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Urinary phosphorus excretion in fish: environmental and aquaculture implications

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Abstract – Global aquaculture production has expanded rapidly in recent decades, resulting in a significant increase in effluent discharge rich in phosphorus and nitrogen. In freshwater and brackish water ecosystems, phosphorus is often the primary limiting nutrient. Environmental sustainability of aquaculture, therefore, hinges on reducing phosphorus in its effluents, particularly urinary phosphorus due to its soluble nature, which can directly contribute to eutrophication and harmful algal blooms such as red tides. In contrast, fecal phosphorus, which is primarily insoluble, poses a lower environmental risk. However, much of the past research has focused on improving phosphorus digestibility to reduce fecal phosphorus excretion, while urinary phosphorus has been largely overlooked. Urinary phosphorus represents the excess phosphorus absorbed from the diet. Therefore, dietary strategies should aim to reduce the available phosphorus content in fish feed to just below their nutritional requirements, ensuring that fish growth and health are not compromised. This approach could significantly reduce or even eliminate urinary phosphorus excretion, greatly enhancing the sustainability of aquaculture. This review highlights key aspects of urinary phosphorus and examines various strategies for its reduction, with a focus on practical in situ techniques.

Keywords: Phosphorus / sustainable aquaculture / environmental sustainability / feed technology / eutrophication / red tides

1 Introduction

World aquaculture production (excluding algae) increased 5.7-fold from 1990 to 2020 (FAO, 2022), leading to a substantial rise in effluents rich in phosphorus (P) and nitrogen (N) (Md Noor and Harun, 2022). In freshwater ecosystems, P is typically the primary limiting nutrient, while N often limits primary productivity in seawater ecosystems. However, seawater fish culture often occurs in areas with significantly lower salinity than the open ocean, such as inner bays, estuaries, and coastal waters. In these low-salinity environments, P is often the primary limiting nutrient. Consequently, in many aquaculture sites, P input can directly stimulate algal blooms and red tides (Smith, 1984; Kato et al., 1985; Cloern, 2001).

Red tides can lead to mass fish mortality, resulting in significant economic losses. For example, red tide-induced fish kills often exceed 1 billion yen (~6.7 million USD) per incident, with multiple occurrences reported annually at net pen facilities in the inner bays of Japan's coastal waters (Imai et al., 2021). Therefore, many aquaculture operations must reduce P in their effluents to achieve environmental sustainability.

The ultimate source of P in aquaculture effluents is fish feed. Approximately 40–60% of the P in typical commercial fish feeds is retained by fish, while the remainder is excreted (Islam, 2005; Tab. 1). Phosphorus excreted by fish falls into two categories: fecal P and urinary P. Fecal P is mostly insoluble and poses a lower environmental risk (Vielma et al., 2000; Fernandes et al., 2007; Bureau and Hua, 2010). In contrast, urinary P is fully soluble and, therefore, particularly harmful to the environment.

Despite its significance, urinary P excretion has not been well studied. This is likely due to the difficulty of collecting fish urine, especially 24-hour samples, which are necessary to account for fluctuations in solute concentrations (Sugiura, 1998; Sarker et al., 2009). Conversely, monitoring P in aquaculture effluents has been extensively researched, but with compromised accuracy since effluents contain both fecal and urinary P (Coloso et al., 2003; McDaniel et al., 2005; Sugiura et al., 2006a).

An additional problem is the limited data available on large fish. In aquaculture, large fish consume the most feed and contribute the most waste to effluent water (Cowey, 1995). Furthermore, large or adult fish require lower concentrations of

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Table 1. Examples of phosphorus (P) mass balance in cultured rainbow trout fed pelleted feed.

Total P (% diet)	P retained by fish (% intake)	Insoluble P excreted (% intake)	Soluble P* excreted (% intake)	Feed type (C or Ex)	FCR	Ref.
0.94–1.01	50–54	32–46	2–14	C	0.74–0.78	1
0.62–0.74	55–62	33–36	2–12	Ex	0.96–1.00	2
0.81	43	34	24	C	1.34	2
0.63	53	31	16	C	1.29	2

* including residual P.
C: commercial feed; Ex: experimental feed. FCR: feed conversion ratio (feed weight / fish weight gain).
Ref. 1 Dalsgaard and Pedersen (2011); 2 Dalsgaard et al. (2023)with some editorial modifications.

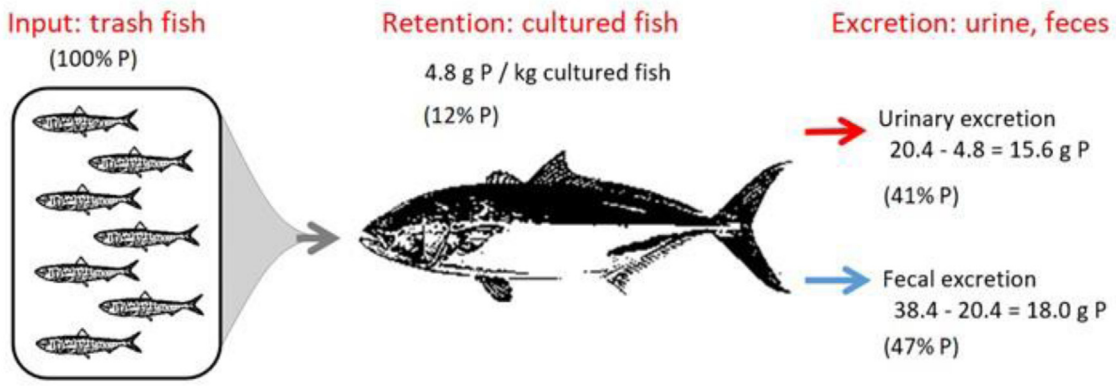

Fig. 1. Environmental cost of farming 1 kg of yellowtail fish: phosphorus (P) mass balance. Yellowtail (amberjack) is the most farmed fish in Japan. To produce 1 kg of yellowtail, approximately 8 kg of trash fish is required. The 8 kg of trash fish provides around 38.4 g of P (0.48% P per wet weight, based on Shearer's 1984 data for rainbow trout). The digestibility of P in trash fish or fish meal has not been reported for yellowtail, but it is estimated to be around 53%, based on recent data from a high-fish meal diet (Horstmann et al., 2023). Thus, 8 kg of trash fish would provide approximately 20.4 g of available P (38.4 g * 0.53 = 20.4 g). However, cultured yellowtail retain only 4.8 g of this P, excreting the remaining 15.6 g as soluble urinary P. These values are in fair agreement with Islam (2005) on a total P basis.

dietary P (Eya and Lovell, 1997; Sugiura et al., 2000), similar to pigs and humans (IOM, 1997; NRC, 1998). However, the precise dietary P requirements of large fish remain under-researched due to technical difficulties (Sugiura, 1998). As a result, the P requirements determined for small juvenile fish are often applied to larger fish (NRC, 2011), which most likely exceeds their physiological needs.

Since urinary P is the most problematic source of pollution in aquaculture effluents, greater attention must be given to reducing its excretion to mitigate aquaculture's environmental footprint (Cho and Bureau, 2001). While many physiological findings contribute to a better understanding of renal P regulation, they do not offer direct applications for mitigating P excretion at the farm level. This review does not seek to introduce advanced but impractical physiological knowledge. Instead, it aims to organize and present problem-solving technologies that support cleaner and more sustainable aquaculture development.

2 Excretion of dietary P

2.1 Mass balance

Phosphorus in aquaculture effluents originates from feed. Some aquaculture facilities use trash fish, while others rely on pelleted feeds, such as dry and moist pellets. Trash fish contain available P in amounts far exceeding the fish's nutritional requirements. They also contain hydroxyapatite-P, which is poorly digested by fish. As a result, both urinary and fecal P excretions are significantly high (Fig. 1).

Pelleted feeds vary widely in composition. When trash fish or fish meal is the primary ingredient, urinary and fecal P excretions remain high, similar to levels observed in fish fed trash fish. This is common in farm-made moist pellets, which are often formulated without considering P excretion into the environment. Generally, as fish meal content in diets decreases, P excretion through urine and feces also declines. This trend is particularly evident in modern commercial feeds, which contain higher proportions of plant-based ingredients and feed oils (Tab. 1).

2.2 Fecal excretion

Dietary P is absorbed in the intestine including pyloric caeca. In freshwater-reared rainbow trout, inorganic P (Pi) in the intestinal lumen is absorbed predominantly via passive, Na-independent transport, which is non-carrier-mediated paracellular diffusion. Pi moves from the luminal to the interstitial compartment through leaky junctions of the proximal intestine and pyloric caeca. This pathway is not regulated by dietary P intake, meaning that even at high dietary P levels, P continues to be absorbed at relatively high rates (Sugiura et al., 2003). Additionally, a serosa-positive electrical

potential generates a downhill gradient across the tight junctions, facilitating paracellular Pi diffusion, similar to what occurs in mammals (Peacock, 2021; Wagner, 2024).

In contrast, marine teleosts tend to generate a serosa-negative transepithelial potential (Loretz, 1995; Sundell and Sundh, 2012). This may limit, to some extent, intestinal Pi absorption and protecting the kidneys, which appears to have a limited capacity compared to those of freshwater teleosts and mammals as discussed below. In marine teleost intestines, excess P may precipitate due to high bicarbonate secretion (leading to elevated luminal pH) and high Mg and Ca concentrations (Wilson et al., 2002).

In flounder (marine fish), Sarker et al. (2006) reported that dietary P absorption increased as dietary KH_2PO_4 levels rose (up to 1.3% available P; the maximum level tested). Apparent P absorption remained high at this level; however, fecal P (which the authors did not calculate) increased as dietary available P exceeded 0.9%. Sarker et al. (2009) observed similar results in yellowtail (marine fish), where apparent P absorption increased with higher dietary P (P source: NaH_2PO_4), but fecal P also doubled at high dietary P intakes. These findings suggest that marine fish increase urinary P excretion while also raising fecal P output at high dietary P intakes. In freshwater-reared rainbow trout, Sugiura et al. (2003) also reported a similar pattern, showing a progressive reduction in fractional Pi absorption in the intestine at high dietary P levels.

Lall and Bishop (1979) reported that rainbow trout reared in freshwater had higher body Ca and P content than those reared in seawater on the same diet, indicating greater dietary P retention in freshwater. Similar results were found in Atlantic salmon (Shearer et al., 1994). Dosdat et al. (1998) observed higher fecal P excretion (~35% of intake) in brown trout reared in seawater compared to those fed the same diet in freshwater (~23% of intake), indicating that seawater reduces intestinal P absorption. Seawater fish drink seawater high in Mg and Ca, and these divalent cations may reduce P absorption in the gastrointestinal tract, similar to their effect in mammals (Sheikh et al., 1989; Tonelli et al., 2010).

2.3 Urinary excretion

The excretory pathway of surplus P varies by species. Herbivorous mammals excrete excess P through feces, whereas in carnivorous mammals, urine is the principal route. In humans, the excretion may be split between feces and urine depending on the Ca content in the diet (Liebig, 1842; Paton et al., 1899–1900; Bergmann, 1901; Sherman, 1919; Maynard and Loosli, 1962). In fish, urine is considered the primary excretory route. In marine teleosts, Smith (1930) noted that nearly all absorbed PO_4, Mg, Ca, and SO_4 are excreted through the kidneys.

Tomiyama et al. (1956) reported that in common carp, about 1% of intramuscularly injected ^{32}P was excreted within 25 hours, with 90% excreted through the kidneys and the remaining 10%, according to the authors' hypothesis, via the gills. Kaune and Hentschel (1987) observed that goldfish regulate P excretion by adjusting renal tubular reabsorption and secretion. When sodium phosphate was intravenously infused, 65% of the P was excreted through the kidneys, with 75% of it eliminated via tubular secretion.

In mammals, urinary Pi reabsorption is tightly regulated by the NaPi-IIa and IIc proteins on the apical membrane of renal tubular brush-border cells (Murer et al., 2003; Peacock, 2021; Wagner, 2024). Gupta and Renfro (1989) found, using flounder proximal tubule monolayer cultures, that renal P secretion also depends on a Na gradient. As such, flounder proximal tubule cells both actively reabsorb and secrete P, depending on P intake and other factors, via Na-dependent Pi transporters located on the brush-border and basolateral membranes, respectively.

In teleosts, especially marine species, glomerular filtration rates (GFR) are lower than in mammals (Natochin, 1996). GFR ($ml\,kg^{-1}\,day^{-1}$) range from 25-200 in freshwater fish and 5-15 in seawater fish, compared to ~2000 in mammals. Similarly, P filtered per day ($\mu moles\,kg^{-1}$) is lower in teleosts (<600) than in mammals (<4000) (Bijvoet and Reitsma, 1977). In marine teleosts, the urine volume is small and highly concentrated, often to the point of supersaturation or precipitation (Marshall and Grosell, 2006). However, seawater fish secrete large amounts of bicarbonate into the intestinal lumen, which in turn reduces the urinary pH. The acidic urine, characteristic of marine fish, can counteract the crystal formation of divalent solutes in the renal tubules and bladder (Smith, 1961; Takvam et al., 2023).

Hickman (1968) analyzed the ion concentrations in the rectal fluid and urine of large flounder ($n = 5$). The rectal fluid contained an average of Mg (180 mM), SO_4 (106 mM), Cl (126 mM), Ca (12 mM), and PO_4 (0.16 mM). Urine contained Mg (142 mM), SO_4 (60 mM), Cl (127 mM), Ca (19 mM), and PO_4 (12 mM). Only 16% of Mg and 11% of SO_4 from ingested seawater were absorbed in the intestine, with the absorbed portions excreted in urine. Hickman estimated that around 32% of ingested Ca remained in rectal fluid, 8% was excreted in urine, and the remainder (~60%) was excreted via the gills or other routes. Seawater fish ingest large amounts of seawater, and some of the Mg and Ca they absorb are excreted renally at high concentrations (Beyenbach, 2004; Islam et al., 2011).

In Atlantic salmon, the gene expression of the bone-derived phosphaturic hormone *fgf23* is upregulated by a high-P diet, suggesting increased urinary P excretion in response to excess P intake. (Fjelldal et al., 2016; Smedley et al., 2018; Drábiková et al., 2023). In the future, this and other molucular markers may be used to monitor the adequacy of dietary P intake in farmed fish (Verri and Werner, 2019).

3 Urinary P monitoring

3.1 Urine sampling

To assess a fish's P status, several indicators may be used, including bone or scale ash content, whole-body Ca content, and the pleural rib shape, among others. If these diagnostic indicators show that the fish's P status is normal, the diet may contain excess P rather than providing the exact amount required. Therefore, after assessing the fish's P status, it is important to measure urinary P excretion, as it reflects excess available P in the diet.

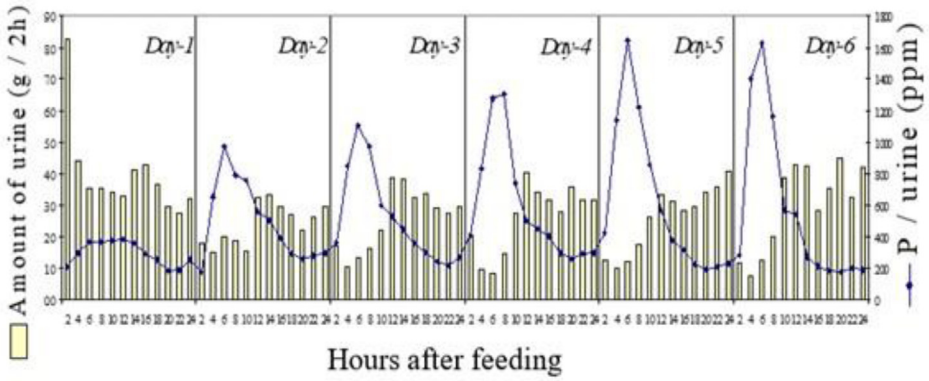

Fig. 2. Urinary phosphorus (P) excretion in freshwater rainbow trout in a metabolic chamber (Sugiura, 1998). Urine was continuously and quantitatively collected from a fish placed in a metabolic chamber of Smith (1967), using a catheter and small open-top vials positioned on a rotating disk. The fish was force-fed once every 24 hours, receiving 1% of its body weight (on a dry basis) from Day 1 to Day 6, using a glass plunger. The diet composition (dry weight basis) was: 35% egg white, 10% gelatin, 14% dextrin, 20% fish oil, 4% vitamin-mineral mix, 2% amino acid mix, and 14% cellulose, with additional 3.51% KH_2PO_4 and 4.21% $CaCO_3$. The total P content was 0.93%, and the total Ca content was 1.89%. Balance summary: Fish body weight: 366 g; Total P fed: 29.1 mg 24 h^{-1}; Total P excreted in feces: 2.29 mg 24 h^{-1}; Urine output: 13.0% of body weight 24 h^{-1}; Total P excreted in urine: 13.7 mg 24 h^{-1}; Total P retained by fish: 13.1 mg 24 h^{-1} (0.374% of dry diet).

However, collecting urine from fish is challenging. Denis (1912–13) quantitatively collected urine from dogfish (elasmobranch) over 24 hours. The fish were restrained with inch-wide bandages and tied to a board about two feet long, which was placed in a large tank of running seawater. The board was positioned so that the cloaca was raised above the water's surface, allowing urine to be collected via a cannula tied to the urinary papilla. Alternatively, some fish were placed in a narrow trough without being fastened to a board. The author noted that pithing (severing) the spinal cord up to the level of the dorsal fin was helpful. The average P concentration in the urine of 10 fasting dogfish was 4520 ppm as P_2O_5 (equivalent to 1973 ppm as P). The urine was acidic, and neutralizing it caused earthy phosphates to precipitate. Denis (1913–14) also analyzed urine from goosefish (teleosts), collected from the bladders of six fish after death, and found that it contained 440 ppm as P_2O_5 (equivalent to 192 ppm as P).

Smith (1929) used a catheter sewn into the urinary papilla to collect urine from several freshwater teleosts. Similar techniques were used by other researchers (e.g., Mashiko and Jozuka, 1961; Post et al., 1965; Holmes and Stainer, 1966; Smith, 1967; Curtis and Wood, 1991; Ng et al., 1996; Deng et al., 2000; Wood et al., 2017; Jin et al., 2022). Of note, some of these studies used bubbled oxygen instead of air in the head tanks to help reduce stress in fish.

Spot sampling of urine is less technical and less stressful for the fish, making it more convenient than catheter or cannula-based collection. Spot sampling can be performed using two methods: tentatively called the forced urination method and the bladder suction method. In the forced urination method, gentle pressure is applied to the fish's abdomen to expel urine directly into a microtube or onto a clean plastic film where it is then collected using a Pasteur pipette. Colored urine should be avoided, as it may indicate fecal contamination. The bladder suction method involves inserting a cannula through the urogenital opening and applying light suction to extract urine directly from the urinary bladder. For both methods, fish should be anesthetized, and water on the body surface should be wiped off before handling.

Spot sampling has been used by several researchers. However, when measuring P, spot sampling may produce inaccurate data, as urinary P concentrations can vary greatly depending on the time of sampling, the source of P, and the diet composition (Fig. 2 Sugiura 1998).

3.2 Urinary P

In humans, Sick (1857) found that ingesting sodium phosphate increased urinary P excretion beyond the amount ingested (Tab. 2). Day (1860, p. 128) wrote, "The quantity of phosphate of lime in the urine is dependent on the quantity of this substance occurring in the food, and on the demands of the organism for this salt". Maurel (1901, 1904) estimated daily P requirements in normal diets through experiments on himself, based solely on urinary P excretion (Tab. 2).

Urinary P excretion is negligible when dietary intake falls below the requirement level of animals including fish. However, once intake exceeds the requirement, the excess P begins to appear in the urine. Miller et al. (1964) demonstrated that urinary P excretion increased proportionately in baby pigs receiving dietary P levels above 0.5%, although P retention remained similar to pigs consuming higher amounts of P. In turkeys, Hurwitz et al. (1978) reported that urinary P excretion was nearly zero when P absorption was below ~200 mg day^{-1}. However, once absorption exceeded that threshold, urinary P excretion (mg day^{-1}) increased proportionally to P absorption.

Bureau and Cho (1999) examined the correlation between plasma P and urinary P concentrations in rainbow trout using tritiated PEG as a glomerular filtration marker and urinary spot sampling. They reported a plasma Pi threshold concentration of approximately 86 mg L^{-1} for urinary Pi excretion and suggested that diets formulated to produce plasma Pi close to this threshold could minimize urinary P excretion.

Table 2. Landmark research of urinary phosphorus (P)*.

Major findings	Source
• Ingesting sodium phosphate greatly increased urinary P excretion. (human)	Sick (1857)
• Ingesting $CaCO_3$ reduced urinary P excretion. (human)	Riesell (1868)
• Dietary fat increased urinary P and decreased fecal P. (human)	Keller (1899, 1900)
• Estimated P requirements based on urinary P excretion. (human)	Maurel (1901, 1904)
• Urinary P excretion was much lower on a P-free diet than during starvation. Dietary protein decreased urinary P excretion. (rat)	Gevaerts (1901)
• Used a catheter to collect urine. Urine is the main route for P excretion. (fish)	Smith (1929, 1930)
• Urinary P excretion increased proportionately in pigs receiving dietary P above 0.5%. (pig)	Miller et al. (1964)

*Excluding mechanism-seeking physiological research that may only remotely related to aquaculture.

Roy and Lall (2004) studied urinary P excretion in haddock and Atlantic salmon reared in seawater (salinity 32‰) and fed the same commercial diet (1.4% total P, 12% ash). Urine was spot-sampled at 0, 3, 6, and 17 hours after feeding using a catheter attached to a syringe. Total urine volume (per body weight) was approximately three times greater in salmon than haddock, but urine P concentration was 4.6 times higher in haddock than in salmon.

3.3 Non-fecal P

An alternative method, though with some compromises, is the use of recirculating tanks (Fig. 3A). In this setup, urine is not directly collected; instead, the recirculating tank water is sampled and analyzed, capturing the combined excretion from urinary, branchial, and surface sources (collectively termed "non-fecal excretions"). Fecal excretion should be carefully excluded using a feces-collection apparatus attached to the main tank. Dalsgaard and Pedersen (2011) and Dalsgaard et al. (2023) employed similar experimental device to determine fecal and dissoved P excretions in rainbow trout.

The advantages of this method include minimal stress to the fish and low technical demand for the researcher. Therefore, despite its limitations, this is one of the most practical methods for measuring urinary P excretion in fish. An important consideration is to collect feces immediately after it is voided to prevent its disintegration and the P leaching, which can be mitigated using appropriate binders such as guar gum (Brinker, 2007, 2009). Similarly, recirculating aquaculture systems may be used where urinary P accumulate in the recirculating water (Huang et al., 2023). Such systems should also be operated with an efficient fecal-separating device to minimize fecal P leaching.

Rodehutscord (1996) found that total P excretion (non-retained P) in rainbow trout remained low up to 0.37% dietary P but increased progressively with higher P intake (P source: Na_2HPO_4). Rodehutscord et al. (2000) further reported that non-fecal P excretion in rainbow trout was low at lower dietary P levels but increased progressively with higher intakes. Non-fecal P excretion was calculated as the amount of diet fed × total P% in the diet × apparent P absorption% − P retained by the fish. Sugiura (1998) and Sugiura et al. (2000) estimated the dietary P requirement of large rainbow trout based on non-fecal or urinary P excretion, measured using a metabolic chamber (Smith, 1967), spot-sampling, and a recirculating system (Fig. 3). Sarker et al. (2009) examined the effects of dietary P content on non-fecal P excretion in yellowtail (marine fish) using diets containing graded levels of NaH_2PO_4. Non-fecal Pi excretion increased linearly once dietary P levels exceeded 0.44%.

3.4 Dissolved P

Dissolved P contains all urinary P and a portion of fecal P that is soluble and therefore leached from voided feces. The amount of leached fecal P varies depending on fecal consistency and the method of fecal collection. Consequently, the lowest dissolved P corresponds to urinary P (or non-fecal P), while the highest dissolved P equals the sum of urinary P and soluble fecal P. Fernandes et al. (2007) reported that approximately 17% of total fecal P was soluble in southern bluefin tuna fed trash fish (sardine), whereas only about 6% was soluble in fish fed pelleted feeds.

Dissolved P monitoring is typically conducted in commercial aquaculture facilities by analyzing effluent P concentrations. Bureau and Hua (2006) estimated the effluent P outputs (g P kg^{-1} fish produced) from Canadian trout farms, reporting values of 23.3 (solid P) and 19.6 (dissolved P) in the 1970s with diets contained 2.5% total P. By the 2000s, these values had decreased to 6.1 (solid P) and 2.4 (dissolved P) with diets containing 1.1% total P. They also projected future reductions to 3.9 (solid P) and 1.0 (dissolved P) with diets containing 0.9% total P. Azevedo et al. (2011) assessed the environmental loadings of P and N across various fractions in the lake-cage culture of rainbow trout (initial body weight ~100 g, final ~800 g) fed for approximately 160 days with a commercial low-pollution feed produced in Canada (protein 44%, fat 22%, total P 1.1%, feed conversion ratio ~1.1). The total P excretion was 8.7 g kg^{-1} fish production, with ~39% in dissolved form.

3.5 Endogenous P (urinary)

Gevaerts (1901) observed that P excretion in the urine of rats on a P-free diet was much lower than during starvation. On a diet of sucrose and edestin or sucrose and ovalbumin, P in the urine was much lower than on a sucrose-only diet (Tab. 2). Wolf and Oesterberg (1911) found that feeding starving dogs a small amount of protein drastically reduced urinary P excretion, while starch and fat had little to no effect. These

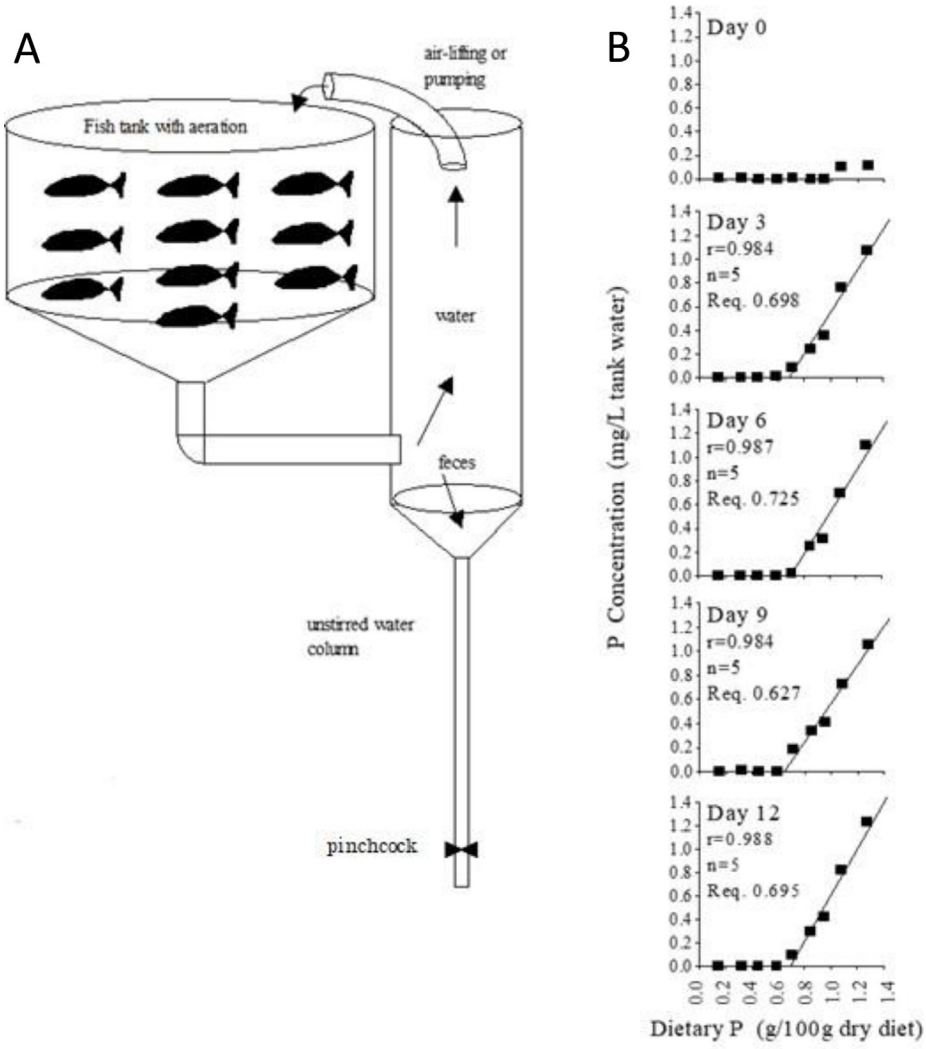

Fig. 3. Non-fecal phosphorus (P) excretion in freshwater rainbow trout measured in a recirculating tank system (Fig. 3A; Sugiura, 1998). Non-fecal P excretion, primarily urinary when properly sampled, provides a rapid and clear response (Fig. 3B), making it useful for estimating dietary P requirements of large or adult fish. In this experiment, 13 fish (average body weight 203 g / fish) were placed in a 150-liter tank for 24 hours without an external water supply. The recirculating tank system continuously collected feces into a long, unstirred water column within minutes of defecation. Although some soluble components in the feces may have leached in the unstirred water column, they remained separated from the tank water. The collected feces, along with water from the unstirred column, were considered fecal excretion, while any changes in the tank water were classified as non-fecal excretion.

mammalian studies indicate that urinary P excretion increases with dietary P intake and decreases with protein intake. In mammals, P loss is known to be lower on a P-free diet than during fasting (Maynard and Loosli, 1962, pp. 351–373).

In fish, urinary P excretion drops to near zero when they are fed a low-P or P-deficient diet (Sugiura, 1998; Sugiura et al., 2000), indicating that renal P reabsorption is highly efficient, with negligible obligatory urinary losses. However, during periods of starvation, fish do excrete P in the urine. The endogenous P loss during starvation may be divided into two phases: an initial phase and a prolonged phase. In prolonged starvation, fish excrete a significant amount of endogenous P in the urine.

In trout, Sugiura (1998) monitored urinary P excretion of non-fed fish, which remained low until day 12 (~0.3 mg P kg fish^{-1} day^{-1}) but increased to 1.8 mg P kg fish^{-1} day^{-1} by day 28. The increase in urinary P excretion during prolonged starvation is likely due to muscle breakdown, as skeletal muscle contains a considerable amount of P.

4 Reducing urinary P

4.1 Approaches

The absorption of dietary P is minimally regulated when the P source is in the form of K- or Na-salts, when the diet is acidified with organic or inorganic acids, or when the P source is not bound to cations. In these cases, any excess P is excreted in the urine. Therefore, the presence of urinary P indicates that its levels can be reduced by lowering the available P content in the diet. Several strategies can be employed to achieve this, including:

- Using commercially available low-P feed containing lower concentrations of available P.
- Mixing the diet with low-P ingredients and re-pelletizing it. Similarly, the diet can be mixed with a dilution feed in an appropriate ratio. Dilution feed is a P-deficient feed made from low-P ingredients (Sugiura, 1998).
- Adding feed oil to the diet, which increases digestible energy (DE) content, and hence feed efficiency (FE), without raising dietary P levels (Satoh et al., 2004; Sugiura, 2018).
- Add P-binders to the diet. P-binders, such as Ca, Al, and Fe (III) compounds, and other chemicals, bind with Pi in the diet or in the gastrointestinal tract, converting available P into unavailable P.

The following subsections mainly describe research on P-binders, a relatively new area with limited studies conducted in fish. As a result, some descriptions remain tentative.

4.2 Ca-based binders

In various animal species, the intestinal absorption of dietary Pi is closely related to dietary concentrations of Ca and other cations, which form insoluble salts with P in the diet or intestinal lumen, reducing the P availability. In 1868, Riesell found that ingesting $CaCO_3$ daily reduced his urinary P excretion by about half, although it returned to normal levels after four days (Tab. 2). Herxheimer (1897) noted that baking 5% $CaCO_3$ into bread decreased urinary P while increasing fecal P. Bergmann (1901) found that while dogs typically excrete most P in urine, a high-Ca diet increases P excretion in feces.

Research has shown that intestinal P absorption decreases as dietary Ca levels increase in various species, including rats (Hoek et al., 1988), pigs (Fox and Care, 1978a), chicks (Fox and Care, 1978b; Al-Masri, 1995), trout (Phillips et al., 1958–1960, 1964; Porn-Ngam et al., 1993), and carp (Nakamura, 1982), among others.

4.3 Insoluble Ca

Since dietary Ca reduces intestinal P absorption, its presence in the diet would be expected to increase the dietary P requirement. However, several studies on fish have reported that dietary Ca levels do not affect the dietary P requirement (e.g., Ogino and Takeda, 1976, 1978; Lovell, 1978; Watanabe et al., 1980). Why is that?

Vielma and Lall (1998) reported that dietary $CaCO_3$ supplementation did not decrease ash, Ca, or P concentrations in the vertebrae and scales of Atlantic salmon, indicating that $CaCO_3$ did not decrease intestinal P absorption. The salmon's high gastric pH (~4) limits dietary $CaCO_3$ solubilization, reducing its negative effect on P absorption. Also in pufferfish, Laining et al. (2011) found that the dietary addition of $CaCO_3$ did not reduce P absorption, which could be due to the intact passage of $CaCO_3$ through the stomach, causing no interaction between Ca and P in the intestine (Sheikh et al., 1989). Since pufferfish lack a functional stomach, dietary $CaCO_3$ was not solubilized in the stomach (Rees and Shroff, 2015). Similarly, many agastric fish consume $CaCO_3$-rich prey without reducing P absorption, as they lack gastric acid secretion (Sugiura, 2018).

In humans, hyperphosphatemia is common among chronic kidney disease (CKD) patients. Thus, various P-binding compounds have been prescribed for CKD patients to inhibit intestinal P absorption. Among Ca-based binders, Ca carbonate and Ca acetate have been used successfully (Tonelli et al., 2010). $CaCO_3$ requires strong gastric acidity (pH ~1.5) to be effective, while highly soluble Ca acetate is effective across a wider pH range in preventing intestinal P absorption (Takahashi et al., 1999; Schucker and Ward, 2005).

Sheikh et al. (1989) studied the P-binding effects of different Ca compounds in vitro. They noted that $CaCO_3$ showed low P-binding (~20%) at pH 6-7, but it increased at more acidic pH. In contrast, Ca-acetate, $CaCl_2$, Ca-lactate, and Ca-gluconate exhibited high P-binding (over 80%) at pH 6 and higher. They also examined the in vivo effects in humans, finding P absorption rates of 77% without Ca, 44% with $CaCO_3$, and 26% with Ca-acetate.

High-fat diets increase intestinal P absorption. Keller (1899, 1900) found that adding fat to the diet decreased fecal P and increased urinary P excretion. He hypothesized that fatty acids combined with Ca to form soaps, excreted in stool mainly as Ca-oleate, freeing P for absorption in the intestine, which resulted in increased urinary P. Kawamoto et al. (2020) reported similar findings without referencing these studies.

4.4 Other binders

Dietary P can also form insoluble compounds with ferric (Fe(III)) and aluminum (Al) compounds (Deobald and Elvehjem, 1935). Al-based compounds are more effective than Ca-based compounds in preventing intestinal P absorption (Lotz et al., 1968; Janssen et al., 1996; Katopodis et al., 2006; Malindretos and Cozzolino, 2016), but they are rarely used in patients with renal failure due to their neurotoxicity (Sheikh et al., 1989; Schucker and Ward, 2005; Kalaitzidis and Elisaf, 2014). Sevelamer, an insoluble anion-exchange resin (Schucker and Ward, 2005), lanthanum (III) (Kalaitzidis and Elisaf, 2014), Boehmite-type Al (B-alm), a crystalline form of Al-hydroxide (Kazama, 2009) effectively bind Pi in the intestine.

In the intestine, P precipitates by combining with co-existing cations, depending on the luminal environment, which is similar to that of a wastewater treatment plant. In such treatments, Al, Fe(III), Mg, Ca, and lanthanum compounds are used to precipitate and remove soluble Pi from wastewater (Haghseresht et al., 2009; Lürling and Tolman, 2010; Li et al., 2021; Akinnawo, 2023).

The dietary addition of P binders reduces urinary P excretion, but increases fecal P excretion. Although fecal P is largely insoluble, it may slowly dissolve in sediment. Therefore, the efficient collection and removal of fecal solids is beneficial. This can be achieved in raceway ponds and recirculating aquaculture systems. In marine cage culture, if tidal flow does not naturally flush away settled feces, artificial removal from the cage bottom may need to be considered (Belle and Nash, 2008; Beveridge, 2008).

5 Achievable goals

When dietary available P falls below the fish's requirement, urinary P becomes undetectable, as noted earlier. In such cases, it would be necessary to assess fish's P status using diagnostic markers such as bone ash, scale ash, and whole-body Ca content (Sugiura, 2018). If the fish are diagnosed as P deficient, increasing the available P content in their diet may or may not be necessary. If the fish are growing normally, particularly in large fish, increasing dietary P is unnecessary (Eya and Lovell, 1997). Bones and scales serve as P reservoirs, and as long as these reservoirs are maintained within normal levels, the fish remain quite healthy. Increasing dietary P is only necessary if the reservoir levels fall below a critical threshold (~74% of saturation in case of rainbow trout) to avoid growth depression and other clinical deficiencies (Hardy et al., 1993).

There are several methods to increase the available P content of fish diets. One direct approach is to fortify the diet with a highly available P source, such as potassium or sodium phosphates. Another option is to mix a high-P diet with the P-deficient diet in an appropriate ratio. Alternate feeding between these diets may be ineffective, as P-deficient fish still excrete excess P in urine when the dietary P level is high (Sugiura et al., 2000). A more sustainable approach is to increase the availability of inherent P in the diets. Techniques such as the use of phytase, acidification, or fermentation (of ingredients) have been studied extensively to enhance P availability in both fish and animal feed (Bureau and Hua, 2010; NRC, 2011).

6 Conclusions

Research on urinary P has been shaped by numerous landmark studies, many of which are inevitably dated. While recent research may appear more advanced due to new technologies, it often reinforces existing knowledge rather than introducing innovative findings. This paper aimed to compile key studies that contribute to addressing long-standing challenges, particularly the environmental sustainability of aquaculture, focusing on practical research rather than mechanistic or physiological investigations.

The continued growth of global aquaculture production relies heavily on its environmental sustainability. Phosphorus excreted by fish via urine is highly soluble; therefore, particularly harmful to the environment. To reduce urinary P excretion, it is essential to lower the dietary available P content to match, or fall slightly below, the fish's dietary requirement by applying in situ techniques discussed in this review.

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Data availability statement

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