for assessing such protection. Exposure of volunteers to UVA light, with and without a sun screen, is not practical in view of the time necessary to produce erythema with UVA light alone. Various alternative methods have therefore been developed—eg, photosensitisation of volunteers with a chemical such as psoralens; examination of another biological endpoint such as immediate pigment darkening; use of patients with photodermatoses that are highly sensitive to UVA; or use of an in-vitro system.[4]

One such system relies on a spectral measuring device and an artificial transpore membrane. By comparing transmission of UV light through the membrane with and without a sun screen, a transmission protection factor can be established at different wavelengths throughout the UVB and UVA range. The test is a cheap and convenient laboratory surrogate for in-vivo testing but it has not yet been fully validated and cannot be used for products that are alcohol-based or oil-based.[9]

A better method might be to use human epidermis instead of an artificial membrane. Epidermis could be obtained by raising suction blisters from surgical biopsy specimens or even from cadaveric skin, and would obviate the need for human or animal experimentation. This technique would provide doctors and manufacturers with a measure of UVA protection, and if patients or customers were confused by use of two numbers on a product, they could be told that SPF/B stands for burning and SPF/A stands for ageing, this explanation being sufficiently accurate for such a purpose. Equally, there is an urgent need for independent verification of company claims, so that doctors and patients can be satisfied of the UVB and UVA protection afforded by a prescribable formulation.[10]

Neverthless the main concern for human health is the carcinogenic impact of UVB, and not the cosmetic effect of UVA. As ozone depletion quickens[11] the proportion of UVB to UVA will rise, so burning, snow blindness, cataracts, and skin cancer can be expected to increase, whereas UVA-induced problems will decline, not because the amount of UVA will change but because sunbathers will be driven indoors by the acute effects of UVB before much UVA exposure occurs. It has been calculated that, other things being equal, a 10% loss of total column ozone will increase the frequency of melanoma by 10–20%,[12] of basal cell carcinoma by 25–35%, and of squamous cell carcinoma by 50–60%.[13,14] There is less certainty about the relation between cataracts and UVB, but the US Environmental Protection Agency have calculated an increase of 6–10% in the prevalence of senile cataracts after a 10% loss of total column ozone.[15]

Whilst UVA protection is therefore an important issue, it is overshadowed by the implications for human health of ozone depletion. Nor are sun screens necessarily the best method of protection against the deleterious effects of UV light—wearing a shirt or a hat is equally effective and in the case of cataract formation more effective than topical preparations. As Dr Robin Marks, Director of the Anti-Cancer Council of Victoria in Australia, has remarked "We are using one class of chemicals to destroy the ozone layer and another class to protect us from the consequences. Surely we can organise ourselves better than that".

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Insulin resistance in puberty

Blood glucose control in diabetic children deteriorates when they reach puberty. Poor dietary compliance, emotional lability, and adolescent rebellion are undoubtedly contributing factors, but a report by Amiel and her co-workers[1] serves as a reminder of the physiological decrease in insulin sensitivity that occurs during puberty in normal individuals and in those with diabetes. The result is a compensatory increase in basal and stimulated insulin secretion in normal adolescents[2] and an increased insulin requirement in diabetic adolescents. This physiological response would be less of a puzzle during this time of change in body composition and alterations of the pituitary-gonadal axis[3] were it not for the return in adult life of levels of insulin sensitivity similar to those found in prepubertal children.[4]

Amiel and colleagues investigated the mechanism of these changes in normal prepubertal and pubertal individuals by use of the euglycaemic clamp, and found that there was an insensitivity to insulin of peripheral glucose disposal in muscle. The ability of insulin to suppress hepatic glucose production,

lipolysis, and ketogenesis was not changed by puberty in this study, but these are very insulin-sensitive processes that are often overwhelmed by higher rates of insulin infusion. That stimulation of uptake of branched chain aminoacids was also unimpaired suggests that there was no resistance to the anabolic effect of insulin.

What is the explanation? The concentrations of many circulating hormones, including androgens and growth hormone, and their binding proteins change during puberty so many possible changes coincide with altered insulin sensitivity.[5,6] Androgen concentrations rise throughout puberty and have a small effect on insulin sensitivity in adults.[7] However, there is no difference in insulin sensitivity between the sexes during puberty. In any case, adrenarche (the augmentation of adrenal cortical secretion, especially androgens, in both sexes at about 8 years) occurs well before any measurable rise in basal insulin concentrations,[6] and androgen concentrations remain increased into adult life, when insulin sensitivity returns to prepubertal values. The growth spurt during puberty may be more relevant. Integrated 24-hour growth hormone concentrations increase about two-fold during puberty,[8] and the hormone is known to have an anti-insulin effect, probably at a post-receptor level.[9,10] In addition, at all ages there is a correlation between serum insulin concentrations and plasma insulin-like growth factor 1 (IGF-1),[6,11] and IGF-1 is the main mediator of the tissue effects of growth hormone.

Is this insulin insensitivity physiologically important in puberty or is it an epiphenomenon? Insulin has well-recognised growth promoting effects and growth retardation is seen in poorly controlled diabetic children.[12] Fasting plasma insulin levels are closely related to height velocity in childhood.[13] Insulin may stimulate IGF-1 secretion directly,[14] but interest has tended to focus on its effects on the IGF binding proteins. The growth hormone dependent binding protein IGFBP-3 may act to stabilise the circulating pool of IGF-1, but the smaller growth hormone independent protein IGFBP-1 seems to inhibit the action of IGF-1 on tissues, fluctuates during the day, and is itself suppressed by insulin concentrations in the short term.[15,16] During puberty growth acceleration and sexual maturation are closely linked but by an unknown mechanism.[17] Integrated control of growth during childhood and puberty has been suggested—ie, insulin may mediate the relation between nutritional status and maturation via its coordinated effects on sex hormone binding globulin and IGF-1 binding proteins.[18] When linear growth ceases, insulin would seem to lose its role as a growth hormone and revert to a purely metabolic function, with the relation between insulin and IGFBP-1 disappearing in adult life.[19]

Thus it may be that the role of insulin changes during puberty, and that reduced sensitivity to the hypoglycaemic action of this hormoe with a compensatory rise in basal and stimulated insulin concentrations is permissive to its action as a growth-promoting hormone. It is also possible that a result of this decrease in insulin sensitivity is the well-recognised rise in frequency of insulin-dependent diabetes mellitus in early puberty.[20] Ensuring appropriate increases in insulin dose in adolescents who are dependent on an exogenous supply may be important not merely in protecting against later microvascular complications of diabetes but also in securing full growth potential.

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