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Diabetic Skin
targeted soothing and support

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Diabetic skin: targeted soothing and support

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ABSTRACT

Excessive inflammatory response, remarkable dryness and disorganization of elastic fibers seem to be major causes of the impairment of cutaneous functions in diabetic skin. Among the molecular pathways causally associated with these aspects, non-enzymatic glycosylation (glycation) stands out as perhaps the single most effective mechanism.

This paper describes in detail some of the causes of cutaneous pathologies associated with diabetes and suggests cosmetic grade ingredients suitable to support the skin of subjects suffering from glycation-induced pathologies as well as for other pathologies encountered in diabetic skin, for which the link with glycation is not immediately obvious.

Introduction

Diabetes-linked pathologies (2014) afflict over 420 million people worldwide, up from 108 million in 1980 [(1)]. That is to say that one person out of sixteen is suffering from a diabetic pathology. In particular, the increase of diabetes in adults over 18 years of age is important and worrying (8.5% in 2014, up from 4.7% in 1980) and remarkable prevalence peaks, twice as much as the world average, are observed in the Eastern Mediterranean regions.

It is well known that the skin of diabetic patients presents specific pathological issues and needs careful and specifically targeted treatments. As a matter of fact, soothing diabetic skin is relevant both from the esthetic as well as from the functional point of view. Successful treatments can be delivered whenever the diagnostics allows an accurate classification of the different manifestations of the disease. As usual, prevention is to be preferred to the repairing intervention, particularly so because of the hyper-reactivity of the cutaneous structures involved in the process.

In this paper we will not discuss the systemic consequences of the diabetic pathology. We will address only the most likely cutaneous aspects of diabetes seen as the outcome of a generalized meiopragic status. In spite of our focus, we feel that it is not unnecessary to maintain that the primary pathology needs to be treated with the appropriate medical therapies and must be accompanied by fitting physical activities under medical control and by an accurate organization of one's lifestyle to avoid behaviors that might worsen the effects of the disease. As a matter of fact, in diabetic patients glucose metabolism is altered and this leads to non-enzymatic glycosylation (protein glycation), impairing of enzymatic activities, micro-angiopathy, atherosclerosis, neuronal degeneration and life-threatening necrotic phenomena.

The most frequently observed cutaneous modification in diabetic patients is accelerated skin aging, ranking from moderate to severe. This is likely to be the consequence of a disorganization of the dermal extracellular matrix, affecting mainly collagen and fibronectin. This is the consequence of the increased activities of Matrix Metallo Proteinases MMP-1 (collagenase) and MMP-2 (gelatinase A), proteolytic enzymes of Collagen, and Lysil Oxydase (LOX) that catalyzes the cross-linking of collagen microfibrils. Currently it is believed that the increased activity of these enzymes be the consequence of the impaired glucose metabolism [(2)].

This is in keeping with what is observed in diabetic skin: fragmentation and disorganization of the scaffold of the dermal structures with concomitant loss of the typical dermal characteristics such as resistance, resilience and elasticity. The above epidermis layer does not present with direct structural damage visible in microscopy. Yet it is affected by the damages and by a de-structured dermis in terms of slowed down or lack of hydration functionalization (adequate transport of water bound to the epidermis) and of the ongoing structural and nutritional support needed to correctly perform the keratinization process. The consequence of this is the aged and withered aspect of the epidermis [(3)].

In this paper we shall describe the cutaneous phenomenology in the diabetic and we shall discuss topical interventions aimed at improving diabetic skin. Appropriate interventions will consist of excipients and active ingredients that are non-toxic to cutaneous enzymes, non-oxidant, non-irritant, with chemical affinities to sebum and that are devoid of negative effects on the biochemical and physiological pathways pertaining to skin structure and functions.

Diabetic Skin

Cutaneous manifestations are known to accompany both insulin-dependent and insulin-independent diabetes mellitus. Often, the clinical observation of one of these manifestations can be of help for the diagnosis of diabetes. These manifestations can be classified into ten major categories[4]. These categories are: Necrobiosis lipoidica diabeticorum, Acanthosis nigricans, Diabetic dermopathy, Diabetic bullae, Yellow skin and nails, Diabetic ulcers, Diabetic cutaneous infections, Perforating dermatosis, Eruptive Xanthomas, and a broader category Other Dermatoses. The etiology of these manifestations is unknown and no established therapy is at hand there are no commonly accepted medical therapies to date.

In recent years, the hypothesis has been put forward that the accumulation of advanced glycation endproducts (AGE) might play a major role in the onset of the pathology of diabetic complications[5, 6].

AGEs are the result of the non-enzymatic glycation, i.e. of the "spontaneous" binding of sugar molecules to free amino groups in proteins. Because of the elevated levels of serum glucose, the rate of formation of AGE can be expected to be larger in diabetic patients than in healthy individuals. The physiological consequences of this binding are manifold.

On the one hand, glycated proteins could have impaired enzymatic activities, i.e. when the glycation occurs in the active site, the enzyme might not be able to perform its catalytic activities at the normal rate. On the other hand, glycation can affect the structural role of fibers by introducing steric distortions in otherwise well-organized structures, and the extracellular matrix might lose its structural functions and viscoelastic properties. Last but not least, glycated proteins acquire a pro-inflammatory status [7] and therefore trigger the recruitment of immune cells from the blood vessels, the consequent oxidative steps and the degradation of dermal elastic fibers by activated matrix metallo-proteinases and myeloperoxidases and are associated to an increased rate of skin aging [8,9].

Glycation phenomena in diabetic skin have been recently summarized [10] and the effect of protein glycation and of AGE on skin aging is being consistently confirmed [11].

The phenomena that can be expected to arise because of accumulated AGE are in keeping with the observed large disorganized collagen bundles in the thickened dermis of diabetic scleroderma as well as with the destruction of the anchoring fibrils and the cleavage of the lamina lucida observed in diabetic bullae.

Last but not least, AGE accumulation can account for both the thickening of the vascular walls and the longitudinal fraying and splitting of elastic fibers and of their disruption into small fragments in the skin of diabetics [12, 13] that are reminiscent of the structural changes observed during skin aging[14].

The accumulation of AGE in diabetic skin has been reported since the end of last century [15, 16]. The formation of pentosidine, an AGE, most likely the first of chemically characterized AGEs, has been shown to correlate with the severity of the diabetic complications in individuals with insulin dependent diabetes mellitus for more than 17 years[17].

It was subsequently pointed out that skin auto-fluorescence correlates quantitatively with the accumulation of AGE[18] and several clinical observations have been reported indicating a correlation between high skin auto-fluorescence and the severity of diabetes-accompanying manifestations such as retinopathy[19] or nephropathy[20] or diastolic functions[21].

These observations seem to point out a causative relationship between the presence of AGE in the skin and the manifestations in diabetes.

The observation that injections of Streptozotocin can induce diabetes in rats[22] oopened a path to the experimental exploration of the mechanisms involved in the onset of the cutaneous manifestations that accompany diabetes and to test the hypothesis that the accumulation of AGE is causally linked to the appearance of these manifestations, or of part of them. Indeed, it had been reported that the accelerated accumulation of AGE in diabetic rats was prevented by oral administration of curcumin, as was the formation of cross links in the collagen of the tail tendon[23].

Studies on endogenous skin lesion in artificially diabetic rats show that 12 weeks after receiving intraperitoneal streptozotocin, male Sprague Dawley rats exhibited decrease of skin thickness, disappearance of the multilayer skin epithelium structure, degenerescence of skin collagen fibers and an increase in infiltration of inflammatory cells[24].

This seems to indicate that the formation of AGE is a crucial mechanism of diabetes-induced early-stage endogenous skin damage since it was known that treatment with insulin improved latent skin lesions in Streptozotocin-induced diabetic rats[25].

It thus appears that diabetic skin is subjected to an accelerated accumulation of AGE, and that this triggers the mechanisms leading to the cutaneous manifestations associated to diabetes. It seems therefore plausible to try and treat diabetic skin with specific soothing agents able to reduce the rate of formation and accumulation of AGE, as well as to improve the loss of elasticity and of moisture that are observed in this pathology.

Possible effective interventions

AGE occur in different tissues of the human body. The ones occurring within blood vessels and arteries have been investigated with particular emphasis because of their potential harmful role in the onset of atherosclerosis, with the aim of finding drugs able to undo the AGE and restore sclerotic arteries to restore a functional state. As of today, there is no pharmacologically acceptable drug treatment capable of removing AGE from the arterial walls.

To preserve and prolong health in the organs of the human body, and particularly of the skin, against the threat of glycation it is therefore necessary to devise a multi-prong strategy, to avoid the onset of the mechanisms leading to the consequences described above. This strategy could consist of topical treatments to prevent the formation of Schiff bases between free sugars and free amino-groups of skin proteins, and their conversion to Amadori products and eventually to AGE, together with anti-inflammatory substances, agents able to stimulate the synthesis of the Natural Moisturizing Factor (NMF) and the synthesis of the collagens connected with the epidermis basement membrane.

A panoply of substances are known to hinder the process of glycation. Their mechanisms of action can be as different as the scavenging of reactive Oxygen species, the chelation of transition metals, the reduction of the concentration of free sugars, the destabilization of the Schiff bases etc. Several vegetal derivatives inhibit the glycation of proteins in vitro and in vivo [26] and oral curcumin was shown to have such an activity in diabetic rats[23]. A new candidate is LR-9, a methylene bisphenylureido derived compound, has been brought to the attention of the scientific community. It scavenges dicarbonyl intermediates and chelates transition metals that catalyze the production of AGE. It inhibits AGE formation when orally administered to rats[27]. Another possible route to reduce the rate of glycation could be the topical application of oligo-peptides with free amino groups (such as oligo-lysine) that could bind free sugar and therefore decrease the concentration of free sugar and therefore reduce the rate of glycation of skin proteins. Other ingredients are endowed with the capability of hindering or inhibiting the formation of AGE, such as the following, which can be successfully used for this purpose thanks to their remarkable anti-lipoperoxidant and bio-regulating actions:

Filagrinol®, a mixture of pollen extract and of unsaponifiable fractions from soybean oil, olive oil and wheat germ oil with a strong protective action against oxidation, Salycuminol®, a local cutaneous modulator of skin inflammatory response with antilipoperoxidant and keratoplastic action, Trioxene-LV®, an anti-free radicals and anti-lipid peroxidation ester of citric acid and ACS-AntiCytoStressor®, a fractional phytoderivative, physiological bioregulator of stress hormones.

Since AGE-carrying proteins have been shown to be pro-inflammatory[7] and since the micro-inflammatory pathway has been pointed out to be the single most efficient mechanism of skin aging[8,9], the topical application of anti-inflammatory substances is expected to alleviate the onset of the inflammation-derived cutaneous manifestation in diabetes. Appropriate inflammation response modulators can be formulated for topical treatments targeting the oversized inflammatory response met in diabetic skin, such as Salycuminol®.

Another aspect of the cutaneous manifestation in diabetes is the severe reduction of hydration. This could be the consequence of random glycation of the proteolytic enzymes whose action is necessary to the degradation of filaggrin and the consequent production of the Natural Moisturizing Factor. It is known that the topical application of Filagrinol®, (an activator of the production of filaggrin) has significant effects on the level of hydration and comfort of the skin[28].

Hydrating products such as Hyaluramine-S®, an active precursor of hyaluronic acid improving deep moisturization, Dermonectin®, a low molecular weight precursor of fibronectin, cutaneous moisturization functionalizer and Filagrinol®, increasing the NMF-Natural Moisturizing Factor, that can be used at an advantage to temperate the dryness of diabetic skin.

Last but not least, it is known that diabetic skin has a brittle aspect (not to be confused with brittle diabetes!) and this may be the consequence of impaired structural functions of glycated collagen IV and VII in the basement membrane. The turnover of collagen IV and of collagen VII is quite rapid and the time for half renewal of Collagen VII in mice is of the order of a few weeks [29]. It is therefore not unreasonable to envision a strategy to increase the

rate of production of these collagens to accelerate the removal of glycated proteins and therefore reducing the time of persistence of a high concentration of glycated proteins in the basement membrane and concomitantly improve the elasticity of the epidermis. Enhancers of the synthesis of collagens can be used to partially restitute resilience and elasticity to diabetic skin.

Among these enhancers one finds Collagenon®, a low molecular weight precursor of collagen that supports, renews and restructures collagen structure and Aminoefaderma®, a balanced mixture of polyproline and E.F.A. with elasticizing and eutrophic effects that promotes the development of new collagen fibrils to improve skin resiliency,

In addition, it is known that diabetic skin is prone to bacterial infections, is sensitive to abrupt temperature changes, is hypersensitive to solar radiation and is subjected to photo-toxicity, and also that it is susceptible to hyperpigmentation and permissible for lipid accumulation, in particular of β-carotene. For these manifestations too, appropriate ingredients are at hand[30].

The table hereunder displays the dermal pathologies associated with diabetes, their anatomical sites of manifestation, the type of associated lesions, the structural and physiological consequences of the pathology as well as the suggested ingredients for topical application.

DERMOPATHY	PREEMINENT CUTANEOUS AREA	SKIN LESIONS	IMPLICATIONS	PREVENTION/ SUPPORT
Necrobiosis Lipoidica Diabeticorum (NLD)	Pretibial area dermis (women > men)	Small papules Large non-desquaming plaques with yellowish atrophic center and edematous rims	Thickening and degeneration of the collagen fibers due to fibroblasts premature aging and slowed down collagen turnover	Collagen and fibronectin active precursors • Collagenon • Dermonectin • Aminoefaderma • Salycuminol
Extensive Granuloma Annulare (GA)	Cutaneous surface, hands and arms back	Oval or circular lesions, from a few millimeters to a few centimeters in diameter, with raised labrum in hyperpigmented or hypopigmented central area	Upper layer of dermis	Slow-release antiseptics • Undelene • Undenat • Iodotrat
Hyperpigmented atrophic lesions	Distal pretibial surface	Small reddish papules with atrophic evolution in 1-2 weeks	Thinning of the epidermis, thickening of the vessels wall that encircle the papillary area of the dermis and the presence of lymphohistiocytic perivascular infiltrates and hemosiderin deposits, facilitated by exposure to hot and cold	Skin protection from sudden temperature leaps and from changes in humidity • Cetacene • PME • Salycuminol
Persistent erythema	Face and neck, occasionally hands and feet	Reddening with dotted areas of variable dimensions, even large sized	Reduced ability of the thickened arteriolar wall to respond with vasoconstriction to physiological stimuli	Protection from solar radiation and prevention of consequences from sun radiation exposure • Megasol System • Filagrinol • Salycuminol

DERMOPATHY	PREEMINENT CUTANEOUS AREA	SKIN LESIONS	IMPLICATIONS	PREVENTION/ SUPPORT
Folliculitis, foruncolosis, impetigo, erysipelas, subcutaneous inflammation, included district hydrolipodystrophy (cellulite)	Bust, arms, face or all body parts	Pustules or serious reddening	Skin bacterial infections due to Staphylococcus aureus and Streptococcus pyogenes (Beta-hemolytic Streptococcus)	• Salycuminol • Braxicina • Sebopessina • Lipoplastidine Solanum Lycopersicum • Filagrinol • ACS-AntiCytoStressor • Azamide • Iodotrat • Undelene • Undamide
Erythrasma	Armpit or between feet fingers	Reddish or brown exfoliating patches with atypical smell in case of sweating (Corynebacterium minutissimum) or periungueal tissue (Candida Albicans)	Cutaneous Bacterial infection due to Corynebacterium minutissimum or Candida Albicans	Intact cutaneous barrier Active precursors of hyaluronic acid, collagen, fibronectin and stimulators of filaggrin production • Hyaluramine-s • Collagenon • Dermonectin • Filagrinol • Undelene • Undamide
Acanthosis Nigricans	Bust, arms, neck, face, groin	Hyperpigmented areas, poorly outlined, mainly brown coloured with thickened and velvety surface	Hyperpigmentation often associated with insulin resistance	Chemical peeling with non-aggressive keratolytics and keratoplastic support • Keresine • Keratoplast • Azamide • Filagrinol
Xanthoma	All over the body, in particular face, limbs and buttocks	More or less prominent and delineated papillae or yellowish nodules	Cutaneous degeneration due to lipid accumulation, in particular of cholesterol and triglycerides	• Iodotrat • Zedomine • Salycuminol • Trioxene LV • Filagrinol • ACS-AntiCytoStressor
Carotenodermia	All over the body, in particular face and limbs	More or less homogeneous yellowing of the cutaneous surface	Carotene deposit due to difficulty in turning into retinol (vitamin A)	• Salycuminol • Trioxene LV • Filagrinol • ACS-AntiCytoStressor • Azamide

DERMOPATHY	PREEMINENT CUTANEOUS AREA	SKIN LESIONS	IMPLICATIONS	PREVENTION/ SUPPORT
Porphyria Cutanea Tarda (PCT)	All over the body, in particular face and limbs	Vesicles, bubbles and ulcers on the areas exposed to sun light	Phototoxic reaction caused by an increase in circulating porphyrins	Protection from solar radiation and prevention of consequences from sun radiation exposure • Megasol System • Filagrinol • Salycuminol
Haemochromatosis or bronze diabetes	All over the body Triad: diabetes mellitus - hepatic cirrhosis - hyperpigmentation	Brown hyperpigmentation	Epidermal deposit of hemosiderin associated with melanin transfer from melanocytes to keratinocytes	Pigmentation modulators, anti-lipoperoxidants and cell turnover normalizers • Azamide • Trioxene-LV • Salycuminol • Filagrinol
Scleredema Diabeticorum	Face, neck, chest, especially back	Swelling and tumefaction	Derma thickened by two to three times compared to normal. Collagen fiber bundles are considerably thickened and with a greater distance each other. Increased amount of acid mucopolysaccharides (hyaluronic acid), increase of bound water and edema.	• Salycuminol • Trioxene LV • ACS-AntiCytoStressor • Zedomine
Glycation	All over the body	Early roughness, more visible on the face and close to the joints, with possible slight hyperpigmentation	Increase of dermal proteins glycosylation and simultaneous increase of cross-linked bonds stabilizing their structure. Progressive increase in quantity of AGEs (Advanced Glycation End-products) and characteristic inflammatory condition	• Filagrinol • Salycuminol • Trioxene LV • ACS-AntiCytoStressor • Azamide

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WHAT IS RELATA TECHNICA?

Starting from the beginning of the human story numberless substances have been applied on the skin to favour wound healing, for the management of skin diseases, or simply and perhaps more often for cosmetic aims. In sharp contrast, only in recent years, and with a great delay as compared with otherfields of pharmacology, the study of the effects of chemicals on the skin moved from art to science; now it is soundly based on a rational approach. Regulatory Authorities classify substances and formulations to be applied on the skin in two distinct categories: drugs and cosmetics. This in order to prevent that harmful or extremely active chemicals, contained in cosmetic preparations, are used without medical control.

Nevertheless, all pharmacologists know that in its widest meaning drug is every substance capable of modifying cell function, and it is difficult to admit that chemicals used in cosmetic preparations are devoid of any influence on biochemical mechanisms of epidermal cells, in particular in the case of long-term treatments. Thus dermopharmacology and cosmetology are at least overlapping disciplines, and there is no doubt that the same methodology should be employed in both fields.

Over the years Relata Technica has achieved a wide readership; at present its aim is to broaden the journal to make it a truly comprehensive dermopharmacology research journal in which articles in all of the most interesting and exciting areas of modern skin care have their forum. As a consequence, Relata Technica should attract manuscripts concerning the pharmacokinetic behaviour and the pharmacodynamic activity of old and new chemicals used to control skin diseases or to prevent skin aging, as well as studies providing insights on which to base rational development of new compounds for medicinal or cosmetic use.

Investigations on the various aspects of the interaction of chemicals with the skin can be analysed by the use of several experimental models: the intact animal, fragments of surviving skin, keratinocytes cultures or the more sophisticated in vitro reconstructed human skin, subcellular fractions and pure enzyme systems. The end point examined in the study may be the macroscopic appearance of the skin, its histological, histochemical or ultrastructural features, and a biochemical or molecular marker.

An important aspect of dermopharmacology, and even more of cosmetology, is safety assessment. Therefore the journal will be also very interested in publishing the results of research dealing with the local and systemic tolerability of new compounds. In this respect, one of the major goals of Relata Technica is to promote studies on the use and validation of the so called alternative assays which should have the final aim of substituting, at least for cosmetics, the use of laboratory animals in the assessment of systemic toxicity, local irritant activity and, in a broader sense, of any possible adverse effect.

Finally, Relata Tecnica should be the natural publication outlet for manuscripts concerning the formulation of dermopharmaceutical and cosmetic preparations, and in particular for those which analyse the influence of the vehicle and other ingredients on the efficacy and tolerability of the active substance.

It is essential that the quality of papers published in Relata Technica be good and, on the other hand, it is important for the journal to process and publish papers promptly. We will make every possible effort to improve and shorten the review process, and I believe that Relata Technica will become a preeminent journal in the field of dermopharmacology.

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