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Toxic Effects of Organotin Species on Algae

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The inhibition by 20 organotin compounds (R_nSnX_{4-n}), which included some newly synthesized pesticides, of the growth of two green algae (*Scenedesmus obliquus* and *Platymonas* sp.) was studied. The influence of the counterion X group on the toxicity of organotins was discussed. The destruction of submicrostructures of the algal cell by organotins was viewed by micrography and electron microscope scanning. It was found that the toxicity of the organotins varied significantly according to their substitution. The order of toxicity is tri- ≫ di- ⩾ mono-organotins. Within the same substituent series, the toxicity depends on the properties of both R and X groups. The larger and the more lipophilic the R group, the more toxic is the organotin. The influence of the X group is more complex: when X is a small easily ionizable group, it has little effect on the toxicity; however, when it is a large organic group, it does change the bioactivity of the organotin. Large organic groups may influence the polarizability of the central tin atom, and so change the toxicity of organotins.

Keywords: organotins; toxicity; algae; submicrostructure; counterions

INTRODUCTION

Organotin compounds, represented by the formula R_nSnX_{4-n} (R are groups attached to the tin atom through C–Sn bonds; X are attached through non-C–Sn bonds), are a series of extensively used organometallic compounds. Mono- and di-organotins are mainly used as thermal stabilizers in PVC products and catalysts in the production of polyurethane foams; triorganotins are used in agriculture, paper-making and the paint industry as biocides.

In recent years, the production of organotins has risen rapidly. The total world production was about 3.5×10^7 kg y^{-1} in 1989, and it was estimated that about 30% would enter into the aquatic environment eventually.[1] There have been many reports on organotin pollution in different aquatic ecosystems, especially in harbours around the world with heavy shipping traffic.[2] Our research group has also detected organotin pollution in several waters in North China.[3,4]

There have been many reports on the toxicity of organotins for various aquatic organisms, providing the information that some organotins are highly toxic to aquatic organisms. However, most studies were carried out on tributyltin and triphenyltin species. There are many kinds of organotins, and they transform into other species through complex chemical reactions after they enter the environment.[5,6] Therefore studies on the toxic effects of the different kinds of organotin is needed.

The toxicity of organotins R_nSn_{4-n} varies with substitution and the R group.[7,8] Little information on the influence of the X group on toxicity has been found. The present workers studied the inhibition by 20 different kinds of organotins, which included some newly synthesized pesticides with large N-containing aromatic X groups, on the growth of two algae (*Scenedesmus obliquus* and *Platymonas* sp.). The influence of the X group on the toxicity of these organotins was investigated. In order to illustrate the destruction of submicrostructures of the algae by organotins, which can help to reveal the toxicity mechanism of organotins, typical toxic symptoms of the algae were viewed by micrography and electron microscope scanning.

EXPERIMENTAL

Chemicals

Tri-n-butyltin chloride (TBTCl), di-n-butyltin chloride ($DBTCl_2$), mono-n-butyltin chloride ($MBTCl_3$), trimethyltin chloride (TMTCl), dimethyltin chloride ($DMTCl_2$), monomethyltin chloride ($MMTCl_3$), triphenyltin chloride (TPTCl), triphenyltin hydroxide (TPTOH), tri-

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phenyltin acetate (TPTOCOMe), diphenyltin chloride ($DPTCl_2$), monophenyltin chloride ($MPTCl_3$), tri-n-propyltin chloride (TPrTCl) and diethyltin chloride ($DEtTCl_2$) were purchased from Vertron and Aldrich Chemical Co. The other organotins were pesticides synthesized by the Research Institute of Elemento-Organic Chemistry of Nankai University. They are formulated as shown in Table 1 (compounds **14–20**; Cy = cyclohexyl).

The purity levels of all the organotins were at least 99% except $DBTCl_2$ (purity 96.5%). Most organotins were dissolved in absolute ethanol to make up 1000 μg Sn l^{-1} stock solutions, and those with lower solubility, usually containing Cy and Ph groups, were made up at 500 μg Sn l^{-1} in ethanol solutions. All stock solutions were stored in a refrigerator in the dark and diluted to suitable concentrations before each experiment.

Instrumentation

This includes an XSP-18 microscope (Jian Nan Optics Instrument Co., China), a THE88-1 shaker (TaiCang Biochemical Instrument Co., China), a BH-2 microscope camera (Olympus Co., Japan), and an EM-400ST electron microscope (Philips Co., USA).

Toxicity tests

The two test algae were *Scenedesmus obliquus* and *Platymonas* sp. *S. obliquus* was cultured in Aquatic-4, and the nutrition medium for *Platymonas* was artificial seawater (salinity 18‰) fortified with trace metals and nutrient salts. The pH values of the nutrition media were 7.6 and 7.8 for *S. obliquus* and *Platymonas* sp., respectively. In order to estimate the toxic effects, the inhibition by organotins of the growth of the algae was determined. The experiment was conducted in a 250 ml Erlenmeyer flask that was sealed with a bacterium-eliminated ventilating film. The medium in the flask was sterilized by pasteurization at 120°C and allowed to cool to room temperature. Algae stock solution, in the logarithmic stage of growth, was transferred into the nutrient medium immediately after the organotin standard solution had been mixed well in it. The volumes of test medium were 100 ml for *S. obliquus* and 60 ml for *Platymonas* sp. Six concentrations of toxicant were used, and each concentration as well as the untreated control was tested several times. Each compound was tested one to three times. Algae were cultured for 96 h at (25±1) °C under cycles of 12 h of light (4000 lux) and 12 h of darkness. Each day the test media were shaken for 5 min on a shaker (100 rpm) at 3 h intervals. The cells were enumerated every morning.

The maximum ethanol concentration in the test media was not more than 3%. An ethanol control test indicates that under this concentration ethanol has no deleterious effect on the growth of the two algae.

Typical poisoning symptoms of the algae were viewed and recorded using microphotography. *Platymonas* contaminated with TBT was observed using an electron microscope.

RESULTS AND DISCUSSION

Inhibition by organotin species of the growth of the algae

Figures 1(a) and (b) are the growth curves of *Scenedesmus obliquus* and *Platymonas* sp. respectively in test media containing different TBTCl concentrations.

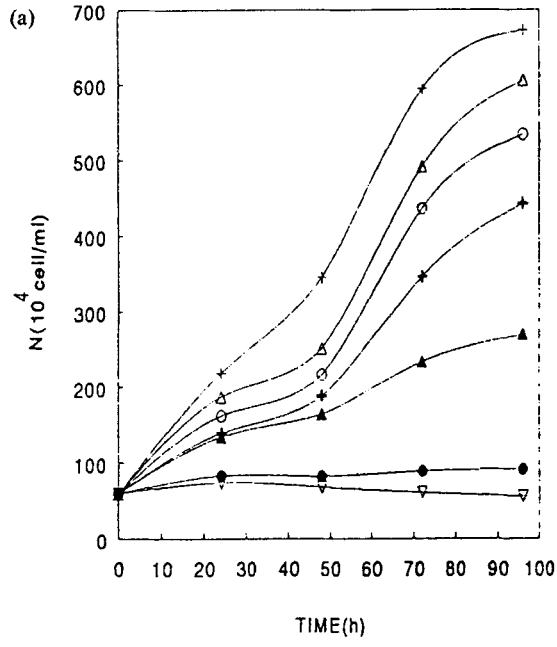
It can be seen that the EC_{50} values of TBT for the two algae are less than 1 ppb. TBTCl is highly toxic to algae. Responses of the two algae to the other 19 organotins are of a similar pattern. Proportions of inhibition (PI) of the growth of the two algae by different concentrations of organotins are linearly correlated with the logarithm of the concentrations. Correlation coefficients r varied from 0.925 to 0.999. Thus EC_{50} values can be calculated from Eqn [1]:

$$PI = a + b \log_C \quad [1]$$

where PI is the inhibition proportion of the growth of algae, C is the concentration of organotin, and a and b are constants. The average 96 h EC_{50} values of 20 organotins for the growth of the two algae are listed in Table 1.

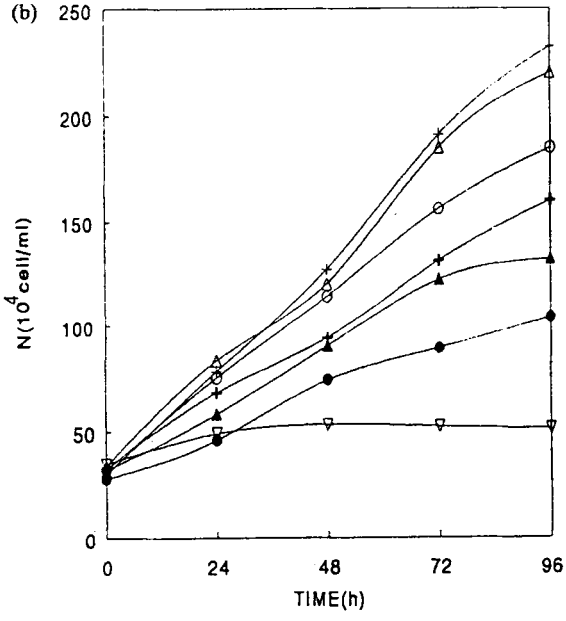
Comparison of toxicity of different organotins

In order to compare the toxicity of different organotins and to interpret the structural factors, toxicity values T were calculated from Eqn [2]:



Data for Fig. 1(a)

Run no.	TBT concn ($\mu g\ l^{-1}$)	$N \times 10^4$ cells/ml 0 h	24 h	48 h	72 h	96 h
0	0.00	60	218	345	595	673
1	0.05	60	187	251	492	606
2	0.20	60	162	216	436	534
3	0.80	60	139	188	346	443
4	1.50	60	134	164	233	269
5	3.00	60	83	82	89	91
6	5.00	60	74	68	61	56



Data for Fig. 1(b)

Run no.	TBT concn ($\mu g\ l^{-1}$)	$N \times 10^4$ cells/ml 0 h	24 h	48 h	72 h	96 h
0	0.00	30	78	127	191	232
1	0.02	34	83	120	185	220
2	0.05	32	75	114	156	185
3	0.20	33	68	94	131	160
4	0.80	32	58	90	122	132
5	1.60	28	46	74	89	104
6	3.00	35	49	53	52	51

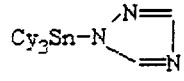
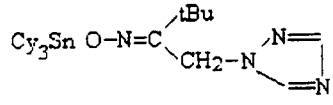
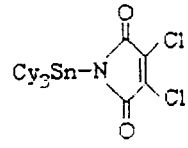
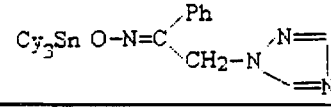
Figure 1 Growth curves of the two algae in media containing different TBTCl concentrations, as follows. (a) *S. obliquus*: + (run 1), 0.0 ppb; △ (run 2), 0.05 ppb; ○ (run 3), 0.2 ppb; + (run 4), 0.80 ppb; ▲ (run 5), 1.50 ppb; ● (run 6), 3.00 ppb; ▽, 5.0 ppb. (b) *Platymonas*: + (run 1), 0 ppb; △ (run 2), 0.02 ppb; ○ (run 3), 0.05 ppb; + (run 4), 0.20 ppb; ▲ (run 5), 0.80 ppb; ● (run 6), 1.60 ppb; ▽ (run 7), 3.00 ppb. N is the number of cells/ml.

$$T = (EC_{50}/M_{Sn})^{-1} \quad [2]$$

where M_{Sn} is the atomic weight of a tin atom. The unit of T is 1 (μmol Sn)$^{-1}$. Bar graphs of toxicity values are shown in Figs 2 and 3, which represent different series of organotin compounds.

It is evident that different species of organotins have different toxicities. First, the toxicity of

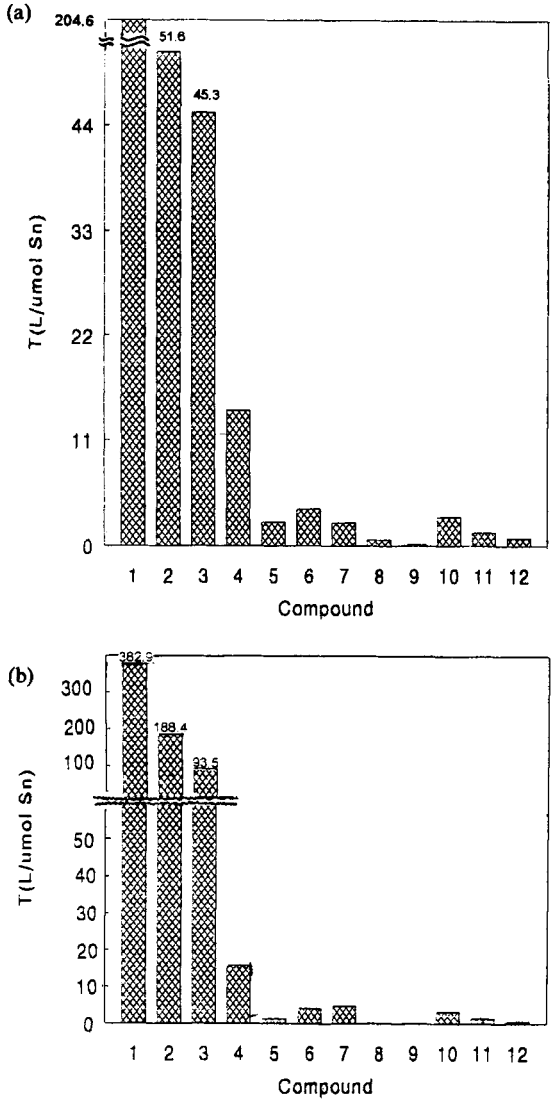
Table 1 96 h EC_{50} values of organotins

Compound[a]	EC_{50} of algae (ng Sn l^{-1})	
	S. obliquus	*Platymonas* sp.
1 TBTCl	0.58	0.31
2 TPrTCl	2.30	1.27
3 TPTCl	2.62	0.63
4 TMTCl	48.02	98.14
5 $DBTCl_2$	31.44	30.04
6 $DPTCl_2$	42.29	24.87
7 $DEtTCl_2$	190.83	635.05
8 $DMTCl_2$	805.84	988.69
9 $MBTCl_3$	39.77	38.35
10 $MPTCl_3$	85.12	83.46
11 $MMTCl_3$	157.86	211.27
12 TPTOH	2.44	0.70
13 TPTOCOMe	2.71	0.61
14 $Cy_2MeSnOCOMe$	8.42	7.51
15 $Cy_2MeSnOCOPr$	8.42	7.47
16 Cy_3SnOH	17.65	0.65
17 	5.56	0.28
18 	21.85	1.18
19 	14.42	0.40
20 	5.20	0.28

[a] Abbreviation: Cy, cyclohexyl.

organotins varies significantly with substitution: triorganotins have the highest toxicity, which has proved to be true for different types of organisms. However, the difference in toxicity for di- and mono-organotins for the two algae is not significant, which is not the case for animals, where diorganotins are more toxic than mono-organotins.[9, 10]

Within the same substitution series, the toxicity of organotins depends on the properties of the groups R and X. In Fig. 2, when the organotins are all chlorides and the R groups are different, it can be seen that the toxicity of organotins depends on the size and lipophilicity of the R group. The larger and the more lipophilic R is, the more toxic is the organotin. In Fig. 3, there are three groups (viz. 1–3, 4–5, 6–10) and within each group the organotin contains the same R group. Few reports exist on the influence of the X group on the toxicity of organotins. The opinion that the X group has little influence on the toxicity of organotins is well accepted. In order to reveal the influence of the X group, we selected different types of this group. From Fig. 3, it is clear that the influence of X is rather complicated. In the first group (see Fig. 3), the X groups are easily ionizable and ionize immediately after the organotins enter into aqueous solution; the remaining organic part of the organotin will bind with dominant anions in aqueous solution (i.e. OH^- and Cl^-), so organotins with the same



Data for Fig. 2

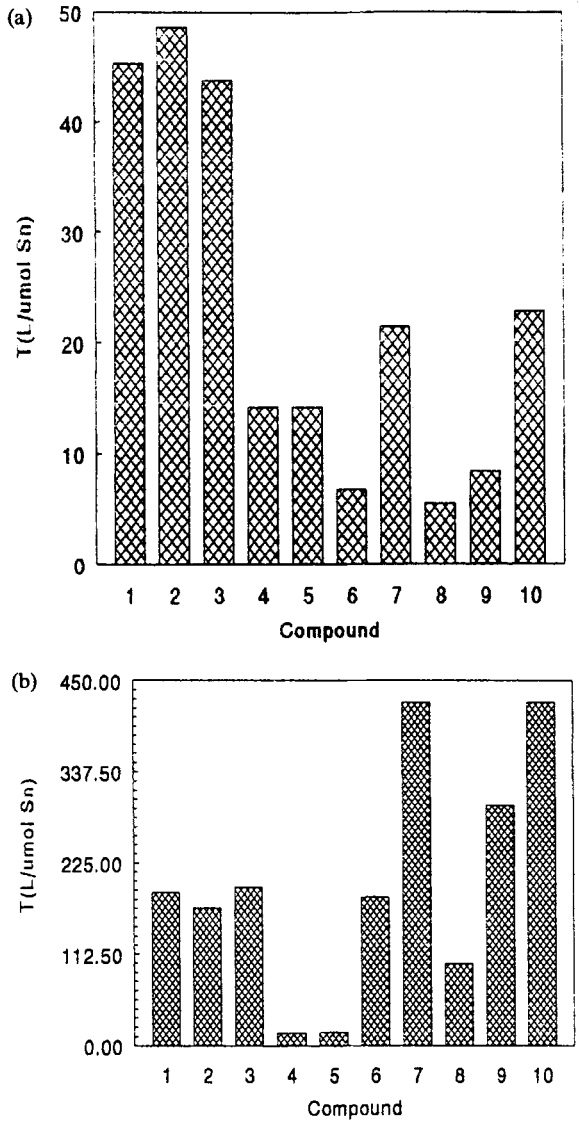
Bar no.	Compound	T (l/μmol Sn) (a) *S. obliquus*	(b) *Platymonas* sp.
1	TBTCl (1)	204.6	382.9
2	TPrTCl (2)	51.6	93.5
3	TPTCl (3)	45.3	188.4
4	$Cy_2MeSnOCOMe$ (14)	14.1	15.8
5	TMTCl (4)	2.47	1.21
6	$DBTCl_2$ (5)	3.77	3.95
7	$DPTCl_2$ (6)	2.14	4.79
8	$DEtTCl_2$ (7)	0.62	0.19
9	$DMTCl_2$ (8)	0.15	0.12
10	$MBTCl_3$ (9)	2.99	3.09
11	$MPTCl_3$ (10)	1.39	1.42
12	$MMTCl_3$ (11)	0.76	0.56

Figure 2 Bar graphs of T values for R_nSnCl_{4-n} (R are aliphatic or aromatic substituents; $n=1, 2, 3$). The compounds corresponding to bar numbers 1–10 are listed in the accompanying table (a) *S. obliquus*. (b) *Platymonas* sp.

organic group always have the same patterns under the same conditions. In this paper, TPTOH, TPTOCOMe and TPTCl have similar toxicities in both *S. obliquus* and *Platymonas* sp. In the second group, 96 h EC_{50} values for $Cy_2MeSnOCOMe$ and $Cy_2MeSnOCOPr$ are almost the same. However, during the experiment it was observed that the toxic effect of $Cy_2MeSnOCOPr$ occurred more slowly than that of $Cy_2MeSnOCOMe$. Thus EC_{50} values after different periods were calculated and are listed in Table 2.

In the earlier stages of the experiments, the toxicity of $Cy_2MeSnOCOPr$ (**15**) was obviously lower than that of $Cy_2MeSnOCOMe$, (**14**) and gradually became equal to that of $Cy_2MeSnOCOMe$ by 96 h. We attributed this effect of $Cy_2MeSnOCOPr$ owing to OCOPr being large in volume so that it decomposes slowly from the tin atom, and consequently obstructs the entry of the whole organotin into the algal cell or the combination of the tin atom with the active sites in biomolecules.

In the third group, X is a large nitrogen-containing aromatic group. For the five Cy_3-containing compounds studied, the T values vary



Data for Fig. 3

Bar no.[a]	T (l/μmol Sn) (a) *S. obliquus*	(b) *Platymonas* sp.
1	45.3 Group I	188.4 Group I
2	48.6	169.5
3	43.8	194.6
4	14.1 Group II	15.8 Group II
5	14.1	15.9
6	6.73 Group III	182.6 Group III
7	21.4	423.8
8	5.43	100.6
9	8.34	296.7
10	22.8	423.8

[a] For key to bar numbers, see caption below.

Figure 3 Bar graphs of T values for several groups of organotins (1–3, 4–5 and 6–10); the members of each group contain the same R substituents. (a) *S. obliquus*; (b) *Platymonas* sp. The numbers represent the same compounds in (a) and (b), as follows: 1, TPTCl (**3**); 2, TPTOH (**12**); 3, TPTCOMe (**13**); 4, $Cy_2MeSnOCOMe$ (**14**); 5, $Cy_2MeSnOCOPr$ (**15**); 6, Cy_3SnOH (**16**); 7–10, **17–20**, respectively, in Table 1.

from 5.20 to 21.85 for *S. obliquus* and from 0.28 to 1.18 for *Platymonas* sp., exceeding the error range of the test. It can be concluded that X groups do influence the toxicity of the organotins. In order to explain the toxicity sequence of these five compounds in terms of the toxicant structure, we intended to relate the toxicity to the lipophilicity parameter $^1x^r$ which has been considered to be the crucial factor governing the toxicity of the organotins.[8] However, the correlation coefficient is very low. Therefore we suggest that the X group influences the toxicity of organotins, not only by changing the lipophilicity of the whole molecule, but also by affecting the polarizability of the tin atom.

According to the soft–hard acid–base theory, a hard (soft) Lewis acid is easy to combine with the complementary Lewis base to form stable compounds. Seeing that most of the active sites in organisms, such as —SH, are soft Lewis bases,

Table 2 EC_{50} values of $Cy_2MeSnOCOMe$ and $Cy_2MeSnOCOPr$ after different periods of time

Alga	Compound	24 h	48 h	72 h	96 h
S. obliquus	$Cy_2MeSnOCOMe$ (**14**)	9.49	8.21	8.24	8.42
	$Cy_2MeSnOCOPr$ (**15**)	10.40	9.95	9.74	8.42
Platymonas sp.	$Cy_2MeSnOCOMe$ (**14**)	8.96	8.67	7.53	7.51
	$Cy_2MeSnOCOPr$ (**15**)	10.32	9.27	7.84	7.47

soft Lewis acids such as $-CH_3Hg$ combine tightly with these sites and thus are highly toxic. Factors which could decrease the positive charge and consequently increase the polarizability of the central tin atom would turn the tin atom softer and hence would increase the toxicity of the organotins. In most toxic compounds, **17** and **20**, the tin-binding atoms N and O belong to a large π-systems. A large π-system can provide more electrons to O and N atoms; hence the intensity of withdrawal of electrons from the tin atoms declines. In general, the positive charge of the tin atoms decreases, polarizability increases, and the toxicity of the whole molecule increases. In the case of the compounds Cy_3SnOH (**16**) and **19** the tin-adjacent oxygen and nitrogen atoms are both electron-attracting, and thus their toxicity is low. The structures of **18** and **20** are similar. However, the toxicity of the former is three times lower than that of the latter. In this case, because Ph is replaced by t-Bu, the oxygen atom cannot share electrons from a large π-system, and so it attracts electrons from the tin atom. This results in a decrease of toxicity. Furthermore, the polarizability of t-Bu itself is very low. It will prevent the organotin from permeating through the organism film or combination of the tin atom with the active sites.

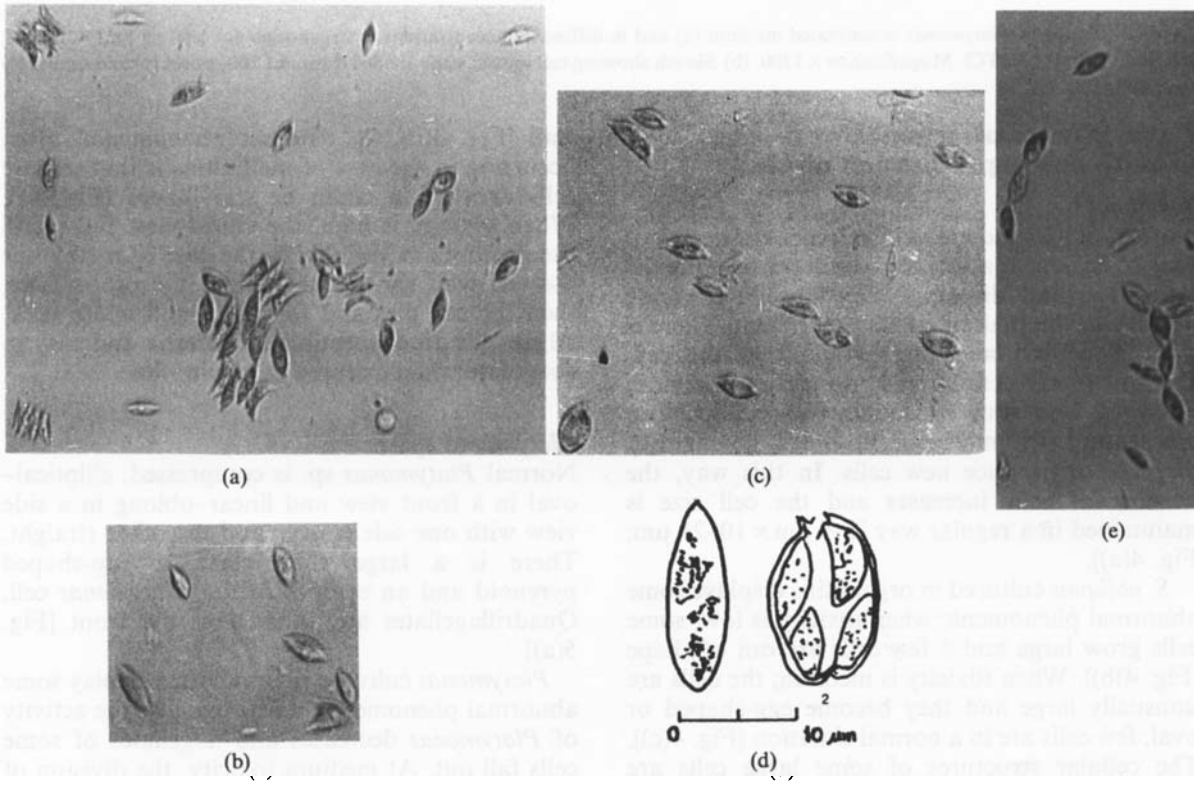


Figure 4 Status of *S. obliquus* in untreated medium (a) and in different concentrations of organotins (b) 1.50 ng Sn l^{-1} TBTCl; (c) 3.00 ng Sn l^{-1} TBTCl; (e) 100 ng Sn l^{-1} TMTCl. Magnification × 1200. (d) Sketch showing cell injury: splitting of the chloroplast (1) and gelation of the mother cell wall (2).

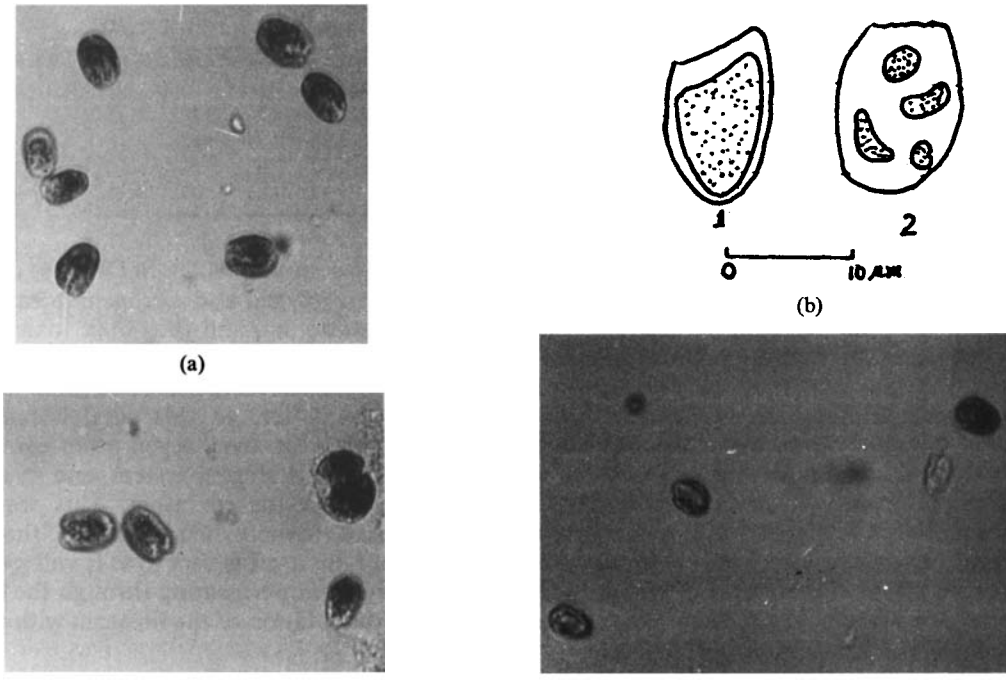


Figure 5 Status of *Platymonas* in untreated medium (a) and in different concentrations of organotins: (c) 1.60 ng Sn l^{-1} TBTCl; (d) 3.00 ng Sn l^{-1} TBTCl. Magnification × 1200. (b) Sketch showing cell injury: semi-divided dormant 200 spores (1) and unusually large cells (2).

Toxic effects of organotins on the growth and reproduction of algae

S. obliquus

Normal *S. obliquus* exists in colonies composed of four or eight cells attached to each other along the long axis, and disperses into unicellular mode because of the shaking in the experiment. There is one chloroplast and one pyrenoid in the cell. During reproduction, the protoplasm divides crosswise into four zoospores. When zoospores are mature, the mother cells break down; they disperse to produce new cells. In this way, the number of cells increases and the cell size is maintained in a regular way [3–9 μm × 10–21 μm; Fig. 4(a)].

S. obliquus cultured in organotins displays some abnormal phenomena: when toxicity is low, some cells grow large and a few cells are out of shape [Fig. 4)b)]. When toxicity is medium, the cells are unusually large and they become egg-shaped or oval; few cells are in a normal situation [Fig. 4(c)]. The cellular structures of some large cells are injured: the chloroplast splits into several small pieces [Fig. 4(d), 1], and mature zoospores cannot disperse because of the gelation of the mother cell wall [Fig. 4(d), 2]. Another phenomenon often occurring in the case of methyltins is that mature cells arrange in chain or star-shapes (Fig. 4e). When toxicity is high, the chloroplast fades and the protoplasm shrinks. In the case of methyltins the cell wall shrinks as well as the protoplasm, then the cell dies and forms a small white stick. Algal cells are susceptible to bacteria and easy to coagulate when cultured in organotins.

Platymonas sp.

Normal *Platymonas* sp. is compressed, elliptical–oval in a front view and linear–oblong in a side view with one side convex and the other straight. There is a large chloroplast, a cup-shaped pyrenoid and an eyespot in the *Platymonas* cell. Quadriflagellates are situated at the front [Fig. 5(a)].

Platymonas cultured in organotins display some abnormal phenomena: at low toxicity, the activity of *Platymonas* decreases and flagellates of some cells fall out. At medium toxicity, the division of cells is inhibited. Dormant zoospores are in semi-divided states [Fig. 5(b), 1], or two dormant spores exist in one mother cell [Fig. 5(c)]. In the case of

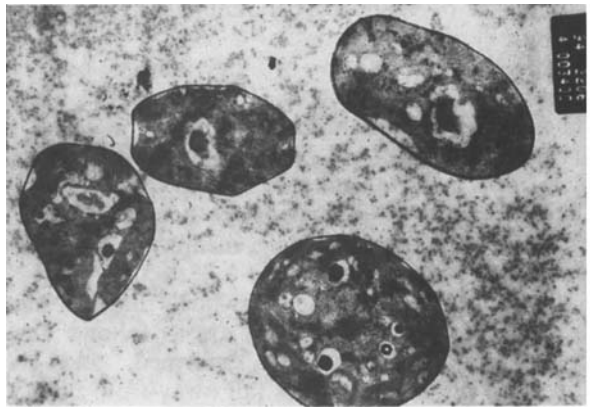


Figure 6 Different sectional structures for normal cells of *Platymonas*.

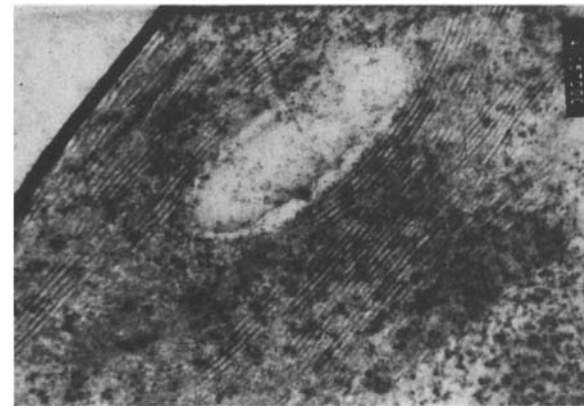


Figure 9 Chloroplast of the normal *Platymonas* sp. cell.

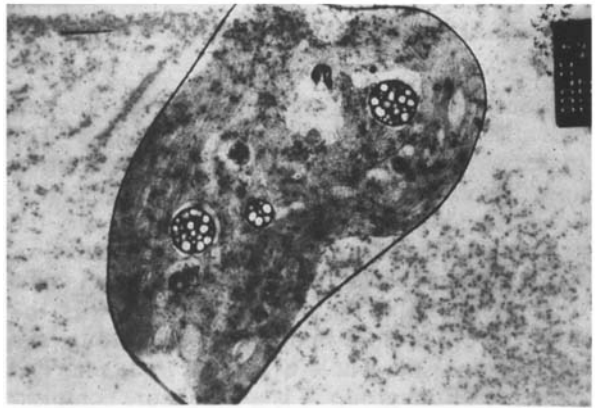


Figure 7 Separation of cell wall and cytoplasm in an injured platymonas cell.

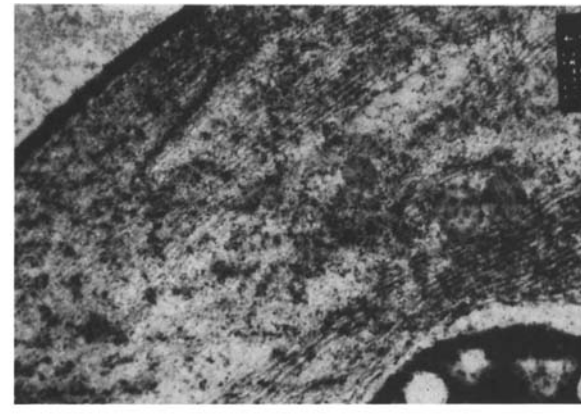


Figure 10 Chloroplast of the exposed *Platymonas* sp. cell.

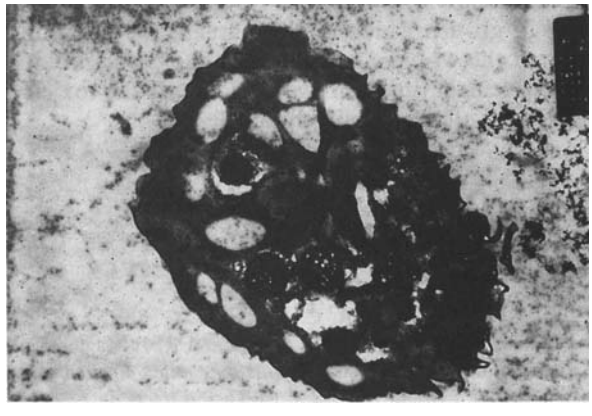


Figure 8 Dead *Platymonas* cell.

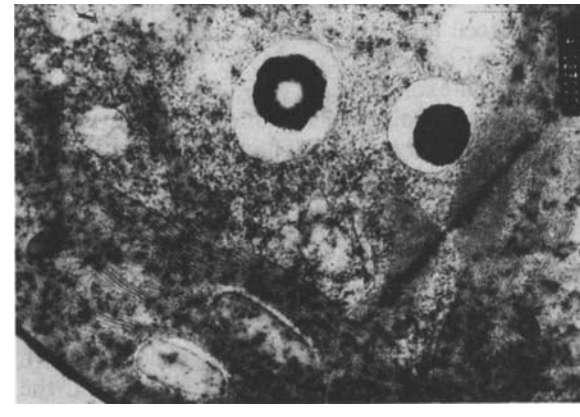


Figure 11 Chloroplast, mitochondrion and endoplasmic reticulum of the normal *Platymonas* cell.

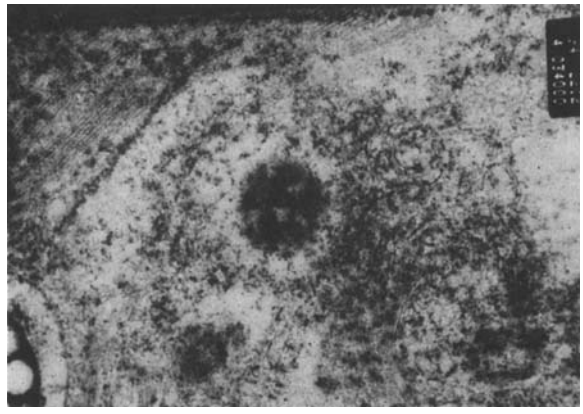


Figure 12 Chloroplast, mitochondrion and endoplasmic reticulum of the exposed *Platymonas* cell.

tripropyltin and methyltins, unusually large cells occur, and cellular structures in these cells are injured [Fig. 5(b), 2]. At high toxicity, organotins directly affect mother cells to produce dormant spores [Fig. 5(d)]. Shrinkage of protoplasm occurs, and interior structures decompose. The cell dies and forms white empty walls. *Platymonas* cultured in organotins are also susceptible to bacteria and easy to coagulate.

Destruction of submicrostructure of Platymonas by TBTCl

There have been few reports on the destruction of submicrostructures of algae by organotions; however, this is very important in the elucidation of toxicity mechanisms. We observed the cells of unexposed and TBTCl (0.2 ppb)-exposed *Platymonas* by electron microscopy. Figures 6–8 are electron micrograms of a normal cell, a TBTCl-exposed cell and a dying cell, respectively. There are several obvious examples of a plasmolysis in the TBTCl-exposed cell (Fig. 7). The exposed *Platymonas* is pale, which is due to the destruction of chlorophyll synthesis. The dead cell is deep in colour. The cell wall shrinks with the cytoplasm and main organelles have dissolved (Fig. 8).

The main organelles of the exposed *Platymonas* cells are all influenced to some extent by TBTCl. First, the chloroplast of the normal *Platymonas* is thick and deep-coloured. The structure of the stroma is compact and in perfect order (Fig. 9). When *Platymonas* sp. is exposed to TBTCl, the stroma lamellae turn loose, intermittent and vague (Fig. 10). Furthermore, the chloroplast is pale in colour, for triorganotins can inhibit the phosphorylation in photosynthesis.

The mitochondrion of the normal cell is dumbell-shaped and dark in colour (Fig. 11). The mitochondrion in an exposed cell swells up. The whole structure of the mitochondrion is deformed and its contents disappear (Fig. 12). This is because TBTCl can affect the membrane of the mitochondrion, causing membrane swelling and increasing penetrability.

The other phenomena which have been observed in exposed samples are as follows: the endoplasmic reticulum is deformed, and in disorder; the ribose bodies fall away from it; the membrane of the nucleus is deformed and becomes rough; the Golgi body is also deformed and internal structures are destroyed.

CONCLUSION

Various species of organotins have different toxicities towards the growth of algae. The toxicity depends not only on the substitution and the properties of R, which have been well-documented in references, but also on the property of the X group. For different substitution, the toxicity is in the order tri-$\gg$ di-$\geqslant$ mono-organotins. Within the same substituent series, the larger and the more lipophilic the R group, the more toxic is the whole organotin molecule. The influence of the group X is a little more complex: when X is a small, easily ionizable group, it has little effect on the toxicity. However, when it is a large organic group, it does change the bioactivity of the organotin. Large organic X groups change the toxicity of organotins through changing the polarizability of the central tin atom. Thus both R and X groups which could increase the lipophilicity of the whole compound, or the polarizability of the central tin atom, would increase the toxicity of the whole organotin molecule.

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