Coronary risk factors in people from the Indian subcontinent living in West London and their siblings in India

Deepak Bhatnagar, Inder S Anand, Paul N Durrington, Deven J Patel, Gurpreet S Wander, Michael I Mackness, Francis Creed, Barbara Tomenson, Y Chandrashekhar, Melanie Winterbotham, Reginald P Britt, Julian E Keil, George C Sutton

Summary

Several reports have shown that migrants from southeast Asia tend to have an increased risk of coronary heart disease when settled in their new country. We compared coronary risk factors in a randomly selected group of 247 migrants from the Indian subcontinent of Punjabi origin living in West London and 117 of their siblings living in the Punjab in India.

The West London cohort had a greater body mass index (p<0·001), systolic blood pressure (p=0·0087), serum cholesterol (p<0·001), apolipoprotein B (p<0·001), lower high-density lipoprotein cholesterol (p<0·05) and higher fasting blood glucose (p<0·05) than their siblings in the Punjab. Insulin sensitivity, derived from the homoeostatic assessment mathematical model, was lower in men in West London than in their counterparts in India (p<0·05). Indians in West London had lower β cell function than those in the Punjab (p<0·001). Serum lipoprotein (a) concentrations were similar in both the West London and Punjab population, but were significantly higher (p=0·01) than those of white European populations in the UK.

Increases in serum cholesterol after migration from India lead to increased coronary risk conferred by high serum lipoprotein (a) concentrations and greater insulin resistance. Such between-country comparisons are an important means of establishing the importance of coronary risk factors.

Lancet 1995; **345:** 405–09

See Commentary page 401

Departments of Cardiology and Haematology, Hillingdon Hospital, Uxbridge, Middlesex, UK (D J Patel MRCP, M Winterbotham BSc, R P Britt FRCPath, G C Sutton FRCP); **University Departments of Medicine and Psychiatry, Manchester Royal Infirmary, Oxford Road, Manchester M13 9WL, UK** (D Bhatnagar MRCP, M I Mackness PhD, F Creed FRCPsych, B Tomenson BSc, P N Durrington FRCP); **Department of Cardiology, Post Graduate Institute of Medical Education and Research, Chandigarh, India** (I S Anand FRCP, G S Wander MD, Y Chandrashekhar MD); **and Department of Epidemiology, Medical University of South Carolina, Charleston, South Carolina, USA** (Prof J E Keil MD)

Correspondence to: Dr D Bhatnagar

Introduction

Several reports show that migrants from the Indian subcontinent have an increased risk of coronary heart disease (CHD).[1] Often their CHD risk rises above that of the society to which they have migrated[2] despite the fact that their serum total cholesterol is similar to or lower than that of the indigenous population.[3] Other coronary risk factors such as cigarette smoking are also similar. McKeigue and co-workers have suggested that the excess mortality rates of CHD that occur in an immigrant Indian population, which cannot be fully explained on the basis of conventional risk factors, may be partly due to decreased insulin sensitivity,[4] which may be the result of genetic differences.[5] However, even insulin sensitivity cannot fully account for the increased coronary mortality in Indians.[6]

Serum lipoprotein (a) (Lp(a)) is another inherited CHD risk factor that has not been investigated in migrant Indians. Serum Lp(a) is largely unaffected by diet or drugs[7] and is a particular risk factor when associated with increased serum low-density-lipoprotein cholesterol.[8,9] Apolipoprotein (a), which is a constituent protein of Lp(a), has considerable homology with plasminogen and may interfere with fibrinolysis. Furthermore this protein is retained in the arterial wall and thus contributes to atherogenesis.[10]

There are scanty data about the prevalence of CHD in India,[11] but in view of the physical geography, size, and cultural diversity of the country there will inevitably be variations. The predominantly rural population in India is, however, likely to have a lower prevalence of CHD than that seen in the UK.[12] The reports of increased mortality in Indian migrants to the UK from such communities reflect the interplay of the newly acquired nutritional or emotional environment against a background of genetic susceptibility.

Subjects and methods

UK

We carried out our study in the Northcote ward of Southall in West London (population 12 858), where 83% (n=8764) of residents in the 1991 census came from the Indian subcontinent.[13] We assessed 485 subjects with Indian surnames who were randomly selected from the electoral register. Fasting blood samples were taken on-site and analysed in Manchester.

Siblings in India

One-third of participants in West London gave sufficiently detailed information for one or more siblings still living in India to be contacted. These siblings were investigated in a similar way to the West London cohort by a team of local investigators (GSW, ISA, YC). Recruitment of siblings was limited to the state of Punjab in India. In India the study was coordinated from the

Department of Cardiology at the Post Graduate Institute of Medical Education and Research, Chandigarh. A field team of doctors and technicians went to a specific area or village in a district after initial postal confirmation of assent for the study. Fasting blood samples were taken on-site and transported at 4°C to the laboratory in Chandigarh.

Protocol

All subjects had a clinical examination. A recording was made of the blood pressure, and patients completed a supervised bilingual questionnaire about symptoms of CHD, neuroses, and environmental stresses. Blood pressure was measured after the subject had been seated for 5 min. Blood samples were taken after an overnight fast for the determination of serum cholesterol, triglycerides, high-density-lipoprotein (HDL) cholesterol, apolipoprotein B (apo B), Lp(a), insulin, and blood glucose. In the UK and India blood was centrifuged on the day of collection and serum separated into aliquots. Before freezing aliquots, we used one of them to isolate HDL using the same batch of reagents for heparin-$MnCl_2$ precipitation in India and Manchester.[14] HDL and serum were then frozen at −70°C before analysis.

Laboratory methods

The frozen samples from West London and Chandigarh were transported to Manchester on dry ice. Cholesterol in serum, and HDL, serum triglycerides, insulin, apo B and Lp(a) were measured in the Department of Medicine in Manchester. To ensure that there were no changes in the lipid concentrations because of transportation, another aliquot of serum was used to analyse samples with similar analytical methods in Chandigarh. Site visits to the laboratory in India were done by one of us (DB). Reagents and standards used for analysis were obtained in the UK and were shipped to India with appropriate precautions. Both laboratories participated in external quality-assurance schemes for serum lipid determinations; their performance was satisfactory. Aliquots of the same quality-control material used in Manchester were sent to India for within-batch analysis and showed no significant differences in performance between the laboratories for any of the variables. There was close agreement between values; mean (SE) cholesterol values measured in Chandigarh and Manchester were 5·3 (1·2) and 5·0 (1·1) mmol/L, respectively, and median (range) triglycerides were 2·10 (2·56–8·44) and 2·08 (2·49–7·34), respectively.

Serum and HDL-cholesterol were measured by a CHOD-PAP method and triglycerides by a GPO-PAP method, both on a discrete analyser. Apo B was measured by rate immunonephelometry on the Beckman Array (Beckman, Palo Alto, USA).[15] Serum Lp(a) was measured by a two-site immunoradiometric assay (Pharmacia, Milton Keynes, UK[16]) calibrated in the Manchester laboratory against the protein concentration of a preparation of Lp(a) isolated from human serum.

Serum insulin was determined by an assay based on a modification of the double antibody method of Morgan and Lazarious[17] and was validated before use by the substitution of standards with the WHO first international reference preparation of human insulin and through human-derived control sera (Lyphochek, Bio Rad Laboratories, Watford, UK). Blood glucose was analysed by the same glucose oxidase method in India as in the UK.

We obtained a measure of insulin sensitivity and β-cell function from fasting blood glucose and insulin by the homoeostasis assessment mathematical model (HOMA).[18] The calculations are based on the assumption that control of plasma glucose and insulin concentrations in the fasting state is determined by a self-contained feedback loop that involves the liver, β-cells, and both insulin-sensitive and insulin-insensitive tissues. Insulin sensitivity is expressed as a percentage of that in a healthy, lean reference population of European descent living in the Oxford area who were assigned a value of 100% for insulin sensitivity and β-cell function by the inventors of the method.[18]

	Men		Women	
	West London	Punjab	West London	Punjab
Number	118	65	129	52
Age (yrs)	46·0 (10·6)	44·4 (9·4)	45·6 (9·4)	45·8 (8·9)
BMI (kg/m²)	26·8 (5·2)	22·9 (4·7)†	27·4 (4·9)	22·7 (4·0)†
Serum cholesterol (mmol/L)	6·5 (1·4)	4·9 (1·1)†	6·2 (1·2)	5·1 (1·0)†
Serum triglycerides‡ (mmol/L)	2·10 (1·88–2·35)	2·06 (1·78–2·38)	1·66 (1·49–1·85)	1·71 (1·45–2·01)
HDL cholesterol (mmol/L)	1·12 (0·45)	1·21 (0·43)	1·16 (0·42)	1·34 (0·39)*
Serum apo B (mg/dL)	103·6 (30·2)	62·5 (17·2)†	91·8 (24·3)	65·5 (18·3)†
Serum Lp(a)‡ (mg/dL)	18·8 (15·3–23·0)	17·4 (13·7–22·1)	20·4 (16·9–24·5)	18·9 (13·9–25·8)
Systolic blood pressure (mm Hg)	146 (23)	132 (22)†	143 (28)	142 (23)
Diastolic blood pressure (mm Hg)	93 (14)	87 (12)	86 (14)	88 (10)

*P<0·05; †p<0·001; ‡geometric mean and 95% CI. BMI=body mass index.

Table 1: **Clinical characteristics, serum lipids, apolipoproteins, and blood pressure in Indians living in West London and their siblings in the Punjab**

Statistical analysis

Data from questionnaires, clinical examination, and laboratory determinations were stored on a computer database at the University of Manchester. Statistical analyses were performed with a Statistical Software Package. Serum triglycerides, Lp(a), insulin, β-cell function, and insulin sensitivity had log normal distributions and are presented as geometric mean with 95% CI. Group means were compared by *t* test or analysis of variance on actual data for normally distributed variables and on log-transformed data for log-normal variables. The association between variables was tested by Pearson's correlation coefficients.

Results

West London population

Of the 485 individuals approached in Southall, 390 agreed to participate in the study (response rate 81%). Complete data were available for 376 participants. One individual who was profoundly hyperlipidaemic (serum cholesterol >12 mmol/L; triglycerides >30 mmol/L) was excluded from the analysis. Since all Indian siblings were from the Punjab, all comparisons are restricted to West London subjects who were originally from the Punjab (n=247; 118 men 129 women). In this Punjabi population 73·7% were Sikhs, 17·8% were Hindus, 6·8% were Muslim, and 1·7% were Christian. The results of the serum lipids and apolipoprotein measurements in the remaining West London population did not differ from the West London cohort who originated from the Punjab.

Siblings in the Punjab

We contacted siblings of 59 West London subjects. There were no differences between these subjects and either the rest of the West London study population or the West London cohort born in the Punjab. On average 2 siblings were contacted for each index subject, so demographic data and blood samples were obtained from 120 siblings from the Punjab. Complete and verifiable data were available on 117 subjects from India (66 men and 54 women), who were the siblings of 59 of the West London sample. 52% were Sikh, 43·1% were Hindu, and 4·6% were Christian.

The mean age of subjects in India and in West London was similar (table 1) and there were no differences in age between the sexes either in India or in West London.

Table 1 shows that men and women from the Punjab had a significantly lower body mass index (kg/m²) than

	Men		Women	
	West London	Punjab	West London	Punjab
Number	118	65	129	52
Blood glucose	5·7±1·4	4·5±1·0†	5·1±1·2	4·7±0·8*
Serum insulin‡	8·4 (7·2–9·8)	6·7 (5·4–8·4)	8·0 (7·0–9·1)	8·9 (7·5–10·6)
Insulin sensitivity‡	45·4 (39·1–52·7)	59·9* (47·8–74·9)	47·9 (42·1–54·6)	45·3 (38·1–54·0)
β-cell function‡	86·0 (75·2–98·3)	134·1† (114·1–157·6)	108·4 (97·4–120·7)	156·7† (135·4–181·3)

*P<0·05; †p<0·001; ‡geometric mean and 95% CI.

Table 2: **Blood glucose, serum insulin, β-cell function, and insulin sensitivity in Indians living in West London and their siblings in the Punjab**

those living in West London (22·8 [95% CI 21·9 to 23·7] *vs* 27·1 [26·5 to 27·8]; p<0·001). They also had significantly lower total serum cholesterol (p<0·001) and higher HDL cholesterol (p=0·018, for women only). Serum triglycerides were similar in both groups. Apo B concentrations were significantly lower in subjects from the Punjab than in those from West London (p<0·001), but serum Lp(a) concentrations were not significantly different (table 1).

Indian men in West London had significantly higher systolic and diastolic blood pressures than men in the Punjab (p<=0·001 and p=0·0068, respectively), but there were no significant differences between women in West London and those in the Punjab.

The mean blood glucose was significantly lower in subjects in the Punjab than in West London both for men and women separately and together (p<0·001). Based on a cut-off point of 6·6 mmol/L, 15% of subjects in West London (n=37) were diabetic compared with 3% in the Punjab (n=4) (χ^2=10·62, df=1, p=0·0011).

Men in the Punjab had significantly greater insulin sensitivity than those in West London (p=0·035), but women had similar results in both locations. β-cell function was significantly higher in Punjab than in West London in both men and women (p<0·001) (table 2).

Blood pressure, insulin sensitivity, and body mass index

In Indian men in both West London and the Punjab insulin sensitivity correlated with diastolic blood pressure (r=−0·16, p=0·035 and r=−0·44, p=0·001, respectively). In addition in the Punjabi men, there was a correlation between insulin sensitivity and systolic blood pressure (r=−0·43, p=0·001). Insulin sensitivity was inversely related to body mass index in Indian men in the Punjab (r=−0·55, p<0·001) and West London (r=−0·20, p<0·05). For Indian women living in the Punjab, insulin sensitivity was not significantly correlated to blood pressure, but in those living in West London correlations with systolic blood pressure (r=−0·17, p=0·029) and diastolic blood pressure (r=−0·17, p=0·022) were observed. However body mass index was correlated with insulin sensitivity in women in West London (r=−0·29, p=0·0001) and in the Punjab (r=−0·33, p=0·032).

Sibling pairs

To investigate further the similarity of Lp(a) concentrations in the two populations we matched 30 subjects from West London with their siblings in the Punjab who were of the same sex and of simlar age (45·7 [9·6] *vs* 44·6 [8·1]) and carried out paired t tests between the two groups for each variable. The Punjab siblings had a lower body mass index (p=0·001), serum cholesterol (p<0·001), and apo B (p<0·001), whereas blood glucose (p=0·069), HDL cholesterol (p=0·55), insulin sensitivity (p=0·12), whereas β-cell function (p=0·55), serum Lp (a) (p=0·11), systolic blood pressure (p=0·13), diastolic blood pressure (p=0·47), and triglycerides (p=0·27) were not statistically different. The only significant correlation observed was between serum Lp(a) of the matched siblings (Pearson's r=0·45; p=0·036) indicating a substantial element of heritability.

Insulin sensitivity and β -cell function

Insulin sensitivity in Indian men in West London was reduced compared with those in the Punjab (p<0·05), but there were no differences in the women. β-cell function was decreased in both men and women in West London compared with those in the Punjab (p<0·001).

Comparison with data from subjects of European descent

Serum Lp(a) Serum Lp(a) concentrations in people from the Indian subcontinent were compared with those available for three European groups, the analysis for which were done in the Manchester laboratory by the same method. All three Europid populations had similar Lp(a) values (table 3); however, Indians in West London and in the Punjab had significantly higher Lp(a) than any of the populations of European descent in the UK (p=0·001).

Discussion

We have shown that migrants from the Indian subcontinent to the UK have a less favourable coronary risk profile compared with their siblings who did not migrate. The migrants had higher serum cholesterol, apo B, and blood glucose and lower HDL cholesterol concentrations. Findings were similar in matched sibling pairs and when migrants from the Punjab as a whole were compared with siblings as a whole. Serum Lp(a) concentrations, which are to a great extent determined genetically, were similar in both Indian populations.

An important observation in this study is the higher serum Lp(a) concentrations in Indians in West London and in the Punjab compared with white European populations in Britain. Serum Lp(a) is generally thought to be a risk factor for coronary artery disease when the serum cholesterol concentrations are as high as in the British European population.[8] In populations originating from Africa serum Lp(a) concentrations are also higher than in those in Europids in Britian,[19] but in both Africans

	Indian populatxions		European populations		
	West London	Punjab	Caerphilly	Oxfordshire	Manchester
Number	376	117	1808	227	137
M/F	194/182	65/52	1808/0	161/66	100/37
Age (yr)	45·3 (30–65)	45·0 (30–65)	56 9 (48–66)	67·6 (20–80)	48·9 (20–74)
Serum Lp(a) (mg/dL)	18·5* (16·5–20·8)	18·1* (15·0–21·8)	10·0 (9·7–10·6)	12·4 (7·5–17·4)	8·2 (5·5–9·0)

*p<0·05. Data are geometric mean and 95% CI.

Table 3: **A comparison of serum Lp(a) in Europeans and in Indians living in West London and their siblings in the Punjab**

living in Africa and those living in the US the incidence of coronary artery disease is less than that seen in caucasians.[20] This is probably because Africans and their descendants have lower LDL cholesterol concentrations than people of European descent. Indians, on the other hand, would appear to have higher Lp(a) and to have the capacity to rapidly acquire the higher LDL cholesterol of Europids on emigrating to a country like Britain. Therefore, diet and lifestyle-induced increases in serum cholesterol in Indians add to the genetic background of a potentially atherogenic and thrombogenic state due to high serum Lp(a).

McKeigue et al[4] have proposed that insulin resistance is an important factor that may explain the increased frequency of coronary artery disease seen in Indians. Our results support earlier investigations showing that Indians are more insulin resistant then Europeans and further suggest that Indians are insulin resistant even before migration. However, this conclusion must be tempered by the knowledge that the European population with which this comparison was made was non-obese. We can, however, draw much stronger conclusions from the comparison of the Indians in Britain and in India and it seems clear from this that there is both a deterioration in insulin resistance and β-cell function associated with migration.

As in other populations, insulin resistance is accompanied by other CHD risk factors such as increased serum triglyceride, low HDL cholesterol, and central obesity.[21] All these factors are more prevalent in Indian imigrants in several geographical locations.[3,22-24] Our study contributes to the question of whether South Asians develop insulin resistance before migration or whether they develop it through unaccustomed nourishment afterwards. Decreased insulin sensitivity was present in the Punjabi population who had not migrated. This finding does not neccesarily mean that Indians are genetically predisposed to insulin resistance because Barker and Hales have suggested that a thrifty phenotype possibly develops in response to in-utero nutritional deprivation and that such imprinting may offer protection during chronic undernutrition.[25] These theories have yet to be tested in people from the Indian subcontinent, most of whom have children of smaller birth weight (even in affluent families) and, as adults, generally have a lower body mass index compared with white Europeans.[26] Especially relevant would be the degree of insulin resistance of the children of Indians resident in the UK. Whatever the explanation of the insulin insensitivity in Indians the likelihood that it will be expressed as glucose intolerance and frank diabetes probably increases when their dietary intake of energy and fat increases on migration to countries such as the UK. The apparent deterioration in pancreatic secretory capacity in the migrant Punjabis in this study would also reward further study in this context.

Immigrant men in West London had higher systolic blood pressures than their counterparts in the Punjab. These observations are consistent with earlier findings in Indians migrating to London and remaining in India.[27,28] As with our study, there was also a relation between body mass index and blood pressure. The inverse relation we found between blood pressure and insulin sensitivity in Indian migrants in the present study has generally been reported in European populations,[29,30] but there is also a report in Hong Kong Chinese.[31]

In conclusion, body weight, serum cholesterol, and blood pressure are increased in Indians, who migrate to the UK. They already have higher serum Lp(a) than the indigenous population and this is unaffected by migration. Their insulin resistance is exposed as an increase in blood glucose and decrease in HDL cholesterol. On migration they acquire a CHD risk profile that is similar to that of the host community and which unmasks the underlying genetic risk of coronary heart disease conferred by high serum Lp(a) and a predisposition to increased insulin resistance. These findings should be helpful in designing public health strategies to reduce underlying CHD risk in Indians in the UK.

We are grateful to Dr C Gordon for carrying out the insulin assays, to Dr S Kumar for HOMA calculations, and to Mrs Hilary Prais and Mrs Barbara Haines for skilled technical assistance.

The data on serum Lp(a) concentrations on subjects of European descent (Europids) is used with the permission of the Oxfordshire Stroke Project and the Medical Research Council Epidemiology Unit in Wales.

References

1 Miller GK, Kotecha S, Wilkinson WH, et al. Dietary and other characteristics relevant for coronary heart disease in men of Indian, West Indian and European descent in London. *Atherosclerosis* 1988; **70:** 63–72

2 Balarajan R. Ethnic differences in mortality from ischaemic heart disease and cerebrovascular disease in England and Wales. *BMJ* 1991; **302:** 560–64.

3 Rajadurai J, Arokiasamy J, Pasamanickam K, Shater A, Mei LO. Coronary artery disease in Asians. *Aust NZ J Med* 1992; **22:** 345–48.

4 McKeigue PM, Shah B, Marmot MG. Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in South Asians. *Lancet* 1991; **337:** 382–86.

5 Schumaker MC, Maxwell TM, Wu LL, Hunt SC, Williams RR. Elbein SC. Dyslipidemia among normoglycemic members of familial NIDDM pedigrees. *Diab Care* 1992; **15:** 1285–89.

6 Editorial. Coronary heart disease in Indians overseas. *Lancet* 1986; i: 1307–08.

7 MBewu AD, Durrington PN. Lipoprotein (a): structure, properties and possible involvement in thrombogenesis and atherogenesis. *Atherosclerosis* 1990; **85:** 1–14.

8 Bhatnagar D, Durrington PN. Does measurement of apolipoproteins add to the clinical diagnosis and management of dyslipidaemias? *Curr Opin Lipidol* 1993; **4:** 299–304.

9 Cremer P, Nagel D, Labrot B et al. Lipoprotein (a) as predictor of myocardial infraction in comparison to fibrinogen, LDL cholesterol and other risk factors: results from the prospective Gottingen Risk Incidence and Prevalence Study (GRIPS). *Eur J Clin Invest* 1994; **24:** 444–53.

10 Editorial. Lipoprotein (a). *Lancet* 1991; **331:** 397–98.

11 Chadha SL, Ramachandran K, Skekhawat S, Tandon R, Gopinath. A 3-year follow-up study of coronary heart disease in Delhi. *Bull World Health Organ* 1993; **71:** 67–72.

12 Ramachandran A, Snehlata C, Dharmaraj D, Viswanathan M. Prevalance of glucose intolerance in Asian Indians. *Diab Care* 1992; **15:** 1348–55.

13 Balarajan R. The ethnic populations of England and Wales: the 1991 Census. *Health Trends* 1992; **24:** 113–16.

14 Mackness MI, Durrington PN. Lipoprotein separation and analysis for clinical studies. In Lipoprotein Analysis: A practical approach Eds Converse CA, Skinner ER. Oxford University Press, Oxford, 1992: pp 1–42.

15 Adolphson JL, Albers JJ. Comparison of two commercial nephelometric methods for apo AI and Apo B with standardised apoprotein AI and B radioimmunoassays. *J Lipid Res* 1989; **30:** 597.

16 Durrington PN, Ishola M, Hunt L, Arrol S, Bhatnagar D. Apolipoprotein (a), AI and B and parental history in men with early onset ischaemic heart disease. *Lancet* 1988; i: 1070–73.

17 Gordon C, Yates AP, Davies D. Evidence for a direct action of exogenous insulin on the pancreatic islets of diabetic mice: islet response to insulin pre-incubation. *Diabetologia* 1985; **28:** 291–94.

18 Matthews DR, Hosker JP, Rudenski A, Naylor BA, Treacher D, Turner RC. Homeostasis model assessment: insulin resistance and beta cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia* 1985; **28:** 412–19.

19 Sandholzer C, Hallman DM, Saha N, et al. Effects of apolipoprotein (a) size polymorphimism on lipoprotein (a) concentration in seven ethnic groups. *Hum Genet* 1991; **86:** 607–14.

20 Watkins LO, Neaton JD, Kuller LH. Racial differences in high-density lipoprotein cholesterol and coronary heart disease incidence in the usual care group of the Multiple Risk Factor Intervention Trial. *Am J Cardiol* 1986; **57:** 538–45.

21 Fontbonne AM, Eschwege EM. Insulin and cardiovascular disease. The Paris Prospective Study. *Diab Care* 1991; **14:** 461–69.

22 McKeigue PM, Marmot MG, Court YDS, Cottier DE, Rahman S, Riemersman RA. Diabetes, hyperinsulinaemia and coronary risk factors in Bangladeshis in East London. *BMJ* 1988; **60:** 390–96.

23 Knight TM, Smith Z, Whittles A, et al. Insulin resistance, diabetes and risk markers for ischaemic heart disease in Asian men and non-Asiann men in Bradford. *Br Heart J.* 1992; **67:** 343–50.

24 Enas EA, Yusuf S, Mehta JL. Prevalence of coronary artery disease in Asian Indians. *Am J Cardiol* 1992; **70:** 945–49.

25 Barker DJ, Hales CN, Fall CH, Osmond C, Phipps K, Clark PM. Type 2 (non-insulin dependent) diabetes mellitus, hypertension and hyperlipidaemia (syndrome X): relation to reduced fetal growth. *Diabetologia* 1993; **36:** 62–67.

26 McKeigue PM. Coronary heart disease in Indians, Pakistanis and Bangladeshis: aetiology and possibilities for prevention. *Br Heart J* 1992; **67:** 341–42.

27 Keil JE, Britt RP, Weinrich MC, Hollis Y, Keil BW. Hypertension in Punjab females: Comparison between migrants to London and natives in India. *Hum Biol* 1980; **52:** 423–33.

28 Keil JE, Weinrich MC, Keil BW. Hypertension in a population sample of female Punjabi Indians in Southall. *J Epidemiol Comm Health* 1980; **34:** 45–47.

29 Ferranini E, Buzzigoli G, Bonadonna, et al. Insulin resistance in essential hypertension. *N Engl J Med* 1987; **317:** 350–57.

30 Smith U. Insulin resistance in hypertension. In: Insulin Resistance: Moller DE, ed. John Wiley & Sons Ltd, Chichester, England, 1993: 335–43.

Social services case-management for long-term mental disorders: a randomised controlled trial

M Marshall, A Lockwood, D Gath

Summary

Case management arose in the USA as a solution to the difficulties of providing community care to people with severe mental disorders. The basic principle of the approach is that a case manager takes responsibility for a client; arranges an assessment of need, a comprehensive service plan, delivery of suitable services, and monitoring and assessment of services delivered. The case-management approach has been widely accepted, to the extent that recent legislation has made case-management the cornerstone of community care in the UK.

We did a randomised controlled trial to evaluate a social services case-management team for people with long-term mental disorders. Subjects were referred from hostels for the homeless, night shelters, a general-practitioner clinic for the homeless, the Oxford City Council homelessness unit, and local voluntary-sector group homes. Of 103 subjects referred, 80 consented to be randomised to treatment or control groups. At 14-month follow-up, as assessed by standardised interviews, there were no significant differences between groups in number of needs, quality of life, employment status, quality of accommodation, social behaviour, or severity of psychiatric symptoms. In the case-management group there was a significant reduction in deviant behaviour on a standardised behaviour rating scale (REHAB) (mean=0·79; 95% CI 0·26–1·32).

It is unfortunate, in view of the limited effectiveness we have shown, that social services case-management was not evaluated in randomised controlled trials before its implementation in the UK.

Lancet 1995; **345:** 409–12

See Editorial page 399

Oxford University Department of Psychiatry, Warneford Hospital, Oxford OX3 7JX, UK (M Marshall MRCPsych, A Lockwood RMN, D Gath FRCPsych)

Correspondence to: Dr M Marshall

Introduction

People with long-term mental disorders have many social and psychiatric needs.[1] Since the closure of psychiatric hospitals in the UK, services required to meet these needs have become dispersed among different sites and providing authorities,[2] with the consequence that many people with mental disorders are unable to obtain the care they need.[3] In the USA, case-management arose as a solution to this problem of dispersion; this approach has been widely accepted[4,5] and recently became the cornerstone of community care in the UK. Under the NHS and Community Care Act 1990,[2,6,7,8] the responsibility for implementing case-management in the UK has been given to social services departments, who promote case-management teams to work with the mentally ill.[4,6,9]

In the UK, as elsewhere, the practice, composition, and organisation of case-management teams vary. Some UK teams work entirely within social services departments; others are jointly managed by social services and mental health services, or by mental health services alone.[10] Nevertheless, teams share basic principles of the case-management approach: a case-manager, who takes responsibility for a client, arranges an assessment of need, a comprehensive service plan, delivery of suitable services, monitoring and assessment of services delivered, and also evaluates results.[2,6] The expectation was that, by working alongside existing services, case-management teams would improve the quality and efficiency of care for patients with long-term mental disorders.[11]

The case-management approach has been described as "intuitively appealing",[12] but there is little evidence that it is efficacious. The only randomised trial of an approach comparable to that practised by UK case-management teams was carried out in the USA and showed that the case-management group received more services than the control group and were more often admitted to mental hospitals, but showed no improvements in quality of life.[12]