



Toxicological effects of sodium dodecyl sulfate

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ABSTRACT

The mass production and application of chemicals in different areas of plant and animal sciences cause a serious contamination to the immediate environment. Sodium Dodecyl Sulfate (SDS) is also known as Sodium Lauryl Sulfate (SLS) and commonly used in household, kitchen and laundry uses as a detergent ingredient and others. The description of SDS is important because it is now entered into the molecular laboratories such as biochemical research involving electrophoresis. It may have some effects on the cell or tissues of plants, animals and microorganisms. Some of the earlier reports suggested for the further research in SDS with higher plants. The higher plants may supply an important hereditary test method for screening and scrutinizing the genotoxic effects of SDS. The available data was so meager that there was a requirement of more research in the area. Therefore, it may be suggested to figure out the effects of SDS on the genome content of the plants, animals and microbes.

Key words: Mini-review, SDS, Plants, Effects, Genotoxic

INTRODUCTION

The environmental disturbances approach in various forms. The flora and fauna differ in their compassion and reaction to environmental disturbances. The organisms have a range of competence for disturbance alarm, signaling and retort. The mass production and application of chemicals in different areas of plant and animal sciences cause a serious contamination to the immediate environment.

Today, Man is apprehensive very much with the contamination of his environment. But, the hygienic environment has been an essential apprehension for human beings from time immemorial. The traditional soaps and detergents were used to prepare from plant or animal fats by the people of prehistoric India. Most probably, the traditional detergents and soaps were used to wash the utensils, laundry, bath and hand. Later on, modern technology has interfered with synthetic detergents and gradually substituted the traditional soaps and detergents. Thus modern man made detergents and soaps manufacturing and progressive production started. Afterward industrial revolutions grasp the other uses of detergents. The present detergent business is not confined to the domestic use and need, accordingly, but also serving to the requirements of trade and other locale where detergents may commonly used at present.

The first synthetic detergents were prepared and used by the Germans during the First World War with a general name called *Nekal* [1]. Most probably, it was prepared by the coupling of propyl or butyl alcohol with naphthalene and later on sulfonation of these two chemicals to produce a detergent of short-chain alkyl naphthalene sulfonate type.

During 1920s and 1930s long-chain alcohols were sulfonated and sold in the market as neutralized sodium salts [2]. During this period, for instance, a long-chain of alkyl aryl alcohols were sulfonated with benzene as the aromatic nucleus and the alkyl portion was made from a kerosene fraction. The growth of this sulfonated detergent became known and sold in the marketplace as cleaning materials, particularly in the USA [3].

EXPERIMENTAL SECTION

The materials and methods include the literature survey of the effects of SDS on different organisms and the information available during the survey on the topic was illustrated here.

RESULTS AND DISCUSSION

The data available on the toxicological effects of SDS in plants, mammals, fishes, human, and fungi were recorded here. SDS has effects on these organisms based on the concentrations and time of application. The data on the effects of SDS were recorded in two broad heads i.e. toxicological effects and environmental fate as under.

TOXICOLOGICAL EFFECTS OF SDS

SDS may be recognized to cause injurious effects on humans and animals. These harmful effects depend on the intensity of detergent concentrations and length of exposure. Some of the effects of SDS in fishes, mammals, humans, bacteria, yeast and plants are illustrated.

A concentration range of SDS (0-15mg/l) induced the morphological changes in kidney and spleen of gilthead (*Sparus aurata* L.) with a significant inhibitory effect on fertilization success [5]. The different concentrations of SDS (3, 5, 7 and 10 mg/l) were exposed on twenty juvenile turbot (*Scophthalmus maximus* L.) which showed 50% mortality at 384, 190, 12 and 4h respectively. There were sub-lethal chronic effects of SDS on the survival, metabolism, and growth of juveniles of *Centropomus parallelus* at three different salinities [6]. There were reports that SDS affects metabolism and swimming capacity of *Cyprinus carpio* L [7]. The acute toxicity of *Daphnia magna* increased with growing alkyl chain length of Alcohol Sulfates [8].

SDS had physical and biochemical effects on cells although the membrane being the primary target structure. It may cause epidermal cell proliferation and differentiation in vitro [9]. It has been reported that frequent revelation of SDS may be the source of skin irritation and hyperplasia in guinea pigs and more sensitive to Rabbit skin cultures than human skin [10]. It was reported that SDS might be unsafe by the oral route in mammals (LD₅₀ 1200 mg/kg bw), by the dermal route in rabbit (LD₅₀ = ~600 mg/kg bw) and guinea pig (>1200 mg/kg bw) with skin and eye irritation in all respectively [11]. The treatment of rats with SDS (100-1000 mg/kg bw/day) showed the augmented level of cholesterol esters and phospholipids but simultaneously reduced the levels of triglycerides, irritation of the gastro-intestinal tract, systemic toxicity on epididymal sperm and slight to moderate maternal toxicity [12].

SDS may be fatal or produce a serious damage to the health of an individual, if consumed ≤150 g [13]. The direct contact to SDS (≤20%) may cause moderate inflammation, irritation of the skin and repeated exposure may able to induce dermatitis like redness, swelling and blistering [14]. SDS may be very reactive in some persons and causing respiratory irritation, difficult breathing and further damage to the lung [15]. The hyperactivity of a body against an antigen (non-allergic condition) is known as reactive airways dysfunction syndrome (RADS).

It has been reported the toxic effects of SDS on gram-negative bacteria [16]. The increased amount of SDS in the cytoplasm contributes to misfolding of denatured protein which could be toxic to the cell with other toxic effects [17].

The SDS has an effect on different cell organelles and showed upregulated and downregulated genes in *Saccharomyces cerevisiae* [18]. The products of up and down regulated genes were localized in the cytoplasm, mitochondria, nucleus, peroxisome and plasma membrane [19].

There were reports on the effects of antibiotics in *Pisum sativum*, paraquat in *Hordeum vulgare* and human Lymphocytes, insecticides organophate and zadirctinbase on *Lathyrus sativus* L, 6- benzylaminopurine on *Cicer arietinum*, BAP and IAA *Vicia faba*, 2, 4-D on *Triticum aestivum* and on other plant systems [20-24]. The exposure of *V. faba* root tips to high concentrations of herbicide paraquat, sodium metabisulfate (SMB) and potassium metabisulfate (KMB) has been suggested the clastogenic, mutagenic, c-mitosis, inhibition of DNA synthesis and effects on the spindle formation [25]. The application of different concentrations of sodium ascorbate (SA), sodium benzoate (SB), boric acid (BA), citric acid (CA), potassium citrate (PC), sodium citrate (SC), potassium metabisulphite (PBS), sodium nitrite (SN) and creatine on meristematic root tips of *Allium cepa* L. and other plant cell at different times showed a progressive reduction in mitotic index, disappearance of protein bands and induction of cytogenetic abnormalities such as laggards, bridges and micronuclei [26-27]. The toxicity of alkyl sulfates towards algae ranged value between 1 and 10 mg/l [28]. Almost all the chemicals has been tested for their mutagenicity on different plant and animal models but there was not a single data available on the effects of SDS on meristematic root tips of plants for cytogenetical and other parameters studied.

ENVIRONMENTAL FATE

The biological degradation of SDS may be initiated with primary alkyl sulfatase and hydrates to 1-dodecanol. This (1-dodecanol) may be hydrolysed into dodecanal in the presence of primary alcohol dehydrogenase. The dodecanal could be converted into dodecanoic acid in the presence of aldehyde dehydrogenase. The carbon molecule dodecanoic acid could be cleaved into smaller carbon molecules by β -oxidation process or it might be elongated in to longer carbon atoms such as tetradecanoic acid. The carbon molecule tetradecanoic acid may be further used for the production of phospholipids or could be used to further elongation of the carbon molecule and desaturation of the carbon molecule. The desaturation of the carbon molecule may produce saturated and unsaturated fatty acids and degraded by β -oxidation which mineralised or incorporated into biomass [29]. There is an uncertainty of harmful concentrations and fractions affected for normal plant and animal species distribution [30].

CONCLUSION

The available data suggested that the use of SDS in various industry, household products, animals and plants is increasing at an alarming rate. Although, there were some reports of the effects of SDS on mammals, fishes, plants, bacteria and yeast but it was not sufficient to conclude anything. The available data was so meager that there was a requirement of more research in the area. Moreover, the available data had not been shown any adverse effects on the genome content of the materials studied except some chromosomal abnormalities in some plants (*Vicia faba* and *Allium cepa*). Therefore, it may be suggested to figure out the effects of SDS on the genome content of the plants, animals and microbes.

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REFERENCES

- [1] D S Painter. *The Journal of American History*, **2012**, 99(1), 24.
- [2] N R Itrich; T W Federle. *Ecotoxicology and Environmental Safety*, **2006**, 64, 30.
- [3] A K Mungray; P Kumar. *Ecotoxicology and Environmental Safety*, **2009**, 64, 14.
- [4] R El-Bakatoushi. *Romanian Journal Biology-Plant Biology*, **2010**, 55(2), 71.
- [5] M Rosety; F J Ordonez; R M Rosety; J M Rosety, I Rosety; C Carrasco; A Ribelles. *Histology and Histopathology*, **2001**, 4, 1091.
- [6] A J Rocha; V Gomes; P V Ngan; M J Passos; R R Furia. *Ecotoxicology and Environmental Safety*, 2007, 3, 397.
- [7] V Bantseev; D McCanna; A Banh; W W Wong; K L Moran; D G Dixon; J R Trevithick; J G Sivak. *Toxicological Science*, **2003**,1, 98.
- [8] J Ahlers; C Riedhammer; M Vogliano; R-U Ebert; R Kühne; G Schüürmann. *Environmental Toxicology and Chemistry*, **2006**, 25 (11), 2937.
- [9] D C L Wong; R J Toy; P B Dorn. *Ecotoxicology and Environmental Safety*, **2004**, 58, 173.
- [10] J C Dunphy; D G Pessler; S W Morrall; K A Evans; D A Robaugh; G Fujimoto; A Negahban. *Environmental Science Technology*, **2001**, 35, 1223.
- [11] S D Dyer; D T Stanton; J R Lauth; D S Cherry. , *Environmental Toxicology Chemistry*, **2000**,19, 608.
- [12] G M Boeije; M L Cano; S J Marshall; S E Belanger; R Van Compernelle; P B Dorn; H Gumbel; R Toy; T Wind. *Ecotoxicology and Environmental Safety*, **2006**,64, 75.
- [13] C V Eadsforth; A J Sherren; M A Selby; R Toy; W S Eckhoff; D C McAvoy; E Matthijs. *International Biodetermination Biodegradation* , **2006**,63 (8), 981.
- [14] D A Basketter; M York; J P McFadden; M K Robinson. *Contact Dermatitis*, **2004**,51, 1.
- [15] T W Federle; N R Itrich. *Ecotoxicology and Environmental Safety*, **2006**, 64, 30.
- [16] S E Belanger; P B Dorn; R Toy; G Boeije; S J Marshall; T Wind; R Van Compernelle; D Zeller. *Ecotoxicology and Environmental Safety*, **2006**, 64, 85.
- [17] S Rajagopal; N Sudarsan; K W Nickerson. *Applied Environment Microbiology*, **2002**, 68, 4117.
- [18] S Sirisaththa; Y Momose; E Kitagawa; H Iwahashi. *Water Research*, **2004**, 1, 61.
- [19] M M Abboud; K M Khleifat; M Batarseh; K A Tarawneh; A Al-Mustafa; M Al-Madadhah. *Enzymes Microbial Technology*, **2007**,41, 432.
- [20] J T Polit; J Maszewki; A Kozmierzak. *Cell Biology International*, **2000**, 27 (7), 559.
- [21] M Usciati; M Codaccioni; J Guern. *Journal of Experimental Botany*, **2004**, 23, 1009.
- [22] K Priti; M T Lalit; K N Tapan; K B Tarun; S Lelit; K S Bibhesh. *Nature and Science*, **2009**, 7 (3), 104.
- [23] J Gabriele; G Svetla; S Mila; K Stanislava. *Environmental Toxicology*, **2010**,25, 294.
- [24] S Kumar. *Cytology and Genetics*, **2010**,44 (2), 14.

- [25] K L Palani; N Panneerselvam. *Medicine and Biology*, **2007**,14(2), 60.
- [26] E A A Abdel-Hady; H M Barakat. *Journal Genetics Cytology*, **2005**,34, 237.
- [27] T Sifa. *Mutation Research*, **2007**,626, 4.
- [28] T Wind; S E Belanger. *Bulletin of Environmental Contamination and Toxicology*, **2006**,76, 218.
- [29] L Dou; Y Yajie; L Dongwei; S Liyan; W Yangqing; T Wei; X Zhonghui. *Research Journal Biotechnology*, **2013**,8(11), 100.
- [30] T Aldenberg; J Jaworska. *Ecotoxicology Environmental Saftey*, **2000**,46, 1.