Int. J. Aquat. Biol. (2026) 14(3): 102-111
ISSN: 2322-5270; P-ISSN: 2383-0956
Journal homepage: www.ij-aquaticbiology.com

Original Article

Developmental toxicity and teratogenic effects of phytic acid on zebrafish (*Danio rerio*) embryos

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Abstract: Phytic acid (myo-inositol-1,2,3,4,5,6-hexakis-dihydrogen phosphate) is a common constituent of oilseeds, legumes, and cereals and is ubiquitously present in plant-based foods. While its antinutritional effects in vertebrates have been well documented, its potential impacts on early embryonic development remain poorly understood. Therefore, this study aimed to investigate the developmental toxicity of phytic acid in zebrafish (*Danio rerio*, ZF) embryos. Developmental toxicity testing was conducted in accordance with OECD Test Guideline 236, in which ZF embryos were exposed to various concentrations of phytic acid and a control treatment. Embryo mortality and developmental abnormalities were evaluated at different hours post-fertilization (hpf). Phytic acid exhibited moderate toxicity to ZF embryos, with an estimated median lethal concentration (LC_{50}) at 96 hpf of 5.0±1.0 ppm. Furthermore, phytic acid-treated embryos and larvae exhibited various developmental deformities, including reduced heart rate, shortened body length, yolk sac edema, tail malformations, delayed hatching, and spinal curvature. Cardiac-related abnormalities, such as pericardial edema and impaired blood circulation, were also observed. Overall, these findings indicate that phytic acid exerts teratogenic effects on zebrafish embryos at certain exposure levels.

Article history:
Received 27 January 2026
Accepted 30 April 2026
Available online 25 June 2026

Keywords:
Developmental toxicity
Teratogen
Phytic acid
Zebrafish embryos

Introduction

Phytic acid (myo-inositol-1,2,3,4,5,6-hexakis-phosphate) is a naturally occurring phosphorus-rich compound that plays an essential role in plants as the primary storage form of phosphorus and inositol (Reddy et al., 1989; Raboy, 2001). In human and animal nutrition, phytic acid contributes to phosphorus homeostasis and has been associated with several beneficial properties, including antioxidant activity and potential protective effects against metabolic disorders. However, when present at high levels, phytic acid acts as an antinutritional factor due to its strong chelating capacity toward essential minerals, thereby reducing their bioavailability (Sandberg, 2002). Phytic acid is widely distributed in oilseeds, legumes, cereals, and nuts, and its extensive presence in plant-based foods leads to broad dietary and environmental exposure.

The antinutritional effects of phytic acid have been extensively documented in vertebrates. Numerous studies have shown that excessive intake of phytic acid can impair mineral absorption, reduce growth performance, disrupt bone mineralization, and decrease digestive efficiency (Vaintraub and Bulmaga, 1991; Caldwell, 1992; Knuckles, 1998). These effects are primarily attributed to the ability of phytic acid to chelate divalent cations such as calcium, iron, zinc, and magnesium, as well as its capacity to interact directly with digestive enzymes, including pepsin, α-amylase, β-galactosidase, and lipase, resulting in reduced enzymatic activity and altered nutrient utilization (Vaintraub and Bulmaga, 1991; Knuckles, 1998). Collectively, these findings indicate that phytic acid is a biologically active compound rather than an inert dietary constituent.

Beyond its nutritional implications, increasing evidence suggests that phytic acid may exert broader biological effects on growth and survival, particularly during early developmental stages. Studies in herbivorous insects have demonstrated that phytic

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DOI: https://doi.org/10.22034/ijab.v14i3.2779

acid acts as an antifeedant, reduces food consumption, suppresses growth rates, and induces mortality in early instars (Jood et al., 1995; Marzo et al., 1997; Modgil and Mehta, 1997). Notably, Green et al. (2000) reported that phytic acid exposure at concentrations naturally encountered in host plant tissues significantly reduced larval growth and caused mortality during early developmental stages, indicating that phytic acid may be toxic under certain exposure conditions.

In addition to its effects on growth and survival, phytic acid has been shown to interfere with detoxification processes. Green et al. (2000) demonstrated that phytic acid inhibited cytochrome P450 monooxygenase–mediated metabolism of xanthotoxin in *Depressaria pastinacella*, a species that relies heavily on P450 enzymes to detoxify host plant allelochemicals. This inhibition was not attributable to reduced iron availability, suggesting a direct effect of phytic acid on detoxification enzymes. Disruption of P450-mediated detoxification pathways may increase susceptibility to toxic compounds and contribute to reduced survival during sensitive developmental periods.

Early embryonic development is a critical window during which exposure to bioactive compounds may cause irreversible structural and functional abnormalities (Divvela et al., 2025). Even substances with relatively low acute toxicity may induce developmental or teratogenic effects by disrupting tightly regulated processes such as mineral homeostasis, enzyme activity, organogenesis, and physiological maturation (Selevan et al., 2000; Hill, 2002). Given its strong chelating properties, documented interactions with enzymes, and demonstrated toxicity during early life stages in other organisms, phytic acid may pose a potential risk to embryonic development.

Despite substantial evidence of the biological activity of phytic acid in insects and adult vertebrates, its potential effects on early embryonic development in vertebrate models remain poorly understood. In particular, experimental data on the developmental and teratogenic effects of phytic acid during early life stages remain limited. This knowledge gap highlights the need for systematic evaluation of phytic acid–induced developmental toxicity using appropriate vertebrate models and standardized testing approaches.

Zebrafish (*Danio rerio*, ZF) are an established vertebrate model for developmental toxicity studies due to their small size, low maintenance cost, and well-characterized embryogenesis (Gautam et al., 2024). Developmental endpoints such as survival, growth, cardiac function, and morphology are widely used in toxicity assessments. In addition, the availability of a fully sequenced genome, genetic manipulation tools, and compatibility with omics-based analyses enable mechanistic investigations of chemical-induced toxicity (Hasanpour et al., 2021a, b, 2025; Radkhah and Eagderi, 2022). Zebrafish embryos develop rapidly ex vivo, with major organ systems forming within 72 hours post-fertilization (hpf) (He et al., 2014). Their optical transparency during early development allows direct observation of internal structures and efficient scoring of developmental abnormalities following chemical exposure (Kanugo et al., 2014). Zebrafish embryo toxicity assays show good concordance with mammalian toxicity outcomes, supporting their use as a bridge between in vitro and higher vertebrate models (Cassar et al., 2019). The fish embryo toxicity (FET) test is increasingly applied to detect developmental and teratogenic effects of environmental contaminants, including pesticides, nanoparticles, and pharmaceuticals (Braunbeck et al., 2015).

Despite evidence that phytic acid exhibits biological activity and toxicity in other organisms, its developmental and teratogenic effects in zebrafish embryos remain poorly understood. Therefore, this study aimed to evaluate the dose- and time-dependent effects of phytic acid on zebrafish embryos and larvae in accordance with OECD Test Guideline 236, focusing on embryo mortality, heart rate, body length, and morphological abnormalities.

Materials and Methods

Reagents: Phytic acid (myo-inositol-1,2,3,4,5,6-

hexakisphosphate; 99% purity) was used in this study. A stock solution was prepared by dissolving phytic acid in deionized water under gentle stirring until completely dissolved, then stored at 4°C until use. Working solutions at nominal concentrations of 5, 2.5, 1.25, and 0.625 ppm were freshly prepared prior to each exposure by serial dilution of the stock solution in E3 embryo medium. All test solutions were prepared immediately before use to minimize potential degradation and to ensure concentration accuracy during embryo exposure.

Zebrafish maintenance and embryo collection: Adult zebrafish were maintained in a glass aquarium (30 L capacity) supplied with continuous aeration. Fish were kept at a controlled temperature of 26±1°C. The fish were fed 2-3 times daily with a combination of commercial dry pelleted feed and live food (*Artemia* nauplii), depending on availability. For breeding, sexually mature zebrafish were selected and acclimated prior to spawning. A spawning ratio of two males to one female (2:1) was employed. Male and female fish were placed in the same aquarium but separated using a fine-mesh net divider to prevent premature spawning. After an acclimation period of approximately 12-15 h, the divider was removed to allow natural spawning. No additional light stimulation was applied during the spawning process. Fertilized eggs were collected within 30-60 min after spawning. The embryos were carefully separated from adult fish and debris and then rinsed several times with E3 embryo medium to remove unfertilized eggs and adherent particles. Only normally developing, transparent embryos were selected for subsequent exposure experiments.

Toxicity assay: The fish embryo toxicity (FET) test was performed in accordance with OECD Test Guideline 236. Fertilized zebrafish embryos were collected within 3 h post-fertilization (hpf) and used for exposure experiments. Embryos were exposed to phytic acid at nominal concentrations of 5, 2.5, 1.25, and 0.625 ppm, along with a control group containing embryo medium only. Exposure assays were conducted using 24-well plates, with one plate assigned to each test condition. Each plate contained 20 wells, with one embryo placed per well and 1 mL of the corresponding test solution added to each well. Thus, 20 embryos were used per condition in each experimental replicate.

The exposure lasted 96 h, and test solutions were renewed every 24 h to maintain consistent exposure conditions. The entire experiment was repeated independently 4 times (n = 4), yielding a total of 80 embryos per treatment condition. Embryo survival and developmental endpoints were evaluated at defined time points during the exposure period.

Deformities scoring and calculation: Embryo mortality and developmental abnormalities were examined using an OptiLab Viewer 4 at 24, 48, 72, and 96 hours-post-fertilization (hpf). Mortality was recorded at each observation time point based on the absence of heartbeat or coagulation of embryos. Morphological abnormalities were evaluated in surviving embryos according to standard zebrafish developmental criteria.

At early developmental stages (24 hpf), general developmental delay and abnormal body axis formation were assessed. At 48 hpf, cardiac-related endpoints, including heart rate alterations and abnormal blood circulation, as well as tail malformations, were evaluated. At 72 hpf, pericardial edema and yolk sac edema were examined, and hatching success was recorded. At 96 hpf, delayed hatching and persistent morphological abnormalities were further assessed. Median lethal concentrations (LC_{50}) of phytic acid were estimated at 96 hpf based on cumulative embryo mortality data. The percentage of deformities was calculated using the total number of surviving embryos at each observation time point. These percentage values were subsequently used to estimate median effective concentrations (EC_{50}) for overall developmental abnormalities. The teratogenic index (TI) was calculated to evaluate the teratogenic potential of phytic acid by dividing the LC_{50} value at 96 hpf by the corresponding EC_{50} value (TI = LC_{50}/EC_{50}). A higher TI value was interpreted as indicative of greater teratogenic potential relative to lethality.

Heart rate analysis: At 48 hours-post-fertilization

Table 1. Deformity percentages observed at 24, 48, 72, and 96 h after phytic acid treatment.

	PA (ppm)	Mortality (%)	No Tail Detachment (%)	Abnormal Somites (%)	Abnormal Eye Pigmentation (%)	Abnormal Tail Morphology (%)	Abnormal Tail Blood Flow (%)
24 hpf	0 (Control)	7.50±6.45	0.00±0.00	0.00±0.00			
	0.625	12.50±6.45	5.00±3.54	3.75±2.50			
	1.25	15.00±5.77	8.75±4.79	6.25±3.54			
	2.5	12.50±11.90	12.50±6.45	8.75±4.79			
	5.0	**52.50±14.43**	**31.25±8.54**	**25.00±7.07**			
48 hpf	0 (Control)	8.75±4.79			0.00±0.00	0.00±0.00	0.00±0.00
	0.625	12.50±6.45			6.25±3.54	5.00±3.54	4.38±2.50
	1.25	15.00±5.77			12.50±6.45	10.00±5.00	8.75±4.79
	2.5	17.50±6.45			25.00±8.66	18.75±7.50	15.00±6.12
	5.0	**43.75±10.31**			**56.25±9.57**	**48.75±8.54**	**45.00±7.91**
		Mortality (%)			**Unhatched Eggs** (%)	**Pericardial Edema** (%)	**Yolk Sac Edema** (%)
72 hpf	0 (Control)	10.00±5.77			7.50±4.79	0.00±0.00	2.50±2.74
	0.625	15.00±7.07			15.00±6.12	6.25±4.79	5.00±3.54
	1.25	17.50±6.45			30.00±9.57	15.00±6.12	12.50±5.00
	2.5	18.75±8.54			60.00±12.25	37.50±9.13	25.00±8.66
	5.0	**50.00±12.25**			**92.50±6.45**	**75.00±10.80**	**62.50±9.57**
96 hpf	0 (Control)	10.00±5.77			2.50±2.74		
	0.625	20.00±0.00			10.00±5.00		
	1.25	21.25±4.79			22.50±7.50		
	2.5	20.00±7.07			55.00±10.80		
	5.0	**56.25±4.79**			**97.50±2.50**		

(hpf), cardiac activity of zebrafish embryos exposed to phytic acid was evaluated under an OptiLab Viewer 4. The heartbeats of individual embryos were counted for 20 s, and the values obtained were converted to beats per minute (bpm). Heart rate measurements were performed using 10 randomly selected embryos per concentration in each experimental replicate. The experiment was conducted in four independent replicates (n = 4).

Body length survey: Body length, defined as the distance from the mouth tip to the end of the tail fin, was measured using Optilab Viewer 4 software under a stereomicroscope. Measurements were performed at 79 h post-fertilization (hpf) using 10 embryos/larvae per test condition in each experimental replicate. The body length assessment was conducted in four independent experiments (n = 4).

Spine deformities scoring: Spinal abnormalities were evaluated at 144 h post-fertilization (hpf) using a stereomicroscope equipped with Optilab Viewer 4. The assessment was performed using 10 embryos/larvae per test condition in each experimental replicate. The experiment was conducted in four independent replicates (n = 4).

Data analysis: Statistical analyses were performed using SPSS software. Data are presented as the mean ± standard deviation (SD). Differences between the control and treatment groups were analyzed using an unpaired t-test. Dose-dependent effects were evaluated using one-way analysis of variance (ANOVA). Statistical significance was defined as $P<0.05$.

Results

Acute toxicity of phytic acid: The results demonstrated that phytic acid–induced mortality increased in a concentration- and time-dependent manner (Table 1). Embryos in the control group exhibited low mortality throughout the exposure period, remaining below 15% at 96 hpf, indicating acceptable test validity. At lower concentrations (0.625-2.5 ppm), embryo mortality remained below 25% at 96 hpf. In contrast, exposure to 5 ppm phytic acid resulted in a marked increase in cumulative mortality, exceeding 50% by 96 hpf (Table 1). Based on cumulative mortality data, phytic acid exhibited moderate acute toxicity to zebrafish embryos, with an estimated median lethal concentration (LC_{50}) of approximately 5.0±1.0 ppm at 96 hpf. No LC_{50} value was reached at earlier time points within the tested concentration range. These findings indicate that phytic acid exerts dose- and time-dependent lethal effects on zebrafish embryos, particularly at higher exposure concentrations.

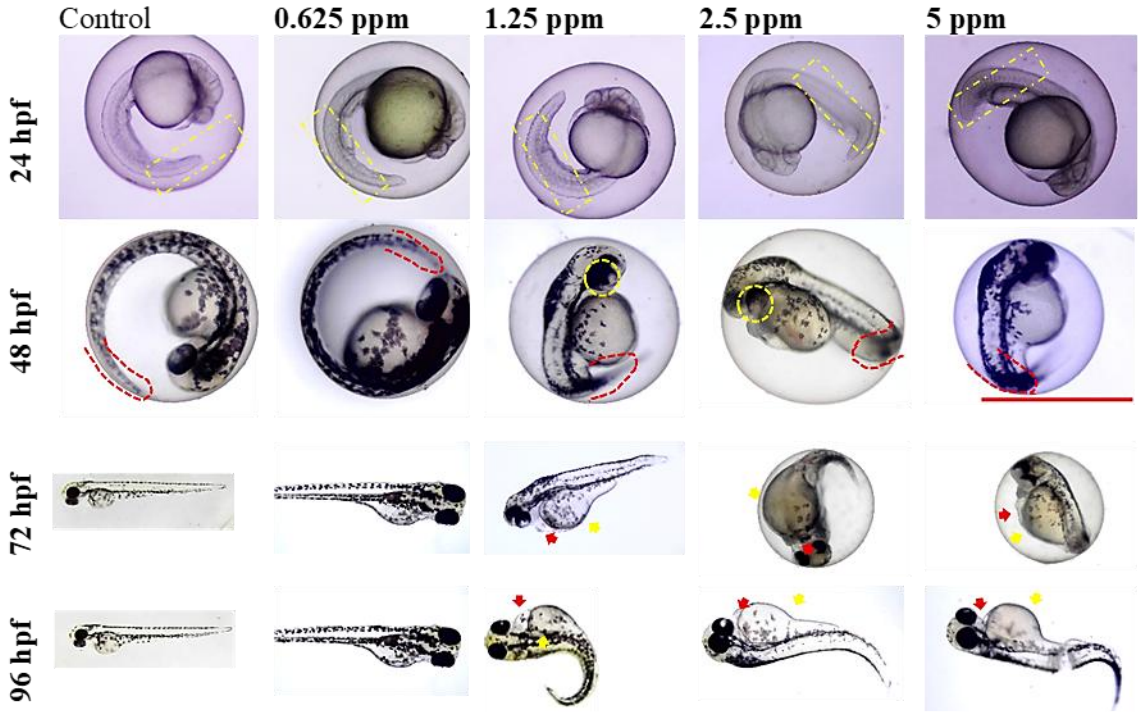

Figure 1. Representative images illustrating phytic acid–induced developmental abnormalities in zebrafish embryos at 24, 48, 72, and 96 h post-fertilization (hpf). The yellow dotted square indicates somite abnormalities; the red dotted line highlights tail malformations; the yellow dotted circle marks alterations in eye development; the red arrow denotes pericardial edema; and the yellow arrow indicates yolk sac edema. Scale bar = 1.0 mm.

Developmental toxicity of phytic acid: To evaluate the developmental toxicity of phytic acid in zebrafish (ZF), morphological abnormalities and malformation frequencies were assessed at 24, 48, 72, and 96 hpf, with additional observations at 144 hpf for growth-related endpoints. Throughout the exposure period, embryos and larvae in the control group and those exposed to the lowest concentration of phytic acid (0.625 ppm) generally exhibited normal development and typical phenotypes (Table 1, Fig. 1). In contrast, embryos and larvae exposed to phytic acid at concentrations of 1.25 ppm or higher exhibited a concentration-dependent increase in developmental abnormalities. The morphological assessment revealed that phytic acid exposure induced a broad spectrum of developmental deformities, including alterations in somite formation, tail morphology, cardiac structure and function, yolk sac edema, delayed hatching, and spinal curvature. The incidence and severity of these abnormalities increased with both concentration and exposure duration. A summary of the observed morphological endpoints is presented in Table 1, while representative deformities observed during the exposure period are illustrated in Figure 1.

Embryos in the control group and those exposed to the lowest concentration of phytic acid (0.625 ppm) exhibited normal somite formation during early development. In contrast, embryos treated with phytic acid at concentrations of 1.25 ppm and above showed a significant increase in abnormal somites (AS) at 24 hpf, indicating disrupted early segmentation (Table 1, Fig. 1). Normal tail detachment was consistently observed in control embryos, whereas abnormal tail detachment (ATD) occurred sporadically at lower phytic acid concentrations and increased markedly at the highest concentration tested. At 5 ppm, approximately one-third of embryos exhibited incomplete or absent tail detachment, a significantly higher proportion than in the control group. Alterations in eye development were also evident following phytic acid exposure. Abnormal eye pigmentation (AEP) was observed at 48 hpf in a concentration-dependent manner (Table 1, Fig. 1). Embryos exposed to phytic acid at concentrations of 1.25 ppm and higher exhibited reduced retinal pigmentation, while those treated with higher

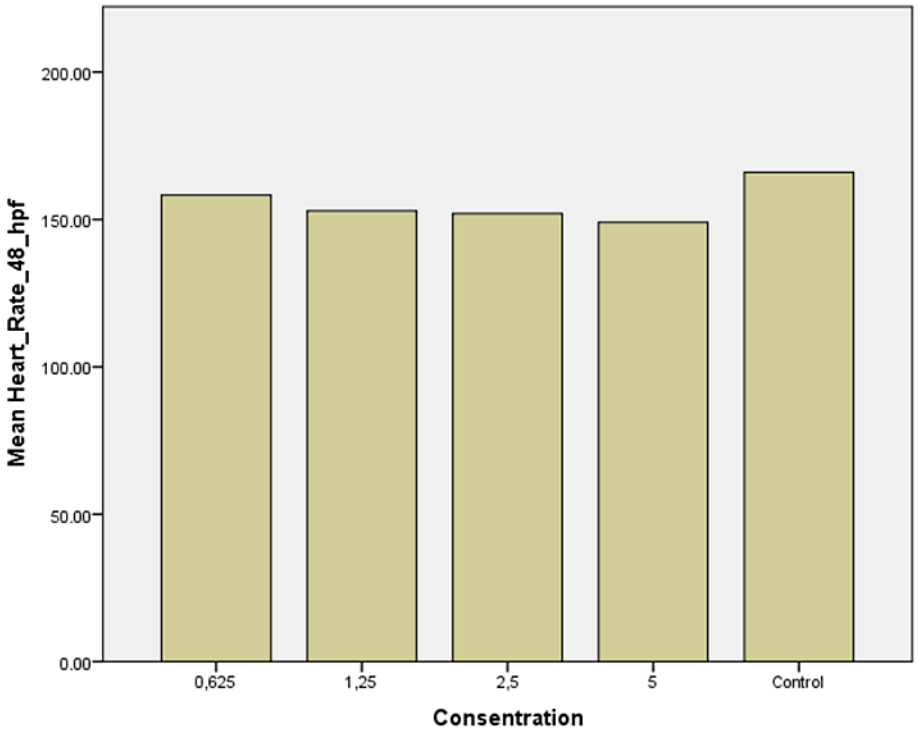

Figure 2. Effects of phytic acid on the mean heart rate of zebrafish embryos at 48 h post-fertilization (hpf). Embryos were exposed to increasing concentrations of phytic acid (0, 0.625, 1.25, 2.5, and 5 ppm). Heart rate values are expressed as mean beats per minute (bpm) calculated from four independent experiments (n = 4).

concentrations showed noticeably smaller eyes compared with controls. In addition, phytic acid exposure induced abnormal tail morphology (ATM), with the incidence of tail malformations increasing significantly at concentrations ≥1.25 ppm.

Phytic acid treatment further affected hatching success at later developmental stages. Embryos in the control and 0.625 ppm groups hatched normally at both 72 and 96 hpf. In contrast, hatching was significantly inhibited at concentrations of 2.5 ppm and above, with less than 10% of embryos successfully hatching by 96 hpf at the highest concentration tested (Table 1, Fig. 1).

Effects of phytic acid on ZF cardiac development and function: To evaluate the effects of phytic acid on cardiac development and function in zebrafish embryos, cardiac-related endpoints, including pericardial edema, blood circulation, and heart rate, were assessed in control and phytic acid–treated groups. Phytic acid exposure induced cardiac alterations in a concentration-dependent manner (Table 1, Fig. 1). At higher concentrations, mild pericardial edema and alterations in blood circulation were observed, indicating impaired cardiovascular development. Heart rate measurements at 48 h post-fertilization (hpf) revealed a clear dose-dependent reduction in cardiac activity following exposure to phytic acid. The average heart rate of control embryos was 166.0±0.82 beats per minute (bpm). Embryos exposed to 0.625 ppm phytic acid showed a slight decrease in heart rate (158.3±2.4 bpm), whereas further reductions were observed at 1.25 ppm (153.0±2.2 bpm) and 2.5 ppm (152.0±2.4 bpm). The lowest heart rate was recorded in embryos treated with 5 ppm phytic acid (149.0±3.7 bpm), representing an overall reduction of approximately 10% compared with the control group (Fig. 2). These results indicate that phytic acid exerts a concentration-dependent inhibitory effect on zebrafish cardiac function during early development.

Effects of phytic acid on overall ZF growth: At 96 hpf, body length measurements were conducted to assess the effect of phytic acid exposure on the early growth of *D. rerio* larvae. The mean body length of

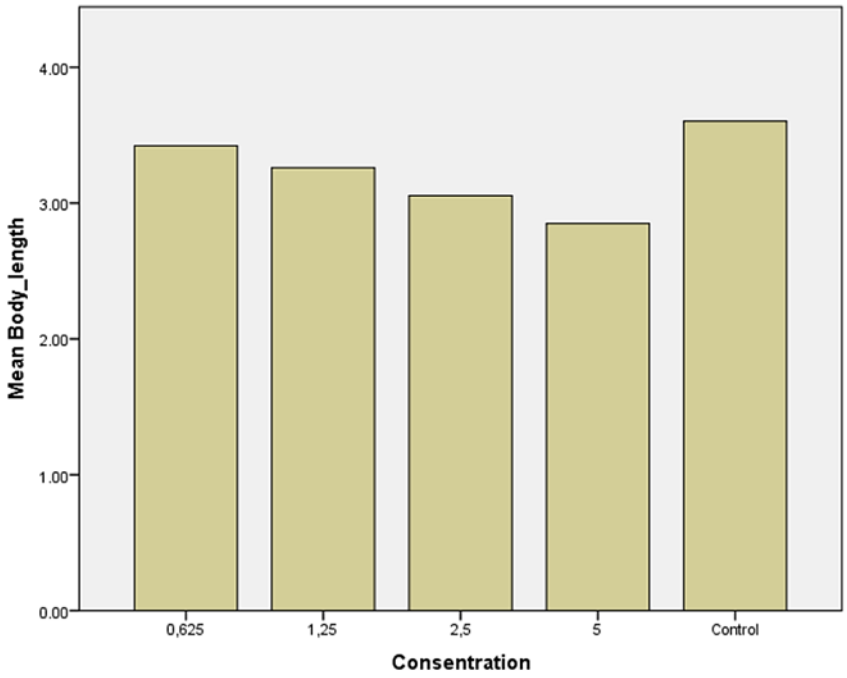

Figure 3. Effects of phytic acid on the mean body length of zebrafish larvae at 96 hours post-fertilization (hpf). Embryos were exposed to increasing concentrations of phytic acid (0, 0.625, 1.25, 2.5, and 5 mg/L). Body length values are calculated from four independent replicates for each treatment group (n = 4).

the control group was 3.60±0.29 mm. In comparison, larvae exposed to phytic acid exhibited progressively shorter body lengths, with average values of 3.42±0.17, 3.26±0.14, 3.06±0.25, and 2.85±0.06 mm at concentrations of 0.625, 1.25, 2.5, and 5 mg/L, respectively. Relative to the control, all treated groups showed a reduction in body length, with the decrease becoming more pronounced at higher concentrations. This trend indicates a dose-dependent inhibition of larval growth, suggesting that phytic acid adversely affects development during early embryogenesis (Fig. 3).

Discussions

The present study demonstrates that phytic acid induces developmental toxicity and teratogenic effects in zebrafish embryos in a concentration- and time-dependent manner. Although phytic acid is widely recognized for its nutritional and antinutritional properties in plants and vertebrates, its potential effects during early embryonic development remain insufficiently investigated. The current findings show that phytic acid exposure causes multiple lethal and sublethal developmental disturbances, even at concentrations that do not immediately induce mortality, highlighting its potential teratogenic risk during sensitive stages of embryogenesis.

Embryo mortality increased progressively with increasing concentration and exposure duration, with cumulative mortality exceeding 50% at 5 ppm by 96 hpf. The estimated LC_{50} value of approximately 5 ppm indicates moderate acute toxicity. The zebrafish Fish Embryo Acute Toxicity (FET) test has been widely validated as a reliable alternative model for evaluating chemical toxicity, as early developmental stages are particularly sensitive to environmental stressors and enable rapid detection of both lethal and sublethal effects (OECD, 2013; Hill et al., 2005). Importantly, mortality at lower concentrations (0.625-2.5 ppm) remained limited, while significant developmental abnormalities were already evident. This separation between lethality and malformation is a typical characteristic of teratogenic compounds and suggests that phytic acid primarily interferes with developmental processes rather than causing nonspecific cytotoxicity (Selderslaghs et al., 2009).

Hatching inhibition was among the most sensitive endpoints observed. Normally, zebrafish embryos

hatch between 48 and 72 hpf, with nearly complete emergence by 96 hpf. In contrast, embryos exposed to higher concentrations of phytic acid exhibited marked delays or failure to hatch. Successful hatching depends on the enzymatic digestion of the chorion and adequate muscular activity of the larvae. Toxicant-induced impairment of metabolic capacity or enzymatic secretion can disrupt these processes, leading to delayed or incomplete hatching (Sant and Timme-Laragy, 2018). Prolonged retention within the chorion may restrict oxygen exchange, delay initiation of feeding, and compromise energy utilization, ultimately increasing developmental stress and mortality risk.

Morphological abnormalities were also frequently observed following phytic acid exposure. These included abnormal somite formation, tail malformations, and yolk sac edema. Somite defects detected at early stages indicate disruption of segmentation and somitogenesis, which are tightly regulated processes essential for proper musculoskeletal development. Tail abnormalities may impair swimming ability and locomotor performance, reducing feeding efficiency and survival potential. Yolk sac edema, a common indicator of osmotic or cardiovascular dysfunction, reflects impaired fluid regulation and reduced circulatory performance (Incardona et al., 2016). Similar malformations have been widely reported in zebrafish embryos exposed to developmental toxicants and are recognized as sensitive indicators of teratogenicity (Nagel, 2002; Selderslaghs et al., 2009).

Cardiac development appeared particularly susceptible to phytic acid exposure. Treated embryos exhibited pericardial edema, impaired blood circulation, and a significant reduction in heart rate. Quantitative measurements at 48 hpf showed a clear concentration-dependent bradycardia, with approximately a 10% reduction at the highest treatment level. Cardiac endpoints are among the most sensitive biomarkers of developmental toxicity in zebrafish because the cardiovascular system forms early and is essential for nutrient and oxygen delivery during rapid embryonic growth (Stainier, 2001; MacRae and Peterson, 2015). Even moderate reductions in heart rate may impair tissue perfusion and organ development, thereby contributing to overall growth retardation and increased susceptibility to stress (Custodis et al., 2010).

Growth inhibition was further confirmed by the reduction in body length at 96 hpf. Body length is a sensitive indicator of overall developmental progress and metabolic efficiency, and decreased length typically reflects impaired cell proliferation or nutrient utilization (Richendrfer et al., 2011). The observed reduction in larval size suggests that phytic acid interferes with normal growth processes, likely through metabolic or nutritional disruption.

The mechanisms underlying these developmental effects are likely multifactorial. Phytic acid (myo-inositol hexakisphosphate) is a potent chelator of essential minerals such as calcium, iron, zinc, and magnesium. Its ability to form insoluble complexes reduces mineral bioavailability, a condition long associated with impaired growth and mineral deficiencies in vertebrates (Schlemmer et al., 2009; Gupta et al., 2013). During embryogenesis, these minerals are critical for enzyme activation, DNA synthesis, muscle contraction, and cardiac function. Consequently, mineral sequestration by phytic acid may disrupt key physiological pathways, resulting in growth inhibition, edema formation, and cardiac dysfunction. Additionally, interactions with digestive and metabolic enzymes, as well as interference with detoxification pathways, such as the cytochrome P450 system, may exacerbate developmental stress (Green et al., 2001).

Early embryogenesis represents a critical window of vulnerability during which even mild biochemical disturbances can produce irreversible structural and functional abnormalities (Lana and Gardner, 2005). In mammals, disruption of mineral homeostasis, cardiac function, or metabolic activity during early gestation has been associated with impaired fetal growth, congenital malformations, and developmental delay (Selevan et al., 2000). Given that phytic acid is a strong chelator of essential minerals and can interfere with enzymatic and metabolic processes, excessive

exposure may theoretically compromise nutrient availability required for normal fetal development. Although phytic acid is generally regarded as safe at typical dietary levels, the present findings suggest that elevated or concentrated exposure could exert sublethal developmental effects, particularly during sensitive stages of organogenesis.

Conclusion

In conclusion, phytic acid induced concentration- and time-dependent developmental toxicity in zebrafish embryos. Exposure resulted in increased mortality, delayed hatching, morphological abnormalities, impaired cardiac function, and reduced growth. The estimated LC_{50} value of 5.0±1.0 ppm at 96 hpf and the presence of significant sublethal effects at lower concentrations indicate that phytic acid possesses teratogenic potential during early embryogenesis. These findings provide new insights into the developmental toxicity of phytic acid and highlight the need for further investigation into its biological effects during early life stages.

Acknowledgments

This project was partially funded by the Doctoral Dissertation Research Grant of Universitas Sebelas Maret (PDD-UNS) Indonesia, awarded to Dewi Puspita Sari under contract number 194.2/UN27 .22/PT.01.03/2024, dated 15th March 2024.

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