ICADA präsentierte das Konzept "Abgrenzung kosmetischer und medizinischer Aknehaut-Pflege" bei der EU-Kommission und bekam breite Zustimmung auch von den Vertretern der Mitgliedsstaaten-Ministerien.

Nachfolgend das ICADA-Gutachten, das der EU-Kommission vorgestellt wurde:

Dermarcation of cosmetic and medicinal acne treatments
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Skin passes several well differentiable phases during lifetime characterized by unique physiological properties. These are phases like baby-skin, juvenile skin, adult skin, skin during pregnancy and old skin is. The skin properties and the skin phase in conjunction with juvenile skin are well perceived by the consumer, since the morphological situation, the skin-properties and the consumer classifi-cation are 1:1 proportional to each other.

Consumers are even able to differentiate the primary and secondary comedo-subcategories of juvenile skin just by optical and organoleptic skin-properties. Scientific explanation can proof, that the consumer-classification and the mor-phological conditions are 1:1 mirror-images. Since juvenile acne skin is only a temporary skin condition which has defined triggers and disappears together with these triggers contrary to diseases, which last even when the trigger

already has disappeared, juvenile acne-skin is just a skin-discomfort to certain degree and turns only into a disease in a late stage. We explain how to differ default from disease and why it is active consumer protection, to differ and to take care with cosmetic anti-acne products first, before the consumer is confronted with severe side effects of drugs like corticosteroids, antibiotics and isotretinoin, to treat just a skin discomfort.

Default sebum and keratin production

Sebum cells are produced in the sebaceous gland. The sebaceous gland basal layer consists of stem-cells. These stem cells produce daughter cells, which sepa-rate and migrate toward the sebaceous gland outlet, while the stem cells stay at the basal layer, to produce the next generation of daughter cells. The daughter cells migrate through the sebaceous gland-orifice, are extruded into the follicle infrainfundibulum (lower part of the hair follicle), pass the acroinfundibulum (upper part of the follicle) and the hair follicle-orifice to spread then over the skin- and hair-surface. (figure:1 sebaceous gland and follicle)

During migration the sebum cells start production of lipids and also keratin to a low degree- They change appearance and physical properties and turn into se-bum cells containing mostly triglycerides, free fatty acids, wax esters, squalene, cholesterol and tonofilaments contrary to the epidermal skin lipids lamellae based on ceramides, cholesterol, cholesterolsulfate and free fatty acids. They fi-nally reach a volume of 15 – 20 times the genuine diameter and burst to poor their content into the sebaceous gland and the follicle. The sebaceous cell walls are not digested, but become a constituent of the follicle-filament. The filament-mixture is a viscous, opaque fluid.

This mixture passes through the infundibulum, which is walled by a lamellar structure of horny cells and skin lipid layers and which scales off very small par-ticles as part of a regular desquamation process. Infrainfundibulum and acroin-fundiblum differ in the amount of epithelium cell layers. In addition the thin epithelial layer of the infrainfundibulum is not continuously distributed over the infundibulum wall but covers only in areas.

Keratin cell from the sebaceous gland orifice and horny flakes from the follicle walls are scaled off into the infundibulum, mixed with sebum and encapsulate P acnis. The resulting liquid (filament) is opaque and usually sufficient fluid enough, to migrate through the follicle orifice onto the skin surface.

Changes in puberty

The sebaceous gland stem cell-proliferation is stimulated in the juvenile acne skin phase by not outbalanced hormone-levels (DHEAs) and PPAR-ligands (per-oxisome-proliferation activating receptor) like 5-lipoxygenase generated leu-kotriens. Acne is only a temporary stage of skin during the usual temporary un-balanced hormone-concentrations. It affects an estimated 80–90% of teenagers regardless of sex (1). These 90% of teenagers are not diseased, of course, but in a default skin-phase corresponding to their age. Acne decreases latest by age 25. Longer lasting acne (post puberty acne) or different physio-pathologic acne is caused by very different triggers and might have diseases underlying (polycystic ovary syndrome, hirsutism, Cushing's syndrome, acne climacterica). Diseases as acne-triggers are not within the scope of this paper and the cosmetic acne treat-ment.

In consequence of dilution by higher sebum-contents the balance between choles-terol and cholesterolsufate might be disturbed and keratinase-activity might be down-regulated. In consequence bigger, solid, not usually pattern desquamated horny flakes are shed off from the follicular wall and change the properties of in-frainfundibulum-content to less fluidity

(hyperkeratinisation 1, 2, 3). Additional keratin-synthesis is reported. Plugs are built up blocking the infrainfundibulum (not the follicle-orifice), the follicle gets clogged. This stage is called microcomedo.

In advertise and even in literature one might find illustration of plugged follicle orifices or acroinfundibula. These figures and treatments based on it (peeling) are wrong approaches and have their origin in an old concept by Unna ("Korkentheo-rie"). The plugs within the infrainfundibulum of the follicle allow only small amounts of filament to pass and the follicle is expanded in the plug-area, i.e. the lower follicle-area and not at the upper end.

Acne in exclusive response to unbalanced hormone levels and PPAR-ligands is only triggered by adrenarche in puberty. Potentially amplifying cofactors like smoking, spicy food, cold, stress, high glycemic load diet, milk and many more are in discussion, but no doubt without any triggering property on their own. Men-strual cycles and pregnancy are of additional influence but no cause. Acne is a default physiological condition of juvenile skin.

When the blockade turns less passable and more filament is kept back by a solid plug, the follicle is enlarged in the area of infrainfundibulum building up a pri-mary comedo or micro comedo. (figure 2: microcomedo). In a later stage primary comedones turn into more enlarged primary comedones like whiteheads (closed comedones) and blackheads, which are open comedones, where oxidized melanin colored horny cell give the dark color (figure 3: whitehead, blackhead). This skin condition of primary comedones is called "acne spot" by the consumer and indus-try.

Follicle orifices are usually colonized by Propionibacterium acnes (P.acnis). This colonization is no sign of any disease but default. If infrainfundibulum turns rich in lipids and proteins, the warm area grants best conditions to start P. acnes to migrate and proliferate with a higher rate. The skin might react to microorgan-ism metabolic products by irritation. Therefore higher colonization and irritation is not a prerequisite for acne, only one of the following results.

The ceramide-moieties of the follicle-wall lamellae become diluted by high sebum contents. The ceramide-1, in which the linoleic acid functions like a stick through the lamellar stacks, is not longer able to keep the well organized condition (4). Thus in the infrainfundibulum the follicle wall turns less protective and metabo-lism products and even P acnis themselves penetrate into the surrounding tissue causing irritation, building skin-ducts and finally even lesion or scars, when re-pair mechanisms are overexpressed. Thus primary comedones turn into secon-dary comedones like pustels, papules, nodules and, if it enlarges into deeper ar-eas, cysts. (figure 4: pustule, papule; figure 5: nodule)

The sebaceous cells penetrated into the surrounding tissue attract immunocom-petent cells like neutrophils resulting in pathologic irritations. These irritations due to tissue-reaction have to be strongly differed from the former irritations caused by P. acnis. These consequences are the border to a disease-stage of acne.

Demarcation between cosmetic and medicinal acne treatment

Dermatologists (1) define

1. acne comedonica: microcomedones and primary comedones like acne spots, pimples, whiteheads, blackheads. This is the default juvenile skin condi-tion at 90% of adolescent population (1)
2. acne papulopustulosa: secondary comedones like pustules, papules (pin-heads), nodules (large papules), which are escalating conditions to status 1.

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