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Anti-inflammatory and Other Bioactivities of Triterpene Esters in Shea Butter

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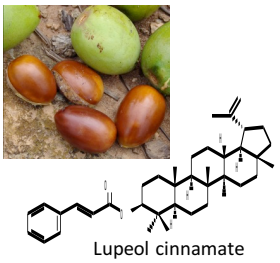
Shea butter is used as a substitute for cocoa butter in chocolate industries due to its similarity in triacylglycerol composition to cocoa butter. In addition, shea butter has recently become a very popular ingredient in cosmetics and personal care applications. The main reason for the use of shea butter in high-value cosmetic formulations is associated with the high levels of non-glyceridic lipids, especially triterpene esters. This article highlights the triterpene ester components of shea butter and their bioactivities including anti-inflammatory and protease-inhibitory activities, as well as cancer chemopreventive properties. The triterpene ester fractions of shea butter are consisted with the cinnamic and acetic acid esters of α-amyrin, β-amyrin, lupeol, and butyrospermol.



Keyword: *Shea butter; triterpene esters; anti-inflammatory activity; chemopreventive property; skincare formulation.*

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anti-inflammatory, cancer chemopreventive, cytotoxic, and melanogenesis- inhibitory activities.

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1. Introduction

The shea tree (*Vitellaria paradoxa* C.F. Gaertner; belonging to the Sapotaceae family) is indigenous to the savanna belt extending across sub-Saharan Africa north of the equator, ranging from Mali in the west to Ethiopia and Uganda in the east (**Figure 1**). The trees grow to a size of 15-22 m, starting to yield commercial quantities of fruit after 25-50 years. The trees produce small whitish flowers in February-March and the fruits mature in June-July. Harvesting of the fruits takes place in June-September. The large (4-8 cm) fleshy fruits contain one or two kernels rich in the fat we know as shea butter.[1-5] Shea butter has been used extensively in Cocoa Butter Equivalents (CBEs) since the 1960s and is currently one of the permitted fats for use in chocolate under the European Union Chocolate Directive. Shea butter has gained increasing popularity as an ingredient for cosmetics and personal care products in international markets. The main reason for this growing interest of shea butter has been the recognition of its therapeutic benefits—ultra-violet light protection, anti-inflammatory, moisturizing, regenerative, anti-eczema, and anti-wrinkle properties—due to the presence of a significant fraction of non-glyceridic lipids (3-12%) that includes many bioactive constituents, especially triterpene (alcohol) esters.[1-5] The present review highlights the triterpene ester components of shea butter and their anti-inflammatory and other bioactivities.

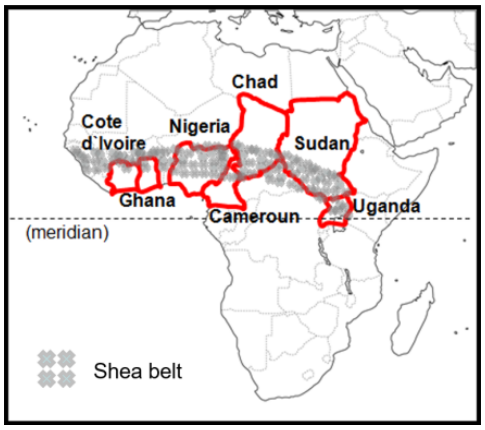

Figure 1. Map of Africa showing the "Shea belt" region, the area that shea tree grows in.

2. Triacylglycerols and Fatty Acids of Shea Butter

The fat content of shea kernels is highly variable depending mainly on the populations of shea tree, and ranged generally from 20 to 58%.[6-9] Thus, upon investigation on the shea kernels for 36 samples from seven sub-Saharan countries, *i.e.*, Cote d'Ivoire, Ghana, Nigeria, Cameroun, Chad, Sudan, and Uganda (**Figure 1**), these have been found to contain fat in the range of 29.7-53.7% with a mean value of 42.4%.[6] Di Vincenzo *et al.*[7] reported the fat content of 158 kernel samples from four

African countries ranged from 41 to 55% (mean 48%), while Gwalia *et al.*[8] investigated 44 ethno-varieties of shea kernels in Uganda and reported their fat content ranged from 43.9 to 58.4%. Maranz *et al.* have investigated shea kernels for 42 shea populations in 11 African countries.[9] The results showed very high variability in fat content both within and between populations, and the fat content range was 20-50%.[9]

2-1. Triacylglycerols (TG): Investigation on the TG fractions for 36 shea butter samples has revealed that these contain SOS and SOO as the principal constituents, followed by POS, OOO, and SLO (**Table 1**), where L: linoleic acid (fatty acid chain length: number of double bonds = 18:2), O: oleic acid (18:1), P: palmitic acid (16:0), and S: stearic acid (18:0).[10] These TG profile range is consistent with that reported elsewhere.[1,3,7,9] The TG profiles showed a strong geographic trend: shea samples from the western area of the shea belt (Cote d'Ivoire, Ghana, and Nigeria) exhibited a higher concentration of high-melting TGs, such as SOS and POS, whereas those from the central (Cameroun and Chad) and eastern areas (Sudan and Uganda) contained higher percentages of low-melting TGs, such as SOO and OOO. Given the substantially higher percentages of SOS, which was highest in samples from western Nigeria and Ghana, these support the chocolate industry's preference for West African shea butters.[10]

Table 1. Triacylglycerol Variation in Shea Butter[a)]

Triacylglycerol (TG)	Percentage of total TG		
	Mean	Min	Max
SOS	31.2	20.3	43.7
SOO	27.7	19.8	37.1
POS	13.1	5.7	18.2
OOO	10.8	3.1	23.6
SLO (+ POO & PLS)	10.3	5.8	15.9

a) Data are from 36 trees samples in seven countries.[10] Fatty acid key: S = stearic, O = oleic, P = palmitic, L = linoleic.

2-2. Fatty Acid Profiles: The fatty acid profile of shea butter is almost reflected with the compositions of the TG fractions. The fatty acid composition is simple, dominated by two acids, oleic and stearic acids, with the decreasing proportions of four acids, linoleic, palmitic, arachidic (20:0), and linolenic acids (18:3).[1,3,9] Stearic and oleic acids together account for 85-90% of the fatty acids in most shea butters.[1,3,7,8]

The fatty acid profile determined for 36 shea nut samples from seven sub-Saharan countries was shown in **Table 2**.[6] Shea butters from Uganda, located at the eastern part of the shea belt region, had substantially higher oleic acid percentages than those from West Africa, with intermediate values seen in central African provenances (**Figure 1**).[3] Generally, the high stearic acid content of shea butter is responsible for the solid consistency that makes shea butter distinctive, while the relative percentage of oleic acid influences how soft or hard the shea butter is.[9]

Table 2. Fatty Acid Variation in Shea Butter[a)]

Fatty Acid		Percentage of total TG		
		Mean	Min	Max
16:0[b)]	Palmitic	4.4	3.3	7.5
18:0	Stearic	40.4	29.5	55.7
18:1	Oleic	49.3	37.2	60.7
18:2	Linoleic	6.6	4.3	8.0
18:3	Linolenic	0.4	0.2	1.7
20:0	Arachidic	1.3	0.8	1.8

a) Data are from 36 trees samples in seven countries.[6]
b) Fatty acid carbon chain length: number of double bond.

3. Non-glyceridic Lipid (NGL) Components of Shea Butter

One of the main differences between shea butter and the majority of other vegetable oils and fats is its high concentration of NGL components. A normally extracted and refined shea butter contains 5-10% by weight of NGL while a typical vegetable oil contains less than 1% of NGL.[5] The NGL fraction ranged from 2.4% to 11.6% (mean 6.2%) for the 36 samples of shea butter analyzed.[6]

3-1. Triterpene Alcohols and Their Esters: The main components of the unsaponifiabls of shea butter are triterpene alcohols,[4,5,6,10] and the triterpene content in the unsaponifiables for the 36 shea butter samples ranged from 22.4% to 71.7% with a mean 48.1%.[6] The major triterpene alcohols were α-amyrin (**1**), β-amyrin (**2**), lupeol (**3**), and butyrospermol (**4**) with the minor levels of ψ-taraxasterol (**5**), taraxasterol (**6**), parkeol (**7**), 24-methylene-24-dihydroparkeol (**8**), 24-methylenecycloartanol (**9**), dammaradienol (**10**), and 24-methylenedammarenol (**11**) (Figure 2; Table 3).[6]

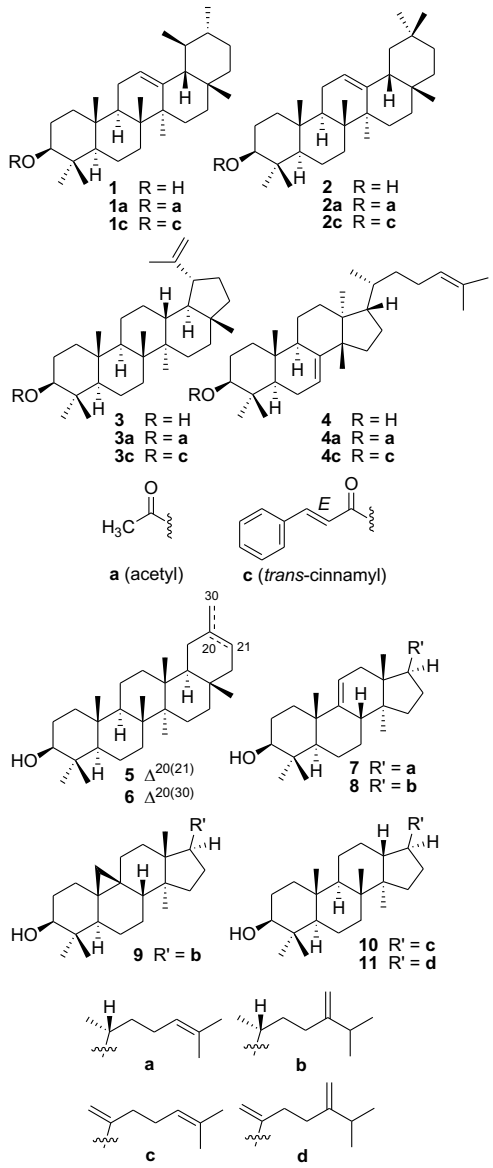

Figure 2. Structures of Triterpene Alcohols and Their Esters found in Shea Butter

Table 3. Compositions (%) of Triterpene Alcohol (TA) Fractions from the Unsaponifiable Fracitons of 36 Shea Butter Samples[a)]

	Compound	% of TA Fraction (mean)
1	α-Amyrin	31.3–41.4 (36.3)
2	β-Amyrin	8.4–13.2 (9.6)
3	Lupeol	17.4–25.1 (21.0)
4	Butyrospermol	14.9–26.3 (20.3)
5	ψ-Taraxasterol	1.4–4.2 (3.1)
6	Taraxasterol	0.7–2.2 (1.6)
7	Parkeol	3.1–6.2 (3.9)
8	24-Methylene-24-dihydroparkeol	trace
9	24-Methylenecycloartanol	trace
10	Dammaradienol	trace
11	24-Methylenedammarenol	trace

a) Taken from ref.[6]

Most of the triterpene alcohols in shea butter occur as the esterified forms with acetic acid and cinnamic acid.[4,5,6,10] The triterpene ester (TE) fractions in the 36 shea butter samples are in the range of 0.5-6.5%, and contain α-amyrin cinnamate (**1c**) as the predominant TE followed by butyrospermol cinnamate (**4c**), α-amyrin acetate (**1a**), lupeol cinnamate (**3c**), β-amyrin cinnamate (**2c**), lupeol acetate (**3a**), butyrospermol acetate (**4a**), and β-amyrin acetate (**2a**) (Figure 2; Table 4).[10] Analysis of the percentages of acetyl and cinnamyl triterpene esters showed strong regional affinity, with the highest values found in Nigerian provenances and the lowest values in Ugandan butters.[7,10] This suggests that West African provenances had significantly higher levels of both acetyl and cinnamyl triterpenes than shea butter from East Africa.

Table 4. Compositions (%) of Triterpene Ester (TE) Fractions of 36 Shea Butter Samples[a)]

	Compound	% of TE Fraction (mean)[b)]	
		Acetate (**a**)	Cinnamate (**c**)
1	α-Amyrin	8.9–18.7 (14.1)	19.9–35.5 (29.3)
2	β-Amyrin	2.7–9.3 (4.9)	4.4–11.1 (7.6)
3	Lupeol	4.5–10.3 (7.2)	4.4–13.4 (9.0)
4	Butyrospermol	3.4–10.3 (5.8)	5.9–19.6 (14.8)

a) Taken from ref.[10]

3-2. Sterols: Sterols constitute a small proportion of the unsaponifiable fraction of shea butter.[6,11,12] The unsaponifiable fraction of shea butter was found to contain 5% of sterols and 2% of methylsterols, in addition to major

triterpene alcohols (75%).[11] The sterol fraction was consisted exclusively of Δ^7-sterols, *i.e.*, spinasterol (**12**; 43% of the total sterols), shottenol (**13**; 37%), avenasterol (**14**; 11%), and 24-methyllathosterol (**15**; 6%) (**Figure 3**).[11]

3-3. Tocopherols: Bup *et al.*[13] studied tocopherols (vitamin E) in shea butter, revealing that the total tocopherol content ranged from 37.2-385.2 μg/g of shea butter, with a mean of 163.1 μg/g. Four isomers of tocopherols [α (**16**), β (**17**), γ (**18**), and δ (**19**)] (**Figure 3**) were detected among which α-tocopherol (**16**) constituted the major type of tocopherol ranging from 53 to 83% of the total tocopherols with a mean of 65.5%. The mean values for β (**17**), γ (**18**), and δ (**19**) were 2.3%, 30.0%, and 2.4%, respectively.

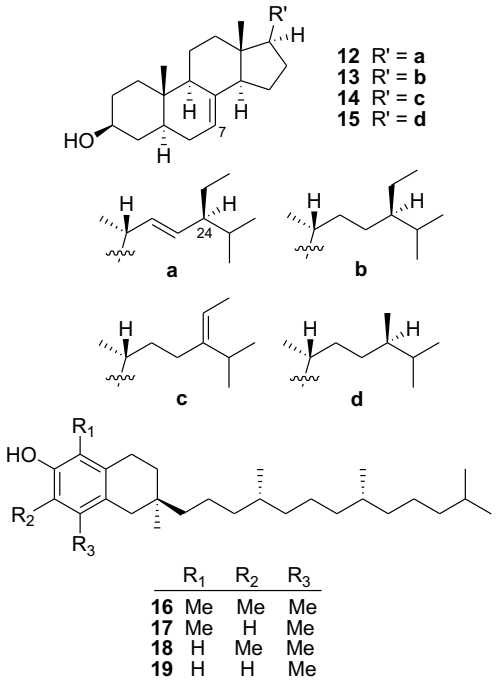

Figure 3. Structures of Sterols and Tocopherols found in Shea Butter

Maranz & Wiesman[14] reported corresponding values of 64%, 7%, 15%, and 14%, respectively, while Honfo *et al.*[15] reported values of 76-79%, 3-5%, 16-17%, and 0.7-0.9%, respectively, for the same parameters. The tocopherol content in shea butter is comparatively low, corresponding to a low level for sensitive polyunsaturated fatty acids in the butter. However, the main shea butter fatty acids, oleic and stearic acids, are sufficiently stable against oxidation. Thus, if carefully purified and correctly handled, shea butter and its extracts show superior oxidative stability and do not require additional antioxidants.[16]

4. Bioactivities of Triterpene Esters from Shea Butter

Due to the semi-solid characteristics and buttery consistency, shea butter itself can be used as great emollient (skin softener) and moisturizer without further processing. In addition, fractionated shea butter especially olein fraction is easily formulated in creams or surfactant based products such as bath products and shampoo to provide the skin, scalp, and hair with well-maintained or increased moisture. While these properties are due to shea butter's characteristic fatty acid composition, many other medicinal properties are related to its NGL constituents, primarily the triterpene esters, which exhibit various bioactivities such as anti-inflammatory, protease-inhibiting, and chemopreventive properties.[1,3,16,17]

4-1. Anti-inflammatory Activity: Traditional uses of shea butter in African folk medicine have been greatly attributed to the anti-inflammatory properties of shea butter, which may be related to the NGL constituents, especially triterpene alcohols and their esters.[18] Several studies show that triterpene alcohols and their esters, and the triterpene ester enriched fraction of shea butter exhibit anti-inflammatory in different models of inflammation. The anti-inflammatory activity against 12-*O*-tetradecanoylphorbol acetate (TPA)-induced inflammation in mice of four triterpene acetates (**1a**-**4a**) and four triterpene cinnamates (**1c**-**4c**), isolated from shea butter,

was investigated, and their inhibitory activities were compared with those of a commercially available anti-inflammatory drug, indomethacin, as shown in **Table 5**.[19] All triterpene esters tested markedly inhibited the TPA (1 μg/ear)-induced inflammation [ID_{50} (50% inhibitory dose) 0.08-0.35 mg/ear], which is almost equivalent with, or more inhibitory than indomethacin (ID_{50} 0.3 mg/ear). The cinnamates (**1c-4c**) exhibited stronger activity (ID_{50} 0.08-0.29 mg/ear) than those of the corresponding acetates (**1a-4a**) (ID_{50} 0.25-0.35 mg/ear). Among the cinnamates, lupeol cinnamate (**3c**) exhibited the strongest inhibitory activity (ID_{50} 0.08 mg/ear).

Table 5. Inhibitory Effects of Triterpene Alcohols and Their Esters Isolated from Shea Butter, and Reference Compound on TPA-induced Inflammation in Mice

Compound	ID_{50} (mg/ear)[a]		
	Free alcohol[b]	Acetate (**a**)[c]	Cinnamate (**c**)[c]
1	0.2	0.28	0.29
2	0.4	0.35	0.15
3	0.6	0.25	0.08
4	0.6	0.33	0.11
5	0.4		
6	0.3		
8	0.4		
9	0.5		
10	0.8		
11	0.5		
Indomethacin[d]	0.3		

a) 50% inhibitory dose. b) Taken from ref.[23] c) Taken from ref.[19] with a slight modification. d) Reference compound.

These results suggest that topical application of triterpene esters exert a strong and rapid onset inhibition of TPA-induced inflammation. These effects seem to be associated with the suppression of skin prostaglandin E_2 (PGE_2) levels by mechanisms involving the suppression of cyclooxygenase (COX)-2 expression,[20] *via* inhibition of upstream protein kinases, namely, extracellular signal-regulated protein kinase (ERK), p38 mitogen-activated protein kinase (MAPK), and protein kinase C (PKC) α, and blocking nuclear factor-κB (NF-κB) activation.[21]

Compound **3c** has further exhibited anti-inflammatory activity on rat hind paw edema induced by carrageenan.[19] Paw edema caused by subcutaneous injection of carrageenan is due to vasodilation and increased vascular permeability. This event is caused by the release of various inflammatory mediators such as PGs and thromboxane (TX).[22] It is suggested that **3c** probably exerted anti-inflammatory activity through the inhibition of those inflammatory mediators of the acute inflammation. Inhibition of the synthesis or release of inflammatory mediators may be the main mechanisms for the action of **3c**.

Ten free triterpene alcohols, **1-6** and **8-11**, isolated from the unsaponifiable fraction of shea butter[6] also have been evaluated for their anti-inflammatory activity against TPA-induced inflammation in mice.[23,24] All these triterpene alcohols showed inhibitory activities (ID_{50} 0.2-0.8 mg/ear) in the same concentration range as the control indomethacin (**Table 5**).

The anti-inflammatory activities of the methanolic extract (this is supposed to contained enriched triterpene esters) of shea butter using lipopolysaccharide (LPS)-induced murine macrophage cell line J774 have been studied.[25] It was observed that the extract significantly reduced the levels of LPS-induced nitric oxide (NO), tumor necrosis factor-α (TNF-α), interleukins (IL), -1β (IL-1β), and -12 (IL-12). Expression of pro-inflammatory enzymes, inducible nitric oxide synthase (iNOS), and COX-2 were also inhibited by the extract. These anti-inflammatory effects were due to an inhibitory action of the extract on LPS-induced iNOS, COX-2, TNF-α, IL-1β, and IL-12 mRNA expressions. Moreover, the shea butter methanol extract efficiently

suppressed IκBα phosphorylation and NF-κB nuclear translocation induced by LPS. This explains the molecular bases of shea butter's bioactivity against various inflammatory conditions.

The triterpene ester concentrate of shea butter (more than 50% triterpene esters) has been investigated for its attenuating effect against osteoarthritis (OA)-induced pain and joint destruction in rats.[26] This showed that the concentrate reduced the swelling of the knee joint with OA and rectified its weight bearing after anterior cruciate ligament transaction (ACLT) with medial meniscectomy (MMx) surgery in rats. Treatment with the concentrate also decreased ACLT plus MMx surgery-induced knee joint matrix loss and cartilage degeneration. The shea butter triterepene ester concentrate relieves the symptoms of OA and protects cartilage from degeneration, and, thus, this has the potential to be an ideal nutraceutical supplement for joint protection, particularly for injured knee joints.[26,27]

4-2. Protease-inhibiting Activity: Collagen and elastin are the major structural proteins providing skin with toughness and plumpness. The production of collagen and elastin decreases with increasing age, resulting in thinner and less elastic skin. The effects of this natural aging process can be alleviated by stimulating collagen and elastin synthesis or by inhibiting the activity of the collagenases and elastases. Some of the triterpene alcohols found in shea butter have been found to contribute to the inactivation of proteases such as metalloprotease (*e.g.*, collagenase) as well as serine protease (*e.g.*, elastase).[1] Recently, Anderson and Alander have demonstrated that triterpene ester enriched shea extract (60-65% triterpenes) possess protease-inhibiting, collagen-enhancing, and skin barrier strength and protection activities as well as anti-inflammatory activity.[16] These findings highlight the triterpene ester enriched shea extract as a potential bioactive lipid complex for protecting the skin against environmental stress conditions and treating aged skin.

4-3. Cancer Chemopreventive Property: Prevention is one of the most important and promising strategies to control cancer. Carcinogenesis is generally recognized as a multistep process in which distinct molecular and cellular alterations occur. From the study of experimentally induced carcinogenesis in rodents, tumor development is considered to consist of several separate, but closely linked, stages: tumor initiation, promotion, and progression. Chemoprevention is defined as the use of natural or synthetic agents that reverse, suppress or arrest carcinogenic and/or malignant phenotype progression towards invasive cancer. Inhibitors on the induction of Epstein-Barr virus early antigen (EBV-EA) induced by TPA in Raji cells (derived from Burkitt's lymphoma) have been demonstrated to act as inhibitors in an *in vivo* two-stage carcinogenesis test using 7,12-dimethylbenz[*a*]anthracene (DMBA) as an initiator and TPA as a promoter.[28]

4-3-1. Inhibitory Effect on *in vitro* EBV-EA Activation: The inhibitory effects on EBV-EA activation induced by TPA have been examined as a preliminary evaluation of the potential antitumor-promoting activities of compounds **1a**-**4a** and **1c**-**4c**.[19] The results are collected in **Table 6**, together with the corresponding data for β-carotene, a vitamin A precursor studied widely in cancer-chemoprevention animal models, and

for retinoic acid, one of the retinoids that has been studied as a cancer-chemoprevention agent for various organ site cancers.[29] Even at a concentration of 32 nmol (molar ratio of compound to TPA 1000:1), high viability (70%) of Raji cells was observed indicating low cytotoxicity of all compounds. All compounds tested showed inhibitory effects with IC_{50} values (concentration for 50% inhibition with respect to positive control) of 373-470 molar ratio/32 pmol TPA. These values are almost equivalent to, or more potent than, retinoic acid (IC_{50} 482 molar ratio/32 pmol TPA). Among the eight compounds tested, both acetates and cinnamates of butyrospermol (**3**) and lupeol (**4**) showed potent inhibitory effects (IC_{50} 373-383 molar ratio/32 pmol TPA), which were almost comparable with, or more potent than β-carotene (IC_{50} 397 molar ratio/32 pmol TPA). Three free triterpene alcohols, α-amyrin (**1**), β-amyrin (**2**), and 24-methylenecycloartanol (**9**), also have been reported to exhibit potent inhibitory effects on EBV-EA activation with IC_{50} values of 393, 470, and 272 molar ratio/32 pmol TPA, respectively.[28] These compounds may have, therefore, potential as antitumor-promoters.

Table 6. Inhibitory Effects of Triterpene Alcohols and Their Esters Isolated from Shea butter on the Induction of Epstein-Barr Virus Early Antigen (EBV-EA).

Compound	IC_{50}[a]		
	Free alcohol[b]	Acetate (**a**)[c]	Cinnamate (**c**)[c]
1	393	401	470
2	470	405	452
3	n.d.	383	379
4	n.d.	380	373
9	272		
β-Carotene[d]	397		
Retinoic acid[d]	482		

a) IC_{50} values represent the molar ratios of compounds, relative to TPA, required to inhibit 50% of the positive control activated with 32 pmol TPA. b) Taken from ref.[28]; n.d. = not determined. c) Taken from ref.[19] d) Reference compounds.

4-3-2. Inhibitory Effect on *in vivo* Two-stage Carcinogenesis Assay on Mouse Skin Papillomas: The inhibitory effect of lupeol cinnamate (**3c**), one of the potent inhibitors on *in vitro* EBV-EA activation assay, has been evaluated in a tumor model in mouse skin, and the inhibitory effect has been compared with that of β-carotene.[19,30] The incidence (%) of papilloma-bearing mice and the average numbers of papillomas per mouse in the two-stage carcinogenesis test in mouse skin using DMBA as an initiator and TPA as a promoter are presented in **Figure 4A** and **4B**, respectively. The incidence of the papilloma-bearing mice was high and 100% at 10 weeks promotion in *Group I* (the positive control). Per mouse at 10 and 20 weeks of the promotion, 3.7 and 8.6 papillomas, respectively, were found. The formation of papillomas in the mouse skin was delayed, and the mean numbers of papillomas per mouse were reduced by the treatment with compound **3c** (*Group II*). Thus, in *Group II*, treated with compound **3c**, the percentage ratios of papilloma-bearing mice were 27% at 10 weeks, and 93% at 20 weeks, and the mean papillomas per mouse were 1.6 at 10 weeks, and 4.1 at 20 weeks. The inhibitory effect of compound **3c** was found to be superior to that of reference β-carotone (*Group III*; papilloma bearers: 33 and 100% at 10 and 20 weeks, respectively; papillomas/mouse: 1.5 and 6.0 at 10 and 20 weeks, respectively).

Several studies have reported that skin applications of TPA result in several histological and biochemical alterations including inflammatory responses such as development of edema, hyperplasia, and induction of COX-2 expression,[31] and generation of reactive oxygen species, which play a critical role in oxidation of many macromolecules and help in initiation as well as promotion of tumorigenesis.[32] TPA induces the enzyme ornithine decarboxylase (ODC) activity, which is a biomarker of skin tumorigenesis.[33] Administration of TPA results in stimulation of Ras signaling pathway along

with activation of a number of kinases subsequently activates the cell proliferation pathway and alters the expression of anti-apoptotic Bcl-2 and proapoptotic Bax protein.[34]

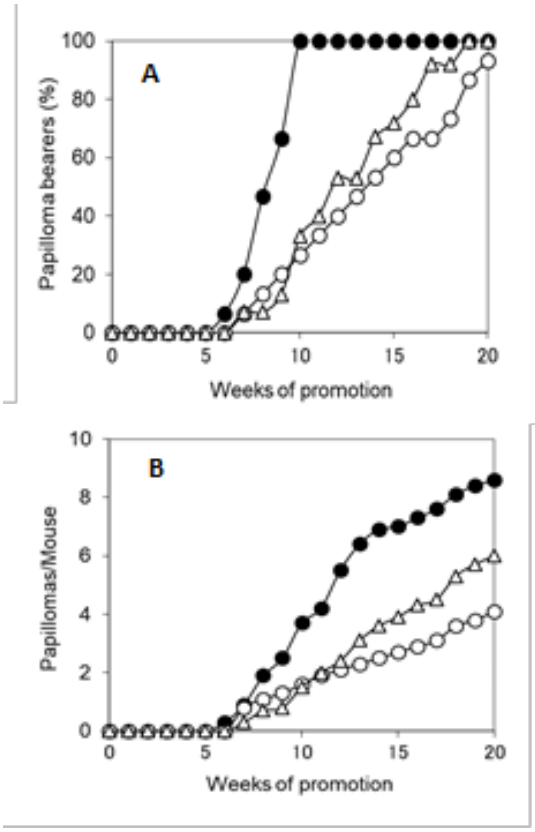

Figure 4. Inhibitory effect of lupeol cinnamate (**3c**) and reference β-carotene on mouse skin carcinogenesis induced by DMBA and TPA. (**A**) Percentage of mice with papillomas; (**B**) average number of papillomas per mouse. Tumor formation in all mice (*n* = 15 for each of groups I, II, and III) was initiated with DMBA (390 nmol) and promoted with TPA (1.7 nmol) twice weekly, starting one week after initiation. Filled circles (●) represent the untreated control group (TPA alone*; Group I*); open circles (○) refers to TPA + **3c** (85 nmol*; Group II*); open triangles (△) refer to TPA + β-carotene (85 nmol; *Group III*). After 20 weeks of promotion, a significant difference in the number of papillomas per mouse between the groups treated with compound **3c** and β-carotene, and the control group was evident ($p<0.05$, using the Student's *t*-test).

5. Conclusions

Shea butter has become a very popular ingredient in cosmetics and personal care applications due to its good emolliency and moisturizing properties. While these properties are due to shea butter's characteristic fatty acid composition, many other medicinal properties are related to its non-glyceridic lipid constituents, primarily the triterpene esters. The triterpene ester fraction of shea butter is consisted of the cinnamic and acetic acid esters of α-amyrin, β-amyrin, lupeol, and butyrospermol. These triterpene esters have been proved to exhibit a large variety of biological activities, especially of anti-inflammatory and protease-inhibiting activities, as well as chemopreventive properties in chemical carcinogenesis. The triterpene esters of shea butter have, thus, wide range of possibilities, especially in skin-care but perhaps also in the field of functional foods.

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