Prospecting for new concepts in clinical science is like panning for gold: all that glitters is not gold. If the clinicians and scientists do not define exactly what they are looking for, they might mistake a bad idea for a good one. The risk is to confuse discovery with creation, bringing into imaginary existence a concept based upon false interpretations. In this respect, working on the subject ‘dry skin’ is like standing on shifting sands.

Despite its common use, the term ‘dry skin’ has never been defined in a repeatable way and often covers misinformation. The different ways to interpret dry skin by the cosmetologic and pharmaceutical industries are enough to persuade us about the pseudoscientific character of that elusive terminology. Claims of some cosmetologists would have us believe that bringing water to the skin with ‘moisturizers’ is the solution to the problems of dry skin. The pharmaceutical industry usually has an opposite view when it proposes an ointment rather than a cream to treat the chronic dry type of various dermatitides.

If there are such discrepancies in the concepts, what does dry skin mean? According to dictionaries, dry skin would mean skin without water. This is the skin of mummies. In fact, ‘dry skin’ does not necessarily apply to a dry structure and does not concern the whole skin. It just refers to the superficial part of the stratum corneum that is harsh and looks like a dry soil [1, 2]. By definition such a brittle scaly surface of the skin should be called rough skin without any reference to its level of hydration [3].

The paper of Saint Léger et al. [4] in this issue of Dermatologica is a scientific approach to clarify the origin of one type of rough skin. It follows a series of papers addressing the same question: what is the biological feature responsible for rough skin?

Some authors relate it to the lack of water in the stratum corneum [5, 6] but this was not confirmed by other studies [7–10]. Xerosis of atopic dermatitis represents such an example where conflicting data about hydration of the stratum corneum have been reported [7, 10, 11]. It should be also noted that many spongiotic and parakeratotic disorders are labelled dry skin although they contain large amounts of water in the stratum corneum, either in the extracellular space with serum or within parakeratotic cells.

An abnormal amount or composition of epidermal and sebaceous lipids have been reported to be one associated feature or the cause of rough skin. Despite some findings [4, 12], there is no definite proof that lipids play a central role in the development of the most common types of these skin conditions [8, 9]. Apart from X-linked ichthyosis and rare drug side effects, there is no other example where an abnormal lipid metabolism may be related to the presentation of xerosis. Obviously there are problems and controversies in the field of rough skin. Some of the shortcomings can be mitigated by further refinements in the clinical classification and rating.
Many of the past studies were inherently flawed because of the bias created by selected groups of patients or volunteers under the influence of markedly different environmental factors. Is winter dry skin of Philadelphia [4] the same as in Liège [8]? The question remains open. In short, there is not a single biologic feature responsible for rough skin. Such an aspect of the skin is a medley of totally unrelated conditions probably induced by a number of biologic pathways [3]. We need a classification of these disorders. If, at the present time we ignore their molecular biology, we should at least attempt to accurately assess the spectrum of rough skin presentations by clinical assessment, dermatopathology, biochemistry and physical methods. The issue of precise, repetitively found criteria for accurate diagnosis of xerosis is a crucial one. Our therapeutic approaches would probably benefit from such evaluations.

Editorial

References