Strategies for the prevention of psychiatric disorder in primary care in south London

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Abstract
Study objective—To compare the potential impact of high risk and population based approaches to the prevention of psychiatric disorder, using a representative sample of general practice attenders as the target population.
Design—This was a prospective cohort study.
Setting—A health centre in south London.
Participants—Three hundred and seven consecutive attenders aged 16–65, recruited at randomly selected general practice surgeries.
Main results—A linear association was found between the number of different types of socioeconomic adversity reported at recruitment (T1) and the prevalence of psychiatric disorder one year later (T2). The population attributable fraction (PAF) for socioeconomic adversity at T1 was 37.4%. In theory, social interventions for high risk individuals at T1 would reduce the prevalence of psychiatric disorder at T2 by 9% at most, compared with a reduction of 18% if just one item of socioeconomic adversity were eliminated among those with any socioeconomic risk factors.
Conclusions—Social interventions targeted at individuals at highest risk of the most common mental disorders are likely to be extremely limited in their capacity to reduce the prevalence of these conditions. A population based risk reduction strategy, modified according to individual risk, represents a potentially feasible and effective alternative.

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A strong argument exists for developing strategies to reduce the prevalence of the most common mental disorders, anxiety and depression. These conditions have a combined prevalence rate in the community of between 15% and 30%,[1-5] and account for one third of days lost from work due to ill health[6] and one fifth of consultations in general practice in the UK.[7] The public health importance of these disorders, even in mild form, is further shown by the finding that low levels of depression resulted in 51% more days lost from work than major depression.[8] The annual cost of the common mental disorders in the UK may amount to £6 billion, of which two thirds results from lost productivity.[9] Ominously, their prevalence may have increased recently.[10]

The most common preventive approach in psychiatry involves targeting individuals at high risk of disorder,[11-14] which Rose[15] likened to, "attempting to control icebergs by sending warships to shoot off their visible portions". Psychiatrists often reject population based interventions, partly through aetiological uncertainty[15] and partly because these are perceived as either inefficient[13] or synonymous with utopian political change.[16] Many would concur with Goldberg's view that, "it is intuitively easy to see that it would be wasteful to devote resources to populations who are unlikely to get depressed".[13]

A recent report commissioned by the US Congress argued for a population based risk reduction approach.[17] The common mental disorders are associated with forms of socioeconomic adversity that could possibly be corrected, including low income and financial hardship,[18-22] unemployment,[23] poor housing,[24-28] lack of education,[29] and social isolation.[22] In randomised trials, risk factors and (in some cases) the prevalence of common mental disorders have been reduced by interventions to address unemployment,[30] marital separation,[31] bereavement,[32] and teenage pregnancy in the context of socioeconomic deprivation.[33] Such interventions could form the basis of a risk reduction package. Primary care, where up to 40% of attenders suffer from common mental disorders,[34] represents an attractive setting in which to evaluate their potential effectiveness. Only one previous study has evaluated a preventive intervention (training in cognitive behavioural skills) aimed at a population of general practice attenders.[34] This study was restricted to preventing incident cases of depression and failed to produce statistically significant findings because of the rarity of this outcome.

This study aims to add data to what is often a polemical debate. Its objective is to compare the potential impact of high risk and population based approaches to the prevention of psychiatric disorder, using a representative sample of general practice attenders as the target population.

Methods
A prospective cohort study was conducted at a health centre in south London. Study methods have been described elsewhere.[35] Consecutive attenders aged 16–65 years were recruited at randomly selected surgeries. Subjects completed a questionnaire comprising the 12 item general health questionnaire (GHQ-12)[36] and a checklist of recent somatic symptoms before

being approached for baseline interview (T1). Those who completed this interview were approached for a further interview 12 months later (T2).

The interview assessments were the following:

● The computer administered clinical interview schedule (CIS-R),[37 38] using a case threshold of 11/12,
● A computer administered social assessment covering marital status, children, education, employment, personal finances, housing, and physical handicap.

In the social assessment, low income was defined as an adjusted[39] household income below the lowest quartile for the sample. Structural housing problems were defined as one or more of damp, rot in woodwork or infestation. Overcrowding was defined as living in a household where the ratio of people:bedrooms was greater than 2. Physical disability was defined as a physical impairment that interfered with activities of daily living.

Social assessment data were used to construct an 11 item index of current socioeconomic adversity (ICSA-11). Items were forms of objective socioeconomic adversity judged a priori by the investigators to involve unequivocal hardship, and which were thought to be potentially remediable. An ICSA-11 score was derived by scoring one point for each type of socioeconomic adversity. A shortened version of the ICSA-11 (the ICSA-2, see Results) was produced by restricting variables to those that were independently associated with being a case of psychiatric disorder at T2, after adjusting for the ten other ICSA-11 items.

STATISTICAL ANALYSES
Unadjusted odds ratios (95% confidence intervals) for each ICSA-11 item were compared with those adjusted for the other 10 items using logistic regression. Likelihood ratio tests were used to compare linear with categorical models and to test for interactions. Analyses were performed using Stata.[40]

The population attributable fraction (PAF) is an estimate of the proportion of cases at T2 attributable to socioeconomic adversity at T1, and therefore the proportion of cases that could be prevented, in theory, if the association in question is not confounded by other risk factors, if the exposure in question were removed, and if in doing so other risk factors remained constant. PAFs were calculated using risk ratios derived from logistic regression coefficients. Since the prevalence of common mental disorder was too great to satisfy the rare disease assumption, odds ratios were numerically greater than corresponding risk ratios and would have led to overestimates of respective PAFs. In general, for an exposure with multiple levels, the fraction of cases attributable to exposure at level k may be calculated by:

$$PAF_k = p_k'(\theta_k - 1)/\theta_k$$

where p_k' is the proportion of cases at exposure level k and θ_k is the risk ratio for individuals exposed to level k (compared with the baseline group). For example, at ICSA-11 score ≥ 4:

$$PAF = 0.142(2.46 - 1)/2.46 = 0.084$$

Alternatively, the PAF at each level of ICSA score may be estimated by calculating the proportion of cases at T2 that could be prevented (in theory) by reducing the ICSA score to 0. The number of cases that might be prevented by incremental reductions in the ICSA score was calculated as the sum of the differences between the observed number of cases at each level of exposure (eg ICSA-11 score ≥ 4) and the expected number of cases for the same subjects if their individual risk (ie the risk ratio) of psychiatric disorder at T2 were reduced to that of subjects in the adjacent stratum (eg ICSA-11 score = 3). Numbers of expected cases were calculated by multiplying the number of subjects at the higher level of exposure (eg ICSA-11 score ≥ 4 at T1) by the risk ratio for subjects at the next lowest level of exposure (eg risk ratio for subjects with ICSA-11 score = 3), and then multiplying this figure by the proportion of cases at the baseline level of exposure (ie ICSA-11 score = 0). For example, the number of cases prevented by a one point reduction in ICSA-11 score among subjects with a score of ≥ 4 at T1 was calculated thus:

$$Observed\ cases - expected\ cases =$$
$$15 - (24 \times 2.08 \times 0.254) = 2.32\ cases$$

This calculation was done for all levels of ICSA-11 score at T1. The sum of the differences between observed and expected numbers of cases was then divided by the total number of cases at T2, and expressed as a percentage reduction in the prevalence of psychiatric disorder. Finally, the potential impact of social interventions on the prevalence of psychiatric disorder at T2 was estimated for two (hypothetical) target groups as follows:

● A group at high social risk at T1, defined as an ICSA-11 score ≥ 4 (or ICSA-2 score = 2),
● Subjects with any social risk, defined as an ICSA-11 (or ICSA-2) score ≥ 1.

Results
Of 426 attenders who were approached, 404 (94.8%) completed the preliminary questionnaire, 307 (72.1%) completed the first interview (T1), and 261 were re-interviewed 12 months later (T2)—a follow up rate of 85.0%. No statistically significant differences were found between those who did and did not participate at T1 in respect of age, sex, GHQ score, or somatic symptom score. Subjects lost to follow up did not differ to a statistically significant degree from those who completed the T2 interview in respect of age, sex, or either ICSA-11 or CIS-R scores at T1. Of those who completed both interviews, 168 (64.4%, 58.6, 70.2) were women. At T2, 106 subjects were cases of psychiatric disorder, a prevalence rate of 40.6% (34.6, 46.6). The estimated annual incidence rate for psychiatric disorder was 15.7% (10.5, 23.4), and the annual rate of

Table 1 Prevalence of exposure and adjusted odds ratios (OR) (95% confidence intervals) for being a case at one year follow up (T2) in relation to each of the variables included in the index of current socioeconomic adversity (ICSA-11) assessed at recruitment (T1), and OR adjusted for each of the other variables in the table

Variable	% exposed (no)	Unadjusted OR (95% CI)	p	Adjusted OR (95% CI)	p
Single parent	7.3 (19)	3.47 (1.18, 11.48)	0.02	2.86 (0.76, 10.82)	0.12
Low income	23.7 (62)	3.54 (1.86, 6.75)	<0.0001	2.68 (1.22, 5.86)	0.01
Structural housing problems	24.9 (65)	2.69 (1.46, 4.98)	0.001	2.07 (1.07, 4.03)	0.03
Overcrowding	1.9 (5)	0.98 (0.08, 8.74)	1.00	1.53 (0.24, 9.95)	0.66
No educational qualifications	42.0 (129)	1.45 (0.85, 2.47)	0.18	1.26 (0.66, 2.38)	0.49
Physical handicap	7.7 (20)	1.29 (0.47, 3.53)	0.76	1.14 (0.40, 3.26)	0.81
Unemployed (self)	15.3 (40)	0.86 (0.40, 1.80)	0.79	0.99 (0.02, 5.45)	0.98
Seperated/divorced/widowed	13.7 (42)	1.66 (0.80, 3.48)	0.20	0.97 (0.39, 2.40)	0.95
Partner/spouse unemployed	7.3 (19)	1.35 (0.48, 3.74)	0.70	0.94 (0.31, 2.90)	0.92
Debt ≥£1000	15.3 (40)	0.97 (0.46, 2.03)	1.00	0.94 (0.42, 2.09)	0.87
Homeless	0.8 (2)	1.47 (0.02, 115.9)	1.00	0.43 (0.02, 11.27)	0.61

maintenance (ie non-remission) of psychiatric disorder was 68.8% (57.6, 77.1).[35] Psychiatric disorder at T2 was associated with a higher somatic symptom score at index consultation (p<0.001) and older age (p = 0.04).

SOCIOECONOMIC ADVERSITY AND PSYCHIATRIC DISORDER

The frequency of exposure to ICSA-11 items ranged from 42.0% (no educational qualifications) to 0.8% (homelessness), and (unadjusted) odds ratios for psychiatric disorder at T2 ranged from 3.54 (1.86, 6.75) (low income) to 0.86 (0.40, 1.80) (subject unemployed). After adjusting for other ICSA-11 items, only low income and structural housing problems remained independently associated with psychiatric disorder at T2. These two items formed the ICSA-2 (table 1).

Statistically significant linear trends were identified between psychiatric disorder at T2 and both ICSA-11 score (χ^2 for linear trend = 14.15, df = 1, p = 0.0002; χ^2 for departure from linear trend = 0.21, df = 3, p = 0.98) and ICSA-2 score (χ^2 for departure from linear trend = 1.11, df = 1, p = 0.29). Adjusting for CIS-R score at T1 did not alter the linearity of these associations, and had only a small effect on their size, reducing the odds ratio for ICSA-11 score from 1.48 (1.20, 1.81) to 1.30 (1.01, 1.68), and that for ICSA-2 from 2.72 (1.80, 4.11) to 2.33 (1.41, 3.83). A statistically significant positive correlation was found between ICSA-11 score and age (r = 0.32, p<0.001), but not with somatic symptom score at index consultation (p = 0.58). Age did not confound the association between ICSA-11 score and psychiatric disorder at T2 to a statistically significant extent.

THE POTENTIAL IMPACT OF PREVENTIVE STRATEGIES

The total PAF for all socioeconomic adversity as assessed by the ICSA-11 was 37.4%, of which 8.4% was attributable to the highest category of social risk (ICSA-11 ≥ 4). Analysis using ICSA-2 scores yielded similar findings (table 2).

Table 3 contrasts the potential impact of interventions targeted at the entire population of attenders on the one hand, and at those with the highest ICSA-11 scores. In theory, a one point reduction in ICSA-11 score among those at high social risk (ICSA-11 ≥ 4) would result in a 2.2% reduction in the prevalence of psychiatric disorder at T2, compared with 18.0% if the ICSA-11 score of *all* attenders who scored 1 or more on this measure were reduced by the same amount. If all socioeconomic adversity were removed, the maximum predicted reduction in prevalence that could be achieved by the high risk approach would be 8.4% using ICSA-11 (or 8.3% using ICSA-2), compared with a 37.4% reduction (30.6% using ICSA-2) if all those with any socioeconomic risk factors were targeted.

High risk for a continuous exposure may be defined by placing the cut off point anywhere along the continuum of risk. Figure 1 shows that the potential reduction in the prevalence of psychiatric disorder at T2 arising from interventions at T1 is directly proportional to the number of subjects eligible for intervention.

Discussion

The prevalence of psychiatric disorder at T2 (40.6%) was higher than in comparable primary care studies.[41-43] This was unlikely to be due to measurement artefact or bias, since

Table 2 Distribution of study sample and the revised clinical interview schedule (CIS-R) cases at one year follow up (T2), risk ratio (RR) (95% confidence interval (CI)), and population attributable fraction (PAF) in relation to longer and shorter versions of the index of current socioeconomic adversity (ICSA-11 and ICSA-2, respectively) scores at recruitment (T1)

ICSA score at T1	ICSA-11 (range 0–4)				ICSA-2 (range 0–2)			
	% sample (no)	% of T2 cases (no)	RR (95% CI)	PAF%	% sample (no)	% of T2 cases (no)	RR (95% CI)	PAF%
0	24.1 (63)	15.1 (16)	1.00	—	59.8 (156)	41.5 (44)	1.00	—
1	29.5 (77)	26.4 (28)	1.43 (0.85, 2.40)	7.9	31.8 (83)	44.3 (47)	2.01 (1.47, 2.75)	22.3
2	23.4 (61)	26.4 (28)	1.81 (1.09, 2.99)	11.8	8.4 (22)	14.2 (15)	2.42 (1.65, 3.53)	8.3
3	13.8 (36)	17.9 (19)	2.08 (1.23, 3.51)	9.3	—	—	—	—
≥4	9.2 (24)	14.2 (15)	2.46 (1.46, 4.16)	8.4	—	—	—	—
Totals	100 (261)	100 (106)	1.79 (1.14, 2.81)*	37.4†	100 (261)	100 (106)	2.09 (1.56, 2.82)*	30.6†

* RR for any socioeconomic adversity (ICSA score ≥ 1)
† Total PAF for all socioeconomic adversity in study population.

Table 3 Predicted potential reduction in the prevalence of psychiatric disorder at one year follow up (T2) resulting from "high risk" and "population based" social interventions in relation to incremental reductions in the longer and shorter versions of the index of current socioeconomic adversity (ICSA-11 and ICSA-2 respectively) score among those targeted for intervention. The final row indicates the proportion of the study sample who would have been eligible for the intervention (s)

Reduction in ICSA score in target group	ICSA-11 (range 0–4)		ICSA-2 (range 0–2)	
	High risk*	Population†	High risk‡	Population§
−1	2.2	18.0	2.4	24.7
−2	3.7	29.1	8.3	30.6
−3	5.9	35.0	—	—
−4	8.4	37.4	—	—
% attenders eligible	9.2	75.9	7.1	40.1

* ICSA-11 score ≥ 4; † All subjects with ICSA-11 score ≥ 1; ‡ ICSA-2 score = 2; § All subjects with ICSA-2 score ≥ 1.
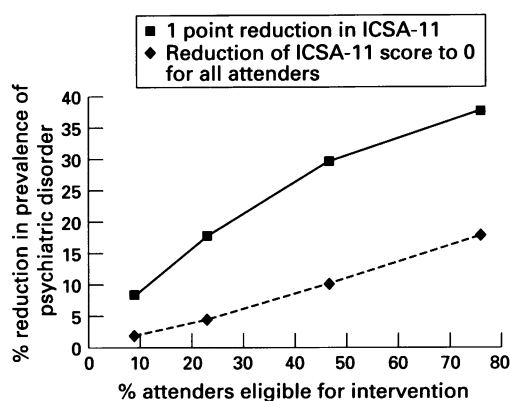

Figure 1 The potential reduction in the prevalence of psychiatric disorder at one year follow up (T2) arising from (1) a one point reduction in the index of current socioeconomic adversity (ICSA-11) score and (2) a reduction in the ICSA-11 to 0 for all eligible subjects against the percentage of attenders eligible for intervention.

the CIS-R has recently been validated in a community survey,[5] and the 12 month incidence rate was similar to that found elsewhere.[44] The high prevalence of psychiatric disorder was most likely to reflect the inner city setting of the study practice,[35] and is unlikely to have influenced either the magnitude or linearity of the association between socioeconomic adversity and psychiatric disorder.

Physical illness was a potential confounder. Despite including physical handicap as a type of socioeconomic adversity (since handicap may be remediable even if the underlying disability is not), we did not find a statistically significant association between the ICSA-11 score and somatic symptom score at T1. Although the severity of somatic symptoms at index consultation did not therefore confound the association of interest, we cannot exclude the possibility of residual confounding by other indices of physical ill health.

PREVENTION OF THE COMMON MENTAL DISORDERS

Our results are consistent with previous evidence of associations between rates of common mental disorders and both low income [18–22 24] and poor housing.[24–28] Previous findings from this study show that while socioeconomic variables such as low household income were associated with a worse outcome among prevalent cases at baseline, such variables were only weakly associated with the incidence of psychiatric disorder after adjusting for potential confounders. By contrast, family psychiatric history and the severity of psychiatric symptoms at T1 were independently associated with the incidence psychiatric disorder after adjusting for other risk factors, including measures of previous psychiatric disorder.[35] Thus, socioeconomic interventions may be of greater importance in secondary than primary prevention.

As expected, most cases of psychiatric disorder at T2 occurred in individuals at low social risk at T1.[45] The association between ICSA-11 score at T1 and the prevalence of psychiatric disorder at T2 was linear and, as figure 1 shows, the potential predicted reduction in the prevalence of psychiatric disorder achievable by socioeconomic interventions was directly proportional to the proportion of attenders targeted for intervention. Thus, interventions targeted at those with the highest social risk in this study (defined as an ICSA-11 score ≥ 4) are likely to be extremely limited in their impact on the prevalence of psychiatric disorder, since only 9% of attenders fell into this category. At the other extreme, it was predicted that 18% of cases at T2 could have been prevented, in theory, if the ICSA-11 scores of the three quarters of attenders with a score of 1 or more at T1 were reduced by just one point.

There are three important caveats. Firstly, the findings are population specific. While the risk ratio is likely to remain constant across populations, the incidence and maintenance rates and the PAF will decline as exposure to socioeconomic adversity falls. The incidence and maintenance of common mental disorders will be lower in less socially deprived areas, and a smaller proportion of all cases will be attributable to socioeconomic adversity. Secondly, we have assumed that effective interventions exist to remove the socioeconomic adversity covered by our index, and that such interventions would reduce the prevalence of psychiatric disorder 12 months later. While we can calculate the maximum potential impact of such interventions, these figures are hypothetical. Clearly, other studies are needed to assess the actual effects of specific interventions. Finally, members of the study population were likely to be at higher risk of psychiatric disorder than individuals living in more affluent areas. In restricting our sample to those attending a single health centre in south London, we have compared the potential impact of high risk and population based approaches to prevention nested within a high risk approach. While this does not affect the validity of our findings, it further highlights the need for caution in generalising these findings beyond similar inner city settings.

Preventive interventions are specific to disorder and depend partly on the shape of the dose-response relationship between the exposure and risk of disorder. A population based approach is likely to have the greatest impact where this is linear, or where the risk of disorder plateaus above a certain level of exposure. By contrast, a high risk approach would be more

appropriate where the risk of disorder increases sharply above a certain level of exposure (ie a threshold effect). Where the dose-response curve is linear, the decision about what proportion of a population should be targeted for intervention will depend on the availability and acceptability of effective intervention,[46] and the associated costs and benefits. The fundamental dilemma, however, is that while the benefits of a population intervention may outweigh its costs, and may greatly exceed the absolute benefits of an intervention restricted to those at the highest risk of disorder, the absolute cost of intervention is likely to be high.

High risk interventions may bring great benefit to the small number of individuals at the highest risk of developing a specific disorder and are an important part of good clinical practice. Although we have compared the (theoretical) impact of high risk and population wide interventions, these are best conceptualised as the extremes of a continuum. As Rose and Lewis pointed out,[47] these strategies should be viewed as complementary components of a needs-led, population based prioritised system of comprehensive risk factor control.

CONCLUSIONS

A high risk approach to prevention alone is incapable of meeting the public health challenge of reducing the prevalence of the most common mental disorders.[45 48] Nevertheless, it might be argued that low income and structural housing problems are intractable forms of socioeconomic adversity, and that a high risk approach represents the most pragmatic use of limited resources.[13 14] What are the alternatives? Egalitarian political interventions, such as changes in taxation or housing policy,[48 49] are likely to be opposed because they are costly and as yet unsupported by empirical evidence. If population based interventions are to gain credibility, interventions must be developed which are both economically feasible and acceptable to populations.[46] Unfortunately, it is not clear what form these should take. There have been no trials of interventions designed to alleviate low income or housing problems, and nor do we know whether it would be more effective to address such fundamental socioeconomic causes or individual characteristics, such as interpersonal relationships or cognitive style, which may mediate their effect on mental health.[50] Further research is needed to identify mediating risk factors, and to explore the feasibility and acceptability of alternative interventions.

Experience from community trials of cardiovascular prevention shows clearly that such interventions must be "owned" locally, since their effectiveness depends on their acceptability to the target population. To this end members of the community, along with its leaders and institutions, must be mobilised during the design phase of any intervention.[46] We suggest that a locally tailored, multi-domain package of risk reduction measures might be developed, in collaboration with users, GPs,

public health physicians, community leaders, local authority social services and housing departments, voluntary organisations, and the local media.[17 47] The ultimate form of the intervention would be determined by both aetiological evidence and local need, and might involve education via the media, plus individual level programmes to provide, for example, advice and advocacy in securing benefits, managing personal finances, and negotiating housing repairs.

1 Cox BD, Blaxter M, Buckle ALJ, et al. *The health and lifestyle survey.* Cambridge: Health Promotion Research Trust, 1987.
2 Robins LN, Regier DA, eds. *Psychiatric disorders in America: the epidemiological catchment area study.* New York: The Free Press, 1991:449.
3 Goldberg D, Huxley P. *Common mental disorders: a biopsychosocial approach.* Routledge: London, 1992.
4 Kessler RC, McGonagle KA, Zhao S, et al. Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States. *Arch Gen Psychiatry* 1994;51:8–19.
5 Meltzer H, Gill B, Petticrew M. *OPCS surveys of psychiatric morbidity in Great Britain. Report No 1. The prevalence of psychiatric morbidity among adults aged 16–64 living in private households in Great Britain.* London: HMSO, 1995.
6 Jenkins R. Minor psychiatric disorder in employed young men and women and its contribution to sickness absence. *Br J Ind Med* 1985;42:147–54.
7 Williams P, Tarnopolsky A, Hand D, Shepherd M. Minor psychiatric morbidity and general practice consultations: the west London survey. *Psychol Med* 1986;**Monograph Suppl 9**:1–37.
8 Broadhead WE, Blazer D, George L, Tse C. Depression, disability days and days lost from work. *JAMA* 1990;264: 2524–28.
9 Croft-Jefferys C, Wilkinson G. Estimated costs of neurotic disorder in UK general practice. *Psychol Med* 1989;19: 549–58.
10 Lewis G, Wilkinson G. Another British disease? A recent increase in the prevalence of psychiatric morbidity in Britain. *J Epidemiol Community Health* 1993;47:358–61.
11 Harris T. Implications of a vulnerability model for the prevention of affective disorder. In: Cooper B, Helgason T, eds. *Epidemiology and the prevention of mental disorders.* London: Routledge, 1989:207–27.
12 Jenkins R. Depression and anxiety: an overview of preventive strategies. In: Jenkins R, Newton J, Young R, eds. *The prevention of anxiety and depression.* London: HMSO, 1992: 11–21.
13 Goldberg D. Early diagnosis and secondary prevention. In: Jenkins R, Newton J, Young R, eds. *The prevention of anxiety and depression.* London: HMSO, 1992:33–38.
14 Paykel ES, Jenkins R, eds. *Prevention in psychiatry.* London: Gaskell, 1994.
15 Rose G. Mental disorder and the strategies of prevention. *Psychol Med* 1993;23:553–55.
16 Scott J, Leff J. Social factors, social interventions and prevention. In: Paykel ES, Jenkins R, eds. *Prevention in psychiatry.* London: Gaskell, 1994:25–31.
17 Mrazek PJ, Haggerty RJ, eds. *Reducing risks for mental disorders.* Washinton, DC: National Academy Press, 1994.
18 Bruce ML, Takeuchi DT, Leaf PJ. Poverty and psychiatric status. *Arch Gen Psychiatry* 1991;48:470–474.
19 Murphy JM, Olivier DC, Monson RR, Sobol AM, Federman EB, Leighton AH. Depression and anxiety in relation to social status. *Arch Gen Psychiatry* 1991;48:223–29.
20 Rodgers B. Socio-economic status, employment and neurosis. *Soc Psychiatry Psychiatr Epidemiol* 1991;26:104–14.
21 Romans SE, Walton VA, McNoe B, Herbison GP, Mullen PE. Otago women's health survey 30 month follow-up. II: remission patterns of non-psychotic psychiatric disorder. *Br J Psychiatry* 1993;163:739–46.
22 Bruce ML, Hoff RA. Social and physical health risk factors for first-onset major depressive disorder in a community sample. *Soc Psychiatry Psychiatr Epidemiol* 1994;29:165–71.
23 Warr P. *Work, unemployment and mental health.* Oxford: Oxford Science Publications, 1987:1–361.
24 Platt S, Martin C, Hunt S. The mental health of women with children living in deprived areas of Great Britain: the role of living conditions, poverty and unemployment. In: Goldberg D, Tantam D, eds. *The public health impact of mental disorder.* Toronto: Hogrefe and Huber, 1990: 124–135.
25 Brown GW, Harris T. *Social origins of depression.* London: Tavistock, 1978.
26 Huxley PJ, Goldberg DP, Maguire GP, Kincey VA. The

prediction of the course of minor psychiatric disorders. *Br J Psychiatry* 1979;**135**:535–43.

27 Birtchnell J, Masters N, Deahl M. Depression and the physical environment: a study of young married women on a London housing estate. *Br J Psychiatry* 1988;**153**:56–64.

28 Goldberg D, Bridges K, Cook D, Evans B, Grayson D. The influence of social factors on common mental disorders: destabilisation and restitution. *Br J Psychiatry* 1990;**156**: 704–13.

29 Blazer DG, Kessler RC, McGonagle KA, Swartz MS. The prevalence and distribution of major depression in a national community sample: the National comorbidity survey. *Am J Psychiatry* 1994;**151**:979–86.

30 Price RH, van Ryn M, Vinokur A. Impact of a preventive job search intervention on the likelihood of depression among the unemployed. *J Health Soc Behavior* 1992;**33**: 158–67.

31 Bloom BL, Hodges WF, Kern MB, McFaddin SC. A preventive intervention program for the newly separated. *Am J Orthopsychiatry* 1985;**55**:9–26.

32 Vachon MLS, Sheldon AR, Lancee WJ, Lyall WAL, Roger J, Freeman SJJ. A controlled study of self-help intervention for widows. *Am J Psychiatry* 1980;**137**:1380–84.

33 Olds DL, Henderson CR, Tatelbaum R, Chamberlin R. Improving the life-course development of socially disadvantaged mothers: a randomised trial of nurse home visitation. *Am J Public Health* 1988;**78**:1436–45.

34 Munoz RF. The prevention of depression: current research and practice. *Applied and Preventive Psychology* 1993;**2**: 21–33.

35 Weich S, Churchill R, Lewis G, Mann A. Do socio-economic risk factors predict the incidence and maintenance of psychiatric disorder in primary care? *Psychol Med* 1997; **27**:73–80.

36 Goldberg DP, Williams P. *The user's guide to the general health questionnaire.* Windsor, Berks. Nfer-Nelson, 1988.

37 Lewis G, Pelosi AJ, Araya R, Dunn G. Measuring psychiatric disorder in the community: a standardised assessment for use by lay interviewers. *Psychol Med* 1992;**22**:465–86.

38 Lewis G. Assessing psychiatric disorder with a human interviewer or a computer. *J Epidemiol Community Health* 1994;**48**:207–10.

39 Hills J. *Inquiry into income and wealth.* York: Joseph Rowntree Foundation, 1995.

40 Stata Corporation. *Stata statistical software: release 4.0.* College Station, Texas: Stata Corporation, 1995.

41 von Korff M, Shapiro S, Burke JD, *et al.* Anxiety and depression in a primary care clinic. *Arch Gen Psychiatry* 1987;**44**:152–56.

42 Barrett JE, Barrett JA, Oxman TE, Gerber PD. The prevalence of psychiatric disorders in a primary care practice. *Arch Gen Psychiatry* 1988;**45**:1100–6.

43 Ormel J, Koeter MWJ, van den Brink W, van de Willige G. Recognition, management and course of anxiety and depression in general practice. *Arch Gen Psychiatry* 1991; **48**:700–6.

44 Kessler LG, Cleary PD, Burke JD. Psychiatric disorders in primary care. *Arch Gen Psychiatry* 1985;**42**:583–87.

45 Rose G. *The strategy of preventive medicine.* Oxford: Oxford University Press, 1992.

46 Koepsall TD, Diehr PH, Cheadle A, Kristal A. Invited commentary: symposium on community intervention trials. *Am J Epidemiol* 1995;**142**:594–99.

47 Lewis B, Rose G. Prevention of coronary heart disease: putting theory into practice. *J R Coll Physicians Lond* 1991; **25**:21–26.

48 Watt GCM. All together now: why social deprivation matters to everyone. *BMJ* 1996;**312**:1026–29.

49 Benzeval M, Judge K, Whitehead M, eds. *Tackling inequalities in health.* London: King's Fund, 1995.

50 Link BG, Phelan J. Social conditions as fundamental causes of disease. *J Health Soc Behav* 1995;(extra issue):80–94.