Fish Protein Promotes Skeletal Muscle Hypertrophy via the Akt/mTOR Signaling Pathways

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(Received July 5, 2021)

Summary Skeletal muscle is the largest organ in the body and has a broad range of plasticity, undergoing atrophy in response to aging or disease and hypertrophy in response to nutritional supplements or exercise. Loss of skeletal muscle mass and force increases the risk of falls, impairs mobility, and leads to reduced quality of life. In a previous study, we demonstrated that taking in Alaska pollock protein (APP) for only 7 d increased the gastrocnemius muscle mass in rats. This study was conducted to identify hypertrophic myofibers and analyze how hypertrophy occurs within them. Twenty male rats were randomly divided into two groups and administered a diet of casein or APP for 7 d. The expression of each myosin heavy chain (MyHC) isoform in a cross-sectional area was then measured. MyHC IIb and IIx isoforms exhibited hypertrophic features in the gastrocnemius muscles of the APP-fed rats. Furthermore, comprehensive proteomic analyses were conducted to identify changes in protein expression due to muscle hypertrophy. Our results, evaluated by pathway analyses, indicated that the activity of the growth factor signaling pathway was significantly impacted by APP consumption. Moreover, APP could promote protein synthesis by activating the protein kinase B/mechanistic target of the rapamycin signaling pathway, which is also promoted by exercise.

Key Words Alaska pollock protein, skeletal muscle, hypertrophy, Akt/mTOR signaling, myosin fiber type

Skeletal muscle is the most extensive muscular system in the human body, accounting for approximately 40% of the total body weight. It is responsible for normal movement and imparts tensile strength, and a balance between protein degradation and synthesis is required to maintain skeletal muscle mass. Therefore, changes in protein degradation and/or synthesis can cause skeletal muscle atrophy and hypertrophy. Skeletal muscle consists of two types of muscle fibers: slow-twitch (type I) and fast-twitch (type II), which are heterogeneously distributed in the muscle. The rate of muscle loss with aging is approximately 1–2% per year past the age of 50 (1, 2); in particular, the amount of fast-twitch fiber tends to decrease (3). Skeletal muscle mass

and strength should be maintained to prevent age-related muscle disorders, which can be achieved by the activation of protein synthesis pathways in muscle cells. Several studies have proposed that resistance training, dietary remedies, and nutritional supplements may serve as effective treatment strategies for age-related muscle disorders (4, 5). Various reports have explored muscle hypertrophy via nutritional supplements. A previous study reported that oral lactate administration increased the tibialis anterior muscle weight and fiber cross-sectional area (CSA) in mice (6), and another indicated that taking in leucine along with exercise can activate protein synthesis signaling in muscle cells (7). Multiple studies have reported the importance of boosting muscle mass and maintaining muscle strength by administering a protein-rich diet (8).

Herein, we studied the ability of fish proteins to promote muscle hypertrophy. Fish consumption has risen worldwide in recent years, and several studies have highlighted the beneficial effects of fish proteins (9, 10). Moreover, the nutritional benefits of fish proteins should be determined because the edible portion of fish is rich in proteins and low in fat. Alaska pollock (*Theragra chalcogramma*) is an integral component of processed sea-

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Abbreviations: Akt, protein kinase B; APP, Alaska pollack protein; Atrogin1, F-box protein 32; CSA, cross-sectional area; FOXO, Forkhead box O; IGF, insulin-like growth factor 1; IPA, ingenuity pathway analysis; mTOR, mechanistic target of the rapamycin; MuRF1, muscle-specific RING finger protein 1; MyHC, myosin heavy chain; Myostatin, growth differentiation factor 8.

food products, such as surimi, fish sausages, and kamaboko. We previously reported that Alaska pollock protein (APP) intake can increase the muscle mass and decrease the serum triglyceride levels in rats (11, 12). Importantly, these effects were not attributed to fish oil, because APP was eliminated from fish oil. Our previous study demonstrated a significant increase in the weight of hypertrophic gastrocnemius muscle in APP-fed rats after 7 d without exercise training (13). However, we did not mention “where” (which type of myofibers) hypertrophic features were observed and “how” hypertrophy occurred.

In this study, we investigated the mechanism of APP-induced muscle hypertrophy. Muscle hypertrophy can be induced by the promotion of the synthesis pathway and/or the suppression of the degradation pathway. Physical exercise and nutrient anabolism can up-regulate insulin-like growth factor 1 (IGF-1) upstream of Akt/mTOR signaling and promote protein synthesis (14). In contrast, the Forkhead box O (FOXO)-dependent ubiquitin-proteasome system promotes protein degradation by augmenting the expression of ligase F-box protein 32 (Atrogin1) and the muscle-specific RING finger protein 1 (MuRF1) (15–17). In our previous work, we reported the effect of APP on canceling the atrophic features of the immobilization of the limbs, but we do not know the effect of APP in normal situation (18). We hypothesized that APP consumption may be linked to muscle hypertrophy under normal conditions in different pathway with immobilization. We used iTRAQ-based quantitative proteomic analysis to detect over 20,000 peptides in the gastrocnemius muscle in order to identify the pathway that may be affected and demonstrate how APP causes hypertrophy.

MATERIALS AND METHODS

Materials. As described in our previous study, male Sprague–Dawley rats aged 5 wk were purchased from Japan SLC, Inc. (Shizuoka, Japan). All rats were housed in stainless-steel wire mesh cages in a temperature-controlled room ($23 \pm 1^\circ\text{C}$) under a 12-h light/dark cycle (dark phase: 15:00–3:00) (18). At 6 wk of age, the animals were assigned to one of two experimental groups: the casein diet group, which was fed an AIN-93 diet with casein as the protein source, or the APP group, which was fed a diet containing APP in place of casein (11). The rats were housed separately for 5 d to acclimate to the environment throughout the 7-d feeding experiment with casein and APP. After 7 d of feeding, the rats were sacrificed and the gastrocnemius muscle was excised. This study was conducted in accordance with the ethical guidelines of the Ehime University Animal Experimentation Committee (08A92) and was in complete compliance with the National Institutes of Health: Guide for the Care and Use of Laboratory Animals. All efforts were made to minimize the number of animals used and limit experimentation to what was necessary in order to generate reliable scientific information. These experiments were conducted in September 2018.

Sample preparation. Following decapitation, the soleus, gastrocnemius, and extensor digitorum longus (EDL) muscles were collected on the final day of the experiment along with a non-fasting blood sample. The muscles were frozen in liquid nitrogen-cooled isopentane and stored at -80°C . Approximately 50 mg of the gastrocnemius muscles were cut into pieces and homogenized after adding 500 μL of a lysis buffer to extract total protein. After sonication on ice, the lysate was centrifuged at $10,000 \times g$ for 40 min at 4°C to form pellets. The total protein concentration of the supernatant was determined using a BCA Protein Assay kit (Pierce, Thermo Scientific, Waltham, MA, USA). The supernatant sample was stored at -30°C , and the blood samples were centrifuged at $1,500 \times g$ and 4°C for 10 min and stored at -60°C until analysis.

Immunohistochemical staining to determine the myosin fiber type. Frozen gastrocnemius muscles were cut at the middle of the tissues, trimmed 10–20 times to 40- μm thickness. Serial 10 μm -thick cross-sections of muscles were prepared using a cryostat (CM 1950; Leica Microsystems, Wetzlar, Germany) and mounted on Matsunami Adhesive-coated slides (Matsunami, Osaka, Japan). To differentiate each muscle fiber type, the muscle sections were stained to identify fiber type-specific antibodies (19). The acetone-fixed cryosections were washed thrice with phosphate-buffered saline containing Tween (PBST), blocked with Blocking One Histo (Nacalai Tesque, Inc., Tokyo, Japan) for 20 min, and incubated overnight at 4°C with primary antibodies against MyHC I (#BA-F8), MyHC IIa (#SC-71), and MyHC IIb (#BF-F3), which were all obtained from the Development Studies Hybridoma Bank, University of Iowa. Thereafter, the tissue sections were washed five times with PBST and incubated for 1 h at 23°C with secondary antibodies conjugated to Alexa Fluor 350 (#A21040), Alexa Fluor 488 (#A21042), and Alexa Fluor 555 (#A21127), which were all purchased from Thermo Fisher Scientific KK, Tokyo, Japan. The fluorescence intensity was detected using a wide-field fluorescence microscope (BZ-9000; Keyence, Osaka, Japan). CSA of each myofiber was measured in three independent animals with 560–570 myofibers selected per section. In all cases, the CSA measurement of each individual myofiber (technical replicates) was averaged to obtain a single biological replicate of each muscle sample.

iTRAQ analysis. A 200- μg protein sample was precipitated with six volumes of cold acetone for 2 h at -30°C . The lysate was then centrifuged at $8,000 \times g$ for 10 min at 4°C (20). The samples were reduced, alkylated, digested, and labeled following the instructions of the iTRAQ Reagent Multi-Plex kit (AS SCIEX iTRAQ® Reagents-Chemistry Reference Guide, P/N 4352135). The labeled peptide samples were dried in a centrifugal concentrator prior to fractionation with a High-pH Reversed-Phase Peptide fractionation kit (Thermo Fisher Scientific, Waltham, MA, USA). The peptide fractions were desalted using a C-18 Spin Column and transferred to autosampler vials. A QExactive

Table 1. Anatomical and muscle parameters.

Measurement	Casein rats	APP rats
Food intake (g/7 d)	131.14±1.65	127.98±3.39
Body weight (g)	223.5±2.88	224.8±3.36
Soleus muscle weight (mg)	82.17±1.39	80.97±2.05
Soleus muscle weight-to-BW (mg/g)	0.368±0.003	0.357±0.007
EDL muscle weight (mg)	114.36±1.74	117.41±2.36
EDL muscle weight-to-BW (mg/g)	0.512±0.005	0.518±0.007
Gastrocnemius muscle weight (mg)	1,259.01±22.69	1,365.49±24.37**
GW-to-BW ratio (mg/g)	5.634±0.073	6.075±0.062**

BW: body weight, GW: gastrocnemius muscle weight. Values are the means±SE. $n=10$ per group. ** $p<0.01$ compared to casein-fed rats.

Orbitrap LC-MS (Thermo Fisher Scientific) was used to conduct LC-MS/MS analysis. The raw MS/MS data were converted into the MGF format using Proteome Discoverer 2.0 (Thermo Fisher Scientific). The search was conducted against the UniProt/Swiss-Prot database using the rat taxonomy filter. The identification parameters were set as follows: mass tolerance of 10 ppm and fragment ion mass tolerance of 0.02 Da.

Ingenuity pathway analysis. Ingenuity pathway analysis (IPA; Qiagen, Hilden, Germany) was used to investigate the functional networks of the proteins expressed in the gastrocnemius muscle for the 7-d feeding experiments with casein and APP. The data used for IPA analysis, including all protein IDs (12,886), were obtained from the rat taxonomy database UniProt/Swiss-Prot Accession; 3,901 analysis-ready IDs were identified after eliminating unmapped IDs and low-expression proteins (cut off: -2.0 to 2.0). These analysis-ready IDs (3,901) were consolidated with the expression value set as “average” and analyzed using IPA Content version 49309495. In the signal transduction analysis using IPA, the p -value and activation z -score were calculated for each potential transcriptional regulator. The z -score indicated the up- or downregulation of signal transduction. The detailed procedure is described in the manual of Ingenuity Upstream Regulator Analysis in IPA (21).

Western blot analysis. SDS-PAGE analysis was conducted as described previously (22). The samples were diluted in Laemmli sample buffer and boiled for 10 min at 98°C . The protein samples were separated by 10% SDS-PAGE and transferred onto PVDF membranes (Millipore, Bedford, MA, USA). The membranes were blocked for 1 h in Tris-buffered saline containing Tween (TBST; 20 mM Tris-HCl [pH 7.6], 137 mM NaCl, 0.1% Tween-20) and 5% skim milk. Thereafter, the membranes were incubated overnight at 4°C against Akt (#4691, Cell Signaling Technology, Danvers, MA, USA), phospho-Akt (Ser473, #4060, Cell Signaling Technology), mTOR (#2972, Cell Signaling Technology), phospho-mTOR (Ser2448, #2971, Cell Signaling Technology), p70 S6 Kinase (#2708, Cell Signaling Technology), phospho-p70 S6 Kinase (Thr389, #9234, Cell Signaling Technology), Fbx32/Atrogin1 (#ab168372, Abcam, Cambridge, UK), TRIM63/MuRF1 (#55456-1-

AP, ProteinTech, Rosemont, IL, USA), GDF8/Myostatin (#19142-1-AP, ProteinTech), and GAPDH (#2708, Cell Signaling Technology) with primary antibodies. The next day, the membranes were washed four times with TBST and incubated with horseradish peroxidase (HRP)-conjugated secondary antibodies (anti-rabbit or anti-mouse IgG: 1 : 1,000, Cell Signaling Technology) for 1 h at 23°C . The protein bands were detected using enhanced chemiluminescence (Luminata Forte Western HRP Substrate; Merck Millipore, Bedford, MA, USA) with an ImageQuant LAS 500 imaging system (GE Healthcare, Amersham, UK). Quantification was conducted using ImageJ software (<http://rsbweb.nih.gov/ij/>). To prevent mis-stripping the proteins, the expression of p-Akt and Akt and p-mTOR and mTOR were separately evaluated with the same samples.

Statistical analysis. All data are expressed as the mean±standard error of the mean (SE). Statistical analyses were conducted using the Student's t -test in StatView 5.0 (SAS Institute, Tokyo, Japan), and the level of significance was set at $p<0.05$ (*) or $p<0.01$ (**).

RESULTS

Muscle hypertrophy observed following APP feeding

We first measured the food intake and body and skeletal muscle [soleus and EDL] weights. There were no significant differences in the above parameters when comparing casein-fed (casein rats) and APP-fed (APP rats) rats during the 7-d feeding period (Table 1). In contrast, the gastrocnemius muscle weight and gastrocnemius muscle/body weight ratio of APP rats were significantly higher than those of casein rats. We then observed hypertrophic features in the gastrocnemius muscle tissues. Four major MyHC isoforms are present in the gastrocnemius muscles of mammals: three fast-twitch isoforms (MyHC IIb, MyHC IIX, MyHC IIA) and one slow-twitch isoform (MyHC I) (23). Figure S1 (Supplemental Online Material) shows muscle sections stained with antibodies against MyHC IIb, IIA, I, and IIX in green, red, blue, and black (unstained), respectively. Owing to differences in the muscle fiber compositions, we divided the gastrocnemius muscle into three analytical regions: surface lateral head gastrocnemius (LGaS), deep lateral head gastrocnemius (LGaD), and deep medial head

gastrocnemius (MGasD). LGasS is the surface region consisting of 90% fast fibers (MyHC IIb and IIx), while LGasD and MGasD are deep regions consisting of both fast (MyHC IIb and IIx) and slow (MyHC IIa and I) fibers. The ratios of slow fibers differ between LGasD and MGasD, which are almost 50% and over 60%, respectively. As shown in Fig. 1 and Table 2, in the APP rats, the muscle fiber CSA in LGasS were significantly larger than those of the casein rats. The analysis of cryosections from the transverse gastrocnemius muscles revealed muscle fiber hypertrophy in LGasS (Fig. 1). The CSA of MyHC IIb-expressing myofibers increased by approximately 1.5-fold. The data indicated that the APP rats exhibited typical muscle hypertrophy in fast-twitch MyHC isoforms (Table 2).

Proteomics analyses revealing the activation of the IGF-1 signaling pathway

We conducted iTRAQ analyses to determine differences in the expression of various skeletal muscle proteins. Over 20,000 peptides were detected and analyzed using Proteome Discoverer 2.0, and the identified pro-

teins were grouped by gene ontology. Proteins categorized into “metabolic process” were mainly detected (Fig. 2A). We then selected 86 proteins as variably expressed proteins with differences of ± 1.5 -fold or more between the casein and APP rats. As shown in Fig. 2B, 40% of the different expressed proteins were categorized as metabolic process-regulating proteins. Ingenuity pathway analysis was also conducted to identify the activated signaling pathways, and pathways related to the proliferation and differentiation of muscle cells were observed. Hence, we observed the downregulation of transcription factor forkhead in rhabdomyosarcoma (FKHR) or FOXO signaling and upregulation of Akt/mTOR signaling, which induces protein synthesis, cell growth, and proliferation (Fig. 2C). Akt and mTOR were identified and increased a significant (shown in red), strongly predicting an increase in protein synthase (shown in orange). p70S6K was not detected, which predicted activation. The expression of phosphoinositide 3-kinase p85 (PI3Kp85) and protein phosphatase 2 (PP2A), which are related to cellular functions, was significantly decreased in APP rats (shown in green), predicting the inhibition of downstream of cell death (shown in blue). Consequently, muscle hypertrophy by APP feeding was related to increased cell proliferation and differentiation. Additionally, significant changes in IGF-1 signaling were detected (Fig. S2 (Supplemental Online Material)). Previous studies have demonstrated that exogenous IGF-1 treatment can rescue the atrophic phenotype of gastrocnemius muscle (24). In our previous study, we reported that APP intake ameliorated atrophic features during a 2-wk limb immobilization period and increased *Igf1* gene expression in soleus muscles (18). Therefore, IGF-1 signaling and/or similar pathways may be affected by APP. We then evaluated the expression of these proteins by western blotting analyses.

APP feeding augmented protein synthesis and enhanced Akt/mTOR signaling in skeletal muscle

The expression of proteins involved in the Akt/mTOR signaling pathway and ubiquitin-proteasome markers was further verified by western blotting. As shown in Fig. 3A, the phosphate Akt (p-Akt), mTOR (p-mTOR),

Fig. 1. Immunofluorescent images of the muscle sections stained with each MyHC isoform-specific antibody (green: MyHC IIb, black: MyHC IIx, red: MyHC IIa, blue: MyHC I). Results on the left and right are those for Casein and APP rats, respectively. Scale bar: 100 μ m.

Table 2. Division of gastrocnemius muscle into three regions.

	Muscle fiber cross-section area (μ m ²)					
	LGasS		LGasD		MGasD	
	Casein rats	APP rats	Casein rats	APP rats	Casein rats	APP rats
MyHC IIb	2,393.6 $\pm$ 131.6	3,627.0 $\pm$ 344.0*	2,276.5 $\pm$ 288.9	3,188.1 $\pm$ 225.0	2,237.9 $\pm$ 56.0	3,725.0 $\pm$ 589.6
MyHC IIx	1,473.9 $\pm$ 111.3	2,086.8 $\pm$ 106.4**	1,671.6 $\pm$ 161.8	2,276.6 $\pm$ 77.9*	1,610.1 $\pm$ 31.9	1,881.3 $\pm$ 217.3
MyHC IIa	n.d.	n.d.	1,275.9 $\pm$ 125.6	1,520.5 $\pm$ 100.5	1,352.3 $\pm$ 139.3	1,480.0 $\pm$ 197.2
MyHC I	n.d.	n.d.	1,471.8 $\pm$ 170.9	1,883.1 $\pm$ 96.3	1,663.1 $\pm$ 123.5	2,223.2 $\pm$ 248.2

The muscle fiber CSA was calculated, and statistical analyses were conducted. n.d.: not detected. Values are the means $\pm$ SE. Three independent animals were used. We calculated 560–570 myofibers and averaged per animal. * p <0.05, ** p <0.01 compared to casein-fed rats.

blotting analyses revealed that p-Akt and p-mTOR were upregulated. p70S6K was not identified by iTRAQ; however, the phosphorylation of S6K was increased. Atrogin1, MuRF1, and Myostatin were not identified by iTRAQ, and the western blotting analyses indicated that there were no significant differences between APP and Casein. IGF-1 played a pivotal role in our model. Hence,

Fig. 3. Analysis of protein expression in rat skeletal muscle. Representative western blot analysis of Akt/mTOR signaling pathway markers (A) and ubiquitin-proteasome markers in gastrocnemius muscle (B). Column charts represent Akt, mTOR, p70S6K, Atrogin1, MuRF1, and Myostatin normalized by GAPDH, and the western blots represent phospho-Akt, mTOR, and p70S6K normalized by the total Akt, mTOR, and p70S6K. p-Akt: phospho-Akt, p-mTOR: phospho-mTOR, p-p70S6K: phospho-p70S6K. Data are presented as the mean $\pm$ SE ($n=4-5$). * $p<0.05$, ** $p<0.01$ compared to casein-fed rats. GAPDH was used as a housekeeping control.

Fig. 4. Expression of IGF-1 in rat gastrocnemius muscle and serum. IGF-1 in muscle was normalized by GAPDH. The data are presented as the mean $\pm$ SE ($n=5$).

the expression of IGF-1 was measured in the gastrocnemius muscle and serum, and there were no significant differences, as shown in Fig. 4.

DISCUSSION

Skeletal muscle is a highly malleable organ that responds to various cues, including physical activity, nutrients, and aging. For example, resistance training promotes skeletal muscle hypertrophy and improves metabolic function (27). Additionally, the surgical ablation of synergistic muscle (28–30), ladder climbing (31), and electric stimulation have also demonstrated skeletal muscle hypertrophy, mimicking resistance exercise (32, 33). Kobayashi et al. reported that the gastrocnemius muscle mass of a trained leg was significantly greater (7.8%) than that of the control legs after 12 isometric resistance training sessions (34). Another previous study demonstrated that hypertrophic features require a few weeks of chronic exercise training to man-

ifest in gastrocnemius muscle (35, 36). In this study, APP intake increased the gastrocnemius muscle weight within a 7-d administration period (Table 1). The period was very short, and there was no significant difference in the body weights; the ratio of gastrocnemius muscle/body weight in APP-fed rats was greater than that in Casein-fed rats, indicating that protein synthesis may have been upregulated in the skeletal muscles.

To investigate the hypertrophic muscle features, we measured the CSA of myofibers. In our previous study, we stained gastrocnemius muscle sections with antibodies against myosin proteins specific for fast and slow fibers and measured the CSA of both fiber types (13). However, gastrocnemius muscle is composed of four major MyHC isoforms, which are difficult to distinguish as they exist heterogeneously within the tissues, and their compositional ratio varies between muscle types and regions (Fig. S1). In this study, we attempted to identify myofiber isoforms with hypertrophic features via immunofluorescent analysis (Fig. 1) and measured their CSA values (Table 2) in all regions (LGasS, LGasD, MGasD). Our data clearly showed that the CSA of MyHC IIb and IIx in LGasS were significantly higher in APP rats than casein rats. This means the APP feed might be a valid reason for the hypertrophy of fast fibers. The significant hypertrophy of MyHC IIx in LGasD might show the process of myofiber isoform conversion from MyHC IIx to MyHC IIb. Aguiar et al. reported that 5 wk of resistance training increased the CSA by approximately 1.4-fold (37), and our model also showed an increase of almost 1.4–1.5-fold. No data regarding whether the hypertrophic features could be consistently prolonged or not were available; therefore, further studies with long-term APP feeding should be conducted.

Signaling pathways downstream of IGF-1 are required for controlling the protein synthesis and degradation balance (Fig. S2). IGF-1 signaling upregulated protein synthesis via protein kinase Akt/mTOR signaling and downregulated protein degradation by suppressing the expression of ubiquitin ligase or autophagy via FOXO signaling (14). Resistance exercise-induced muscle hypertrophy is associated with the upregulation of anabolic signaling pathways (p-Akt, p-mTOR, p-p70S6K), and also results in a reduced rate of autophagy with a decrease in the LC3-II/I ratio and an increase in the level of p62/sequestosome 1 (38). Kwon et al. reported that 8 wk of resistance training could result in a 3.2-fold increase in muscle mass and muscular strength (38). Additionally, they also reported the enhancement of the activity of anabolism-related proteins (Akt, mTOR, p70S6K). In this study, we used iTRAQ-based proteome and western blotting analyses to examine the differences in protein expression due to APP feeding (Figs. 2 and 3). As shown in Fig. 3, APP promoted skeletal muscle hypertrophy by activating the Akt/mTOR signaling pathway to enhance protein synthesis rather than inhibit protein degradation, and the underlying mechanism was very similar to that governing resistance training. Although IGF-1 expression remained unaltered (Fig. 4), downstream Akt/mTOR

signaling was upregulated by APP. Other signaling factors that altered Akt/mTOR signaling without IGF-1 may exist. For example, a previous study also identified that mechanical loading can induce muscle hypertrophy and activate Akt signaling without the involvement of the IGF-1 receptor (39, 40), such as insulin receptor and Ca^{2+} (41). The expression of these factors in our models and the experiments using mTOR inhibitors will be evaluated in future studies to investigate factors that affect muscle hypertrophy.

Authorship

Research conception and design: KU, TK, MF, and NGI; experiments: MM, MF, KK, and NGI; statistical analysis of the data: MM, TM, and NGI; interpretation of the data: MM, MF, TM, and NGI; writing of the manuscript: MM, EY, MF, TM and NGI.

Disclosure of state of COI

This study was funded by Nippon Suisan Kaisha, Ltd. Eriko Yoshida and Kenji Uchida are current employees of Nippon Suisan Kaisha, Ltd.

Acknowledgments

This study was supported in part by JSPS KAKENHI Grant Number 21H03330, “Nanotechnology Platform Project” operated by the Ministry of Education, Culture, Sports, Science and Technology (MEXT), Japan (No. JPMXP09 S19NM0048). We would like to thank Editage (www.editage.com) for English language editing.

Supporting information

Supplemental online material is available on J-STAGE.

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