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Review Article

Effects of Trace Elements - Tin or Tin Compounds on Animals

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Abstract

There is a close relationship between trace elements and animal health. Tin, as a common trace element, which is widely distributed in nature, plants, and the body of animals. Tin pose important position in animal production, but the effect of tin on animals needs to be clarified, so this review aims to clarify the effect of tin or tin compounds on the animals in the aspects of the absorption, distribution and metabolism, biological functions, the requirement of tin in animals, tin deficiency and toxicity, and then the relationship between tin and immunity, and the perspective research in animals were discussed. We hope this review could provide theoretical knowledge and guiding in future research and application of tin in animal production and health.

Keywords: Tin; Tin Compounds; Biological Functions; Deficiency; Toxicity; Immunity

Introduction

Tin (Sn), as an old element, is one of the earliest element found in the nature, it was used as tools in people's daily life from two or three thousand years ago. In modern days, tin or tin compounds is widely applied in the automotive industry (steel plate), atomic energy industry (protection material), fusible alloy (reagents and catalysts), chemical industry (drugs), plastic industry (synthetic rubber), polyester industrial (stabilizer and contact agent) and daily life (food packaging and tooth fillings materials). More and more studies have been proven that tin is an essential trace element for plants [1,2], microorganisms [3,4] and animals [5], which has many biological functions. Tin deficiency would cause negative effect on animals. However, high dose exposure to tin or tin compounds is harmful to people or animals. The purpose of this review is to clarify the effect of tin or tin compounds on the animals with respect to the absorption, distribution and metabolism of tin, the biological functions of tin or tin compounds, the requirement dose of the tin in animals, the deficiency and toxicity of tin, the relationship between tin and immunity and so on. Finally, we hope that this review could provide theoretical knowledge and guiding lines in future research and application of tin in animal production.

Absorption, Distribution and Excretion

Absorption

Tin compounds exist in two major groups of organotin and in organotin compounds. In organotin is poorly absorbed from the gastrointestinal tract in animals (2.8% of Sn^{2+} compounds and 0.64% of Sn^{4+} compounds). In laboratory species, more than 90% of an ingested dose of in organotin is recovered in the faeces [6,7]. Organotin compounds can generally enter the body through the respiratory, digestive tract and skin mucosa. Many factors can affect the absorption of tin, such as valence (Sn^{2+} salt is about four times more readily absorbed than the corresponding Sn^{4+} salt), anion complement [8], citric acid [9,10] and some others.

Distribution

Tin distributes throughout of the animal body, and accumulates in most of the organs, particularly in the bone, and to a lesser extent in the liver, lung, tongue, lymph nodes and kidney [7,11].

Excretion

Clearance of the absorbed tin is depend on its biological half-life, such as in bone, the half-life of tin is estimated to be 34 days (Sn^{2+}) and 40 days (Sn^{4+}) in the rat, as well as 10-20, 50 and 85 days in the kidney, spleen and the liver, respectively [7,12]. The organotin which has been absorbed in the body is mainly transformed by liver microsomal enzymes, and then most of the metabolites get into the kidney and the digestive tract, finally eliminated from saliva, milk, or eliminated through the respiratory tract mucosa [13].

Tin Compounds with Physiological Activity

Tin compounds can be divided into two main classes, inorganotin salt (valency +2 or +4) and organotin compounds. Inorganotin salts are generally considered to have a low level of toxicity, and tin oxides are considered to be non-toxic. For the organotin compounds, which have the general formula R_4SnX_{4-n} (n=1-4), the toxicological properties are affected by both the number and the nature of the organic groups (R) attached to the tin atom [14]. In generally, there are four styles of organotin compounds, which are monohydroxyltin compounds (RSnX3), dihydroxyltin compounds (R2SnX2), trialkyltin compounds (R3SnX) and tetralkyltin compounds (R4Sn) [15]. Physiological activity is in the order R3SnX>R4Sn>R2SnX2>RSnX3, and when R is butyl or propyl, the physiological activity of the tin compounds are up to the most. Among these compounds, the most contaminated and most studied organotin is Tributyltin compounds (TBT) and Triphenyltin (TPT). Several classes of organotin compounds--carboxylates, amino-acids and their derivatives, oxamates, organotin thioamides, organotin dithiocarbamates, organotin drug conjugates and organotin schiff base complexes are known to exhibit significant cytotoxicity to tumor cells *in vitro* and *in vivo* [16,17].

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Biological Function of Tin or Tin Compounds

Anti-bacteria and anti-corrosion

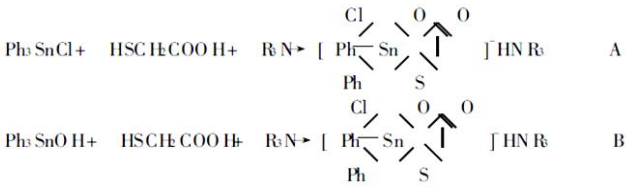
Organotins are usually used for industrial and agricultural purpose and antibiologic agent. A number of bacterial processes can be inhibited by organotins, whose inhibition effect is relate to the bacterial membrane functions [4]. Some of those microorganisms have been used for bioremediation studies and bio-reporters for detecting TBT pollution in the environment [18]. Tin compounds are also used in the synthesis of a variety of pesticides, fungicides, and pharmaceuticals. In the industry, tin is used as mildew preventive and preservatives [19].

Promote the growth, nucleic acid and protein synthesis

It is reported that tin is an essential trace element in life, the supplement of appropriate dose of tin has a significant growth effect and promote the metabolic reactions of proteins and nucleic acids [20]. For example, 50μg/kg TBT could increase the weight and promote the growth of the male mouse [21], and higher level of tin significantly reduced the growth rate, feed intake, and efficiency of feed utilization of broilers [22], and finally the growth performance was significantly affected by dietary tin supplementation in broilers.

Anti-tumor activities of tin compounds

There are four types of tin compounds with anti-tumor activity, R2SnX2, R2SnX2·L2, porphyrin derivatives, steroid derivatives and other tin compounds. It has been reported that the tin existing in the thymus hormone has anti-tumor effect, because it can produce one or more steroid or peptides compounds in the thymus [13]. Pellerito et al. (2006) [23] reported that the anti-tumor activity of organotin compounds may depend on the ability of release of Rn-Sn^{+} in the complex hydrolysis before formation of Sn-DNA, while the ability is related to the structure type of the hydrocarbon base, the space and electron effect of the hydrocarbon, bonding styles of ligand and tin atoms [24], and the bond length, such as the configuration B is not as stable as A, and the B compounds are better than A compounds in the inhibitory effect on tumor cells, as showed in below formula A and B.



As far as 1980s, studies have been found that some tin compounds exhibited anti-tumour activity and may play important roles in the cancer diagnosis and chemotherapy, or in controlling of the hyperbilirubinaemia [25]. Now, more and more tin based anti-tumour drugs have been synthesized [26-28] and nonplatinum based antitumor drugs containing tin (IV) illustrate the prominent strategies and applied prospect [29]. These functionalized organotin complexes incorporate with glycosyl substituents can enhance the tissue targeting, and act as an efficacious anti-proliferative drug by inducing morphological changes and apoptotic of the tumor cell [16]. Though the anti-tumor effect of organotin compounds or its derivatives has been widely recognized, but the development and application of organotin drugs have been hampered by their toxicity.

Relationship between tin and other trace element

Tin as an essential trace element, it can affect the mechanism of other elements. In general, tin has adverse effects on the metabolism of essential trace elements such as iron, copper, zinc, calcium, and selenium in rats or rabbit [30,31]. Tin ($SnCl_2$) consumption in rats resulted in a significant depression of zinc in liver, iron in kidney, copper in liver and kidney, and increased calcium concentration after tin administration in the liver and kidneys in rabbits [32]. Fe, Cu and Zn status can be influenced by dietary Sn ($SnCl_2$) concentrations lower than 50mg/kg in plasma, kidney, spleen or tibia in male rats [33].

Other functions

Recent study has reported that tin protoporphyrin can mitigates acute kidney injury [34]. Tin pose a positive effect on the wound healing, and promote tissue growth and wound healing and energy metabolism [35].

Requirement of Tin in Animals

Tin is an essential trace element for plants [36,37] and fungi [38]. The essentiality of tin for animals has also been confirmed, which has growth effect on rats [5]. Recommended allowance of tin in dietary of pig, poultry, ruminants is ≤ 50mg/kg according to the national standards of the People's Republic of China (GB26434-2010). Rats required 1μg tin as stannic sulfate per gram of experimental diet for optional growth, but this observation has not been confirmed [39].

Tin Deficiency

It has been reported that dietary tin deficiency caused the considerable changes in tissue mineral concentrations in rat [5], and the animals showed lack of growth, seborrhea, shaggy fur, and loss of hair and hearing [40]. The weights of the liver and tibia of the rats were less, and decrease in food efficiency by tin deficiency [5].

Toxicity of Organotin Compounds in Animals

Most of the inorganotin compounds is considered to have a low level of toxicity or non-toxic, but majority of the organotin compounds is thought to be have high level of toxicity, and the toxicity level is depended on the molecular structure of the organotin.

Reproductive toxicity

Many studies have reported that the organotin compounds have high toxicity to the reproductive systems of animals, for example, the Dibutyltin Chloride (DBTCl) (intraperitoneal injection ≥0.05μg/kg) induce the decrease of the testicles weight, sperm survival/density, and increase the rate of sperm deformation in mouse [41-43], and significantly decrease the sperm motility in rats [44]. Gracel et al. [45] reported that dibutyltin dilaurate (DBTDL) pose negative effect on the reproductive ability of rats. Tributyltin Compounds Chloride (TBTCL) caused increased of the number of cell fragments and deciduous cells in the seminal vesicles and epididymis [46]. In addition, the female animals are easily affected by organotin compounds. TBTCL can induce the infertility of the rats when exposed at the early stage of pregnancy [47,48], cause the abortion and fetal malformation when exposed at the middle stage of pregnancy [49], and induced the decrease in number of litter, weight loss, increase anus and genitals

distance when exposed at the whole stage of pregnancy [50,51].

The experimental study of the embryonal toxicity *in vitro* found that TBTCL (0.05mg/L) can inhibit the vitellicle growth and vascular differentiation, and increase the abnormal rate of embryo development [52].

Genotoxicity

It has been reported that all of the butyltin has genotoxicity to animals, among these compounds the Dibutyltin (DBT) (Sn20~40 μg/L) is the most toxic compound, and less toxic is the Tributyltin compounds (TBT) (Sn20~90 μg/L), and the tetrabutyl tin and Monobutyltin (MBT) have the lowest genotoxicity to animals [53,54]. The genotoxicity effect mainly performed in the following aspects, for example, butyltin compounds can induce the genetic mutations [55], chromosomal or chromosomal aberrations [56], and the oxidative injury and DNA damage [57-59].

Neurotoxicity

Trimethyl Tin (TMT), as an organotin compound, which has the high level of neurotoxicity to animals. The toxicity of TMT is mainly focus on the central nervous system, TMT can get through the blood-brain barrier, and resided in the central nervous system, and then result in a series of neurologic lesions and symptoms. TMT can lead to animal paraplegia, ataxia, excitement, attack behavior and epilepsy and other neurological symptoms, such as paraplegia, ataxia, agitation, aggression, epilepsy and other neurological symptoms [60,61]. In addition, animal studies indicate that TMT could impair the learning ability and cause the loss of memory [62]. The target organs of TMT neurotoxicity are hippocampus, dentate gyrus, caudate nucleus and cerebellum, brainstem and so on. TMT poses some negative effects on these organs by changing the neurotransmitter in the conduction process of the central nervous system [63,64], membrane potential [65,66], gene expression [67], and induced free radical damage, mitochondrial dysfunction [61], neuronecrosis (induced oxidative damage and neurodegeneration) [68,69], and at the same time cause the neurogenic inflammation [70], excitatory glutamate toxicity [71] and others.

Immunotoxicity

The immunotoxicity of tin compounds mainly performed in the effects on the immune cells, immune organs and immune immunoreactive substances, the detailed contents are reviewed in section 7.

The mechanisms of tin (take tmt for example) toxicity to the cells

The exposure of TMT causes the excessive release and accumulation of glutamate salts, and hence induces the excitatory toxicity of glutamate to cells [72]. The imbalance oxidation/antioxidant in cells which induced by oxidative stress caused by TMT is another important reason of cellular injury. TMT can also promote the increase of intracellular calcium ion concentration and thus impaired the activity of the Calpain [73]. More importantly, the Stannin protein on the mitochondrial outer membrane is the main target of TMT specificity action target [74], so the mitochondria is always the first organelle impaired by TMT. The mitochondrial membrane rupture cause the cytochrome C released into the cytoplasm, and then after the cytochrome C combined with Apaf1,

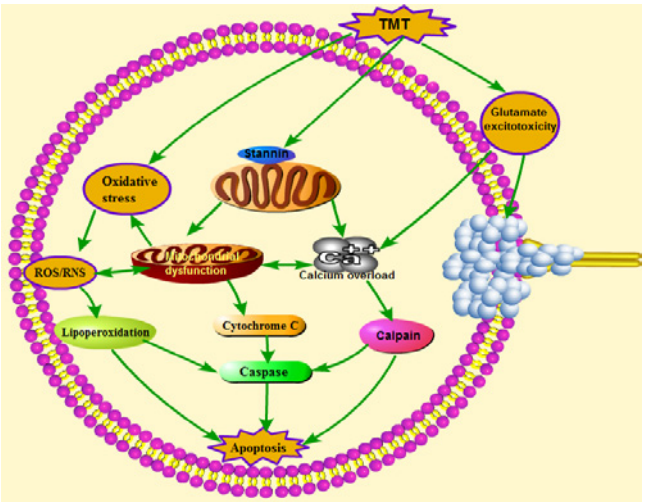


Figure 1: Mechanisms of TMT toxicity to the cells (remake according reference [71]).

they can stimulate the caspases activity, lastly activated the self-destruct program and eventually result in the apoptosis of the cells [75], as showed in Figure 1.

Tin or Tin Compounds and the Immunity

A lot of studies have been reported that tin or tin compounds pose effects on the immune organs, immune cells, the immunoactive substance and intestinal mucosal immunity [76-78].

Effect of tin compounds on the immune organs

The immune organs is the place where immune cells are formed, mature or concentrated, include the bone marrow, thymus, spleen, lymph node, and bursa of Fabricius (unique to birds). Most studies suggested that TBT and TBTCL could induce the atrophy of the thymus and spleen of mice [79-81], TBTCL induced the thymic atrophy of the crimson-spotted rainbowfish (Melanotaenia fluviatilis) [82]. TBTCL can also decrease the density of cortical cells in the thymus, and induce the substantial damage of the thymic structure and the formation of apoptosis body in mice [83].

Effect of tin compounds on the immune cells

The immune cells mainly consist of T lymphocyte, B lymphocyte, K lymphocyte, NK lymphocyte, mast cell and mononuclear phagocytic system and so on. Some studies indicated that low level of the TBT has positive effects on the B and T lymphocyte, and increase the antibody production of the B lymphocyte, and increase the number of the T lymphocyte [84]. However, high level of the TBT could decrease the ANAE positive rate of T lymphocyte in peripheral blood and the conversion rate of T lymphocytes in the spleen [80]. TBT can also affect the B lymphocyte, decrease the survival rate and its proliferation activity *in vitro* [53]. Harford et al. (2007) [85] reported that the murray cod (Maccullochella peelii peelii) were injected with TBT significantly decrease the B lymphocyte proliferation index *in vivo*, and the inhibiting effect was also observed *in vitro*.

The high dose of TBTCL can affect the phagocytic activity of macrophages in the liver and spleen of mice [80], and reduce number of natural killer cells of rats [86], decrease the macrophage

phagocytosis rate [83]. Other researches also reported that TBTCL can inhibit the NK cell activity, produce irreversible inhibition effects on NK cells [87,88].

Effect of tin compounds on the immunoactive substance

The immunoreactive substances are produced by immune cells or other related cells, there are three main types, which are antibodies (immunoglobulins), lymphokine (such as cytokines, interferon and other factors), and lysozyme. It has been reported that the appropriate dose of TBT can increase the mRNA expression of TNF-α in cells [89], and increase the production of the interleukin-6 (IL-6) in pig [90]. TBTCL inhibit the activities of Acid Phosphatase (ACP), Alkaline Phosphatase (AKP) [91,92] in rat or clam, and decrease the activity of lysozyme in serum and kidney of fish [93]. TBT can decrease the immune response to vibrio anguillarum and inhibit the production of specific antibody in channel catfish [94], and pose great effects on the level of serum IgM, IgG [80].

Effect of tin compounds on the intestinal mucosal immunity

The intestinal mucosal immune system, which is consisted of intraepithelial lymphocyte, lamina propria lymphocyte and Peyer Patch (PP) and other lymphoid tissues, which is the first line of defense against infection. A study has reported that TBTCL can inhibit the immune related genes expression in foregut and hindgut hepcidin of carp, and then the carp mucosal immune was suppressed [95].

Future Perspectives of Tin or Tin Compounds on Animals

At present, most of the researches suggested that the organotin compound can be used as the cancer chemotherapy drug because their lower toxicity and price, we believe that a lot of organotin drugs with antitumor activity will be found. However, the organotin pollution level in the environment and food safety issues to people health should be considered. So in the future studies, we should pay more attention on the research of microcosmic aspects, such as genes or proteins level, to explore the mechanism of tin or tin compounds (especially organotin) on animals or people, and to find a suitable biomarkers to monitor and to effectively avoid organotin pollution. The inorganotin is now begin to used in treatment of some diseases, but the toxicity and the mechanisms of them should be considered.

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References

1. Müller FL, Cyster LF, Raitt LM, Aalbers J. The effects of tin (Sn) additions on the growth of spinach plants. Phyton, International Journal of Experimental Botany. 2016; 84: 461-465.
2. Shaheen SM, Tsadilas CD, Rinklebe J. A review of the distribution coefficients of trace elements in soils: Influence of sorption system, element characteristics, and soil colloidal properties. Advances in Colloid and Interface Science. 2013; 201: 43-56.
3. Pardo T, Clemente R, Bernal MP. Effects of compost, pig slurry and lime on trace element solubility and toxicity in two soils differently affected by mining activities. Chemosphere. 2011; 84: 642-650.
4. Cooney JJ, Wuertz S. Toxic effects of tin compounds on microorganisms. Journal of Industrial Microbiology. 1989; 4: 375-402.
5. Yokoi K, Kimura M, Itokawa Y. Effect of dietary tin deficiency on growth and mineral status in rats. Biological Trace Element Research. 1990; 24: 223-231.
6. Mehri A, Marjan RF. Trace elements in human nutrition: a review. International Journal of Medical Investigation. 2013; 2: 115-128.
7. WHO. Guidelines for Drinking-Water Quality. Volume 2: Health Criteria and Other Supporting Information. World Health Organization, Geneva. 1996.
8. Hiles RA. Absorption, distribution and excretion of in organotin in rats. Toxicology and Applied Pharmacology. 1974; 27: 366-379.
9. Cima F. Tin: environmental pollution and health effects. Encyclopedia of Environmental Health. Elsevier, Burlington. 2011; 351-359.
10. Schafer SG, Femfert U. Tin a toxic heavy metal? A review of the literature. Regulatory Toxicology and Pharmacology. 1984; 4: 57-69.
11. Ge R, Wang YJ, Tang L, Cheng JM, Han LG, Li YL, et al. Preparation, antitumor activity in mice, pharmacokinetics and tissue distribution in rats of dinbutyl-di-(4-chlorobenzohydroxamato) tin (IV) liposome. Applied Organometallic Chemistry. 2014; 28: 204-210.
12. Nagano K, Nishizawa T, Umeda Y, Kasai T, Noguchi T, Gotoh K, et al. Inhalation carcinogenicity and chronic toxicity of indium-tin oxide in rats and mice. Journal of Occupational Health. 2011; 53: 175-187.
13. Li T, Zhan XL. Trace element tin and health. Guangdong Trace Elements Science. 2003; 10: 7-12.
14. Okoro HK, Fatoki OS, Adekola FA, Ximba BJ, Snyman RG. Sources, environmental levels and toxicity of organotin in marine environment-a review. Asian Journal of Chemistry. 2011; 23: 473-483.
15. Xiao X, Liang C, Zhao W. Organotin carboxylates: from structures to antitumour activities. European Journal of Bio Medical Research. 2015; 1: 23-27.
16. Arjmand F, Parveen S, Tabassum S, Pettinari C. Organo-tin antitumor compounds: their present status in drug development and future perspectives. Inorganica Chimica Acta. 2014; 423: 26-37.
17. Hadjikakou SK, Hadjiliadis N. Antiproliferative and anti-tumor activity of organotin compounds. Coordination Chemistry Reviews. 2009; 253: 235-249.
18. Cruz A, Anselmo AM, Suzuki S, Mendo S. Tributyltin compounds (TBT): a review on microbial resistance and degradation. Critical Reviews in Environmental Science and Technology. 2015; 45: 970-1006.
19. Yan XT, He B, Jiang GB. Cytotoxicity study of organotin compounds. The tenth national biological medicine chromatography and related technical academic symposium. 2014.
20. Elangovan A, Shim KF. The influence of replacing fish meal partially in the diet with soybean meal on growth and body composition of juvenile tin foil barb (Barbodes altus). Aquaculture. 2000; 189: 133-144.
21. Li BS. Effects of tributyltin compounds on Glucose Homeostasis in male mice and the recovery feasibility (Doctoral dissertation). Xiamen University, Xiamen. 2017.
22. Sun LH, Zhang NY, Zhai QH, Gao X, Li C, Zheng Q, et al. Effects of dietary tin on growth performance, hematology, serum biochemistry, antioxidant status, and tin retention in broilers. Biological Trace Element Research. 2014; 162: 302-308.
23. Pellerito C, Nagy L, Pellerito L, Szorcsik A. Biological activity studies on organotin (IV) n^+ complexes and parent compounds. Journal of Organometallic Chemistry. 2006; 691: 1733-1747.
24. Nath M, Vats M, Roy P. Design, spectral characterization, anti-tumor and anti-inflammatory activity of triorganotin (IV) hydroxycarboxylates, apoptosis inducers: *in vitro* assessment of induction of apoptosis by enzyme, DNA-fragmentation, acridine orange and comet assays. Inorganica Chimica Acta. 2014; 423: 70-82.
25. Barbieri F, Viale M, Sparatore F, Schettini G, Favre A, Bruzzo C, et al. Antitumor activity of a new orally active organotin compound: a preliminary

study in murine tumor models. Anti-cancer Drugs. 2002; 13: 599-604.

26. S Basu Baul T, Dutta D, de Vos D, Hopfl H, Singh P. Advancement Towards Tin-based Anticancer Chemotherapeutics: Structural Modification and Computer Modeling Approach to Drug-Enzyme Interactions. Current Topics in Medicinal Chemistry. 2012; 12: 2810-2826.

27. Gielen M, Biesemans M, de Vos D, Willem R. Synthesis, characterization and *in vitro* antitumor activity of di-and triorganotin derivatives of polyoxa- and biologically relevant carboxylic acids. Journal of Inorganic Biochemistry. 2000; 79: 139-145.

28. Gielen M, Biesemans M, Willem R. Organotin compounds: from kinetics to stereochemistry and antitumour activities. Applied Organometallic Chemistry. 2005; 19: 440-450.

29. Kaluderovic GN, Paschke R. Anticancer metallotherapeutics in preclinical development. Current Medicinal Chemistry. 2011; 18: 4738-4752.

30. Chiba M, Ogihara K, Inaba Y, Nishima T, Kikuchi M. The organ distribution of tin and the effect of tin on concentrations of several essential elements in rabbit. Toxicology. 1984; 31: 23-32.

31. Johnson MA, Greger JL. Tin, copper, iron and calcium metabolism of rats fed various dietary levels of inorganotin and zinc. The Journal of Nutrition. 1985; 115: 615-624.

32. Zaręba G, Chmielnicka J. Effects of tin and lead on organ levels of essential minerals in rabbits. Biological Trace Element Research. 1989; 20: 233-242.

33. Pekelharing HL, Lemmens AG, Beynen AC. Iron, copper and zinc status in rats fed on diets containing various concentrations of tin. British Journal of Nutrition. 1994; 71: 103-109.

34. Johnson AC, Delrow JJ, Zager RA. Tin protoporphyrin activates the oxidant dependent NRF 2-Cytoprotective pathway and mitigates acute kidney injury. Translational Research. 2017; 186: 1-18.

35. Wu MJ. The Element of Tin and Human Health. Studies of Trace Elements and Health. 2013; 30: 66-67.

36. Kabata-Pendias A. Trace elements in soils and plants, Fourth Edition. CRC press. Taylor & Francis Group, Boca Raton, London, New York. 2010.

37. Pilon-Smits EA, Quinn CF, Tapken W, Malagoli M, Schiavon M. Physiological functions of beneficial elements. Current Opinion in Plant Biology. 2009; 12: 267-274.

38. Miano T, D'Orazio V, Zaccone C. Trace elements and food safety. In PHEs, Environment and Human Health. Springer Netherlands. 2014; 339-370.

39. Aggett JP. Essential and toxic trace elements in human health and disease. Progress in Clinical & Biological Research. 1993; 380: 436-437.

40. Lan H, Wen Y, Shi Y, Liu K, Mao Y, Yi T. Fluorescence turn-on detection of Sn2+ in live eukaryotic and prokaryotic cells. Analyst. 2014; 139: 5223-5229.

41. Annanie D, Huang YY. Effects of dibutyltin chloride on testis and sperm quality in mice. Acta Zoologica Sinica. 2000; 46: 392-398.

42. Omura M, Ogata R, Kubo K, Shimasak Y, Aou S, Oshima Y, et al. Two-generation reproductive toxicity study of tributyltin compounds chloride in male rats. Toxicological Sciences. 2001; 64: 224-232.

43. Grote K, Stahlschmidt B, Talsness CE, Gericke C, Appel KE, Chahoud I. Effects of organotin compounds on pubertal male rats. Toxicology. 2004; 202: 145-158.

44. Yu WJ, Lee BJ, Nam SY, Kim YC, Lee YS, Yun YW. Spermatogenetic disorders in adult rats exposed to tributyltin compounds chloride during puberty. Journal of Veterinary Medical Science. 2003a; 65: 1331-1335.

45. Graceli JB, Sena GC, Lopes PFI, Zamprogno GC, da Costa MB, Godoi AFL, et al. Organotins: a review of their reproductive toxicity, biochemistry, and environmental fate. Reproductive Toxicology. 2013; 36: 40-52.

46. Yu WJ, Nam SY, Kim YC, Lee BJ, Yun YW. Effects of tributyltin compounds chloride on the reproductive system in pubertal male rats. Journal of Veterinary Science. 2003b; 4: 29-34.

47. Harazono A, Ema M, Ogawa Y. Pre-implantation embryonic loss induced by tributyltin compounds chloride in rats. Toxicology Letters. 1996; 89: 185-190.

48. Harazono A, Ema M. Suppression of decidual cell response induced by tributyltin compounds chloride in pseudopregnant rats: a cause of early embryonic loss. Archives of Toxicology. 2000; 74: 632-637.

49. Ema M, Kurosaka R, Amano H, Ogawa Y. Further evaluation of the developmental toxicity of tributyltin compounds chloride in rats. Toxicology. 1995; 96: 195-201.

50. Ogata R, Omura M, Shimasaki Y, Kubo K, Oshima Y, Aou S, et al. Two-generation reproductive toxicity study of tributyltin compounds chloride in female rats. Journal of Toxicology and Environmental Health. 2001; 63: 127-144.

51. Adeeko A, Li D, Forsyth DS, Casey V, Cooke GM, Barthelemy J, et al. Effects of in utero tributyltin compounds chloride exposure in the rat on pregnancy outcome. Toxicological Sciences. 2003; 74: 407-415.

52. Zeng HC, Chen F, Long DX, Li DY, He XP, Wu CQ, et al. Toxicity study on tributyltin compounds chloride on cultured mouse embryos *in vitro*. China Occupational Medicine. 2005; 32: 11-13.

53. Antizar-Ladislao B. Environmental levels, toxicity and human exposure to tributyltin compounds (TBT)-contaminated marine environment. A review. Environment International. 2008; 34: 292-308.

54. Dubey SK, Roy U. Biodegradation of tributyltin compoundss (organotins) by marine bacteria. Applied Organometallic Chemistry. 2003; 17: 3-8.

55. Cima F, Ballarin L. Genotoxicity and immunotoxicity of organotins. Biochemical and Biological Effects of Organotins. Bentham Science Publishers, Sharjah, UAE. 2012; 97-111.

56. Wu L, Chen Z, Zhu J, Hu L, Huang X, Shi H. Developmental toxicity of organotin compounds in animals. Frontiers in Marine Science. 2014; 1: 39-42.

57. Liu HG, Wang Y, Lian L. Tributyhin induces DNA damage as well as oxidative damage in rats. Environmental Toxicology. 2006; 21: 166-171.

58. Morales M, Martínez-Paz P, Ozáez I, Martínez-Guitarte JL, Morcillo G. DNA damage and transcriptional changes induced by tributyltin compounds (TBT) after short *in vivo* exposures of Chironomus riparius (Diptera) larvae. Comparative Biochemistry and Physiology Part C: Toxicology & Pharmacology. 2013; 158: 57-63.

59. Gabbianelli R, Villarini M, Falcioni G, Lupidi G. Effect of different organotin compounds on DNA of gilthead sea bream (Sparus aurata) erythrocytes assessed by the comet assay. Applied Organometallic Chemistry. 2002; 16: 163-168.

60. Kim JK, Choi SJ, Bae H, Kim CR, Cho HY, Kim YJ, et al. Effects of methoxsalen from Poncirus trifoliata on acetylcholinesterase and trimethyltin-induced learning and memory impairment. Bioscience, Biotechnology, and Biochemistry. 2011; 75: 1984-1989.

61. Zhang L, Zhao Q, Chen CH, Qin QZ, Zhou Z, et al. Synaptophysin and the dopaminergic system in hippocampus are involved in the protective effect of rutin against trimethyltin-induced learning and memory impairment. Nutritional Neuroscience. 2014; 17: 222-229.

62. Choi GN, Kim JH, Kwak JH, Jeong CH, Jeong HR, et al. Effect of quercetin on learning and memory performance in ICR mice under neurotoxic trimethyltin exposure. Food Chemistry. 2012; 132: 1019-1024.

63. Chen J, Huang C, Zheng L, Simonich M, Bai C, Tanguay R, et al. Trimethyltin chloride (TMT) neurobehavioral toxicity in embryonic zebrafish. Neurotoxicology and Teratology. 2011; 33: 721-726.

64. Park HJ, Shim HS, Choi WK, Kim KS, Shim I. Neuroprotective effect of Lucium chinense fruit on trimethyltin-induced learning and memory deficits in the rats. Experimental Neurobiology. 2011; 20: 137-143.

65. Geloso MC, Corvino V, Michetti F. Trimethyltin-induced hippocampal degeneration as a tool to investigate neurodegenerative processes. Neurochemistry International. 2011; 58: 729-738.

66. Harkins AB, Armstrong DL. Trimethyltin alters membrane properties of CA1 hippocampal neurons. Neurotoxicology. 1991; 13: 569-581.

67. Toggas SM, Krady JK, Billingsley ML. Molecular neurotoxicology of trimethyltin: identification of stannin, a novel protein expressed in trimethyltin-sensitive cells. Molecular Pharmacology. 1992; 42: 44-56.

68. Yu J, Ding D, Sun H, Salvi R, Roth JA. Neurotoxicity of trimethyltin in rat cochlear organotypic cultures. Neurotoxicity Research. 2015; 28: 43-54.

69. Viviani B, Corsini E, Galli CL, Marinovich M. Glia increase degeneration of hippocampal neurons through release of tumor necrosis factor-α. Toxicology and Applied Pharmacology. 1998; 150: 271-276.

70. Corvino V, Marchese E, Michetti F, Geloso MC. Neuroprotective strategies in hippocampal neurodegeneration induced by the neurotoxicant trimethyltin. Neurochemical Research. 2013; 38: 240-253.

71. Yu JT, Ding DL, Sun H, Yin SK, Salvi R. Neurotoxicity and Ototoxicity of Trimethyltin. Chinese Journal of Otology. 2013; 11: 582-582.

72. Dawson R, Patterson TA, Eppler B. Endogenous excitatory amino acid release from brain slices and astrocyte cultures evoked by trimethyltin and other neurotoxic agents. Neurochemical Research. 1995; 20: 847-858.

73. Piacentini R, Gangitano C, Ceccariglia S, Fà AD, Azzena GB, Michetti F, et al. Dysregulation of intracellular calcium homeostasis is responsible for neuronal death in an experimental model of selective hippocampal degeneration induced by trimethyltin. Journal of Neurochemistry. 2008; 105: 2109-2121.

74. Davidson CE, Reese BE, Billingsley ML, Yun JK. Stannin, a protein that localizes to the mitochondria and sensitizes NIH-3T3 cells to trimethyltin and dimethyltin in toxicity. Molecular Pharmacology. 2004; 66: 855-863.

75. Tomiyama K, Arakawa Y. Zinc- and tin-induced apoptotic mechanisms in immune system and cranial nerve system. Nihon Rinsho Japanese Journal of Clinical Medicine. 2016; 74: 1111-1119.

76. Lawrence S, Reid J, Whalen M. Secretion of interferon gamma from human immune cells is altered by exposure to tributyltin compounds and dibutyltin. Environmental Toxicology. 2015; 30: 559-571.

77. Ueno S, Kashimoto T, Susa N, Asai T, Kawaguchi S, Takeda-Homma S, et al. Reduction in peripheral lymphocytes and thymus atrophy induced by organotin compounds *in vivo*. Journal of Veterinary Medical Science. 2009; 71: 1041-1048.

78. Wu WF, Chen LL, Li YJ, Zhu CH. Effects of tributyltin compounds on the Activities of Immunologic Enzyme in Blood Serum of the Macrobrachium rosenbergill. Journal of Guangdong Ocean University. 2014; 34: 17-21.

79. Im E, Kim H, Kim J, Lee H, Yang H. Tributyltin compounds acetate-induced immunotoxicity is related to inhibition of T cell development in the mouse thymus. Molecular & Cellular Toxicology. 2015; 11: 231-239.

80. Wu HH, Zhang ZJ, Chen Q, Sun XY. Effects of TBTCL on Immune Function of Mice. Journal of Environment and Health. 2006; 23: 137-139.

81. Aw TY, Nicotera P, Manzo L, Orrenius S. Tributyltin compounds stimulates apoptosis in rat thymocytes. Archives of Biochemistry and Biophysics. 1990; 283: 46-50.

82. Harford AJ, O'Halloran K, Wright PF. The effects of *in vitro* pesticide exposures on the phagocytic function of four native Australian Freshwater Fish. Aquatic Toxicology. 2005; 75: 330-342.

83. Chen Q, Zhang ZJ. The immune toxicity and its mechanism of TBTCL on mice. Journal of Toxicology. 2007; 19: 222-222.

84. Ananie D, Chen XL, Zhang YQ, Dai SG. The effects of different organotin compounds on peripheral blood T and B lymphocytes in mice. Acta Scientiarum Naturalium Universitatis Nankaiensis (Science Edition). 2000; 33: 67-69.

85. Harford AJ, O'Halloran K, Wright PF. Effect of *in vitro* and *in vivo* organotin exposures on the immune functions of murray cod (Maccullochella peelii peelii). Environmental Toxicology and Chemistry. 2007; 26: 1649-1656.

86. Whalen MM, Loganathan BG. Butyltin exposure causes a rapid decrease in cyclic AMP levels in human lymphocytes. Toxicology and Applied Pharmacology. 2001; 171: 141-148.

87. Aluoch AO, Odman-Ghazi SO, Whalen MM. Pattern of MAP kinases p44/42 and JNK activation by non-lethal doses of tributyltin compounds in human natural killer cells. Archives of Toxicology. 2007; 81: 271-277.

88. Pavlikova N, Arukwe A. Immune-regulatory transcriptional responses in multiple organs of Atlantic salmon after tributyltin compounds exposure, alone or in combination with forskolin. Journal of Toxicology and Environmental Health. 2011; 74: 478-493.

89. Nakano K, Tsunoda M, Konno N. Tributyltin compounds (TBT) increases TNF-α mRNA expression and induces apoptosis in the murine macrophage cell line *in vitro*. Environmental Health and Preventive Medicine. 2004; 9: 266-271.

90. Bernardini C, Zannoni A, Bertocchi M, Bianchi F, Salaroli R, Botelho G, et al. Deleterious effects of tributyltin compounds on porcine vascular stem cells physiology. Comparative Biochemistry and Physiology Part C: Toxicology & Pharmacology. 2016; 185: 38-44.

91. Mitra S, Gera R, Singh V, Khandelwal S. Comparative toxicity of low dose tributyltin compounds chloride on serum, liver, lung and kidney following subchronic exposure. Food and Chemical Toxicology. 2014; 64: 335-343.

92. Ranilalitha P, Sukumaran M, Raveendran S. Effect of TBTCL on phosphatases activity in estuarine edible clam, Anadara rhombea born (Bivalvia: Mollusca). International Journal of Pure and Applied Zoology. 2014; 2: 315-320.

93. Dalmo RA, Ingebrigtsen K, Bøgwald J. Non-specific defence mechanisms in fish, with particular reference to the Reticuloendothelial System (RES). Journal of Fish Diseases. 1997; 20: 241-273.

94. Regala RP, Rice CD, Schwedler TE, Dorociak IR. The effects of tributyltin compounds (TBT) and 3, 3′, 4, 4′, 5-pentachlorobiphenyl (PCB-126) mixtures on antibody responses and phagocyte oxidative burst activity in channel catfish, Ictalurus punctatus. Archives of Environmental Contamination and Toxicology. 2011; 40: 386-391.

95. Liu JC. Effects of chlorotributylstannane tributyltin compounds chloride on the mucosal immunity of carp (Cyprinus carpio L). (Master's thesis), Shandong Normal University. 2011.

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