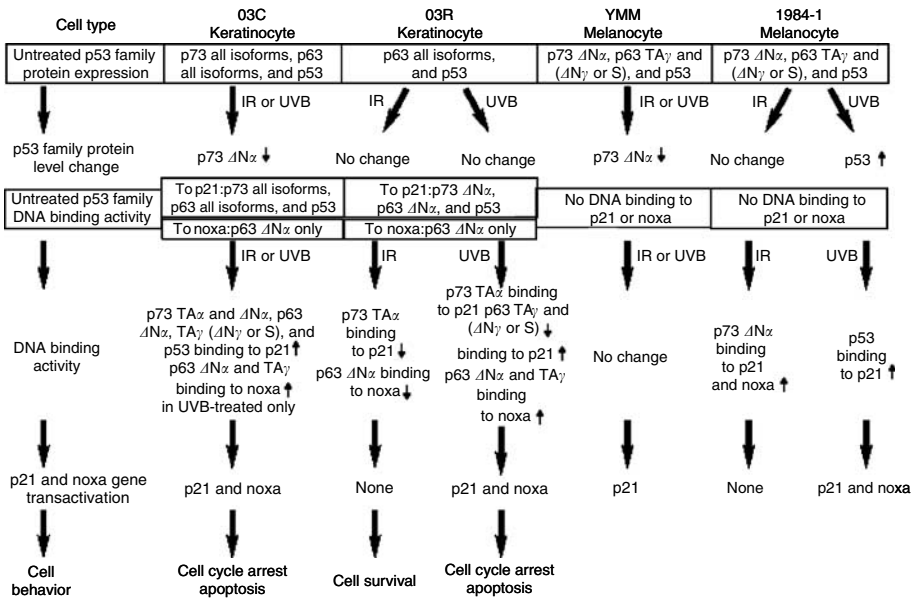


**Figure 5**

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**Female Pattern Hair Loss and its Relationship to Permanent/Cicatricial Alopecia: A New Perspective**
*Journal of Investigative Dermatology* (2007) **127,** 1827–1828. doi:10.1038/sj.jid.5700950

**Correction to:** *Journal of Investigative Dermatology Symposium Proceedings* (2005) **10,** 217–221. doi:10.1111/j.1087-0024.2005.10109.x

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 etiological 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.

Although it is clear that MPHL is less common and less extensive in men of African versus European descent (Smith and Wells, 1964; Setty, 1970), there appears to be a further decrease in the incidence of FPHL in African-American compared with Caucasian women (E.A. Olsen, unpublished data). Potential explanations for this disparity include a difference in the hereditary potential for pattern hair loss in African-American women compared with Caucasian women, different hormonal characteristics in the two races, an intrinsically different clinical presentation of FPHL in African-American women, or another hair loss process superimposed on FPHL in African-American women that obliterates the usual non-scarring presentation. The possibility that women with CCCA could have underlying FPHL, albeit with a degree of superimposed inflammation and fibrosis not typically seen in Caucasian women with FPHL, deserves consideration (Olsen, 2003). A controlled trial of anti-androgens or 5 αR inhibitors early in the process may be of value to help sort out whether there is any androgen relationship to this process.