Research Article

Sodium hypochlorite in water treatment: Toxicity-induced oxidative stress in male albino rats

Beno O. Onunkwor¹, David O. Babayemi¹, Regina N. Ugbaja¹, Adio J. Akamo¹, Patience O. Basorun¹, Opeoluwa Awonubi²

¹Department of Biochemistry, College of Biosciences, Federal University of Agriculture, Abeokuta.
²Pharmaceutical Technology, School of Science and Technology, Moshood Abiola Polytechnic, Ojere, Abeokuta.

ABSTRACT: Chlorination is a common method used in domestic water treatment. However, there are concerns about its safety. This study investigated the probable toxicological effects associated with prolonged oral administration of sodium hypochlorite (NaOCl), a domestic water disinfectant. Thirty-two male albino rats (180-220g) were evenly segregated into four groups of eight rats each. Group 1 (control) were orally administered 100 µl physiological saline, Group 2-4 were orally administered 50 µl, 100 µl and 150 µl/kg body weight of 1.0% NaOCl respectively bi-daily for 12 weeks. This was followed by the assay for indices of oxidative stress, as well as hepatic and renal dysfunctions. The results showed significant (p<0.05) dose dependent elevations in activities of catalase, superoxide dismutase, xanthine oxidase and lactate dehydrogenase, and level of lipid peroxidation. Plasma activities of aspartate and alanine transaminases as well as urea and creatinine concentrations were significantly increased (p<0.05) suggesting liver and kidney compromise. The present study suggests that the use of NaOCl in treating water domestically may present with subtle deleterious biochemical effects.

KEYWORDS: Domestic water treatment, Chlorination, Sodium hypochlorite, Oxidative stress.

Correspondence: Beno Onunkwor; benovinadict@gmail.com; +23408068618104

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INTRODUCTION

Drinking water, also known as potable water, is water that is safe to drink or use for food preparation. The quest for potable water has been in existence for as long as man could distinguish odour, taste, cloudiness, and colours in available water (Ibrahim et al., 2016).

Countries throughout the world are concerned with the effects of unclean drinking water because water-borne diseases are a major cause of morbidity and mortality (Clasen et al., 2007; WHO, 2010). Clean drinking water is important for overall health and plays a substantial role in infant and child health and survival (Anderson et al., 2002; Fewtrell et al., 2005; Vidyasagar, 2007).

Despite numerous efforts by government at various levels and other agencies interested in water and its safety, waterborne diseases are still a major public health and environmental concern (Nwabor et al., 2016). Access by households to sufficient and safe water could result in a substantial reduction of the deaths due to diseases associated with unsafe water (WHO, 1992). In developing countries, poor water quality is the leading cause of health problems. Over eighty percent of all illnesses in developing countries are caused by polluted water (WHO, 2008), hence the utmost need for water treatment. Olukanni et al. (2014) submitted that before water can be totally considered safe for drinking, further treatment is required at the household level. In the rural areas where there are no accesses to potable water, the quantity of water that needs to be treated is the quantity required for drinking and preparing uncooked foods (Ibrahim et al., 2016).

Because drinking water is one of the important sources of transmission of a variety of infectious diseases, disinfection of drinking water is the primary concern for water treatment which plays a pivotal role in the control of water-borne infectious diseases. Chlorine compounds have broad-spectrum antimicrobial activity against bacteria, fungi, and viruses and are fast acting (Hamdullah et al., 2010) and as a result, the addition of germicides in drinking water has been an effective measure to control many infectious diseases (Khan et al., 2008). The bactericidal effect of chlorinated water is based on the penetration of the chemical and its oxidative action on essential enzymes in the cell (Hamdullah et al., 2010). Sodium hypochlorite (NaOCl), a halogenated compound, is the best example of a chlorine compound used as a water disinfectant. The use of NaOCl in household water treatment has been recognized as a cost-effective means of reducing the heavy burden of bacteria, leading to diarrhoea and other waterborne diseases, especially among populations without access to improved water supplies (Claesen and Edmondson, 2006).

Sodium hypochlorite solutions release free chlorine and hypochlorous acid, which are known to be very active in killing most of the bacteria, fungi, and viruses (Abuhaimed, and Abou Neel 2017; Hamdullah et al., 2010).

Hypochlorite ion, a product of sodium hypochlorite, is a highly destructive, selective oxidant, which reacts avidly with all biomolecules and is able to oxidize nucleotides, inactivate enzymes and electron transport systems, disrupt cell membranes and fragment proteins which lead eventually to cell death (Hidalgo et al., 2002). In living organisms, hypochlorite is a useful biomolecule synthesized from hydrogen peroxide and chlorine ions in a chemical reaction catalysed by the enzyme myeloperoxidase (MPO) secreted by activated phagocytes in zones of inflammation (Furtmüller et al., 2000). Hypochlorous acid is a key microbicidal agent, used as a natural defence owing to its great potency as a nucleophilic non-radical oxidant and its efficacy lies in the fact that neither bacteria nor mammalian
cells can counteract its toxic effect since they lack the enzymes required for its catalytic detoxification (Hidalgo et al., 2002).

Hypochlorite is one of the most aggressive oxidants amongst the reactive oxygen species (Liu et al., 2017). Reactive oxygen species are chemically reactive molecules and free radicals generated from molecular oxygen that, if produced in excess, cause damage to tissues and different components of the cells. Yet, if produced in physiological balance, reactive oxygen species have been shown to play a principal role in normal cell signal transduction pathways, including apoptosis, gene expression, and activation of different cell signalling cascades. Hypochlorous acid (HOCl) is known to react with primary amines and other N-compounds to rapidly yield chloramines and nitrogen–chlorine derivatives (Hidalgo et al., 2002). Hypochlorous acid generates superoxide radicals that cause oxidative injury and cell death (Peck et al., 2011).

Sodium hypochlorite may be associated with deleterious biochemical effects particularly on prolonged use especially among the unsuspecting populations, who employ its use domestically to make potable drinking water available. Hence, a contemporary issue of biochemical, pathophysiological, toxicological and clinical concern, attracting critical attention towards the possible subtle effects associated with NaOCl. The present study focused on ascertaining the effects of sodium hypochlorite on certain biologic indices of toxicity, including oxidative stress, liver, and kidney function tests in male albino rats.

**MATERIALS AND METHODS**

**Chemicals**

Sodium hypochlorite (1.0% NaOCl was obtained from local pharmaceutical store ready for use). Glutathione, 1- chloro- 2,4, dinitrobenzene (CDNB) 5, 5-dithiobis (2-nitrobenzoic acid) (DTNB), hydrogen peroxide, pyrogallol, and hypoxanthine were products of Sigma-Aldrich Missouri, USA. Lactate dehydrogenase, alanine transaminase (ALT), aspartate transaminase (AST), blood urea nitrogen (BUN) and creatinine kits were purchased from cypress diagnostics, Belgium.

**Animals**

All conditions of animal experimentation conformed to the NIH guidelines as outlined in NIH publication (National Institutes of Health, 2011), and were approved by the Animal Ethical Committee of Biochemistry Department, Federal University of Agriculture, Abeokuta, Nigeria.

Thirty-two male Wistar rats (bred in the Faculty of Veterinary Medicine, University of Ibadan, Ibadan Nigeria) with a mean body weight of 200g were used for the investigations. They were maintained at 25°C in a well-ventilated animal house on a 12:12 hour light-dark cycle with free access to standard rodent laboratory chow and clean water ad libitum. The rats were acclimatized for two weeks prior to experimental treatments.

**Experimental design**

Animals were randomly and evenly segregated into four groups each with 8 rats: Group one served as control, orally administered 100µl physiological saline; Groups two to four were administered: 50µl, 100µl, and 150µl of 1.0 % NaOCl respectively. Treatments were orally administered bi-daily for 12 weeks, using oral gavage. At the end of NaOCl treatments, all the animals were fasted overnight, subjected to light anaesthesia and then sacrificed. Blood samples were collected via cardiac puncture.
into precooled heparinized tubes. An aliquot of each whole blood sample was kept for some immediate oxidative stress assays while the remaining blood samples were centrifuged to separate plasma and the erythrocytes. All samples not immediately used for assay were stored at -20°C until analysed.

**Superoxide dismutase activity assay**

Superoxide dismutase was determined according to the modified Marklund and Marklund (1974) method of Xican, (2012). This method is based on inhibition of pyrogallol (1, 2, 3–benzenetriol) by superoxide dismutase at 420 nm.

100 µl of potassium phosphate buffer (0.05M; pH 7.4), 830 µl of distilled water was pipetted into each of two test tubes ‘B’ and ‘T’. 50 µl of the phosphate buffer was added to tube ‘B’, while 50 µl of diluted whole blood was added to tube ‘T’. The tubes were incubated for 10 minutes at 25°C and 20 µl of 0.01 M pyrogallol (prepared in 0.01 M HCl) was added to both tubes. They were mixed by inversion and absorbance was recorded at 30 seconds intervals for 5 minutes. One unit is equal to one micromole of pyrogallol inhibited per minute under specified conditions at 25°C. The activity of SOD is expressed as units/ml.

**Catalase (CAT) activity assay**

The catalase activity was measured by the method of Beers and Sizers (1952) in which the rate of decomposition of H₂O₂ was measured spectrophotometrically from changes in absorbance at 240 nm. 2.0 ml of phosphate buffer (0.05 M, pH 7.0) diluted whole blood was added to the cuvette. The reaction was initiated by the addition of 1.0 ml of freshly prepared 59 mM H₂O₂ and decreasing absorbance read for 1 minute at 10 seconds interval. One unit is equal to one micromole of hydrogen peroxide decomposed per minute under specified conditions at 25 °C. The activity of CAT is expressed as units/ml.

**Estimation of lipid peroxidation**

Lipid peroxidation in plasma was estimated colorimetrically by thiobarbituric acid reactive substances (TBARS) method of Buege and Aust (1978). In brief, 0.1 ml of the test sample (plasma and red blood cell) was treated with 2.0 ml of TBA-TCA-HCl, 1:1:1 reagent (thiobarbituric acid 0.37%, 0.25 N HCl and 15% TCA) and incubated in a water bath at 95 °C for 15 min. The tube was then placed on ice, centrifuged and the absorbance of the clear supernatant was measured against blank at 535 nm. TBARS (malondialdehyde MDA) content was determined using the extinction coefficient of 155 nM⁻¹cm⁻¹.

The plasma concentration of lactate dehydrogenase, alanine transaminase (ALT), aspartate transaminase (AST), blood urea nitrogen (BUN) and creatinine were determined spectrophotometrically using Cypress diagnostic kits.

**Statistical analysis**

The results obtained are expressed as mean ± standard error of mean (SEM). One-way analysis of variance (ANOVA) test was performed to check the significance of differences among the groups followed by Duncan’s post hoc test to compare the treated groups with the control group. Values of p<0.05 were regarded as significant, using the Statistical Package for Social Sciences (SPSS) version 20.0.
RESULTS

Superoxide dismutase (SOD) and catalase (CAT) activities in whole blood

Figure 1 depicts the SOD and CAT activities in whole blood of rats treated with 1.0% NaOCl. SOD activity was lowest amongst the control group (140.77 ± 3.69 U/ml). The 100 µl and 150 µl NaOCl treated groups 3 and 4 (179.95 ± 5.25 U/ml and 181.40 ± 5.72 U/ml respectively) had SOD activities that were significantly (p<0.05) higher than the control group by 22%.

An almost similar trend in SOD activity was observed for the CAT activity among the four experimental groups. The 50 µl, 100 µl, and 150 µl NaOCl treated groups (136.10±2.5 U/ml, 137.52 ± 2.92 U/ml and 140.84 ± 3.40 U/ml respectively) showed an average of 18% significantly (p<0.05) increased activities when compared with the control group (113.20 ± 2.71 U/ml).

Lipid peroxidation in plasma

A dose-dependent significant (p<0.05) increase was observed amongst NaOCl treated groups 2 to 4 (0.16 ± 0.01, 0.32 ± 0.01 nM, and 0.41 ± 0.02 nM) on comparing with the control value (0.09 ± 0.01 nM) as represented in Figure 2. The 50 µl, 100 µl, and 150 µl NaOCl treated groups had 1.7, 3.5, and 4.4 folds higher plasma lipid peroxidation than the control group.

Figure 1: Whole blood superoxide dismutase and catalase activities of albino rats exposed to sodium hypochlorite. Each bar represents Mean ± SEM of 8 rats. Bars having different letters are significantly different at p< 0.05.
Figure 2: Plasma lipid peroxidation pattern of albino rats exposed to sodium hypochlorite. Each bar represents Mean ± SEM of 8 rats. Bars having different letters are significantly different at p< 0.05.

Figure 3: Plasma xanthine oxidase activity of albino rats exposed to sodium hypochlorite. Each bar represents Mean ± SEM of 8 rats. Bars having different letters are significantly different at p< 0.05.
Xanthine oxidase activity in plasma

The result of plasma xanthine oxidase (Figure 3) presented significant (p<0.05) 26% increased activity among the 50µl NaOCl treated group (1.1163 ± 0.04 U/dl) while both the 100 µl and 150 µl NaOCl treated groups had 44% elevated activities (1.4488 ± 0.03 U/dl and 1.5138 ± 0.03 U/dl respectively) compared to the control group (0.8300 ± 0.67 U/dl).

Lactate dehydrogenase (LDH) activity in plasma

The Plasma LDH activity of groups 2: (30.36 ± 2.01 U/L); 3: (42.61 ± 2.04 U/L) and 4: (46.42 ± 3.76 U/L) fed with NaOCl were significantly higher (p<0.05) than that of the control group (21.66 ± 1.83). The 50µl NaOCl treated group 2 had an activity that was 1.4 fold higher while the 100 µl and 150 µl NaOCl treated groups 3 and 4 showed 35.6% increase in LDH activity upon comparing with the control group as depicted in Figure 4.

![Figure 4: Plasma lactate dehydrogenase activity of albino rats exposed to sodium hypochlorite. Each bar represents Mean ± SEM of 8 rats. Bars having different letters are significantly different at p< 0.05.](image-url)
Alanine transaminase (ALT) and Aspartate transaminase (AST) activities in plasma

Figure 5 expresses the Alanine transferase activities, which were significantly higher (p<0.05) in 50 µl, 100 µl, and 150 µl NaOCl treated groups 2 (by 1.5 fold), 3 (by 2.6 folds) and 4 (by 2.3 folds respectively) than that of the control rats.

The Plasma AST activities (figure 5) was lowest among the control group (26.16 ± 7.51 U/L), followed by 50 µl NaOCl treated group 2 (29.92 ± 11.06 U/L) with a non-significantly (p>0.05) elevated activity compared to the former. Groups 3 and 4 however, had a 36% increased activity that was significantly higher (p<0.05) than that of the control group.

Creatinine and blood urea nitrogen (BUN) concentrations in the plasma

Comparing the control rats (1.19 ± 0.21 mg/dl) with the three groups treated with NaOCl, the concentration of creatinine in the plasma of rats in all the latter were significantly increased. Group 2 (1.61 ± 0.10) by 26 % while groups 3 and 4 (2.52 ± 0.10 and 2.58 ± 0.10 respectively) had the highest concentration increases of 52% represented in figure 6 chart.

The urea result as depicted in figure 6, showed that, of the three groups treated with NaOCl, only the 150 µl NaOCl treated rats (27.82 ± 1.50) had a significant increase (p<0.05) by 21% in the urea concentration when compared with the control group that had the mean value of 22.13 ± 0.55. The urea concentrations of the 50 µl and 100 µl NaOCl treated groups (26.51 ± 1.30 and 27.82 ± 1.50 respectively) were non-significantly increased (p<0.05).
DISCUSSION

Contaminated water causes epidemic diseases (Gul et al., 2009; Nwabor et al., 2016) and there are worries about the effects of unclean drinking water because water-borne diseases are a major cause of morbidity and mortality (Clasen et al., 2007, WHO, 2010). Clean drinking water is important for overall health (Anderson et al., 2002; Fewtrell et al., 2005; Vidyasagar, 2007).

To prevent water-based diseases, chlorine compounds are added to water and are present in most disinfected drinking water at concentrations of 0.2 – 1 mg/l (Gul et al., 2009). Due to water treatment, the proportion of households that use unclean drinking water source has declined throughout the less developed part of the world (Nwabor et al., 2016). However, the use of chlorination especially NaOCl in the treatment of water may predispose and or expose unsuspecting individuals to subtle deleterious effects. Guzzella et al., (2004) reported that drinking chlorinated water greatly affects the proliferation and growth system.

In this study, oral administration was a simulation of the main route of NaOCl ingestion as a disinfectant in drinking water. The whole blood result of rats fed NaOCl solution, showed significant (p<0.05) increase in the activity of superoxide dismutase and catalase.

Superoxide dismutase (SOD) is a metalloprotein and is the first enzyme involved in the antioxidant defence that catalyzes the dismutation of superoxide to oxygen and hydrogen peroxide thereby
lowering the steady-state level of O$_2$ (Sapakal et al., 2008). Superoxide is highly reactive, has a short half-life, cannot cross the cell membrane, and is therefore acted on by the scavenging enzyme, superoxide dismutase (SOD), which converts it to hydrogen peroxide (H$_2$O$_2$). The findings of this study showed a significant increase in SOD activity suggestive of superoxide radical generation, which is a potent and imminent oxidative stressor that needed to be neutralized and made more water soluble for onward detoxification.

Hydrogen peroxide (H$_2$O$_2$) is more stable compared to superoxide and it diffuses through the lipid bilayer. It is further acted upon by another scavenging enzyme-catalase that works synergistically with SOD. CAT is a hemeprotein, localized in the peroxisomes or the microperoxisomes. This enzyme catalyses the decomposition of H$_2$O$_2$ to water and oxygen and thus protecting the cell from oxidative damage by H$_2$O$_2$ and OH$^-$ radical. CAT is a key component of the antioxidant defense system. Inhibition of these protective mechanisms results in enhanced sensitivity to free radical-induced cellular damage (Joshi et al., 2013; Kilikdar et al., 2011; Sapakal et al., 2008). A concomitant significant increase (p<0.05) in CAT activity due to NaOCl exposure was also observed, further indicating that oxygen radicals were generated. The plausible reason for elevated catalase activity may be the generation of reactive oxygen species, especially H$_2$O$_2$ from SOD reaction, which necessitated more molecules of catalase involvement in peroxide breakdown. Hydrogen peroxide, after oxidation by myeloperoxidase, gives rise to another extremely reactive oxygen species - hypochlorous acid, a dissociation product of NaOCl. Hypochlorous acid generates superoxide radicals that cause oxidative injury and cell death (Peck et al., 2011). Hence, furthering increased oxidative stress due to NaOCl as observed in the present study with concomitant increased response by SOD and CAT.

Lipid peroxidation (LPO) is an autocatalytic process, which is a common consequence of cell death. Malondialdehyde (MDA) is one of the end products in the lipid peroxidation process (Sapakal et al., 2008). MDA is formed during oxidative degeneration as a product of free oxygen radicals (Neilsen et al., 1997) which is accepted as an indicator of lipid peroxidation (Ohkawa et al., 1979). In this study, a dose-dependent significant increase (p <0.05) was observed in the malondialdehyde concentration of groups exposed to sodium hypochlorite which corroborate with the result carried out by Iji et al., (2013). The increase in MDA levels in groups fed with 50µl, 100µl and 150µl 0.1% NaOCl compared with the control group may be related to the elevated level of H$_2$O$_2$. The increased levels of H$_2$O$_2$ mediate the toxic effect through the formation of hydroxyl radical, a potent activator of lipid peroxidation.

Xanthine oxidase is a form of xanthine oxidoreductase, an enzyme that generates reactive oxygen species such as superoxide radicals and hydrogen peroxide when it catalyzes the oxidation of hypoxanthine to xanthine, leading to the production of uric acid (Zhang et al., 2010; Ying et al., 2014). An increase in the activity of this enzyme potentiates oxidative stress due to the formation of superoxide free radicals (Kilikdar et al., 2011; Zhang et al., 2010). However, Munzel and Gori, (2012) reported that its role in mediating increased oxidative stress in the setting of hypertension is not quite clear. The significantly (p<0.05) elevated xanthine oxidase activity observed in this study translates to more generation of reactive oxygen species, and in particular superoxide ion and hydrogen peroxide which in turn evoked and or contributed to the significant increases in the activities of SOD and CAT as observed.

L-lactate dehydrogenase, EC 1.1.1.27, (LDH), a pyridine-linked enzyme expressed extensively in virtually all animal and human tissues, functions primarily in the metabolism of glucose, catalyzing the reduction of free pyruvate to lactate during the last step of glycolysis, as well as catalyzing the
conversion of lactate via oxidation of lactate by NAD$^+$ to produce pyruvate and NADH during gluconeogenesis (Emad et al., 2003). Its concentration is highest in the liver followed in descending order by skeletal muscle, heart, and kidney (Puc et al., 1985). The extracellular appearance of lactate dehydrogenase is a pointer to cell damage or cell death and is found to be at elevated levels where cell damage has occurred (Tope et al., 2016). Lactate dehydrogenase is an index widely used in toxicology and in clinical chemistry to diagnose cell, tissue and organ injury, including heart, liver, muscle, kidney, lung, and blood. Alterations in normal LDH activity pattern were found after exposure to xenobiotics and oxygen stress (Diamantino et al., 2001). Since tissue breakdown releases LDH, therefore LDH can be measured as a surrogate for tissue breakdown. Karthikeyan and Bavani, (2009) reported that the enzyme appears to be a reliable marker for metabolic abnormalities. In the present study, the significantly increased (p<0.05) lactate dehydrogenase activity in the plasma of the NaOCl fed rats, suggests tissue or organ damage hence its leakage into the blood.

The results of the present study suggest that prolonged exposure to the different doses (50 µl, 100 µl, and 150 µl) of NaOCl may be associated with liver damage as shown by significant increases (p<0.05) in plasma marker enzymes- aspartate transaminase (AST) and alanine transaminase (ALT). When the liver is damaged, it often results in a marked increase in the activities of the enzymes that are more confined within the hepatic cells. Such enzymes include alanine transaminase (ALT) and aspartate transaminase (AST), and so are marker enzymes to estimate the extent of damage in the liver (Onunkwor et al., 2015). Transaminases are responsible for detoxification processes, metabolism, and biosynthesis of energetic macromolecules for different essential functions and used as specific indicators for liver damage (Mossaa et al., 2015). When a hepatocellular injury occurs, the liver enzymes, aspartate transaminase, and alanine transaminase leaks into the extracellular space and enter the blood. An elevation of plasma ALT activity is often reflective of liver cell damage. In line with the present study, Iji et al., (2013) reported a significant increase in the activity of aspartate aminotransferase and alanine aminotransferase of birds exposed to the prolonged oral administration of calcium hypochlorite. The significant increase in AST and ALT recorded in this study indicates that the orally administered sodium hypochlorite in water is hepatotoxic.

The present study showed that NaOCl may also present adverse effect on the kidneys by compromising the renal efficiency in the removal of wastes such as urea, thus making the metabolites to be found in concentrations higher than normal in vivo (Ponce-Canchihuaman et al., 2010). Plasma urea and creatinine concentrations were significantly increased (p<0.05) especially among the rats fed 150µl NaOCl compared to the control rats as in other parameters investigated, suggesting dose-dependent aggravations in toxicity. Plasma creatinine and urea concentrations are biomarkers of renal injury and the elevated concentrations of these biomarkers in the blood are usually associated with impairment of renal function. The increase in urea may be due to degradation of purines and pyrimidines or increase urea concentration by either overproduction or the inability of excretion (Mossa and Abbasy, 2012), and significant (p<0.05) elevation of creatinine level in the blood is thus suggestive of possible impaired kidney function (Mossa et al., 2015).

Sodium hypochlorite is one of the chlorine compounds commonly used domestically in the treatment of water. It is a strong oxidizing agent that is highly and rapidly effective against a wide range of microbes and has been used as a universal disinfectant. Sodium hypochlorite when dissolved in water slowly decomposes, releasing sodium ions, chloride ions, hydroxyl radicals and hypochlorous acid (Estrela et al., 2002). However, the toxicity of sodium hypochlorite used for water disinfection is not due to chlorine,
but the formation of disinfectant by-products that results from the interaction of chlorine with organic materials in the source water (Hamdullah et al., 2010). Mixed with water, NaOCl combines to generate highly reactive hypochlorous acid (HOCl) that confers its potent antibacterial and antifungal properties. Hypochlorous acid generates superoxide radicals that cause oxidative injury and cell death (Peck et al., 2011). It is a key microbicidal agent, used as a natural defence owing to its great potency as a nucleophilic non-radical oxidant and its efficacy lies in the fact that neither bacteria nor mammalian cells can counteract its toxic effect since they lack the enzymes required for its catalytic detoxification (Hidalgo et al., 2002). Hypochlorite is a highly destructive, selective oxidant that reacts avidly with all biomolecules and is able to oxidize nucleotides, inactivate enzymes and electron transport systems, disrupt cell membranes and fragment proteins which lead eventually to cell death (Hidalgo et al., 2002). NaOCl is toxic by oral route, primarily due to the corrosive properties of the hypochlorite moiety. When the balance between reactive oxygen species production and antioxidant defences is lost, ‘oxidative stress’ results, which through a series of events deregulates the cellular functions leading to various pathological conditions (Sapakal et al., 2008). Oxidative stress is a broad term referring to several changes in vivo such as the presence of free reactive oxygen or nitrogen species, decrease in the count or activity of antioxidants and antioxidant enzymes, increased lipid peroxidation, among others (Raafat et al., 2009).

Conclusion

The present study concludes that prolonged exposure to 1.0% sodium hypochlorite may be inevitably associated with subtle toxic biochemical effects due to oxidants generation. The extent of damaging effects of NaOCl treated water, may present with mild to fatal consequences relative to dosage and the duration of oral exposure amongst unsuspecting population whose primary concern is access to portable safe water. Therefore, there is need for protection against treated water-related hazardous effects associated with NaOCl toxicity.

REFERENCES


