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Journal of Biology and Medicine
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JOURNAL OF Biology and Medicine
DOI: https://dx.doi.org/10.17352/jbm

José Mesquita-Guimarães*

Porto Faculty of Medicine, Porto, Portugal

Received: 26 June, 2019
Accepted: 20 August, 2019
Published: 21 August, 2019

***Corresponding author:** José Mesquita-Guimarães MD, PhD, Porto Faculty of Medicine, Porto, Portugal, E-mail: clindermmesquitaguimaraes@gmail.com

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Editorial

Human skin biology and the search for the truth

Editorial

1. Human skin consists of epidermis, pilosebaceous follicles, eccrine and apocrine sweat glands, melanocytes, Langherans cells, Merkel cells, connective tissue of the dermis and hypodermis, nerves and lymphatic and blood vessels. It means that the skin is an extremely complex organ, made up of multiple cell types and structures, with remarkable morphofunctional diversity. But the skin is a single, interdependent, unified whole, without "skin appendages", which evolves from birth to senile age and death, depends on sex and the environment and all its cells and structures are under genetic determination, so no two skins are alike.

2. Lamellar or Odland bodies [1] are produced in the spinous cells and granulosa cells of the epidermis and, at the junction of the granulosa cells with the corneal cells, they fuse with the more superficial membrane of the granular cells and empty their contents forming part of the intercellular cement between the corneal cells.

A study on the proteic composition of lamellar bodies of the human epidermis was published 11 years ago, in which the authors found 984 proteins, including proteins, glycoproteins, lipoproteins and acid hydrolases, so that lamellar bodies are secretory lysosomes [2].

And for three decades it has been known that there are lipids in the intercellular cement between corneal cells, such as ceramides, phospholipids, glycolipids, free steroids, cholesterol and sphingolipids, also carried by lamellar bodies [3-5]. It turns out that the intercellular cement between the corneal cells is glicolipoproteic and not only lipid, with remarkable complexity and numerical advantage of the proteins.

3. The junction zone between granulosa cells and corneal cells is not a barrier zone because it is the most fragile zone of the epidermis. And it is fragile because in it occurs the death of the granulosa cells and their transformation into corneal cells, which are totally different, without nucleus, without organelles, only formed by amorphous matrix, filaments and a thick wall. And this zone is not even more fragile because the transformation of granulosa cells into corneal cells occurs at an extraordinary, astonishing speed. In fact with the optical microscope it is impossible to see what happens in this transformation even in the *stratum lucidum* of very thick epidermis. Another detail is that the death of granulosa cells does not occur by lysosomal lysis. The lysosomes formed in the epidermis are the lamellar bodies which empty their contents out of the granulosa cells.

4. For 40 years we have demonstrated that the holocrine involution of the human skin sebaceous glands, the rat skin sebaceous glands and the rat preputial sebaceous glands occur by a mechanism of autophagocytosis [6-8]. Autophagocytosis takes place in completely differentiated cells, with nuclear degradation and cytoplasmic disintegration. In this disintegration, the organelles begin by being focally sequestered, leading to the formation of multiple vesicular bodies surrounded by membrane and containing hyaloplasm and degraded organelles. Vesicular bodies increase in number and size, the degradation progressively accentuates, and finally, they become residual bodies resembling myelin [7].

In sebaceous secretion there is an increasement of lipid droplets and residual bodies predominantly proteic [8]. In addition, holocrine involution is the sum of the sebaceous cells plus the lipid droplets they produce, so the sebum is hydroglycolipoproteic and not just a fat secretion.

5. In the human and rat skin sebaceous glands there are no lysosomes involved in the process of holocrine involution [7]. And in the rat preputial sebaceous glands, the perinuclear granules, which are very numerous and large primary lysosomes [9], remain unchanged in the course of involution [6,7].

6. It is also by autophagocytosis that occurs the differentiation of the stratified squamous epithelium of the main excretory canal of the rat preputial sebaceous glands,

Citation: Guimarães JM (2019) Human skin biology and the search for the truth. J Biol Med 3(1): 053-054.DOI: http://dx.doi.org/10.17352/jbm.000016

which is an epithelium *stratum corneum* identical to that of the human epidermis [6]. This finding is of remarkable importance especially if we associate it with the fact, as already mentioned in point 3., that the death of human epidermis granulosa cells does not occur by lysosomal lysis. So it is our belief that the differentiation of the epidermis and hairs also happens by autophagocytosis similarly than in sebaceous gland differentiation. In fact, in the epidermis, in the hairs and in the sebaceous glands the differentiation is similar because they have the same ectodermal origin. It does not make sense that nature works one way in one case and differently in the other.

It means that one of the auspicious futures in Dermatology research will be how to influence autophagocytosis in these structures.

7. The set of secretions of the epidermal lamellar bodies, the sebaceous glands and the sweat glands form a film between the corneal cells and on top of the epidermis. Its composition is hydroglicolipoproteic, extremely complex, and its purpose is the cohesion of the corneal cells and the existence and maintenance of the cutaneous microbiome.

Regarding the cohesion of the corneal cells, they do not have desmosomes nor hemidesmosomes hence the one that unites them is known by intercellular cement. The same is true on our beaches where the sand grains are dry and loose except where the sea water unites them, leaving the sand to be wet, aggregate and hard.

The cutaneous microbiome is inseparable from the skin. Today one can not speak of the skin without paying attention and mentioning its microbioma, given its remarkable antimicrobial, immunological and biological activities [10]. The same is true, for example, for the human gut microbiome [11].

Finally, the skin is only normal if it is not subjected to interventions that cause dependence, atrophy and alteration of the skin film and microbiome.

Skin dependence and atrophy induced by continued application of creams and emollients are clinically known. In 1976, Kirby and Munro showed skin atrophy, averaging 4.1%, with the application of vaseline with 2% liquid paraffin for 5 and 7 days in rat ears and human forearms [12].

It turns out that everything that is applied to the skin causes alteration of the skin film and the microbioma, because what we apply has nothing to do with reality, that is, with its remarkable physicochemical richness.

I suppose that the data presented above is a reason for reflection and deserves further study in the search for the truth.

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Citation: Guimarães JM (2019) Human skin biology and the search for the truth. J Biol Med 3(1): 053-054.DOI: http://dx.doi.org/10.17352/jbm.000016