plines, and their care should be based in a few specialist centres that can offer the appropriate skills.

RICHARD W E WATTS Royal Postgraduate Medical School Visiting Professor, Department of Medicine. Royal Postgraduate Medical School, London W12 OHS MARTIN A MANSELL

Consultant Nephrologist. St Peter's Group of Hospitals and Institute of Urology, London WC2A 2EZ

- 1 Marshall RW, Robertson WG, Nomograms for the estimation of the saturation of urine with calcium oxalate, calcium phosphate, magnesium ammonium phosphate, uric acid, sodium acid urate, ammonium acid urate and cystine. Clin Chim Acta 1976;72:153-60.
- 2 Robertson WG, Peacock M. Risk factors in the formation of urinary stones. In: Chisholm GD, Williams DI, eds. Scientific foundations of urology. 2nd ed. London: Heinemann Medical, 1982:276-7
- Williams HW, Smith LH Jr. L-glyceric aciduria. A new genetic variant of primary hyperoxaluria. N Engl J Med 1968;278:233-9
- 4 Mistry J, Danpure CJ, Chalmers RA. Hepatic D-glycerate hydrogenase and glyoxylate reductase deficiency in primary hyperoxaluria type 2. *Biochem Soc Trans* 1988;16:626-7. 5 Yendt ER, Cohanim M. Absorptive hyperoxaluria: a new clinical entity – successful treatment with
- hydrochlorothiaxide. Clin Invest Med 1986;9:44-50. 6' Danpure CJ, Jennings PR, Watts RWE. The enzymological diagnosis of primary hyperoxaluria type 1 by measuring the alanine:glyoxylate aminotransferase activity in hepatic p needle biopsies. Lancet 1987;i:289-91.
- 7 Morgan SH, Purkiss P, Watts RWE, Mansell MA. Oxalate dynamics in chronic renal failure. Comparison with normal subjects and patients with primary hyperoxaluria. Nephron 1987;46: 253.
- 8 Watts RWE, Morgan SH, Purkiss P, Mansell MA, Baker LRI, Brown CB. Timing of renal transplantation in the management of pyridoxine resistant type 1 primary hyperoxaluria. Transplantation 1988;45:1143-5.
- 9 Baker LRI, Tucker B, Wood RFM, Gillard MG, Purkiss P, Watts RWE. Successful pregnancy in a renal transplant recipient with type 1 primary hyperoxaluria. Transplantation 1990;49: 811-2
- 10 Watts RWE, Calne RY, Rolles K, et al. Successful treatment of primary hyperoxaluria type 1 by
- combined hepatic and renal transplantation. Lancet 1987;i:474-5.
 11 Watts RWE, Calne RY, Williams R, et al. Primary hyperoxaluria (type 1): attempted treatment by combined hepatic and renal transplantation. QJ Med 1985;57:697-703.
- 12 Cochat P, Faure JL, Divry P, et al. Liver transplantation in primary hyperoxaluria type 1. Lancet 1989;i:1142-3
- 13 McDonald JC, Landreneau MD, Rohr MS, Devault GA Jr. Reversal by liver transplantation of the omplications of primary hyperoxaluria as well as the metabolic defect. N Engl \mathcal{J} Med 1989;**321**:1100-3.

Tanning with ultraviolet A sunbeds

Should be discouraged

Up to a fifth of British adults have used ultraviolet A sunbeds to induce artificial sun tans (CCE Meulemans, unpublished observations).¹ Yet a growing body of evidence indicates that such exposure may be harmful. To determine what the hazards are the British Photodermatology Group recently examined the data on the health effects of artificial ultraviolet A radiation and produced a set of guidelines for exposure.

Despite the sales talk ultraviolet A radiation is not uniformly effective in producing a tan. Ultraviolet A sunbeds generally produce a tan in people who tan well in sunlight (sun reactive skin types III and over),² but those who tan poorly or not at all or who are burnt easily by the sun (skin types I and II) are likely to be disappointed with the cosmetic results.^{3,4} Moreover, up to half of all users develop minor annoying cutaneous effects such as redness, itching, and dryness.³⁴

Some users have potentially more serious effects. People taking drugs or applying cosmetics with photosensitising potential and who then use ultraviolet A sunbeds may develop a photosensitivity reaction, generally an itchy or painful rash, sometimes followed by pronounced pigmentation.⁵ Sunbeds can also cause the common photodermatosis polymorphous light eruption-a transient, irritating, papular reaction⁴⁶and they exacerbate light aggravated dermatoses, such as systemic lupus erythematosus.7 Immunological changes, both cutaneous and systemic, have been seen after exposure to ultraviolet radiation from a sunbed.489 Although these changes diminish immunological responses and, theoretically, immunological surveillance, their actual biological importance is unknown.

Excessive use of ultraviolet A sunbeds-defined as exposure for 30 minutes or more a week over several months-produces increased skin fragility and blistering.¹⁰¹¹ It may also cause melanocytic lesions with malignant potential,^{12 13} though these lesions have resulted primarily from using sunbeds at home, where the duration and frequency of use are likely to be greater than in a salon. In mice long term exposure to ultraviolet A radiation causes premature photoaging of the skin.^{14 15} Although this effect has not been shown in human skin, it would be expected. Likewise, the nonmelanoma skin cancer that has been induced in animals after long term exposure to ultraviolet A would also be expected in humans.^{16 17} Extrapolation from animal studies and from epidemiological data on the incidence of non-melanoma cancer and exposure to sunlight suggests that the relative risk is probably small (<2) if sunbeds are used for no more than 20 half hour sessions a year through adult life,18 19 but no data on humans support this estimate.

The data suggest that the use of ultraviolet A sunbeds is a weak risk factor in inducing melanoma.^{20 21} Further studies are needed to confirm this and to establish the causal relation between pattern of exposure, the nature of the ultraviolet lamp, and melanoma.

Although many gaps in the knowledge of the effects of ultraviolet A radiation remain, the accumulating evidence suggests ever more strongly that the radiation has deleterious effects. The British Photodermatology Group has therefore recommended that the use of ultraviolet A sunbeds for cosmetic tanning should be discouraged. In particular several groups should not use them at all: children aged under 16; people who burn easily, do not tan, or tan poorly; those taking drugs or using cosmetics thought to be photoactive; those suffering from a skin disorder induced or aggravated by exposure to sunlight; those with a history of skin cancer; and those with risk factors for cutaneous melanoma. The risk factors include more than 20 benign pigmented naevi above 2 mm in diameter; a tendency to freckle; clinically atypical naevi; a history of severe sunburn, particularly in childhood or adolescence; and a family history of cutaneous melanoma. People who, despite this advice, want to use ultraviolet A sunbeds should not exceed two courses a year, each of no more than 10 sessions. Each session should last no longer than the time that it takes to produce just perceptible reddening of the skin eight to 24 hours later, up to a maximum of 30 minutes.

Head of Medical Physics, Dryburn Hospital, Durham DH1 5TW

Members of the British Photodermatology Group who contributed to this report are B L Diffey, P M Farr, J Ferguson, N K Gibbs, F R de Gruijl, J L M Hawk, B E Johnson, G Lowe, R M MacKie, A F McKinlay, H Moseley, G M Murphy, P G Norris, A R Young.

- Consumers' Association. The truth about tanning. Which? 1987 May:214-6.
- 2 Gange RW, Park YK, Auletta M, et al. Action spectra for cutaneous responses to ultraviolet radiation. In: Urbach F, Gange RW, eds. The biological effects of UVA radiation. New York: Praeger, 1986:57-65.
- Diffey BL. Use of UVA sunbeds for cosmetic tanning.Br J Dermatol 1986;115:67-76.
- 4 Rivers JK, Norris PG, Murphy GM, et al. UVA sunbeds: tanning, photoprotection, acute adverse effects and immunological changes. Br J Dermatol 1989;120:767-77.
- 5 Hawk JLM. Photosensitizing agents used in the United Kingdom. Clin Exp Dermatol 1984;9:
- 300-2.
 6 Devgun MS, Johnson BE, Paterson CR. Tanning, protection against sunburn and vitamin D formation with a UV-A "sunbed." *Br J Dermatol* 1982;107:275-84.
 7 Stern RS, Docken W. An exacerbation of SLE after visiting a tanning salon. *JAMA* 1986;255:3120.
 8 Hersey P, Hasic E, Edwards A, Bradley M, Haran G, McCarthy WH. Immunological effects of the transmission of the salout sector. solarium exposure. Lancet 1983;i:545-8.
- 9 Hersey P, MacDonald M, Henderson C, et al. Suppression of natural killer cell activity in humans by radiation from solarium lamps depleted of UVB. J Invest Dermatol 1988;90:305-10.

BL DIFFEY

- 10 Farr PM, Marks JM, Diffey BL, Ince P. Skin fragility and blistering due to use of sunbeds. Br Med 7 1988;296:1708-9
- 11 Murphy GM, Wright J, Nicholls DSH, et al. Sunbed-induced pseudoporphyria. Br J Dermatol 1989;120:555-62. 12 Jones SK, Moseley H, MacKie RM. UVA-induced melanocytic lesions. Br J Dermatol 1987;117:
- 111-5.
- Williams HC, Salisbury J, Brett J, du Vivier A. Sunbed lentigines. *Br Med J* 1988;296:1097.
 Kligman LH, Kaidbey KH, Hitchens VM, Miller SA. Long wavelength (>340 nm) ultraviolet-A induced skin damage in hairless mice is dose dependent. In: Passchier W, Bosnjakovic BFM, eds. Human exposure to ultraviolet radiation: risks and regulations. Amsterdam: Elsevier, 1987.77-81
- 15 Bissett DL, Hannon DP, Orr TV. Wavelength dependence of histological, physical and visible changes in chronically UV-irradiated hairless mouse skin. *Photochem Photobiol* 1989;50:763-9. 16 Sterenborg HJCM, van der Leun JC. Tumorigenesis by a long wavelength UV-A source.
- Photochem Photobiol 1990;51:325-30. 17 Van Weelden H, de Gruijl FR, van der Putte SCJ, et al. The carcinogenic risks of modern tanning
- equipment: is UV-A safer than UV-B? Arch Dermatol Res 1988;280:300-7. 18 Diffey BL. Analysis of the risk of skin cancer from sunlight and solaria in subjects living in northern
- Europe. Photodermatology 1987;4:118-26. 19 Slaper H, van der Leun JC. Human exposure to ultraviolet radiation: quantitative modelling of skin cancer incidence. In: Passchier W, Bosnjakovic BFM, eds. Human exposure to ultraviolet radiation: risks and regulations. Amsterdam: Elsevier, 1987:155-71.
- 20 Swerdlow AJ, English JSC, MacKie RM, et al. Fluorescent lights, ultraviolet lamps, and risk of cutaneous melanoma. Br Med J 1988;297:647-50.
- 21 Walter SD, Marrett LD, From L, Hertzman C, Shannon HS, Roy P. The association of cutaneous malignant melanoma with the use of sunbeds and sunlamp. Am J Epidemiol 1990;131: 232-43.

Milk for babies and children

No ordinary cows' milk before 1 year

What milk should a child drink? For the suckling infant the answer is clear – breast milk; or failing that an infant formula. Recently, however, our nutritional priorities have moved on from suckling babies to weanlings and toddlers. New products have arrived-the follow on milks. Another factor is that health conscious families are buying skimmed and semiskimmed milk. So what advice should be given to mothers, living in developed countries, who want to know which milk is best for their children?

The table gives the composition of the milks from which the choice has to be made. Most infant formulas and follow on milks are reconstituted from powders, but some are now available as liquids. All infant formulas and follow on milks available in Britain are fortified with iron and vitamins A and D.

From birth to 6 months

Up to 6 months the child should receive breast milk or an infant formula. Few will require solid weaning foods before 3 months, but almost all will want something extra by 6 months. When compared with bottle fed babies those who have been breast fed for 13 weeks or more have fewer gastrointestinal upsets and fewer admissions to hospital.¹ If an infant formula is chosen one of the whey based products is preferable, though casein predominant formulas are acceptable. Mothers, health visitors, and doctors commonly switch babies from one type of milk to the other; such switching is unnecessary but is probably harmless.

Vitamin supplements are not formally recommended by the Department of Health for children under 6 months.

Ideally mothers should have received vitamin D supplements in pregnancy but few do. If there is any doubt about the mother's vitamin D state during pregnancy—as, for example, in Asian mothers, winter pregnancies, and women living in northern Britain-then a breast fed baby should be given a vitamin D supplement.

Between 6 and 12 months

Between 6 and 12 months breast feeding may continue. Bottle fed babies should continue with their infant formula or they can have a follow on milk (see table); there is little to choose between them. Theoretically, the lower protein: energy ratios in infant formulas would not adequately support a mixed diet that was very low in protein—one made up of fruit and sweets, for example. In practice and in careful studies this does not seem to be a problem, but if there is any doubt then use a follow on formula. I advise mothers who are bottle feeding to continue with an infant formula. Some mothers, however, wish to move on from an infant formula, seeing this as a welcome sign of development of their babies; for them a follow on milk should be recommended rather than cows' milk.

All babies between 6 and 12 months given breast milk will need vitamin supplements. The recommended dose of supplementary vitamin D is 7 µg daily. This is provided by one Department of Health vitamin supplement five drops daily (not prescribable on FP10) and by many proprietary preparations. Vitamin policies have changed several times over the years and there are many different views.²⁻⁴ Those babies given infant formulas or follow on milks will not need vitamin supplementation. Special efforts should be made to ensure that children having only limited exposure to the sun-those in northern urban areas, those not having a sunny holiday, Asian children, those taking vegetarian diets, and others with cultural, social, or medical reasons limiting exposure-should receive vitamin D supplements or drink a milk containing vitamin D.

I do not recommend ordinary cows' milk before the age of 1 year. It contains little vitamin D and iron and causes subclinical but appreciable gastrointestinal bleeding in about a third of children.⁵ Other possible disadvantages are its higher concentrations of saturated fat and sodium, but the importance of this for the child's future is not clear. The extra cost of using an infant formula or a follow on milk rather than ordinary cows' milk (10-15p a day) is small compared with the price of other baby products.

Between 12 and 24 months

After the age of 1 year the choice is between cows' milk or a follow on milk; both are acceptable as part of a mixed diet. Semiskimmed and skimmed milk are not recommended at this age because of their limited energy content. Follow on milks are not used nearly as much in Britain as in some other countries, but they may have some advantages: they contain

Content of available milk for babies and children per 100 g feed (made up with water according to manufacturer's instructions where necessary)

	Energy in kJ (kcal)	Protein (g)	$Vitamin \; D\left(\mu g\right)$	Iron (mg)	Saturated fat (g)	Sodium (mmol)	Cost (pence)	Earliest age for use
Breast milk	290 (70) 285 200 (67 70)	1·3 1·5-1·9	0.01	0.08	2·1 1·0-1·9	0.6 0.6-1.1	6-7	From birth From birth
Infant formulas* Follow on milks†	285-290 (67-70) 270-285 (65-67)	2.0-2.9	1.1-1.2	0.4-0.7	1.2	1.3-1.5	6-7	6 Months
Cows' milk:								
Ordinary	285 (67)	3.4	0.05	0.02	2.5	2.2	4.5-6	12 Months
Semiskimmed	200 (48)	3.4	0.05	0.02	1.1	2.2	4.5-6	2 Years
Skimmed	140 (34)	3.4	0.02	0.05		2.2	4.5-5	5 Years

*Infant formulas available in Britain: whey based – Aptamil, Ostermilk, Premium, SMA Gold; casein predominant – Milumil, Ostermilk 2, Plus, SMA White. +Follow on milks available in Britain: Junior Milk, Progress.