Br.

Jolles (1966) reported that after 2 h of Br administration the proportional amounts of Br accumulated within the thyroid gland was 1, for the kidney it was 0.5 and the liver 0.4. This indicates that the highest accumulation of Br should have occurred within the thyroid gland, but in this trial the highest accumulation occurred within the kidney. On the other hand, Abelin and Poretti (1952), Bosshardt et al. (1956), Huff et al. (1956), Mack and Shipley (1952) and Winnek and Smith (1937) could demonstrate no preferential uptake by the thyroid gland. Cole and Patrick (1958) measured the kidney and intestines as the most active organs to have taken up ^{82}Br after a 2 hour intraperitoneal administration. Hellerstein et al. (1960) reported that Br accumulation does not occur in any particular organ or tissue. Abelin and Poretti (1952) and Winnek and Smith (1937) showed through chemical analyses that supplemented dietary Br increased Br levels in the blood, liver, kidney, adrenals, muscle, spleen and the brain. Golomb (1999) also reported that because of slow excretion of bromide through the kidneys, Br may accumulate in the body when continuously ingested. Masoud et al. (1973) reported acute renal failure cases leading to death by a direct nephrotoxic effect of the bromate ion. These reports emphasize the potential problems arising with Br in water at concentrations > 0.01 mg/L.

The long half-life of bromide is 14 to 94 h for the thyroid gland, 88 to 235 h for the liver and 22 to 197 h for the whole body (Pavelka et al., 1999). Frances et al. (2003) reported a 10-day half-life elimination period for Br in the blood. The prolonged half-life in the thyroid gland, liver, whole body and blood indicate why the vast quantities of Br accumulation were seen in these three organs. The I concentrations within all three of the tested organs decreased as Br treatments increased, but were non-significant. The I concentrations for the kidney were less than for the other two organs, indicating the ameliorating effect of I on the kidney. This observation can be explained by Pavelka et al. (2002) as high levels of Br intake influence the I metabolism of the animal by increasing the iodide excretion by the kidneys. Fortunately for a 42-day production period I did alleviate the effect of Br not to the expense of I. The I concentrations were the highest for the thyroid gland and less for the liver and kidney. This is a typical observation as reported by Perlman et al. (1941), as the thyroid gland has 100 times the ability of the liver and the kidney to retain I.

The histopathological assessment established explicit damage to the livers that received the elevated Br treatments; severe damage in the form of hepatocellular hypertrophy and vacuolar degeneration were reported. It was also clear that the alleviator element (I) compensated for the damage caused by Br, by comparing the mild damage in T3 with the more severe damage in the T6. A longer exposure period can possibly indicate significant I reduction within the thyroid gland, liver and kidney. At 6 weeks of age the damage had

augmented and a new symptom, intracytoplasmic fatty change was reported for both of the treatment groups, where the elevated treatment group was moderately damaged and the alleviator group mildly damaged. It was clearly stated in the histopathology report that the factor of time did play a role in the degeneration of the histopathology of the organs receiving high Br treatments.

Although the highest Br concentration was found to be within the kidney, the liver was most severely damaged according to the histopathology report. A longer exposure period might have indicated histopathological severity to the thyroid gland and the kidney. A study by Velický et al. (2004) noted the adverse effect Br had on the ultra-structure of rat thyroid glands. The administration of 10, 50 and 100 mg Br/L for 16 or 60 days resulted in marked hypertrophy and hyperplasia of the thyroid gland, micro follicular rearrangement and lower colloid volume. A sub-clinical condition due to Br could have had a detrimental effect on the chickens as shown by the effect on production parameters (Du Toit and Casey, 2010) that might be observed clinically after a longer period of exposure. The concern with a sub-clinical condition is that the lack of observable adverse effects can create the impression that the water has no possible hazardous effect, i.e. no PHCC. The same obscured risk would apply to animal products for human consumption as very high levels of Br accumulated within the three observed organs, the thyroid gland, liver and kidney.

5. Conclusions

The results obtained in this study indicate that the current guideline level of 0.01 mg/L Br in drinking water may not be too restrictive. The trial confirmed that concentrations of the PHCC (Br) in the drinking water of broilers can accumulate to concentrations that exceed a maximum acceptable concentration of 1 mg Br/L in animal tissues used for human consumption, regardless of a short exposure period. Explicit histopathological damage was caused in the livers of broilers that received the elevated Br treatments of 1 and 3 mg Br/L, or the equivalent ingestion levels of 1.59 and 4.44 mg Br/L per bird per day. It is shown that I has a potential ameliorative effect on Br toxicity in broilers.

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