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Androgenic alopecia may have evolved to protect men from prostate cancer by increasing skin exposure to ultraviolet radiation

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Summary Androgenic alopecia affects populations adapted to colder climate, and individuals at an age and hormonal status susceptible to prostate cancer. Male pattern baldness enhances absorption of UV radiation on the top of the head, an area directly exposed to sunlight during everyday activities. Ultraviolet radiation is reported to reduce the risk of advanced prostate cancer. Here I propose that progression of androgenic alopecia rather than being a risk factor is a finely tuned mechanism evolved to protect against prostate cancer.
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Introduction

Ethologists perceive androgenic alopecia (AGA) as a social signal evolved to indicate senescence [1], whereas in the medical literature it is discussed as an abnormal benign condition with no physiological effect [2].

Social signal theory has been supported by image manipulation experiments, where pictures of bald men are perceived older than the same faces with full hair [3]. The exact nature of the information conveyed, however, has been debated. Some believe that baldness is a signal of social competence and aggressive dominance [4], others suggest it indicates maturity and a non-threatening form of dominance [1].

In any case, young women perceive bald men sexually less attractive and less active [1]. As AGA affects about 30% of Caucasian males at the age of 30 years [5], signalling senescence at the age of the highest reproductive potential might be premature and therefore costly for many men. Even though the social signal theory has not been incorporated into mainstream medical science, the notion that AGA is not simply a disordered condition but has specific functions deserves attention, considering the genetically pre-programmed nature of the process.

Hair follicles react to androgens in a site-specific manner. In man, with genetic predisposition to AGA, terminal hairs in the temporal and frontal regions and later over the vertex of the scalp are

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miniaturized into vellus hairs, whereas follicles at other areas are not affected under the influence of androgens. Such differential reaction to the same hormone is pre-programmed in the individual follicles: follicles transplanted from non-balding areas to the bald vertex develop into full hair. Although the mechanism of differential reaction of hair follicles to androgens is not fully understood [6], it is very likely that Type 2 5α reductase (5αR-2) converting testosterone to more potent dihydrotestosterone in the follicles is involved. Besides hair follicles, 5αR-2 is expressed in the prostate, testes, seminal vesicles, and the liver. Inherited deficiency in 5αR-2 activity prevents AGA and results in small prostate. Finasteride, a 5αR-2 inhibitor has been useful in the treatment of AGA as well as prostatic hypertrophy [7]. As changes in the level of 5αR-2 activity affect AGA and prostatic hyperplasia in a similar manner, AGA as a risk factor for prostate cancer has been a target of epidemiological studies. Although findings have been inconsistent, one of the largest study including 1446 prostate adenocarcinoma cases and 1390 controls depicted an association between vertex baldness and prostate cancer [8].

Another line of epidemiological studies suggest reduced risk of prostate cancer associated with sun exposure, outdoor activity, highly active vitamin D receptor variants [9], high level of vitamin D [10], childhood sunburn, regular foreign holidays, sunbathing score, high exposure to UVR [11] or living at lower latitude [12], results all pointing at the protective role of sun exposure.

The hypothesis

Male pattern baldness exposes a relatively large skin area to sunlight during everyday activity performed in a vertical position. I propose that AGA evolved to elevate UV absorbance and thus to provide some protection against prostate cancer. Moreover, the shared androgen pathways leading to AGA and prostate cancer indicate a finely tuned mechanism of inducing baldness in men susceptible for prostate cancer at an age preceding the predicted onset of the disease. According to this interpretation if baldness is a social signal, then it has gained this function secondarily.

This hypothesis comprises two testable relations. Whether AGA was positively selected for in populations susceptible for prostate cancer can be directly tested when genetic background of AGA as well as of predisposition to prostate cancer are known. Indirect evidence of shared genetic control might be revealed by possible genetic correlations between AGA, sun exposure and prostate cancer in family history studies. The possible protective role of AGA against prostate cancer can be easily resolved. Epidemiological studies on risk factors for prostate cancer should involve questions on AGA status as well as on the level, and surface area exposed to sunlight.

Consequences of the hypothesis

This hypothesis might be relevant for the design of epidemiological studies as well as for the treatment of AGA and prostate cancer. Epidemiological studies on the link between AGA and prostate cancer can be confounded by the varying level of sun exposure of bald men with high or low outdoor activity, wearing or not wearing a sun protective hat, and living at lower or higher altitudes. Similarly, sun exposure measured as difference in pigmentation between areas protected or not protected from the sun without considering the surface area exposed may weaken the power of testing the cancer-protective role of UV radiation. Controlling for both AGA and sun exposure in both lines of studies might reveal stronger associations between sun exposure and lower risk of non-skin cancers.

Benign prostatic hyperplasia has been treated with finasteride, a potent 5αR-2 inhibitor [7]. As such treatment also halts or reverses AGA, treated men with prostatic hyperplasia might receive less UV radiation during everyday activities than without finasteride treatment. Careful studies are needed so that advice on compensating for reduced sunlit areas could be given to patients.

As association between low level of sun exposure with various forms of non-skin cancers, coronal heart disease, and hypertension has been suggested [13,14], links between predisposition to baldness and such conditions would be worthy to seek.

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