Melanocyte and Keratinocyte Carcinogenesis: p53 Family Protein Activities and Intersecting mRNA Expression Profiles
Journal of Investigative Dermatology (2007) 127, 1826–1827; doi:10.1038/sj.jid.5700928


In the publication by Kulesz-Martin et al., the authors reported that the TAp63α isoform was expressed in a keratinocyte model system in vitro, based upon reverse transcription-PCR analysis. We have since confirmed by immunoblotting using isoform-specific antibodies (provided by Dr Wendy Weinberg) and comparison with known molecular weight p63 isoforms ectopically expressed in keratinocytes that the TAp63α isoform is not expressed in the keratinocyte model but that the p63 isoforms present are identical to those in primary keratinocyte cultures. Thus, the p53 family members in keratinocytes are as follows in order of apparent molecular weight from greatest to least: ΔNp63α, TAp63β, TAp63γ, and either ΔNp63γ or ΔNp63α with ΔNp63α predominating. In the melanocyte model, the p63 isoforms expressed are TAp63γ, and either ΔNp63γ or ΔNp63α with the lower molecular weight isoform predominating.

The corrected Figures 2, 3 (keratinocytes in left panel), and 5 (summary of changes in p53 family expressions during carcinogenesis of keratinocytes and melanocytes) are shown. All text in the paper referring to TAp63α expression in keratinocytes and melanocytes should instead read ΔNp63α and discussion of TAp63β should read TAp63γ, a more transcriptionally active p63 isoform.
Female Pattern Hair Loss and its Relationship to Permanent/Cicatricial Alopecia: A New Perspective


In the publication by Olsen, there is an error on page 219. The section title “Central Center CCCA” is incorrect and should read “Central Centrifugal Cicatricial Alopecia (CCCA)”. This section is reprinted here with the correct section title. The publisher (Blackwell Publishing Inc.) regrets the error.

CENTRAL CENTRIFUGAL CICATRICIAL ALOPECIA (CCCA)
This common type of hair loss was first reported under the term “hot comb alopecia” by LoPresti et al. (1968). This particular hair loss occurs almost exclusively in African Americans, most commonly in African-American women. As it became apparent that African-American men may also be affected as well as those individuals who have not used hot combs (a method by which oiled or greased hair is straightened with a heated iron) (Sperling and Sau, 1992; Sperling et al., 1994; Headington, 1996), the terminology evolved to “follicular degeneration syndrome” per Sperling and Sau (1992) and more recently to CCCA (Olsen et al., 2003), the descriptive term adopted by the North American Hair Research Society (NAHRS). Whether hair-grooming methods specific to the African-American culture, such as hot combs, relaxers, tight braids, heavy extensions, and a variety of oils and pomades, could cause or at least contribute to the hair loss remains an unsubstantiated assumption.

The hair loss in CCCA begins in the central midline scalp and is slowly progressive centrifugally (Figure 3). The condition may progress to a Hamilton Norwood Type VI or VII pattern or may only involve the top of the scalp. Inflammation may or may not be obvious clinically. Perifollicular erythema or follicular keratoses are not typical findings. Histologically, a perivascular and perifollicular lymphocytic infiltrate, concentric lamellar fibrosis, sebaceous gland loss, as well as premature disintegration of the internal root sheath are typical (Whiting, 2001a,b). Granulomatous inflammation secondary to follicular rupture may also be seen (Sperling and Sau, 1992). Recently, these histological findings have been given the moniker of “pseudopelade”, which, unlike the entity of pseudopelade described by Brocq, does not typically have the clinical counterpart of discrete non-inflammatory patches of hair loss.

No effective therapy for CCCA has been definitively identified. Discontinuation of the use of hot combs, relaxers, and/or excessive heat, all factors that have been identified as possibly causing this condition, has, in most cases, not led to cessation of progressive hair loss. Whether hair care products that are used to moisturize the hair and scalp could be etiologic factors has never been addressed. Both bacterial and fungal scalp infections need to be searched for and treated, as these may be contributing factors. Although anti-inflammatory medications such as topical steroids and/or systemic antibiotics may slow hair loss in many cases, they are not uniformly effective treatments. Well-controlled clinical trials are needed to address these issues.

Figure 5

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For the rest of the document, the text is too small to be read clearly. However, based on the mentioned corrections and the context, it seems to discuss the correct terminology and the clinical aspects of CCCA, including its etiology, histological features, and the lack of effective treatments.