Hair care and anti-dandruff

**GENERAL ASPECT OF DANDRUFF**

Dandruff is a common but troublesome and sometimes stubborn condition which manifests itself exclusively on the scalp. Dandruff scale is a cluster of corneocytes, which have retained a large degree of cohesion with one another and which become detached as such from the surface of the stratum corneum. It is not unfrequently centred by a pilosebaceous follicle (1). Parakeratotic cells often make up part of dandruff (2). Their density is related to the severity of the clinical manifestations. Seborrhoea also influences the clinical presentation. Pityriasis simplex indicates the presence of fine squames, usually of a small size, conferring an asteatotic and farinaceous appearance of the process. Steatoid pityriasis differs by the seborrhoeic, thick and adhesive nature of the dandruff, and the scale of which is often larger in size (1). Both the size and the abundance of the scales are heterogeneous from one site of the scalp to another, and vary over time (2). The condition may also fluctuate with the environment, particularly the climate as it often worsens in cold season. Ethnicity possibly influences dandruff severity. As an example, some Africans seem to have particularly abundant dandruff. In these individuals, scales are probably trapped in meshes of crowded terminal hairs thus preventing them from being lost. This hair-dandruff relationship may conversely explain the absence of dandruff on bold pates, and applies equally to hairless or shaved sites and to regions that sport only vellus hairs.

**Deciphering the origins of dandruff**

Clinically, dandruff appears as a non-inflammatory condition although histologically lymphoid cells are abutted to the distal portion of the hair follicles (1). It is generally acknowledged that a continuum exists between dandruff and seborrhoeic dermatitis representing more severe the inflammatory pole with further extension of the lesions outside the scalp. Dandruff is likely caused by several aetiopathogenic pathways with complex mechanisms and interactions. Among them, the microbial aetiology of the common type of dandruff is straightforward. The evidence implicating yeasts of the genus *Malassezia* in dandruff and seborrhoeic dermatitis has accumulated over time (3, 4). However, to add to the difficulty of interpretation, the quantitative microbiological assessments do not always point to the role of the yeast, the abundance of which might be proportional to the volume of the colonized scales, or be responsible for the altered desquamation (2). One other aspect that has not yet received much attention is the relationship between dandruff and the distinct species of *Malassezia*. There is ample evidence that the various *Malassezia* spp. are present in different amounts and proportions according the body site and to the nature of the *Malassezia*-driven disorders (5).

The *Malassezia* distribution is not uniform at the skin surface and inside the stratum corneum in dandruff and seborrhoeic dermatitis (6, 7). The yeasts appear clumped only over some corneocytes whereas other corneocytes in the vicinity harbour few of these yeasts. Perhaps, the *Malassezia*-binding sites differ in number on corneocytes. Another possibility calls for the intervention of the natural antimicrobial peptides of the innate immunity. The yeasts should normally boost the expression of β-defensin-2 by human keratinocytes (8). In dandruff, however, their expression could be impaired at specific sites thus allowing the *Malassezia* yeasts to accumulate. Further considerations should be given to the fact that *Malassezia* spp. express antigenic and pro-inflammatory properties stimulating both the innate and acquired immune responses, and neuroimmune sensorial responses as well (9). Controlling or ideally eradicating the accumulation of *Malassezia* spp. currently appears to be a clever strategy for treating dandruff (1-4). However, a puzzling problem is raised by the actual in vivo mode of action of anti-dandruff shampoos claiming an in vitro antifungal activity. Indeed, most of the active compounds are barely soluble in water, and their casual residence time on the scalp is very short (10). These conditions are at variance with those respected in the treatment of any other pityrosporosis and dermatomycosis. Hence, one cannot exclude other major or ancillary modes of action of these active anti-dandruff products, including some direct biological effects on epidermal cells (1, 7).

A dandruff condition without a primary fungal involvement is possible. The most obvious example is...
the scalp desquamation following excessive exposure to sunlight. The intercorneocyte cohesion is then affected. The same phenomenon occurs in minimal chronic irritation of the scalp. Daily routine cumulative minimal insults including hard brushing, over-shampooing, hair frictions (11) and certain cosmetic hair products may also be responsible for this event, as well as airborne xenobiotics from the environment. Other non-fungal agents may lead to minimal chronic irritation such as sebum-derived products, as well as sunlight activation of follicular-photosensitizing agents such as porphyrins synthesized by Propionibacterium spp. Some neuroimmune influence may be also operative. It is believed that psychological stress commonly amplify the severity of the condition. There are, however, no experimental and factual observations supporting this assumption.

**ASSESSMENT OF DANDRUFF SEVERITY**

Dandruff can be assessed through visual scoring performed under controlled procedures followed by adequately trained observers. It is wise to secure a wash-out period by using a bland shampoo for at least 2 weeks prior to the start of any clinical trial. Because of the removal of dandruff scales by shampooing, the visual assessment should be performed at defined intervals after shampooing. A 2-day period is a compromise between sufficient scaling recurrence and a loss of scales from external factors (combing, brushing, contact with pillows, etc..) during this period (12). The whole scalp or instead a defined specific part, usually the most severely affected, is to be examined. In most subjects, the vertex is the most scaly area, whereas the nape is less involved and possibly scale-free. A left-right split-head procedure is valid for intra-individual comparative trials as dandruff globally shows lateral symmetry. However, it may cause bias by cross-diffusion of the test products (12). Self-assessments and related parameters (itching, tolerance, stress, medications, environmental conditions, etc..) are often informative for the interpretation of data. The clinical evaluation should ideally be supported by some quantitative and objective bioinstrumental methods (1, 12). Among the many ways of collecting dandruff, we have designed the squamometry method (13). Because of its ease and reproducibility, the collection of dandruff is performed using a transparent self-adhesive disc left in place for a few seconds. Sampling should ideally be performed with controlled pressure on the scalp. The overall density of dandruff is estimated by squamometry, which is a reflectance colorimetric method, after staining the sample with a solution of toluidine blue and basic fuschin for 1 min. Examination by computerized image analysis under a microscope is another means of quantifying dandruff severity. At the same time, this enables yeasts of the Malassezia spp. to be counted. Live fungal cells are conveniently identified by vital staining with neutral red.

It is common observation that dandruff adhere to hair. It is possible to quantify this feature in order to assess any effect of hair product on this adhesion process (14). In our experience, the amounts of dandruff on the scalp and on hair are not always correlated.

**IMPACT OF DANDRUFF ON HAIR LOSS**

The presence of dandruff and its *Malassezia* load may precede or accompany hair loss under the form of telogen effluvium (2). It may thus exacerbate some types of alopecia (15-17). On a 2-day collection, non-dandruff scalps normally lead to a 50-100 hair loss whereas those affected by dandruff may range from 100 to 300, although a positive correlation with clinical grades of dandruff cannot be drawn (12). This hair shedding process associated with some cases of dandruff may result from alterations in the teloptosis process (exogen phase) and in the hair cycle phenomenon (18-20). This event is perhaps related to the subclinical inflammation demonstrated under the microscope (19). It has been shown that specific products used to combat dandruff may limit the progression of androgenic alopecia (21).

**CONCLUSION**

Dandruff is basically a reactive response of the epidermis of the scalp to various stimuli. Converging features support the role of *Malassezia* spp. collected over some corneocytes as the main causative agent. Some environmental factors such as ultraviolet light and airborne irritant xenobiotics or those applied deliberately to the scalp are also potential protagonists. Irrespective of the causative factor, the biological events involved in dandruff could be summarized into a few successive steps involving the intervention of the triggering agent, particularly the increased *Malassezia* load, discrete subclinical inflammation, parakeratosis and corneocyte cohesion. The process can also ensue in hair cycle disturbance and increased hair loss.

**REFERENCES**


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