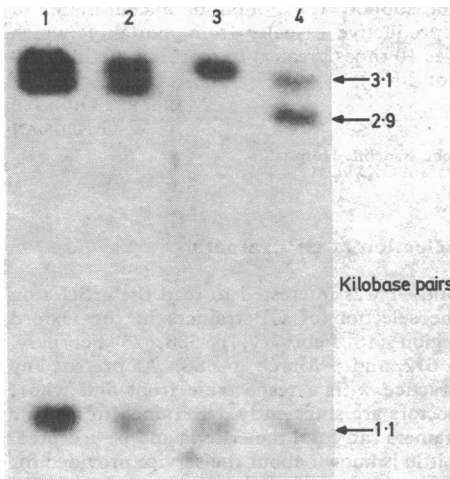


- ¹ Sykes BC. A high frequency Hind III restriction site polymorphism within a collagen gene. *Disease Markers* 1983;1:141-6.
- ² Cheah KSE quoted in Sykes BC. Collagen and inherited connective tissue disease. *Nature* 1983; 305:764.

*Dr Pope and Dr Nicholls reply below.—
Ed, *BMJ*.

SIR,—As there was no indication that broad boned lethal osteogenesis imperfecta is more common in any particular racial group we considered the combined control samples we used were adequate. We had seen at least three affected Anglo-Saxon patients with the deletion, but no Anglo-Saxon control carried a similar change. The probability that three consecutively affected patients would show the deletion is greater than chance alone and would be at least $(1/400)^3$ —that is, 1/64 000 000. If Dr Sykes and Mr Ogilvie's observation is confirmed, however, then a frequency of 10% among Asian Indians would increase the chance of three consecutive babies with osteogenesis imperfecta carrying the deletion to the much less impressive figure of $(1/10)^3$ —



Double Eco RI and Bam HI digestions of DNA from patients with osteogenesis imperfecta congenita and control. The 4.3 kb Eco RI fragment shown in the figure of Dr Sykes and Dr Ogilvie is cleaved into 3.1 kb and 1.1 kb fragments. Similarly the 4.0 kb fragment becomes 2.9 kb and 1.1 kb respectively. Tracks 1, 2, and 4 show patients with osteogenesis imperfecta. Track 3 is a normal control. The pattern shown in track 4 is similar to that described in our earlier paper. Tracks 1 and 2 show variations in which the abnormal allele produces 3.0 and 1.1 kb fragments.

that is, 1/1000 in that population. There are nevertheless several potential flaws in the argument.

Firstly, joint hypermobility and blue sclerae are common among Asian Indians so that perhaps collagen gene abnormalities are also frequent.¹ Their effects need not necessarily be disadvantageous in heterozygotes as the heterozygote parents in our study were to all intents and purposes clinically normal. Our evidence suggests that it is a combination of the 300 base pair deletion with another unidentified gene defect which may result in osteogenesis imperfecta. We obviously have no indication as to the frequency of the latter mutant in the general population.

Secondly, in our patients digestion with Eco RI, Bam HI, and other enzymes² has confirmed an actual deletion of a segment of DNA.² We assume that Dr Sykes and Mr Ogilvie's studies are similar, although they illustrate only a single Eco RI digestion. Although their patients show apparently clinically neutral changes there is no certainty that these are identical to our observations in osteogenesis imperfecta congenita. We already have evidence for at least two separate deletions in this disease group (figure), and others (relevant or irrelevant) are certainly possible. Cloning and sequencing of the deletion that we have observed will finally answer the speculation whether it lies within the collagen gene. Similar studies will also be required to assess the importance of Dr Sykes and Mr Ogilvie's observation.

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- ² Pope FM, Weiss E, Nicholls AC. Collagen $\alpha 1(I)$ -like gene defects in Ehlers-Danlos syndrome and severe lethal osteogenesis imperfecta. *J Invest Dermatol* 1983;80:324-5.

Psychosis after cannabis abuse

SIR,—We question the assertion by Dr M W P Carney and others (7 April, p 1047) that "Psychosis after abuse of cannabis is well recognised. . ."

Most published studies, including those cited by Dr Carney and others, are either anecdotal single case studies or unsystematic series. Altman and Evenson highlighted the pitfalls inherent in these types of study by showing that of 158 psychiatric admissions 38 were preceded by cannabis intake and 123 by watching a late night television movie.¹ No attempt is made in such series to establish that "psychosis" is more common in cannabis users. They fail to show a statistically significant correlation between the two, let alone a causal link.

Such criticisms might partially be offset if the psychoses had enough common features to warrant the label of a clinical syndrome. A causal mechanism would then be possible (as in amphetamine psychosis). The published studies, however, including that of Dr Carney and others, describe a heterogeneous group of reactions with no clue to the cause.

Even assuming that there is an association between cannabis and psychiatric disturbance it is possible that in an established cannabis user a condition with hypomanic features might lead to an increased intake. We have observed two such patients recently, and an account of four others has been published.²

There are two valid reports of attempts to correlate cannabis use with mental illness.^{3,4} One study of longstanding users and matched controls found no significant differences between the groups in the incidence of psychiatric problems.³ The other study found a higher incidence of psychopathology in cannabis users, but this preceded the first use of the drug.

There is a worrying tendency for psychotic episodes in Afro-Caribbeans, especially Rastafarians, to be attributed to cannabis. This may lead the psychiatrist to overlook a treatable

primary functional psychotic disorder or an acute psychotic reaction.⁵

We are concerned that the term "cannabis psychosis" has crept into medical reports without justification. We suggest that the methods used to show its existence are as valid as those used in the last century to establish "masturbation insanity."

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- ² Harding T, Knight F. Marijuana-modified mania. *Arch Gen Psychiatry* 1973;29:635-7.
- ³ Halikas JA, Goodwin DW, Guze SB. Marijuana use and psychiatric illness. *Arch Gen Psychiatry* 1972; 27:162-5.
- ⁴ Beaubrun MH, Knight F. Psychiatric assessment of 30 chronic users of cannabis and 30 matched controls. *Am J Psychiatry* 1973;130:309-11.
- ⁵ Littlewood R, Lipsedge M. Some social and phenomenological characteristics of psychotic immigrants. *Psychol Med* 1981;11:289-302.

SIR,—I am pleased to see that British psychiatry is at last recognising psychosis after cannabis use. The experience of Dr M W P Carney and others is exactly that of most psychiatrists in the Caribbean.

Between March 1983 and March 1984 I saw five cases of psychosis following heavy use of cannabis in an island population of 6500. The patients were all men in their 20s. Two presented with violent manic bouts, and three had schizophreniform symptoms. All had normal premorbid personalities, and one had had a similar manic episode two years previously—again following a bout of heavy cannabis smoking. All the episodes resolved in under three weeks.

Such events are a normal part of psychiatry in the Caribbean, and I wonder why this condition is not recognised so readily in Britain. One possible explanation is the widespread home cultivation of *Cannabis sativa* L, particularly among Rastafarians. The cannabis here is therefore readily available in an unadulterated form giving a higher average dose than in Britain. This research should provide interesting material for the debate about cannabis legalisation.

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Psychiatry and violent offenders

SIR,—Dr Richard Smith (28 January, p 310) points out that both prisoners and judges have inflated ideas about what psychiatry might achieve in treating the mentally abnormal offender. As an example of this point I recently had a prisoner who had referred himself for treatment.

The prisoner was a 29 year old single, unemployed man who was serving a three month sentence for assault. He came from a united but socially and emotionally deprived background; he had suffered from secondary enuresis until 15, and had often truanted from school. He joined the army at 17 and was often in trouble due to violence. He was eventually discharged from the army at 26 after assaulting and threatening to shoot an officer. For the past three years he has been unemployed, living rough, and abusing both alcohol and illicit drugs. He has frequently been convicted of violent crimes. On examining his mental state