Mechanism of Acute Toxicity of Linear Alkylbenzene Sulfonate (LAS) in Carp

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To evaluate the mechanism of acute toxicity of Linear Alkylbenzene Sulfonate (LAS) in freshwater teleosts, I exposed carp (*Cyprinus* carpio) to LAS at sublethal concentration, 4.0mg/ℓ for 48h and compared the hematological parameters and branchial histology with those in control fish.

In the LAS-exposed fish, severe injuries in the gills were clearly observed upon histological observation. And hematological evaluation revealed dysfunctions in both external respiration and excretion of metabolites attributable to injury of the gills. Furthermore, intense hemolysis was observed in all fish, indicating that the respiration was also impaired by the hemolysis. In contrast to the respiratory dysfunction, increased blood erythrocyte count was noted. In osmoregulation, hepatopancreas function or kidney function, no abnormality was observed.

These results suggest that the acute toxic symptoms of LAS in freshwater teleosts appear in the form of respiratory and excretory dysfunctions at the gills, and that the death of fish exposed to LAS at lethal concentration is due to these dysfunctions.

In Shiga Prefecture, the use of detergents containing phosphorus has been prohibited by "Ordinance to prevent Eutrophication of Lake Biwa in Shiga Prefecture" since July, 1980. In response to the ordinance, home soap consumption instead of synthetic detergents increased. However, after phosphorus-free synthetic detergents were put on the market, the conversion from soap to these detergents in home use has gradually increased.*

A surfactant commonly contained in these detergents is linear alkylbenzene sulfonate (LAS). This material is readily degraded by bacterial attack, and partial biodegradation significantly reduces its specific toxicity ; [1-3] it is considered that LAS has no effect on aquatic life. However, intact LAS flows in an uninterrupted path from home drainage pipe through rivers directly into Lake Biwa because municipal sewage systems around the lake are incomplete. Thus, the deleterious impact of surfactant on aquatic life is a concern. Therefore, the author investigated the effects. In one experiment in the series, a few findings relevant to the mechanism of the acute toxicity were obtained by the observation of hematological and changes in carp exposed to LAS at sublethal concentration. The author wishes to present those findings in this report.

Materials and Methods

Experimental surfactant Sodium n-Dodecylbenzene sulfonate (LAS) made by Wako Pure Chemical Industries, Ltd. was used.

Experimental fish Carp (*Cyprinus* carpio) 335 - 400mm in standard body length and 1,100-1,600 g in body weight were used. The fish were reared at a commercial farm in Shiga Prefecture, and transported to Shiga Prefectural Fisheries Experimental

* Shiga Prefecture : Survey and Investigation on the Environmental Impacts of Synthetic Detergents-Report of Task committee (Chairman:T.Sueishi), 1984.

Station, where they were fed artificial diet for carp culture and kept in a tank 5 × 2 × 0.5m (depth) containing air-saturated running well water for about six months prior to the experiment. Surfactant was never detected in the form of methylene blue active substance (MBAS) in this well water.

LAS exposure The experimental fish were exposed to LAS for 48h in the apparatus schematically doagrammed in **Fig. 1.** In this apparatus, the dense LAS solution prepared with distilled water together with the well water as diluent was added through the mixing tube into the test chamber at the rate of 60mℓ/h. Distilled water was added to the control chamber in place of the dense LAS solution in the same manner as described above. The properties of the well water used as diluent are shown in **Table 1.** LAS was never detected as MBAS in this diluent. The flow rate of the diluent into both the experimental and control chambers was adjusted to 180 ℓ /h. The LAS concentration in the experimental chamber was adjusted to 4.0mg/ℓ, which is 63% of the 48h-LC_{50} (6.3mg/ℓ) in carp,[4] by setting the concentration of the dense LAS solution at 12,000 mg/ℓ. After food was withheld for one day, eight fish were introduced into each chamber from the rearing tank. Water temperature, DO and pH range in the chambers during the experiment was 19-21 degrees Centigrade, 68-81%, and 7.68-7.78, respectively.

Analysis After the exposure to LAS, fish were carefully taken out of the chambers one at a time, and 5-10m ℓ of blood was immediately collected into a heparinized syringe by inserting a needle into the heart thruugh the thoracic wall. Plasma was separated by centrifugation of blood at 3,000rpm for 10min. A few samples of whole

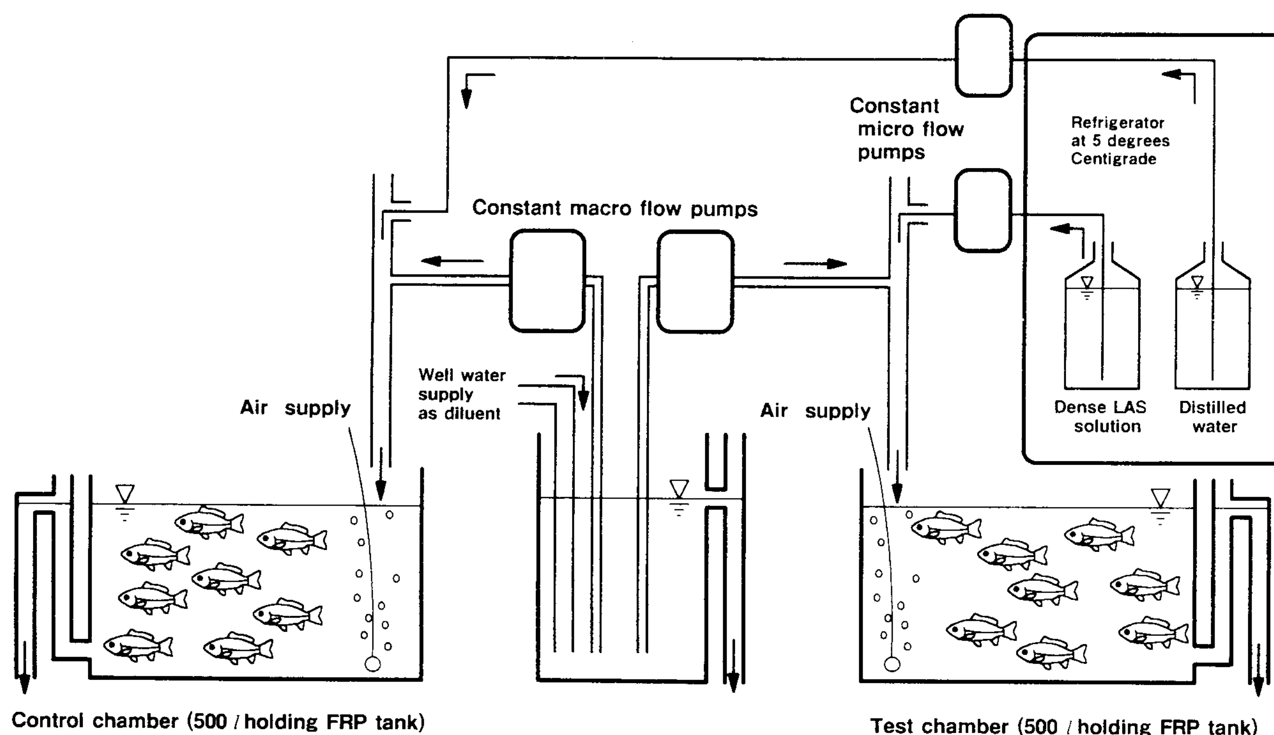

Fig. 1. Shematic diagram of LAS exposure apparatus. Arrows indicate the flow directions.

Table 1. Properties* of the well water used as the diluent in the LAS exposure test

temperature	DO	pH	Ca	Mg	Na	K	MBAS
℃	%		mg/ℓ	mg/ℓ	mg/ℓ	mg/ℓ	
19.6	86.0	7.70	16.3	3.8	16.7	0.5	Not detected

* Values just before the supply into the tanks.

blood were also analyzed. The whole blood and plasma parameters and methods of analysis used are shown in **Table 2.** Hematocrit value and lactic acid level were determined immediately after blood collection, and the other parameters within one day in samples which had been kept at low temperature. After blood collection, fish were killed instantly by medullary amputation, the body weight and standard body length were measured and the center of the fourth gill arch, spleen and hepatopancreas were extracted. The extracted gill was instantly fixed with Carnoys solution for a few hours. The fixed specimen was dehydrated and embedded in paraffin by routine histological techniques. Tissue sections cut at thickness of 5 μ m were stained with hematoxylin-eosin or PAS reaction, and the prepared specimen was then observed by light microscopy.

Table 2. Blood, plasma and hepatopancreas analysis : Parameters ans Methods

Parameter	Abbreviation	Sample	Method
Red blood cell count	RBC	Whole blood	hematocytometer
Hemoglobin	Hb	Whole blood and Plasma	Sodium lauryl sulfate - hemoglobin method*1
Hematocrit value	Ht	Whole blood	Micro - hematocrit method
Mean corpuscular hemoglobin concentration	MCHC	–	Calculated usung the formula : (Hb / Ht) × 10^2
Mean corpuscular volume	MCV	–	Calculated using the formula : (Ht / RBC) × 10^7
Mean corpuscular hemoglobin	MCH	–	Calculated using the formula : (Hb / RBC) × 10^7
Glutamic pyruvic transaminase	GPT	Plasma and hepatopancreas	Reitman - Frankel method*1 : incubated at 25 ℃
Glutamic oxaloacetic transaminase	GOT	Plasma and hepatopancreas	Reitman - Frankel method *1 : incubated at 25 ℃
Lactate dehydrogenase	LDH	Plasma	Cabaud - Wróblewski method*1 : incubated at 25 ℃
Alkaline phosphatase	AlP	Plasma	Phenyl phosphate method*1 : incubated at 25 ℃
Calcium	–	Plasma	*o* - Cresolphtaleincomplexone method*1
Magnesium	–	Plasma	Atomic absorption spectrophotometry
Sodium	–	Plasma	Flame spectrophotometry
Inorganic phosphorus	–	Plasma	Taussky method*1
Chloride	–	Plasma	Schales - Schales method*1
Glucose	–	Plasma	*o* - Toluidine - boric acid method*1
Lactic acid	–	Whole blood	Noll modified method*2
Total cholesterol	–	Plasma	*o* - Phthalaldehyde method*1
Total protein	–	Plasma	Biuret method*1
Albumin	–	Plasma	Bromocresol green method*1
Globulin	–	–	Calculated using the formula : Total protein - albumin
Albumin-globulin ratio	–	–	Calculated using the formula : Albumin / globulin
Urea	–	Plasma	Diacetyl monooxine method*1
Uric acid	–	Plasma	Uricase・4 - aminoantipyrine (4 - AA)・3 - methyl - N - ethyl - N- (β - hydroxyethyl) - anilline method*1
Ammonia	–	Whole blood	Fujii - Okuda modified method*1
Creatinine	–	Plasma	Folin - Wu method*1

*1 Clinical examination kit made by Wako Pure Chemical Industries, Ltd. was used.
*2 A clinical examination kit made by Boehringer Mannheim was used.

The extracted spleen was weighed immediately, and spleen-somatic index (spleen-to-body weight percentage, hereinafter abbreviate SSI) was calculated. The extracted hepatopancreas was rapidly refrigerated, and activities of glutamic oxaloacetic transaminase (GOT) and glutamic pyruvic transaminase (GPT) in the tissue were measured within the day, as follows : the refrigerated tissue was homogenized in a glass homogenizer, the homogenate was diluted 1,000 times (v/v) with distilled water, and the activities per tissue volume were then measured by Reitman-Frankel method. The activities per tissue weight were calculated using the specific gravity measured individually with the homogenate.

Results and Discussion

Hypersecretion of mucus from the body surface and bleeding from the fins and gills were observed in LAS-exposed fish. No symptoms of this sort were observed in control fish. Such symptoms have been observed in other studies of LAS-exposed fish.[5,6] These are apparently typical findings of acute toxicity associated with LAS. No mortality was observed in either the LAS-exposed or control group.

Light micrographs of gill tissue sections are shown in **Fig. 2.** Manifest secondary gill lamellae were observed in the control fish, but, in the LAS-exposed fish, collapse of the tops of the secondary gill lamellae and bleeding from the parts, as well as abnormal proliferation of the basal epithelium between the secondary gill lamellae were clearly noticed. Furthermore, marked congestion was observed between the pavement cells in the partial secondary gill lamellae. Cells positive on PAS reaction, conjectured to be mucous cells, were present

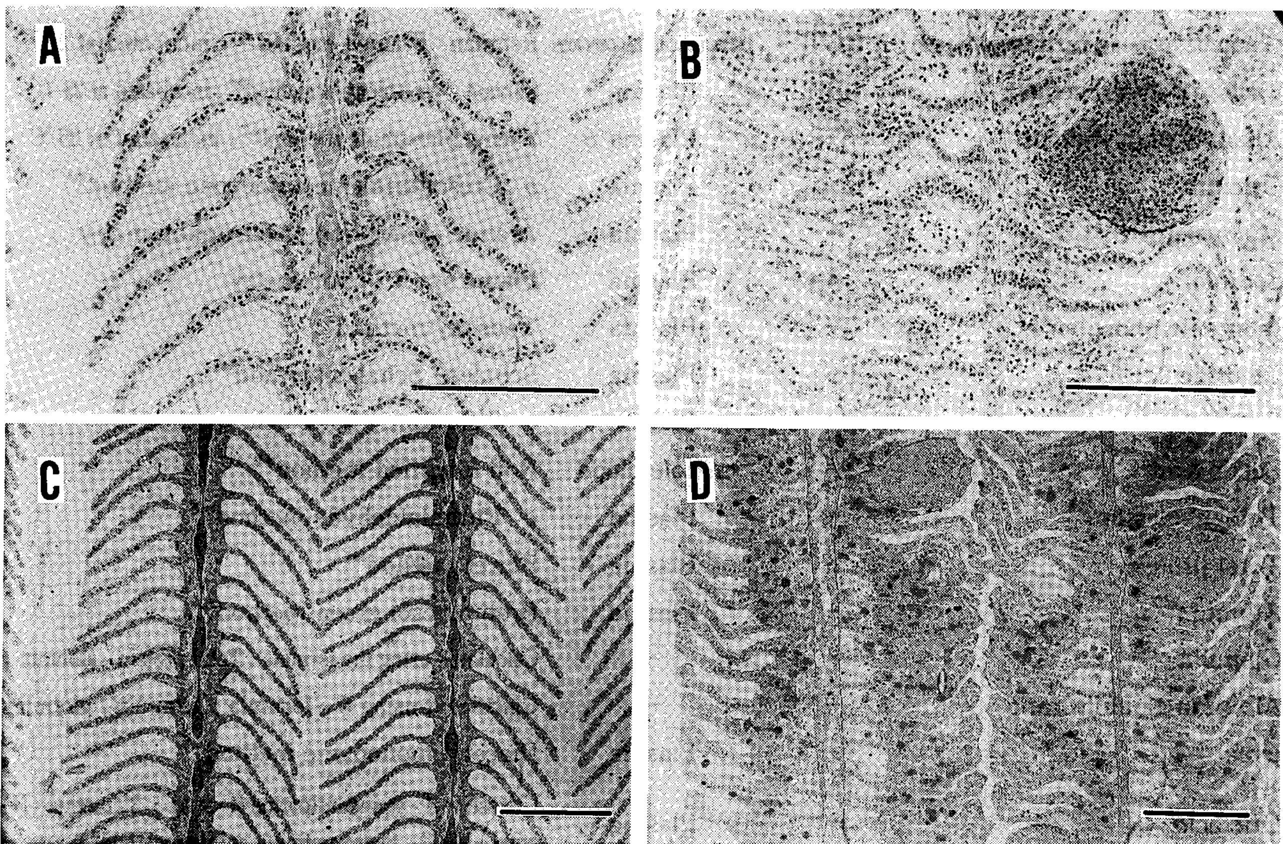

Fig. 2. Light micrographs of the lamellae of carp.
A : Gill lamella of the control fish. Plain secondary gill lamellae are shown. B : Gill lamella of the LAS - exposed fish. Collapse of the tops of secondary gill lamellae, bleeding, abnormal proliferation of the basal epithelia between the secondary gill lamellae and vehement congestion are observed. C : Gill lamellae of the control fish . D : Gill lamellae of the LAS-exposed fish. Mucous cells, positive to PAS reaction, are observed. Bars represent 50 μ m. The section A and B were stained by H. E., and C and D were by H. E. and PAS reaction.

throughout the gills of LAS-exposed fish, but no particular reaction was noted in the gills of control fish. Although the LAS-exposure conditions in the studies were different, almost identical changes of LAS-exposed gills have been observed in several species of fish, including carp,[7] crucian - carp (*Carassius auratus*),[8] goldfish(*C. a.*),[9,10] and bluegill(*Lepomis macrohirus*)[2].

The changes observed in the gills of LAS - exposed fish are typical symptoms of LAS intoxication in fish. In this examination, it is expected that the external respiration and the excretion of metabolites are inhibited by the abnormal changes of gills.

The results of analyses of the plasma, blood and the hepatopancreas and SSI are shown in **Table 3.** The number of fish that has been used for this experiment was restricted by capacity of tanks. The number was smaller to do a statistical analysis. But, in this report, the data that have been obtained with this experiment were statistically analyzed, and an argument was fromed as being based on the result, in principle, then. However, in some cases that differences were not admitted with the analysis also, consideration was done regarding the differences.

The LAS-exposed group showed significantly higher red blood cell count (RBC) and hematocrit value (Ht) than that in the control group ($p < 0.01$ and $p < 0.05$, respectively). Similar findings were observed for hemoglobin (Hb), but this difference was not significant. On the other hand, the LAS - exposed group showed lower SSI than the control group ; this difference was also not significant. The similar finding has been observed in the yellowtail (*Seriola quinqueradiata*) during heavy exercise.[11] In that study, it was inferred that many erythrocytes had been released into the circulating blood from the spleen in response to increased demand for oxygen induced by the heavy exercise. The findings in the present study suggest that the oxygen supply to the tissue in the LAS-exposed fish was obstructed by the diminution of gas exchange area on gills, unfavorable circulation of the blood in the gills, and hemolysis, as described below. The increase in RBC, Ht and Hb and decreased SSI observed in the present study could be accounted for as due to the release of many erythrocytes from the spleen into the circulating blood in compensation for oxygen supply obstruction.

No significant differences between the groups were observed in mean corpuscular hemoglobin concentration (MCHC), mean corpuscular volume (MCV), or mean corpuscular hemoglobin (MCH). However, the LAS-exposed group showed lower mean values of MCV and MCH than the control group. Intense hemolysis was observed in all plasma samples from the LAS - exposed fish but not observed in those from control fish. Hemolysis was reflected in the higher value of plasma hemoglobin in the LAS-exposed group than in the control group. It is well known that surfactants, such as LAS, induce hemolysis and hemoglobin degeneration, [12] and it has been confirmed that LAS is highly concentrated in the gill, gall bladder and blood of fish in bioaccumulation studies using radioactive isotopes.[13] Such facts suggest that the hemolysis observed in the LAS-exposed fish was caused by LAS which had infiltated into the blood, and it is thought that the slightly lower MCV and MCH in the LAS-exposed group than in the control group were due to the partial destruction of erythrocyte accompanying the hemolysis.

Regarding enzymes, no significant difference was observed between the control group and the LAS-exposed group in GPT, GOT, lactate dehydrogenase (LDH) or alkaline phosphatase (A1P) activity in the plasma or in GOT activity in the hepatopancreas. However, the GPT activity in the hepatopancreas was significantly higher in the LAS-exposed group than in the control group ($p < 0.05$). Of these enzymes, GPT and GOT are abundant in the hepatopancreas and the kidney.[14] Accordingly, if these tissues are injured, effluence of these enzymes into the blood and rise in their activities in the blood with ensue. However, no rise in their activities in blood (plasma) was observed in the LAS-exposed ; therefore it is deduced that the hepatopancreas and the kidney of LAS-exposed fish are not damaged severely.

In regard to inorganic content, no significant

Table 3. Comparison of whole blood, plasma and hepatopancreas parameters and spleen-to-body weight percentage in control and LAS-exposed carp

Item*1	Sample	Unit	Mean value ± Standard deviation*3		Judgement*4	Significance level*5
			control carp	LAS exposed carp		
RBC	Whole blood	10^4 · cells/mm^3	144.1 ± 20.6	183.6 ± 28.1	<	0.01
Hb	Whole blood	g/dℓ	9.3±1.4	10.3±1.3		
Hb	Plasma	g/dℓ	0.0±0.1	0.4±0.5		
Ht	Whole blood	%	36.8±4.5	42.0±5.1	<	0.05
MCHC	–		25.3±2.1	24.5±1.4		
MCV	–	μm^3	258±37	231±31		
MCH	–	10^{-9} · mg	64.8±7.9	56.6±7.9		
GPT	Plasma	IU/ℓ	7.6±2.9	8.8±3.4		
GPT	Hepatopancreas	IU/g	9.9±1.4	11.6±1.4	<	0.05
GOT	Plasma	IU/ℓ	10.2±2.6	10.7±3.4		
GOT	Hepatopancreas	IU/g	4.7±1.0	4.6±1.0		
LDH	Plasma	IU/ℓ	191±28	189±81		
AlP	Plasma	IU/ℓ	27.1±15.8	32.2±20.6		
Calcium	Plasma	mg/dℓ	13.8±4.4	14.6±5.4		
Magnesium	Plasma	mg/dℓ	3.00±0.31	2.86±0.32		
Sodium	Plasma	mEq/ℓ	122.6±16.9	125.7±11.6		
Inorganic phoshorus	Plasma	mg/dℓ	6.59±0.68	6.65±0.58		
Chloride	Plasma	mEq/ℓ	112.6±3.5	103.2±10.2	>	0.05
Glucose	Plasma	mg/dℓ	78.3±16.9	143.2±65.4	<	0.02
Lactic acid	Whole blood	mg/dℓ	3.76±1.44	4.17±1.31		
Total cholesterol	Plasma	mg/dℓ	219±28	214±28		
Total protein	Plasma	g/dℓ	3.6±0.3	3.6±0.6		
Albumin	Plasma	g/dℓ	1.7±0.2	1.7±0.3		
Globulin	Plasma	g/dℓ	1.9±0.3	2.0±0.4		
Albumin - globulin ratio	Plasma		0.88±0.18	0.86±0.13		
Urea	Plasma	mg−N/dℓ	3.31±1.57	5.27±1.25	<	0.02
Uric acid	Plasma	mg/dℓ	0.41±0.12	0.48±0.11		
Ammonia	Whole blood	mg/dℓ	648±82	845±174	<	0.02
Creatinine	Plasma	mg/dℓ	0.52±0.16	0.49±0.10		
SSI*2	–	%	0.35±0.08	0.29±0.06		

* 1 Abbreviations are defined in **Table 2.**
* 2 Spleen-somatic index (spleen-to-body weight percentage).
* 3 Each value is mean and standard deviation for eight samples.
* 4 ">" : Significantly lower than the control mean.
"<" : Significantly higher than the control mean.
* 5 By t - test or modified t - test (Cochran - Cox method).

difference between the groups in calcium, magnesium, sodium or inorganic phosphorus content was observed. Since half the fish in eigher group were females with relatively developed ovaries, calcium content of both groups was high. The chloride content of the LAS-exposed group was significantly lower than that in the control group ($p < 0.05$). It is considered that sodium and chloride are indicators of osmotic asynergia in fish. The concentrations of these elements would be decreased upon the influx of external water into the body during rapid osmotic asynergia in freshwater fish. That no significant difference between the groups in sodium concetration was observed in this experiment suggests that osmotic asynergia did not occur in the LAS-exposed fish, whereas the decreased chloride concentration in this group would suggest asynergia. These resullts are contradictory. Therefore, to clarify this question, I exposed carp to 6.0mg/ℓ LAS in isotonic ($\Delta = -0.53$ degree Centigrade[15]) saccharose water for 48h and compared plasma chloride value by the method described above with that in control fish placed in the same isotonic solution. For further comparison, chloride level in fish exposed to 6.0 mg/ℓ LAS in the fresh well water mentioned above was also measured. LAS exposure was by the still water method under the conditions shown in **Table 4.** As shown in **Table 5,** plasma chloride value in the LAS-exposed fish in both the isotonic solution and the fresh water was significantly lower than that of control fish in the respective diluent (both $p < 0.001$). Furthermore, the degree of the decrease in the fish in fresh water was larger than that in the main experiment (**Table** 3). This difference is probably due to the differece in conditions, such as LAS

Table 4. Conditions of LAS exposure*1 in carp*2 in isotonic or fresh water

Diluent	LAS concentration*3	Exporsure period	No. of carp	Volume of solution
	mg/ℓ	h		ℓ
Isotonic saccharose well water*4	0.0	48	8	100
Isotonic saccharose well water*4	6.0	48	8	100
Well water	0.0	48	8	100
Well water	6.0	48	8	100

* 1 Still water method with aeration.
* 2 Body weight : 86 - 140 g.
* 3 Initial value.
* 4 $\Delta = -0.53$ ℃[15]

Table 5. Comparison of plasma chloride value in control carp and LAS-exposed carp in isotonic water and fresh water

Diluent	Mean value of choloride ± Standard deviation*1		Judgment*2	Significance level*2
	control carp	LAS exposed carp		
	mEq/ℓ	mEq/ℓ		
Isotonic saccharose well water	112.4±4.6	74.1±14.3	>	0.001
Well water	116.1±3.1	92.0±9.0	>	0.001

* 1 Each value represents mean and standard deviation for eight carp.
* 2 See **Table 3.**

concentration, exposure methods, and fish size. Osmotic asynergia did not occur in isotonic solution ; it is therefore thought that the decrease of plasma chloride value is caused by a factor other than osmotic asynergia. This finding and the lack of significant decrease in sodium value in LAS-exposed fish suggest that osmotic asynergia,which is caused by the external water invasion, did not occur in the LAS-exposed fish.

Laboratory diuresis, which in known in experimental fishes, is induced by experimental stress, and in fish with laboratory diuresis, abnormally large quantity of chloride is released from the blood into the urine and excreted,[16] and the chloride level in the blood is reduced. It is thought that the decrease in plasma chloride level observed in this experiment was due to laboratory diuresis triggered by LAS-exposure.

Glucose level in the LAS-exposed group was significantly higher than that in the control group ($p < 0.02$), as shown **Table 3**. It is well known that glucose level in fish species is markedly increased by many kinds of stresses.[17-22] The hyperglycemia observed in this experiment clearly indicates that the LAS-exposed fish were under severe stress.

It is known that lactic acid in blood is increased by progressive anaerobic respiration under asphyxia. As there was no significant differece between the groups in this study in whole blood lactic acid level,it is thought that the anaerobic respiration of LAS-exposed fish was not progressing and that they were not in a state of asphyxia.

There was no significant difference between the control and LAS-exposed groups in plasma total cholesterol, total protein, albumin, or globulin level or in albumin-globulin ratio. However, the LAS-exposed group had significantly higher plasma urea and whole blood ammonia than the control group (both $p < 0.02$). As urea and ammonia are excreted into the surrounding water mainly through the gills,[23] it is thought that the increase in urea and ammonia concentration in the blood of the LAS-exposed fish was due to excretory dysfunction associated with the histological changes in the gills described above. On the other hand, uric acid and creatinine are excreted mainly through the kidney in urine.[24] Because no significant decrease in these values or increase in plasma GPT or GOT values in the LAS-exposed fish was observed, it is thought that the kindney function of LAS-exposed fish was not affected.

Synthesizing the experimental results of this paper, the acute toxic mechanism of LAS in this fresh water teleost can be explained as follows. When the fish is exposed to LAS at sublethal concentration, damage occurs mainly in the gills, which disturbs both respiration and excretion functions. Furthermore, the hemolytic anemia impairs respiration. In opposition to the respiratory dysfunction, erythrocytes are released into the circulating blood from the spleen. However, a rise of LAS concentration or prolongation of duration of LAS exposure will induce death caused by respiratory and excretory dysfunctions.

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コイにおける直鎖型アルキルベンゼンスルホン酸塩（LAS）の急性毒性の作用機構

藤原　公一

淡水産硬骨魚類に対する直鎖型アルキルベンゼンスルホン酸塩（LAS）の急性毒性の作用機構を知るため、コイに亜致死濃度のLAS（4㎎/ℓ）を48時間曝露し、血液性状と鰓組織像を対照魚と比較した。その結果、曝露魚には著しい鰓の損傷がみられ、血液性状からはその損傷が原因となった外呼吸障害と排泄障害が認められた。さらに全曝露魚には溶血がみられ、外呼吸障害の進行がうかがわれた。以上から、LASによる急性致死は外呼吸障害と鰓からの排泄障害により起こると考えられた。