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THE BARIATRIC IMPROVEMENT AND TOXICTY OF BOTANICAL OILS FOR SKIN CARE

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Abstract-Botanical oils are increasingly used in traditional and cosmetic applications, but also as dietary supplements and pharmaceutical-grade enhancers for bariatric surgery improvement. The focus of this review is to evaluate the existing knowledge regarding the differential biological and toxicological effects of major bioactive constituents of plant oils, and to correlate them to the compositional changes in their fatty acid profiles. Multiple skin diseases that result in depletion or disturbance of skin lipids may require tailored mixtures of several botanical oils to simultaneously maintain natural skin-barrier function, promote repair and regeneration of wounded tissues, and achieve corrective modulation of immune disorders. Furthermore, as bioactive constituents of botanical oils enter the human body by oral or topical application and often accumulate in measurable blood concentrations, there is a need for monitoring their hazardous effects to reduce the possible over-added toxicity and promote maximal normal tissue sparing.

Keywords:- Bariatric surgery improvement, Botanical oils, fatty acid, toxicology

Introduction

Botanical oils are lipids or fats derived from one or more plant parts and can be broadly classified into fixed (vegetable) and essential (volatile) oils (Johnson, 1961). Four agricultural crops (oil palm, soybeans, rapeseed and sunflower) serve as major sources of fixed oil for nutritional applications (Dyer et al., 2008). These oils are a combination of saturated (no double bonds), monounsaturated (1 double bond), and polyunsaturated (2 or more double bonds) fatty acids of varying carbon chain length, attached to a glycerol molecule. Natural mono- (MUFA) and poly- (PUFA) unsaturated oils contain double bonds in less thermodynamically stable cis configuration that is prone to oxidative deterioration. As such, unsaturated oils can be industrially processed to remove (saturate) double bonds by partial hydrogenation. This process, however, introduces trans configuration into the fatty acids that has detrimental health effects (Ascherio, 2006). The degree of fatty acid saturation defines the fluidity, molecular packing, lipoxidative damage, and integrity of cell membranes (Pamplona, 2008). Essential oils are volatile and aromatic complexes of terpene, terpenoid, or phenylpropene origins that evaporate when exposed to heat. The characteristic flavor and aroma that they impart can directly attract pollinators, repel herbivores, and protect plants from biotic and abiotic environmental stresses (Unsicker et al., 2009). As different plants have evolved in diverse ecogeographical areas and developed unique repellent or bactericidal properties, the chemical constituents of essential oils show a much greater structural variety when compared to fixed oils (Bakkali et al., 2008). A single essential oil typically consists of 20-80 constituents of various concentrations, with 2-4 primary structures that largely define its physiochemical and biological properties (Islam et al., 2016). Terpenes (or hydrocarbons) consist of different number of isoprene units such as monoterpenes (C10), sesquiterpenes (C15), and diterpenes (C20), and represent over 90% of essential oil constituents in most plants (Tongnuanchan and Benjakul, 2014). The remaining components of essential oils are broadly identified within the groups of oxygenated (esters,

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aldehydes, ketones, alcohols, phenols, and oxides), aromatic, and sulfur- containing compounds (Dhifi et al., 2016).As complex bioactive constituents in fixed and essential oils coevolved to mediate plant- animal interactions, they likely contain functional, biologically relevant chemical spaces and pharmacophores that were selected to interact with animal and human cell targets (Sharifi-Rad et al., 2017). Botanical oils are widely used to prevent or ameliorate human diseases, especially in the form of topical applications to promote skin health, heal injuries and burns, decrease scarring, improve cosmetic outcomes, reduce social stigmatizing, and promote wellbeing (Vaughn et al., 2018). Plant oils can rapidly penetrate through the lipid structures of the skin and interact with the cell membrane proteins to induce their conformational modifications (Herman and Herman, 2015). Their unique physiochemical properties are utilized for natural enhancement of skin penetration during transdermal drug delivery (Edris, 2007). This review therefore summarizes recent evidence on utility of fixed and essential botanical oils for topical skin care, including maintenance of natural skin-barrier function, repair and regeneration of wounded tissues, and modulation of immune skin disorders. As bioactive constituents of fixed and essential oils achieve measurable blood concentrations following the oral absorption, skin penetration, or inhalation routes, there is a need for monitoring hazardous effects of these bioactive and their metabolites to reduce the possible over-added toxicity and promote maximal normal tissue sparing.

Structure and function of the skin

Skin functions to maintain temperature and hydration, while protecting the body from environmental injuries and microbial infections. Damaged skin allows entry of chemical irritants, microorganisms, and allergens that promote and amplify skin inflammatory and immune responses (Chen and DiPietro, 2017). Skin consists of two major structural layers, the epidermis with embedded sebaceous glands, hair follicles, and sweat glands (epithelium) and the dermis (a mixture of loose and dense connective tissues). While the epidermis consists predominantly of continuously replenished and shed, terminally differentiated keratinocytes, dermis is populated by a variety of cell types including fibroblasts, immune cells, and white adipocytes surrounded by the fibrillar collagen.

Basement membrane comprising type IV collagen and laminin separates these two layers and serves as an anchor for dermal papillae and the smooth muscles that control the hair follicle (Watt and Fujiwara, 2011). The epithelial layer is also colonized by neural crest-derived melanocytes responsible for melanin production (skin color and UV protection) and dendritic immune antigen-presenting cells (monocyte-derived Langerhans and thymus-derived T cells) that respond to injury or infection with the production of proinflammatory cytokines, thus maintaining the innate immune response of the skin (Ohteki and Koyasu, 2001). Upper epidermis (stratum corneum) also contains keratohyalin protein granules and lamellar lipid bodies that prevent water loss. The major proteins of stratum corneum are type I (acidic) and type II (basic) keratins, filaggrin (proteolytically cleaved to release the amino acids as a moisturizing factor), loricrin and involucrin (cross-linking factors), and small proline rich proteins (SPRs) (Wickett and Visscher, 2006). Main components of lamellar lipid bodies are ceramides (sphingolipids linked to long-chain fatty acids, 50%), cholesterol (25%), and free fatty acids (cleaved from keratinocyte membrane phospholipids, 15%) that maintain the acidic skin surface pH of 4.0-5.5 and the diversity of skin microbiome (Elias and Choi, 2005). Cosmetic- grade glycerin (a natural component of triglyceride lipids) and petroleum-distilled mineral oil (Baby Oil as branded by Johnson & Johnson) or petrolatum (Vaseline as branded by Unilever) are effective skin-conditioning agents that increase hydration and improve elasticity of the epidermis (Rawlings and Lombard, 2012).

The dermis contains stromal cells, with fibroblasts making up the major cell type.

Structural cells of the peripheral nervous, immune, vascular, and lymph systems either reside or temporarily migrate through the skin (Rognoni and Watt, 2018). Upper papillary (proliferative) and lower reticular (secretory) dermal layers are separated by the vascular plexus. Fibroblast residing in each layer are epigenetically modified to either proliferate or secrete extracellular matrix (ECM) (Collins et al., 2011).

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Type I, III and V collagens are the most abundant fibrillary ECM proteins. Additional fibril-associated collagens connect collagen I and III fibrils with decorin and perlecan proteoglycans (Reed and Iozzo, 2002). The elastic fiber network and glycosaminoglycans such as hyaluronic acid allow for functional interaction and capture of water to generate osmotic pressure responsible for skin turgor (Juhlin, 1997). As the dermis is critical in maintenance of healthy and repair of wounded skin by means of functional and nutritional support of the epidermis (Waller and Maibach, 2006), it often serves as a primary target for therapeutic and cosmetic interventions targeting collagen and elastin production, or cellular responses within the dermal tissue (Badenhorst et al., 2014).

Botanical oils for topical skin care

Inexpensive and readily available botanical oils are routinely used for topical skin applications. They may enhance skin function by forming a physical barrier, supplying fatty acids to different skin layers, activating peroxisome proliferator-activated receptor-α (PPAR-α) signaling, or decreasing cutaneous inflammation (De Luca and Valacchi, 2010). Chemical diversity found in botanical oils leads to a variety of pharmacological activities and modes of action depending on quantities and proportions of individual chemical constituents in these complex mixtures. In general, it appears high linoleic acid 18:2(n-6) containing botanical oils (i.e., sunflower) are more beneficial to skin health (Hanley et al., 1998) when compared to the high oleic acid 18:1(n-9) counterparts (Jiang et al., 2000). Therefore, different ratios of individual fatty acids present in botanical oils often result in opposite, either beneficial or detrimental, effects on epidermal barrier function and comedogenicity and merit detailed inquiry (Darmstadt et al., 2002).

Results

Lipids of healthy skin

Skin surface lipids derived from the epidermis and sebaceous glands are found in decreasing order on scalp > face > back > chest > abdomen > arms > legs > palms and soles. The latter do not contain sebaceous glands but receive small amounts of carryover lipids from other areas of the body (Downing and Strauss, 1974). Human sebaceous glands are a unique source of wax esters and squalene (Table 1.1). The composition of human skin lipids also differs from that of other mammals by higher content of triacylglycerols and free fatty acids (Cheng and Russell, 2004). Fatty acids naturally present in human stratum corneum are mostly saturated docosanoic acid 22:0, lignoceric acid 24:0, and hexacosanoic acid 26:0 that are often branched, methylated, and/or hydroxylated, although smaller quantities of oleic acid 18:1(n-9) and linoleic acid 18:2(n-6) have been also reported (Bouwstra and Honeywell-Nguyen, 2002; Vicanová et al., 1997). The perceived oiliness of the skin, however, does not depend on total surface lipids nor the proportion of free fatty acids, but rather correlates with the larger ratios of unsaturated fatty acids and wax esters in the sebum. As the most abundant saturated fatty acid in human sebum, palmitic acid 16:0 is metabolized by both fatty acid desaturase 2 (FADS2) and stearoyl-CoA (SCD) to sapienic acid 16:1(n-10) and sebaleic acid 18:2(n-10). Desaturation of stearic acid 18:0 by the SCD enzyme also results in accumulation of oleic acid 18:1(n-9) in human sebum (Park et al., 2016). Small amounts of two essential fatty acids, linoleic acid 18:2(n-6) and α-linolenic acid 18:3(n-3), as well as a conditionally essential arachidonic acid 20:4(n-6) that becomes essential if a deficiency in linoleic acid develops, are also found in human sebum (Table 1.2).The extracutaneous traffic of lipids into the epidermis plays a significant role in permeability barrier formation. Dietary fatty acids (Reynolds et al., 1978), sterols (Bhattacharyya et al., 1983), and glucosylceramides (Tsuji et al., 2006) traffic through the extracutaneous tissues to contribute to the epidermal lipid pool. Antimicrobial lauric acid 12:0 and sapienic acid 16:1(n- 10), antifungal caprylic acid 8:0 and capric acid 10:0, and antioxidant vitamin E are delivered to the skin surface to naturally reduce oxidative damage and provide basic antimicrobial defenses (Fischer et al., 2014). The epidermis also lacks d6 and d5 desaturase activity and imports arachidonic acid 20:4(n-6) from the extra epidermal sites (Chapkin and Ziboh, 1984). Finally, essential fatty acids critical for the efficient structure and function of the skin must be also delivered from the diet and incorporated into ceramides (Kendall et al., 2017) (Table 1.3).Deficiency

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of essential fatty acids leads to scaliness of the skin and an increased water consumption, mainly due to disruption of the water permeability barrier and an increase in trans- epidermal water loss (Basnayake and Sinclair, 1956). Linoleic acid 18:2(n-6) is also selectively targeted for β-oxidation by the sebaceous cells as a unique energy source for their function (Pappas et al., 2002), while application of nicotinamide (Tanno et al., 2000) and L-lactic acid (Rawlings et al., 1996) produce similar effects.

Lipids in skin diseases and wound healing

External damage by physical (mechanical injury, UV-irradiation, heat, excessive moisture, pressure, or friction), chemical (solvents, irritants, or allergens) or microbial assaults (bacteria, fungi, or viruses) results in injuries in the form of wounds, burns, calluses, or scars. Dry, cracked, or fissured skin is often presented with major changes in its lipid profile, resulting in excessive water loss and direct exposure to allergens and microbes that further irritate and inflame skin.Injured skin heals in four overlapping stages that include hemostasis (blood clot formation), inflammation (infiltration of immune cells), proliferation (angiogenesis, granulation, epithelialization, and extracellular matrix (ECM) remodeling by proliferating and migrating fibroblasts and keratinocytes), and maturation (wound contraction and resolution of inflammation) of skin layers. When these processes are disturbed due to an underlying genetic or clinical disorder, the healing pathology results in an ulcerative chronic wound, a hypertrophic scar, or a keloid (Eming et al., 2014).Proinflammatory cytokines and lipid mediators synthesized and released by neutrophils at the site of the wound must be tightly regulated and resolved, otherwise leading to persistent inflammation and uncontrolled proliferation and collagen secretion by skin cells. This pathology directly interferes with contraction of the wound that comprises of proliferation and migration of keratinocytes into the wounded area, and differentiation of fibroblasts into myofibroblasts (Leoni et al., 2015). While some organisms are fully capable of repairing and regenerating injured tissues, in humans this process occurs only in fetal skin and is partially preserved in the gut epithelium and hematopoietic system (Lorenz et al., 1992).

Direct supplementation or replacement of skin lipids can be explored in the prevention or treatment of skin pathologies. Lipid-based barrier repair creams like EpiCeram® by PromiusPharma (ceramides, cholesterol, and free fatty acids, 3:1:1), Lipobase by AstellasPharma (sorbitanoleate, carnauba wax, ceramide 3, oleic acid, palmitic acid, and cholesterol), CeraVe® by L'Oréal (ceramides 6II, 3, 1, phytosphingosine, hyaluronic acid), Triceram® by Dentaurum (lanolin, ceramides, soybean sterol, linoleic acid, hyaluronic acid), Atopiclair® by Alliance Pharma (glycyrrhetinic acid 2%, hyaluronic acid, grapevine extract, telmesteine, shea butter), andMimyX® by Stiefel Laboratories(acannabinoid agonist N-palmitoylethanolamine, olive oil, palm glycerides, vegetable oil, squalene) have been cleared for marketing by the Food and Drug Administration (FDA) as 510(k) medical devices with no defined active ingredients. The synthetic sebum mixture consisting of 45% triglycerides, 25% wax monoesters (jojoba oil), 17% fatty acids, and 12% squalene has been also proposed (Wertz, 2009).

Additional mixtures of unsaturated fatty acids that modulate skin proliferative and immune responses may also promote wound closure by direct effects on skin inflammation and permeability (Cardoso et al., 2004). Majority of skin diseases also present with variation or depletion of the major skin lipids as reported for atopic dermatitis (reduced ceramides and C20-26 fatty acids), psoriasis (reduced ceramides), type 2 Gaucher disease (increased glucosylceramides), acne vulgaris (reduced sphingolipids), atopic eczema (increased short-chain ceramides), and aged dry skin (reduced ceramides) (Sahle et al., 2015).

Pathological microbial infection of the skin also alters skin lipid profiles as shown for Propionibacterium infections observed in acne (Saint-Leger et al., 1986) and Pityrosporum folliculitis infections associated with seborrheic dermatitis (Bergbrant et al., 1991). Stress and other physiological factors often exacerbate skin conditions and healing processes by changes in neurohormone and steroid hormone

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levels that directly affect blood flow, metabolic and immune status of the skin, and function of hair follicles (Hunter et al., 2015).

Table 1.1. Lipid class composition of various skin sites (% total lipid).

Lipid class[1]	Human skin surface					Other mammals	
	Sebum	**Scalp**	**Face**	**Forehead**	**Back**	**Mouse**	**Sheep**
Basic analysis							
Squalene	12	12-14	12	12	12-16	--	--
Wax esters	26	21-23	23	26	22-23	5	10
Fatty acids (total)	58	56-65	65	62	61-66	6	--
Extended analysis							
Squalene	12	12-13	12	12	11-16	--	--
Wax esters	26	20-22	23	25	22	5	10
Triglycerides (bound)	--	29-32	35	43	**43-46**	6	--
Fatty acids (free)	--	**29-33**	**27**	16	16	--	--
Sterol esters	3	3	3	2	3	10	46
Sterols	2	2	1	1	1-2	13	12
Diesters	--	--	--	--	--	**65**	**21**
Hydrocarbons	--	1	1	--	1-2	--	--

1 Adapted with modifications from (Downing and Strauss, 1974).

Table 1.2. Fatty acid composition from various body sites (%).

Fatty acid	Scalp[1]	Sole[2]		Forearm[3]			Erythrocyte[4]	Plasma[5]
	Surface	**Live**	**SC**	**N**	**PU**	**PI**	**Membrane**	**Lipids (µmol/L)**
Lauric	0.1-1.9	*t*-0.1	0.2	nr	nr	nr	--	--
Myristic	4-8	1.4-2.5	1.1-1.9	1.1	1.5	0.8	--	16.2-325.7
Palmitic	**18-29**	40.6-48.6	24.6-25.1	14	13.9	12	**23-26**	285.4-4064.5
Palmitoleic	--	--	--	2.3	2.6	3.9	--	27.7-555.9
Sapienic		--	--				nr	--
Stearic	2-5	33.9-34.8	18.6-19.3	11.1	10.9	10.1	**14-21**	110.2-1013.7
Oleic	**11-19**	86.2-86.0	83.6-80.0	15.1	13.7	16.8	**14-19**	178.7-3210.5
Linoleic	1-2	96.7	96.1-96.7	21.5	21	15.7	11-12	279.7-4970.5
α-Linolenic	--	--	--	nr	nr	nr	0.1-0.3	--
Arachidic	0.2-1.8	1.0-1.2	3.9-4.8	1.6	1.7	1.8	--	*t*-29.8
Mead	--	--	--	1.5	1.4	1.4	1-2	--
Arachidonic	--	--	--	6.2	6.5	5.0	**14-16**	42.7-882.8
EPA	--	--	--	nr	nr	nr	1	4.4-215.4
Behenic	0.1-1.2	1.2-2.9	7.5-7.8	2.7	2.9	1.4	--	*t*-39.0
DPA	--	--	--	nr	nr	nr	2-3	*t*-88.5
DHA	--	--	--	nr	nr	nr	4-7	7.2-237.5
Lignoceric	0.3-1.2	1.0-3.7		10	10.5	4.2	--	*t*-15.7

Adapted with modifications from: 1 scalp skin surface (Koch et al., 1982); 2 sole skin epidermis (Ansari et al., 1970); 3 normal (N), uninvolved (PU), and involved (PI) psoriatic forearm skin (Chapkin et al., 1986); 4 erythrocyte membrane (Akinyemi et al., 2017); 5 plasma lipids (Abdelmagid et al., 2015).

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Table 1.3. Compositions of skin bound fatty acids by various lipid class (%).

Fatty acid[1]	Lipid number	Free	Ester-linked		Long-chain bases		Amide-linked		Cholesteryl esters
			Cer 1	Cer 6I	Cer 2	Cer 3	Cer 2	Cer 6I	
Myristic	14:0	0.8	2	--	--	--	0.2	--	--
Myristoleic	14:1	--	2.3	--	--	--	--	--	--
Palmitic	16:0	7.4	**18.0**	**30.2**	0.6	3.1	3.6	1.7	9.9
Palmitoleic	16:1(n-7)	0.7	4.8	--	0.7	--	--	--	3.0
Margaric	17:0	0.8	1.2	--	2.1	8.0	0.4	1.4	1.1
Stearic	18:0	9.1	9.1	4.8	11.7	12.8	4.4	9.9	4.6
Oleic	18:1(n-9)	5.7	11.6	--	**35.6**	--	--	--	**68.2**
Linoleic	18:2(n-6)	1.4	**20.7**	--	--	--	--	--	--
Nonadecylic	19:0	1.1	0.1	--	1.6	16.0	1.0	1.7	--
Arachidic	20:0	5.9	3.5	3.6	6.5	14.4	3.8	1.6	6.6
Heneicosylic	21:0	1.9	0.2	3.1	20.7	10.1	1.2	1.4	1.1
Behenic	22:0	15.3	4.8	3.3	1.5	21.4	8.7	1.7	1.3
Tricosylic	23:0	6.2	--	--	--	2.1	5.7	1.3	1.0
Lignoceric	24:0	**26.9**	**8.4**	**20.2**	10.5	5.1	**30.4**	**27.9**	1.4
Pentacosylic	25:0	5	1.8	6.3	--	2.1	7.8	11.3	1.1
Cerotic	26:0	8.5	4.1	18.5	--	4.9	**18.3**	**34.8**	1.1

1 Adapted with modifications from (Wertz et al., 1987).

Table 1.4. Fatty acid composition of botanical oils sorted by their major constituents.

Common name and part used ***Latin name***	Coconut kernel *Cocosnucifera*	Palm kernel *Elaeisguineensis*	Laurel fruit *Laurus nobilis*	Babassu seed *Attaleas peciosa*	Murumuru seed *Astrocaryum murumuru*	Nutmeg nut *Myristicafragrans*
Saturation ratio	93:6:2	82:16:3	48:37:15	80:17:3	90:7:3	88:8:2
% Major fatty acid composition	Lauric 48% Myristic 19% Palmitic 9% Caprylic 8%	Lauric 46% Myristic 18% Oleic 16% Palmitic 8%	Lauric 43% Oleic 37% Linoleic 15% Palmitic 5%	Lauric 34% Myristic 19% Oleic 17% Palmitic 11%	Lauric 49% Myristic 30% Palmitic 7% Oleic 7%	Myristic 79% Oleic 7% Palmitic 6% Lauric 2%

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Common name and part used ***Latin name***	Ucuhuba seed *Virolasurinamensis*	Palm pulp *Elaeisguineensis*	Coffee bean *Coffea arabica*	Buckthorn fruit *Hippophaerhamnoides*	Kokum seed *Garcinia indica*	Sal fruit *Shorearobusta*
Saturation ratio	93:4:1	50:39:11	44:7:49	28:47:25	61:38:1	64:37:1
% Major fatty acid composition	Myristic 71% Lauric 16% Palmitic 4% Oleic 4%	Palmitic 44% Oleic 39% Linoleic 10% Stearic 4%	Linoleic 48% Palmitic 33% Stearic 7% Oleic 7%	Palmitic 27% Palmitoleic 25% Linoleic 16% Oleic 15%	Stearic 59% Oleic 38% Palmitic 2% Linoleic 1%	Stearic 48% Oleic 37% Arachidic 8% Palmitic 7%

Common name and part used ***Latin name***	Mango seed *Mangifera indica*	Shea nut *Vitellaria paradoxa*	Cupuacu bean *Theobromagrandiflorum*	Cocoa bean *Theobroma cacao*	Kusum seed *Schleicheraoleosa*	Tallow fat *Sevum*
Saturation ratio	49:45:3	45:48:6	45:48:6	61:35:3	35:62:1	58:36:3
% Major fatty acid composition	Oleic 45% Stearic 42% Palmitic 7% Linoleic 3%	Oleic 48% Stearic 40% Palmitic 5% Linoleic 6%	Oleic 39% Stearic 38% Palmitic 11% Arachidic 8%	Oleic 35% Stearic 33% Palmitic 28% Linoleic 3%	Oleic 43% Arachidic 21% Gadoleic 15% Palmitic 8%	Oleic 47% Palmitic 26% Stearic 14% Myristic 3%

Table 1.4 (continued).

Common name and part used ***Latin name***	Rambutan seed *Nepheliumlappaceum*	Peanut bean *Arachishypogaea*	Ben seed *Moringaoleifera*	Rice bran *Orysa sativa*	Brazil nut *Bertholletiaexcelsa*	Oat seed *Avena sativa*
Saturation ratio	40:57:1	11:56:26	14:71:2	26:38:34	24:39:36	17:40:39
% Major fatty acid composition	Oleic 51% Stearic 15% Arachidic 15% Palmitic 8%	Oleic 56% Linoleic 26% Palmitic 8% Stearic 3%	Oleic 66% Palmitic 9% Behenic 4% Stearic 7%	Oleic 38% Linoleic 34% Palmitic 22% Stearic 3%	Oleic 39% Linoleic 36% Palmitic 13% Stearic 11%	Oleic 40% Linoleic 39% Palmitic 15% Stearic 2%

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Common name and part used ***Latin name***	Sesame seed *Sesamumindicum*	Jatropha seed *Jatropha curcas*	Argan fruit *Arganiaspinosa*	Neem seed *Azadirachtaindica*	Pecan nut *Caryaillinoinensis*	Avocado seed *Perseaamericana*
Saturation ratio	15:40:43	16:44:34	15:46:34	39:46:12	9:50:39	22:58:12
% Major fatty acid composition	Linoleic 43% Oleic 40% Palmitic 10% Stearic 5%	Oleic 44% Linoleic 34% Palmitic 9% Stearic 7%	Oleic 46% Linoleic 34% Palmitic 14% Linolenic 1%	Oleic 46% Palmitic 21% Stearic 16% Linoleic 12%	Oleic 50% Linoleic 39% Palmitic 7% Linolenic 2%	Oleic 58% Palmitic 20% Linoleic 12% Palmitoleic 8%
Common name and part used ***Latin name***	Macadamia nut *Macadamia Integrifolia*	Canola seed *Brassica napus*	Pistachio nut *Pistacia vera*	Peach kernel *Prunus persica*	Apricot kernel *Prunusar meniaca*	Plum kernel *Prunusdo mestica*
Saturation ratio	14:79:2	6:61:21	12:63:25	8:65:25	6:66:27	3:68:23
% Major fatty acid composition	Oleic 59% Palmitoleic 19% Palmitic 9% Stearic 5%	Oleic 61% Linoleic 21% Linolenic 9% Palmitic 4%	Oleic 63% Linoleic 25% Palmitic 11%	Oleic 65% Linoleic 25% Palmitic 6% Stearic 2%	Oleic 66% Linoleic 27% Palmitic 6%	Oleic 68% Linoleic 23% Palmitic 3%

Table 1.4 (continued).

Common name and part used ***Latin name***	Olive fruit *Oleaeuropaea*	Almond nut *Prunus dulcis*	Buriti fruit *Mauritiaflexiosa*	Hazel nut *Corylusavellana*	Marula fruit *Sclerocaryabirrea*	Papaya seed *Carica papaya*
Saturation ratio	17:69:12	7:71:18	19:71:7	8:75:10	18:75:4	18:76:3
% Major fatty acid composition	Oleic 69% Palmitic 14% Linoleic 12% Stearic 3%	Oleic 71% Linoleic 18% Palmitic 7%	Oleic 71% Palmitic 17% Linoleic 7% Stearic 2%	Oleic 75% Linoleic 10% Palmitic 5% Stearic 3%	Oleic 75% Palmitic 11% Stearic 7% Linoleic 4%	Oleic 76% Palmitic 13% Stearic 5% Linoleic 3%

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Common name and part used ***Latin name***	Tea seed *Camellia sinensis*	Pataua fruit *Oenocarpusbataua*	Carrot seed *Daucus carota*	Camelina seed *Camelina sativa*	Jojoba seed *Simmondsiachinensis*	Mustard seed *Brassica napus*
Saturation ratio	11:77:8	17:78:3	4:80:13	8:77:10	8:92:0	12:60:21
% Major fatty acid composition	Oleic 77% Palmitic 9% Linoleic 8% Stearic 2%	Oleic 78% Palmitic 13% Stearic 4% Linoleic 3%	Oleic 80% Linoleic 13% Palmitic 4%	Gondoic 33% Oleic 14% Linolenic 10% Linoleic 9%	Gadoleic 75% Erucic 15% Oleic 10% Nervonic 3%	Erucic 42% Linoleic 15% Oleic 12% Linolenic 6%

Common name and part used ***Latin name***	Buckthorn seed *Hippophaerhamnoides*	Cranberry seed *Vacciniummacrocarpon*	Borage seed *Boragoofficinalis*	Black currant seed *Ribesnigrum*	Rosehip fruit *Rosa canina*	Pumpkin seed *Cucurbitapepo*
Saturation ratio	10:24:66	8:23:69	14:20:66	8:13:76	6:12:82	19:33:48
% Major fatty acid composition	Linoleic 36% Linolenic 28% Oleic 24% Palmitic 7%	Linoleic 37% Linolenic 32% Oleic 23% Palmitic 6%	Linoleic 43% gLinolenic 24% Oleic 20% Palmitic 10%	Linoleic 46% Linolenic 29% gLinolenic 19% Oleic 13%	Linoleic 46% Linolenic 31% Oleic 12% Palmitic 4%	Linoleic 50% Oleic 33% Palmitic 11% Stearic 8%

Table 1.4 (continued).

Common name and part used ***Latin name***	Soybean seed *Glycine max*	Corn seed *Zeamays*	Cotton seed *Gossypiumarboreum*	Raspberry seed *Rubusidaeus*	Hemp seed *Cannabis sativa*	Black cumin seed *Nigella sativa*
Saturation ratio	18:75:4	14:32:51	26:18:52	3:13:81	8:12:57	16:22:60
% Major fatty acid composition	Linoleic 50% Oleic 24% Palmitic 11% Linolenic 8%	Linoleic 51% Oleic 32% Palmitic 12% Stearic 2%	Linoleic 52% Oleic 18% Palmitic 13% Stearic 12%	Linoleic 55% Linolenic 26% Oleic 13% Palmitic 3%	Linoleic 57% Linolenic 21% Oleic 12% Palmitic 6%	Linoleic 60% Oleic 22% Palmitic 13% Stearic 3%

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Common name and part used ***Latin name***	Walnut seed *Juglans Regia*	Watermelon seed *Citrullusla natus*	Grape seed *Vitisvin ifera*	Poppy seed *Papaverso mniferum*	Passion seed *Passiflora edulis*	Sunflower seed *Helianthus annuus*
Saturation ratio	9:18:60	21:18:60	12:20:68	12:17:69	13:15:70	11:16:70
% Major fatty acid composition	Linoleic 60% Oleic 18% Palmitic 7% Stearic 2%	Linoleic 60% Oleic 18% Palmitic 11% Stearic 10%	Linoleic 68% Oleic 20% Palmitic 8% Stearic 4%	Linoleic 69% Oleic 17% Palmitic 10% Stearic 2%	Linoleic 70% Oleic 15% Palmitic 10% Stearic 3%	Linoleic 70% Oleic 16% Palmitic 7% Stearic 4%

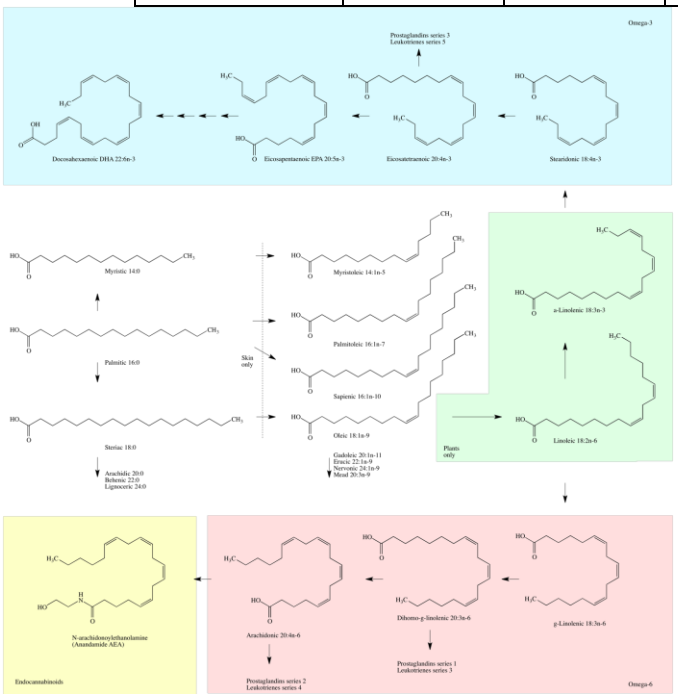

Figure 1.1 Schematic biosynthesis of MUFA and PUFA fatty acids.

Discussion

Botanical oils contain complex mixtures of both saturated and unsaturated fatty acids, typically esterified in the form of triglycerides, that act as powerful lipophilic solvents to selectively extract and accumulate nonpolar secondary metabolites produced by the source plants. Although data is limited, the most efficient solvent-like molecules in the fixed (oleic and linoleic acids) and essential (limonene) botanical oils are likely of low toxicity. However, these compounds may cause mild skin irritation, while their oxidation products may produce dermal sensitization in humans (DeWitt and Bebarta, 2004). The potentially detrimental effects may also vary with dose, form (water or fat soluble), and site of application (sebaceous gland, hair follicles), which will define absorption rates and accumulation of the bioactive compounds in various layers of the skin and systemic circulation.Most of botanical oils are generally well tolerated in adults, with occasional allergic skin reactions occurring, and are "generally recognized as safe" (GRAS) as a food (dietary supplement) by the U.S. Food and Drug Administration. This designation, however, does not require the manufacturer to prove the safety and efficacy of the product prior to marketing. Botanical oils produced by different manufacturers may also contain different ingredients that do not match the actual ingredients, or their amounts listed on the label. As such, botanical oils are considered as alternative (complimentary) strategies used to supplement the perceived failures and side effects of conventional medicines.

Site of application

Regional permeability of the human body is not uniform and is typically ranked as follows: scrotum > face/scalp > trunk/extremities > palm/sole > nail. Within those regions, further variations in stratum corneum thickness, the number of

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sebaceous glands, and hydration status can all affect absorption and metabolism of botanical oils and their bioactive components. Understanding the parameters that affect the permeability of the skin barrier is essential for achieving correct dosing and adherence regimens with a goal of reaching therapeutic targets within the local skin environment (ointment or cream) or systemic uptake via dermal capillary beds (Prausnitz and Langer, 2008). The use of botanical oils as vehicles for therapeutic drug delivery provides a wide variety of choices between achieving optimal drug potency and therapeutic effectiveness, as well as the risk of over-added toxicity, as the same drug may appear in different potency classes when formulated in different vehicles or applied to different target site (Williams and Barry, 2004).

Neonatal skin sensitization

Infant skin is susceptible to dryness and irritation from external factors, including topical skin care products not formulated for the infant's skin (Kuller, 2016). Topical products with adverse effects on skin barrier function, however, carry a potential to develop atopic dermatitis or eczema (Danby et al., 2013). The practice of recommending and using topical oils for the prevention or treatment of baby dry skin or for massage, including the increased societal interest in natural interventions, often ignores the fact that specific topical oils may have an adverse effect on skin barrier function (Cooke et al., 2011). While oils with the lowest oleic acid content provide a lower risk of irritant contact dermatitis (Kuller, 2016), sunflower-based oils may also may retard postnatal skin barrier maturation in infants (Kanti et al., 2014). Skin ointments containing components of food origin also carry the risk of possible percutaneous sensitization to food proteins that may promote development of contact dermatitisand persistent eczema, as it was shown for almond oil (Guillet and Guillet, 2000).

Secondary metabolites and biological reactive intermediates

While most botanical oils can be considered safe, a few contain compounds that can be converted to biological reactive intermediates, causing toxicity (Llana-Ruiz-Cabello et al., 2015). Although health promoting effects of secondary metabolites coextracted into the botanical oils may be beneficial, they may also have potential toxic effects and local higher levels of exposure due to topical application. For example, rosemary oil has been demonstrated to induce lipid and protein oxidation at high doses (Estévez and Cava, 2006). High doses of the monoterpenoid phenols, carvacrol and thymol, increase the levels of malondialdehyde, resulting in membrane damage, and 8-hydroxy-deoxyguanosine, causing cell DNA damage (Ozkan and Erdogan, 2012). Eugenol present in clove oil can be oxidized to phenoxyl radicals that induce reactive oxygen species-mediated apoptosis in human cells (Yoo et al., 2005). Borage plant parts contain pyrrolizidine alkaloids that are toxic to the liver and lungs, and maybe coextracted into borage seed oil (Low Dog, 2009). Raw botanical oil materials often originate from different sources and storage timeframes, complicating comparisons of bioactive ingredients and lack of potentially toxic contaminants in them.

Genotoxicity and photosensitivity

Some of secondary metabolites coextracted with botanical oils may form genotoxic DNA adducts or activate detoxification enzymes, as it was shown for safrole and quinones in sassafras oil or epoxides found in pennyroyal oil (Dietz and Bolton, 2011). Other botanical oils and their constituents may exhibit a dual genotoxic and antigenotoxic effect, as it was shown for β-caryophyllene (Di Sotto et al., 2010). Adverse cutaneous responses to the combined action of the botanical oil or its bioactive constituent and UV radiation may cause phototoxic reactions that result in sunburn, edema, hyperpigmentation, photoaging and cancer (Gould et al., 1995). Some of these effects, however, may be beneficial in alleviating multiple symptoms of psoriasis, vitiligo, and cutaneous T-cell lymphoma (Bark et al., 2010).

Overdose in pregnant women and children

In rare instances, some commercially marketed hemp seed oils could lead to mild cannabinoid poisoning in children (Chinello et al., 2017) and pregnant women (Yang et al., 2017). While food-grade strains of hemp must contain less than 0.3% THC by weight (whole plant), hemp seeds or stems used to produce hemp oil may become contaminated by THC-rich trichomes of hemp flowers and thus acquire THC (Yang et al., 2017). Due to the polymorphic nature of cytochrome P450 enzymes that

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can be further affected by age, liver impairment, or potential drug interactions, people consuming hemp products may gradually accumulate THC due to its slow metabolism or relatively long half-life in the body, resulting in potentially higher concentrations (Watanabe et al., 2007).

Conclusions

Both topical and dietary interventions with botanical oils may produce different functional outcomes according to their phytochemical composition and the pathophysiological state of the target tissue. The depletion or disturbance of any of the skin lipid classes results in a rapid disruption of skin integrity and leads to a variety of structural (barrier), physiological (repair and regeneration), and pathological (inflammation) changes that allow further entry of microbial and chemical irritants and deterioration of the affected, aged or diseased skin. Replenishment of those lipids by direct replacement or enhancement of their in-situ production with botanical oils may restore skin function and reduce pathophysiological symptoms associated with the disease. Among inexpensive, widely available oils, sunflower oil high in the omega-6 linoleic acid, and flax or hemp oil enriched with the omega-3 α-linolenic acid, offer an attractive combination of enhanced metabolic and reduced inflammatory and comedogenic effects. On the other hand, application of olive oil with high oleic acid content is warranted when deep transdermal penetration is desired, and the target skin site can be further sealed off with the application of highly saturated coconut or shea butters. The presence of dissolved bioactive secondary metabolites that target a specific health outcome will further substantiate the use of a particular plant source within the group of botanical oils with similar physiochemical properties.To become established in clinical settings, the required mixtures and doses should be individually determined in randomized controlled trials that simultaneously monitor for hazardous effects of botanical oil supplementation to reduce the possible over-added toxicity associated with the interventions.

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