7 November 2019

Dr. Paulette Gaynor
Office of Food Additive Safety (HFS-200)
Center for Food Safety and Applied Nutrition (CFSAN)
Food and Drug Administration
5001 Campus Drive
College Park, MD
20740 USA

Dear Dr. Gaynor:

Re: GRAS Notice for Shea Butter

In accordance with 21 CFR §170 Subpart E consisting of §§ 170.203 through 170.285, AAK USA Inc. [499 Thornall Street, Edison, NJ], as the notifier, is submitting one hard copy and one electronic copy (on CD), of all data and information supporting the company's conclusion that refined Shea butter and its fractionated derivative products (*e.g.*, shea olein and shea stearin), are GRAS on the basis of scientific procedures, for use in specified food and beverage products across multiple categories; these food uses of Shea butter are therefore not subject to the premarket approval requirements of the *Federal Food, Drug and Cosmetic Act*. Information setting forth the basis for AAK's GRAS conclusions also are enclosed for review by the agency.

I certify that the enclosed electronic files were scanned for viruses prior to submission and are thus certified as being virus-free using Symantec Endpoint Protection 12.1.5.

Should you have any questions or concerns regarding this GRAS notice, please do not hesitate to contact me at any point during the review process so that we may provide a response in a timely manner.

Sincerely,

James S. Jones, Ph.D.
Vice President Customer Innovation
AAK USA Inc.
499 Thornall Street, Edison, NJ
08837 USA
t: 973 344 1300
e: jim.jones@aak.com

AAK

#89

GRAS NOTICE FOR REFINED SHEA BUTTER

SUBMITTED TO:
Office of Food Additive Safety (HFS-200)
Center for Food Safety and Applied Nutrition (CFSAN)
Food and Drug Administration
5001 Campus Drive
College Park, MD
20740 USA

SUBMITTED BY:
AAK USA Inc.
499 Thornall Street, Edison, NJ
08837 USA

DATE:
6 November 2019

GRAS Notice for Refined Shea Butter

TABLE OF CONTENTS

List of Appendices

List of Figures and Tables

GRAS Notice for Refined Shea Butter

Part 1. § 170.225 Signed Statements and Certification

In accordance with 21 CFR §170 Subpart E consisting of §170.203 through 170.285, AAK USA (AAK) hereby informs the United States (U.S.) Food and Drug Administration (FDA) that refined shea butter and its fractionated derivative products (*e.g.*, shea olein and shea stearin), as manufactured by AAK, is not subject to the premarket approval requirements of the Federal Food, Drug, and Cosmetic Act based on AAK's view that the notified substance is Generally Recognized as Safe (GRAS) under the conditions of its intended use described in Section 1.3 below. In addition, as a responsible official of AAK, the undersigned hereby certifies that all data and information presented in this notice represents a complete, representative, and balanced submission, and considered all unfavorable, as well as favorable, information known to AAK and pertinent to the evaluation of the safety and GRAS status of shea butter as a food ingredient for addition to various conventional food products, as described herein.

Signed,

November 6, 2019

James S. Jones, Ph.D.
Vice President Customer Innovation
AAK USA Inc.
499 Thornall Street, Edison, NJ
08837, USA
t: 973 344 1300
e: jim.jones@aak.com

Date

1.1 Name and Address of Notifier

AAK USA Inc.
499 Thornall Street, Edison, NJ
08837 USA

1.2 Common Name of Notified Substance

Shea butter; shea oil; shea nut butter; shea nut oil.

1.3 Conditions of Use

Refined shea butter is intended for use in the following food categories as an alternative or partial replacement to vegetable and animal fats currently used in the following food and beverage categories at levels limited to current Good Manufacturing Practice (cGMP):

- Baked goods and baking mixes, as defined in 21 CFR §170.3(n)(1) of this chapter (U.S. FDA, 2018a);
- Beverages and beverage bases, non-alcoholic, including only special or spiced teas, soft drinks, coffee substitutes as defined in 21 CFR §170.3(n)(3) of this chapter;
- Breakfast cereals, including ready-to-eat and instant and regular hot cereals as defined in 21 CFR §170.3(n)(4) of this chapter;
- Cheeses, including curd and whey cheeses, cream, natural, grating, processed, spread, dip, as defined in 21 CFR §170.3(n)(5) of this chapter;
- Cheese analogs / substitutes / imitation cheese;
- Condiments and relishes, including plain seasoning sauces and spreads, olives, pickles, and relishes, but not spices or herbs as defined in 21 CFR §170.3(n)(8) of this chapter;
- Confections and frostings, including candy and flavored frostings, marshmallows, baking chocolate, and brown, lump, rock, maple, powdered, and raw sugars as defined in 21 CFR §170.3(n)(9) of this chapter;
- Dairy product analogs, including non-dairy milk, frozen or liquid creamers, coffee whiteners, toppings, and other non-dairy products, as defined in 21 CFR §170.3(n)(10) of this chapter;
- Dairy product analogs, including dairy products such as yoghurt or sour cream with partial or full milk fat replacement with vegetable fat/oil;
- Fats and oils, including margarine, dressings for salads, butter, salad oils, shortenings and cooking oils as defined in 21 CFR §170.3(n)(12) of this chapter;
- Fish products (excluding catfish), including all prepared main dishes, salads, appetizers, frozen multicourse meals, and spreads containing fish, shellfish, and other aquatic animals, but not fresh fish as defined in 21 CFR §170.3(n)(13) of this chapter;
- Frozen dairy desserts and mixes, including ice cream, ice milks, sherbets, and other frozen dairy desserts and specialties as defined in 21 CFR §170.3(n)(20) of this chapter;
- Gelatins, puddings, and fillings, including flavored gelatin desserts, puddings, custards, parfaits, pie fillings, and gelatin base salads as defined in 21 CFR §170.3(n)(22) of this chapter;
- Grain products and pastas, including macaroni and noodle products, rice dishes, and frozen multicourse meals, without meat or vegetables as defined in 21 CFR §170.3(n)(23);

- Gravies and sauces, including all meat sauces and gravies, and tomato, milk, buttery, and specialty sauces as defined in 21 CFR §170.3(n)(24) of this chapter;

- Hard candy and cough drops, including all hard type candies as defined in 21 CFR §170.3(n)(25) of this chapter;

- Herbs, seeds, spices, seasonings, blends, extracts, and flavorings, including all natural and artificial spices, blends, and flavors as defined in 21 CFR §170.3(n)(26) of this chapter;

- Herbs, seeds, spices, seasonings, blends, extracts, and flavorings, including all natural and artificial spices, blends, and flavors as defined in 21 CFR §170.3(n)(29) of this chapter;

- Milk products, including flavored milks and milk drinks, dry milks, toppings, snack dips, spreads, weight control milk beverages, and other milk origin products, as defined in 21 CFR §170.3(n)(31) of this chapter;

- Nuts and nut products, including whole or shelled tree nuts, peanuts, coconut, and nut and peanut spreads as defined in 21 CFR §170.3(n)(32) of this chapter;

- Plant protein products, including the National Academy of Sciences/National Research Council "reconstituted vegetable protein" category, and meat, poultry, and fish substitutes, analogs, and extender products made from plant proteins, as defined in 21 CFR §170.3(n)(33) of this chapter;

- Processed vegetables and vegetable juices, including all commercially processed vegetables, vegetable dishes, frozen multicourse vegetable meals, and vegetable juices and blends as defined in 21 CFR §170.3(n)(36);

- Snack foods, including chips, pretzels, and other novelty snacks as defined in 21 CFR §170.3(n)(37) of this chapter;

- Soft candy, including candy bars, chocolates, fudge, mints, and other chewy or nougat candies as defined in 21 CFR §170.3(n)(38) of this chapter;

- Soups and soup mixes, including commercially prepared meat, fish, poultry, vegetable, and combination soups and soup mixes as defined in 21 CFR §170.3(n)(40) of this chapter; and

- Sweet sauces, toppings, and syrups, including chocolate, berry, fruit, corn syrup, and maple sweet sauces and toppings as defined in 21 CFR §170.3(n)(43) of this chapter (U.S. FDA, 2018a).

1.4 Basis for GRAS

Pursuant to 21 CFR §170.30 (a)(b) of the Code of Federal Regulations (CFR) (U.S. FDA, 2018b), AAK has concluded that the intended uses of refined shea butter as described herein are GRAS on the basis of scientific procedures.

1.5 Availability of Information

The data and information that serve as the basis for this GRAS Notification will be sent to the U.S. FDA upon request, or will be available for review and copying at reasonable times at the offices of:

AAK USA
1145 Harbour Way South
Richmond, CA
94804 USA

Should the FDA have any questions or additional information requests regarding this Notification, AAK USA will supply these data and information upon request.

1.6 Freedom of Information Act, 5 U.S.C. 552

It is AAK's view that all data and information presented in Parts 2 through 7 of this Notice do not contain any trade secret, commercial, or financial information that is privileged or confidential, and therefore, all data and information presented herein are not exempted from the Freedom of Information Act, 5 U.S.C. 552.

Part 2. § 170.230 Identity, Method of Manufacture, Specifications, and Physical or Technical Effect

2.1 Identity

The ingredient that is the subject of this GRAS Notice is refined shea butter and its fractionated derivative products (*e.g*., shea olein and shea stearin) (See Figure 2.2-3). Shea butter is a refined triglyceride fat from the shea tree. Shea (*Vitellaria paradoxa C.F. Gaertn;* synonyms*: Butyrospermum paradoxum, Butyrospermum parkii*) is an agro-managed tree crop from the Sapotaceae family, which is found in the wild, growing in parklands in large parts of Sub-Saharan Africa and savannah ecosystems of northern Ghana (Figure 2.1-1). The shea tree provides fruits for direct consumption and is an important source of nutrients and energy for local inhabitants. The shea seeds/kernels are isolated from the fruit bodies (Figure 2.1-1), which are collected from the ground in accordance with good practices for shea fruit harvesting and processing and are then typically processed by roasting or boiling are sold as kernels or further processed into crude, or unrefined shea butter. Since the 1960s, refined shea butter and its fractions have been used in Europe as a food ingredient, mainly in chocolate products. Around half of the crude Shea butter produced (600,000 to 700,000 ton per annum) is used locally and the other half is exported (Jasaw *et al.*, 2015). Out of the total shea butter exported from Africa, 85 to 90% is used in food products and the rest is used in the cosmetic sector (CBI, 2015). The fractioned product shea stearin is the most important product derived from shea butter and is used as a cocoa butter equivalent (CBE) and cocoa butter improver (CBI) in the chocolate and confectionary industry. More recent opportunities for shea fat products include the use of refined olein and whole shea butter as a palm oil replacer (CBI, 2015).

Figure 2.1-1 **Images of (A) Shea Tree Habitat, Cultivation, and Harvesting; (B) Shea Fruit; and (C) Shea Kernel**

(A)

(B)

(C)

Shea butter is characterized by a high level of stearic and oleic acid and contains a higher content of unsaponifiables than most vegetable oils. The main fatty acid constituents of the oil are comprised of palmitic, stearic, oleic, and linoleic acids. Shea butter has no toxic minor components and the risk of contamination with hazardous chemicals in the supply chain is low.

Unrefined shea butter is obtained from the kernel by thermal or cold pressing in a manner that does not alter the nature of the fat and can be semi-purified by washing with water, settling, filtering and

centrifuging. Shea butter is often consumed in its unrefined form in areas where it is harvested locally, but it is more typically subjected to refining, which reduces impurities to produce a clean tasting white paste. A regional standard for shea butter was established by Codex in 2017 (CXS 325R-2017 – CODEX, 2017a). The standard applies to unrefined shea butter intended for direct consumption, or as an ingredient in the manufacture of food products.

Food uses of shea butter outside of Africa are largely limited to refined products and includes products refined into fractions such a shea stearin. As is the case for other food grade vegetable oils and fats, shea butter predominantly consists of lipids, or generically fat (avg. 92 g fat/100 g product). The fat phase is then characterized by mainly triglycerides (avg. 99.7% of the fat phase), unsaponifiable matter (avg. 8 g/100 g product) and traces of monoglycerides and diglycerides. The unsaponifiable matter (avg. 8%) of shea butter consists of triterpene alcohols (5.2 to 6.0% of total butter), hydrocarbons (0.16 to 0.4% of total butter), sterols, and tocopherols.

The triglycerides present in shea butter are predominantly (>80%) the following (Di Vincenzo *et al.*, 2005):

- 1,3-distearoyl-2-oleoylglycerol (StOSt)
- 1-stearoyl-2,3-dioleoylglecerol (StOO)
- triolein (OOO)
- 1-palmitoyl-2-oleoyl-3-stearoylglycerol (POSt)
- 1-stearoyl-2-linoleoyl-3-oleoylglycerol (StLiO)
- 1,2-dioleoyl-3-palmitoylglycerol (POO)

They are the same triglycerides found in common vegetable oils and fats (Gunstone and Harwood, 2007; Padley *et al.*, 1986).

Following the European Inventory of Existing Commercial Chemical Substances (EINECS) nomenclature, the material can also be described as shown in Table 2.1-1, reproduced from Table III.2 – A, entitled "Nomenclature of Shea butter".

Table 2.1-1 Nomenclature of Shea Butter

EC number:	293-515-7
EC name:	Shea tree, ext.
CAS number (EC inventory):	91080-23-8
Synonym	Glycerides C16-18 and C18 unsatd. EC number: 266-948-4 CAS number: 67701-30-8
Molecular formula:	Not applicable
Molecular weight range:	*ca*. 833-889 (predominantly)

2.1.1 Triglycerides and Fatty Acids

The fatty acid composition of shea butter is genetically controlled by the shea tree, and only minor variances in fatty acid composition are reported in shea butter produced from kernels of shea trees growing in different geographical origins. For example, it has been found that oleic acid is dominant in butters from Uganda, while stearic acid is dominant in samples of West Africa provenances (Maranz *et al*., 2004; Di Vincenzo *et al*., 2005; Akihisa *et al*., 2010a). Nevertheless, shea butter is always characterized by a high level of stearic and oleic acid, with little linoleic acid and even less palmitic acid (Table 2.1.1-1). The Codex standard for unrefined shea butter is provided in Table 2.1.1-2.

Table 2.1.1-1 Fatty Acid Composition of Shea Butter from Across Geographical Locations (Nahm, 2011)

Country of Origin	Palmitic Acid (C16:0)	Stearic Acid (C18:0)	Oleic Acid (C18:1)	Linoleic Acid (C18:2)
Benin	3.8	44.1	43.8	6.7
Burkina Faso	3.8	44.1	44.0	6.4
Ghana	4.0	45.6	43.3	6.3
Cote d'Ivoire	6.6	46.8	51.4	8.4
Mali	3.3	43.3	44.6	6.0
Nigeria	3.4	43.8	44.3	5.8
Chad	5.4	32.3	54.8	5.4
Uganda	4.2	28.9	57.8	6.3
Average	4.3	41.1	48.0	6.4

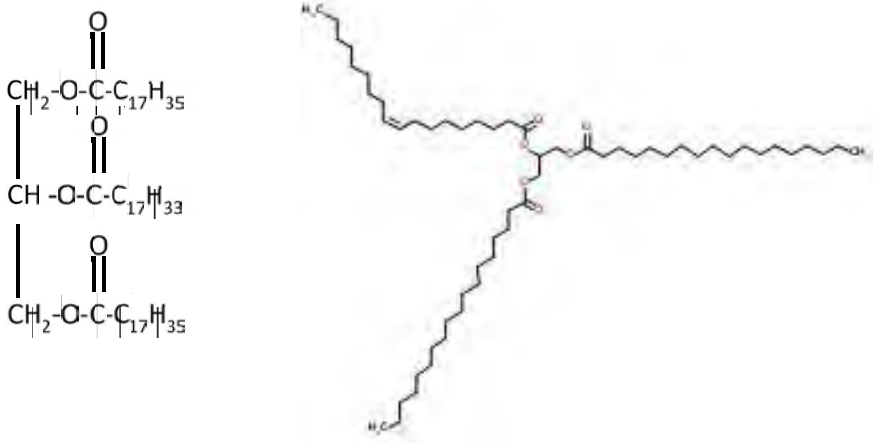
Table 2.1.1-2 Codex Standard CXS 325R-2-17 – Fatty Acid Composition of Unrefined Shea Butter (CODEX, 2017a)

Lauric acid (C 12:0)	<1
Myristic acid (C 14:0)	<0.7
Palmitic acid (C 16:0)	2 to 10
Palmitoleic acid (C 16:1)	<0.3
Stearic acid (C 18:0)	25 to 50
Oleic acid (C 18:1)	32 to 62
Linoleic acid (C 18:2)	1 to 11
Linolenic acid (C 18:3)	1 to 11
Arachidonic acid (C 20:0)	<3.5

Three groups of triglycerides have been identified in shea butter: polyunsaturated, di-unsaturated, and mono-unsaturated; no saturated triglycerides have been reported. The main polyunsaturated triglyceride is OOO (10.8%), while the principal di-unsaturated and mono-unsaturated are StOO (26.8%) and StOSt (40.9%), respectively (Padley *et al.*, 1986; Di Vincenzo *et al.*, 2005). The triglycerides present in shea butter are predominantly (>80%) StOSt, StOO, OOO, POSt, StLiO, and POO. They are further described in the tables of the following sections.

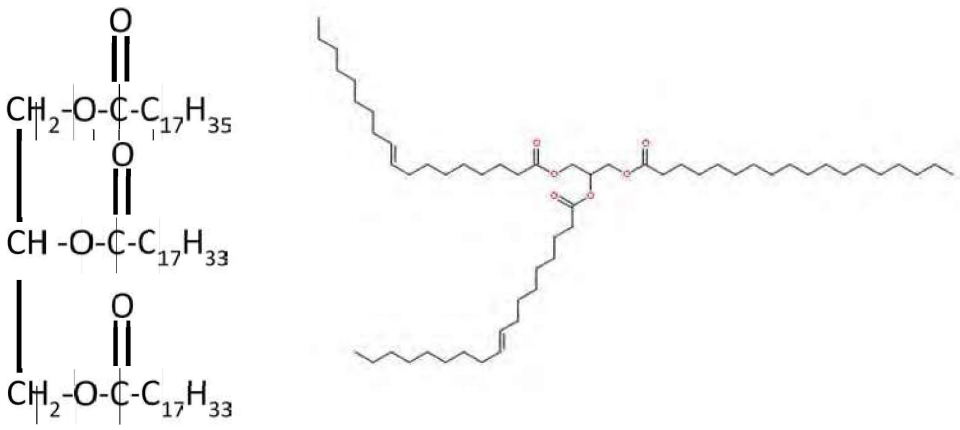
2.1.1.1 1,3-distearoyl-2-oleoylglycerol (StOSt)

Table 2.1.1.1-1 Description of 1,3-distearoyl-2-oleoylglycerol (StOSt)

CAS Number:	2846-04-0
Structure:	
Empirical Formula:	$C_{57}H_{108}O_6$
Molecular Weight:	889.464 g/mol
Synonyms:	1,3-bis(octadecanoyloxy)propan-2-yl (9Z)-octadec-9-enoate, 1,3-bis(stearoyloxy)-2-propanyl (9Z)-9-octadecenoate, 1,3-bis(stearoyloxy)propan-2-yl (9Z)-octadec-9-enoate, 1,3-distearo-2-olein.
Physical Characteristics:	Physical state (20°C and 101.3 KPa): Solid Melting/freezing point: 36.5°C (β'-form) Color: White to very faint yellow Density: 0.9±0.1 g/cm3 Boiling point: 817.6±35.0 °C at 760 mmHg
Found in (mol %):	shea butter (40.9 %), cocoa butter (25.2 %), illipe fat (44 %), tallow (4.8 %) (Padley *et al.*, 1986; Di Vincenzo *et al.*, 2005).

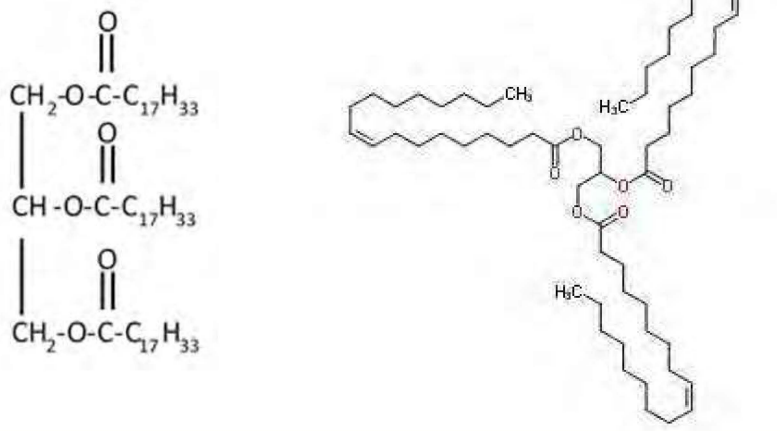
2.1.1.2 1-stearoyl-2,3-dioleoylglecerol (StOO)

Table 2.1.1.2-1 Description of 1-stearoyl-2,3-dioleoylglecerol (StOO)

CAS Number:	2680-59-3
Structure:	
Empirical Formula:	$C_{57}H_{106}O_6$
Molecular Weight:	892.48 g/mol
Synonyms:	1,2-dioleoyl-3-stearoyl-rac-glycerol, 1,2-di(cis-9-octadecenoyl)-3-octadecanoyl-rac-glycerol, glycerol 1,2-di-(9Z-octadecenoate) 3-octadecanoate, 1-O-stearoyl-2-O,3-O-bis[(E)-9-octadecenoyl] glycerol.
Physical Characteristics:	Physical state (20°C and 101.3 KPa): Liquid Melting/freezing point: No data available Color: Colorless to very faint yellow Density: 0.917g/cm3 Boiling point: 819.5°C at 760 mmHg
Found in (mol %):	shea butter (26.8 %), illipe fat (6.0 %), tallow (5.9 %), lard (6.1 %), olive oil (3-7 %), cocoa butter (4.9 %), and butter (1.2 %) (Padley *et al.*, 1986; Di Vincenzo *et al.*, 2005; Gunstone and Harwood, 2007).

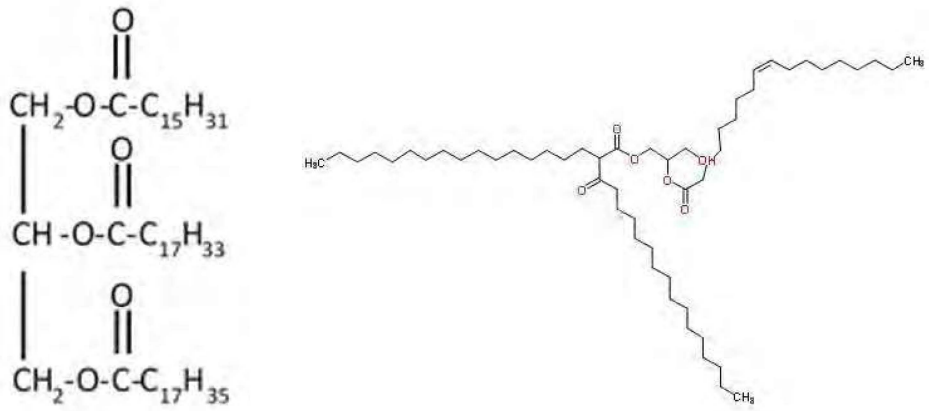
2.1.1.3 Triolein (OOO)

Table 2.1.1.2-3 Description of Triolein (OOO)

CAS Number:	122-32-7	
Structure:		
Empirical Formula:	$C_{57}H_{104}O_6$	
Molecular Weight:	885.43 g/mol	
Synonyms:	1,2,3-propanetriyl (9Z,9'Z,9''Z)tris(-9-octadecenoate), 1,2,3-tri-(9Z-octadecenoyl)-sn-glycerol, 1,2,3-Tri(cis-9-octadecenoyl)glycerol, glycerin trioleate.	
Physical Characteristics:	Physical state (20°C and 101.3 KPa)	Liquid
	Melting/freezing point	-5.5°C
	Color	Colorless to very faint yellow
	Density	0.8988 g/ml (40 °C)
	Boiling point	554.2 °C at 760 mmHg
Found in (mol %):	olive oil (40-59%), rapeseed oil (22.4 %), shea butter (10.8%) (Padley *et al.*, 1986; Di Vincenzo *et al.*, 2005; Gunstone and Harwood, 2007)	

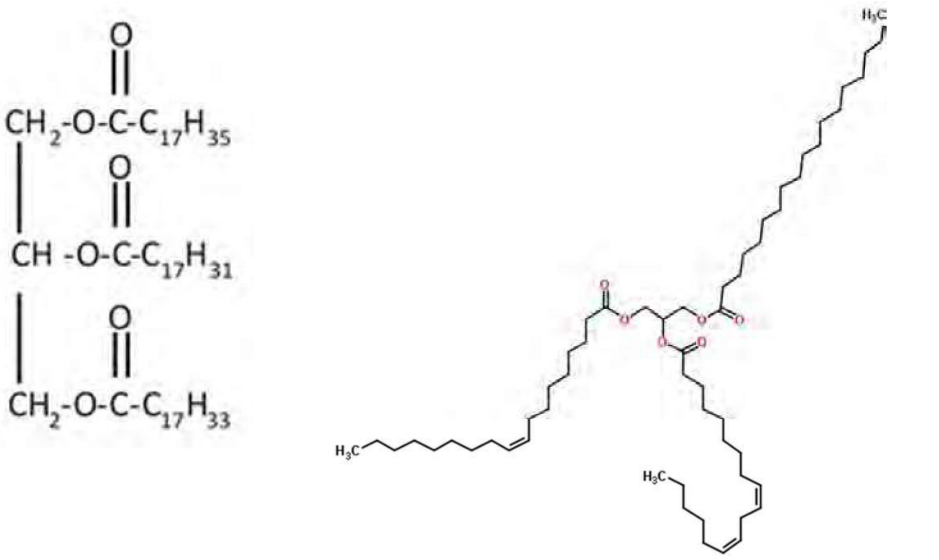
2.1.1.4 1-palmitoyl-2-oleoyl-3-stearoylglycerol (POSt)

Table 2.1.1.4-1 Description of 1-palmitoyl-2-oleoyl-3-stearoylglycerol (POSt)

CAS Number:	2190-27-4
Structure:	
Empirical Formula:	$C_{55}H_{104}O_6$
Molecular Weight:	861.41 g/mol
Synonyms:	1-palmito-3-stearo-2-olein; 1-(((1-oxohexadecyl)oxy)methyl)-2-((1-oxooctadecyl)oxy)ethyl (Z)-9-octadecenoate; 1-palmitoyl-2-oleoyl-3-stearin; 2-oleo-3-palmito-1-stearin; 2-oleo-3-stearo-1-palmitin; 2-oleopalmitostearin; 9-octadecenoic acid (Z)-, 1-(((1-oxohexadecyl)oxy)methyl)-2-((1-oxooctadecyl)oxy)ethyl ester.
Physical Characteristics:	Physical state (20°C and 101.3 KPa): Solid Melting/freezing point: 39°C (β-form) Color: White to very faint yellow Density: 0.915g/cm3 Boiling point: 800.3°C at 760 mmHg
Found in (mol %):	Cocoa butter (46 %), shea butter (5.3%), palm oil (5.1%) (Padley *et al.*, 1986; Di Vincenzo *et al.*, 2005).

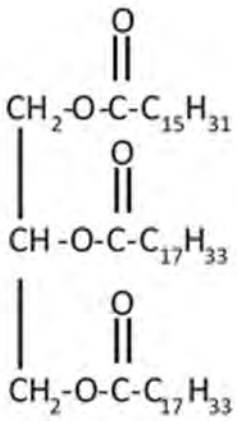
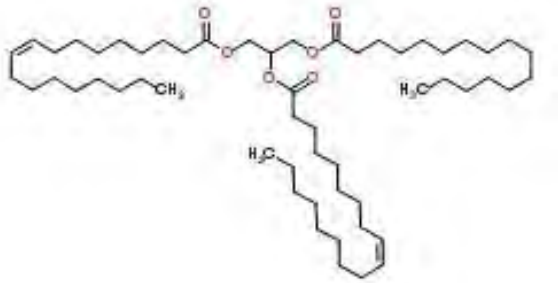
2.1.1.5 1-stearoyl-2-linoleoyl-3-oleoylglycerol (StLiO)

Table 2.1.1.5-1 Description of 1-stearoyl-2-linoleoyl-3-oleoylglycerol (StLiO)

CAS Number:	Not available	
Structure:		
Empirical Formula:	$C_{57}H_{104}O_6$	
Molecular Weight:	885 g/mol	
Synonyms:	1-oleoyl-2-linoleoyl-3stearoyl glycerol.	
Physical Characteristics:	Physical state (20°C and 101.3 KPa)	Liquid
	Melting/freezing point	No data available
	Color	Colorless to very faint yellow
	Density	0.9±0.1 g/cm3
	Boiling point	801.4±55.0 °C at 760 mmHg
Found in (mol %):	Shea butter (5.2 %), olive oil (<3%) (Di Vincenzo *et al.*, 2005).	

2.1.1.6 1,2-dioleoyl-3-palmitoylglycerol (POO)

Table 2.1.1.6-1 Description of 1,2-dioleoyl-3-palmitoylglycerol (POO)

CAS Number:	2190-30-9	
Structure:	 	
Empirical Formula:	$C_{57}H_{102}O_6$	
Molecular weight:	859.39 g/mol	
Synonyms:	1,2-Di(cis-9-octadecenoyl)-3-hexadecanoyl-rac-glycerol, 3-(hexadecanoyloxy) propane-1,2-diyl (9Z,9'Z) bis-octadec-9-enoate, 3-(palmitoyloxy)-1,2-propanediyl (9Z,9'Z) bis(-9-octadecenoate).	
Physical Characteristics:	Physical state (20°C and 101.3 KPa)	Liquid
	Melting/freezing point	No data available
	Color	Colorless to very faint yellow
	Density	0.9±0.1 g/cm3
	Boiling point	802.2±45.0 °C at 760 mmHg
Found in (mol %):	Palm oil (22.8 %), olive oil (20.1 %), shea butter (3.1 %) (Di Vincenzo *et al.*, 2005; Ollivier *et al.*, 2006; Gunstone and Harwood, 2007).	

2.1.2 Unsaponifiable Composition

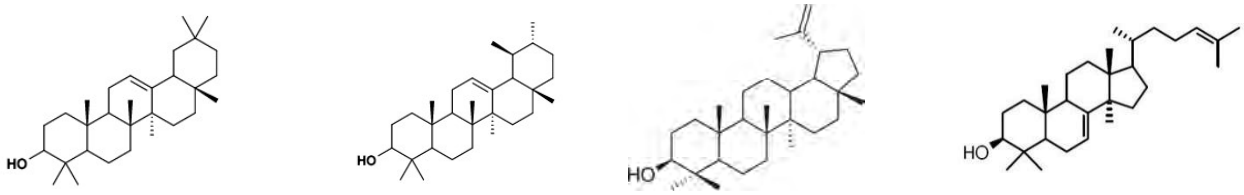
The unsaponifiable fraction of an oil includes all of oil components that remain insoluble after alkaline hydrolysis (saponification). Shea butter is rich in unsaponifiable matter and is a characteristic feature of lipids obtained from shea kernels. Unrefined shea butter has a yellow coloration imparted by the carotenoid composition of the oil, which is removed during refining of the oil (Figure 2.1.2-1). The unsaponifiable content of refined shea butter is typically in the region of 8%, which is higher than that found in most vegetable oils and fats and can range from 1.2 to 17.6% depending on the level of refining (Honfo *et al.*, 2104). In commercial varieties used for food use the amount of unsaponifiables ranges from 4 to 11% (Nahm, 2011). The unsaponifiable fraction of Shea butter is dominated mostly by triterpene alcohols (65 to 75% of total unsaponifiables), followed by hydrocarbons (2 to 5% of total unsaponifiables), sterols and tocopherols (Nahm, 2011).

Figure 2.1.2-1 Refined *vs*. Unrefined Shea Butter

2.1.2.1 Triterpene Alcohol Compounds

The main components of the unsaponifiable fraction of Shea butter are triterpene alcohols (0.8 to 6.2% of total butter), and their acetic and cinnamic acid esters (0.5 to 6.5% of total butter) (Akihisa *et al.*, 2010a,b). 95% of the total triterpene alcohols are made of α-amyrin, β-amyrin, lupeol and butyrospermol, all common triterpene alcohols of other vegetable oils and fats (Ito *et al.*, 1974; Itoh *et al*, 1980; Padley *et al.*, 1986; Akihisa *et al.*, 2010b, 2011).

Figure 2.1.2.1-1 Structure of Major Triterpene Alcohols of Shea Butter



α-amyrin β-amyrin lupeol butyrospermol

2.1.2.2 Hydrocarbons

Shea butter contains hydrocarbons in the form of a polyisoprenoid hydrocarbon, a natural gum-like material also known as karitene (Padley *et al.*, 1986). Hydrocarbons make up 2 to 5% of total unsaponifiables (0.16 to 0.4% of total butter) (Nahm, 2011).

2.1.2.3 Sterols

Major sterols in Shea butter are α-spinasterol and Δ-7-stigmasterol, found also in common vegetable oils and fats (Padley *et al.*, 1986).

2.1.2.4 Tocopherols

The α-tocopherol in shea butter is largely predominant with an average of 112 mg/kg of butter; the γ-tocopherol is second with an average of about 11 mg/kg. β- and δ-tocopherols are almost absent. Despite the lower level of tocopherols compared to that found in palm oil, for example, their amount is still very relevant to the intake of vitamin E locally (Allal *et al.*, 2013).

2.1.3 Contaminants

The contaminants specifications, as given in Appendix A, entitled "Standard contaminants specifications", apply to the Refined Shea butter commercialized by AAK. Specifications are in line with the Codex Alimentarius and FEDIOL (The EU Vegetable Oil and Protein Meal Industry, http://www.fediol.be/). Contaminants analyses of 3 batches of refined shea butter, as given in Appendix A, entitled "Contaminants analysis of Refined Shea butter", show that refined shea butter of AAK is within contaminants specifications.

2.2 Manufacturing

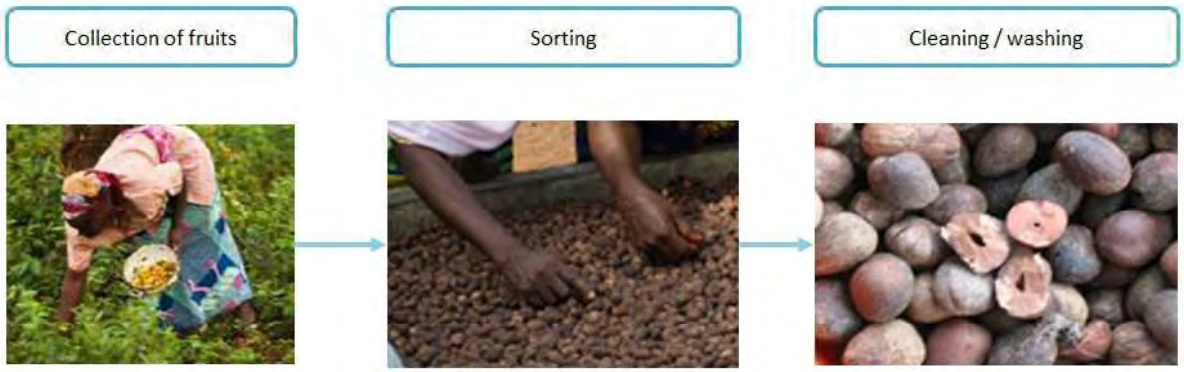
The production process can be divided into 3 parts: curing, extraction, and refining (Figure 2.2-1). The processing of shea fruits starts with curing, which occurs soon after the picking of ripened wild fruits from the fields. Fruits are collected and the green pulp exterior is removed, and the seeds are cleaned to remove any foreign matter from the shea kernels such as dust, stones and metal. Of the total shea kernels collected, half is exported to be further processed, and the other half is extracted in Africa. Of the shea butter extracted in Africa, half is exported to be further processed and the other half is consumed locally as cooking fat.

For the extraction of shea butter, the kernels are crushed. Rolling the seeds fractures the seed coat and directly ruptures many oil cells. Shea kernel grits are then boiled for overall 30 minutes to denature the proteins present in the seeds and to favor the extraction of the oil. Manual or partially mechanized extraction is still very common since more than half of the shea butter is consumed locally as cooking oil. Nevertheless, shea butter for the European and North American markets is extracted industrially in an industrial press mechanically or with the aid of hexane[1], as done for edible seed oils.

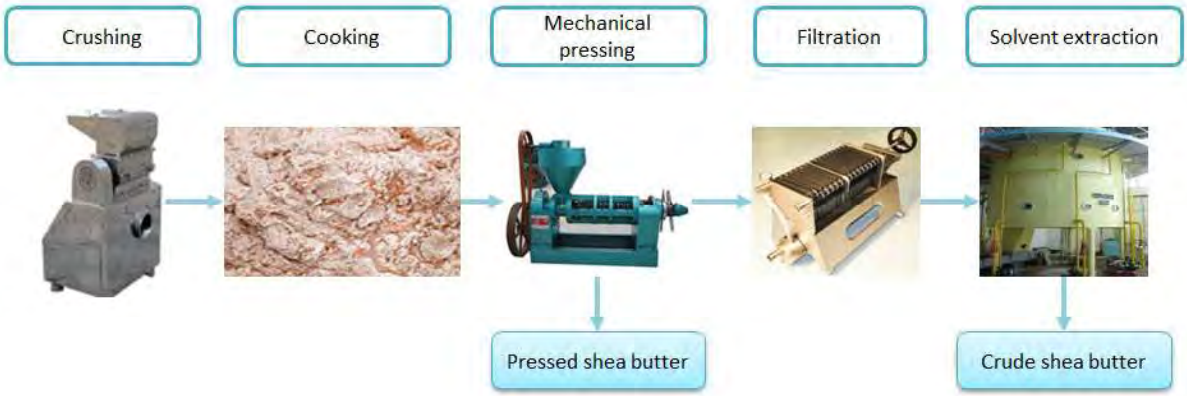
[1] AAK notes that "Commercial hexane" containing about 50–85% n-hexane has been in major use as an oilseed extraction solvent since the 1940's and therefore is GRAS based on history of use prior to 1958. Regulations permitting the use of n-hexane as a solvent in the production of several food ingredients have been promulgated and include limits of 25 ppm for hop extracts (21 CFR §172.560), 25 ppm for marigold extracts (21 CFR §73.295), and 5 ppm in fish protein isolates (21 CFR §172.340) to name a few. Most notable, n-hexane has GRAS status as a minor constituent (not more than 5 ppm) in cocoa butter substitute under 21 CFR 184.1259. In the European Union a maximum residue limit (MRL) for n-hexane in vegetable oils is 5 ppm and in Australia/New Zealand Food Standards Code 1.3.3 imposes limits of 20 ppm for residual levels of hexanes in all foods when used as an extraction solvent. AAK notes that It is generally recognized that no n-hexane residue remains refined oil after processing owing to its high volatility. Moreover, animal-feeding studies with expeller and solvent-extracted meals demonstrate that residual levels of hexane are without adverse health effects in the animals (Wakelyn and Wan, 2004). AAK therefore considers the use of n-hexane in the manufacture of shea butter to be GRAS.

Figure 2.2-1 Production of Unrefined Shea Butter

(A) Curing



(B) Extraction



After the extraction of shea butter, various options exist for refining the butter, which typically occurs in European installations by applying standard refining steps typical of all edible vegetable oils and fats. In all circumstances it involves the removal of free fatty acids and some unsaponifiable matter present in the unrefined shea butter. Most often refining consists of the traditional bleaching and deodorization steps (Addaquay, 2004).

Optionally treatment with alkali (neutralization) and the removal of gums (de-gumming) are also carried out. If applied, de-gumming is the first step of the refining process. The process involves a single-stage phosphoric acid treatment and a single-stage hot water treatment, followed by continuous removal of the hydrated gums in a de-gumming centrifuge. The de-gummed oil is then neutralized with alkali (caustic soda solution 7 to 12%) at about 66 to 77°C to remove free-fatty acids and then washed to reduce the soap content of neutral oil. Effective neutralization results in enhanced effectiveness of subsequent steps (bleaching and deodorization) and furthermore results in high yields of a quality product (Addaquay, 2004).

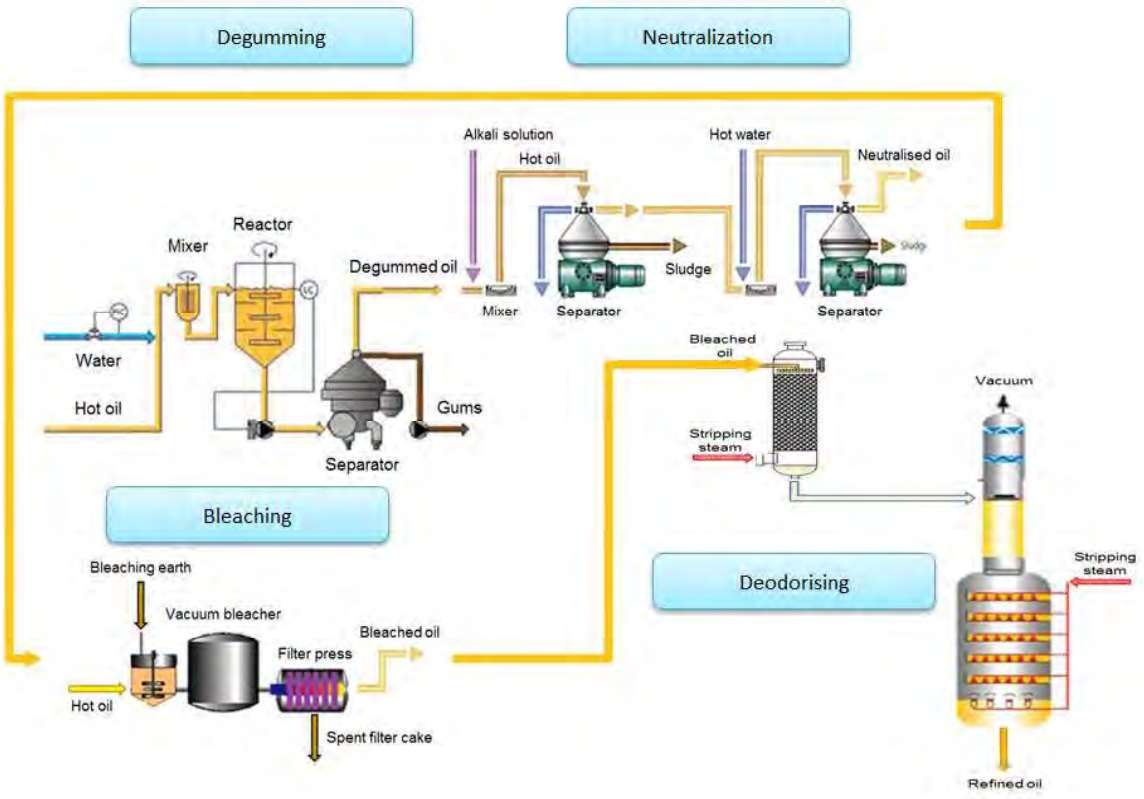
The neutral, washed and dried shea butter still contains some color bodies that have to be removed. Bleaching is then carried out with bleaching earth. The oil is cold-mixed with metered quantities of bleaching earth and thereafter heated to the correct temperature and pumped to a bleaching chamber operating under vacuum where an adequate retention time is provided to ensure effective bleaching. The oil/earth slurry is further pumped through hermetic leaf filters operating in sequence to enable continuous bleached oil (filtrate) discharge (Addaquay, 2004).

Deodorization represents the last major processing step in refining of shea butter and removes compounds that cause undesirable odor, flavor and color. It removes fatty acids and monoglycerides, esters, oxidative reaction products (aldehydes, ketones and peroxides) and many unsaponifiable compounds such as hydrocarbons, sterols and triterpene alcohols (Addaquay, 2004).

Deodorization uses steam distillation under vacuum. The bleached oil passes through a de-aerator where the oil is degassed. The oil is heated to the stripping temperature in a pre-heater. The oil then flows to a flash chamber and descends counter-current to the stripping steam in the form of a very thin film and becomes completely deodorized. The oil from the bottom is further filtered and cooled down (Addaquay, 2004).

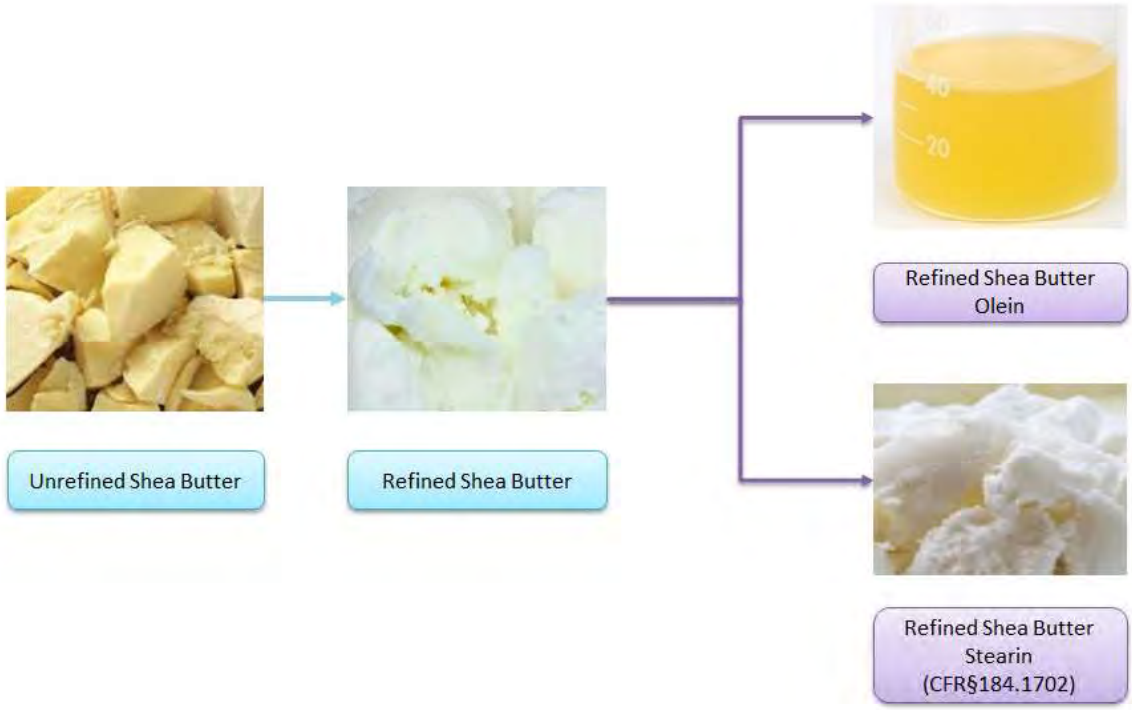
All processing chemicals used in the manufacture of refined shea butter are appropriate for food-use. AAK USA has certified all production processes.

Figure 2.2-2 Refining of Shea Butter



Shea butter is composed of higher and lower melt point triglycerides, which can be separated (fractionated) into a stearin fraction (creamy fat), and an olein fraction (liquid oil). The olein fraction is most commonly used for cosmetic applications and the stearin fraction is used for production of food products such as margarines and confectionaries (Figure 2.2-3). Fractionation of shea butter can be conducted by chemical/mechanical methods or by physical separation. Chemical fractionation is typically conducted under a vacuum and separation is assisted by a solvent to separate the olein from the stearin at different temperatures. Once separated, the olein fraction can be decanted from the stearin fraction. Physical separation methods include sedimentation and centrifugation (WATH, 2004).

Figure 2.2-3 Fractionation of Shea Butter



AAK USA has certified all production processes, and all processing chemicals, food contact articles and additives used in the manufacture of refined shea butter are food grade and are used in accordance with an appropriate federal regulation, have been determined to be GRAS for their respective food uses or have been the subject of an effective food contact notification. Unrefined shea butter used by AAK meets quality standards set forth by Codex for unrefined vegetable oil (CXS 325R-2017 – CODEX, 2017a). The African Organization for Standardization also has released standards for unrefined product (https://www.arso-oran.org/approved-african-standards/).

2.3 Specifications

Refined shea butter meets the quality characteristics specifications mentioned in the "Standard for edible fats and oils not covered by individual standards" (CODEX, 2017b). Specifications for shea butter products marketed by AAK are presented in Table 2.3-1. All analyses are conducted in accordance with validated international standards. Certificates of Analyses demonstrating compliance of refined Shea butter

manufactured by AAK with the ingredient specifications are presented in Appendix B. Certificates of analyses for the fractionated products shea olein and shea stearin are also included in Appendix B. As discussed in Section 2.1.3 refined shea butter products manufactured by AAK are subject to quality control standards for environmental toxins and include routine analyses for dioxins and dioxin-like substances, polychlorinated biphenyls (PCB's), pesticides, and heavy metals such as lead, cadmium and copper (See Appendix A).

Table 2.3-1 Specification – Refined Shea Butter

Parameter		Specification	Methods
Specifications	Color	Off-white	Visual assessment
	Odor and taste	Characteristic of oils and fats and free from rancid odor and taste	Smell and taste
	Acid value mg KOH/g	Max 0.5	IUPAC 2.201; ISO 660: 1996
	Peroxide value meq/kg	Max 10	AOCS cd.8b-90; IUPAC 2.501; ISO 3960:2001
	Iodine value	50 to 75	ISO 3961
Typical values	Matter volatile at 105°C % w/w	Max 0.2	IUPAC 2.601; ISO 662:1998
	Insoluble impurities % w/w	Max 0.05	ISO 663:2000 IUPAC 2.604
	Soap content % w/w	0.005	BS 684 Section 2.5
	Relative density g/ml (40°C)	0.89 – 0.93	IUPAC 2.101
	Melting point °C	25 – 40	ISO 6321:2002
	Refractive index at 44°C	1.455–1.465	IUPAC 2.102; ISO 6320:2000
Compositional analyses	Unsaponifiable matter %	4–10	ISO 18609
	Trans fatty acids (g/100 g)	Max 1	IUPAC 2.304
	Fatty acid composition % w/w		
	Lauric (C12:0)	< 1	ISO 5508:1990 IUPAC 2.201 IUPAC 2.301 IUPAC 2.302 IUPAC 2.304
	Myristic (C14:0)	< 0.7	
	Palmitic (C16:0)	2–10	
	Palmitoleic (C16:1)	< 0.3	
	Stearic (C18:0)	25–50	
	Oleic (C18:1)	36–62	
	Linoleic (C18:2)	1–11	
	Linolenic (C18:3)	< 1	
	Arachidic	< 3.5	

2.4 Stability

Edible fats and oils are not highly perishable ingredients because of the absence of water. Microorganisms require water to grow. Nevertheless, fats and oils have variable shelf lives during which minor changes of their sensory characteristics occur, due to rancidity. Two types of rancidity are known: hydrolytic rancidity and oxidative rancidity.

Hydrolytic rancidity results in the formation of free fatty acids and soaps (salts of free fatty acids) and is caused by either the reaction of lipid and water in the presence of heavy metals or by the action of lipase enzymes. Low levels of free fatty acids are not necessarily objectionable, particularly if they are 16 or

18 carbon fatty acids as commonly found in refined shea butter, since these fatty acids have limited taste. The acid value in fat is used to check progress of hydrolytic rancidity during storage (IUPAC 2.201 or ISO 660: 1996).

Oxidative rancidity occurs in fats and oils that contain unsaturated fatty acids, mostly because unsaturated fats are less stable than saturated fats. Oxidation produces an accumulation of aldehydes and ketones, which are compounds that are also responsible for the unfavorable flavors and odors. One of the most widely used tests for oxidative rancidity is the measure of the peroxide value (AOCS cd.8b-90 or IUPAC 2.501 or ISO 3960:2001).

Storage data show that no oxidative changes are seen in refined shea butter after 12 months storage at ambient temperature in a sealed container.

Part 3. Dietary Exposure

3.1 Current Uses of Shea Butter in the United States

Under 21 CFR §184.1702 'sheanut oil' (aka shea stearin) meeting specifications set forth in the Food Chemicals Codex for sheanut oil is permitted for use in the following food categories at levels not to exceed current good manufacturing practice: Confections and frostings as defined in §170.3(n)(9) of this chapter, coatings of soft candy as defined in §170.3(n)(38) of this chapter, and sweet sauces and toppings as defined in §170.3(n)(43) of this chapter (U.S. FDA, 2018a,c).

During review of GRASP 8G0343, the FDA calculated mean estimated daily intakes of sheanut butter (Fuji Oil Co. Ltd., 1988 as reviewed in U.S. FDA, 1998). The mean and 90th percentile total population intakes of sheanut butter were estimated to be 2 and 2.2 and 4.4 g/person/day for the general U.S. population of consumers >2 years of age. A daily intake of 1.8 g/person/day was estimated from consumption of confections and candies by children 2 to 5 years of age, with 90th percentile estimates of 4.3 g/person/day in this age group (63 FR 28895 – U.S. FDA, 1998).

3.2 Estimated Intake of Shea Butter

Shea butter is intended for use as an alternative fat source to existing food uses of semi-solid vegetable oils and fats (*e.g.*, hydrogenated vegetable oil, coconut oil, palm oil) that are in use in the U.S. marketplace. Specific food use categories are outlined in Section 1.3 and use-levels will be in accordance with cGMP. AAK notes that shea butter will be completely substitutional to existing oils that are used in food and therefore the introduction of shea butter to the U.S. marketplace will not increase the overall intakes of dietary fat by American consumers of these food products. The major fatty acids of shea butter include stearic acid, oleic acid, linoleic, and palmitic acid. As shown in Table 3.2-1 below, with the potential exception of stearic acid, the major fatty acids in shea butter (palmitic, oleic, and linoleic) are compositionally similar to levels that are present within common food oils that shea butter will replace in the diet (*e.g.*, cocoa butter, palm oil and coconut oil). Accordingly, an estimation of the impact of introducing shea butter to the U.S. marketplace on fatty acid intake within the U.S. population from GRAS uses of shea butter should focus exclusively on characterizing the anticipated increases in stearic acid intake.

Table 3.2-1 Fatty Acid Composition of Shea Butter and Comparison to Common Oils and Fats

Fatty Acid	Shea Butter[a]	Cocoa Butter[b]	Palm Oil[c]	Coconut Oil[c]	Beef Tallow[d]
Lauric acid (C12:0)	<1	-	<0.5	45.1–53.2	ND
Myristic acid (C14:0)	<0.7	0.02–0.16	0.5–2.0	16.8–21.0	2–6
Palmitic acid (C16:0)	2 to 10	23.6–30.5	39.3–47.5	7.5–10.2	20–30
Palmitoleic acid (C16:1)	<0.3	-	<0.6	ND	1–5
Stearic acid (C18:0)	25–50	30.2–36.5	3.5–6.0	2.0–4.0	15–30
Oleic acid (C18:1)	32–62	33.2–38.6	36.0–44.0	5.0–10.0	30–45
Linoleic acid (C18:2)	1–11	2.2–4.8	9.0–12.0	1.0–2.5	1–6
Linolenic acid (C18:3)	1–11	0.1–0.2	<0.5	<0.2	< 1.5
Arachidonic acid (C20:0)	<3.5	0.7–1.4	<1.0	<0.2	<0.5

ND = not detected
[a] CXS 325R-2017 (CODEX, 2017a).
[b] Padley *et al*. (1986).
[c] CODEX-STAN 210 – 1999 (CODEX, 2015a).
[d] CODEX-STAN 211 – 1999 (CODEX, 2015b).

An estimation of the intake of stearic acid in the diet from all potential food uses of shea butter is difficult to estimate as shea butter would be unlikely to be present in all potential food applications that a consumer may ingest in a day. It is also impossible to predict the degree to which shea butter would replace current fats that are used in the U.S. marketplace. The existing production volumes of shea butter also are insufficient to completely replace all sources of saturated fats in the U.S. marketplace. Conducting intake estimations of the impact of substituting specific fatty acids in the diet from the partial replacement of current fats with an alternative oil in the diet is highly complicated and theoretical estimates are likely to be gross overestimates of the real-life situation. Given the high level of expected variability (*i.e*., partial replacement to full replacement of existing oils) in the manner by which food manufacturers will use shea butter in food, the data generated from intake estimates are expected to be highly unreliable relative to the real-life situation. Nevertheless, it is noted that existing food uses of solid fats in the U.S. marketplace for bakery products and margarines currently use animal fats (lard, tallow, butter), and tropical oils such as palm, palm kernel and coconut oils. These oils contain high levels of the hypercholesterolemic fats lauric (C 12:0), myristic (C 14:0), and palmitic (C 16:0) fatty acids. The replacement of these fats with shea butter is expected to reduce the intakes of C12:0, C14:0, C16:0 fatty acids and produce a proportional increase in stearic.

Attempts to model the effect of substituting a high stearic acid low linolenic acid soybean oil for hydrogenated soybean oil on fatty acid intake have been reported by DiRienzo *et al*. (2008). The high stearic acid soybean oil used in the intake modeling contained 30% stearic acid and 3% linolenic acid and therefore compares reasonably well with the stearic acid and linolenic acid composition of shea butter (Table 3.2-2).

Table 3.2-2 Fatty Acid Compositions of Two Versions of HSLL Oil *vs*. Regular Soybean Oil and Hydrogenated Soybean Oil (DiRienzo *et al*., 2008).

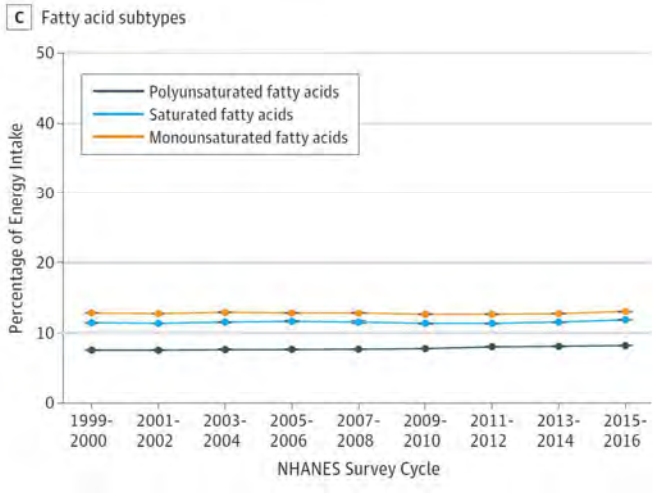
Fatty Acid	Regulator Soybean Oil	Hydrogenated Soybean Oil	HSLL1	HSLL2	Shea butter
Palmitic	10.1	10.8	9.3	8.0	2–9
Stearic	3.6	12.1	18.7	30.0	20–50
Oleic	21.2	30.2	19.8	23.2	40–60
Linoleic	51.3	4.4	44.5	37.1	3–11
Linolenic	6.8	0.2	3.1	3.0	<1
TFA	0.7	32.8	0.39	0.5	-

HSLL1 = high stearic low linolenic soybean oil version 1; HSLL2 = high stearic low linolenic soybean oil version 2; TFA = trans fatty acids.

The authors used National Health and Nutrition Examination Survey (NHANES) 1999–2002 survey data and the following food categories: all baked goods, including yeast-breads and rolls, quick breads, cakes, cookies, pies, pastries, crackers, salty snacks, pancakes and waffles; home use shortenings; French fries and all fried meat, poultry and fish; and margarines. The fatty acid composition for the hydrogenated soybean oil component of the foods was extrapolated from the United States Department of Agriculture (USDA)s's database of fatty acid profiles for 214 foods, and from data provided by the fats and oils industry. Using SAS statistical software, the authors estimated that substitution of hydrogenated soybean oil with a high stearic acid low linolenic acid oil would increase the dietary intake of stearic acid from baseline levels of 3% total energy in the diet at the 90th percentile, to *ca*. 4 to 5% total energy. The intakes of palmitic and linolenic acids were unchanged. Intakes of oleic acid did not appreciably change (9.8 to 10.1% total energy) and linoleic acid intakes decreased from 7.4 to 6.8% of total energy.

AAK considered the applicability of data generated using NHANES 1999-2000 survey data to current food use trends. As reported by Rehm et al., (2016) dietary intake of saturated fat as a % of total energy was reported to be 10.9 in the 1999-2000 NHANES, a value that was unchanged in 2011-2012 survey, which reported saturated fat intakes of 10.7% of energy. Similar findings were reported by Shan et al., (2019) where the authors reported no appreciable changes in saturated fat intake between the 1999-2000 and the 2015-2016 NHANES surveys (See Figure 3.2-1).

Figure 3.2-1 **Trends in Estimated Percentage of Energy Intake from Fat Among U.S. Adults Aged 20 Years or Older by NHANES Survey Cycle From 1999-2000 to 2015-2016.**



Based on the compositional similarity of shea butter to the experimental oils investigated by DiRienzo *et al*. (2008), extrapolation of the findings reported by the authors to the effects of introducing shea butter to the U.S. marketplace will likely produce a similar pattern of changes in fatty acid intakes.

Part 4. Self-Limiting Levels of Use

No known self-limiting levels of use are associated with refined shea butter.

Part 5. Experience Based on Common Use in Food Before 1958

The GRAS status of shea butter as described herein is based on scientific procedures in accordance with 21 CFR 170.30(a)(b) and therefore experience based on common use in food before 1958 is not applicable (U.S. FDA, 2018b). A discussion of the history of safe consumption of shea butter and its derivative products is discussed in Section 6.2.

Part 6. Narrative and Safety Information

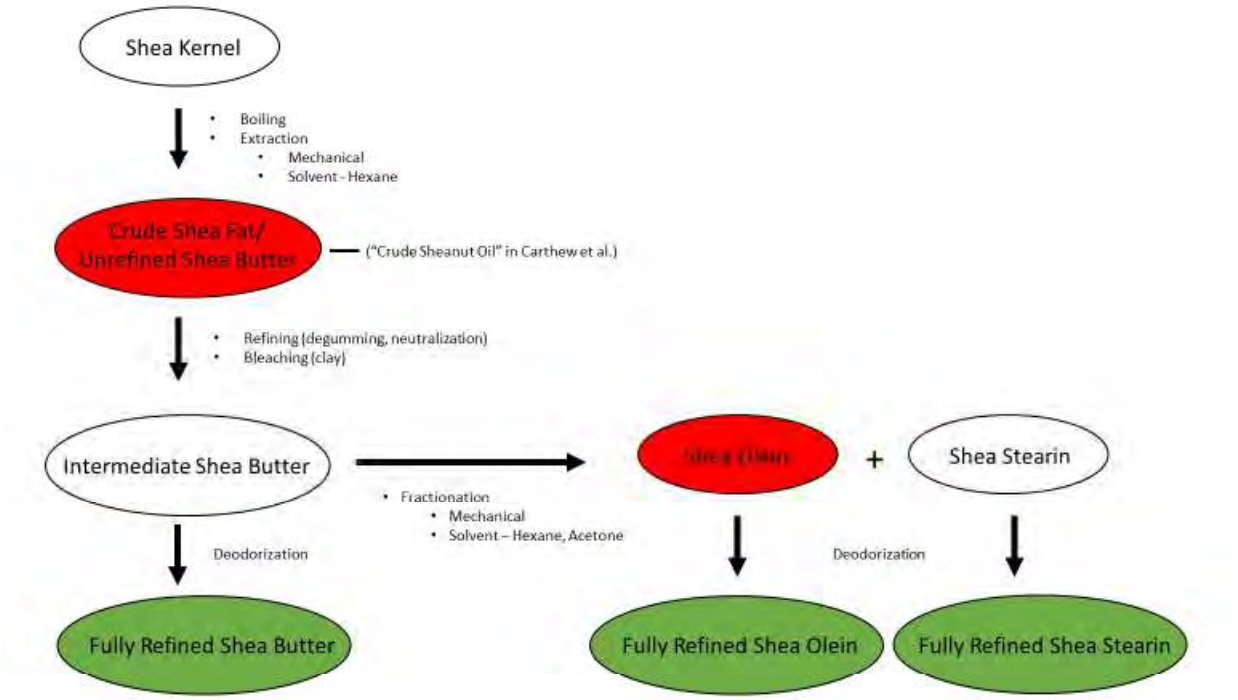
6.1 Introduction

The subject matter of this GRAS evaluation is refined shea butter obtained from the fruit of *Vitellaria paradoxa.* Shea butter is composed of triglycerides containing oleic acid, stearic, linoleic and palmitic acids as the major fatty acids. All fatty acids in shea butter are common fatty acids in the diet and the triglyceride composition is similar to other fats that have a history of safe consumption in the diet (Table 3.2-1). During digestion shea triglycerides are hydrolyzed to mainly oleic acid, stearic acid, linoleic acid, palmitic acid and 2-monoglycerides. These components are naturally found as part of glycerides, lipids, lipoproteins, and membranes of both plants and animals. Moreover, these fatty acids, monoglycerides and glycerol components as are found in a broad range of edible fats, oils, and emulsifiers that are GRAS, and therefore are part of typical human diet. The synthesis and metabolism of these substances are well understood and are documented in biochemistry textbooks (Nelson and Cox, 2008).

Shea butter will serve as an alternative to existing sources of saturated fats, including hydrogenated vegetable oils, that are used in the U.S. food supply. The introduction of shea butter will therefore not increase intake of dietary fat in the U.S. population. Based on the fatty acid composition of shea butter, substitution of current saturated oils with shea butter could potentially result in small increases in stearic acid intake at the expense of palmitic acid (see Section 3.2). AAK notes that stearic acid is a major fatty acid constituent of beef tallow and hydrogenated and partially hydrogenated vegetable oils, oils that have a long-history of use in the U.S. diet. Although health concerns related to trans-fat have resulted in significant removal of beef tallow and hydrogenated fats from the U.S. marketplace (with GRAS status removed for partially hydrogenated oils), it should be recognized any changes in stearic acid intake from the introduction of shea butter to the U.S. marketplace will likely fall within ranges that are represented by the historical uses of beef tallow and hydrogenated vegetable oils in the U.S. diet.

Outside of the higher content of stearic acid, which differentiates shea butter from other common vegetable oils, shea butter is unique in its high content of unsaponifiable matter. As discussed throughout this notice, unrefined, and various refined and fractionated (*e.g.*, shea olein, shea stearin) preparations of shea butter have a long history of safe use globally. Consumption of unrefined shea butter rich in unsaponifiables continues to be a staple source of dietary fat for large-populations of Africans where shea trees are harvested. Commercial preparations of refined shea butter and other fractionated derivatives such as shea olein also contain significant quantities of unsaponifiable matter and these ingredients have a long history of safe use globally in confectionary products. AAK is not aware of any documented evidence of shea butter containing toxic or other undesirable substances that would render commercial oil preparations containing usual quantities of unsaponifiable matter as unsuitable for food use. During the FDA's GRAS affirmation of 'sheanut oil' the agency established specifications to ensure removal of "excessive" unsaponifiable material from the oil (63 FR 28895 – U.S. FDA, 1998). Under 21 CFR §184.1702 the content of unsaponifiable matter in sheanut oil is not to exceed 1.5% (U.S. FDA, 2018c). The agency did not further elaborate on the rationale for reducing the unsaponifiable matter content of the oil, nor did the agency make any reference to the unsaponifiable matter being unsafe. During review of the GRAS status of "sheanut oil" the FDA primarily relied on published data and information establishing that history of safe consumption of shea oil preparations in the diet prior to 1958. However, the agency also reviewed a series of unpublished studies characterizing the hazard of 2 commercial preparations of crude/unrefined shea butter and shea butter olein (see Figure 6.1-1).

Figure 6.1-1 Shea Butter Preparations Evaluated for Toxicity by Carthew *et al*. (2001)



The test articles (red circles) used in the study included an unrefined shea butter ("crude sheanut oil") preparation representative of commercial products consumed throughout Africa, as well as olein, a fractionated preparation high in oleic acid. These preparations contained 5% and 6.8% unsaponifiable matter respectively. Shea butter (green circle) marketed by AAK is a refined fat produced from the crude fat. Shea butter can be fractionated to produce stearin and olein fractions (green circles).

The Agency stated that these studies corroborated the safety of sheanut oil and

> "[…] *establish that sheanut oil has absorbability comparable to that as the other tested GRAS oils and fats. These studies also establish that the growth rates for the subject animals were comparable to those for animals fed other GRAS oils and fats. The fourth study, which is unpublished, is a 104-week toxicity/carcinogenicity study of sheanut oil and other oils in rats. The results of this 104-week study demonstrate that there is no carcinogenic potential for sheanut oil*" (63 FR 28895 – U.S. FDA, 1998).

These studies have since been published in the peer-reviewed literature (Baldrick *et al*., 2001; Carthew *et al*., 2001; Earl *et al*., 2002a,b), and therefore AAK considers these findings to be pivotal to establishing the GRAS status of shea butter in accordance with scientific procedures. These studies are discussed further in Sections 6.4. The findings reported by the authors are consistent with FDA conclusions above that shea butter is without evidence of toxicity in animal safety studies at high dietary inclusion rates and support a conclusion that the unsaponifiable component of typical commercial preparations of the oil is without safety concern. AAK notes that shea butter ingredients manufactured by the company are largely characterized by products rich in stearic acid or oleic acid. Published animal toxicity studies characterizing the hazard of shea butter have included studies conducted using unrefined shea butter rich in stearic acid, studies conducted using the oleic acid rich fraction (shea olein), as well as hydrogenated shea olein

preparations. These test articles are compositionally representative of the spectrum of fatty acid profiles that can be produced from refined shea butter, and also includes hazard characterization of the unsaponifiable fraction at levels of 5 to 6%, which are similar to the unsaponifiable content of shea butter manufactured by AAK (See Appendix B). As the refining process for shea butter does not produce significant compositional changes to the oil but largely serves to reduce the level of impurities (*e.g.*, free fatty acids, phospholipids, environmental contaminants, color bodies and odorous aldehydes and ketones) in the oils giving them a neutral bland color and flavor, studies conducted using unrefined shea butter preparations were relevant to the safety of AAK's shea butter ingredients.

AAK notes that animal toxicity studies of shea butter (*i.e.,* Baldrick *et al*., 2001; Carthew *et al*., 2001; Earl *et al*., 2002a,b) have been published for almost 20 years and therefore have been generally available to the general scientific community for a considerable period of time without reports from qualified experts or authoritative bodies questioning the authors findings or the suitability of shea butter for food use. Based on the longstanding availability of these studies in the literature, combined with the current widespread use of shea butter and shea butter derivatives in the diet globally, it can be concluded that there is no generally available data that would call into question conclusions that historical, current, or future uses of shea butter would be unsafe for food use.

As discussed, the introduction of shea butter to the U.S. marketplace as an alternative to existing food uses of saturated fats that are low in stearic acid may result in a small increase in dietary intake of stearic acid. No changes in dietary intakes of other major fatty acids (oleic, palmitic, linoleic) were anticipated as the compositions of these fatty acids in shea butter are largely similar to levels in oils they will replace. The safety assessment of exposures to the major fatty acids of shea butter from all proposed food uses therefore focused exclusively on the impact of potential changes in stearic acid intake. Based on intake simulations that have been done for other stearic acid rich oils, replacement of current saturated fat sources with shea butter is likely to increase the percent contribution of stearic acid in the diet (as % total energy) from 3 to 4% in mean consumers and between 3.3 to 5.4% in 90th percentile consumers. For an individual consuming a 3,000-calorie diet this increase in stearic acid intake would roughly approximate to an additional 6 g of stearic acid assuming an energy contribution of stearic acid of 9 kcal/g fat. These increases in stearic acid intake would likely occur at the expense of palmitic and oleic acids. AAK reviewed published clinical studies evaluating the effects of shea butter and stearic acid rich oils on various physiological outcomes including measures of lipid metabolism, inflammatory and hemostatic risk factors (see Section 6.7.4). There were no findings in these studies to suggest that consumption of stearic acid would adversely impact human nutrition in a manner that is different from other vegetable oils. General recognition of this conclusion is supported by a scientific opinion from the EFSA on the substantiation of health claims related to stearic acid and maintenance of normal blood cholesterol concentrations pursuant to Article 13(1) of Regulation (EC) No 1924/20061 (EFSA, 2010). Although EFSA was unable to establish a dose-response relationship between stearic acid and the claimed effects on lipid metabolism, the agency did conclude, based on the available evidence, that the "[…] *clinical trials show that stearic acid does not increase total and LDL* [low-density lipoprotein]-*cholesterol concentrations as much as SFA* [saturated fatty acid] *with 12-16 carbon atoms".* As stearic acid is anticipated to be substitutional to palmitic, lauric and myristic acids in the diet, AAK has concluded that introduction of shea butter to the U.S. marketplace as an alternative to current saturated fats in the diet would not be nutritionally disadvantageous to U.S. consumers.

Based on the above publicly available data and information AAK has concluded that the intended uses of shea butter in food, as described in Section 1.3 above can be concluded to be GRAS in accordance with scientific procedures. Further discussion of the basis for this conclusion is presented in the sections that follow below.

6.2 History of Safe Consumption

Refined and unrefined shea butter has been used extensively in Africa as cooking oil since at least the 19th century (Fuji Oil Co. Ltd., 1988). It is assumed that 40% of African people leaving in the shea tree growing zone (= 42.8 million people) consume shea butter on a daily basis for an equivalent of about 21 g of shea butter per day (Levitt, 2013).

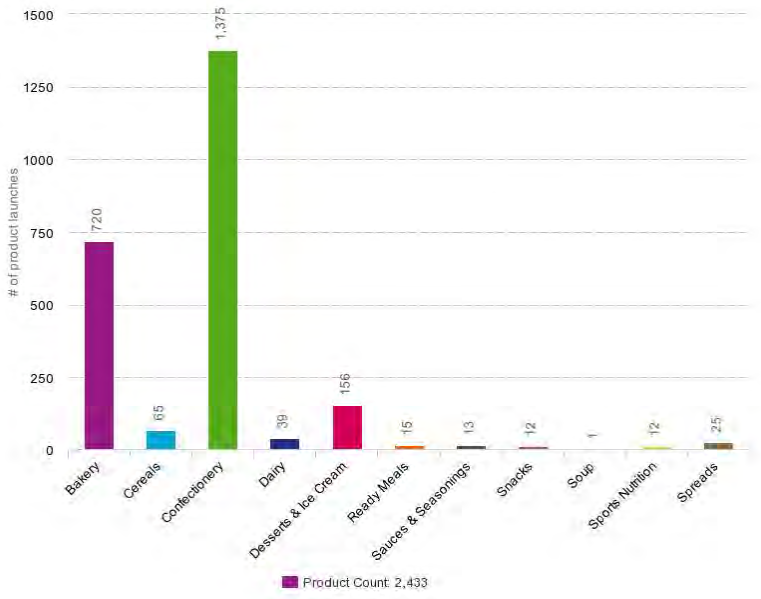
Exportation of shea butter from Africa to Europe has occurred since the latter part of the 19th century and it became more significant in the 1930s. In Europe, the oil was refined or fractionated and used both as a cooking oil, as a cocoa butter substitute and also for making margarines. Available trade statistics show that, except for parts of World Word II, European imports of shea kernels were steady in the late 1930s and the 1940s and have continued since then (Fuji Oil Co. Ltd., 1988).

In the 1950s, the solvent fractionation process for refining and fractionating shea butter was developed. This process, which resulted in at least 2 patents, allowed the manufacture of fractions suitable for the chocolate business (Fuji Oil Co. Ltd., 1988).

6.2.1 Europe

In 2000, the European Union provided regulatory clearance for the use of refined shea butter as a partial substitute for cocoa butter fats used in chocolate production (up to 5% content by weight is allowed) (Directive 2000/36/EC) (EC, 2000). Since then food uses of refined shea butter and its fractions has continued to increase and is now a popular fat for a number of food applications (Figure 6.2.1-1). Figure 6.2.1-1 represents the new product launches between January 2013 and December 2015 and it is an underestimation of the total amount of food products in Europe containing refined shea butter. In almost all products shown refined shea butter is used in blends with other vegetable oils (Fuji Oil Co. Ltd., 1988).

Figure 6.2.1-1 Products Launched in the European Union between January 2013 and December 2015 Containing Shea as an Ingredient (Innova Database)



6.2.2 United States

Shea oil was commercialized in U.S. since 1998 following the GRAS affirmation petition status of Sheanut oil (GRASP 8G0343) submitted by Fuji Oil Company Ltd., 1988. Under 21 CFR §184.1702 'Sheanut oil' is defined as an oil product from the Shea tree *Butyrospermum parkii* and is composed principally of triglycerides containing an oleic acid moiety at the 2-position and saturated fatty acids, usually stearic or palmitic acids, at the 1- and 3-positions (U.S. FDA, 2018c). Under the regulation Shea oil is characterized as having an iodine value of between 28 and 43, and an unsaponifiable matter content of <1.5%. The regulation is therefore is largely limited to the use of stearin fractions produced from shea butter.

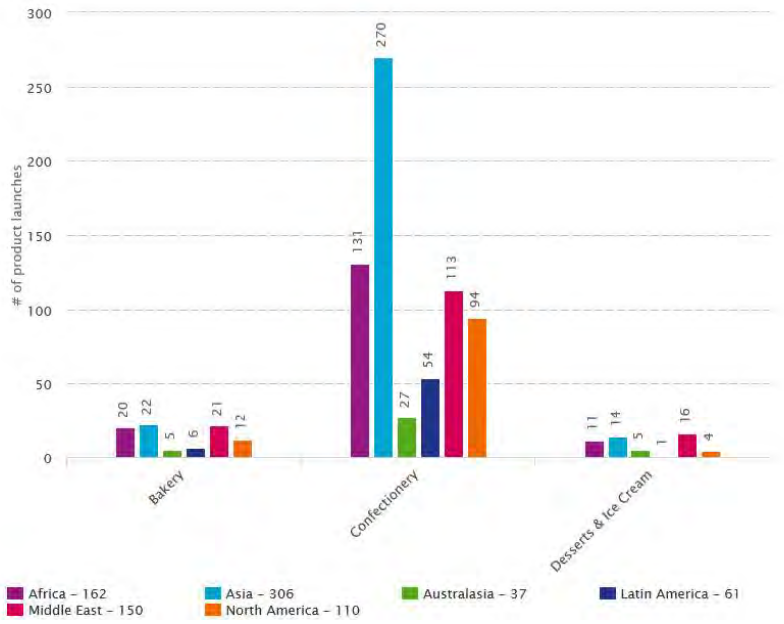
The ingredient is permitted for use in the following food categories at levels not to exceed current good manufacturing practice, except that the ingredient may not be used in a standardized food unless permitted by the standard of identity: Confections and frostings as defined in §170.3(n)(9) of this chapter, coatings of soft candy as defined in §170.3(n)(38) of this chapter, and sweet sauces and toppings as defined in 21 CFR §170.3(n)(43)\.

During review of GRASP 8G0343, the FDA calculated mean estimated daily intakes of shea butter (Fuji Oil Co. Ltd., 1988). The mean and 90th percentile total population intakes of shea butter were estimated to be 2 and 2.2 and 4.4 g/person/day for the general U.S. population of consumers >2 years of age. A daily intake of 1.8 g/person/day was estimated from consumption of confections and candies by children 2 to 5 years of age, with 90th percentile estimates of 4.3 g/person/day in this age group (63 FR 28895 – U.S. FDA, 1998).

6.2.3 Other Parts of the World

As shown in Figure 6.2.3-1 shea butter is well represented in the bakery, confectionary and desserts & ice cream food categories also in Middle East, Latin & North America, Australasia, and Asia.

Figure 6.2.3-1 Products Launched in the World between January 2013 and December 2015 Containing Shea Butter as an Ingredient (Innova Database)



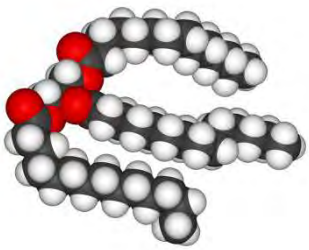
6.3 Absorption, Distribution, Metabolism, and Excretion

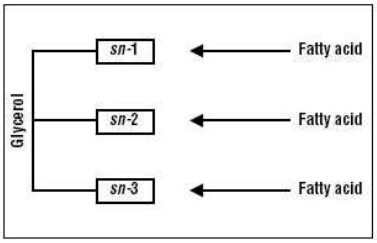
6.3.1 Absorption, Distribution, Metabolism, and Excretion of Triglycerides

Triglycerides, the main component of oils and fats, are organic molecules and are derived from both plant and animal origin. Triglycerides are mostly made up of carbon and hydrogen and are not very polar and thus insoluble in water. Dietary triglycerides (and more importantly their metabolite free fatty acids) serve several important biological functions. Triglycerides serve as a major energy source for the body as 1 g of fat supplies 9 kcals (37.8 kJ), which is more than twice the energy supplied by an equivalent amount of carbohydrates or proteins. However, the biological function of triglycerides goes far beyond energy supply. Triglycerides play major roles in the absorption of nutrients, appetite, cognitive function, immune function, cellular signaling pathways and are important dietary constituents for infants for optimal growth and development.

Triglycerides consist of a glycerol backbone bound to 3 fatty acids (Figure 6.3.1-1). A numbering system has been recommended to describe these forms. The prefix "*sn*" is placed before the stem name of the compound, when the stereochemistry is defined (*sn* = stereospecific numbering).

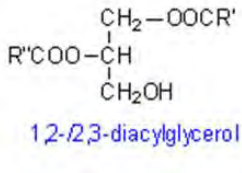
Figure 6.3.1-1 Typical Triglyceride Structure

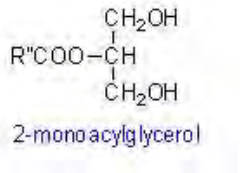




Diglycerides and monoglycerides exists and contain 2 or 1 bound fatty acids per glycerol, respectively, and exist in various isomeric forms, *i.e.* the fatty acid can exist as various positions on the glycerol as shown in Figure 6.3.1-2. Although monoglycerides and diglycerides exist in trace amounts in animals and plants, both are key intermediates in the biosynthesis of triglycerides and other lipids, and act are vital cellular messengers.

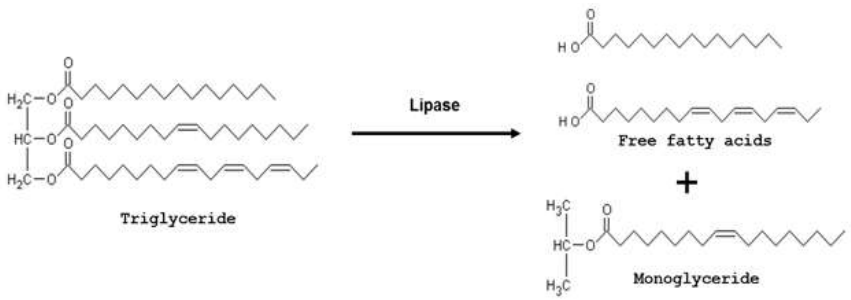
Figure 6.3.1-2 Typical Structure of a Di- and Mono-glyceride





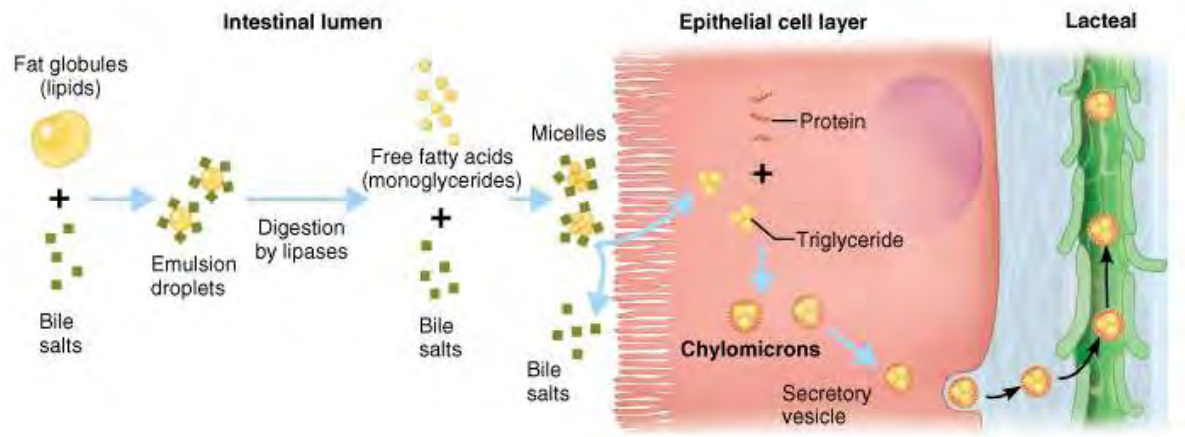
When triglycerides are digested, they are metabolized into free fatty acids and a monoglyceride by pancreatic lipases. Lipases also exist in saliva which can metabolize triglycerides. Lipases typically act on the *sn*-1 and *sn*-3 positions and yield a sn-2 monoglyceride as described in Figure 6.3.1-3.

Figure 6.3.1-3 Specific Gut Lipase Hydrolysis



Some fatty acids are more readily absorbed as a monoglyceride than as a free fatty acid. The chain length of the free fatty acids governs the subsequent uptake, which is either *via* the portal or lymphatic system. Small and medium chain fatty acids can be directly absorbed into the blood stream through the portal vein (portal system). Dietary fatty acids of short and medium chain-length are not usually esterified but are oxidized rapidly in tissues as a source of fuel to support biological functions. However, long chain fatty acids are absorbed into the intestinal wall, reassembled into triglycerides and secreted into the blood stream as chylomicrons *via* the lymphatic system. In view of the fatty acid composition of refined shea butter, its absorption follows this latter route represented in Figure 6.3.1-4.

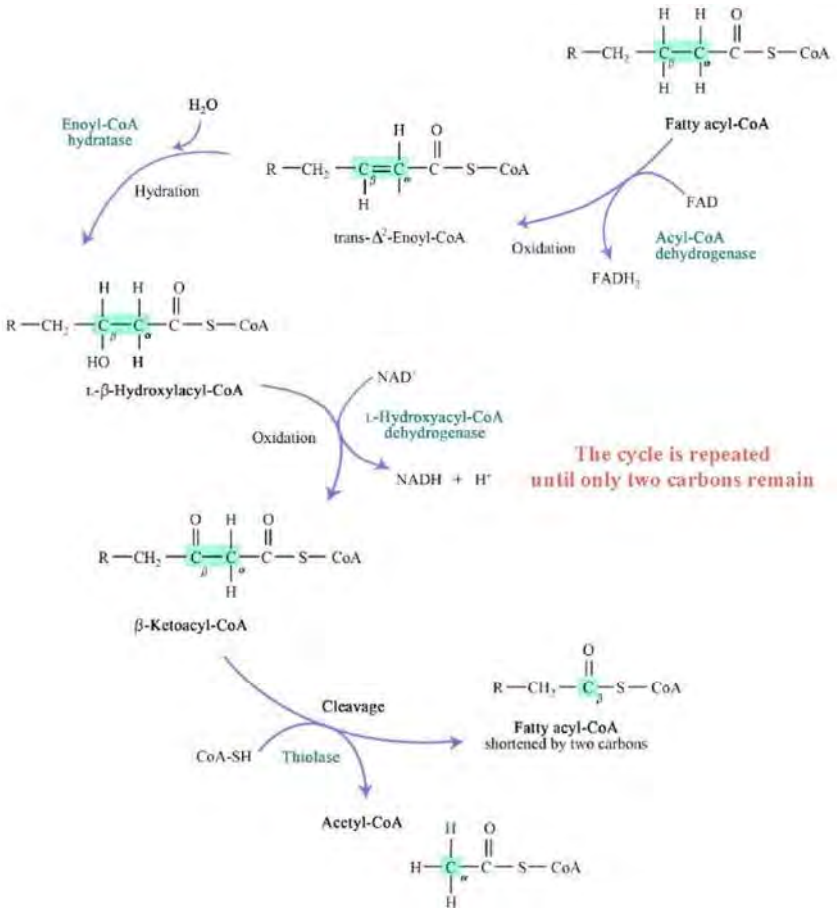
Figure 6.1.3-4 Digestion and Absorption of Lipids in Humans



Triglycerides are the primary storage form of long-chain fatty acids for energy and structural purposes, and free acids can be mobilized quickly when required for transport in an appropriate form to the heart, liver and other tissues where they can be oxidized. Whereas the brain typically relies of carbohydrates for fuel, the heart and skeletal muscle prefer fatty acids as a source of energy. Fatty acids can then be burned as fuel (*beta*-oxidation) or used for other biological processes throughout the body.

Beta-oxidation occurs in the mitochondria and/or in peroxisomes to generate acetyl-coenzyme A (acetyl-CoA). The process is the reverse of fatty acid synthesis: 2-carbon fragments are removed from the carboxyl end of the acid, as shown in Figure 6.1.3-5. This occurs after dehydrogenation, hydration, and oxidation to form a *beta*-keto acid. The acetyl-CoA then converts to ATP, CO_2, and H_2O using the citric acid cycle and releases energy of 106 ATP. Unsaturated fatty acids require additional enzymatic steps for degradation.

Figure 6.1.3-5 *Beta*-Oxidation of Fatty Acids



6.3.2 Animal Studies with Shea Butter

Thomasson (1956) examined the rate of absorption of shea butter administered orally by gavage to male Wistar rats at 400 mg per 100 cm^2 body surface; It was presumed that more consistent results would be achieved by expressing the amount of absorbed fat as a function of body surface. The levels of fat recovered and the distribution within the gastrointestinal tract at various time points are outlined in Tables 6.3.2-1 and 6.3.2-2.

Table 6.3.2-1 Recovery of fats from the gastrointestinal tract of rats following administration of 400 mg/100 cm^2 Shea Butter (adapted from Thomasson, 1956)

Type of Fat:	Percentage of fat recovered (% of dose)			Mean AT_{50}[a] (min)
	3 hours	8 hours	9.5 hours	
Shea butter	76	64	40	477 minutes

[a] AT_{50} is the number of minutes after which 50% of the fat administered had disappeared from the gastrointestinal tract.

Table 6.3.2-2 Distribution of fats within the gastrointestinal tract of rats following administration of 400 mg/100 cm^2 Shea Butter (adapted from Thomasson, 1956)

Type of Fat:	Percentage of fat recovered (assume 100% recovered in entire gastrointestinal tract)								
	3 hours			8 hours			9.5 hours		
	Stomach	Intestine		Stomach	Intestine		Stomach	Intestine	
		Small	Large		Small	Large		Small	Large
Shea butter	66	29	5	60	22	18	50	35	15

6.3.3 Absorption, Distribution, Metabolism, and Excretion of Unsaponifiable Material

6.3.3.1 Animal Study with Shea Butter Olein

In an oral absorption and excretion study, groups of Wistar male rats were fed shea butter olein in a semisynthetic diet (Earl *et al.*, 2002a). In a low-dose experiment, groups of 24 rats received control feed, feed containing 0.5% shea butter olein, or feed containing 5% shea butter olein for 1 week, with control feed administered to all rats the week prior and the week following the exposure week. In a high-dose experiment, 2 groups of 15 male and 15 female rats received either 10% or 20% shea butter olein in the feed for 3 weeks. Shea butter olein contained approximately 8% triterpene alcohols and < 1% of other shea butter sterols.

In the first experiment, feces were collected and pooled weekly for each treatment group throughout Weeks 2 and 3. In the second experiment, feces were collected and pooled for each treatment group in Week 3 only. The dried fecal matter of the rats was then analyzed with thin-layer and gas-liquid chromatography for fecal lipid, total sterol, differential sterol levels, and, specifically, triterpene alcohols. Excretion of triterpene alcohols increased with the consumption of shea butter olein. Apparent absorption was estimated from the disappearance of triterpene alcohols from the feces. The majority of the triterpene alcohols was excreted unchanged.

6.3.3.2 Human Study with Shea Butter Olein

The same study above (Earl *et al*., 2002a) also examined the oral absorption and excretion of shea butter olein in 4 male volunteers. On Day 3 of an 8-day period, the subjects consumed a single 25 g portion (approximately 0.4 g/kg) of shea butter olein in mayonnaise. No other vegetable fats were consumed during the course of the study. Shea butter olein contained approximately 8% triterpene alcohols and <1% of other shea butter sterols.

Feces were collected on Days 3 to 8 inclusively, freeze-dried and weighed. The dried fecal matter was analyzed in the manner described above. Excretion of triterpene alcohols increased with the consumption of shea butter olein, with a marked increase from baseline on Days 4 and 5 and a return to approximate baseline on Day 8. Absorption of triterpene alcohols was estimated to be 13 to 49%. The majority of the triterpene alcohols was excreted unchanged.

6.4 Animal Studies

6.4.1 Safety of Shea Butter and its Fractionated Derivatives

6.4.1.1 Acute Toxicity Studies

Results of an acute toxicity study on an extract from shea butter olein containing 50% unsaponifiable matter was reported by BSP Pharma A/S as part of the Premarket Notification for BSP-201 and SheaNature (U.S. FDA, 2004). Trial was carried out in Wistar rats following Organisation for Economic Co-operation and Development (OECD) Test Guideline 420 (OECD, 2001). Single gavage doses of 2,000 mg test substance/kg body weight were administered to male and female rats (5/sex); animals were observed for at least 1,3, and 6 hours after dosing and daily thereafter for 14 consecutive days. There were no deaths or other signs of toxicity and body weight gains were normal during the study period. Piloerection was observed in 3 animals 1 hour after treatment and in 5 animals 3 hours after treatment, though the authors suggested this may have been related to treatment and handling procedures. Erythema was observed in the intestine of 1 male and discoloration of the liver, spleen, and lungs was seen in 1 female during gross necropsy examination. The authors concluded that shea butter olein containing 50% unsaponifiable matter was not acutely toxic; the minimal lethal dose was above 2,000 mg of the test substance/kg body weight (U.S. FDA, 2004).

6.4.1.2 Sub-Acute Studies

The effect of feeding various concentration of shea oil on biochemical parameters such as lipid profile (total cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides), blood glucose, total serum protein and serum albumin levels were studied by Akinwale *et al.* (2012). Twenty-five adult rats where divided into 5 groups of 5 rats each. Group I Control was given growers marsh and water *ad libitum*. Groups II, III, IV, and V were given 250 mg, 500 mg, 1,000 mg, and 1,500 mg (wet weight) of shea oil, respectively, in addition to growers marsh and water *ad libitum*. The feeding and intubation lasted for 28 days after which the rats were euthanized and blood samples were collected. The blood samples were assayed for lipid profile, glucose, total serum protein and serum albumin content (Table 6.4.1.2-1).

Table 6.4.1.2-1 Results Showing Effect of Feeding Various Concentrations of Shea oil on some Biochemical Parameters

Parameter / Groups	I (Control)	II	III	IV	V
HDL (mmol/L)	1.5 ± 0.2	1.1 ± 0.2**	1.1 ±0.1**	1 ± 0.1 **	1 ± 0.0**
LDL (mmol/L)	3.4 ± 0.5	2.5 ± 0.5*	2.5 ± 0.5*	2.3 ± 0.1*	2.2 ± 0.1*
Total Chol (mmol/L)	5.5 ± 0.9	3.9 ± 0.5**	3.8 ± 0.3**	3.7 ± 0.1**	3.7 ± 0.05**
TG (mmol/L)	2.9 ± 0.1	2.4 ±0.5	2.4 ± 0.1	2.3 ± 0.1*	2.3 ± 0.05
Albumin (U/L)	35.3 ±1.5	30.7 ± 0.57*	30.7 ± 0.5*	31.7 ± 1.53*	30 ± 1.0*
Total Protein (U/L)	75.0 ± 1	70.3 ± 1.5	62.3± 11.3	71.3 ± 1.5	68.0 ± 3
Glucose (mmol/L)	3.1 ± 1	4.4 ± 1	4.6 ± 0.2	4.5 ± 0.2	4.0 ± 0.8

HDL = high-density lipoprotein; LDL = low-density lipoprotein; TG = triglyceride.
Mean ± standard deviation n=3
Values with different superscript horizontally are statistically different.

The results showed a very significant decrease in HDL, total cholesterol and LDL when the test groups were compared to the control ($P<0.05$), with insignificant decrease when comparison was made within the test groups ($P>0.05$). A significant decrease in the albumin level as compared to the control group ($P<0.05$), but an insignificant decrease when comparison was done within the test groups. An insignificant increase in blood glucose as compared to the control groups ($P>0.05$) was reported as well as and an insignificant decreased in total protein as compared to the control ($P>0.05$).

The effect of feeding shea butter-based diet on plasma, liver and kidney enzymes as well as the plasma lipid profile was studied by Israel *et al*., (2014). Twenty-one weaned male rats were divided into 3 groups: control, Test 1 and Test 2, each containing 7 rats. The control group was given feed containing soya bean oil as the lipid source *ad libitum.* In Test Groups 1 and 2, shea butter was incorporated in the diet at levels of 5% and 15% (w/w) respectively, replacing soya bean oil. The feeding lasted for 28 days after which the rats were sacrificed and the plasma as well as tissue samples from liver and kidney were collected. From the plasma, lipid profile; aspartate and alanine aminotransferases, alkaline phosphatase and total protein were assayed. From the tissue samples, aspartate and alanine aminotransferases, alkaline phosphatase and total protein were assayed. A significant decrease ($P<0.05$) was observed in the total cholesterol (TC), HDL, LDL, and triglyceride (TG) upon feeding with shea butter-based diet (Table 6.4.1.2-2). Feeding with shea butter did not produce undesirable effects in hepatic or renal tissues (Table 6.4.1.2-3).

Table 6.4.1.2-2 Plasma Lipid Profile of Rats Fed with Butter Based Diet

	Control	5% Shea butter	15% Shea butter
HDL(mmol/L)	0.90±0.03^{a}	0.70±0.10^{b}	0.55±0.07^{c}
TG (mmol/L)	1.20±0.00^{a}	0.70±0.20 ^{b}	0.55±0.07^{b}
LDL (mmol/L)	0.80±0.09^{a}	0.57±0.12 ^{b}	0.48±0.07^{c}
TC(mmol/L)	2.50±0.14^{a}	1.45±0.21^{b}	1.20±0.30^{b}
TC/HDL ratio	2.78±0.11^{a}	2.07±0.09^{b}	2.18±0.08^{b}
LDL/HDL ratio	0.89±0.02^{a}	0.81±0.05^{a}	0.87±0.03^{a}

HDL = high-density lipoprotein; LDL = low-density lipoprotein; TG = triglyceride; TC = total cholesterol.
Values are mean ± standard deviation.
Mean(s) with the same super script in each row are not significantly different at 5% level of significance ($P<0.05$).

Table 6.4.1.2-3 Plasma, Hepatic and Renal Enzyme Activities of Rats Fed with Butter Based Diet

	Parameter	Control	5% Shea butter	15% Shea butter
Plasma	ALT(U/L)	4.00±0.00^{b}	8.00±0.00^{a}	9.00±0.00^{a}
	AST (U/L)	44.00±4.24^{a}	33.50±3.54 ^{b}	33.00±5.90^{b}
	ALP (U/L)	427.88±16.89^{a}	153.10±5.00 ^{b}	235.50±3.54^{b}
	TP (g/100ml)	77.35±5.44^{a}	62.50±4.80^{b}	65.10±2.40^{b}
Liver	ALT(U/L)	116±5.66^{b}	138.5±3.53^{a}	118.0±2.82^{b}
	AST (U/L)	106.0±5.66^{a}	82.5±7.78^{b}	74.5±3.54^{b}
	ALP (U/L)	74.3±5.80^{a}	39.5±3.30^{b}	46.02±1.01^{b}
	TP (g/100ml)	32.9±2.70^{a}	32.9±0.00^{a}	13.55±2.70^{b}
Kidney	ALT(U/L)	4.00±0.00^{b}	6.70±.20^{a}	4.00±0.00^{b}
	AST (U/L)	100.50±2.12^{a}	96.00±0.00 ^{a}	33.00±5.90^{b}
	ALP (U/L)	2416.82±194.71^{a}	1471.69±13.77 ^{c}	1825.00±185.80^{b}

Table 6.4.1.2-3 Plasma, Hepatic and Renal Enzyme Activities of Rats Fed with Butter Based Diet

Parameter	Control	5% Shea butter	15% Shea butter
TP (g/100ml)	18.67±1.10[a]	15.50±0.30[b]	10.65±1.34[c]

ALP = alkaline phosphatase; ALT = alanine aminotransferase; AST = aspartate aminotransferase; TP = total protein.
Values are mean ± standard deviation.
Mean (s) with the same super script in each row are not significant different at 5% level of significance (P<0.05).

6.4.1.3 Subchronic Toxicity - Shea Olein

In a 13-week rat feeding study reported by Earl *et al*. (2002b) Wistar rats received a diet containing 20 wt % (10 to 15 g/kg/day) shea butter olein or hydrogenated shea butter olein. Groups were comprised of 15 male and 15 female rats. Equivalent groups of rats were fed either 20 wt % palm oil, soybean oil, or the hydrogenated equivalents. Given the reported 7.44% and 6.52% unsaponifiable material of the shea butter olein and the hydrogenated shea butter olein it is estimated the rats consumed approximately 0.7 to 1.1 g/kg body weight/day of unsaponifiable material. During the exposure period, body weight, food and water consumption, urine chemistry, and clinical pathology were assessed. Gross necropsy and microscopic examination of select tissues and organs were performed at study completion.

The shea butter olein diets produced biological effects similar to those of palm oil and soybean oil diets. Slightly reduced body weight gain was observed in rats fed either of the shea butter olein diets when compared to diets with palm oil and soybean oil. No significant differences in body weight gains were observed between rats fed hydrogenated shea butter olein *versus* non-hydrogenated shea butter olein. Slightly reduced cholesterol levels, increased aminotransferase levels, and lower triglyceride and alanine aminotransferase values were observed in rats fed nonhydrogenated diets, as were increased in liver weights and reduced liver-lipid values. These changes were not biologically significant and were considered related to the high fat content of the diets. Also considered biologically insignificant by the authors were raised alkaline phosphatase levels and increased food consumption in rats fed hydrogenated shea butter olein. The authors concluded that all diets were well tolerated in the rat and that none of the findings in this study were considered adverse.

The authors concluded that shea butter olein given at 20% of the diet (10 to 15 g/kg/day, equivalent to levels of up to 1.1 g/kg body weight/day of unsaponifiable matter) was well tolerated and appeared to have no adverse effect on the growing rat. The results of this study demonstrate that no unsafe level of shea butter olein and its associated unsaponifiable fraction could be identified in growing rats, a finding that supports the use of shea olein as a macronutrient food ingredient.

6.4.1.4 Developmental and Reproductive Toxicity (DART) Studies – Shea Olein

The reproductive toxicity of shea butter olein and hardened (hydrogenated) shea butter olein was investigated by Baldrick *et al*. (2001) in 2 dietary studies in rats during pre-mating, mating, pregnancy and weaning. The studies compared 7 or 15% hardened shea butter olein with 7 or 15 wt % of the unhardened material (approximately equivalent to 3.5 or 7.5 g/kg/day); additionally, toffee powder and cocoa butter (STUDY 1), and crude shea butter, unhardened palm oil and hardened palm oil (STUDY 2) were used for comparison. Toffee powder was selected for comparison in STUDY 1 since, at the time, it was a commercially available material comprised of 20% hardened shea butter olein. The authors noted that STUDY 1 and STUDY 2 were conducted years apart and different comparator oils were employed in each.

Though the STUDY 1 diets were not analyzed, analysis of STUDY 2 diets revealed the levels of unsaponifiable material in the shea butter, shea butter olein, and hydrogenated shea butter olein to be 5%, 6.4%, and 3.5%, respectively, as compared to 0.1% for both the palm oil and the hardened palm oil.

In STUDY 1, groups of Colworth-Wistar rats (n=40; 20 animals/sex) received diets containing 7% of shea butter olein, 7% of a hardened shea butter olein, 7 wt % of cocoa butter or 35 wt % of toffee powder for 20 consecutive weeks. During Week 12 of the 20-week feeding period, rats were mated, and following gestation, all dams were allowed to litter. At weaning, offspring were fed the parental diet for 7 to 10 days prior to sacrifice. In addition, 12 male and 12 female parental animals from each group were sacrificed following the 20-week treatment period (after weaning) for examination. Parental animals were assessed for general condition and health, body weights, food consumption, clinical pathology evaluation, gross necropsy examination and weighing of selected organs while assessment of pups included a clinical chemistry evaluation, as well as skeletal (using X-ray) and macroscopic examinations. In STUDY 2, groups of Colworth-Wistar rats (n=100; 50 animals/sex) received diets containing 15 wt % crude shea butter, shea butter olein or palm oil, or 15 wt % of a hydrogenated ('hardened') shea butter olein and palm oil, for 10 consecutive weeks.

Experimental diets were introduced 2 weeks prior to mating; dams were allowed to litter and at weaning, offspring were fed the parental diet. Endpoints observed included assessment of general health and measurement of litter and weaning parameters. In addition, at the end of the 10-week treatment period, liver, lymph glands, serum and red blood cells were examined for fatty acid composition, and livers and mesenteric lymph nodes were examined for evidence of lipogranulomas in F_0 animals.

For parental animals, results from STUDY 1 demonstrated that there were no differences in the general condition of animals fed the 4 diets. Actual test material intake ranged from 3 to 6 g/kg/day across the groups. Compared to rats fed the cocoa butter diet, a statistically significant reduction in mean body weight gain was observed in males receiving unhardened shea butter olein and in both sexes receiving toffee powder; a marginal but statistically significant increase in gain was noted in females fed the unhardened shea butter olein diet. In contrast, the hardened shea butter olein diet elicited no effect on body weight gains. The authors attributed the reported difference in body weight gain between groups to differences in the calorific value of the diets.

No differences in hematological parameters were noted among the groups after 20 weeks of treatment. Results of clinical chemistry analyses showed a trend for reduced mean cholesterol in both sexes fed the unhardened shea butter olein, toffee powder and hardened shea butter olein diets, compared to those receiving cocoa butter. Increased mean alkaline phosphatase (ALP) values were also observed in both sexes of the unhardened shea butter olein, hardened shea butter olein and toffee powder groups. The authors reported that both findings resulted from the feeding of high fat diets. With respect to organ weights, a higher absolute mean heart weight was reported for females fed the unhardened and hardened shea butter olein diets, compared to those receiving the cocoa butter diet. In offspring, none of the diets had an effect on the number of litters born or weaned. Litter size, number of pups at birth and weaning, survival and body weight at weaning were similar among groups. Results of clinical chemistry analyses showed an increase in mean ALP values for both sexes in the hardened shea butter olein and toffee powder groups; a smaller increase was also observed in the unhardened shea butter olein group. Macroscopic and x-ray examination of animals showed no findings related to the test materials. For parental animals in STUDY 2, no unusual health problems were noted. Results from fatty acid analyses of the liver, lymph node and blood serum, as well as the membrane phosphatidyl choline (PC) analyses showed no unexpected results for the diets administered. Likewise, histopathological examination of the liver and mesenteric lymph nodes

showed no evidence of lipogranulomas in any of the groups. For offspring, none of the diets had any effect on the number of litters born. No significant differences were noted in pup body weights at birth and weaning among groups.

Based on the results of STUDY 1 and STUDY 2, the authors concluded that there was no evidence of reproductive toxicity for unhardened or hardened shea butter olein in the rat at levels equating to approximately 7.5 g/kg/day. The results of this study demonstrate that no unsafe level of shea olein and its associated unsaponifiable fraction was observed in reproductive and developmental toxicity studies, a conclusion that is supportive of the use of shea olein as a macronutrient ingredient.

6.4.2 Carcinogenicity – Unrefined Shea Butter and Shea Olein

The carcinogenicity of unrefined shea butter (referred to as "crude sheanut oil" within the study) and shea butter olein was evaluated in a dietary study in Wistar rats (Carthew *et al*., 2001). Groups of 50 male and 50 female rats received diets containing 15% (*ca*. 7.5 g/kg/day) unrefined shea butter, 15% shea butter olein, or 15% palm oil. All test articles were prepared by accepted commercial processing methods. The free fatty acid content of the materials was 11.3%, 0.2%, and 0.2% respectively, and the unsaponifiable matter was measured as 5.0%, 6.4% and 0.1%. The rats used in the study were offspring of the animals used in the reproduction study described above (STUDY 2) and the test diets were administered at weaning (21 days of age) for a duration of 104 weeks. The following parameters were assessed: mortality, clinical signs of toxicity, body weight, food intake, clinical pathology, organ weights and macroscopic and histopathological changes plus tumor type and incidence evaluation.

Some isolated differences among the oils were observed. Final mortality values for both sexes were approximately 28 to 30% for the shea butter olein and unrefined shea butter groups, and 40% for the palm oil group. No clinical signs were found in any animals that could be associated with a particular diet. Likewise, terminal body weights and mean food intake values were not significantly different among groups. Analysis of hematological parameters revealed a statistically significant reduction in the differential monocyte count, for male rats fed shea butter olein ($0.5x10^9$/liter *vs*. $0.8x10^9$/liter for palm oil), and females fed unrefined shea butter and shea butter olein ($0.4x10^9$ and $0.3x10^9$/liter, respectively, *vs*. $0.6x10^9$/liter for palm oil); however, these changes were slight in magnitude and not considered to be of immunological significance. With respect to organ weights, mean absolute heart weights for both sexes fed shea butter and shea butter olein diets were significantly reduced compared to the palm oil diet. Relative heart weights were also significantly reduced, although only for males fed shea butter and shea butter olein. Mean relative liver weights were higher for female rats fed the shea butter diet; however, the authors attributed this trend to the feeding of a high fat diet. There were no significant histopathological findings in rats of any group.

Although pulmonary lipidosis (as indicated by focal accumulation of lipid-laden alveolar macrophages associated with thickening and increased cellularity of adjacent alveolar walls) was observed in both sexes of all diet groups, the highest incidence and severity was noted in the shea butter olein and unrefined shea butter diet groups; thus, variations in pulmonary lipidosis were reported to be diet-related. However, the authors reported that the incidences of pulmonary lipidosis similar to incidences reported previously in life-time studies of Colworth-Wistar rats administered 7.5% sunflower seed oil in the diet; for unknown reasons, palm oil appears to behave differently than other oils in this regard.

The tumor profile was as expected for Colworth-Wistar rats in lifetime studies with high fat diets. An overall summary of tumor findings showed a similar pattern between diets (palm oil/shea butter/shea butter olein) of rats with neoplasms, multiple neoplasms, malignant neoplasms and metastatic neoplasms plus total number of neoplasms.

Tumor type analysis showed that the incidence of liver, pancreas and skin tumor varied with different diets. A statistically significant increase was found in the incidence of pancreas exocrine adenomas and skin keratoacanthomas for males fed the SO diet and hepatomas for females fed SNO and SO diets. The incidence of liver hepatomas in the SNO and SO treatment groups was reported by the authors as being consistent with effects of high fat diets in Colworth-Wistar rats (*i.e.*, similar incidences of liver hepatoma were reported in 5 separate in-house studies with control diet groups fed 15% fat containing diets). The pancreatic exocrine adenomas and keratoacanthomas were also found to be within ranges for these tumors in historical data on the control Wistar rats (Poteracki and Walsh, 1998). These incidences thus were not considered to be significantly different from the historical range of these tumor types in Colworth-Wistar rats.

Findings from this study demonstrate that commercial shea butter preparations containing high levels of unsaponifiable matter (*i.e.*, unrefined shea butter and shea butter olein) were well tolerated in Colworth Wistar rats following long-term (104 weeks) consumption at levels up to 15% in the diet and no evidence of tumorigenic potential was reported by the authors. The absence of any undesirable effects of unrefined shea butter and shea butter olein at levels of up to 15% in the diet provide strong supporting evidence that macronutrient food uses of shea butter and its fractionated derivative products are safe.

6.5 Animal Studies of Major Fatty Acids

Oleic, palmitic and stearic acids were tested for acute oral toxicity in rats. Administration of doses up to 21.5 ml/kg (equivalent to 19.1 g/kg) of oleic acid and up to 10 g/kg of palmitic acid by gavage to albino rats resulted in no deaths and no significant gross lesions at necropsy. Dose of 25% (w/v) stearic acid in corn oil produced the deaths of 1 rat. At necropsy of the rat, congested lungs and kidneys and advanced autolytic changes were observed. No significant gross lesions were found at necropsy of 2 rats of the 0.464 and 4.64 g/kg triple-pressed stearic acid (40 to 47% stearic acid/40 to 60% palmitic acid fat) dose group. Transient signs of toxicity were observed in rats at doses of 10 g/kg 25% stearic acid in corn oil and the 4.64 and 10.0 g/kg triple-pressed stearic acid. Signs of toxicity included slight depression, depressed righting and placement reflexes, oily and unkempt fur, mucoid diarrhea, excessive salivation, and sero-sanguineous discharge from the muzzle and eyes (CIR, 1987). A summary of the chemical safety information of palmitic and stearic acid can be found in the ECHA website (ECHA, 2018, 2019). Additional safety studies were carried out in the 1970s to 1980s mainly with cosmetic formulations. A safety assessment of oleic, lauric, stearic, myristic and palmitic acids published by the Cosmetics Ingredient Review Expert Panel was published in 1987 (CIR, 1987). The results of acute, subchronic and chronic studies in animals administered oleic, and stearic acids by gavage or *via* the diet were summarized by the Expert Panel. AAK notes that there were no findings in these studies to suggest that intake of the major fatty acids from shea butter (stearic acid and oleic acid) would be unsafe.

6.5.1 Chronic and Sub-chronic Studies of Oleic and Stearic Acid

Feeding of 5% oleic acid or 50% stearic acid diets to chicks for 4 weeks had no adverse effects. Rats fed diets containing 4.6 g/kg/day palmitic acid for 6 weeks developed hyperlipemia. A diet containing 50% stearic acid fed to rats for 8 weeks resulted in microscopic "foreign body-type reaction" in adipose tissue. Feeding 15% oleic acid diets to rats for 10 to 16 weeks had no adverse effects on growth or general health. Of 4 female weanling rats fed the diet for 16 weeks, all 4 were able to become pregnant; however, 2 died at parturition, a litter was eaten at birth, and the remaining litter died within 3 days of birth. Mating of 7 adult female rats fed the diet for 16 weeks resulted in production of 52 young, 44 of which survived 1 week and 11 of which survived 3 weeks. Mammary development was retarded, and a few rats had ovarian cysts. No lesions were found in other organs (CIR, 1987).

6.6 Human Studies

Data accumulated during the past 50 years indicate that stearic acid (C18:0) is unique among the SAFA since unlike other predominant long-chain SAFA (palmitic (C16:0), myristic (C14:0), and lauric (C12:0) acids) which increase blood cholesterol levels, when carbohydrates are used as reference, stearic acid has been shown to have a neutral effect on blood total and low-density lipoprotein (LDL) cholesterol levels (Kris-Etherton *et al.*, 2005; Mensink, 2016). The neutral effect of stearic acid on blood total and LDL cholesterol levels implies that this long-chain SAFA may not increase the risk for cardiovascular disease. For this reason, it is a suitable alternative to other dietary sources of saturated fat that are rich in (C12:0), myristic (C14:0) & palmitic (C16:0) such as palm oil, coconut oil, and hydrogenated vegetable oils.

6.6.1 Studies in Humans Administered Shea Butter or Stearic Acid

Manual searches of the Pubmed database identified 15 clinical studies and several comprehensive reviews evaluating the nutritional impact of stearic acid intake on various measures of cardiovascular health (Dougherty *et al.*, 1995; Kelly *et al.*, 2001; Sanders *et al.*, 2001; Judd *et al.*, 2002; Tholstrup *et al.*, 2003; Baer *et al.*, 2004; Lefevre *et al.*, 2004; Bysted *et al.*, 2005; Kris-Etherton *et al.*, 2005; Mensink, 2005; Tholstrup, 2005; Thijssen and Mensink, 2005; Thijssen *et al.*, 2005a,b; Berry *et al.*, 2007; Maljaars *et al.*, 2009; Robinson *et al.*, 2009; Tholstrup *et al.*, 2009; Karupaiah *et al.*, 2011; Gebauer *et al*., 2014). Although interpretation of the study findings across all studies is complicated by the differences in study designs and lipid compositions of the control and test diets that are evaluated, in general, the totality of data tend to support a conclusion that substitution of significant proportions of dietary fat with stearic acid does not adversely impact biochemical measures of cardiovascular health (See Table 6.6.2-1). Some studies reported findings to suggest that stearic acid intake may adversely affect hemostatic risk factors such as factor VIIc coagulant activity (Sanders et al., 2001) and plasma fibrinogen (Baer *et al*., 2004) A review of the literature on the effect of stearic acid on hemostatic risk factors in humans was conducted by Tholstrup, (2005). The authors evaluated findings from three randomized crossover studies evaluating the hemostatic effects of stearic acid-rich test diets in healthy young men. The authors concluded that dietary stearic acid was not more thrombogenic compared with other long-chain fatty acids. The authors also stated that the slightly increased effect on fasting plasma fibrinogen may be biologically insignificant.

Table 6.6.2-1 Human Studies of Stearic Acid

Reference	Study Population	Test Article and Dose	Duration of Stearic Acid Intake	Relevant Observations
Tholstrup *et al.* (1994)	Healthy males (n=15), Mean age= 25	(1) Shea butter (42% stearic acid) (2) Palm-kernel oil (3) Palm-kernel + sunflower oil	3 weeks	Diet 1 resulted in significant reduction in total, LDL, and HDL cholesterol, along with reduced FVIIc compared to diet 2
Dougherty *et al.* (1995)	Healthy males (n=10) ages 32-56	(1) High stearate: Shea butter (25%) (2) Low stearate: Palm oil and butter (6%)	40 days	Diet 1 significantly lowered total, LDL, and HDL cholesterol
Kelly *et al.* (2001)	Healthy males (n=13), Mean age = 35	(1) Stearic acid-enriched spreading margarine (29%), baking margarine (34%), and biscuits (31%) (2) Palmitic acid-enriched spreading margarine (31%), baking margarine (42%), and biscuits (42%)	4 weeks	Plate volume, FVIIc, and plasma lipid concentrations decreased significantly with consumption of stearic acid-rich diet (19 g/day)
Sanders *et al.* (2001)	Healthy males (n=17) and females (n=18) ages 40-60	(1) Structured TAG containing stearic acid (16%) (2) Cocoa butter (11% stearic acid) (3) High-oleic acid sunflower oil	3 weeks	Mean increase in plasma TAG 3 h after eating was lowest for meal 1; Plasma FVIIc significantly increased after meals 2 and 3, but not 1
Judd *et al.* (2002)	Healthy males (n=50) ages 25-60	(1) Stearic acid (44%) (2) Oleic acid (3) TFA (4) LMP (5) TFA+ stearic acid (23%) (6) Carbohydrates (control)	5 weeks	TAG concentrations were highest after consumption of diet 1; Diet 1 did not affect LDL cholesterol but lowered HDL cholesterol; Plasma HDL was lowest following diets 1 and 3
Tholstrup *et al.* (2003)	Healthy males (n=10) ages 21-28	Cakes, rolls, juice, and marmalade enriched in 1 of 2 test fats (1.2g/kg bodyweight): (1) Myristic acid (2) Shea butter (42% stearic acid)	24 hours	Consumption of diet 1 resulted in higher postprandial HDL TAG; No differences seen for other lipoproteins
Baer *et al.* (2004)	Healthy males (n=50) ages 25-60	(1) Stearic acid (38%) (2) Oleic acid (3) TFA (4) LMP (5) TFA+ stearic acid (24%) (6) Carbohydrates (control)	5 weeks	Fibrinogen concentrations were significantly higher after consumption of stearic acid-rich diet compared to control diet
Bysted *et al.* (2005)	Healthy males (n=16), Mean age= 23	5 Interesterified fats containing 43-47% of the target fatty acid: (1) Palmitic acid (2) Stearic acid (3) *Trans*-18:1 isomers (4) Oleic acid (5) Linoleic acid	8 hours	Fatty acids in test fats were extensively incorporated into chylomicron TAG, but reflected less in VLDL tag; no preferential clearing of chylomicron TAG was observed
Thijssen and Mensink (2005)	Healthy males (n=18) and females (n=27) ages 18-65	(1) Stearic acid (39%) (2) Oleic acid (3) Linoleic acid	5 weeks	No significant changes in LDL-cholesterol, HDL-cholesterol, or size of lipoproteins between the 3 diets
Thijssen *et al.* (2005)	Males (n=18) and females (n=27) with	(1) Stearic acid (8%) (2) Oleic acid	5 weeks	Mean platelet volume was lowest following diet 1;

Table 6.6.2-1 Human Studies of Stearic Acid

Reference	Study Population	Test Article and Dose	Duration of Stearic Acid Intake	Relevant Observations
	slightly elevated plasma cholesterol, ages 28-66	(3) Linoleic acid		FVIIa, fibrinogen, and plasminogen activator inhibitor-1 did not differ between the 3 diets
Maljaars *et al.* (2009)	Healthy males (n=2) and females (n=13), Mean age= 24	6 g fat emulsion of shea oil (59% stearic acid), canola oil (2% stearic acid), and safflower oil (2% stearic acid)	4 days	Compared to the control (saline), the stearic acid-rich emulsion did not significantly increase satiety
Robinson *et al.* (2009)	Obese males (n=11) with BMI >30 and non-obese males (n=10) with BMI <30	Stearic acid-rich spread which was: (1) Non-interesterified (2) Chemically interesterified (3) Enzymatically interesterified; And a control meal (bread and water)	6 hours	Interesterification did not affect post-prandial glucose, insulin, free fatty acids, or cholesterol Postprandial TAG concentrations in non-obese subjects did not change based on diet; Obese subjects had an 85% increase in TAGs with chemically-interesterified stearic acid compared to non-interesterified
Karupaiah *et al.* (2011)	Healthy males (n=10) and females (n=10) ages 22-38	(1) Stearic acid (33%) (2) Lauric + myristic acids (3) Palm olein	7 days	Plasma TAG was significantly increased after consuming a stearic acid-rich diet; At 2 hours, plasma HDL-cholesterol increased significantly in diets 2 and 3 compared to diet 1 Lipemic response after stearic acid consumption did not resume postabsorptive levels by 8 hours
Gebauer *et al.* (2014)	Healthy males (n=50) ages 25-60	Test articles and doses are identical to those in study by Baer *et al.*	5 weeks	Compared to control diet, the stearic acid-rich diet did not induce significant changes in FVIIc, plasminogen activator inhibitor-1, or plasmin alpha-2-antipalsmiin

LDL= low-density lipoprotein; HDL= high-density lipoprotein; TAG= triacylglycerol; TFA= *trans* fatty acid; LMP= sum of lauric, myristic, and palmitic acids; FVIIa= activated clotting factor VII; FVIIc= factor VII coagulant activity; BMI= body mass index (kg/m^2)

6.7 Investigations on the Unsaponifiable Fractions from Shea Butter

6.7.1 Genotoxicity

Results of an Ames bacterial mutagenicity assay and an *in vivo* mouse micronucleus test on an extract from shea butter olein containing 50% unsaponifiable matter was reported by BSP Pharma A/S as part of the Premarket Notification for BSP-201 and SheaNature (U.S. FDA, 2004). Both studies were conducted according to the methods of the OECD. In the Ames assay, the test substance in dimethylsulfoxide (DMSO) was not toxic to *Salmonella* Typhimurium strains TA102, TA100, TA98, TA1537, or TA1535 at dose levels of 50, 160, 500, 1,600, and 5,000 µg/plate (equivalent to 25, 80, 250, 800, and 2,500 µg unsaponifiable matter/plate). In addition, no biologically or statistically significant increases in the number of revertant colonies were observed in any tester train after treatment with the test substance at any dose level, either in the presence or absence of rat liver metabolic fraction (S-9), as compared with negative controls (U.S. FDA, 2004).

In the *in vivo* mouse micronucleus assay, male mice were treated with a single dose 2,000 mg/kg body weight dose of BSP-201 (equivalent to 1,000 mg of unsaponifiable material/kg bw) or controls by oral gavage. Five mice from each group were sacrificed 24 hours after dosing while 5 additional mice from the shea butter extract and the negative control groups were sacrificed 48 hours after dosing. No adverse reactions to treatment with the test substance were observed, nor were any biologically or statistically significant increases in the frequency of micronucleated polychromatic erythrocytes in mice treated with the shea butter extract as compared with negative controls. It was concluded that the test substance was not genotoxic under the conditions of this study (U.S. FDA, 2004).

6.7.2 Other Studies

The effect of the unsaponifiable fractions of shea fat was investigated in 2010 by Akihisa *et al*. (Akihisa *et al*., 2010b). This study evaluated 8 triterpene esters for their inhibitory effects on Epstein-Barr virus early antigen (EBV-EA) in Raji cells as a primary screening test for inhibitors of tumor promoters. All the compounds showed moderate inhibitory effects. Furthermore, lupeol cinnamate exhibited inhibitory effect on skin tumor promotion in an *in vivo* 2-stage carcinogenesis test using 7,12-dimethylbenz [*a*] anthracene (DMBA) as an initiator and 12-*O*-tetradecanoylphorbol-13-acetate (TPA) as a promoter.

The inhibitory effects on EBV-EA activation induced by TPA were examined as a preliminary evaluation of the potential antitumor-promoting activities for 4 triterpene acetates and 4 triterpene cinnamates. All of the compounds tested showed significant inhibitory effects while preserving the high viability of Raji cells. The IC_{50} values were reported to be equivalent to or more potent than retinoic acid. Among the 8 compounds tested, both acetates and cinnamates of butyrospermol and lupeol showed potent inhibitory effects.

Subsequently, the inhibitory effects of lupeol cinnamate were determined in a 2-stage carcinogenesis test on mouse skin using DMBA as an initiator and TPA as a promoter. The incidence of papillomas in Group I (untreated) was 100% in mice after 11 weeks of promotion. Further, more than 4 and 8 papillomas were formed per mouse at 11 and 20 weeks of promotion, respectively. In contrast, the formation of papillomas in mouse skin was delayed and the mean number of papillomas per mouse was reduced by treatment with lupeol cinnamate. Thus, in Group II (treated with lupeol cinnamate), the percentage ratios of papilloma-bearing mice were only 33% at 11 weeks, and 93% at 20 weeks, and the mean papillomas per mouse were 1.9 at 11 weeks, and 4.1 at 20 weeks.

6.7.3 Animal Studies

Studies of gastrointestinal effects of SheaFlex70™ compared with ibuprofen using a rat-based model showed that SheaFlex70™ produced no gastrointestinal lesions compared with the ibuprofen-treated group that exhibited a statistically significant number of gastric and intestinal lesions (U.S. FDA, 2004). SheaFlex70™ is a patented (EP 1 200 106) concentrate containing approximately 75% triterpenes derived from shea butter. The most abundant triterpenes are butyrospermol, lupeol, and the α- and β-amyrine plus their dihydro-derivatives.

Twelve Sprague-Dawley rats were administered 2,000 mg/kg doses of SBE dissolved in arachid oil by oral gavage daily for 4 days. Other rats were doses with the control vehicle or with 200 mg/kg of ibuprofen, which served as a positive control. Following an overnight fast, the rats were received intravenous injections of 1 ml of % Evans blue saline 30 minutes prior to sacrifice. The stomach and small intestines were scored for gastrointestinal lesions. No adverse clinical signs were recorded, and no adverse effects on

body weight were seen with the exception of 1 rat treated with ibuprofen. No gastrointestinal lesions were observed in control rats or in rats treated with the test material. In contrast, a significant number of lesions were observed in rats treated with ibuprofen as compared to those in the control group ($p = 0.0018$ for gastric lesions, $p<0.0001$ for intestinal lesions). The majority of these small intestinal lesions were seen in the aboral part of the jejunum and in the ileum. SBE, at oral doses of 2,000 mg/kg/day administered for 4 days, was found to have no ulcerogenic effect in the rat.

From the results of the *in vivo* anti-inflammatory tests, *in vitro* EBV-EA induction test, and *in vivo* 2-stage carcinogenesis, it can be concluded that the triterpene cinnamates and triterpene acetates isolated from shea fat do not have undesirable biological effects on tumor promotion. Shea-derived terpines (Shea-flex70) also did not produce adverse gastrointestinal effects in a rat model following 4 days of consumption at a dose of 2,000 mg/kg body weight per day.

6.7.4 Humans

6.7.4.1 Blood Clinical Chemistry & Hematology

In the study reported by Sierksma *et al.* (1999), the effects of a margarine enriched with shea butter triterpene alcohols taken daily for 3 weeks were compared with those of a margarine fortified with soybean sterols and with those of a non-fortified table spread. The mean daily shea butter triterpene alcohols intake was 3.2 g. Thirty-nine men and thirty-seven women completed the study. The spread enriched with shea butter triterpene alcohols did not lower plasma total, LDL- and HDL-cholesterol levels. None of the spreads induced changes in blood clinical chemistry or hematology.

Visser *et al.* (2000) reported no adverse effects no adverse effects and no effect of lipoprotein concentration in healthy normocholesterolemic subjects (28 men and 32 women) treated with 2.6 g/day of triterpene alcohol intake from shea butter margarine for 3 weeks.

Although the data with regards to its effect on serum lipoprotein concentrations were inconclusive, both studies lend support to the safety of shea butter unsaponifiables in humans.

6.7.4.2 Anti-inflammatory Effects

A single-site, 15-week randomized, double-blind, parallel, placebo-controlled study (Cheras *et al.*, 2010) examined a range of biomarkers in 89 patients with osteoarthritis of the knees and/or hips to determine potential modes of action of SheaFlex70™. SheaFlex70™ is a patented shea butter unsaponifiable matter concentrate containing approximately 75% triterpenes. The most abundant triterpenes are butyrospermol, lupeol, and the α- and β-amyrine plus their dihydro-derivatives (Weidner, 2003; http://www.flexnowsheaflex75.com/english/index.php).

After a minimum washout period of 3 weeks, participants were randomized to either placebo or SheaFlex70™ once daily for 15 weeks. Blood and urine samples for safety and biomarker assays were taken at baseline, Week 1, Week 10, and Week 15, the conclusion of the study. Treatment comprised either 100% SheaFlex70™ or placebo which comprised 100% canola oil. Daily dosage was three 750 mg softgel capsules (2,250 mg/day) taken in the morning.

In the group of participants with levels of osteoarthritis biomarkers in the upper quartile at baseline, there were significant decreases in inflammation and cartilage breakdown and trend level decreases in bone

remodeling in the SheaFlex70™ group *versus* placebo between commencement and completion of the study. Inflammation marker TNF-alpha fell 23.9% *vs.* 6% (treatment *vs.* placebo), p = 0.041. Cartilage degradation marker CTX-II fell 28.7% *vs.* an increase of 17.6% (treatment *vs.* placebo), p = 0.018. This marker also showed significant falls across the entire study group, 10.6% *vs*. an increase of 11.6%, (treatment *vs.* placebo), p = 0.016. Osteocalcin levels fell 9.2%, p = 0.014 (treatment) *vs*. 1.2%, ns (placebo), p = 0.096 (treatment *vs*. placebo). These findings, coupled with the finding of decreased pain in the treatment group, point toward a group of potentially beneficial pharmacological effects on the part of SheaFlex70™ in key areas associated with arthritis pathophysiology. No adverse effects of the treatment were reported.

6.7.4.3 Muscle Function

In the non-randomized control intervention study published by Chen *et al.* (2013) on the effects of SheaFlex75™ on quadricep strength, impaired in osteoarthritis patients, 39 patients were given 6 pills/day for 16 weeks of SheaFlex75™, equivalent to 2,160 mg of shea butter triterpene concentrate (75%). The morphological changes of muscles around the knees and the ability to control muscles in different tasks were examined at baseline, after 8 weeks and after 16 weeks.

Improved muscle function was observed, including greater control and an increase in-muscle strength to achieve a functional goal; nevertheless, the subjective feeling of improvement in the activities of daily living was not significant. No adverse effects of the treatment were reported.

6.8 Allergenicity

Shea kernels are obtained from the shea tree that is indigenous to many parts of Africa. The shea seed is the kernel of the fruit of the shea tree. The fruit portion is typically removed to retrieve the hard-shelled kernel which is pressed to obtain the shea butter. The shea tree, *Vitellaria paradoxa*, is a member of the *Sapotaceae* family. According to Judd *et al*., 1999 in Plant Systematics A Phylogenetic Approach and Zomlefer *et al*. 1994 in Flowering Plant Families all members of the *Sapotaceae* family are botanically berries. In the case of *Vitellaria* the fruit is a single seeded berry. Nuts are whole fruits, derived from a single ovary. Weir *et al.*, Botany, An Introduction to Plant Biology, defines a typical nut as "*a one-seeded, indehiscent dry fruit with a hard or stony pericarp, the shell*". A berry, according to the same source is "*a fleshy type of fruit derived from a compound ovary*". Usually, but not always many seeds are embedded in a flesh. Weir goes on to cite the date as an example of a 1-seeded berry. Another 1-seeded berry is the avocado (Storey, 1973). The structure of avocado serves to illustrate the structure and use of shea. The pulp on the outside of the shea seed is similar to the part of the avocado & used as a food source. In case of shea seed, it is used to extract shea butter.

When the U.S. Congress passed the Food Allergen Labeling & Consumer Protection Act in 2004, they appropriately designated tree nuts as among the most commonly allergenic foods in the U.S. However, Congress failed to provide a list of tree nuts when they passed this law. Later in October 2006, the FDA provided a list of tree nuts in an attempt to clarify the uncertainty left by Congress. That list included shea nuts and consequently the FDA requires listing of products derived from Shea as allergenic. Authors of various publications about "Shea Nut Oil" discuss the shea fruit, then add the "shea nut" as the part of the fruit which is used to extract shea butter. This is a misuse of the word "nut" since botanically the nut is the entire fruit, not part of the whole thing.

In Europe, shea kernels are not classified as nuts and consequently are not listed among the nuts that are subject to mandatory allergen labelling. A search of the worldwide clinical literature provided no evidence

to indicate that any allergic reactions have ever been reported to shea butter. Allergic reactions to shea kernels have not been described either.

Furthermore, recent research (Chawla *et al.*, 2011) indicates that shea butter does not contain any detectable protein residues and does not contain detectable residues of proteins from peanut or various known allergenic tree nuts (walnut, almond, pecan, hazelnut). Since allergens are proteins, this research indicates the absence of detectable allergens in shea butter.

In addition, since the material is refined, there is no allergenic risk to consumers including individuals with pre-existing peanut or tree nut allergies.

6.9 Conclusion

Based on the above data and information presented herein, AAK has concluded that the intended uses of refined shea butter in conventional food and beverage products, as described in Section 1.3, is GRAS based on scientific procedures. Shea butter manufactured by AAK therefore may be marketed and sold for its intended purpose in the U.S. without promulgation of a food additive regulation under Title 21, Section 170.3 of the Code of Federal Regulations.

Part 7. List of Supporting Data and Information

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General Information:

Sample No.:	160180800
Sample:	Refined veg. oils and fat 1653483-10 Lipex 102
Date of receipt:	19.02.2016
Testing period (begin / end):	19.02.2016 / 29.02.2016
Quantity:	ca 500 g
Packaging	Plastic bottle

Test Results:

Parameter	Method	Lab	Unit	Result	Limit of quantification	Requirements
Minerals/metals:						
Lead	DIN EN 15763 mod., ICP/MS	HH	mg/kg	< 0,01	0,01	
Cadmium	DIN EN 15763 mod., ICP/MS	HH	mg/kg	< 0,005	0,005	
Mercury	DIN EN 15763 mod., ICP/MS	HH	mg/kg	< 0,01	0,01	
Arsenic	DIN EN 15763 mod., ICP/MS	HH	mg/kg	< 0,02	0,02	
Iron	DIN EN 15763 mod., ICP/MS	HH	mg/kg	< 0,20	0,20	
Copper	DIN EN 15763 mod., ICP/MS	HH	mg/kg	< 0,05	0,05	
Nickel	DIN EN 15763 mod., ICP/MS	HH	mg/kg	< 0,05	0,05	
Tin	DIN EN 15763 mod., ICP/MS	HH	mg/kg	< 0,05	0,05	
Chromium	DIN EN 15763 mod., ICP/MS	HH	mg/kg	< 0,04	0,04	

Sample 160180800	Refined veg. oils and fat; 16534ϕ3-10; Lipex 102					
Parameter	Method	Lab	Unit	Result	Limit of quantification	Requirements
PAH						
Benzo(a)anthracene	SOP M 2920, GC/MS	HH	µg/kg	< 0,2	0,2	
Benzo(c)fluorene	SOP M 2920, GC/MS	HH	µg/kg	< 0,5	0,5	
Chrysene	SOP M 2920, GC/MS	HH	µg/kg	< 0,2	0,2	
Cyclopenta(c,d)pyrene	SOP M 2920, GC/MS	HH	µg/kg	< 0,2	0,2	
5-Methylchrysene	SOP M 2920, GC/MS	HH	µg/kg	< 0,2	0,2	
Sum light PAH	calculated	HH	µg/kg	< 0,2	0,2	
Benzo(b)fluoranthene	SOP M 2920, GC/MS	HH	µg/kg	< 0,2	0,2	
Benzo(k)fluoranthene	SOP M 2920, GC/MS	HH	µg/kg	< 0,2	0,2	
Benzo(j)fluoranthene	SOP M 2920, GC/MS	HH	µg/kg	< 0,2	0,2	
Benzo(a)pyrene	SOP M 2920, GC/MS	HH	µg/kg	< 0,2	0,2	
Indeno(1,2,3-cd)pyrene	SOP M 2920, GC/MS	HH	µg/kg	< 0,2	0,2	
Dibenzo(ah)anthracene	SOP M 2920, GC/MS	HH	µg/kg	< 0,2	0,2	
Benzo(ghi)perylene	SOP M 2920, GC/MS	HH	µg/kg	< 0,2	0,2	
Dibenzo(a,l)pyrene	SOP M 2920, GC/MS	HH	µg/kg	< 0,5	0,5	
Dibenzo(a,e)pyrene	SOP M 2920, GC/MS	HH	µg/kg	< 0,5	0,5	
Dibenzo(a,i)pyrene	SOP M 2920, GC/MS	HH	µg/kg	< 0,5	0,5	
Dibenzo(a,h)pyrene	SOP M 2920, GC/MS	HH	µg/kg	< 0,5	0,5	
Sum heavy PAH	calculated	HH	µg/kg	< 0,2	0,2	
Sum PAH 4 (Benzo(a)anthracene, Benzo(a)pyrene, Benzo(b)fluoranthene, Chrysene)	calculated	HH	µg/kg	< 0,2	0,2	
Sum PAH total	calculated	HH	µg/kg	< 0,2	0,2	
Mycotoxins:						
Aflatoxin B1	LC/MS	HH	µg/kg	< 0,01	0,01	
Aflatoxin B2	LC/MS	HH	µg/kg	< 0,01	0,01	
Aflatoxin G1	LC/MS	HH	µg/kg	< 0,01	0,01	
Aflatoxin G2	LC/MS	HH	µg/kg	< 0,01	0,01	
Sum Aflatoxins B/G	calculated	HH	µg/kg	< 0,01	0,01	

Sample 160180800	Refined veg. oils and fat; 1653493-10; Lipex 102					
Parameter	Method	Lab	Unit	Result	Limit of quantification	Maximum residue level

Parameter	Method	Lab	Unit	Result	Limit of quantification	Maximum residue level
Pesticides - Multimethod:						
Acephate	ASU L 00.00-34	B2	mg/kg	< 0,020	0,020	-
Aldrin	ASU L 00.00-34	B2	mg/kg	< 0,005	0,005	-
Azinphos-ethyl	ASU L 00.00-34	B2	mg/kg	< 0,020	0,020	-
Azinphos-methyl	ASU L 00.00-34	B2	mg/kg	< 0,020	0,020	-
Bifenthrin	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Bromocyclen	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Bromophos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Bromophos-ethyl	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Cadusafos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Carbophenothion	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Oxy-chlordane	ASU L 00.00-34	B2	mg/kg	< 0,005	0,005	-
Chlordan, sum	ASU L 00.00-34	B2	mg/kg	< 0,005	0,005	-
Chlorfenson	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Chlorfenvinphos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Chlormephos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Chlorpyrifos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Chlorpyrifos-methyl	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Chlorthion	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Chlorthiophos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Coumaphos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Cyfluthrin	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Cyhalothrin, lambda-	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Cypermethrine, alpha-	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Cypermethrin, sum	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
DDT, sum calc.	ASU L 00.00-34	B2	mg/kg	< 0,005	0,005	-
DEF	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Deltamethrin	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Demeton-S-methyl	ASU L 00.00-34	B2	mg/kg	< 0,020	0,020	-
Dialifos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Diazinon	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Dichlofenthion	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Dichlorvos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Dicofol, sum	ASU L 00.00-34	B2	mg/kg	< 0,030	0,030	-
Dicrotophos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Dieldrin, in total calc.	ASU L 00.00-34	B2	mg/kg	< 0,005	0,005	-
Dimefox	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Dimethoate	ASU L 00.00-34	B2	mg/kg	< 0,020	0,020	-
Omethoat	ASU L 00.00-34	B2	mg/kg	< 0,020	0,020	-
Sum (Dimethoate, Omethoat) expressed as Dimethoate	ASU L 00.00-34	B2	mg/kg	< 0,020	0,020	-
Dioxabenzofos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Dioxathion	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Disulfoton, in total calc.	ASU L 00.00-34	B2	mg/kg	< 0,020	0,020	-

Sample 160180800	Refined veg. oils and fat; 1653493-10; Lipex 102					
Parameter	Method	Lab	Unit	Result	Limit of quantification	Maximum residue level
Ditalimfos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Endosulfan, in total	ASU L 00.00-34	B2	mg/kg	< 0,005	0,005	-
Endrin	ASU L 00.00-34	B2	mg/kg	< 0,005	0,005	-
Endrin ketone	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
EPN	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Ethion	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Ethoprophos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Etrimfos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Fenamiphos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Fenchlorphos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Fenitrothion	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Fenpropathrin	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Fenson	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Fensulfothion, sum	ASU L 00.00-34	B2	mg/kg	< 0,020	0,020	-
Fenthion	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Fenvalerate	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Flucythrinate	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Fluvalinate, tau-	ASU L 00.00-34	B2	mg/kg	< 0,020	0,020	-
Fonofos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Formothion	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
HCH, sum	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Heptachlor, in total calc.	ASU L 00.00-34	B2	mg/kg	< 0,005	0,005	-
Heptenophos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Hexachlorobenzene	ASU L 00.00-34	B2	mg/kg	< 0,005	0,005	-
Iodofenfos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Iprobenfos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Isocarbophos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Isodrin	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Isofenphos	ASU L 00.00-34	B2	mg/kg	< 0,020	0,020	-
Isofenphos-methyl	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Lindane (HCH, gamma-)	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Malathion	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Malaoxon/Malathion, in total	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Mecarbam	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Methacrifos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Methamidophos	ASU L 00.00-34	B2	mg/kg	< 0,020	0,020	-
Methidathion	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Methylpentachlorphenylsulfide	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Mevinphos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Mirex	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Monocrotophos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Naled	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Oxydemeton-methyl, in total calc.	ASU L 00.00-34	B2	mg/kg	< 0,020	0,020	-
Paraoxon	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Parathion (Parathion-ethyl)	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Parathion-methyl, in total calc.	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Pentachloranisol (PCA)	ASU L 00.00-34	B2	mg/kg	< 0,005	0,005	-
Pentachlorobenzene	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Permethrin	ASU L 00.00-34	B2	mg/kg	-(1)	0,020	-

Sample 160180800	Refined veg. oils and fat; 1653493-10; Lipex 102					
Parameter	Method	Lab	Unit	Result	Limit of quantification	Maximum residue level
Phenkapton	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Phenthoate	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Phorate, in total calc.	ASU L 00.00-34	B2	mg/kg	< 0,020	0,020	-
Phosalone	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Phosmet	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Phosphamidon	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Phoxim	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Pirimiphos-ethyl	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Pirimiphos-methyl	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Profenofos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Propetamphos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Prothiofos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Pyrazophos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Pyridaphenthion	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Quinalphos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Quintozen, in total calc.	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
S421	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Sulfotep	ASU L 00.00-34	B2	mg/kg	< 0,020	0,020	-
Sulprofos	ASU L 00.00-34	B2	mg/kg	< 0,020	0,020	-
Tecnazene	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Tefluthrin	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
TEPP	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Terbufos, in total calc.	ASU L 00.00-34	B2	mg/kg	< 0,020	0,020	-
Tetrachlorvinphos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Tetradifon	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Tetrasul	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Thionazin	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Tolclofos-methyl	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Triamiphos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Triazophos	ASU L 00.00-34	B2	mg/kg	< 0,010	0,010	-
Trichlorfon	ASU L 00.00-34	B2	mg/kg	< 0,020	0,020	-
Vamidothion	ASU L 00.00-34	B2	mg/kg	< 0,020	0,020	-

(1) traces below limit of quantification

Sample 160180800	Refined veg. oils and fat; 1653493-10; Lipex 102					
Parameter	Method	Lab	Unit	Result	Limit of quantification	Maximum residue level
non-dioxinlike PCBs:						
PCB 28	Regulation (EU) No. 589/2014(1)	ZfD	µg/kg	< 0,10	0,1	
PCB 52	Regulation (EU) No. 589/2014(1)	ZfD	µg/kg	< 0,10	0,1	
PCB 101	Regulation (EU) No. 589/2014(1)	ZfD	µg/kg	< 0,10	0,1	
PCB 138	Regulation (EU) No. 589/2014(1)	ZfD	µg/kg	< 0,10	0,1	
PCB 153	Regulation (EU) No. 589/2014(1)	ZfD	µg/kg	< 0,10	0,1	
PCB 180	Regulation (EU) No. 589/2014(1)	ZfD	µg/kg	< 0,10	0,1	
Sum indicator PCBs (upper bound)		ZfD	µg/kg	0,60		

(1) HRGC/HRMS, Result related to original substance
(2) subcontracted.

The laboratory sites of the SGS group Germany according to the abbreviations mentioned above are listed at http://www.institut-fresenius.de/filestore/89/laborstandortkuerzelsgs2.pdf.

To whom it may concern

STATEMENT CONCERNING CONTAMINANTS
This document states the maximum residual levels (MRL) of contaminants in fully refined vegetable oils and fats, which are delivered from AAK*, with reference to relevant EU legislation, WHO Codex Alimentarius Codex Stan, MVO (Product Boards for Margarine, Fats and Oils) "Specification for Refined Vegetable and Marine Oils excluding Olive Oil" and FEDIOL Code of Practices. MRLs based on legislation in bold text in the tables below.

AAK's overall food safety system is based on risk analysis (HACCP) of the entire supply chain. In the monitoring program raw materials, additives and final products are regularly sampled and analysed according to a schedule, and analytical results for potential contaminants are monitored. This program is a part of and serves as a supplementary check on the effectiveness of the food safety system.

The statement concerns:

Dioxins, furans, dioxin-like PCBs	Microorganisms	Radio activity
Glycidyl Fatty Acid Esters	Mineral Oils	Solvents
Non dioxin-like PCBs	PAHs,	
Metals	Pesticides	

Dioxins and dioxin-like PCBs

Component	MRL according to AAK standard (pg/g fat)	MRL according to standard (pg/g fat)	Reference
Sum of dioxins and furans (WHO-PCDD/F-TEQ)	0.75	**0.75**	EU Regulations 1881/2006, 1259/2011
Sum of dioxins, furans and dioxin-like PCB's (WHO-PCDD/F-PCB-TEQ)	1.25	**1.25**	EU Regulations 1881/2006, 1259/2011

Glycidyl Fatty acid esters

Component	MRL according to AAK standard (µg/kg)	MRL according to standard (µg/kg)	Reference
Glycidyl fatty acid esters expressed as glycidol	1000	**1000**	EU Regulation 1881/2006, 2018/290

Non dioxin-like PCBs

Component	MRL according to AAK standard (ng/g fat)	MRL according to standard (ng/g fat)	Reference
Sum of PCB28, PCB52, PCB101, PCB138, PCB153, PCB180	40	**40**	EU Regulations 1881/2006, 1259/2011

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Metals

Component	MRL according to AAK standard (mg/kg)	MRL according to standard (mg/kg)	Reference
Arsenic (As)	0.1	0.1	WHO Codex Alimentarius Codex Stan 210
Cadmium (Cd)	0.05	Oils: - **Soya beans: 0.2**	EU Regulations 1881/2006, 488/2014
Copper (Cu)	0.05	0.05	MVO
Iron (Fe)	0.5	0.5	MVO
Lead (Pb)	0.1	**0.1**	EU Regulation 1881/2006
Mercury (Hg)	0.02	**Oil seeds: 0.02**	EU Regulation 396/2005 including amendments
Nickel (Ni)	0.2	0.2	MVO

Microorganisms

The deodorisation step, in which the oil is heated to above 200°C under vacuum, effectively eliminates microbiological activity.

Indicator organism	MRL according to AAK standard	MRL according to standard	Reference
General			
Total plate count	100 cfu/g	-	AAK Standard
Yeast	10 cfu/g	10 cfu/g	MVO
Mould	10 cfu/g	10 cfu/g	MVO
Enterobacteriacae	10 cfu/g	10 cfu/g	MVO
Pathogenic			
Salmonella	Absent in 25 g	Absent in 25 g	MVO
E. coli	Absent in 1 g	-	AAK Standard

Mineral Oils

Component	MRL according to AAK standard (mg/kg)	MRL according to standard (mg/kg)	Reference
Mineral oils/Diesel/Long chain hydrocarbons Range C10-C56	50	-	AAK Standard

Polycyclic Aromatic Hydrocarbons (PAHs)

Component	MRL according to AAK standard (µg/kg)	MRL according to standard (µg/kg)	Reference
Bens(a)pyrene (BaP)	2.0	**2.0**	EU Regulation 835/2011
Sum of benzo(a)pyrene, benz(a)anthracene, benzo(b)fluoranthene and chrysene (PAH 4)	10	**10**	EU Regulation 835/2011

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Pesticides

Component	MRL according to AAK standard (mg/kg)	MRL according to standard (mg/kg)	Reference
Endosulfan (Sum of α- and β-isomers and endosulfan-sulphate, expressed as endosulfan)	Soybean oil and blends thereof: 0.3 Other oils: 0.1	**Soya beans: 0.5** **Other raw materials: 0.05-0.3 depending on raw material**	EU Regulations 600/2010, 839/2008, 149/2008, 396/2005
Other organochlorine and organophosphorus pesticides	0.01 per pesticide	**0.01-0.1 per pesticide, depending on raw material**	EU Regulations 600/2010, 839/2008, 149/2008, 396/2005

Radio activity

Isotops	MRL according to AAK standard (Bq/kg)	MRL according to standard (Bq/kg)	Reference
Cs^{134} + Cs^{137}	600	**600**	EU Regulation 733/2008

Solvents

Component	MRL according to AAK standard (mg/kg)	MRL according to standard (mg/kg)	Reference
Acetone	1	-	AAK Standard
Hexane	1	**1**	EU Directive 2009/32
Methanol	10	**10**	EU Directive 2009/32

Yours faithfully,

AAK Netherlands BV

Xenophon Koukouvinos
Quality Assurance Director

*This statement concerns products produced at the AAK sites in Aarhus (Denmark), Zaandijk (Netherlands), Karlshamn (Sweden), and Hull (UK). This statement does not cover the Business Area Technical Products & Feed and therefore excludes products from Tefac and Feed.

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CERTIFICATE OF ANALYSIS

To:

Attention: Quality Control

Product: Lipex 102-25Kg Solid
Lot #: 1869488 **Mfg. Date:** 11-Dec-17
Sample #: W180502-140418 **Ship Date:**
Order #: **Container #:**
Contract #:

Customer PO/Release:
Customer Product:

Label Ingredient Statement
USA: INCI:Butyrospermum Parkii (Shea) Butter (US)

Method - Test Parameter	Result	Units	Min	Max
Acid Value (IUPAC 2.201(m)) - Acid Value (mg KOH/g)	0.05	mg KOH/g		0.50
Color 5 1/4" Red (AOCS Cc 13j-97) - Color Red (Lovibond Tintometer)	1.2			2.0
Fatty Acid Composition (IUPAC 2.304) - C18:1	45.6	%	42.0	48.0
Fatty Acid Composition (IUPAC 2.304) - C18:2	6.0	%	5.0	8.0
Fatty Acid Composition (IUPAC 2.304) - C18:0	41.6	%	39.0	44.0
Fatty Acid Composition (IUPAC 2.304) - C16:0	4.5	%	3.0	6.0
Peroxide Value (AOCS Cd 8b-90(m)) - Peroxide Value (meq/kg)	0.1	meq/kg		1.0
Iodine Value (IUPAC 2.205(m)) - Iodine Value (Wijs)	66.4	wijs	60.0	70.0
Unsaponifiable Matter (AOCS Ca 6a-40) - Unsaponifiable matter (%)	6.60	%		

Shelf Life: When stored in unopened original container according to recommended storage conditions, the recommended shelf life is a minimum of two years from the production date but may be extended based on product re-test results.

Recommended Storage: Material should be stored in dark, dry and odour free conditions. Recommended storage temperature is 15-20°C or below.

Note: the reported test results pertain to the sample submitted for analysis.
This certificate is produced electronically and is valid w/o a signature.

Lisa Washakowski

Print Date: 14-May-2018 13:49 CAT#: 19 Document #: CA-12153/1-1

CERTIFICATE OF ANALYSIS

To:

Attention: Quality Control

Product: Lipex 102-25Kg Solid
Lot #: 1895712 **Mfg. Date:** 12-Feb-18
Sample #: W180502-140701 **Ship Date:**
Order #: **Container #:**
Contract #:

Customer PO/Release:
Customer Product:

Label Ingredient Statement
USA: INCI:Butyrospermum Parkii (Shea) Butter (US)

Method - Test Parameter	Result	Units	Min	Max
Acid Value (IUPAC 2.201(m)) - Acid Value (mg KOH/g)	0.19	mg KOH/g		0.50
Color 5 1/4" Red (AOCS Cc 13j-97) - Color Red (Lovibond Tintometer)	1.4			2.0
Fatty Acid Composition (IUPAC 2.304) - C18:1	45.8	%	42.0	48.0
Fatty Acid Composition (IUPAC 2.304) - C18:2	6.0	%	5.0	8.0
Fatty Acid Composition (IUPAC 2.304) - C18:0	42.6	%	39.0	44.0
Fatty Acid Composition (IUPAC 2.304) - C16:0	3.3	%	3.0	6.0
Peroxide Value (AOCS Cd 8b-90(m)) - Peroxide Value (meq/kg)	0.2	meq/kg		1.0
Iodine Value (IUPAC 2.205(m)) - Iodine Value (Wijs)	65.8	wijs	60.0	70.0
Unsaponifiable Matter (AOCS Ca 6a-40) - Unsaponifiable matter (%)	6.20	%		

Shelf Life: When stored in unopened original container according to recommended storage conditions, the recommended shelf life is a minimum of two years from the production date but may be extended based on product re-test results.

Recommended Storage: Material should be stored in dark, dry and odour free conditions. Recommended storage temperature is 15-20°C or below.

Note: the reported test results pertain to the sample submitted for analysis.
This certificate is produced electronically and is valid w/o a signature.

Lisa Washakowski

Print Date: 14-May-2018 13:49 CAT#: 19 Document #: CA-12189/1-3

CERTIFICATE OF ANALYSIS

To:

Attention: Quality Control

Product: Lipex 102-25Kg Solid
Lot #: 1902883 **Mfg. Date:** 27-Feb-18
Sample #: W180529-141627 **Ship Date:**
Order #: **Container #:**
Contract #:

Customer PO/Release:
Customer Product:

Label Ingredient Statement
USA: INCI:Butyrospermum Parkii (Shea) Butter (US)

Method - Test Parameter	Result	Units	Min	Max
Acid Value (IUPAC 2.201(m)) - Acid Value (mg KOH/g)	0.15	mg KOH/g		0.50
Color 5 1/4" Red (AOCS Cc 13j-97) - Color Red (Lovibond Tintometer)	2.0			2.0
Fatty Acid Composition (IUPAC 2.304) - C18:1	46.2	%	42.0	48.0
Fatty Acid Composition (IUPAC 2.304) - C18:2	6.1	%	5.0	8.0
Fatty Acid Composition (IUPAC 2.304) - C18:0	42.0	%	39.0	44.0
Fatty Acid Composition (IUPAC 2.304) - C16:0	3.7	%	3.0	6.0
Peroxide Value (AOCS Cd 8b-90(m)) - Peroxide Value (meq/kg)	0.2	meq/kg		1.0
Iodine Value (IUPAC 2.205(m)) - Iodine Value (Wijs)	67.8	wijs	60.0	70.0
Unsaponifiable Matter (AOCS Ca 6a-40) - Unsaponifiable matter (%)	6.90	%		

Shelf Life: When stored in unopened original container according to recommended storage conditions, the recommended shelf life is a minimum of two years from the production date but may be extended based on product re-test results.

Recommended Storage: Material should be stored in dark, dry and odour free conditions. Recommended storage temperature is 15-20°C or below.

Note: the reported test results pertain to the sample submitted for analysis.
This certificate is produced electronically and is valid w/o a signature.

Lisa Washakowski

Print Date: 30-May-2018 06:03 CAT#: 19 Document #: CA-12154/1-1

CERTIFICATE OF ANALYSIS

To:

Attention: Quality Control

Product: Lipex 102-25Kg Solid

Lot #: 1906520 **Mfg. Date:** 13-Mar-18

Sample #: W180716-100158 **Ship Date:**

Order #: **Container #:**

Contract #:

Customer PO/Release:

Customer Product:

Label Ingredient Statement

USA: INCI:Butyrospermum Parkii (Shea) Butter (US)

Method - Test Parameter	Result	Units	Min	Max
Acid Value (IUPAC 2.201(m)) - Acid Value (mg KOH/g)	0.11	mg KOH/g		0.50
Color 5 1/4" Red (AOCS Cc 13j-97) - Color Red (Lovibond Tintometer)	1.5			2.0
Fatty Acid Composition (IUPAC 2.304) - C18:1	45.6	%	42.0	48.0
Fatty Acid Composition (IUPAC 2.304) - C18:2	6.0	%	5.0	8.0
Fatty Acid Composition (IUPAC 2.304) - C18:0	42.6	%	39.0	44.0
Fatty Acid Composition (IUPAC 2.304) - C16:0	3.7	%	3.0	6.0
Peroxide Value (AOCS Cd 8b-90(m)) - Peroxide Value (meq/kg)	0.1	meq/kg		1.0
Iodine Value (IUPAC 2.205(m)) - Iodine Value (Wijs)	66.5	wijs	60.0	70.0
Unsaponifiable Matter (AOCS Ca 6a-40) - Unsaponifiable matter (%)	6.60	%		

Shelf Life: When stored in unopened original container according to recommended storage conditions, the recommended shelf life is a minimum of two years from the production date but may be extended based on product re-test results.

Recommended Storage: Material should be stored in dark, dry and odour free conditions. Recommended storage temperature is 15-20°C or below.

Note: the reported test results pertain to the sample submitted for analysis.
This certificate is produced electronically and is valid w/o a signature.

Lisa Washakowski

Print Date: 16-Jul-2018 10:16 CAT#: 19 Document #: CA-12480/2-2

CERTIFICATE OF ANALYSIS

To:

Attention: Quality Control

Product: Lipex 205 - 18Kg
Lot #: P1834396 **Mfg. Date:** 21-Feb-18
Sample #: W180405-142912 **Ship Date:**
Order #: **Container #:**
Contract #:

Customer PO/Release:
Customer Product:

Label Ingredient Statement
USA: INCI: Butyrospermum Parkii (Shea) Butter (US)

Method - Test Parameter	Result	Units	Min	Max
Acid Value (IUPAC 2.201(m)) - Acid Value (mg KOH/g)	0.07	mg KOH/g		0.50
Color 5 1/4" Red (AOCS Cc 13j-97) - Color Red (Lovibond Tintometer)	1.0			2.0
Peroxide Value (AOCS Cd 8b-90(m)) - Peroxide Value (meq/kg)	0.2	meq/kg		1.0
Iodine Value (IUPAC 2.205(m)) - Iodine Value (Wijs)	74.0	wijs	70.0	80.0

Shelf Life: When stored in unopened original container according to recommended storage conditions, the recommended shelf life in drums is a minimum of two years and in plastic pails a minimum of one year from the production date but may be extended based on product re-test results.

Recommended Storage: Material should be stored in dark, dry and odour free conditions. Recommended storage temperature is 15-20°C or below.

Note: the reported test results pertain to the sample submitted for analysis.
This certificate is produced electronically and is valid w/o a signature.

Lisa Washakowski

Print Date: 07-May-2018 17:19 CAT#: 19 Document #: CA-12182/1-3

CERTIFICATE OF ANALYSIS

To:

Attention: Quality Control

Product: Lipex 205 - 18Kg
Lot #: P1923657-1 **Mfg. Date:** 02-Jul-18
Sample #: W180709-095527 **Ship Date:**
Order #: **Container #:**
Contract #:

Customer PO/Release:
Customer Product:

Label Ingredient Statement
USA: INCI: Butyrospermum Parkii (Shea) Butter (US)

Method - Test Parameter	Result	Units	Min	Max
Acid Value (IUPAC 2.201(m)) - Acid Value (mg KOH/g)	0.07	mg KOH/g		0.50
Color 5 1/4" Red (AOCS Cc 13j-97) - Color Red (Lovibond Tintometer)	1.9			2.0
Peroxide Value (AOCS Cd 8b-90(m)) - Peroxide Value (meq/kg)	0.2	meq/kg		1.0
Iodine Value (IUPAC 2.205(m)) - Iodine Value (Wijs)	72.9	wijs	70.0	80.0

Shelf Life: When stored in unopened original container according to recommended storage conditions, the recommended shelf life in drums is a minimum of two years and in plastic pails a minimum of one year from the production date but may be extended based on product re-test results.

Recommended Storage: Material should be stored in dark, dry and odour free conditions. Recommended storage temperature is 15-20°C or below.

Note: the reported test results pertain to the sample submitted for analysis.
This certificate is produced electronically and is valid w/o a signature.

Lisa Washakowski

Print Date: 09-Jul-2018 10:08 CAT#: 19 Document #: CA-12320/1-1

CERTIFICATE OF ANALYSIS

To:

Attention: Quality Control

Product: Lipex 205 - 18Kg
Lot #: P1944451-1 **Mfg. Date:** 13-Sep-18
Sample #: W180921-111540 **Ship Date:**
Order #: **Container #:**
Contract #:

Customer PO/Release:
Customer Product:

Label Ingredient Statement
USA: INCI: Butyrospermum Parkii (Shea) Butter (US)

Method - Test Parameter	Result	Units	Min	Max
Acid Value (IUPAC 2.201(m)) - Acid Value (mg KOH/g)	0.06	mg KOH/g		0.50
Color 5 1/4" Red (AOCS Cc 13j-97) - Color Red (Lovibond Tintometer)	1.6			2.0
Peroxide Value (AOCS Cd 8b-90(m)) - Peroxide Value (meq/kg)	0.2	meq/kg		1.0
Iodine Value (IUPAC 2.205(m)) - Iodine Value (Wijs)	72.9	wijs	70.0	80.0

Shelf Life: When stored in unopened original container according to recommended storage conditions, the recommended shelf life in drums is a minimum of two years and in plastic pails a minimum of one year from the production date but may be extended based on product re-test results.

Recommended Storage: Material should be stored in dark, dry and odour free conditions. Recommended storage temperature is 15-20°C or below.

Note: the reported test results pertain to the sample submitted for analysis.
This certificate is produced electronically and is valid w/o a signature.

Lisa Washakowski

Print Date: 21-Sep-2018 11:16 CAT#: 19 Document #: CA-2-226747/1

CERTIFICATE OF ANALYSIS

Product: Illexao HS 90 (Shea Stearin)
Lot #: 18D31236 **Mfg. Date:** 06-Jul-18
Sample #: W190117-180608

Attention: Quality Control

Kosher

Label Ingredient Statement

USA: INCI: Butyrospermum Parkii (Shea) Butter (US)
FDA: Sheanut Oil

Method - Test Parameter	Result	Units	Min	Max
FFA - As Oleic	0.070	%		0.070
Peroxide Value	0.0	meq/kg		1.0
Color - Red	0.4	Lovibond		2.5
Color - Yellow	2.4	Lovibond		40.0
IV	36.2		35.5	38.5
Flavor	Pass			

For reference, the AAK RSPO certificate # is: BMT-RSPO-000035.
The RSPO certificate number only applies to sales contracts of certified sustainable products (Segregated or Mass Balance).

Note: the reported test results pertain to the sample submitted for analysis.
This certificate is produced electronically and is valid w/o a signature.

Lisa Washakowski

Print Date: 17-Jan-2019 18:09 CAT#: 13 Document #: CA-2-246944/1

CERTIFICATE OF ANALYSIS

Product: Illexao HS 90 (Shea Stearin)

Lot #: 18D32095 **Mfg. Date:** 09-Nov-18

Attention: Quality Control **Sample #:** W190117-181927

Kosher

Label Ingredient Statement

USA: INCI: Butyrospermum Parkii (Shea) Butter (US)
FDA: Sheanut Oil

Method - Test Parameter	Result	Units	Min	Max
FFA - As Oleic	0.059	%		0.070
Peroxide Value	0.0	meq/kg		1.0
Color - Red	0.5	Lovibond		2.5
Color - Yellow	3.0	Lovibond		40.0
IV	35.9		35.5	38.5
Flavor	Pass			

For reference, the AAK RSPO certificate # is: BMT-RSPO-000035.
The RSPO certificate number only applies to sales contracts of certified sustainable products (Segregated or Mass Balance).

Note: the reported test results pertain to the sample submitted for analysis.
This certificate is produced electronically and is valid w/o a signature.

Lisa Washakowski

Print Date: 17-Jan-2019 18:25 CAT#: 13 Document #: CA-2-246954/1

CERTIFICATE OF ANALYSIS

Product: Illexao HS 90 (Shea Stearin)

Lot #: 19D30067 **Mfg. Date:** 14-Jan-19

Attention: Quality Control **Sample #:** W190117-165046

Kosher

Label Ingredient Statement

USA: INCI: Butyrospermum Parkii (Shea) Butter (US)
FDA: Sheanut Oil

Method - Test Parameter	Result	Units	Min	Max
FFA - As Oleic	0.060	%		0.070
Peroxide Value	0.0	meq/kg		1.0
Color - Red	0.3	Lovibond		2.5
Color - Yellow	1.9	Lovibond		40.0
IV	36.3		35.5	38.5
Flavor	Pass			

For reference, the AAK RSPO certificate # is: BMT-RSPO-000035.
The RSPO certificate number only applies to sales contracts of certified sustainable products (Segregated or Mass Balance).

Note: the reported test results pertain to the sample submitted for analysis.
This certificate is produced electronically and is valid w/o a signature.

Lisa Washakowski

Print Date: 17-Jan-2019 16:53 CAT#: 13 Document #: CA-2-246931/1

Viebrock, Lauren

From: Jeffrey Fine <Jeffrey.Fine@aak.com>
Sent: Monday, May 18, 2020 9:41 PM
To: Viebrock, Lauren
Subject: AAK Submission GRN 892

Follow Up Flag: Follow up
Flag Status: Flagged

Dear Dr. Viebrock:

I am writing on behalf of AAK to request that FDA terminate its review of our GRAS Notification for Shea (GRN 892). Based on comments received from FDA we recognize that the precise identity of the ingredient under consideration was unclear and perhaps ambiguous.

Thank you for communicating this to us and for the clarification you provided in our recent discussion. We intend to use this feedback constructively to meet the requirements of the Agency in a "revised" subsequent Notification.

Sincerely,

Jeffrey Fine, PhD.

Jeffrey Fine:: R&D
AAK USA Inc. :: Edison NJ, 08837
m: 973-369-5002