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Duct Disruption, a New Explanation of Miliaria

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From argument and a few personal observations, the hypothesis has crystallised that the miliaria commonly occurring in unacclimatised Caucasians visiting hot climates is caused by exposure to ultraviolet irradiation, by an effect on the cells of the upper epidermis, which eventually allows a split to develop between them and the new stratum corneum that grows up beneath, into which sweat from the disrupted ducts can collect as microcysts. This dehiscence is the probable explanation of sunburn peeling and photo-onycholysis. It is concluded that duct disruption, not blockage or dysfunction, is the immediate cause of the miliarias.

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Miliaria, a common problem for Caucasians in hot climates, is generally attributed to sweat duct blockage (1). I have never found this explanation convincing, because I consider the histological evidence of blockage (2–5) to be poor and because miliaria cannot be produced by stimulation of sweat glands which have ducts blocked by disease (6) or experiment (7, 8); indeed, during such blockage, the coil continues to secrete sweat and the duct is able to absorb all that is secreted (7, 8). I therefore concluded that the immediate cause of miliaria was not duct blockage but a failure of the duct to absorb all the sweat secreted by the coil (6, 9). These two opposing hypotheses have had as little effect on one another as on the disorder they attempted to explain, and this continued mechanistic sterility, together with a few personal observations, compelled a reconsideration of the problem which produced a new hypothesis with promise of a better outcome.

Role of UV irradiation

I usually develop miliaria crystallina in tropical climates. The lesions appear rapidly when I sweat heavily and are easily stopped by taking a cold shower. But I have noticed that the lesions never occur before the end of the first week after arrival, and that they have a clear relationship to sun exposure, the miliaria being confined to the areas of mild sunburn (I rarely burn severely), never occurring in unexposed skin. As I have made this observation many times on myself and found, by questioning, the identical phenomenon in others, I can only conclude that exposure to ultraviolet irradiation (UVR) is the common cause of miliaria crystallina. The production of lesions by UVR has long been known, and it is therefore surprising that the prime role of UVR has been largely ignored, or indeed refuted (1–5).

The opportunity to examine the relationship of miliaria to UVR came in the course of an unrelated study (10) in which a 4×6 cm area of my forearm was exposed to 5 joule/sq.cm of 311 nm UVR. When heavy thermogenic sweating was induced 3 and 6 days later, by playing squash with marginally better opponents, no miliaria occurred at 3 days; but, just as I had noticed with natural sunlight in the tropics, miliaria developed at 6 days at the site of UV exposure, but not in the unexposed, normal skin. To confirm the unsurprising guess that the lesions had formed in the stratum corneum, that layer was removed as completely as possible with cellophane tape, on a separate occasion, in half of the area exposed to the same dose of UVR 7 days earlier. As before, miliaria occurred with thermally induced sweating in the UV-exposed skin, but not where the stratum corneum had been removed. From these observations, I can only conclude that exposure to UVR induces miliaria crystallina by an effect which leads to a change in the stratum corneum 5–6 days later.

What might this change be? The long-accepted view (2) that the primary change in miliaria is "keratotic plugging of the sweat duct orifices" (3) can be rejected on general grounds because miliaria does not occur with superficial sweat duct blockage (6–8). It can likewise be rejected as the explanation of UV-provoked miliaria: UVR could not evoke the manufacture of plugs from the dead keratinocytes of the surface, and a plug of keratinocytes newly formed in the epidermis would take longer than 5–6 days to grow out to the surface (11). Furthermore, in the former case, the occurrence of miliaria would be synchronous with exposure or, at any rate, more rapidly than 5–6 days, as it would likewise if blockage were caused by material in the lumen of the terminal duct produced by damage more deeply in the skin. An explanation other than duct blockage is required.

Although the lesions of UV-induced miliaria are discrete, I found them to be easily spread by *gentle* pressure, and they join up with one another in the stratum corneum. They can then be peeled off as a sheet, revealing a new layer of normal-looking stratum corneum beneath, in which the miliaria do not occur. Thus, the sweat, which initially forms discrete vesicles, is collecting in a potential plane of separation in the stratum corneum induced by UVR damage, and the ease with which the vesicles can be spread into this plane by pressure reveals this potential split. The commonly observed, but equally commonly unremarked upon, *uniformity* of the sheet of "sunburn peeling" fits comfortably with this interpretation. The thickness of one such sheet, measured by micrometer, was 50 μm, which is more than the thickness of the normal stratum corneum, suggesting that the primary action of UVR is on the epidermis, the first living cells to be encountered by UVR penetrating the skin. Histological examination of several sheets of sunburn peeling and the outer walls of a group of fresh miliarial vesicles, produced 7 days after UV exposure of my flexor forearm (Fig. 1), confirmed this site of effect, the outer wall of the miliarial microcysts (and the sun-burn peeling) consisting of the old stratum corneum lying on 2–3 layers of damaged epidermal cells which had separated from the new stratum corneum beneath.

Mechanism

As discussed in rejecting terminal duct blockage in the stratum corneum, the delay in onset of lesions excludes the possibility

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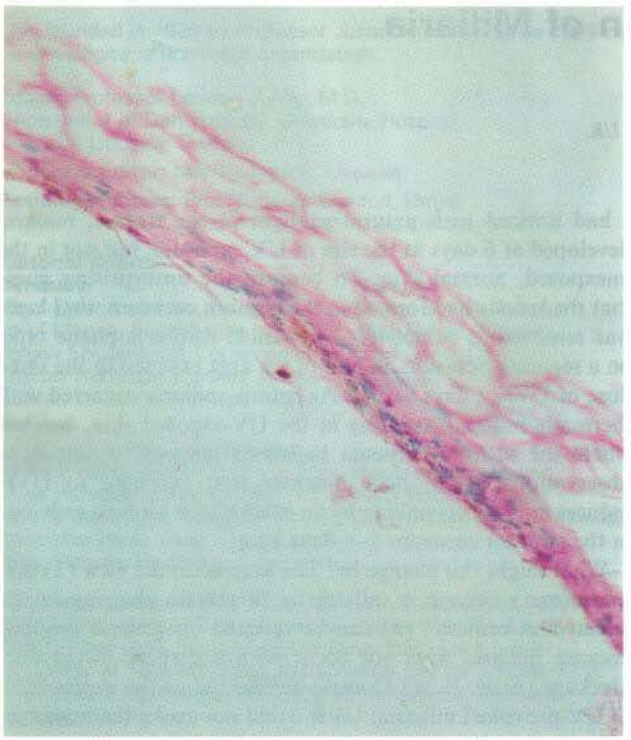

Fig. 1. Outer wall of miliarial microcyst, consisting of the old stratum corneum and a layer of 2–3 damaged epidermal cells that has split away from the new stratum corneum (not shown), which forms the base of the microcyst.

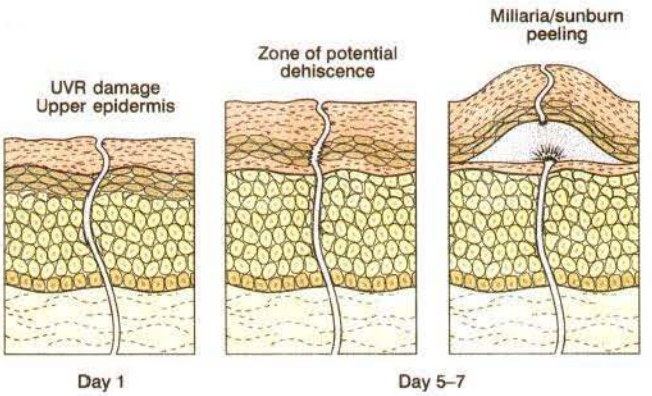

Fig. 2. Diagramatic explanation of UV-provoked miliaria crystallina by duct disruption; 1) UVR damage to cells of upper epidermal cells; 2) The UVR-damaged epidermal cells are extruded, sandwiched between the old and new stratum corneum cells; 3) The potential for dehiscence of the UVR-damaged epidermal cells from the new stratum corneum becomes apparent 5–7 days later when, during heavy sweating, sweat escapes from the disrupted ducts into the plane of dehiscence, forming miliaria. The plane of dehiscence may also reveal itself as sunburn peeling.

that UVR has a *direct* dehiscent effect on the epidermis. Thus, whatever the initial effect of UVR on the cells of the upper epidermis, it is not detected as miliaria until the affected epidermal cells are moved out by the proliferating cells beneath them, and a plane of separation develops in the stratum corneum, between the UV-damaged epidermal cells and the cells of the new stratum corneum beneath them. Clearly, such a plane of separation, which may later progress to a sheet of sunburn peeling, would destroy the continuity of the sweat ducts, the complex spiral structure of which is maintained up to the surface of the stratum corneum (12); sweat would then leave the disrupted ducts and collect as miliaria in the plane of dehiscence, as shown diagrammatically in Fig. 2. Thus it appears that the immediate cause of miliaria crystallina is *duct disruption*, and this duct disruption is primary, not secondary as previously believed; duct blockage and inadequate absorption, the elderly mechanistic predecessors of this hypothesis, can now be laid to rest. Although the direct evidence for this conclusion comes from self-observations, these were consistently reproducible on a number of occasions, and therefore show, at least, why *I* develop miliaria crystallina in the tropics (maceration miliaria is discussed later). But since, personal oddities not withstanding, I am unlikely to differ in the relevant respects from other members of my species, this personal evidence can reasonably be generalised. More importantly, the same conclusion can be reached on theoretical grounds and a reinterpretation of published evidence: thus it is now apparent that my new hypothesis resolves the conflict dormant, or avoided, in previous studies (2–5), and also explains the clinical features of crystalline miliaria. It is interesting, and not entirely explicable, why previous investigators, including myself, chose a different conclusion (2–6), despite the contrary evidence which was equally available then as now. In particular, Lowenthal (5) (although less concerned with crystalline than other miliarias, and despite a curious attribution of the disorder to saline tonicity) showed that the keratin "plugs" which had long been associated with duct blockage were a late event (5); yet despite this they were not generally dismissed as secondary phenomenon.

New hypothesis

The new hypothesis has obvious practical and theoretical implications. As ever, the former require less supportive verbal dressing. In brief, I have found that gradually increasing UV exposure and use of sun screens prevents miliaria, as well as being more enjoyable than avoiding the sun. Could *duct disruption*, the essence of the new hypothesis, account for the long recognised, but unexplained, occurrence of miliaria crystallina after iontophoresis (13)? Certainly, as I have now belatedly recognised, it occurs with a similar latency to that of UVR-induced lesions. However, since the peeling after iontophoresis is variable and irregular, if the action of the iontophoretic current is on the cells of the upper epidermis and sweat ducts, leading to disruption of their continuity as new cells of the stratum corneum (and sweat ducts) grow out, the effect must be more focal than produced by UV exposure. *Duct disruption* would likewise explain the miliaria which follows the application of "irritants" and diseases which damage the epidermis (2, 4), leading ultimately to a layered desquamation. The unifying hypothesis of *duct disruption* into a plane of potential dehiscence also explains the miliaria crystallina which follows superficial hydration. Of course, it is well known that hydration of the skin surface occludes the sweat duct orifices (14), but for reasons already discussed this would not produce miliaria so long as the ducts remain intact. Thus in the miliaria crystallina which follows surface occlusion (therapeutically, or in hot, humid climates), the causal *duct disruption* is likely to be due to dissolution by maceration damage to the stratum corneum (15) (rather than secondary

to changes in the epidermis beneath), allowing sweat from the disrupted ducts to collect in the space opened by maceration. Finally, although *duct disruption* appears universally applicable to the various forms of miliaria crystallina, it should be equally applicable to other miliarias, except that a zoned dehiscence would not be a prerequisite. However, and moving out of the sweat ducts, the zone of dehiscence does provide a novel explanation of drug-induced photo-onycholysis: since the nail is now known to be formed continuously along its length (16), and not just in the matrix as previously believed, dehiscence following UVR damage to epidermal cells of the nail plate would separate nail from its active site of growth in the bed. Photo-onycholysis is the simple analogue of sunburn peeling.

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