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AIDS: act now, don't pay later

SIR,—Dr D E B Powell (20 September, p 758) maintains that the acquired immune deficiency syndrome is "first and foremost related to male homosexuality" and that health education should carry the wording "homosexual activity may damage your health." He is correct that most cases in the United Kingdom and the United States occur in homosexual men, but this is not so world wide. In certain African countries the sex distribution of sufferers is virtually equal. Surely the important point about the disease is that the main mode of transmission is through sexual intercourse with an infected partner. Health education should concentrate on ways in which this message can be made clear. To use emotive terms about homosexuality damaging health is shortsighted since (a) it supposes that all homosexuals behave in the same way and are at equal risk of acquiring human immunodeficiency virus (HIV) infection and (b) it suggests to heterosexuals that they are not at risk; this is going to be increasingly incorrect.

Dr Powell also raises the question of whether three cases of AIDS in drug abusers constitutes enough of a problem to allow us also to concentrate on this group. The important issue is not the total number of cases but the prevalence of antibodies to HIV in such populations. So far HIV infection in this group has been more of a problem in the United States and Europe. Last year surveys indicated that 76% of addicts in Italy (the figure was 6% in 1980) and 32% in Switzerland had antibodies to HIV. In the UK this was 10% (a fourfold increase in one year) but with pockets of much higher rates, such as 50% in Edinburgh. The next wave of the epidemic of AIDS in the UK will undoubtedly occur in addicts. This is why we need to concentrate on them and produce effective health education.

We must not allow ourselves to make the same mistake as occurred with the rising prevalence of HIV in homosexual men. AIDS did not start to occur in homosexual men in America until the prevalence of HIV had reached 20-25% in 1982. At that time probably only about 4% of London's homosexual population had antibodies to HIV, yet we did not mount a national health education programme until 1986, when the prevalence was over 25% and more than 300 cases had occurred. We ignored the American experience and did not act at an early stage; and we must not let this happen again. We need to act now with effective health education and not when the three cases are 30 or 300. Then control will be so much harder.

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SIR,—Dr D E B Powell (20 September, p 758) does a great disservice to medicine and the acquired immune deficiency syndrome (AIDS) by assuming that homosexual activity and community are the only sources of AIDS. Attitudes like this in the

United States delayed research grants and work on AIDS by years. We have only to look at the accounts of AIDS in Africa^{1,2} and the United States to avoid making fatuous assumptions and comments. If we are unable to learn from the experience of other nations no doubt we will end up paying more, as you suggest in your excellent and timely leading article (9 August, p 348). We have to accept that AIDS is here and does not respect sex or sexuality. Available information indicates that 3-4% of the current caseload in North America and Europe has arisen from heterosexual spread.¹ How can Dr Powell assume that if homosexuality was "cured" AIDS would disappear? We have to be dispassionate about this debate and not make the sexuality of an individual an issue. One of the reasons why help (financial and otherwise) has not been forthcoming is because the "moral majority" dictates terms. It is not a homosexual disease. Had the disease initially attacked heterosexuals and then spread to other groups I wonder if Dr Powell would have reacted by saying: "Heterosexual activity may damage your health"?

Sufferers of AIDS need help and they need it now, along with our compassion and understanding. Everyone needs information and education on AIDS and needs it now if we are to make any headway in combating the disease. The medical profession with its longstanding liberal traditions needs to be educated perhaps even more than the man on the Clapham omnibus.

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Psoriasis

SIR,—Dr R H Champion's admirable review of psoriasis (28 June, p 1693) and the subsequent letter from Dr C E M Griffiths and colleagues (26 July, p 266) has highlighted the difficulty investigators engaged in psoriasis research face in deciding whether the particular anomaly they are investigating represents a genuine primary defect or simply a secondary effect. Unfortunately, despite claims to the contrary,^{1,2} the cause of the characteristic epidermal proliferation present in psoriasis remains elusive.

Dr Janet Marks's comments (23 August, p 509) on the clinical effects of cyclosporin A are interesting, particularly her reference to clinical efficacy at dosages unlikely to affect the immunological mechanisms postulated for the pathogenesis of psoriasis.³ A possible alternative explanation for cyclosporin A's activity in psoriasis already exists, since cyclosporin A has been shown to be a potent inhibitor of the intracellular calcium dependent regulator protein calmodulin—a property which may be crucial to its action on T lymphocytes.⁴ This may be relevant, because the calmodulin content of psoriatic epidermis has been shown to be about twice that found in normal controls⁵ and to fall once the psoriasis is treated.⁶ Recently dithranol, one of the principle drugs used topically in psoriasis, has been reported to be a powerful calmodulin inhibitor.⁷ Calmodulin regulates a wide range of calcium dependent processes within the cell,⁸ and binding of cyclosporin A to calmodulin may prevent effective activation of the second messengers and enzymes required for keratinocyte proliferation.

Calmodulin also activates phospholipase A₂, the enzyme responsible for the release of arachidonic acid from cell membranes, thereby regulating the production of the inflammatory eicosanoids. Since

phospholipase A₂ levels are increased in psoriatic epidermis,⁹ calmodulin inhibition should theoretically be able to modify the inflammatory component of psoriasis. This may explain the rapid effect of cyclosporin A on erythrodermic psoriasis noted by Dr R A Wakeel and Dr D C Disk (26 July, p 266).

It is of interest that the antifungal drug ketoconazole has recently been shown to be an effective treatment for scalp psoriasis.¹⁰ Although the clinical improvement seen has been attributed to inhibition of the growth of pityrosporon yeasts, calmidazolium (R-24571, Janssen Pharmaceuticals), a derivative of another antimycotic imidazole, miconazole, has proved to be such a potent calmodulin antagonist that it is marketed purely for this purpose (as a laboratory reagent).

A wide range of drugs possess calmodulin inhibitory properties, offering a number of considerably less toxic alternatives to cyclosporin A. Whether or not other calmodulin inhibitors may prove to be clinically useful in psoriasis remains uncertain, however, since some drugs with calmodulin antagonistic activity, such as propranolol, appear to aggravate psoriasis. Nevertheless, the remarkable therapeutic effectiveness of cyclosporin A in psoriasis shows that these agents deserve further investigation.

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Discrimination against singlehanded general practitioners

SIR,—We find it incredible that the latest amendment to the Statement of Fees and Allowances for General Practitioners (the Red Book), of July 1986 (SFA 126), has included yet another incentive for GPs to join together to form a group. Page 18a of the Red Book now states that doctors in selected areas (essentially those recognised by the government's inner city policy) who join together to form a group of three or more doctors after 1 April 1984 will be eligible for an incentive payment of up to £4000 per doctor spread over a year.

We are not aware of any evidence that states clearly that group practices provide a better stan-