



## Determination of the lethal concentration 50% (LC<sub>50</sub>) of lead chloride and its accumulation in different organs of *Gambusia affinis* fish

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### Abstract

In the present research, mosquito fish *Gambusia affinis* have been exposed to lead chloride during 24, 48, 72 and 96 hours in order to evaluate the lead chloride lethal 50 (LC<sub>50</sub>) concentration and the Its residue in certain organs of fish. Usage of the EPA computer software based on Finney Probit Analysis method has been statistically tested for the data collected LC<sub>50</sub> values of *G. affinis* if 24, 48, 72 and 96 hours were found to be 59.4, 55.9, 51.1 and 49.0 mg/L, respectively. LC<sub>50</sub> decreased as mean exposure times. 20 fish were placed in each concentration of four sublethal concentrations 20 and 25 mg/L for two acute periods 24 and 96 hours as well as 10 and 5 mg/l for chronic periods 15 and 30 hours. The testes were carried out as three replications, the accumulation of lead in various fish organs was determined by Atomic Absorption Spectrophotometer. The finding revealed that the accumulation of PbCl<sub>2</sub> on different organs of *G. affinis* be time dependent fashion and Pb-content in organs increased significantly time dependent at chronic exposure as compared as acute- exposure.

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### Introduction

Owing to their toxicity, heavy metal is considered to be environmental contaminants, persistence in the ecosystem and bio accumulative existence (1). It is characterized as a metal with density is bigger than 5 g/cm<sup>3</sup> and their atomic weight is 63.5-200.5 g/mol (2). In the environment there are many sources of heavy metals for example lead, arsenic, mercury, cadmium, selenium, nickel, chromium, and copper lead, heavy metals in general, are commonly categorized as foundation and non-core minerals (3). Heavy metals are an important source of pollution because of their toxic nature and their capacity to accumulate (4). The potential of heavy metals to build up in the aquatic environment due to its failure to decompose (5). In different manufacturing methods Lead is a commonly used metal in various industrial processes and in the marine world is very persistent (6). Lead

is a highly poisonous metal, and its routine use has caused excess contamination of the environment and associated health problems in many parts of the world (7). Fish have been recognized as the main aquatic organisms that accumulate considerable amounts of certain metals exceeding their concentrations in the aquatic ecosystem (8). Individual growth rates, physiological functions, reproduction and mortality in fish (9). Heavy metals penetrate fish bodies in 3 possible ways: through gills, through the digestive track and through the surface of the body. The gills are known to be the important site of direct absorption of water metal (10,11), Although it is generally calculated the body surface take a small part in absorption of heavy metals in fish (12). Levels in fish usually reflect levels found in sediment and water of the particular aquatic environment from which they are sourced (3) levels in fish usually reflect levels found in sediment and water of the

particular aquatic environment from which they are sourced (13). At the end of the aquatic food chain fish may collect heavy metals and transfer them through the food chain to people who cause acute or chronic diseases (14). The purpose of the present study was to determine the LC<sub>50</sub> of lead chloride in acute and chronic treatment of mosquito fish lead accumulation in various *G. affinis* organs.

## Materials and methods

Fishes were collected from the Tigris River banks on the left side of Mosul, Iraq. Experimental fishes which measured an average length 2.025 cm and weighed 0.18 g experimental fish have been exposed to metal concentration. First phase of repair laboratories included a quarantine the duration during which the fish were acclimatized to the requirements of the laboratory for the experiment for two weeks at least. The animals were housed in glass aquariums filled with dechlorinated tap water. There were 16:8 pgotoperoids (16 h. light/8 h darkness) and fish were fed twice daily with commercially available fish food.

Ten fish have been placed in clean tap water, acting as monitors, and there were 10 fish put in aquariums 25\*25\*30 cm at 20 cm in depth; 20 and 25 mg/L for chronic exposure and chloride (PbCl<sub>2</sub>) for acute and every procedure had 10 replicates. Animals were then traded for 10 and 5 mg/L PbCl<sub>2</sub> as lead quantitative metal head, liver, gills, muscle, and, intestine dissected. In Pyrex test tubes, tissues of fish organs have been dried at a stable weight of 48 hrs at 60°C. Analysis was carried out according to the procedure described by (15). Dried tissues with concentrated sulphoric acid and perchloric acid were weighed and digested at 350 rpm. When the gases were white and clear the solution was transparent, the specimens were cooled to room temperature and ten ml of tubes filled with ultra-pure water. All samples were analyzed a graphite furnace AAS technique is used to assess the PbCl<sub>2</sub> concentration (ZEEnit700). Triplicate samples were analyzed. The coefficient of variance was generally less than 10 percent. Metal concentrations in the tissues there were measured on a dry basis of weight and express as dry weight of µg/g.

## Conditions

Metal toxicity testing was carried out under laboratory conditions. This experiment was conducted with six metal treatments in a fully randomized system. A glass tank was for water substitution. Fish density was 10 fish per tank. Stock solutions of lead chloride a were prepared in double distilled water by dissolving the analytical grade PbCl<sub>2</sub>. 10 Fishes were per concentration.

## LC<sub>50</sub> determination

For determination of the LC<sub>50</sub> values, the following ranges were tested, six PbCl<sub>2</sub> 10, 20, 40, 60, 80 and 100 mg/L concentrations were chosen for mosquito fish. Metal solutions were prepared by dilution of a stock solution with dechlorinated tap water. A control with dechlorinated tap water only was also used. The number of dead fish was counted every 12 hours and removed immediately from tanks. The mortality rate was determined at the end of 24, 48, 72 and 96 hours. The acute toxicity test was conducted in accordance with standard methods (16).

## Statistical analysis

The acute toxic effect of lead chloride on mosquito fish was calculated in this study by the use of Finney's method of determining Probit Analysis LC<sub>50</sub> (17). In order to Graphpadprism 5. The statistical significant differences between different treatments and control are reported by different letters a, b, c, d. The values with different letters in the same row are significantly different (Tukey test,  $P \leq 0.05$ ).

## Result

Acute lead toxicity shows that mortality is directly proportional to the heavy metal lead chloride concentration while the mortality rate is virtually absent in the control (Tables 1-5).

Table 1: Correlation between lead chloride concentration and mortality rate for mosquito fish on time (24 -96 h)

Concentration of PbCl <sub>2</sub> (mg/l)	Mortality rate (%) on time (24-96)				
	N	24 hours	48 hours	72 hours	96 hours
0.0	10	0	0	0	0
10.0	10	0	0	0	1
20.0	10	2	2	2	2
40.0	10	3	3	3	4
60.0	10	4	4	4	5
80.0	10	5	5	6	7
100.0	10	9	10	10	10

Table 2: The correlation between the concentration of lead chloride and *G. affinis* mortality rate(24hours)

Concentrations of PbCl <sub>2</sub> (mg/L)	Amount of the exposed fish	Number of deadly fish	Death in the place Bioassay	Expected death	Estimation death
10	10	0	0.0	0.0	0.0209
20	10	2	0.2	0.2	0.1068
40	10	3	0.3	0.3	0.3255
60	10	4	0.4	0.4	0.5043
80	10	5	0.5	0.5	0.6328
100	10	9	0.9	0.9	0.7237

Table 3: The linkage between the concentration of lead chloride and *G. affinis* mortality rate for 48hours

Values of PbCl <sub>2</sub> (mg /L)	Count of exposed fish	Totalr of dead fish	Death in the site Bioassay	Death Suspected	Estimating death
10	10	0	0.0	0.0	0.0155
20	10	2	0.2	0.2	0.0986
40	10	3	0.3	0.3	0.3368
60	10	4	0.4	0.4	0.5346
80	10	5	0.8	0.8	0.6727
100	10	10	1.0	1.0	0.7664

Table 4: The interaction between the PbCl<sub>2</sub> concentrations and *G. affinis* survival rate (72 hours)

Levels pbcl <sub>2</sub> (mg / L)	Number of fishthat are exposed	Amount of fish dead	Death at the assay system	Death Awaited	Estimating Fatality
10	10	0	0.0	0.0	0.0130
20	10	2	0.2	0.2	0.0962
40	10	3	0.3	0.3	0.3516
60	10	4	0.4	0.4	0.5630
80	10	6	0.6	0.6	0.7059
100	10	10	1.0	1.0	0.7991

Table 5: The association between lead chloride concentration and mortality rate of *G. affinis* (96 hours)

Quantities of PbCl <sub>2</sub> (mg/L)	Number of fish reported	Number of killed fish	Killing at the assay system	Dying Expected	Analyzing Fatality
10	10	1	0.1	0.1	0.0040
20	10	2	0.2	0.2	0.0629
40	10	4	0.4	0.4	0.412
60	10	5	0.5	0.5	0.5974
80	10	7	0.7	0.7	0.8584
100	10	10	1.0	1.0	0.7664

### LC<sub>50</sub> of lead for mosquito fish

Mosquito fish susceptibility to the impact of lead toxicity was found to increase mortality with an increase in lead concentration, while mortality was virtually absent in the control (Table 1). Analytical results showed that the mean lethal concentration (LC<sub>50</sub>) of lead to mosquito fish for exposure was 24, 48, 72 and 96 hours 59.443, 55.978, 53.256 and 500.514 mg/L respectively. It soon became evident that an improvement in the duration of exposure led to a rise in mortality (Table 6). Fish are capable of acquiring and both

active ingredients absorb metals from water and passive procedures in their bodies; the accumulation of metals in fish tissues is also based on metal absorption, tissue distribution and deposition. In our research, the amounts of lead accumulated in *Gambusia affinis* tissues varied dependent on the time of exposure, the concentrates and the form the tissue. Atomic absorption analysis showed that the *Pb* content in different measured organs increased significantly based on time and PbCl<sub>2</sub> concentration. In this analysis, we observed variations in PbCl<sub>2</sub> accumulation between control

and treatment. Our results indicated that PbCl<sub>2</sub> the accumulation in tissues of the brains > gill > intestine > liver > muscles in fish exposed to lead chloride 20 mg/L, period 96 hours of significant differences and more impact than 24 hours and control fall (Table 7). Similar letters show the statistically significant differences between different treatment and regulation. There are significantly different values of different letters in the same row (Tukey test, P≤0.05) Different letters. show statistically significant differences between different treatments and regulation. The values with different letters in the same row differ significantly. We also observed variations in PbCl<sub>2</sub> accumulation between control and treatment. Our research found that PbCl<sub>2</sub> collection in tissues in gills the order of the > liver > intestine > brain > muscle in fish exposure to lead chloride 25 mg/L, period 96hours significant differences and greater impact than 24hours and control fall (Table 8). Whereas the concentration of lead chloride 5 mg/L shows the gills sequence > liver, intestine > brain > muscles. There is no significant difference between The period of 30 days and 15 days while both of two period differ from control (Table 9). Finally, the concentration of lead chloride 10 mg/L shows the sequence gills > intestine > liver > brain > muscles. The

accumulation of lead chloride in the period 30 is greater and significant difference between The period 15 days control groups while both of two period differ from control (Table 10). Similar letters show the statistically significant differences between different treatment and regulation. There are significantly different values of different letters in the same row (Tukey test, P≤0.05) Different letters. show statistically significant differences between different treatments and regulation. The values with different letters in the same row differ significantly.

### Discussion

Results of the present study showed that the LC<sub>50</sub> value of 24, 48, 72 and 96 hours of lead exposure in Gambusia were 59.443, 55.978, 53.256 and 50.514 mg/L respectively, no death among the control group. The use of the LC<sub>50</sub> test as a general indicator of chemical toxicity has become popular and has been questioned as inaccurate and in sightful criteria for decades. It is thus useful to reconsideration the repeated identification of the LC<sub>50</sub> before conducting laboratory tests (9).

Table 6: Lethal concentration and upper and lower limits of lead chloride period for mosquito fish

Point	Concentration levels of PbCl <sub>2</sub> (mg/ L), (95 % limits of trust)			
	24 hours	48 hours	72 hours	96 hours
LC <sub>50</sub>	59.443 (42.349-91.876)	55.978 ( 40.427-80.615)	53.256 (38.668-73.847)	50.514 (38.273-66.973)

Table 7: Concentration of lead chloride (µg/mg) dry weight in various *G. affinis* organs exposed to 20 mg/L at 24 hours and 96 hours exposure period

Time	Brain	Gills	Liver	Intestine	Muscles	Effect of time
control	0.018±0.002 <sup>a</sup>	0.041±0.001 <sup>dklf</sup>	0.038±0.001 <sup>f</sup>	0.015±0.001 <sup>ac</sup>	0.015±0.001 <sup>ag</sup>	0.254 <sup>A</sup>
24h	0.160±0.01 <sup>b</sup>	0.092±0.002 <sup>e</sup>	0.099±0.05 <sup>i</sup>	0.124±0.04 <sup>k</sup>	0.021±0.01 <sup>am</sup>	0.0992 <sup>B</sup>
96h	0.202±0.002 <sup>c</sup>	0.347±0.13 <sup>f</sup>	0.190±0.02 <sup>j</sup>	0.218±0.055 <sup>l</sup>	0.025±0.01 <sup>mn</sup>	0.196 <sup>C</sup>
Effect of organs	0.126 <sup>A</sup>	0.16 <sup>B</sup>	0.109 <sup>C</sup>	0.119 <sup>AC</sup>	0.0203 <sup>C</sup>	

Table 8: Concentration of lead chloride 25 mg/L dry weight in the various organs of *G. affinis* at 24 hours and 9 6hours

Time	Brain	Gills	Liver	Intestine	Muscles	Effect of time
control	0.018±0.002 <sup>a</sup>	0.041±0.011 <sup>dklf</sup>	0.038±0.01 <sup>f</sup>	0.015±0.03 <sup>ac</sup>	0.015±0.021 <sup>c</sup>	0.02 <sup>a</sup>
24 h	0.193±0.003 <sup>b</sup>	0.400±0.1 <sup>d</sup>	0.231±0.001 <sup>igc</sup>	0.240±0.01 <sup>i</sup>	0.03±0.021 <sup>k</sup>	0.218 <sup>b</sup>
96 hr	0.236±0.01 <sup>c</sup>	0.415±0.005 <sup>e</sup>	0.319±0.001 <sup>h</sup>	0.265±0.11 <sup>j</sup>	0.04±0.022 <sup>l</sup>	0.255 <sup>c</sup>
Effect of organs	0.149 <sup>a</sup>	0.285 <sup>b</sup>	0.196 <sup>c</sup>	0.173 <sup>d</sup>	0.028 <sup>e</sup>	

Table 9: Concentration of lead chloride 5 mg/L dry weight in different *G. affinis* organs at 15and 30 days exposure period

Time	Brain	Gills	Liver	Intestine	Muscles	Effect of time
Control	0.018±0.002 <sup>a</sup>	0.041±0.001 <sup>d</sup>	0.038±0.01 <sup>df</sup>	0.015±0.01 <sup>a</sup>	0.015±0.005 <sup>a</sup>	0.0254 <sup>a</sup>
15 days	0.288±0.01 <sup>b</sup>	0.465±0.004 <sup>ie</sup>	0.380±0.1 <sup>g</sup>	0.324±0.2 <sup>l</sup>	0.043±0.022 <sup>dk</sup>	0.3 <sup>b</sup>
30 days	0.297±0.01 <sup>c</sup>	0.487±0.002 <sup>f</sup>	0.397±0.11 <sup>h</sup>	0.462±0.12 <sup>j</sup>	0.066±0.033 <sup>l</sup>	0.2624 <sup>bc</sup>
Effect of organs	0.201 <sup>a</sup>	0.331 <sup>b</sup>	0.271 <sup>c</sup>	0.267 <sup>c</sup>	0.041 <sup>d</sup>	

Table 10: Concentration of lead chloride 10 mg/L dry weight in different *G. affinis* organs at 15 and 30 days exposure period

Time	brain	gills	liver	intestine	muscles	Effect of time
control	0.018±0.002 <sup>a</sup>	0.041±0.01 <sup>ag</sup>	0.038±0.01 <sup>abg</sup>	0.015±0.01 <sup>ab</sup>	0.015±0.005 <sup>ab</sup>	0.0254 <sup>a</sup>
15 days	0.334±0.13 <sup>ab</sup>	0.642±0.18 <sup>bg</sup>	0.424±0.14 <sup>abg</sup>	0.521±0.13 <sup>bceg</sup>	0.073±0.022 <sup>abg</sup>	0.3988 <sup>b</sup>
30 days	0.431±0.16 <sup>g</sup>	0.720±0.19 <sup>beg</sup>	0.528±0.22 <sup>bdg</sup>	0.714±0.24 <sup>bfcg</sup>	0.1±0.0055 <sup>abeg</sup>	0.4986 <sup>c</sup>
Effect of organs	0.261 <sup>a</sup>	0.467 <sup>b</sup>	0.33 <sup>c</sup>	0.417 <sup>d</sup>	0.188 <sup>e</sup>	

For all tissues, the PbCl<sub>2</sub> concentration was lower in the control group than in the treatment group at the periods 24, 15 and 30 days. It is well known that heavy metals like Cd and Pb are potentially deposited in marine organisms and sediments, where they are subsequently passed to humans via the food chain (18). The influence of the metal depends on the animal, size and the form of the species. Although the organisms survive the initial attack of pollutants due to their defense adaptations, the injuries caused by the gradual exposure will be observed even in small doses at later stages when the resistance of the organism is reduced due to ageing. In addition, the test organism's state and reaction to the amount of metal entering its body, the degree of retention and the rate of excretion affect the toxic effect of heavy metal (7). We studied lead accumulation in the brain, gill, liver, intestine, and muscles tissues of mosquito fish. The tendency of each organ to accumulate lead chloride in this study was brain > gills > intestine > liver > muscle in fish exposure to lead chloride 20 mg/L, period 24 and 96 hours, while the concentration of PbCl<sub>2</sub> in fish exposure to 24 mg/L gills > liver > intestine > brain > muscle. The accumulation of lead chloride in *G. affinis* exposure to 5 mg/L was gills sequence > liver, intestine > brain > muscles and in the concentration 10 mg/L was gills > intestine > liver > brain > muscles. The accumulation of metal in fish tissues depends on the concentration and time of exposure, as well as other factors such as temperature, age, contact with other metals, water chemistry and fish metabolic activity (19). Pb is a non-essential factor and in aquatic species close to anthropogenic sources, high concentration can occur. Even at low concentrations, it is toxic and has no known role in biochemical processes (7). Fish have the capacity to store heavy metals in their tissues to higher levels than environmental concentration through absorption along the gills surface and kidney, liver and gut tract wall (20). The present study revealed that the lead was collected in the various fish organs, the highest concentration of lead was detected in the gills tissue of *G. affinis*, while in the muscle tissue the lowest was found. Gills has the highest levels of lead accumulation, this finding was being contradictory to the work of Mahboob *et al* who reported lead was greater accumulation in gills, kidney, liver and muscles in *Cyprinus carpio* (21). Gills are important location for heavy metals to join (22); and it is the first target organ for fish exposure. The large concentration of metals in the gills is related to complexation of metals with the mucus, which is difficult to

extract from the tissue completely prior to the analysis. The metal concentration in the gill shows the level of the metals in waters where fish live, while the concentration in kidney and liver contraction reflect the storage of metals. Therefore, gills in fish are more commonly recommended as environmental markers of fish organs (23). Of great significance is the deposition of Lead in fish tissues. Lead in the gills has genotoxic and cytotoxic damage (24).

Liver was the target organ for Pb accumulations (25). Many experiments have shown that the liver accumulates more metals than other tissues. The bioaccumulation of metals in liver may be linked to its function of metabolism, liver is the primary detoxifying organ and the target for the accumulation in fish of most metals (26,27). The accumulation of metals in the liver is a very rapid process suggesting the presence of a non-saturable ion channel and lysosomal system where metals usually arrived coupled to metallothioneins (7). Metallothioneins (MTs) are proteins bound to metal that are upregulated upon metal exposure. In contrast, MTs can attenuate the detrimental effects of the metal to the system of the organism to a certain extent and concentrations of exposure (28,29). Also, (30) reported the accumulation of Pb in the Pangus fish has the decreasing order of gill > liver > kidney > gonad > muscle.

Lead toxicity is targeted towards processes of brain memory and learning, and can be regulated by three processes. Lead can impair brain learning and memory by inhibiting the N-methyl-d aspartate (NMDAR) receptor and may block neurotransmission by inhibiting the release of neurotransmitters, block the neuronal voltage gated calcium (Ca<sup>2+</sup>) channels (VGCCs) and reduce brain derived neurotrophic factor (BDNF) (31), however it is apparent in this study that there is a relatively little accumulation of Pb in brain tissues compared to the Gills and liver. Similarly, (6) determined that bioaccumulation of lead was Pb liver > gills > kidney > brain > muscles in *Cirrhina mrigala*.

Fish intestines are charged with digestion, nutrient absorption, digested food excretion, and Metabolic active tissues such as gills, liver and kidneys absorb heavy metals in concentrations comparatively higher than the rest of the body's tissues such as skin and muscles (1,28). Muscles are not the primary target for accumulation (6,18). Furthermore, Das *et al* reported the effects of sublethal concentrations exposure of Pb in nervous system (32). After different exposure durations 3-42 days, the accumulation profile of Pb was brain > liver > kidney > gills > muscles > skin. The bioaccumulation of Pb in the Katla fish *Gibelion catla* has

the decreasing order of liver > gill > kidney > gonad > muscle (30).

Among animals, muscle tissue doesn't play a very active role among metal accumulation. In our study the lowest rate of accumulation of lead was in muscle tissue, which is the fish's edible component. Many studies have found the same result (33,34). The low concentrations of metals in the muscle of fish species can indicate the low levels of binding proteins in the muscle (35).

## Conclusions

Present investigation revealed variable toxicity of PbCl<sub>2</sub> to the *G. affinis* in LC<sub>50</sub> and bioaccumulation in some organs of mosquito fish. LC<sub>50</sub> decreased as mean exposure times. All the fish tissues showed significantly variable exposure sublethal concentration and time-dependent greater accumulation of PbCl<sub>2</sub> observed in gills while the trace PbCl<sub>2</sub> was least in the muscles. Lead has harmful health effects even at lower levels, and there is no known safe exposure level.

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## Conflict of interest

The current study involved researchers' collaboration, as the work was done to complete the research findings and compose it with the involvement of all researchers.

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## تحديد التركيز المميت الوسطي لكلوريد الرصاص وتراكمه في أعضاء مختلفة لأسماك البعوض *Gambusia affinis*

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### الخلاصة

في الدراسة الحالية، تم تعريض أسماك البعوض إلى كلوريد الرصاص في الفترات ٢٤، ٤٨، ٧٢ و ٩٦ ساعة لتحديد التركيز القاتل لكلوريد الرصاص لنصف العدد الكلي للأسماك ومخلفاته في بعض أعضاء الأسماك. تم تقييم البيانات التي تم الحصول عليها إحصائياً ووجد أن قيم التركيز القاتل لكلوريد الرصاص لنصف العدد للفترات ٢٤ و ٤٨ و ٧٢ و ٩٦ ساعة في اسماك البعوض كانت ٤,٥٩، ٩,٥٥ ملغم/لتر و ١,٥١ ساعة وكذلك ١٠ و ٥ ملغم/لتر للفترتين التأثير المزمّن ١٥ و ٣٠ ساعة. تم إجراء التجارب باستخدام ٣ مكررات، تم تحديد تراكم الرصاص في أعضاء الأسماك المختلفة عن طريق جهاز الامتصاص الذري. أوضحت النتائج أن تراكم كلوريد الرصاص في الأعضاء المختلفة لأسماك البعوض يكون بطريقة تعتمد على الوقت وأن محتوى الرصاص في الأعضاء ازداد بشكل كبير بالاعتماد على الوقت عند التعريض المزمّن بالمقارنة مع التعريض الحاد.