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RESEARCH ARTICLE

Soybean (*Glycine max* L. Merr.) Hexane Extracts Inhibit Cellular Fatty Acid Uptake by Reducing the Expression of Fatty Acid Transporters

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Abstract Intake of saturated and *trans*-fatty acids is a strong risk factor for coronary heart disease. We investigated the inhibitory effects of 2 hexane extracts from white (WBE) and black soybeans (BBE) on cellular fatty acid uptake *in vitro*. Transcellular uptake of elaidic acid (t18:1), a major *trans*-fatty acid present in processed foods, in Caco-2 monolayers was significantly reduced by 28.3 and 16.7% 60 min after WBE and BBE treatment, respectively. Results of flow cytometry (FACS) analysis showed significant reductions in boron-dipyrromethene (BODIPY) fluorescence-labeled fatty acid uptake by 35.4 and 40.2% with WBE and BBE treatment, respectively. BBE treatment significantly reduced the expression of fatty acid transport protein-4 and CD36 in Caco-2 cells, as determined by quantitative real time-polymerase chain reaction (qRT-PCR). Similar trends were found in WBE treatment, although to a lesser degree. These observations suggest that soybean extract may reduce fatty acid uptake and cellular fat accumulation by altering fatty acid transporter expression.

Keywords: soybean, fatty acid uptake, fatty acid transport protein-4, CD-36

Introduction

Triglycerides are neutral lipids consisting of 3 long-chain fatty acids (LCFA, number of carbons >14) esterified to a glycerol backbone. In the digestive process, the free fatty acids are liberated from triglycerides with several lipases and are absorbed by intestinal enterocytes and then re-esterified to triglycerides, incorporated into triglyceride-rich chylomicrons, and secreted into the circulation (1). In fat digestion, intestinal cells take up LCFA via cellular transporters, such as fatty acid transporter protein-4 (FATP-4) or CD36. In addition, it was suggested that fatty acid-binding protein-2 (FABP-2) and acyl-CoA synthetase (ACS) as well as FATP-4 promote cellular fatty acid uptake by accelerating fatty acid acylation (1-3). Thus, the regulation of these proteins will be important in regulation of dietary fatty acid uptake.

High intake of dietary LCFA can cause obesity and heart disease (4, 5). It is well known that high caloric intake from dietary fat directly increases lipid accumulation in adipose tissue and is thus a cause of obesity in humans (6). Among the different types of LCFA, *trans*-fatty acids, which contain at least 1 double bond in the *trans* configuration, are of particular interest due to their potent adverse effects on human health. The *trans*-fatty acids are primarily formed during food processing and heat treatments, including the partial hydrogenation of vegetable oils, commercial cooking, and baking processes (4).

Fat intake in the USA and several European countries averages between 30 and 40% of the total calorie consumption, and *trans*-fatty acid consumption accounts for up to 2 to 3% of total calories consumed (1,4,7). Although the average consumption of *trans*-fatty acids is small, it has a potent metabolic impact. High intake of *trans*-fatty acids significantly affects plasma lipid levels by increasing total and low density lipoprotein (LDL) cholesterol and plasma triglyceride levels (8). *Trans*-fatty acids also increase the levels of serum lipoprotein(a), a strong risk factor for coronary heart disease (CHD) (5). Indeed, it has been reported that a 2% increase in energy

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intake from *trans*-fatty acids was associated with a 23% increase in the incidence of CHD (4).Thus, intake of small amounts of *trans*-fatty acids may have a large impact, and the control of *trans*-fatty acid intake is therefore important for human health.

Soybean (*Glycine max L.* Merr.) has been ingested as a protein supplement in East Asian countries for hundreds of years due to its high-quality amino acid profile. Numerous reports have suggested hypolipidemic effects of soybean, indicating effects in reducing the risk of CHD and atherosclerosis (9-11). Meta-analysis showed that intake of soybean, especially soy protein, is associated with reduced levels of total and LDL cholesterol and lowered plasma triglyceride concentrations (12), and recent reports also showed that anthocyanins, phytochemicals in soybean, can also reduce LDL cholesterol and plasma triglyceride levels (13, 14).

Many studies have indicated that soybean or soybean-derived compounds can reduce plasma cholesterol levels by multiple mechanisms, such as inhibition of bile acid resorption from the intestine and modulation of hepatic cholesterol gene expression (15-17). For example, soy protein has been shown to induce hepatic LDL-receptor expression and to inhibit the expression of HMG-CoA reductase (15). These effects result in removal of cholesterol from the blood by the liver and inhibition of hepatic biosynthesis of cholesterol in mice, thus contributing to the reduction in total and LDL cholesterol in rodents (18). Although the mechanism of cholesterol reduction by soybean has been investigated extensively, the biological mechanism underlying the soybean-induced reduction of plasma triglyceride levels is not well understood.

Plasma triglyceride reduction could be achieved in several ways, and soybean has been suggested to inhibit the uptake of dietary fat, especially *trans*-fatty acids, from the intestine. In the present study, we prepared hexane extracts of white soybean and black soybean and investigated their effects on the uptake of elaidic acid, a major *trans*-LCFA present in processed foods, by Caco-2 human intestinal cells. We also examined the uptake of boron dipyrromethene (BODIPY) fluorescence-labeled fatty acid in 3T3-L1 cells stably expressing hormone-sensitive lipase, fatty acid transporter-1, and perilipin, which were engineered to accumulate in neutral fat droplets.

Materials and Methods

Materials, cell culture, and the preparation of black soybean extract (BBE), and white soybean extract (WBE) Caco-2 cells were purchased from the Korean Cell Line Bank (Seoul, Korea) and 3T3-L1 cells stably expressing hormone-sensitive lipase, fatty acid transport protein-1, and perilipin (HFP cells) were provided by Dr. Andreas Stahl (Department of National Science and Toxicology, UC Berkeley, CA, USA). Elaidic acid, bovine serum albumin (BSA, fatty acid-free), and Lucifer yellow were purchased from Sigma-Aldrich (St. Louis, MO, USA). Dulbecco's modified Eagle's medium (DMEM), fetal bovine serum (FBS), trypsin-ethylenediamine tetraaceticacid (EDTA), penicillin, and streptomycin were obtained from Hyclon (Logan, UT, USA). Hank's balanced salt solution (HBSS) was obtained from Gibco Co. (Grand Island, NY, USA), and gentamycin was purchased from Cassion (Logan, UT, USA). All cells were cultured and incubated at 37°C in a humidified atmosphere of 5% CO_2. The HFP cells were grown in DMEM with 4 mM L-glutamine, 10% FBS, 100 units/mL penicillin, and 100 μg/mL streptomycin, whereas Caco-2 cells were grown in DMEM containing 4.5 g/L glucose, 4 mM glutamine, and 1% non-essential amino acid (NEAA) supplemented with 20% FBS, 25 mM HEPES (pH >6.0), 100 units/mL penicillin, 100 μg/mL streptomycin, and 0.1%(v/v) gentamycin. Black soybeans, white soybeans, and soybean oil (SBO) were obtained from Nonghyup (Suanbo, Korea). The samples were finely ground with a high-speed mixer, and the soybean powder was then incubated with 3 volumes of hexane for 12 hr. After centrifugation, the solvent in the supernatant was removed with a rotary vacuum evaporator, and samples were freeze dried. Soybean powder was extracted 3 times for preparation of hexane extracts for the experiments.

Measurement of total phenolic compounds and total flavonoid contents The amounts of total phenolic compounds in the extracts were determined spectrophotometrically using Folin-Ciocalteu reagent. Briefly, samples were placed in test tubes, followed by the addition of 1.0 mL of 2 N Folin-Ciocalteu reagent and 0.8 mL of 7.5% sodium carbonate. The tubes were mixed and allowed to stand for 30 min. Absorption was measured at 765 nm (UV mini-1240; Shimadzu, Kyoto, Japan). Total phenolic content was expressed as gallic acid equivalents (GAE) in mg/g of dry material. Flavonoid concentration was determined as follows. The samples (50 μL) were diluted in 80% aqueous ethanol (450 μL), and 0.5 mL aliquots were added to test tubes containing 0.1 mL of 0.1% aluminum nitrite, 0.1 mL of 1 M potassium acetate, and 4.3 mL of 80% ethanol. After 40 min at room temperature, the absorbance was determined with a microplate reader (Model 680; Bio-Rad, Hercules, CA, USA) at 425 nm. Total flavonoid concentration was calculated using a quercetin standard curve.

Monolayer evaluation and elaidic acid uptake assay Caco-2 cells were cultured as described previously (19, 20). Cells were plated at a density of 1.4-1.8×10^5 cells/cm^2 onto collagen-coated polytetrafluoroethylene (PTFE) Transwell

filter inserts (12-well plates, 12-mm in diameter, 0.4-μm pore size; Corning, Corning, NY, USA). The medium was changed on alternate days. The integrity of the cell monolayer was verified by measuring the transepithelial electrical resistance (TEER) using a Millicell ERS meter (Millipore, Billerica, MA, USA). Lucifer yellow was employed to check the junction integrity of Caco-2 cells, and luminescence was quantified with a Victor 3 multi-label plate reader (Perkin-Elmer, Shelton, CT, USA) at 485 and 535 nm. Cell monolayers with TEER values >300 Ω/cm^2 (20) and less than 5% Lucifer yellow transmission were subjected to further fatty acid uptake analysis (21). For the uptake assay, confluent Caco-2 cells in Transwell inserts were rinsed. After 1 hr of incubation with HBSS buffer, the cells in the apical compartment were incubated with 0.5 mL of 500 μM elaidic acid-BSA complex solution (pH 7.4) at 37°C. Elaidic acid-BSA complex was prepared as described previously (19,22). Spent HBSS from the basolateral compartment was collected, with a sampling interval of 30 min.

Quantification of elaidic acid uptake by gas chromatography (GC) Elaidic acid was extracted according to the method of Folch (23) and methylated to FAME with boron trifluoride-ester complex. Analysis of elaidic acid methyl ester was performed using an Agilent 7890 coupled with a flame ionization detector (FID) system (Agilent Technologies, Wilmington, DE, USA) with a polar fused-silica capillary column SP-2560 (100 m×0.25 mm, 0.25-μm film thickness; Supelco Inc., Bellefonte, PA, USA) programmed from 140 to 240°C at 4°C/min, held for 5 min at 140°C and for 15 min at 240°C, with the detector at 260°C and helium as the carrier gas. A 2 μL aliquot of the sample solution was injected in splitless mode at 260°C. Reproducibility of injections and performance of the column were satisfactory over the period of investigation. Data were assessed by one-way analysis of variance (ANOVA) using Chemstation software (Agilent Technologies).

Quantification of fatty acid uptake with fluorescence-activated cell sorter (FACS) HFP cells were plated in 6-well plates at a density of 1×10^5 cells/well and grown for 24 hr. BODIPY fluorescence-labeled fatty acid (C_1-BODIPY 500/510 C_{12}, 4,4-difluoro-5-methyl-4-bora-3a,4a-diaza-s-indacene-3-dodecanoic acid, 2 μM in HBSS with 0.1% BSA) was added to each well with a blank solution or one of the treatment solutions, including various concentrations of BBE or WBE, and the culture plates were then incubated for 1 min at 37°C. The assay was terminated by addition of ice-cold 0.2% BSA containing HBSS to each sample. Cells were resuspended with FACS buffer and analyzed by flow cytometry (FACSCalibur; BD Biosciences, San Jose, CA, USA) by determining FL-1 fluorescence, and data analysis was performed with CellQuestPo software (BD Biosciences).

Quantitative real time-polymerase chain reaction (qRT-PCR) Total RNA was extracted from Caco-2 cells using Trizol reagent (Invitrogen, Carlsbad, CA, USA) according to the manufacturer's instructions. The cDNA was synthesized from 2 μg aliquots of total RNA using reverse transcriptase (Mbiotech, Seoul, Korea) with 50 pmol of oligo dT. Gene expression levels were measured with a Bio-Rad iQ5 iCycler system (Bio-Rad) with Bio-Rad iQ SYBR Green Supermix reagent (Bio-Rad). The PCR conditions were 95°C for 3 min followed by 60 cycles of 95°C for 20 sec, 55-61°C for 15 sec, and 68°C for 20 sec. The primers were designed using OligoPerfect Designer software (Invitrogen). The expression levels were calculated by comparison with GAPDH using iQ5 Optical System Software (version 2; Bio-Rad).

Statistical analysis Each experiment was performed at least three times, and all data are expressed as the means and standard error (SEM) except the values shown in Fig. 3, for which variations are expressed as standard deviation (SD). Student's *t*-test was performed for unpaired group comparisons. Statistical significance was defined as $p<0.05$ or $p<0.01$.

Results and Discussion

Total polyphenol and flavonoid contents in BBE and WBE Both BBE (20.0 mg GAs/g, 22.2 mg QEx/g) and WBE (53.8 mg GAs/g, 36.1 mg QEx/g) contained significant total amounts of polyphenol and flavonoid, whereas the amounts in SBO (0.4 mg GAs/g, 7.62 mg QEx/g) were negligible (Table 1). These observations suggest that both BBE and WBE contain significant amounts of antioxidative phytochemicals that may affect cellular fatty acid uptake.

Inhibition of elaidic acid uptake Effects of both soybean extracts on intestinal elaidic acid uptake were quantified with Caco-2 cell monolayers in a Transwell system using the GC-FID method. BBE treatment significantly inhibited transcellular uptake of elaidic acid at 30 min (34.8%, $p=0.009$), whereas WBE did not. However, at 60 min, both BBE (16.7%, $p=0.003$) and WBE treatments (28.3%, $p=0.007$) showed significant reduction of transcellular elaidic acid uptake (Fig. 1A, 1B). SBO showed marginal effects. Both BBW and WBE showed much greater inhibitory effects on the endocellular uptake of elaidic acid in Caco-2 cells (85.3%, $p=0.02$ and 80.2%, $p=0.001$, respectively) than did SBO (Fig. 1C). Although some inhibition of elaidic acid uptake may have been due to competition between lipids in hexane extracts and elaidic

Table 1. Total phenolics and flavonoid contents of BBE, WBE, and SBO

Component[1)]	BBE	WBE	SBO
Total phenolics (mg GAEs/g extract)	20.0	22.2	0.4
Total flavonoids (mg QEs/g extract)	53.8	36.1	7.62

[1)]GAEs, gallic acid equivalents; QEs, quercetin equivalents

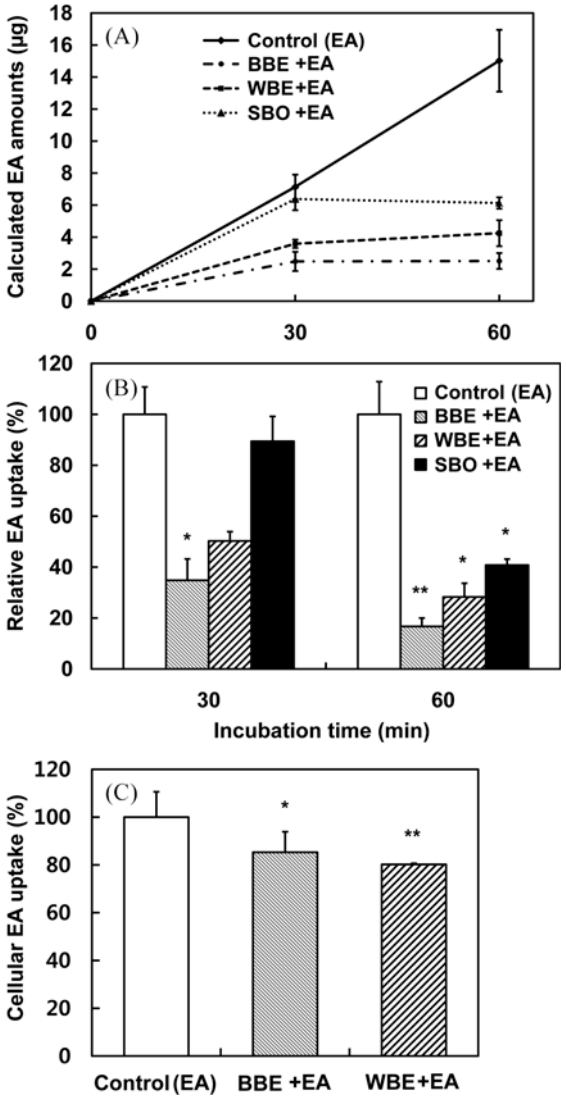

Fig. 1. Elaidic acid uptake by Caco-2 monolayers. Time-course comparison of elaidic acid (EA) uptake with Caco-2 cells grown on membrane inserts (A), relative elaidic acid uptake with Caco-2 cells at 30 and 60 min in BBW- and WBE-treated cells compared with controls (B), relative endocellular uptake of elaidic acid after BBE and WBE treatment in Caco-2 cells (C). All error bars represent SEM; *p<0.05, **p<0.01. BBE, black bean hexane extract; WBE, white bean hexane extract; SBO, soybean oil

acid for cellular uptake, our data strongly suggest that phytochemicals in both BBE and WBE contribute significantly to the inhibition of intestinal uptake of elaidic

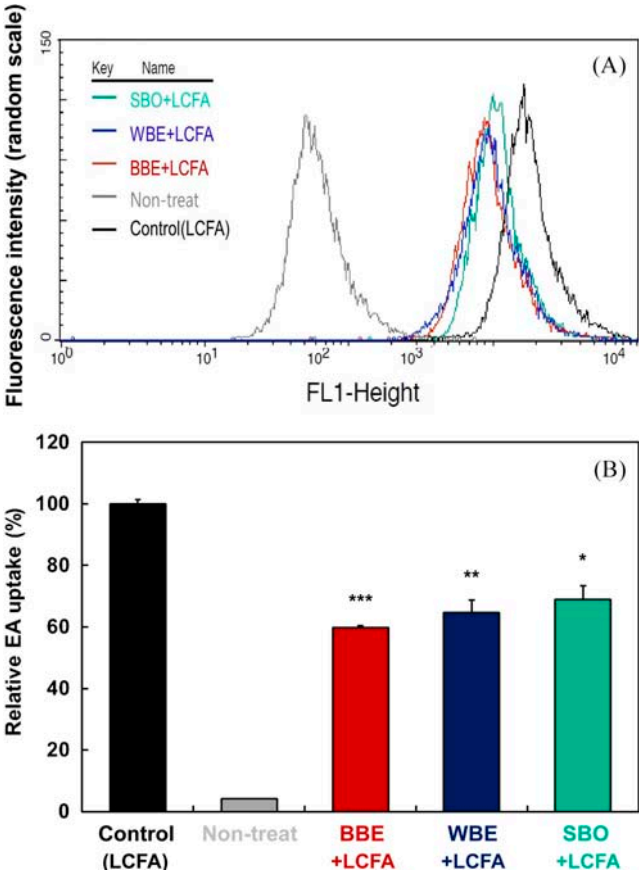

Fig. 2. Inhibitory effects of BBW and WBE on the uptake of LCFA by HFP cells. Intracellular fluorescence of HFP cells quantified with FACS after BBE and WBE treatment. LCFA indicates BODIPY-labeled long-chain fatty acid (A), relative uptake of BODIPY-labeled long-chain fatty acid uptake by HFP cells with BBE and WBE treatment (B). All error bars represent SEM; *p<0.05, **p<0.01, ***p<0.001

acid, as SBO did not show marked effects.

Inhibition of long-chain fatty acid uptake The effects of BBE and WBE on LCFA uptake were quantified in HFP cells using fluorescently labeled LCFA (C_1-BODIPY 500/510 C_{12}). As indicated above, HFP cells were derived from 3T3-L1 cells stably expressing perilipin, fatty acid transporter-1, and hormone-sensitive lipase. These 3 proteins represent the minimum requirements for the accumulation of intracellular fat droplets. HFP cells showing stable expression of these 3 genes were specifically constructed to investigate cellular fatty acid transport. Preadipocyte 3T3-L1 cells take up free fatty acids at marginal levels. However, HFP cells can take up large amounts of free fatty acids and subsequently show accumulation of distinctive cytosolic fat droplets. Incubation of HFP cells with BODIPY-labeled fatty acid increased cytosolic BODIPY-fluorescence, which could be detected by FACS. Compared with controls incubated only with BODIPY-labeled fatty acid, cells incubated with BBE and WBE showed significant

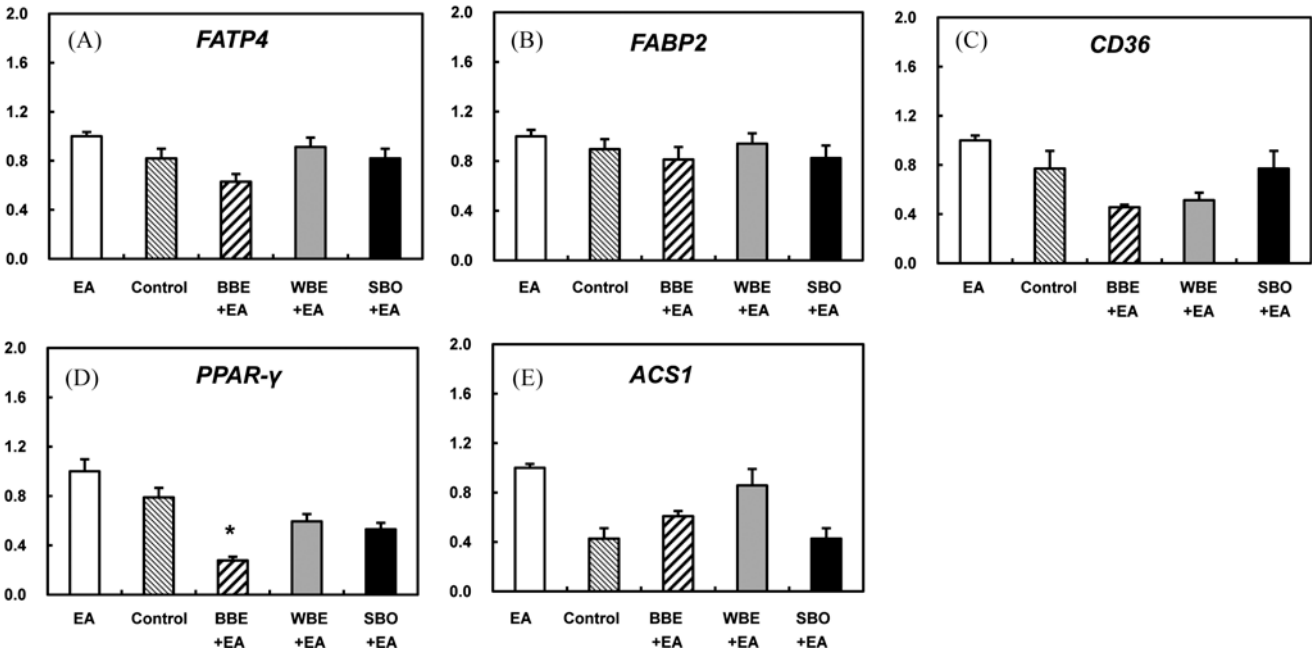

Fig. 3. FATP4, CD36, ACS1, FABP2, and PPAR-γ gene expression in Caco-2 cells incubated with BBE and WBE. The fold change of each mRNA species was calculated as the ratio of the level of mRNA in treated cells to that of the corresponding mean value in control cells. The level of gene expression in each sample was normalized relative to GAPDH and α-tubulin. All error bars represent SD; *$p<0.05$

reduction in cellular fatty acid accumulation by 40.2% ($p=1.2\times10^{-5}$) and 35.4% ($p=0.001$), respectively (Fig. 2A, 2B). SBO showed some reduction, by 31.1% ($p=0.002$), but to a lesser degree. These observations suggest that both BBE and WBE inhibit the cellular uptake of LCFA.

Expression of genes involved in fatty acid uptake To investigate the mechanisms underlying the inhibitory effects of soybean extracts on fatty acid uptake, a total of 5 genes involved in intestinal fatty acid uptake and metabolism were targeted to analyze their transcriptional changes in Caco-2 cells by qRT- PCR (Fig. 3). FATP4 gene expression was reduced by 37% ($p=0.35$) after BBE treatment, whereas WBE treatment resulted in a decrease of only 7% ($p=0.81$). Both BBE and WBE treatments marginally altered FABP-2 gene expression. However, CD36 gene expression was markedly downregulated by both BBE (45.6%, $p=0.25$) and WBE (51.3%, $p=0.25$). Similarly, PPAR-γ gene expression was reduced by approximately 3-fold by BBE treatment (27.8%, $p=0.02$) and by slightly less than 2-fold by WBE treatment (59.4%, $p=0.20$). Expression of the gene encoding ACS1, which facilitates cellular fatty acid uptake mediating TG synthesis, was also downregulated by BBE (39%, $p=0.80$) and WBE (14%, $p=0.20$).

In general, the uptake of fatty acids from the circulation into cells involves several steps: 1) the release of free fatty acids from triglycerides by enzymatic hydrolysis and binding of fatty acids to albumin; 2) transfer from albumin to membrane proteins or the lipid bilayer; 3) uptake across the plasma membrane; and 4) intracellular association with FABP and ACS (1,19). The mechanism of fatty acid uptake across the plasma membrane has been a matter of debate for many years. Initially, it was proposed that fatty acids could traverse the membrane by simple diffusion according to a flip-flop mechanism (24). However, many organs and cell types show a rapid, saturable, substrate-specific, and hormonally regulated LCFA uptake mechanism indicative of protein-mediated processes (25,26). It is widely accepted that both diffusion and protein-mediated mechanisms likely contribute similarly to intestinal uptake, as free fatty acid concentrations in the intestinal lumen are much greater than the levels in blood, whereas protein-mediated mechanisms may be more important in other organs.

Several proteins have been proposed to be involved in the cellular LCFA uptake process, including FATPs, fatty acid translocase (CD36/FAT), FABPs, and ACS (1-3). These proteins are distinctive in their expression pattern in the body as well as their subcellular localization. FATP4 is a major fatty acid transporter in the intestinal epithelium and mediates dietary fatty acid uptake (3). CD36 is a scavenger receptor highly expressed in the intestinal cells that also takes up fatty acids with high affinity and has also been shown to play an important role in cellular fatty acid uptake (1). Our data showed that both BBE and WBE reduced cellular elaidic acid uptake with concomitant downregulation of fatty acid transporters FATP-4 and

CD36 in Caco-2 cells. These observations suggest that the inhibition of fatty acid uptake in Caco-2 cells is mediated by reduced levels of membrane fatty acid transport proteins at significant levels.

In summary, we showed that transcellular uptake of elaidic acid (t18:1) was significantly reduced by both WBE and BBE treatment in Caco-2 monolayers. The results of FACS analysis also showed significantly reduced BODIPY fluorescence-labeled fatty acid uptake associated with both WBE and BBE treatment. Finally, qRT-PCR indicated that BBE treatment significantly reduced the expression of FATP-4 and CD36 in Caco-2 cells. Similar trends were found in WBE treatment, although to a lesser degree. These observations suggest that soybean extract may reduce fatty acid uptake and cellular fat accumulation by altering the expression of fatty acid transporters.

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Erratum

In the February 2011 issue of Food Science and Biotechnology (20-1), acknowledgment was not included:
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On page p.242, Acknowledgments:

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From: shyoon@kfri.re.kr
Sent: 2011년 8월 26일 금요일 오후 1:04
To: kosfost2
Cc: 윤석후; choiyj@snu.ac.kr; kang7820@snu.ac.kr; junelee@korea.ac.kr; sungjoon.lee@gmail.com
Subject: 전체답장 : [식품과학회] Erratum 확인 요청
Attachments: Erratum.pdf

은미씨,

잘 된것 같네요.

윤석후

----- 원본 메시지 -----

제목: [식품과학회] Erratum 확인 요청

정보: kosfost2 <kosfost2@kosfost.or.kr> (2011-08-26 오전 11:54)주소록 등록

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식품과학회입니다.

영문지 이번 8 월호에 나갈 Erratum 입니다.

내용 확인부탁드립니다.

최종 ok 가 나면 바로 인쇄하도록 하겠습니다.

김은미 드림

KOSFOST

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