Papers

Effectiveness of antismoking telephone helpline: follow up survey

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Abstract

Objective: To evaluate the effectiveness of an antismoking campaign conducted by the Health Education Board for Scotland.

Design: Descriptive survey of adult callers to a telephone helpline (Smokeline) for stopping smoking; panel study of a random sample of adult callers; assessment of changes in prevalence of smoking in Scotland before and after introduction of the helpline

Setting: Telephone helpline.

Subjects: Callers to Smokeline over the initial one year period. Detailed information was collected on a 10% sample (n = 8547). A cohort of adult smokers who called Smokeline (total n = 848) was followed up by telephone interview three weeks, six months, and one year after the initial call.

Main outcome measures: Numbers of adult smokers calling helpline; changes in smoking behaviour, especially stopping smoking among cohort members; and changes in prevalence of smoking in the general population.

Results: An estimated 82 782 regular adult smokers made genuine contact with Smokeline over the year, representing about 5.9% of all adult smokers in Scotland. At one year 143 of the cohort of 848 callers (23.6%; 95% confidence interval 20.2% to 27.0%) reported that they had stopped smoking, and 534 (88.0%; 85.4% to 90.6%) reported having made some change. About 19 500 (16 700 to 22 350) adult smokers, equivalent to 1.4% (1.2% to 1.6%) of the mean adult smoking population, stopped smoking with direct help from Smokeline. During the second year of the campaign (1994) smoking prevalence among 25-65 year olds in Scotland was 6% (2.0% to 10.0%) lower than it had been before the start of the campaign.

Conclusion: The Health Education Board for Scotland's antismoking campaign reached a high number of adult smokers, was associated with a highly acceptable quit rate among adults given direct help through Smokeline, and contributed considerably to an accelerated decline in smoking prevalence in Scotland.

Introduction

Smoking remains the largest single cause of preventable death and serious ill health in Scotland.[1] It is estimated that some 10 600 people in Scotland die each year as a result of their smoking,[2] and the extent of damage to health related to smoking has been acknowledged in recent national policy statements,[3 4] in which smoking is identified as a first order priority for health education. Targets for reduction of smoking in Scotland have been set at 30% (from 30% to 21%) among 12-24 year olds and at 20% (from 40% to 32%) among 25-65 year olds between 1986 and 2000.

Box 1
Campaign objectives

- To remind smokers and their families and friends of the negative consequences of smoking
- To challenge complacency about smoking
- To motivate and support smokers and their families and friends towards stopping smoking or encouraging and helping others to do so
- To provide direct advice, help, and support to smokers and families and friends to enable them to stop smoking

Paid advertising has been identified as a leading intervention to promote stopping smoking and as a comparatively cost effective way of reaching most smokers.[1 5] Available empirical evidence suggests that mass media campaigns, using either paid or unpaid advertising to reach the general smoking population, can be effective in encouraging and helping people to change their smoking behaviour in some way—for example, to reduce consumption[6-8] or stop smoking altogether.[5 9-11]

We report on the evaluation of the Smokeline campaign launched in October 1992 by the Health Education Board for Scotland. We examined the effectiveness of the campaign during its first year of operation in three respects: adult (age ≥16 years) uptake of the service provided; change in smoking behaviour among adult users of the service; and impact on overall prevalence of smoking among adults in Scotland.

The overall aim of the campaign is to make an important contribution to the national effort to reduce the prevalence of cigarette smoking. The more specific campaign objectives are set out in box 1.

The campaign has three intertwined strands: Smokeline, a telephone service; *You Can Stop Smoking*, a self help guide to stopping smoking, available exclusively through Smokeline; and mass media advertising,

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Box 2
Campaign elements

Smokeline is a free interactive telephone service, available from noon to midnight each day. It is staffed by trained counsellors and provides advice and information, including tips on how to give up and stay off cigarettes; general support and encouragement when people want it; and, if required, a free copy of *You Can Stop Smoking*, for the caller or an adult friend or relative.

You Can Stop Smoking was designed to be a realistic, eye catching, step by step guide that reinforces the smoker's fundamental belief that responsibility for stopping lies squarely with him or her and offers practical advice to increase the likelihood of successfully stopping.

Mass media advertising has been widely used to promote the campaign. Smokeline was launched on 23 October 1992 through two television advertisements. The first of these was a hard hitting, realistic portrayal of a mother, seriously ill in a hospital bed, imploring her young son never to smoke. The second was shown at the end of the same commercial breaks as the first. It was highly positive in tone, featuring a friendly and sympathetic female "counsellor" encouraging smokers and their families to contact Smokeline. The advertisements were shown regularly for four weeks (first burst) and again from weeks 16-23 (second burst).

A further television advertisement, featuring a man who has been told he is dying of a disease related to smoking who expresses his regrets that he will miss seeing his family growing up, was shown intermittently from weeks 46-52 (third burst). Again, it was paired with the positive advertisement.

The television advertising was combined with three striking outdoor posters: two billboard sized, the other smaller. All were displayed intermittently from week 7. In addition, four press advertisements were published intermittently from week 12.

including television, outdoor posters, and press. Further details of all these elements are given in box 2.

Methods

Calls to Smokeline—Information on sex, smoking, nature of call (first or repeat), reason for calling, and source of obtaining Smokeline telephone number was recorded by the counsellor for each interactive call. (A call is recorded as interactive when a counsellor has offered information to a caller or has begun a process of encouraging the caller to clarify his or her request for information.) At the inception of the line it was not anticipated that children would call, and age data were not at first systematically recorded. It became apparent, however, that considerable numbers of callers were aged under 16 years, and all interactive callers were asked to give their ages from the ninth week onwards. Information on smoking habits and history, intentions to stop smoking, and sociodemographic characteristics was sought from a 10% systematic random sample of all calls from adults over the whole 12 month period (n = 8547).

Panel study—Behavioural outcomes were assessed by means of a panel study of adult callers. From the 10% sample of adult callers to Smokeline (see above) a group of 970, of whom 848 (87.4%) were current smokers, was randomly selected for follow up at three points in time (three weeks, six months, and 12 months after the initial call). All had consented to participate at the initial interview. Follow up interviews were conducted over the telephone by an independent research team.

Smoking in the general population—Data on prevalence of smoking in Scotland are issued by the Scottish Office Department of Health, drawing on information collected by the Office for National Statistics for the general household survey and survey on smoking among school aged children.

Results

Adult smokers using Smokeline

The best estimate of the total number of interactive calls from adults during the first to the eighth week was 36 060 (out of 37 370). During the 44 remaining weeks of the year there were 59 664 interactive calls from adults. The calculated total for the year was thus 95 724 (actual total for all ages was 129 717). To this total, we adjusted for repeat calls (6%) and calls by non-smokers (8%) to obtain the number of adult callers who were smokers—82 782, representing about 5.9% of all adult smokers in Scotland (estimated at 1.4 million).

Characteristics of callers

We calculated that over the period under review 49 660 (60%) of the 82 782 adult callers to Smokeline were women, compared with 52% of the general adult population of Scotland.[12] Callers to Smokeline were younger than the Scottish adult (16-44 years) general population as a whole[13] (72% *v* 54%, respectively), more likely to be unemployed (16% *v* 10%), and less likely to be home owners (41% *v* 52%). Over two thirds of callers had tried to stop in the past, while 58% were desperate to stop at the time of their call, and one third claimed that they would do so immediately. The consumption of cigarettes among callers was particularly high, with 56% smoking 20 or more a day (compared with 42% of smokers in the adult Scottish population[12]).

Change in smoking behaviour

Among the 848 adult smokers in the combined initial panel sample, attrition was evident over the course of the follow up period, although it was not exceptionally high. At one year follow up 607 (71.6%) of the original sample were interviewed again. The group successfully followed up was found to be representative of the original sample in respect of motivational factors (for example, intention to stop smoking), previous successful attempts at stopping and (baseline) cigarette consumption. The groups differed, however, with respect to sociodemographic features: those followed up were significantly more likely to be women, older, and owner occupiers and they were less likely to be unemployed.

Table 1 shows point prevalence measures of smoking status, based on respondents' self reports at each interview. The non-smoking rate among those followed up at one year was 23.6% (95% confidence interval 20.2% to 27.0%). Of those smoking at one year, 42.5% reported having stopped at some point during

Table 1 Smoking status (point prevalence) and behavioural change among Smokeline panel members at three week, six month, and one year follow up interviews.* Figures are numbers (percentages) of respondents

Detail	Three weeks	Six months	One year
Baseline:			
No of subjects	819	701	607
Not smoking	177 (21.6)	132 (18.8)	143 (23.6)
Cigarette smoker	639 (78.0)	565 (80.6)	454 (74.8)
Cigar/pipe smoker	3 (0.4)	4 (0.6)	8 (1.3)
Don't know	0	0	2 (0.3)
Smokers at follow up†:			
No of subjects	639	565	454
Smoking less‡	313 (49.0)	95 (16.8)	89 (19.6)
Lower tar‡	105 (16.4)	126 (22.3)	98 (21.6)
Stopped at some time	209 (32.7)	270 (47.8)	193 (42.5)

*Callers were 848 who were smoking on initial call to Smokeline.
†Three categories are not mutually exclusive.
‡Compared with last interview.

Table 2 Any positive action reported by Smokeline panel members at three week, six month, and one year follow up interviews

Follow up	Total No	No who reported positive action (%; 95% CI)*
Three week	819	579 (70.7; 67.6 to 73.8)
Six month	701	610 (87.0; 84.5 to 89.5)
One year	607	534 (88.0; 85.4 to 90.6)

*Not smoking at follow up, stopped smoking at any time during follow up, reduced cigarette consumption, switched to smoking lower tar cigarettes.

Table 3 Time spent not smoking according to smoking status at 12 months in callers to Smokeline. Figures are numbers (percentages) of respondents

Percentage of year spent as non-smoker	Non-smoker at 12 months	Smoker at 12 months	All
⩾80	48 (34.3)	1 (0.2)	49 (8.3)
60-79	3 (2.1)	7 (1.6)	10 (1.7)
40-59	26 (18.6)	21 (4.7)	47 (8.0)
20-39	27 (19.3)	42 (9.4)	69 (11.8)
1-19	36 (25.7)	296 (66.2)	332 (56.6)
0	0	80 (17.9)	80 (13.6)
Total	140 (100.0)	447 (100.0)	587* (100.0)

*Data missing for 20.

the preceding six months, 19.5% were smoking less, and 21.6% had switched to a lower tar brand compared with that smoked six months earlier. If we consider a broader measure of any positive action taken with respect to smoking (defined to include non-smoking at follow up, stopping smoking at any time during follow up, reduced cigarette consumption, or switching to lower tar cigarettes) 88.0% (85.4% to 90.6%) had made some behavioural change in a positive direction by the time of the one year follow up (table 2).

Table 3 provides an analysis of time spent during the year as a non-smoker by respondents' smoking status at 12 months. Just over a third (34.3%; 26.4% to 42.2%) of those who were non-smokers at 12 months had stoppped smoking for at least 80% of the period. This figure equates to 8.2% (6.0% to 10.4%) of the total sample (data missing for 20) and is taken as the period prevalence measure of stopping smoking.

We estimated the number of adult Scottish smokers who stopped smoking with direct help from Smokeline. Given that 82 782 adult smokers called Smokeline over the one year period, the point prevalence measure of stopping smoking (23.6% (SD 3.4%)) gives a rough total of 19 500 adult smokers (range 16 700-22 350), equivalent to 1.4% (range 1.2%-1.6%) of the adult smoking population. When we applied the period prevalence figure (8.2% (SD 2.2%)) the rough total was 6800 adult smokers (range 5000-8600), equivalent to 0.5% (range 0.4%-0.6%) of the adult smoking population.

Change in smoking prevalence in the general population

Table 4 gives the trends in smoking prevalence among Scottish adults during 1984-94. Among all adults aged ⩾16 there was a decline in the percentage of smokers, from 39% in 1984 to 30% in 1994. The decline of 4% (0.7% to 7.3%) between 1992 and 1994 is particularly noteworthy. Among those aged 25-65 the decline was even more pronounced, from 38% in 1992 to 32% in 1994, a reduction of 6% (2% to 10%), thus reaching the national target for this age group six years ahead of the year 2000 deadline.

Discussion

The Health Education Board for Scotland's campaign seems to have been highly effective in encouraging use of the telephone helpline and facilitating positive behavioural change among adult smokers. At follow up one year after first calling Smokeline, nearly nine in 10 smokers using the service had taken some form of positive action on their smoking; just under a quarter were non-smokers. If we generalise from these findings to the total sample of adult callers to Smokeline we get a one year point prevalence estimate of 19 500 adult smokers (1.4% of the adult smoking population) who stopped smoking with direct help from Smokeline.

Possibility of bias

Confidence in these estimates depends largely on dealing satisfactorily with the problems of bias. The first

Table 4 Prevalence (percentages (95% confidence intervals)) of cigarette smoking, Scotland 1984-94

Age (years)	1984* (n=1801)	1986† (n=1699)	1988† (n=1752)	1990† (n=1590)	1992† (n=1607)	1994‡ (n=1492)
16-19	35 (27 to 43)	40 (29 to 51)	31 (23 to 39)	28 (19 to 37)	27 (18 to 36)	26 (17 to 35)
20-24	42 (35 to 49)	37 (29 to 45)	38 (30 to 46)	36 (28 to 44)	39 (31 to 47)	33 (25 to 41)
⩾ 25	39 (37 to 41)	36 (34 to 38)	37 (35 to 39)	34 (31 to 37)	34 (31 to 37)	30 (27 to 33)
25-64	NA	40 (37 to 43)	NA	38 (35 to 41)	38 (35 to 41)	32 (29 to 35)
⩾ 16	39 (37 to 41)	37 (35 to 39)	37 (35 to 39)	34 (32 to 36)	34 (32 to 36)	30 (28 to 32)

NA=data not available.
*Scottish health statistics 1986.[19]
†Scottish health statistics 1995.[12]
‡General household survey 1994 (Scottish Office Department of Health, unpublished data).

Table 5 Summary of rates of stopping smoking achieved in antismoking interventions (mass media and face to face)

Type of intervention	Type of prevalence	Achieved rate of stopping smoking (%)
Mass media[9]		
Baseline*	Point (one year)	6
	Period (one year)	0.5
Standard†	Point (one year)	18
	Period (one year)	5
Ultimate standard‡	Period (3-12 months)	20-30
Face to face[26-28]		
Brief general practitioner advice	Period (one year)	Median 6 (range 3-13); 12 studies
Brief general practitioner advice reinforced with other interventions and follow up	Period (one year)	Median 22.5 (range 13-38); 10 studies
Nicotine replacement:		
Chewing gum	Point (one year)	Experimental group: median 19 (range 7-60)
		Control group: median 13 (range 2-44); 28 trials
Patches	Point (one year)	Experimental group: median 17.5 (range 13-26)
		Control group: median 9.5 (range 4-12); 6 trials

*Natural or underlying rate of stopping not attributable to any intervention.
†Based on an experimental study in which effects of four different combinations of materials from American Lung Association's *Freedom From Smoking in 20 Days* were assessed.[23]
‡Based on participants using face to face clinics.

consideration is possible attrition and other bias in the panel sample. There are no relevant data on the whole Smokeline sample with which to compare the successfully followed up group. The one year sample was found to be representative of the original panel sample in respect of motivational factors (for example, intention to give up), previous successful attempts at stopping, and cigarette consumption, all of which have been shown to be key predictors of stopping smoking in these cohorts.[14] There was bias in respect of certain sociodemographic characteristics, but of these only socioeconomic status seems to be a predictor of successfully stopping smoking and by no means one of the strongest.[14] On the basis of this evidence we conclude that biased sample attrition is unlikely to be large enough to invalidate our findings, and we defend the use of actual denominators (rather than the original denominator) in calculating rates of stopping smoking and other measures of behavioural change.

Another potential source of bias is the use of uncorroborated self reports to measure current smoking behaviour. Biochemical data on stopping smoking are difficult to obtain in large scale campaigns, without considerably inflating the costs of the intervention. Even with such investment the level of confirmation may be unsatisfactory.[15] In any case, there is evidence that self reports of smoking status are fairly accurate.[16-18]

Direct impact of the campaign

The underlying trend in the prevalence of adult smoking during 1975-92 was a decline of about 0.8% a year (47% to 34%).[12 19] The reduction in adult smoking between 1992 (before campaign) and 1994 (during campaign) was 4% (table 4)—much more than the underlying reduction in smoking prevalence that is not attributable to an intervention (1.6%). The cumulative direct impact of Smokeline (between 1.4% (all change achieved within the first year and no effect in the second year) and 2.8% (rate of change in the first year continues into the second year)), together with the underlying reduction in smoking prevalence (1.6%) yields an overall estimated reduction of 3.0% to 4.4% (over two years). The actual decrease of 4% lies within this range. There is thus no evidence that the effect of Smokeline on overall smoking prevalence among Scottish adults arose otherwise than through its direct impact on service users.

This discussion assumes that all of the excess reduction in prevalence of smoking can be attributed (directly or indirectly) to the campaign. A well known difficulty in assessing the success of a mass media health education campaign, however, is how to separate out the true impact of the campaign from other extraneous factors. Naturalistic designs (particularly before and after studies without control groups, such as ours) are subject to confounding period effects, which hamper interpretation of the findings. Three such possible confounders have been identified: cigarette advertising, pricing of tobacco, and advertising of products to help people stop smoking.

Possible confounding effects

In comparison with the Health Education Board for Scotland's expenditure of about £550 000 on television, print, and poster advertising during the first year of the adult Smokeline campaign,[21] the tobacco companies are estimated to have spent about 10 times this amount (£5.6m) on above the line advertising in Scotland.[22] Such advertising is unlikely to have exerted a positive impact on rates of stopping smoking from October 1992 to October 1993. Indeed, it would probably have had the opposite effect.

The cost of a packet of 20 cigarettes rose by 6.5% in March 1993 as a result of increased taxation. This may have had a positive bearing on the rates of stopping smoking at six months and one year.

The marketing of products to stop smoking—for example, transdermal patches—has grown in recent years in response to the demand for practical support from smokers who want to stop. There is no evidence to suggest, however, that their advertising presence correlates with use of Smokeline. Nicorette was the only product advertised during the launch of Smokeline, while total advertising for products to stop smoking remained at a comparatively constant level before and after Smokeline came into existence.[22]

Comparison with other antismoking interventions

The success of the campaign needs to be assessed against other antismoking interventions, as well as in terms of its overall impact on smoking trends among adults in Scotland. Table 5 presents the baseline, standard, and ultimate standard measures against which, as Flay suggests,[9] the results of interventions should be compared; the table also shows the achieved rates of stopping smoking for face to face interventions[26 27] and nicotine replacement programmes.[28] The outcome of the campaign compares well with these other types of intervention.

Conclusions

The Scottish antismoking campaign provided direct help to an exceptionally high number of adult smokers; use of Smokeline by adults was associated

Key messages

- There was an unprecedented response to the antismoking campaign of the Health Education Board for Scotland, with an estimated 5.9% of adult smokers in Scotland responding to the invitation to call Smokeline, a free telephone helpline, in its first year of operation
- A panel study of callers to Smokeline, with telephone interviewing, obtained an acceptable response rate of 71.6% at one year follow up
- Nearly a quarter (23.6%) of smokers who called the helpline were not smoking at the one year follow up, a success rate that exceeds a proposed standard for comparable health education and promotion interventions
- The Smokeline campaign contributed considerably to an accelerated (6%) reduction in smoking among people aged 25-65 in Scotland during 1992-4
- The study findings provide further evidence of the efficacy of mass media antismoking initiatives with a social support component

with a one year rate of stopping smoking that compares highly favourably with those for other interventions; and the campaign made a significant contribution to an accelerated reduction in the prevalence of smoking in Scotland. The study provides further evidence of the efficacy of mass media campaigns with a social support component in motivating and enabling positive behavioural change among adult smokers.

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One hundred years ago

Scientific temperance teaching in schools

The progress of legislative enactments for the compulsory teaching of the nature and properties of alcoholic intoxicants has made rapid progress in the Legislatures of the United States. Initiated in Vermont in 1882, within four years thereafter the Federal Government passed this provision for all the schools in national territory over which the Government exercised control. Now 41 states have been conquered, only 4 remaining without such legislation, with every prospect of these latter following the suit of the other 41 Legislatures. In Illinois, where this law was approved on June 9th last, the provision is to the effect that "the nature of alcoholic drinks and other narcotics, and their effects on the human system," must be taught, in the course of physiology and hygiene, "in all schools supported wholly or in part by public money." There must be a minimum number of four lessons a week for ten weeks in the year for the higher grades, and of three lessons a week for the lower grades. There must also be textbooks graded to each class of scholars. If the teachers fail to obey this instruction, a penalty of £1 is incurred. Before the amount of the State's contribution is handed over to the local commissioners, an affidavit has to be made by a responsible official that the law has been duly carried out.

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Mothers' birth weight and survival of their offspring: population based study

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Abstract

Objective: To test the hypothesis that a baby's survival is related to the mother's birth weight.
Design: Population based dataset for two generations.
Setting: Population registry in Norway.
Subjects: All birth records for women born in Norway since 1967 were linked to births during 1981-94, thereby forming 105 104 mother-offspring units.
Main outcome measures: Perinatal mortality specific for weight for offspring in groups of maternal birth weight (with 500 g categories in both).
Results: A mother's birth weight was strongly associated with the weight of her baby. Maternal birth weight was associated with perinatal survival of her baby only for mothers with birth weights under 2000 g. These mothers were more likely to lose a baby in the perinatal period (odds ratio 2.3, 95% confidence interval 1.4 to 3.7). Among mothers with a birth weight over 2000 g there was no overall association between mother's weight and infant survival. There was, however, a strong interaction between mother's birth weight, infant birth weight, and infant survival. Mortality among small babies was much higher for those whose mothers had been large at birth. For example, babies weighing 2500-2999 g had a threefold higher mortality if their mother's birth weight had been high (≥4000 g) than if the mother had been small (2500-2999 g).
Conclusion: Mothers who weighed less than 2000 g at birth have a higher risk of losing their own babies. For mothers who weighed ≥2000 g their birth weight provides a benchmark for judging the growth of their offspring. Babies who are small relative to their mother's birth weight are at increased risk of mortality.

Introduction

Low birth weight is strongly associated with poor perinatal survival and may be related to poor health outcomes much later in life.[1 2] A woman's birth weight is known to be directly related to the weight of her babies.[3-5] It is unclear, however, whether a mother's birth weight has any predictive value for the survival of her babies. We evaluated the direct relation between maternal birth weight and the perinatal survival of her babies. We also investigated the influence of maternal birth weight on the baby's perinatal survival specific for birth weight.

Subjects and methods

Data were extracted from the Medical Birth Registry of Norway, a population based registry that compiles data on all births in Norway. The registry started in 1967 and comprises data on over 1.6 million births—more than one third of Norway's total population.

In Norway a unique personal number is given at birth to all citizens. Thus, it is possible to link the birth records of women born early in the registry period to the birth records of their children.

All birth records of women born in Norway in 1967 or later were linked to the birth records for the period 1981-94, thereby forming 105 104 units of mother and child (maternal age 14-28 years). Most of the babies (71%) were first births. These data were previously analysed with data up to 1989, comprising 18 342 mother-offspring units.[5]

Perinatal mortality was defined as all registered stillbirths plus live born babies who died within the first week of life. Stillbirths are reportable to the Medical Birth Registry of Norway from 16 weeks of gestation, and gestational age is calculated from the last menstrual period.

Linked units where the mother or the child was a twin, triplet, etc, were excluded from the analysis, leaving 101 579 mother-offspring units. Birth weight of the baby was missing in 0.10%, and birth weight of the mother was missing in 0.22%.

Logistic regression analyses were carried out by using BMDP.[6] All odds ratios were calculated with the LR program in BMDP, and χ^2 tests (maximum likelihood ratio tests) were used to test associations. Most analyses were done with categorical variables. As part of our data comprised non-independent births to the same mother the subset of mothers with first and second births (25 463 mother-offspring units) were analysed with generalised linear models for dependent data by using Oswald[7] as available through S-PLUS.[8]

Results

Perinatal mortality by maternal birth weight

Table 1 shows perinatal mortality in the babies by categories of maternal birth weight. Mortality increased threefold among the babies of mothers who weighed less than 1500 g and twofold among babies of mothers who weighed 1500-1999 g at birth compared with the babies of mothers with higher birth weights. Among the maternal birth weights of ≥2000 g there were hardly any differences in mortality of the babies. Figure 1 shows the effects of maternal birth weight, with 3500-3999 g as reference category, as odds ratios (95% confidence intervals).

Perinatal mortality specific for weight in groups of maternal birth weight

Table 2 presents data on all registered perinatal deaths in the second generation, stratified by birth weight of both mother and child (500 g categories). Although some groups had few or no deaths, a pattern of differentiated mortality by maternal birth weight emerged. Among mothers with birth weight ≥2000 g, weight specific mortality of small babies increased with increasing maternal birth weight. This was the case for

Table 1 Perinatal mortality by maternal birth weight. Data from Medical Birth Registry of Norway

Maternal birth weight (g)	No of babies	No of perinatal deaths	Perinatal mortality (per 1000 births)
<1500	148	5	33.8
1500-1999	626	13	20.8
2000-2499	2 798	35	12.5
2500-2999	14 181	174	12.3
3000-3499	38 050	438	11.5
3500-3999	33 062	349	10.6
4000-4499	10 571	125	11.8
⩾ 4500	1 919	19	9.9
Total	101 355*	1158	11.4

* Includes babies with unknown birth weight.

every category of babies weighing <3500 g, which is close to the mean birth weight in Norway. For example, babies weighing 2500-2999 g had a threefold higher mortality if their mother's birth weight had been high (⩾4000 g) than if the mother had been small (2500-2999 g).

Smaller than average babies

Using logistic regression we analysed perinatal mortality by maternal weight among babies with birth weight <3500 g (for categories, see table 2). Table 3 summarises the results presented as odds ratios (95% confidence intervals). The corresponding odds ratios for all babies are also given for comparison.

Unlike the pattern for births as a whole, there was a strong association between mothers' birth weight and survival of babies weighing <3500 g, even after we excluded the lowest category of maternal birth weight (χ^2 32.4; 4 df; P<0.0001). The association was strengthened after we adjusted for babies' birth weight (χ^2 48.0; 4 df; P<0.0001). Thus, the effect of maternal birth weight among babies with birth weight <3500 g was considerably stronger than in the total.

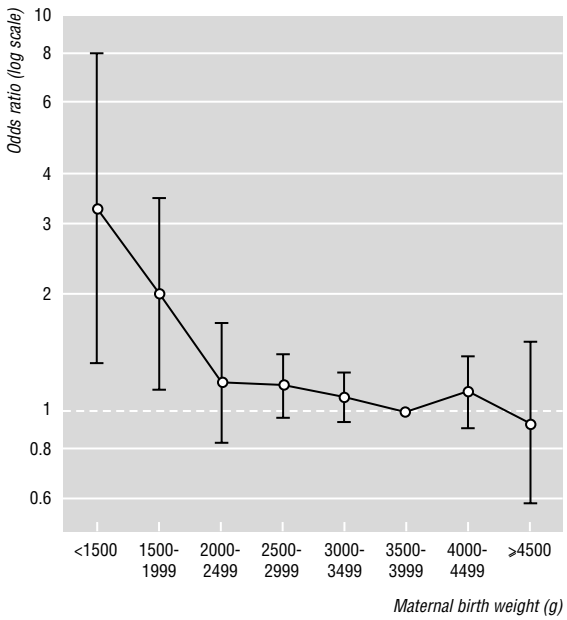

Fig 1 Perinatal mortality by eight categories of maternal birth weight expressed as odds ratios with 95% confidence intervals; reference category 3500-3999 g

Figure 2 shows the perinatal mortality specific for weight by categories of maternal birth weight. (Rates based on fewer than two deaths are not shown.) Although we used categorical variables in the models (table 3), the predicted rates followed a near linear trend for the maternal weight categories for ⩾2000 g. The estimated regression coefficient corresponded to an odds ratio of 1.29 (1.20 to 1.39) for each 500 g increase—that is, an estimated odds ratio of 2.8 for perinatal mortality for equal sized babies for mothers with birth weight ⩾4000 g *v* 2000-2499 g at birth.

Only the first category of maternal birth weight (<2000 g) departed from this trend. Practically all deaths (15 of 17; table 2) for mothers with birth weight <2000 g were among babies who themselves weighed <2000 g. Of the 15 babies who died, 11 were preterm (<37 weeks' gestation).

We could not find any effect of maternal birth weight when the babies weighed above the mean (3500 g) (χ^2 1.1; 4df; P=0.89). We found a significant interaction (P=0.04) when we compared the regression slopes of the four groups with birth weight <3500 g with this last category.

Table 2 Perinatal mortality specific for weight by categories of maternal birth weight. Data from Medical Birth Registry of Norway

Infant birth weight (g) according to categories of maternal birth weight	No (%) of births	No of perinatal deaths	Perinatal mortality (per 1000 births)
Maternal < 2000 g			
< 2000	34 (4.4)	15	441.2
2000-2499	29 (3.8)	1	34.5
2500-2999	129 (16.7)	0	0.0
3000-3499	270 (34.9)	0	0.0
⩾ 3500	311 (40.2)	1	3.2
Maternal 2000-2499 g			
< 2000	99 (3.5)	26	262.6
2000-2499	173 (6.2)	2	11.6
2500-2999	536 (19.2)	2	3.7
3000-3499	997 (35.7)	1	1.0
⩾ 3500	986 (35.3)	1	1.0
Maternal 2500-2999 g			
< 2000	412 (2.9)	120	291.3
2000-2499	569 (4.0)	16	28.1
2500-2999	2523 (17.8)	14	5.6
3000-3499	5519 (39.0)	9	1.6
⩾ 3500	5139 (36.3)	6	1.2
Maternal 3000-3499 g			
< 2000	857 (2.3)	281	327.9
2000-2499	1050 (2.8)	31	29.5
2500-2999	4780 (12.6)	37	7.7
3000-3499	13668 (35.9)	42	3.1
⩾ 3500	17670 (46.5)	29	1.6
Maternal 3500-3999 g			
< 2000	585 (1.8)	236	403.4
2000-2499	620 (1.9)	17	27.4
2500-2999	2848 (8.6)	18	6.3
3000-3499	9951 (30.1)	31	3.1
⩾3500	19029 (57.6)	25	1.3
Maternal ⩾ 4000 g			
< 2000	189 (1.5)	95	502.7
2000-2499	183 (1.5)	9	49.2
2500-2999	687 (5.5)	12	17.5
3000-3499	2768 (22.2)	10	3.6
⩾ 3500	8653 (69.3)	12	1.4
Total	101 264*	1099	10.9

*Total is lower than in table 1 because of babies with missing birth weight.

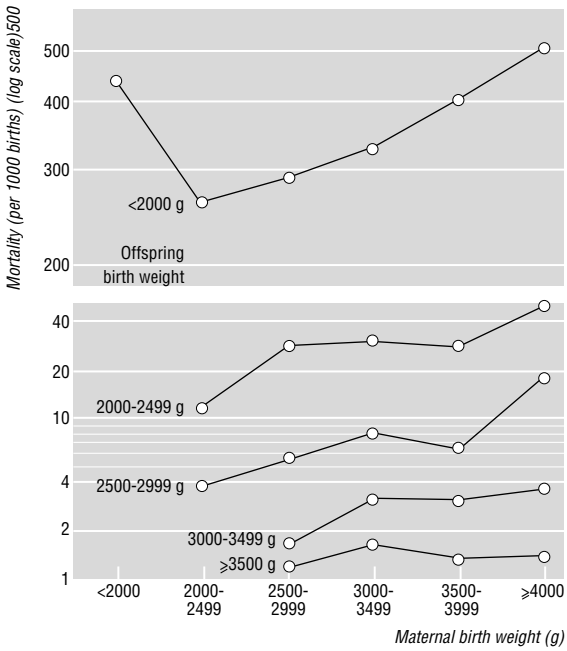

Fig 2 Observed perinatal mortality (per 1000 births) by maternal birth weight in categories of birth weight of offspring

An analysis of the effect on perinatal mortality of maternal birth weight in families with two births by using generalised linear models,[7 8] modelling the non-independence between the births, gave an odds ratio of 1.22 (1.08 to 1.36) for each 500 g increase. In this analysis we excluded mothers with birth weight <2000 g. Also, in an analysis of second births with adjustment for birth weight of the first birth the effect of maternal birth weight persisted (1.33 (1.13 to 1.57) for each 500 g).

Table 3 Odds ratios (95% confidence intervals) for effects of maternal birth weight on perinatal mortality among all babies and among those with birth weight < 3500 g. Data from Medical Birth Registry of Norway

		Among babies < 3500 g	
Maternal birth weight (g)	Among all babies	Crude effects	Adjusted for child's birth weight*
< 2000	1.9 (1.1 to 3.5)	2.1 (1.3 to 3.8)	2.0 (1.0 to 4.0)
2000-2499	1.0 (reference)	1.0	1.0
2500-2999	1.0 (0.7 to 1.5)	1.0 (0.7 to 1.5)	1.3 (0.8 to 2.0)
3000-3499	1.0 (0.7 to 1.4)	1.1 (0.8 to 1.6)	1.6 (1.1 to 2.4)
3500-3999	0.9 (0.6 to 1.2)	1.3 (0.9 to 1.8)	2.0 (1.3 to 3.0)
⩾ 4000	1.0 (0.7 to 1.4)	2.0 (1.3 to 2.9)	3.1 (2.0 to 4.8)
Effects of maternal birth weight			
χ^2 Test	10.9; 5 df; P=0.05	36.1; 5 df; P<0.0001	48.3; 5 df; P<0.0001
χ^2 Test†	3.9; 4 df; P=0.42	32.4; 4 df; P<0.0001	48.0; 4 df; P<0.0001

*Categories as in table 2 (four categories).
†Excludes maternal birth weight < 2000 g.

Table 4 Centiles for birth weight of babies by categories of maternal birth weight. Data from Medical Birth Registry of Norway

Maternal birth weight (g)	Median birth weight of babies (g)	Babies' birth weight (g)				
		2000	2500	3000	3500	4000
< 2000	3330	4.5	8.4	26.0	61.3	89.0
2000-2499	3300	3.8	10.1	30.1	65.6	91.0
2500-2999	3330	3.0	7.2	25.6	64.8	91.7
3000-3499	3450	2.3	5.2	18.3	54.6	87.6
3500-3999	3580	1.8	3.7	12.8	43.6	81.1
⩾ 4000	3740	1.6	3.1	8.8	31.8	71.0

Importance of relative birth weight

A mother's birth weight is a strong predictor of her child's birth weight. In our data the birth weight of the babies increased an average of 28 g for every 100 g of maternal birth weight. This linear relation seems to apply well for maternal birth weights ⩾2000 g. As a consequence, a baby of 3000 g might be on the 30th centile for mothers who had themselves been small at birth but only on the 9th centile for mothers who were large at birth (table 4). Thus, at exactly the same birth weight, one baby might be small for gestational age while another might be appropriate for gestational age and with correspondingly lower mortality, all depending on the birth weight of the mother. This large difference in relative birth weight among babies of the same absolute birth weight presumably explains the large differences in mortality by maternal birth weight.

Discussion

Adverse generational effect

Perinatal events are linked through generations; a baby's birth weight is strongly associated with the mother's birth weight.[3-5] Previous studies have suggested the possibility of higher mortality among offspring of mothers born small, but those studies were based on small numbers without significant results.[9 10] A recent study reported an association between maternal birth weight and infant survival for black but not white subjects.[11] In this large registry of Norwegian births we found that a mother's birth weight directly predicts the survival of her offspring if the mother was very small at birth (<2000 g). This may provide an example of an "adverse generational effect" in which prenatal experience affects a woman's subsequent reproductive success.[12] Most perinatal deaths in babies of mothers who had been small at birth occurred among babies with similarly low birth weight (<2000 g). When the baby is heavier the prognosis is better: only two of the overall 17 losses to mothers who had weighed <2000 g were among babies weighing ⩾2000 g.

Deviation of birth weight from expected weight

In addition to the effect of very low maternal birth weight on survival of the baby, there is an effect in the opposite direction. Mothers who were heavier at birth were more likely to lose their baby at any given weight below the mean. Among these smaller babies there is about a threefold higher relative risk as mother's birth weight increases. This suggests that a baby's survival is related not only to its absolute birth weight but also to its weight relative to its "expected" weight.

The higher risk among small babies of heavier mothers is counterbalanced by the smaller number of low weight babies born to mothers who had been large at birth. Thus, the overall perinatal risk to babies of heavier mothers is not increased.

Mothers who were large at birth tend to have children who are large at birth. If a woman with high birth weight has a small baby, that baby is small not only in absolute terms but also in relation to its expected size. Given two babies with the same small birth weight the one that is also relatively small—that is, in relation to mother's birth weight—has the higher risk of mortality. These data show the importance of relative birth

weight—that is, weight relative to the expected weight for that infant—in addition to absolute birth weight.[13 14] This concept of relative birth weight adds another dimension to the interpretation of birth weight. In our study mothers' birth weight was a strong predictor of an infant birth weight, and therefore infant survival depends in part on how small the baby is in relation to its weight as predicted by the mother's birth weight.

While the concept of relative birth weight may not be familiar to clinicians, it is in fact implicit in the widely used "standards" for birth weight by gestational age. Weight by gestational age is used to identify infants who are small for gestational age for special attention and follow up during pregnancy and after birth. "Small for dates" is defined by centiles, which are units of relative weight. Moreover, the criteria for small for dates are commonly adjusted for factors that are predictive of birth weight, such as parity or sex of the infant.[15] Maternal birth weight is a much stronger predictor of infant weight than either parity or sex and thus could be valuable in setting additional criteria for small for dates. What is "relatively small" for a mother who was small at birth herself is not the same for a mother who was large at birth. Accordingly, the criteria for small for dates differ among women, and this notion of relative birth weight, or heterogeneity in expected birth weight between women, can be extended to standards for fetal growth. In a previous study data on weight by gestational age that used the Norwegian medical birth registry[16] we proposed an adjustment of standards of small for dates according to the weights of previous children to the same mother. This suggestion stemmed from the high correlation in birth weight between siblings born to the same mother. While this adjustment is useful, it is not applicable to first born infants. Mother's birth weight may be useful not only to identify "small" infants born to mothers who are expected to deliver larger babies but also to reduce the number of "false positive" cases of small for dates among mothers expected to deliver smaller babies.

Biological mechanisms

The biological mechanisms by which a mother's birth weight affects her infant's weight are not known. Broadly, three sets of factors are possible.

Firstly, environmental influences (for instance, diet and smoking) may persist across generations.

Secondly, genetic factors may contribute to normal variability in growth. Reports of a parallel association between father's and baby's birth weight[3 17] suggest that fetal genes have a role in the correlation between birth weights of parents and children. In a study of birth weights of babies of twins, fetal genes accounted for more than half of the total variance.[18]

Thirdly, a set of factors may operate through maternal body size or metabolism.[17 19] Sanderson *et al* found that adjustment for maternal height and weight in pregnancy reduced, but did not eliminate, the risk for infant death associated with low maternal birth weight.[11] These data were not available in the Norwegian material. Consideration of these mechanisms is important for understanding the normal variability in fetal growth and judging the effects of relative birth weight on morbidity and mortality.

These same maternal and paternal factors that affect babies' birth weight presumably contribute to the correlation of birth weights among siblings.[20 21] The correlation between birth weights of siblings ($r=0.50$)[22] is considerably greater than seen here between maternal and infant weights ($r=0.21$). Not surprisingly, a baby's birth weight relative to its sibling's weight is also important to its risk of mortality.[23] At any given birth weight, weight relative to sibling weight makes a strong additional contribution to survival. This effect is especially apparent among babies weighing <3000 g, similar to the present analysis.[23] Wang *et al* recently showed that maternal birth weight and the birth weight of a previous sibling are at least partial independent predictors of low birth weight (<2500 g).[24] This result is confirmed in our larger study. Especially, we found the odds ratios for low birth weight in second births when only the mother was of low birth weight, only the first sibling was of low birth weight, and both the mother and the first sibling were of low birth weight were 2.1, 6.1, and 12.4, respectively. The perinatal mortality among the low birth weight infants in families without previous low birth weight, however, was higher than in the three family types with low birth weight (odds ratio 1.3).

Barker has proposed that intrauterine growth is related to cardiovascular and other diseases in the adult.[1 2] The excess perinatal mortality among babies of mothers born small may be an example of early life causes, where prenatal experience may affect later health.[25] The aspects of maternal birth weight that lead to higher perinatal mortality of her babies may also contribute to a higher risk of adult diseases, but other explanations (as discussed above) are possible.

Population based dataset

Our data come from a large population based birth registry. Mothers and babies are linked by unique personal numbers, which do not rely on the mothers' recall. In other studies non-responders are common among categories of mothers with high risk for an adverse outcome of pregnancy.[9] As our data require the mother to have been born in Norway the increased immigration to Norway during the past decade does not influence the data. The distribution of maternal age, however, may represent a bias. Among the linked births we identified mother-offspring units only for the mothers born in 1967 or later, thus excluding babies of mothers older than 28 years. Adjustment for five year categories of maternal age, within the available range, did not change the results. As 28% of mothers were included more than once in this dataset (because they had more than one birth) not all mother-offspring units were independent. Modelling this non-independence gave no substantive effect on the rates or confidence intervals. If anything, the effects of maternal birth weight among the smaller babies were stronger among second births than among first births.

Exclusion of early stillbirths between 16 and 21 weeks (or birth weight <500 g) slightly reduced the direct effect of a perinatal loss for the smaller mothers (birth weight <2000 g) from 2.31 to 2.26 (odds ratios with 3500-3999 g as reference). If anything, the relative effect of birth weight—that is, as reported in table 3—was slightly increased.

Key messages

- A mother's birth weight influences perinatal survival of her own babies but only for mothers with birth weight <2000 g
- There was a strong interaction between mothers' birth weight, infant birth weight, and infant survival
- Babies that are small relative to their mother's birth weight are at increased risk of perinatal death

Variation in mortality

Despite our large sample, some groups of maternal and child birth weight provided only sparse data for perinatal mortality. Variation in mortality may be more complicated than the modelled results suggest. Still, mothers who weighed <2000 g at birth are at substantially increased risk of losing their own baby. Furthermore, high maternal birth weight is indicative of a large baby, and small babies born to those mothers are at higher risk than their weight alone would suggest. To the extent that a woman's birth weight can be obtained easily and accurately, it may offer clinically useful information on the risk of mortality of her babies. The biological pathways by which maternal birth weight affects the weight of her babies, however, need to be better understood.

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Any questions

Is there a molecular explanation of heart rhythm?

Most physiological phenomena are now being explained at molecular or atomic level. What explains the regular action of the sinoauricular node, which initiates the contraction of the heart?

The generation of the rhythm of the heart is a good example of a physiological function that cannot be explained at the molecular level. There is no molecular, still less an atomic, oscillator that generates the heartbeat. Instead many molecular (protein) mechanisms that transport charged atoms (ions) through the cell membrane combine to produce the overall electrical oscillation. These include proteins that specialise in the transport of sodium, potassium, and calcium ions. When the properties of these channels and carriers are represented mathematically in a model of the sinoauricular node cell, and the resulting set of equations is integrated using a computer, regular rhythmic electrical activity emerges as a property of the complete cell system. It is then possible to investigate how modification of each transporter will modulate the rhythm.[1] This is the way in which we have been able to determine how the heart is accelerated by adrenaline and slowed by acetylcholine. In both cases these substances act simultaneously on several different transporters.[2]

Thus it is necessary to study the mechanism of cardiac rhythm at a cellular, not at a molecular level. Moreover, in the whole heart, the sinoauricular node cells are also strongly influenced by the surrounding atrial cells. Hundreds of thousands of cells interact electrically to produce the wave of excitation that invades the rest of the heart and so initiates contraction.[3]

There are many other examples of important physiological functions that cannot be understood at a molecular level. Some medical scientists are now suggesting that we should recognise the existence of "physiome"—that is, a logic of physiological systems—whose study is just as important as that of the genome.

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Cost effectiveness of day and inpatient psychiatric treatment: results of a randomised controlled trial

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Abstract

Objective: To compare direct and indirect costs of day and inpatient treatment of acute psychiatric illness.
Design: Randomised controlled trial with outcome and costs assessed over 12 months after the date of admission.
Setting: Teaching hospital in an inner city area.
Subjects: 179 patients with acute psychiatric illness referred for admission who were suitable for random allocation to day hospital or inpatient treatment. 77 (43%) patients had schizophrenia.
Interventions: Routine inpatient or day hospital treatment.
Main outcome measures: Direct and indirect costs over 12 months; clinical symptoms, social functioning, and burden on relatives over the follow up period.
Results: Clinical and social outcomes were similar at 12 months, except that inpatients improved significantly faster than day patients and burden on relatives was significantly less in the day hospital group at one year. Median direct costs to the hospital were £1923 (95% confidence interval £750 to £3174) per patient less for day hospital treatment than inpatient treatment. Indirect costs were greater for day patients; when these were included, overall day hospital treatment was £2165 cheaper than inpatient treatment (95% confidence interval of median difference £737 to £3593). Including costs to informants when appropriate meant that day hospital treatment was £1994 per patient cheaper (95% confidence interval £600 to £3543).
Conclusions: Day patient treatment is cheaper for the 30-40% of potential admissions that can be treated in this way. Carers of day hospital patients may bear additional costs. Carers of all patients with acute psychiatric illness are often themselves severely distressed at the time of admission, but day hospital treatment leads to less burden on carers in the long term.

Introduction

The Manchester Royal Infirmary's psychiatric day hospital has treated acutely ill patients as an alternative to inpatient treatment for over a decade.[1-3] Roughly 40% of potential admissions can be treated in this way. Other workers have reported similar findings.[4-6] Day patient and inpatient treatment lead to similar social and clinical outcomes.

This paper compares the costs of day patient and inpatient treatment. Other studies have either included only neurotic and personality disorders,[7] or shown no difference because inpatient beds were held open for day patients,[8 9] or shown day hospitals to be an unwarranted, expensive alternative to outpatient treatment for milder illness.[10 11] In our previous study some day patients were so ill that they were transferred to the inpatient unit.[2] Such "failures" of day hospital treatment were included in this study in the intention to treat analysis of costs. These costs were assessed over one year because we find that inpatients with a brief admission have a higher rate of readmission over the subsequent year than day patients.

Day hospital treatment for acutely ill patients may place an excessive burden on the carers. Hence costs to these family members were included both as monetary variables (for example, loss of income and travel costs) and non-monetary variables (for example, stress symptoms in the carer).

Patients and methods

The study was conducted over three years. Randomisation was as in our previous study, by randomly assorted cards in sealed envelopes opened by an independent administrator.[2] After randomisation clinicians managed the patients as usual, determining discharge dates and readmissions independent of the researchers.

All patients aged 18-65 years presenting to the service for admission were considered for the study. Exclusions were compulsory admissions or patients too ill for day treatment, patients discharged in under five days, admissions solely for detoxification of drugs and alcohol, and patients with a diagnosis of organic brain disease, personality disorder, or mania. Psychiatric diagnosis was assessed at admission (by PM and BW) with the present state examination,[12] and the severity of psychiatric symptoms was measured with the comprehensive psychopathological rating scale[13] at admission and after two weeks and one, two, three, six, and 12 months after admission. Demographic data, previous psychiatric admissions, and mode of referral were recorded at the first interview.

The social behaviour assessment schedule[14] was administered by the social research worker (SL) to an informant at admission and at one, two, three, six, and 12 months. This instrument assesses the patient's social performance, abnormal behaviours, and burden on relatives. Disturbed behaviour after admission was assessed with the modified social behaviour schedule,[15] completed by the patient's key nurse. Distress in carers was also assessed by the informant completing the general health questionnaire.[16]

Use of resources

Direct costs to Central Manchester Health Care Trust—The duration of the first and any subsequent inpatient or day hospital admissions, number and length of interviews with medical staff and community psychiatric nurses, and investigations at the hospital were costed at local rates. Costs of the mental health service in central Manchester, including "hotel" and staffing costs, were identified down to unit of service. These were then linked to appropriate clinical activity to give the unit costs for the study. The cost of drugs was based on *British National Formulary* figures,

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adjusted to take account of Central Manchester Health Care Trust overheads. Day hospital admissions were costed (staffing and overheads) by using the number of days that patients actually attended the day hospital.[2]

Direct costs to other agencies—Information on direct costs to other agencies was collected from the records of general practitioners and social workers. Costs of visits were based on national unit costs of community care,[17] as detailed local cost information was not available for these services.

Indirect costs to patients and carers—Indirect costs to patients and carers were estimated from interviews with the main carer. The carer recalled travelling costs (number of journeys, mode of transport, and return mileage) related to the patient's illness, and an estimate was also made of any increased household expenditure and reduction in the patient's or carer's income (that is, as a result of time lost from work due to the illness). The time that the patient and carer spent travelling and in outpatient and other departments during appointments with medical, nursing, and social work staff was estimated but could not be costed. The average time per day that the carer spent in direct care of the patient (while ill) was also estimated and expressed as hours per day.

All the above monetary costs were adjusted to 1994-5 prices by using the relevant price index.

During the second half of the study additional support was provided to help the treatment of acutely ill day patients. A community psychiatric nurse worked specifically to aid their attendance at the day hospital and a nurse was available on call during evenings and weekends. This increased contacts with the community psychiatric nurses but did not significantly affect overall costs.

Ethics—The study was approved by central Manchester's ethical committee.

Data analysis and statistics

An intention to treat analysis compared inpatient and day hospital groups. Clinical and social outcome and costs were compared over 12 months after the first admission. The comprehensive psychopathological rating scale measures psychiatric symptoms, and the social behaviour assessment schedule measures social role performance, abnormal behaviours observed by the informant, and burden on carer. The general health questionnaire completed by the informant measures distress experienced by the carer. These separate outcome measures reflect separate dimensions.[18]

The statistical package for the social sciences was used. Baseline measures were compared by χ^2 and t tests as appropriate. Scores at all follow up assessments were compared by analysis of covariance to control for any baseline differences. In order to check differences between the groups the area above and below the curve (see fig 1) was calculated for each patient and compared by t test.

Costs had a highly positively skewed distribution, so the Mann-Whitney U test was used for analysis. Table 3 gives the resulting P values and also shows the median figures (and 95% confidence intervals) for each cost category for inpatients and day patients and the difference. The data were also analysed by log transformation, the overall pattern of results being identical with those shown (data available on request).

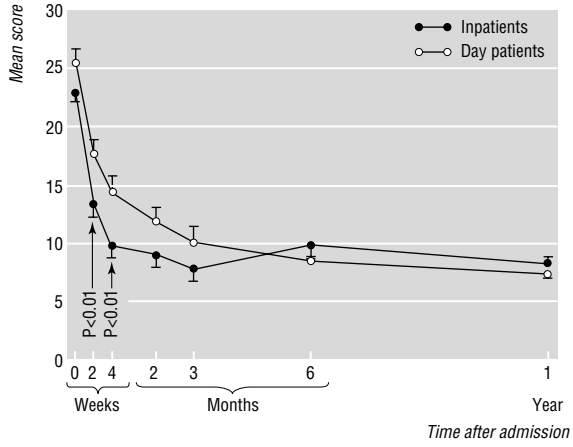

Fig 1 Mean comprehensive psychopathological rating scale scores (psychiatric symptoms) for day patients and inpatients at admission (time 0) and two and four weeks and two, three, six, and 12 months after admission. Bars are SEM

Results

Ninety three inpatients and 94 day patients were randomised. Eight were excluded because of diagnosis or early discharge, leaving 89 inpatients and 90 day patients. Five randomised inpatients were transferred to the day hospital because of lack of beds, and 11 randomised day patients were transferred to the inpatient unit because they were too ill for the day hospital. Only 103 patients (52 inpatients, 51 day patients) had a resident carer who was available for repeated interviews. Patients with and without a carer had similar comprehensive psychopathological rating scale and present state examination scores (severity of psychiatric symptoms).

At admission inpatients and day patients showed no significant differences in sex (49 (55%) and 53 (59%) male, respectively); mean age (37 (SD 12) and 39 (14) years); ethnic origin (69 (78%) and 78 (87%) white); employment status (38 (43%) and 39 (43%) unemployed); marital status (26 (29%) and 36 (40%) married or cohabiting); diagnostic categories (schizophrenia 41 (46%) and 36 (40%), depression 28 (32%) and 32 (36%), neurosis 20 (23%) and 22 (25%); $\chi^2 = 0.68$, $df = 2$, $P = 0.71$); and severity of illness (mean comprehensive psychopathological rating scale scores 23.02 (SD 8.58) and 25.3 (11.72); mean disturbed behaviour social behaviour assessment schedule scores 11.64 (SD 7.13) and 12.66 (7.30)).

Clinical and social outcome

Scores for psychiatric symptoms, social behaviour, and role performance were all significantly ($P < 0.001$) reduced six and 12 months after admission and there was no significant difference between day patients and inpatients at these times (table 1; fig 1). At two and four weeks inpatients showed fewer psychiatric symptoms and fewer abnormal behaviours, indicating more rapid recovery than day patients. Social behaviour assessment schedule burden scores showed no difference in the early months of treatment, but day hospital patients were less of a burden to carers at one year (table 2). General health questionnaire scores of carers were not

Table 1 Comprehensive psychopathological rating scale symptom scores and social behaviour assessment schedule behaviour and social role performance scores at admission and follow up assessments for inpatients and day patients

	Inpatients			Day patients			Estimated difference	95% CI	P value
	Mean	SEM	No	Mean	SEM	No			
Comprehensive psychopathological rating scale scores									
Admission	23.0	0.920	87	25.4	1.242	89	–2.3	–5.4 to 0.7	0.13
2 Weeks	14.2	1.085	72	17.1	1.211	82	–2.9	–5.7 to –0.1	0.048
4 Weeks	10.7	1.113	70	13.9	1.306	76	–3.2	–6.4 to –0.01	0.050
2 Months	9.2	1.118	63	11.6	1.248	69	–2.4	–5.7 to 0.9	0.16
3 Months	8.2	1.042	60	9.8	1.329	63	–1.6	–5.4 to 2.2	0.35
6 Months	10.3	1.211	65	8.4	1.369	69	1.9	–1.7 to 5.5	0.30
1Year	8.6	1.123	65	7.2	1.334	67	1.4	–2.0 to 4.8	0.44
Social behaviour assessment schedule scores									
Behaviour:									
Well	2.67	0.646	52	3.44	0.710	50	–0.77	–2.65 to 1.14	0.43
Admission	16.62	0.922	52	18.06	0.963	51	–1.44	–4.09 to 1.20	0.28
4 Weeks	7.48	1.145	49	10.66	1.190	45	–3.18	–6.19 to –0.17	0.039
2 Months	6.56	1.013	47	9.14	1.150	49	–2.62	–5.47 to 0.31	0.081
3 Months	6.61	0.939	47	7.34	1.007	48	–0.73	–3.33 to 1.87	0.58
6 Months	7.82	1.046	44	6.06	0.952	44	1.76	–0.88 to 4.40	0.19
1 Year	7.78	1.031	44	6.72	0.958	46	1.06	–1.61 to 3.73	0.44
Role:									
Well	6.46	0.712	52	5.92	0.722	51	0.54	–1.47 to 2.55	0.60
Admission	16.48	0.985	52	17.63	0.996	51	–1.15	–3.93 to 1.63	0.42
4 Weeks	17.98	1.152	48	14.30	1.055	45	3.70	1.29 to 6.07	0.003
2 Months	13.49	1.193	47	11.86	1.035	49	1.60	–1.03 to 4.29	0.23
3 Months	10.85	1.115	47	10.12	0.923	48	0.70	–1.91 to 3.37	0.59
6 Months	9.56	1.003	44	8.64	0.943	44	0.90	–1.60 to 3.44	0.47
1 Year	10.24	1.214	43	9.08	1.153	46	1.20	–2.06 to 4.38	0.48

Results at two weeks and later are adjusted for corresponding baseline values by analysis of covariance.

Table 2 Social behaviour assessment schedule score for burden on carers and carers' general health questionnaire scores

	Inpatients			Day patients			Estimated difference	95% CI	P value
	Mean	SEM	No	Mean	SEM	No			
Social behaviour assessment schedule scores—burden on carers									
Well	2.83	0.441	52	2.32	0.376	50	0.51	–0.65 to 1.66	0.39
Admission	9.56	0.852	52	9.57	1.000	51	–0.01	–2.61 to 2.59	0.99
2 Weeks	9.30	0.921	44	9.05	1.123	41	0.25	–0.89 to 1.39	0.66
4 Weeks	7.77	0.746	49	7.75	1.036	46	0.02	–1.51 to 1.55	0.98
2 Months	5.53	0.650	45	6.16	0.789	49	–0.57	–2.30 to 1.04	0.45
3 Months	5.33	0.710	46	5.06	0.689	48	0.27	–1.53 to 2.07	0.77
6 Months	5.72	0.746	43	4.56	0.694	44	1.16	–0.50 to 2.82	0.17
1 Year	5.79	0.694	43	3.93	0.502	43	1.86	0.25 to 3.48	0.024
Carer's scores on general health questionnaire									
Admission	7.7	0.903	46	8.7	1.187	41	–0.99	–3.92 to 1.94	0.51
2 Weeks	7.8	10.23	38	7.6	1.287	32	0.15	–1.46 to 1.86	0.86
4 Weeks	6.6	1.111	42	5.7	1.018	36	0.95	–1.54 to 3.34	0.44
2 Months	4.6	0.973	39	4.8	0.887	39	–0.16	–2.63 to 2.23	0.90
3 Months	4.1	0.845	39	4.0	0.847	38	0.02	–2.09 to 2.29	0.98
6 Months	4.1	0.885	36	3.3	0.976	33	0.77	–1.67 to 3.27	0.54
1 Year	3.7	0.810	31	4.3	1.558	24	–0.69	–3.70 to 2.50	0.67

Results at two weeks and later are adjusted for corresponding baseline values by analysis of covariance.

significantly different at any time (table 2). Calculating the area above and below the curve for each patient as a summary statistic confirmed that there was no significant difference on any clinical or social outcome measure (data available on request).

Resource use and costs

Direct costs—Randomised inpatients accumulated a mean of 62 inpatient days and seven day hospital days over the 12 months. For day patients the figures were 32 day hospital days and 21 inpatient days. Table 3 gives the costs. The duration of interviews with medical staff (11 hours *v* 10 hours; $P = 0.44$) was similar but day patients spent more time with community psychiatric nurses ($P < 0.05$). Costs of hospital investigations and drugs were similar. The median overall difference in costs to the Central Manchester Health Care Trust was £1923 (95% confidence interval £750 to £3174) less for day patients (table 3). Direct costs to other agencies showed no significant differences (table 3). Loss of patients' income through illness absences from work or unemployment was similar. Not surprisingly, patients' travel costs were greater for the day hospital group ($P < 0.05$). Total costs given monetary value showed day hospital treatment to be significantly cheaper (median difference £2165 (£737 to £3593); $P = 0.001$).

Costs to carers—There was a significantly greater loss of income among the carers of day hospital patients,

Table 3 Average costs for patients and carers in inpatient and day hospital groups for 12 months after admission to study

	Inpatient group		Day hospital group		Inpatient *v* day hospital difference and significance	
	Activity median	£ Median (95% CI)	Activity median	£ Median (95% CI)	£ Median (95% CI)	P value (Mann-Whitney U test)
Costs given monetary value						
Direct treatment costs—Central Manchester Health Care Trust:						
Inpatient (days)	48	5040 (3255 to 6615)	0	0 (0 to 0)	3375 (2100 to 5145)	< 0.001
Day hospital (attendances)	0	0 (0 to 0)	24	1392 (754 to 2030)	–1044 (–1566 to –580)	< 0.001
Medical staff (hours)	11	183 (138 to 214)	10	171 (141 to 187)	12 (–21 to 43)	0.44
Community psychiatric nurses (hours)	0	0 (0 to 0)	0	0 (0 to 16)	0 (0 to 0)	0.035
Tests (No)	4	42 (26 to 69)	4	42 (33 to 72)	0 (–16 to 11)	0.98
Drugs (No)	10	66 (45 to 104)	7	55 (23 to 80)	9 (–8 to 34)	0.28
Total		5296 (3845 to 6763)		2370 (1782 to 3259)	1923 (750 to 3174)	< 0.001
Direct treatment costs—other agencies:						
General practitioners (contacts)	13	99 (76 to 130)	14	107 (99 to 122)	–8 (–38 to 15)	0.51
Drugs prescribed by general practitioners (issues)	14	60 (37 to 77)	9	40 (18 to 61)	9 (–6 to 31)	0.20
Tests requested by general practitioners (No)	0	0 (0 to 0)	0	0 (0 to 0)	0 (0 to 0)	0.26
Social workers (contacts)	0	0 (0 to 0)	0	0 (0 to 0)	0 (0 to 0)	0.50
Home helps (contacts)	0	0 (0 to 0)	0	0 (0 to 0)	0 (0 to 0)	0.89
Day care centre (contacts)	0	0 (0 to 0)	0	0 (0 to 0)	0 (0 to 0)	0.91
Total		180 (123 to 231)		163 (122 to 236)	11 (–42 to 62)	0.63
Indirect costs (patients):						
Loss of patients' income		0 (0 to 0)		0 (0 to 0)	0 (–81 to 40)	0.17
Patients' travel costs		13 (7 to 24)		28 (16 to 50)	–12 (–28 to –1)	0.043
Total		3 (0 to 9)		0 (0 to 18)	0 (–16 to 0)	0.66
Total patient costs given monetary value		5931 (4595 to 7586)		2880 (2189 to 3548)	2165 (737 to 3593)	0.0011
Indirect costs (carers):						
Loss of carers' income		0 (0 to 0)		0 (0 to 0)	0 (0 to 0)	0.017
Carers' travel costs		9 (5 to 21)		0 (0 to 2)	6 (1 to 12)	< 0.001
Increased household expenditure		3 (0 to 121)		6 (0 to 76)	0 (–17 to 0)	0.95
Total carer costs given monetary value		9 (1 to 98)		5 (0 to 76)	0 (–66 to 6)	0.76
Costs not given monetary value						
Indirect costs (patients):						
Patients' travel time (hours)		12 (7 to 17)		21 (15 to 33)	–9 (–16 to –2)	0.011
Patients' consultation time (hours)		8 (5 to 12)		5 (4 to 8)	3 (0 to 4)	0.027
Indirect costs (carers):						
Carers' travel time (hours)		11 (7 to 15)		4 (2 to 8)	5 (1 to 10)	0.018
Carers' care time (hours per day)		5 (2 to 11)		8 (1 to 14)	0 (–7 to 2)	0.47
Carers' consultation time (hours)		26 (9 to 31)		4 (3 to 14)	8 (0 to 23)	0.029

No patient in study had contact with social work assistants, health visitors, meals on wheels, family aid, or private health care.

primarily as a result of two carers becoming unemployed. This loss of employment could not be attributed directly to the patient's illness or its treatment, but the costs were included in the cost analysis. Travel costs were significantly greater for the carers of inpatients (table 3). When all direct and indirect costs to patients and carers were considered the median costs for day hospital treatment were £1994 less than for inpatient treatment (95% confidence interval £600 to £3543).

Costs not given a monetary value

Time spent travelling was significantly greater for day patients, but carers' travelling time and outpatient and consultation times with the general practitioner were all significantly greater for inpatients (table 3). A considerable amount of time was spent by carers on the care of their mentally ill relatives but there was no significant difference between inpatient and day hospital groups.

This analysis does not include accommodation costs. Sixty seven (35%) patients were living with a spouse or cohabitee, 51 (27%) were living with a family member, 42 (22%) were in lodgings or a hostel, and 29 (15%) lived alone. There was no significant difference between the inpatient and day hospital groups in this respect.

Discussion

So far as we know this is the first cost effectiveness study of day hospital treatment as an alternative to inpatient psychiatric treatment. There were two principal methodological problems. The first was the attrition rate of the sample over 12 months; this was similar for day patients and inpatients and comparable with other studies in an inner city area.[2 19 20] Secondly, only 103 (55%) patients had a resident carer to interview; this reflects the small social network of such patients and compares favourably with the 28-32% in other studies.[19 21 22]

This study shows that day hospital treatment is cheaper than inpatient treatment. This remained true despite increased costs associated with a few day patients being transferred to inpatient care, additional travelling costs and community psychiatric nurse time for day patients, and a few carers of day patients who incurred considerable loss of income (though in this series it was not clear that this was related to the psychiatric treatment). Carers of day patients recorded lower burden scores at 12 months, possibly because

Key messages

- When inpatient treatment is avoidable day hospital treatment is cheaper for acutely ill psychiatric patients
- Carers of acutely ill psychiatric patients experience severe distress and warrant help in their own right
- Inpatient treatment relieves symptoms more rapidly than day hospital treatment but may lead to increased burden on carers one year later

day hospital care leads to better coping with the illness by patients and relatives.[23] Early in treatment, however, most carers had high general health questionnaire scores—64% (66/103) were above the threshold for probable "cases" of psychiatric disorder in their own right. The more rapid improvement of inpatients is a new finding. This may reflect their removal from a stressful environment, the increased support from staff, or the increased use of drug treatment.[24]

This study included patients with acute symptoms presenting to an inner city psychiatric service. Most presented as emergencies and nearly half (77; 43%) had schizophrenia. These patients represented only 30-40% of potential admissions[2] and were therefore not as ill as patients included in other intensive community treatment programmes in Britain.[19 25] They were more comparable to patients in early intervention studies[22 26 27] and cannot be generalised to all acutely ill patients.

These patients were more ill than those in our previous study[2] (data not shown), reflecting increased confidence of our day hospital staff to treat acute illness, the proximity of the inpatient unit for rapid transfer when necessary, and considerable input from medical and community psychiatric nursing staff. These factors probably helped to prevent "burn out" of staff, which is a feature of some alternatives to hospitalisation for psychiatric patients.[28-31]

This study confirms that day hospital treatment is feasible and cheaper to the health service than inpatient treatment for some acute psychiatric illnesses. Relatives of patients with acute mental illness warrant further support to reduce their distress and enhance their caring role.

We thank Dr Max Marshall for helpful comments on an earlier draft of this paper and Mrs Joan Bond for invaluable secretarial work. The results of log transformation and calculation of the area above and below the curve for each patient as a summary statistic for differences in clinical or social outcome measures may be obtained by writing direct to FC.

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Conflict of interest: None.

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Correction

Drug delivery from inhaler devices

An author error occured in this paper by Carolyn DiGuiseppi and colleagues (8 March, pp 710-3). The fourth and fifth sentences in the third paragraph of the results should have read: "Girls showed substantially larger declines in walking (23% [not 30%]) and cycling (39% [not 64%]) and smaller increases in car travel (35%) than did boys (15%, 23%, [not 18%, 30%] and 45% respectively). Children aged 10-14 showed the largest decline in walking (26% [not 35%]) compared with children aged 0-4 (9% [not 10%] and 5-9 (17% [not 20%]." These changes do not alter the meaning of the two sentences or affect any of the subsequent discussion.

Concordance of phenprocoumon dosage in married couples

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Coumarin derivatives are used in large numbers of patients. However, patients vary considerably in the dosage required to achieve a given level of anticoagulation.[1] Whether this variation is due to differences in diet (for example, intake of vitamin K) or other factors (for example, liver metabolism) is unclear. To determine the influence of life circumstances such as diet on anticoagulant dosage we studied couples in whom both spouses used phenprocoumon. In addition, we compared the dosages used in these patients with those in other subjects, matched for age and sex.

Patients, methods, and results

Our thrombosis service treats around 10 000 patients a year with coumarin derivatives, prothrombin times being monitored by a computerised system. Three target levels of anticoagulation are aimed at depending on the diagnosis. We identified all couples (n = 33; 66 patients) in whom both spouses used phenprocoumon for anticoagulation. Median ages were 72 years (range 61-87) for men and 70 years (62-84) for women. Twenty five couples shared the same target levels of anticoagulation. We then sought 66 controls matched for age, sex, and anticoagulation target level. Thirty three "matched couples" were formed with mean ages of 72 years (range 62-84) for "matched husbands" and 69 years (56-87) for "matched wives."

Individual mean dosages and international normalised ratios[2] between October 1993 and May 1994 were analysed. Only one batch of samples had been used for prothrombin time determinations. Data are given as medians and ranges. Paired Wilcoxon tests were used. Differences in phenprocoumon dosage between spouses and between "matched spouses" were analysed with calculation of 95% confidence intervals for the difference between differences in dosage.[3]

Median anticoagulation levels in the 33 couples did not differ significantly between spouses (international normalised ratio 3.3 (range 2.3-4.0) in men *v* 3.2 (1.8-4.7) in women; P = 0.77) or between the matched spouses (3.4 (2.5-4.1) *v* 3.4 (2.2-4.1); P = 0.74). Median phenprocoumon dosage did not differ between husbands and wives (1.98 (range 0.87-3.29) *v* 2.03 (0.71-3.10) mg/day respectively; P = 0.88), and the dosage in matched husbands (2.27 (0.98-4.65) mg/day) was not significantly higher than the dosage in matched wives (1.98 (0.99-3.19) mg/day; P = 0.094). Similarly, couples with identical target levels of anticoagulation showed no differences in dosage. There was a significant linear correlation of phenprocoumon dosage between spouses ($r = 0.57$ (n = 33); P = 0.0008) (fig 1), which was not found in matched couples. After excluding the eight couples with different target levels of anticoagulation the correlation was not altered ($r = 0.59$ (n = 25); P = 0.0022) and again not significant in the matched couples.

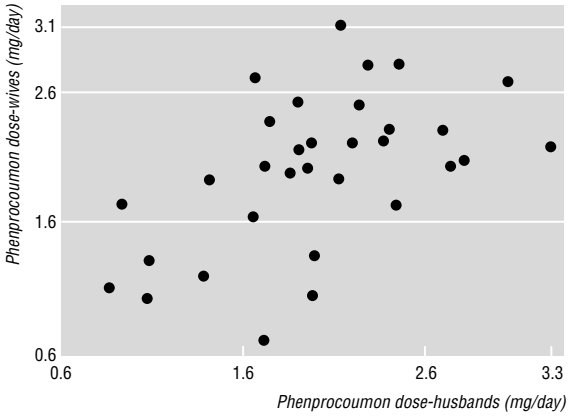

Fig 1 Linear correlation of phenprocoumon dosage in 33 husbands and their wives

The dosage difference within couples was smaller than the dosage difference within matched couples (mean difference of dosage differences -0.49 (95% confidence interval -0.78 to -0.20) mg/day). Similarly, the dosage differences within the 25 couples with identical target levels of anticoagulation were smaller than within the 25 matches (mean difference -0.44 (-0.77 to -0.07) mg/day). In none of the four groups was phenprocoumon dosage significantly correlated with age (all P > 0.30).

Comment

Coumarin derivatives act by inhibiting vitamin K dependent synthesis of several coagulation factors.[4] We found a significant relation in phenprocoumon dosage between spouses, which we assume was due to diet. A diet rich in vitamin K (broccoli, spinach, Brussels sprouts, or lettuce) leads to increased coagulation activity whereas a diet poor in vitamin K leads to stable anticoagulation.[5]

Our findings suggest that diet (and dietary vitamin K) is more important for individual coumarin requirements than is generally assumed.

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Drug points

Acute angle closure glaucoma associated with paroxetine

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We report an episode of acute angle closure glaucoma associated with the use of paroxetine.

An 84 year old woman was taking frusemide, digoxin, lisinopril, and ranitidine to control longstanding hypertension, atrial fibrillation, angina, and oesophagitis. She developed symptoms of depression, and treatment with paroxetine (Seroxat) was started at a dose of 10 mg daily, increasing after 10 days to 20 mg/day. After three days of treatment with the higher dose her right eye became painful and red, with blurring of vision. Ocular examination showed hypermetropia, the left eye having a narrow drainage angle predisposed to angle closure. The right eye showed established angle closure, with corneal oedema and a raised intraocular pressure of 40 mm Hg. Paroxetine was withdrawn, and the eyes responded to a standard regimen of medical treatment followed by laser iridotomies.

In this case there was a clear temporal link between paroxetine administration and the development of acute angle closure glaucoma. The latency of its development correlates with the expected rise in blood concentrations of the drug after the dose increase. This latency also correlates with the mechanism of action of selective serotonin reuptake inhibitors, which induce a gradual rise in postsynaptic concentrations of serotonin through desensitisation of the feedback systems controlling the rate limiting enzyme in serotonin synthesis.[1]

Acute angle closure glaucoma occurs in predisposed eyes with narrow drainage angles; the mid-dilated pupil blocks the circulation of aqueous. This can be induced by mydriatic (pupil dilating) agents, typically those with adrenergic or anticholinergic activity. Selective serotonin reuptake inhibitors, including paroxetine, have comparatively weak anticholinergic and adrenergic activity,[1] although stimulation with serotonin itself induces mydriasis and raises intraocular pressure in some animal studies,[2] and an effect on serotonin receptors should be considered as a possible alternative mechanism. In patients mydriasis is associated with administration of fenfluramine, which induces release of serotonin,[3] and with the selective serotonin reuptake inhibitor indalpine.[4] Mydriasis has been described with paroxetine itself, particularly in overdose,[5] although this could be due to enhanced anticholinergic action in high dose. The manufacturers are aware of a further case of acute angle closure glaucoma associated with paroxetine administration (SmithKline Beecham, personal communication).

This episode of acute angle closure glaucoma therefore probably occurred as a direct result of paroxetine administration. The most likely mode of action is a direct effect on the iris or ciliary body muscle through serotoninergic or anticholinergic mechanisms, or both. We think that further investigation into the association between selective serotonin reuptake inhibitors and acute angle closure glaucoma is warranted.

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Paroxetine and hepatotoxicity

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We report a case of biopsy confirmed chronic active hepatitis that was probably attributable to paroxetine. So far as we are aware, no other similar cases have been published, although there are a few reported cases of hepatitis associated with fluoxetine.[1-2]

A 54 year old woman was treated for depression. She had been receiving thyroxine 100 µg daily, isosorbide mononitrate 10 mg three times a day, atenolol 100 mg daily, and aspirin 300 mg daily for 22 months, drugs she continued to take over subsequent years. After two months of treatment with amitriptyline 50 mg daily, paroxetine 20 mg daily was substituted, a drug that she had not taken before. Results of liver function tests 10 months later were abnormal: aspartate transaminase concentration was 256 IU/l (normal 13-42 IU/l), with normal bilirubin and alkaline phosphatase concentrations. Liver function tests had given normal results two months before the start of paroxetine treatment. Results reached a maximum six months later (aspartate transaminase concentration 299 IU/l and alkaline phosphatase concentration 254 IU/l (normal 30-130 IU/l)). Subsequently, she received ethamsylate for six weeks, and she was diagnosed as having diabetes mellitus (treated by dietary intervention) 18 months after she started taking paroxetine.

Liver ultrasonography yielded normal results, and serological tests for hepatitis A, B, or C and for autoantibodies gave negative results. A liver biopsy specimen taken after 17 months of paroxetine treatment showed chronic active hepatitis with eosinophilic infiltration, suggesting a drug related cause. Paroxetine treatment was therefore stopped. Aspartate transaminase concentrations fell five weeks later to 63 IU/l, returning to normal after a further 13 weeks, although alkaline phosphatase concentration remained minimally increased at 150 IU/l. Alkaline phosphatase concentration subsequently returned to normal, and the results of liver function tests were within normal limits 15 months after paroxetine was stopped. A repeat liver biopsy could not be justified on ethical grounds.

The Committee on Safety of Medicines has been notified of six possible cases of hepatitis associated with paroxetine, although only two of them were confirmed by biopsy (personal communication). Transient abnormalities in liver function tests have, however, been recognised before in association with paroxetine,[3] and in 54 cases abnormal results without other evidence of hepatic disease have been reported to the committee. In our patient the temporal relation between paroxetine and confirmed hepatitis and its reversal after stopping the drug suggests a causal association.

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