erythema by absorbing ultraviolet B rays. The efficacy of some of these compounds, however, is being questioned.³ Some sunscreens may themselves be phototoxic⁷—for example, amyl dimethylaminobenzoate (Padimate A, Escalol 506). Some can prevent erythema, but not premalignant changes in the epidermis⁸—for example, mexenone (2-hydroxy-4-methoxy-4-methylbenzophenone). Furthermore, a 5% ethanolic solution of para-aminobenzoic acid (PABA), though an effective sunscreen, cannot protect against drug-induced phototoxic reactions.⁹ Broad-spectrum sunscreens combining a PABA ester with a benzophenone ester appear currently to provide the most protection from ultraviolet light.⁹

The Council of Europe is currently considering sunscreens and one hopes that in the future proper standardisation and toxicological tests will be required for all sunscreens and tanning aids, studies which would dispel much concern regarding their safety.

INGRID HOOK

Department of Pharmacognosy,
School of Pharmacy,
Trinity College,
Dublin 4

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Distalgesic poisoning—cause for concern?

SIR,—Dr Robert Wall and others are right to draw attention to the dangers of dextropropoxyphene (17 May, p 1213).

Some years ago I became aware that patients for whom Distalgesic had been prescribed frequently requested further supplies in circumstances which, in my opinion, did not warrant this level of analgesia. It was also noticeable that the difficulty of weaning patients off this preparation increased in proportion to the duration of medication. I now rarely prescribe Distalgesic or similar drugs, and never for more than a short period.

In my view any preparation containing dextropropoxyphene should be controlled under the Misuse of Drugs Act, and the dangers of dependence should be publicised widely.

H W K ACHESON

University Department of
General Practice,
Manchester M13 0FW

SIR,—Any Glasgow graduate of my vintage will have clear memories of the depressing remark by our brilliant professor of "materia medica," the late Professor Ralph Stockman, when at the end of nearly an hour's discourse on some drug he would sum it all up with a deprecating "But it is of no use whatever."

Younger generations can hardly realise how few were the really effective drugs that were at our disposal in these presulphonamide days of the early 1930s. The corollary that the highly effective drugs of today result in many

unexpected and dangerous side effects appears to occasion surprise and sometimes what seems undue alarm. The correct action is surely to accept their benefits while retaining an alert outlook for any unwanted reaction.

With regard to the specific warnings on the dangers resulting from Distalgesic, although I do not rank as a "controlled experiment" yet I can speak from personal experience, having suffered from severe disability from back pain and sciatica, which started a year ago and which eventually put me on my back for some weeks. A slipped disc and any more sinister cause having been excluded, it may be guessed that almost all the available range of analgesics was tried (hiatus hernia and a tendency to peptic ulceration being a problem). I can assure Drs R J Young and A A H Lawson (12 April, p 1045) that Distalgesic, in association with the peripherally acting aspirin in the form of Nu-seals, was quite the most effective, avoiding the disastrously constipating effect of codeine. Certainly the pain was such that I at one time required a daily total of 390 mg of dextropropoxyphene, 3.9 g paracetamol, and 3.9 aspirin, in addition to various physical forms of treatment in combination with the anti-inflammatory indomethacin.

Despite this large dosage, there were no ill effects and I had no difficulty in gradually reducing it as the condition improved, until now I am down to single capsules a day with no withdrawal symptoms or apparent development of dependence. Is it not true, with perhaps the exception of the hard drugs (although after a month of codeine I stopped it with alacrity), that much of addiction depends as much on a patient's personality as on the particular drug? The patient of Dr Robert Wall and others (17 May, p 1213) seems to be a case in point.

Let us not throw out the baby with the bath water but retain Distalgesic for what it can do, without forgetting the caution in the use of any drug that Stockman would have approved.

W O G TAYLOR

Ayr

Investigating constipation

SIR,—Your leading article (8 March, p 669) "Investigating constipation," which described the pathological and iatrogenic causes of the condition, contained some questionable statements—for example: "From infancy the British are brought up to regard a daily bowel action almost as a religious necessity and to believe in autointoxication from the cesspool of the unemptied colon; so it is little wonder that their doctors see vast numbers of patients obsessed with the frequency, consistency, diameter, and appearance of their stools." This, surely, is an exaggeration and carries with it the unfortunate implication that regular bowel habits are of little importance.

This illustrates once again that physiological yardsticks, in numerical terms, for human bowel function are badly needed.¹ Recently, a number of investigators²⁻⁶ have compared the effects of high- and low-fibre diets on bowel function. Their data suggest that in healthy subjects good fibre-containing diets produce at least one motion daily, stool wet weight of 150-200 g/day, and a transit time of approximately 48 hours. Multicentre studies are now necessary to confirm these findings; and close co-ordination is essential to secure

uniformity of methodology, which should include an objective method of measuring consistency.⁷ Reference values established in this way would have wide applications in pharmacological research, clinical practice, and health care and would also provide the basis for a scientific definition of constipation. In the mean time Painter⁷ has provided a useful working definition, which states: "Any patient who strains to defecate and does not pass at least one soft stool daily without effort is constipated."

With the present enthusiasm for the "bulk" principle it has become somewhat fashionable to denigrate chemical laxatives as unnecessary evils—as is evident in your leading article. That there has been gross abuse of laxatives in the past and is some continued misuse today is unquestioned. However, there is ample evidence to show that a chemical laxative, carefully selected and used, has a valuable place in many cases of disordered gut motility.⁸ Painter⁷ illustrates the complementary actions of a chemical laxative and dietary fibre when he states: "Idiopathic slow transit constipation, megacolon and megarectum are a persistent problem. Besides dietary fibre they require standardised senna tablets (Senokot). Usually 2-4 tablets daily are sufficient, but the dosage must be found by trial and error. It may be possible to reduce the initial dose when the high-fibre diet has taken effect."

EDMUND W GODDING

Totland, Isle of Wight PO39 0EB

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Diagnosis of infectious mononucleosis

SIR,—Your leading article (10 May, p 1153) dealt with a common problem—the accurate diagnosis of infectious mononucleosis—in which there is much misunderstanding. Pfeiffer's¹ "glandular fever" was a different disease from that of Sprunt and Evans.² In the former the disease occurred in infants, was very contagious and of short duration, and atypical lymphocytes were not described. The infectious mononucleosis of Sprunt and Evans occurred in adolescents, was not very contagious and lasted longer, and atypical lymphocytes were present.

The diagnosis of infectious mononucleosis formerly depended on finding clinical, haematological, and serological evidence of the disease. Recently, the presence of specific IgM anti-Epstein-Barr virus antibody has been accepted as diagnostic of the disease. This test is of particular value in children and in the elderly, where the Paul-Bunnell-Davidsohn test may be negative and the illness atypical. However, many laboratories use only commercial screening kits (for example, Monospot), which are associated with a number of false-positive results. We have studied the serology of 100 patients with clinical and haematological evidence of infectious mononucleosis.³ All patients had atypical lymphocytes constituting over 20% of the total leucocyte count. Positive