

in 1850.³ Others have drawn attention to the difference between the cretinism associated with endemic goitre (classic cretinism) and hypothyroid (sporadic) cretinism.⁴ McCarrison⁵ noted that there were two types of cretinism, which he labelled neurological (the classic form) and myxoedematous, which came into use after Curling's description.

Nelson notes that iodine deficiency is responsible for 5·7 million people with cretinism and another 43 million with intellectual handicap. This misuses the term handicap, which is defined by the World Health Organization as a disadvantage for a particular individual, resulting from a disability or impairment that limits or prevents the fulfilment of a role that is normal (depending on age, sex, and social and cultural factors) for that individual. The 43 million who have some degree of intellectual impairment are not necessarily handicapped. Use of the term handicap is especially inappropriate when referring to those living in village communities in developing countries, where most of the intellectual impairment due to iodine deficiency is to be found. In these societies, literacy and numeracy are not prerequisites and intellectual impairment with its learning disability may not be a serious handicap. On the other hand, deaf-mutism and diplegia do impair a person's ability to have a normal role in those and other societies. Thus, deaf-mutism and diplegia are disabilities that are also serious handicaps: this is not to deny the importance of preventing all impairments attributable to iodine deficiency, and the efforts of international agencies are to be commended. Nevertheless, a community's appreciation of which impairments and their disabilities are handicapping may better determine the most appropriate direction for a preventive programme. For example, deaf-mutism and diplegia can bar entry into marriage, and community involvement in prevention efforts will be easily achieved. Yet the prevention of intellectual impairment or learning disability may not be a priority in a salt iodisation programme in a community that does not recognise the learning disability as being a handicap.

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Allergy to peanut

SIR—As the UK distributors of the Balneum range of bath additives for dry skin conditions, we were interested by Weeks' report (Sept 14, p 759)¹ of allergy to peanut oil in tropical products. Weeks maintains that arachis oil may be contaminated with allergenic proteins from the peanuts themselves and implies that this gives rise to topical hypersensitivity with arachis-oil-containing skin products. Although the risk of peanut allergy after ingestion of peanut-containing foods is well recognised,² the role of topical arachis oil as an allergen is controversial.³ Refined arachis oil should not have the allergenic potential of peanuts because it

should not contain the allergenic proteins found in the shells and kernels of peanuts; this would be especially true of the pharmaceutical-grade arachis oil used in dermatological products.

We are aware of data suggesting that this is also the case for soya oil, which is the oil present in Balneum products marketed in the UK. A study has been undertaken in soya sensitive individuals indicating that soya oil does not cause an allergic reaction in the same way as crude soya extract, as evidenced by skin prick test and oral challenge.⁴ Furthermore, this study was done with culinary soya oils, which may be expected to be less pure than soya oil BP, which is used in Balneum products. Adverse event data available from the Committee on Safety of Medicines for Balneum and Balneum Plus are negligible. There have been two topical adverse events reported for Balneum since it was launched in 1983—one case of "application site disorder" and one of "rash". There have been two topical adverse events reported for Balneum Plus, since it was launched in 1991. Even allowing for under-reporting, this suggests a favourable profile of local tolerability for the Balneum products.

Another implication of Weeks' letter is that there is some degree of cross-sensitivity between arachis oil and soya oil. Although peanut allergies might coexist with allergies to tree nuts,⁵ there is no evidence of any cross-reactivity of arachis oil and soya oil

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SIR—Peanut (groundnut, *Arachis hypogaea*) allergy is becoming more common, possibly because of an increase in infants' exposure to peanut in topical preparations as well as in foods.¹ Another possibility is that peanut allergy is becoming more common because, at least in the more prosperous regions of the world, the prevalence of atopy is increasing.² Recent reports mention anaphylaxis after eating peanut, and contact urticaria to peanut, but the problem of allergic reactions to airborne foodstuff exposure is mentioned only in passing.³ In the past 6 years we have seen four patients with anaphylaxis triggered by airborne peanut vapour.

Case 1. A 21-year-old woman with a family history of atopy developed contact urticaria to peanut as an infant. She later had recurrent episodes of angio-oedema on accidental ingestion of peanut-containing foods. During the past 6 years, despite strict avoidance of dietary peanut, she has had anaphylactic episodes, with stridor and dyspnoea sometimes followed by loss of consciousness. These events required adrenaline self-injection, and seven have necessitated documented emergency hospital care. All recent episodes have followed airborne exposure, such as when she walked past someone who had been eating peanuts and when a relative ate a biscuit, later found to contain peanut. Another episode followed entering a kitchen where peanut oil was used for cooking.

Case 2. A 10-year-old asthmatic boy presented with a 7-