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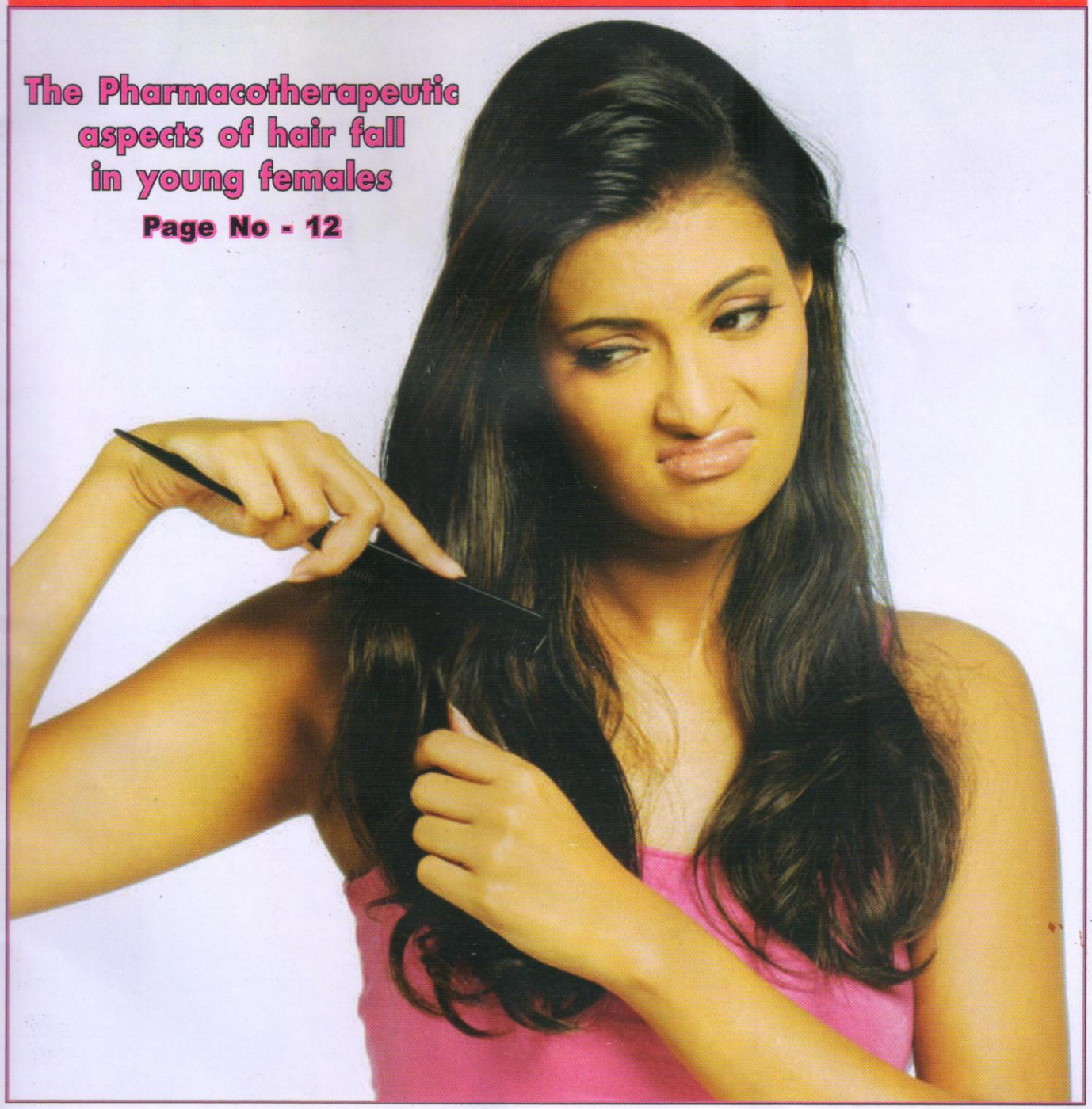

The Pharmacotherapeutic aspects of hair fall in young females

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Oxidative stress in Ageing of Hair

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The study of hair focuses on two main streams of interest: On one hand, the esthetic problem of hair and its management, in other words everything that happens outside the skin; on the other hand, the biological problem of hair, in terms of microscopic, biochemical (hormonal, enzymatic), and molecular changes, in other words the 'secret life' of the hair follicle in the depth of the skin. Basic scientists interested in the biology of hair growth and pigmentation have exposed the hair follicle as a highly accessible and unique model that offers unequalled opportunities also to the gerontologist for the study of environmental and age-related effects. Its complex multicell type interaction system involving epithelium, mesenchyme, and neuroectoderm, and its unique cyclical activity of growth, regression, rest, and regrowth provides the investigator with a range of stem, differentiating, mitotic and post mitotic terminally differentiated cells, including cells with variable susceptibility to apoptosis, for study. Finally, a number of intrinsic and extrinsic modulating factors for hair growth and pigmentation have been identified and are being further tested in vitro.[1]

Ageing of hair

Ageing is a complex process involving various genetic, hormonal, and environmental mechanisms. As the rest of the skin, the scalp and hair are subject to intrinsic or chronologic ageing, and extrinsic ageing due to environmental factors. Both occur in conjunction with the other and are superimposed on each other. Intrinsic factors are related to individual genetic and epigenetic mechanisms with inter individual variation. Examples of intrinsic factors are familial premature graying and androgenetic alopecia (AGA). Extrinsic factors include ultraviolet radiation (UVR), smoking, and nutrition.

Experimental evidence supports the hypothesis that oxidative stress plays a major role in the ageing process. As early as 1956, Harman et al.[2] first proposed this 'free radical theory of aging'. Today it is one of the most widely accepted theories used to explain mechanisms underlying the ageing process. Free radicals are highly reactive molecules with unpaired electrons that can directly damage various cellular structural membranes, lipids, proteins, and DNA. The damaging effects of these reactive oxygen species are induced internally during normal metabolism and externally through exposure to various oxidative stresses from the environment. The body possesses endogenous defence mechanisms, such as antioxidative enzymes (superoxide dismutase, catalase, glutathione peroxidase) and non-enzymatic antioxidative molecules (vitamin E, vitamin C, glutathione, ubiquinone), protecting it from free radicals by reducing and neutralizing them.[3] With age, the production of free radicals increases, while the endogenous defense mechanisms decrease. This imbalance leads to the progressive damage of cellular structures, presumably resulting in the ageing phenotype. The ageing phenotype of hair manifests as decrease of melanocyte function or graying, and decrease in hair production or alopecia.

Hair graying

Hair graying (canities) is a natural age-associated feature. The hair graying trait correlates closely with chronological ageing, but it occurs to varying degrees in all individuals. Hair is said to gray prematurely if it occurs before the age of 20 in Caucasians and before 30 in Africans. While premature canities more commonly appear without underlying pathology, presumably inherited in an autosomal dominant manner, it has been linked to a similar cluster of autoimmune disorders observed in association with vitiligo, i.e., pernicious anemia and autoimmune thyroid disease, and several rare premature ageing syndromes, such as Werner's syndrome. Although graying is understood as a loss of pigment in the shaft, its cellular and molecular origins are incompletely understood.[1] Theories for the gradual loss of pigmentation include exhaustion of enzymes involved in melanogenesis, impaired DNA repair, loss of telomerase, antioxidant mechanisms, and antiapoptotic signals.

Androgenic alopecia

The AGA is a heritable, androgen- and age-dependent process resulting in progressive decline in visible scalp hair density in a sex-dependent defined pattern. It affects at least 50% of

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men by the age of 50 years, and up to 70% of all males in later life.[5] Estimates of its prevalence in women have varied widely, though recent studies claim that 16% of women aged under 50 years are affected, increasing to a proportion of 30–40% of women aged 70 years and over. It is assumed that the genetically predisposed hair follicles are the target for androgen-stimulated hair follicle miniaturization, leading to gradual replacement of large, pigmented hairs (terminal hairs) by barely visible, depigmented hairs (vellus hairs) in affected areas. Affected men typically develop bitemporal recession of hair and vertex balding (male pattern AGA), while women present with diffuse thinning of the crown and an intact frontal hair line (female pattern AGA). Though AGA may manifest as early as at the age of 16 years (before 16 years, it is called by definition premature alopecia), balding has traditionally been considered as an attribute of ageing at all times and in all cultures. Indeed, evidence is emerging that AGA may be considered a form of organ-specific premature ageing.

Effect of smoking & UVA

Besides being the single most preventable cause of significant morbidity and an important cause of death in the general population, tobacco smoking has been associated with adverse effects on the skin. While smoke-induced premature skin ageing has long attracted the attention of the medical community, Mosley and Gibbs were the first to indicate a relationship between smoking and graying of hair and alopecia in men. The number of studied women was not sufficient enough to draw any significant conclusion. An association of smoking status with AGA was recently confirmed in an Asian community: Smoking status, current amount of cigarette smoking, and smoking intensity were statistically significant factors responsible for AGA in men after controlling for age and family history. So far, no data exist for women or partners of heavy smokers via secondary inhalation. The mechanisms by which smoking causes hair loss are multifactorial, and probably related to effects of cigarette smoke on the microvasculature of the dermal hair papilla, smoke genotoxicants causing damage to DNA of the hair follicle, smoke-induced imbalance in the follicular protease/ antiprotease systems controlling tissue remodeling during the hair growth cycle, pro-oxidant effects of smoking leading to the release of pro-inflammatory cytokines resulting in follicular microinflammation and perifollicular fibrosis, and finally increased hydroxylation of estradiol creating a relative hypoestrogenic state (overview). The fact that cigarette smoke-associated hair loss is of the androgenetic type again indicates that genetic factors contribute. The recent findings of Bahta et al. point to the fact that DPCs of androgenetic hair follicles are more sensitive to environmental oxidative stress.[6]

Potential role of oral supplementation therapy

Systemic photoprotection has been the focus of more recent investigation, in as much as this would overcome some of the problems associated with the topical use of sunscreens. Since the antioxidant defense mechanisms decrease as part of the natural ageing process, and are inhibited by UVR, while the production of reactive oxygen species increases, it seems reasonable to substitute antioxidants. Preclinical studies illustrate some photoprotective properties of supplemented antioxidants, though the effect is weak. Moreover, there is a paucity of controlled clinical trials in humans examining the role of antioxidants in preventing or decelerating skin ageing. Therefore, further experimental data need to be generated. Current research suggests that combinations of different antioxidants may have synergistic effects and better efficacy, when compared with single antioxidants used for photo protection.[7]

Since concentration-dependent H_2O_2 - mediated oxidation of tyrosinase in hair follicle melanocytes, in association with the loss of functioning methionine sulfoxide repair, sheds a new light on the slowing down of hair pigmentation as observed in the age-dependent graying process, and under in vitro condition, methionine oxidation can be prevented by l-methionine, it would be interesting whether l-methionine could be useful for intervention or reversal of the hair graying process.[8]

With respect cigarette smoke-induced alopecia, d'Agostini et al. demonstrated that high doses of environmental cigarette smoke induce alopecia in mice. This effect was prevented by the oral administration of a mixture of l-cystine with vitamin B6. Combinations of l-cystine and B vitamins are traditionally used OTC products for the treatment of hair loss. Their effect on humans exposed to cigarette smoke and losing hair has so far not been studied. Oral supplementation with l-cystine, pantothenic acid, thiamine nitrate, and medicinal yeast has been shown to increase the anagen rate in apparently healthy women with telogen effluvium in a placebo-controlled study.

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Scrub typhus is an acute febrile, infectious illness caused by rickettsia O. tsutsugamushi. It was first described in detail by Hashimoto from Japan in 1810. Scrub typhus is endemic to a part of world known as the "tsutsugamushi triangle" which etends from northern Japan and far-eastern Russia in the north, to northern Australia in the south, and to Pakistan and Afghanistan in the west. In India rickettsial infections have been documented from the states of Jammu and Kashmir, Himachal Pradesh, Uttarakhand, Rajasthan, Assam, West Bengal, Maharashtra, Kerala and Tamil Nadu. Himachal Pradesh is a mountainous state in northern India, with the altitude of 350-7000 meters above mean sea level. During the rainy seasons, areas at lower altitudes experience average temperature between 20 to 35^{0}C which is suitable for the spread of arthropod vector. Scrub typhus is a zoonosis and human are accidental hosts. The incubation period ranges from 6 to 21 days. The onset of disease is characterized by high fever (104-105^{0}F, headache, myalgia, cough, conjunctival suffusion and gastrointestinal symptoms. An eschar at the site of chigger bite, regional lymphadenopathy and a maculopapular rash may provide a clue to diagnosis. Mortality rates in untreated patients range from 0-30% and tend to vary with the patient's age and region of infection and if severe complications such as ARDS arise, mortality may still be higher.

- Journal of The Association of Physicians of India

Cutaneous larvae migrans (CML) is predominant in the tropical zone in hot and humid regions, is provoked by the migration of larvae of ankylostomas, which are, in the vast majority of cases, the larvae of Ancylostoma caninum and Ancylostoma braziliense which naturally contaminate dogs or cats. The lesions are localised in areas of the skin which are in contact with the soil since contamination of human beings by the larvae take place during contact of the skin with the ground or dirty sand : the soil is contaminated with feces of animals containing a large number of eggs, which give rise to larvae in favourable conditions 9humidity, sand, temperature greater than 250C). The penetration of larvae entails an allergic skin reaction with pruritis, erythema and a papulous eruption for twenty-four to forty-eight hours. Two to four days late a characteristic symptom appears: a skin furrow of a few centimetres long, which advances by a few centimetres each day, with a constant pruritus (itching). It could be a cutaneous parasitic impasse due to larvae of ankylostomas of dogs which are non pathogenic for human beings.

- La Medecine en France