Randomised placebo controlled trial of effect on mood of lowering cholesterol concentration

J Wardle, J Armitage, R Collins, K Wallendszus, A Keech, A Lawson, for the Oxford Cholesterol Study Group

Abstract

Objective—To evaluate the effects on mood of a substantial and prolonged reduction in total cholesterol concentration.

Design—Randomised placebo controlled comparison of patients who had been allocated to receive simvastatin 20 mg or 40 mg daily versus those allocated matching placebo in a ratio of 2:1. Follow up at an average of 152 weeks after randomisation.

Subjects—Men and women aged between 40 and 75 years at entry with blood total cholesterol of 3.5 mmol/l or greater, who were considered to be at higher than average risk of coronary heart disease based on medical history.

Main outcome measures—The shortened profile of mood states questionnaire, reported use of psychotropic medication, and symptoms possibly related to mood.

Results—Simvastatin reduced total cholesterol by 1.9 mmol/l (26.7%) at the time of follow up. Among all 621 patients randomised to simvastatin (414 patients) or placebo (207 patients) there were no significant differences in the use of psychotropic medication or in reports of symptoms possibly related to mood. Of these patients, 491 (334 simvastatin, 157 placebo) completed the mood questionnaire, and there were no significant differences between the treatment groups in total or subscale scores, even when patients with low baseline cholesterol concentrations or elderly subjects were considered separately.

Conclusion—These results do not support the hypothesis that treatment to lower cholesterol concentration causes mood disturbance.

Introduction

The apparent association between low¹⁻³ or lowered⁴ plasma cholesterol concentrations and death from causes other than illness, particularly suicide, has prompted suggestions that low cholesterol concentration might cause mood disturbances.⁵ In cross sectional studies associations between cholesterol concentration and depressive symptoms have been inconsistent, with a higher prevalence of depression among the group with the lowest concentrations in some studies or in selected subgroups⁶⁻⁸ but not in others.⁹⁻¹¹ An alternative explanation for these associations is that poor or declining health might cause both a fall in blood cholesterol concentrations and an increase in depressive illness.^{8, 12}

Evidence for the effects of lowering blood cholesterol on mood states and behaviours is limited. Studies in monkeys have shown low fat diets to be associated with aggressive behaviour.¹³ One intervention study in humans evaluated the effects on mood of dietary treatment to lower cholesterol concentration and found no adverse effects, but the study lacked any control group and the reduction in cholesterol was minimal.¹⁴

Inhibitors of 3-hydroxy-3-methylglutaryl-coenzyme A (HMG CoA) reductase, a rate limiting enzyme in hepatic cholesterol synthesis, produce large, sustained reductions in cholesterol concentrations and are now widely used to treat hypercholesterolaemia.¹⁵ Recently, a randomised placebo controlled trial of one of these agents, lovastatin, showed no effect on patients' ratings of subjective health or emotional wellbeing during one year's treatment of older adults with mild to moderate hypercholesterolaemia.¹⁶ The present study concerns data on mood states and use of psychotropic drugs after about three years of treatment in the Oxford cholesterol study,¹⁷ a randomised placebo controlled study of simvastatin, another HMG CoA reductase inhibitor.

Methods

A detailed description of the design and three year follow up results of the study is reported elsewhere,¹⁷ and details relevant only to the present study are presented here. The Central Oxford Research Ethics Committee approved the study protocol.

ELIGIBILITY AND ROUTINE DATA COLLECTION

The participants in the study were 621 men and women aged between 40 and 75 years with blood total cholesterol concentration of 3.5 mmol/l or greater, who were considered to be at higher than average risk of coronary heart disease (because of a history of myocardial infarction, angina pectoris, stroke, transient ischaemic attack, peripheral vascular disease, treated diabetes mellitus, or hypertension) and for whom there were neither contraindications to, nor specific indications for, HMG CoA reductase inhibitors. Subjects were also excluded if there was evidence of alcohol or drug misuse or of psychiatric disability which might limit compliance. At the initial screening visit to the clinic for the study, patients completed a detailed questionnaire about their medical and treatment history, blood pressure was measured, and a non-fasting blood sample was taken for laboratory analysis. All screened subjects were given dietary information similar to that contained in the American Heart Association step 1 diet guidelines. After an eight week, single blinded placebo run in period, subjects who were compliant with medication and were willing to continue with treatment for at least five years were evenly randomised to take 40 mg simvastatin daily, 20 mg simvastatin, or placebo. Blinded follow up of mortality and major morbidity still continues.

After the randomisation clinic visit (study week 0) all patients were to be seen at eight weeks, then at 12 weekly intervals for about one year, and then at 24 weekly intervals. At each follow up visit, patients were asked whether they had experienced any of a prespecified list of symptoms (including insomnia or fatigue). Any other symptoms volunteered (such as depression or irritability) were recorded. All hospital admissions, possible adverse drug reactions, and

Imperial Cancer Research Fund, Health Behaviour Unit, University College London, London WC1E 6BT
J Wardle, reader in health psychology

British Heart Foundation/ Medical Research Council/ Imperial Cancer Research Fund Clinical Trial Service Unit, Nuffield Department of Clinical Medicine, University of Oxford, Radcliffe Infirmary, Oxford OX2 6HE
J Armitage, senior registrar in public health medicine
R Collins, senior research fellow
K Wallendszus, research officer
A Keech, honorary senior registrar in cardiology
A Lawson, research nurse

Correspondence to: Dr Wardle.

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reasons for non-compliance with treatment were recorded. Additionally, at annual visits, details of other medication (including psychotropic drugs) being taken at the time of follow up were recorded. Non-fasting blood samples were taken for laboratory analysis from all patients attending follow up visits. Patients were encouraged to attend their scheduled follow up visits even if they had stopped study treatment. Those who declined further clinic follow up had their study treatment stopped but were still to be followed at the scheduled time by telephone or through their general practitioner.

PROFILE OF MOOD STATE QUESTIONNAIRE

Between April and October 1992, at an average of about 152 weeks after randomisation, mood was assessed with a shortened version of the profile of mood state questionnaire. This abbreviated version was quicker and simpler to complete than the full questionnaire and was sent to all surviving patients in the study who were attending the clinic or being followed up by telephone for their completion and return. The questionnaire has six subscales: tension/anxiety, anger/hostility, fatigue/inertia, confusion/bewilderment, depression/dejection, and vigour/activity. The six items from each subscale that loaded highest on each factor in the factor analysis¹⁸ were used in the shortened mood scale.¹⁹ This reduces the burden on the patients without seriously compromising the validity of the scale.²⁰ Patients were given a list of the 36 adjectives (for example, "fatigued") and were asked to rate them in the standard fashion on a scale ranging from 0 (not at all) to 4 (extremely) according to how they had been feeling during the past week. Factor analysis of the results in the present study (data not shown) revealed a questionnaire structure closely similar to that reported for the full scale in the manual.¹⁸ The scores for each set of related adjectives were added together to produce the subscale scores, and a summary total score for each patient was calculated by adding the results for the first five subscales and subtracting the last subscale (that is, vigour/activity).

STATISTICAL ANALYSIS

The principal assessment of the effects of the treatment to lower cholesterol concentration on mood

was based prospectively on the comparison of the results of patients allocated to either of the two simvastatin groups (as both doses had been shown to produce similar large reductions in cholesterol concentration¹⁷) versus the placebo group. A subsidiary assessment entailed comparisons of 40 mg simvastatin daily versus 20 mg simvastatin. All comparisons were made by allocated treatment group among patients completing the questionnaire irrespective of compliance (that is, "intention to treat" except that non-survivors and some other patients did not complete the questionnaire; see below). χ^2 Tests for heterogeneity and trend were used to compare the proportion of patients with different mood scores between the treatment groups. As these data tended to be skewed, medians with upper and lower quartiles are given. Use of psychotropic medication after randomisation was used as another index of psychological disturbance. Two sided P values are used throughout, and differences are described as non-significant if $2P > 0.05$. The study had good power to detect a shift in the distribution of the total score of the profile of mood questionnaire among patients in the combined simvastatin group, such that at least two thirds (rather than half) had scores above the median score of the placebo control group.

Results

PATIENT CHARACTERISTICS

At the time of administration of the questionnaire 49 of the original sample of 621 patients had died and nine were not being followed up in the clinic or by telephone. Completed questionnaires were obtained from 491 (87%) of the 563 survivors offered the questionnaire. The proportions completing the mood questionnaires were similar in each treatment group (166 allocated to 40 mg simvastatin, 168 to 20 mg simvastatin, and 157 to placebo). Baseline characteristics of the 491 patients who completed the questionnaire (table 1) were similar to those of the 621 originally randomised.¹⁷ Non-completers, however, were more likely to be smokers (22% v 12%) and to have a history of myocardial infarction (72% v 59%) and less likely to be married (76% v 86%). The average blood cholesterol concentration at screening was 7.0 mmol/l, the average age was 63.5 years, and 84% of the patients were men. All characteristics recorded were well balanced between groups at randomisation, although slightly more of those allocated to simvastatin were already taking psychotropic medication.

Compliance with study treatment (defined as more than 90% of scheduled tablets being taken) was good, and the numbers who had stopped study treatment were evenly balanced between the treatment groups (26 of those allocated 40 mg simvastatin versus 19 allocated 20 mg simvastatin versus 20 allocated placebo). Changes in lipids were similar to those in the study as a whole¹⁷ with mean (SE) total cholesterol concentration reduced at the time of the questionnaire by 2.0 (0.1) mmol/l (28.3 (1.9)% reduction) among those allocated 40 mg daily simvastatin and by 1.8 (0.1) mmol/l (25.3 (1.6)% reduction) among those allocated 20 mg daily simvastatin compared with 0.1 (0.1) mmol/l (1.0 (1.5)% reduction) in the placebo group. Sustained reductions of this magnitude (an average of 1.9 mmol/l or 26.7% among those allocated simvastatin) maintained for nearly three years provide a strong basis on which to evaluate any possible adverse effects of lowering cholesterol concentration on mood.

ASSESSMENT OF MOOD

Table 2 shows the distribution of subscale scores on the profile of mood state questionnaire and the total mood disturbance scores for those patients who completed the questionnaire. There were no significant

Table 1—Baseline characteristics of all randomised patients. Figures are numbers (percentages) of subjects unless stated otherwise

Detail	Patients completing mood questionnaire		Patients not completing mood questionnaire	
	Simvastatin 20 mg or 40 mg (n = 334)	Placebo control (n = 157)	Simvastatin 20 mg or 40 mg (n = 80)	Placebo control (n = 50)
Mean age (years)	63.3	63.8	63.7	63.6
Mean total cholesterol (mmol/l)	7.0	6.9	7.1	7.2
Mean calculated low density lipoprotein cholesterol (mmol/l)	4.8	4.7	4.8	4.7
Mean high density lipoprotein cholesterol (mmol/l)	1.17	1.14	1.14	1.21
Mean triglycerides (mmol/l)	2.52	2.59	2.69	2.77
Men	282 (84)	132 (84)	70 (88)	42 (84)
Current smokers	44 (13)	16 (10)	14 (18)	14 (28)
Prior myocardial infarction	195 (58)	95 (61)	60 (75)	33 (66)
Prior cerebrovascular accident	30 (9)	17 (11)	7 (9)	3 (6)
Prior peripheral vascular disease	32 (10)	14 (9)	9 (11)	7 (14)
Treated diabetes	11 (3)	6 (4)	2 (3)	1 (2)
Married	286 (86)	137 (87)	64 (80)	35 (70)
Any psychotropic drugs before entry:				
Minor tranquillisers	16 (5)	6 (4)	9 (11)	4 (8)
Major tranquillisers	4 (1)	0	0	0
Prochlorperazine	3 (1)	0	3 (4)	0
Antidepressants	12 (4)	6 (4)	5 (6)	1 (2)
Lithium	0	0	0	0

Table 2—Distribution of scores on profile of mood state for patients completing questionnaire*

Subscale and treatment	Profile of mood state score							Any simvastatin v placebo (χ^2 tests)	
	0	1 to 4	5 to 8	9 to 12	13 to 16	17 to 20	21 to 24	Median (quartiles)	Heterogeneity
Tension/anxiety:									
Simvastatin	2%	46%	32%	12%	5%	2%	1%	5.0 (3.0,8.0)	3.57
Placebo	3%	40%	34%	16%	5%	1%	0%	5.0 (3.0,8.0)	0.07
Anger/hostility:									
Simvastatin	24%	46%	22%	6%	2%	1%	0%	3.0 (1.0,5.0)	3.60
Placebo	23%	49%	17%	10%	1%	1%	0%	3.0 (1.0,5.0)	0.01
Fatigue/inertia:									
Simvastatin	7%	29%	29%	17%	12%	3%	2%	6.0 (3.0,10.0)	10.28
Placebo	5%	28%	36%	18%	5%	7%	2%	6.0 (3.0,10.0)	0
Confusion/bewilderment:									
Simvastatin	5%	54%	29%	7%	4%	0%	0%	4.0 (2.0,6.0)	5.49
Placebo	3%	55%	30%	9%	2%	1%	0%	4.0 (2.0,6.0)	0.11
Depression/dejection:									
Simvastatin	43%	41%	12%	2%	0%	1%	0%	1.0 (0.0,3.0)	1.14
Placebo	43%	43%	11%	1%	1%	1%	0%	1.0 (0.0,3.0)	0.15
Vigour/activity:									
Simvastatin	1%	7%	20%	35%	24%	11%	2%	11.0 (8.0,14.0)	7.43
Placebo	2%	6%	23%	28%	32%	7%	1%	11.0 (8.0,14.0)	0.17
Total score:	-24 to -1	0	1 to 24	25 to 48	49 to 72	73 to 96	97 to 120		
Simvastatin	25%	2%	53%	16%	3%	1%	0%	8.0 (-0.5,22.5)	1.66
Placebo	25%	4%	52%	15%	4%	1%	0%	9.5 (-1.0,20.0)	0.08

*POMS questionnaires were completed by 334 patients allocated simvastatin and 157 allocated placebo. All χ^2 tests were non-significant.

Table 3—Medians (quartiles) of profile of mood state total score subdivided by baseline characteristics

Characteristic	Total score*	
	Simvastatin 20 mg or 40 mg	Placebo control
Baseline cholesterol (mmol/l):		
< 7.0 (mean 6.1)†	9.0 (0.0,24.0)	8.5 (-2.0,23.0)
≥ 7.0 (mean 8.0)†	7.0 (-2.0,20.0)	10.0 (1.0,20.0)
Sex:		
Male	8.5 (-1.0,23.0)	9.0 (-1.0,20.0)
Female	5.0 (0.0,22.0)	10.0 (5.0,25.0)
Age at randomisation (years):		
<70	8.0 (-1.0,22.0)	9.0 (0.0,20.0)
≥70	10.0 (1.0,23.0)	12.5 (-6.0,20.0)

*No significant differences between treatment groups in any of these subgroups.

†Mean (SE) reduction in cholesterol concentration with simvastatin was 1.5 (0.1) mmol/l for those with a mean baseline concentration of 6.1 mmol/l and 2.4 (SE 0.2) mmol/l for those with a mean baseline concentration of 8.0 mmol/l.

differences in the distribution of any of these scores between the two simvastatin groups combined and the placebo group. Nor were there any differences in the subsidiary comparison between those allocated 40 mg daily simvastatin and those allocated 20 mg daily simvastatin (data not shown). To evaluate the possibility that only people with lower baseline cholesterol concentrations would be susceptible to any adverse effects on mood of a reduction in concentration, the results for the 258 patients with baseline concentrations below the mean of 7.0 mmol/l were analysed separately. We found no significant differences between subjects allocated to simvastatin or placebo (table 3). Nor were there significant differences between the treatment groups among men and women analysed separately nor among patients aged 70 years or above, whom, it has been suggested,⁶ might be more susceptible to any effects on mood of lowering cholesterol concentration.

By the time of the administration of the questionnaire patients had been followed up an average of 10 times since randomisation. The proportions of patients completing the questionnaire who had reported any

symptoms that might possibly be related to mood were evenly balanced between the treatment groups (table 4), as were the proportions who had stopped study treatment for any reason. Adverse effects on mood were also evaluated by comparison of the proportions of patients in each treatment group who reported the use of psychotropic medication after randomisation. Neither among patients completing the mood questionnaire nor among all randomised patients were there significant differences between the simvastatin and placebo groups, either in the proportions of patients using psychotropic drugs at any time after randomisation (despite a non-significant chance excess of use before randomisation in the simvastatin groups) or in the proportions starting such drugs after randomisation. (Nor were significant differences between the treatment groups observed in any of these outcome measures with continued follow up to November 1995—that is, an average of six years after randomisation.)

Discussion

The data for these analyses were obtained by taking the opportunity provided by an ongoing randomised placebo controlled trial of a drug treatment that produces substantial and sustained reductions in plasma cholesterol concentrations.¹⁷ A comparison of mood scores derived from a shortened version of the profile of mood state questionnaire allowed us to evaluate the possibility that lowering cholesterol concentration is associated with adverse effects on mood.⁵⁻⁸ There was a high response rate to the mood questionnaire (87%) and, although completers differed from non-completers on certain baseline characteristics, there was no difference in response rate between the simvastatin and placebo groups and therefore no reason to suspect any bias in the results. In this study, there was no evidence after nearly three years that the substantial reduction in cholesterol concentration produced by simvastatin was associated with any adverse mood effects. Nor was there any evidence of adverse effects on patients whose baseline cholesterol concentration was lower than average and so would have been reduced to lower absolute concentrations. There was a slightly, though non-significantly, higher use of psychotropic drugs in the simvastatin group before randomisation

Table 4—Any reports of symptoms possibly related to mood or use of psychotropic drugs at clinic visits up to 31 October 1992. Figures are numbers (percentages) of subjects

Reported event	Patients completing mood questionnaire		Patients not completing mood questionnaire	
	Simvastatin 20 mg or 40 mg (n = 334)	Placebo control (n = 157)	Simvastatin 20 mg or 40 mg (n = 80)	Placebo control (n = 50)
Insomnia*	165 (49)	84 (54)	37 (46)	28 (56)
Difficulty getting to sleep	16 (5)	7 (4)	2 (3)	1 (2)
Disturbed sleep	16 (5)	10 (6)	3 (4)	5 (10)
Early waking	18 (5)	6 (4)	2 (3)	0
Fatigue*	194 (58)	91 (58)	41 (51)	33 (66)
Depression	18 (5)	6 (4)	5 (6)	5 (10)
Irritability	5 (1)	1 (1)	0	0
Personality change	2 (1)	0	0	0
Stopped study treatment and main reason:				
Fatigue	47 (14)	20 (13)	32 (40)	16 (32)
Personality change	3 (1)	0	0	0
Any other reason	2 (1)	0	0	0
Psychotropic drugs†:				
Started before randomisation	42 (13)	20 (13)	32 (40)	16 (32)
Started after randomisation	19 (6)	6 (4)	8 (10)	1 (2)
Any use after randomisation	11 (3)	4 (3)	3 (4)	1 (2)
Any use after randomisation	30 (9)	10 (6)	11 (14)	2 (4)

*Routinely sought symptoms.

†Includes major tranquillisers, prochlorperazine, antidepressants, and lithium; excludes minor tranquillisers.

and the start of study treatment but no evidence for differences between groups in new use of psychotropic drugs during the treatment period (up to more than five years after randomisation).

The selection procedures for this trial should be considered in the interpretation and generalisation of the results. Patients entered in this study had to have cardiovascular disease and to be free from psychiatric conditions which might compromise compliance. Moreover, only those who were successfully compliant in the placebo "run in" period before randomisation were included in the trial. This may have produced a group with a combination of cardiovascular disease and unusually good psychological health in whom vulnerability to any adverse effects of a reduction in cholesterol concentration might be different from that of other populations. In view of the fact that long term compliance is required for clinical use of cholesterol lowering drugs, however, these results are directly relevant to other patients who comply with a pharmacological regimen.

Overall, these results give little support to the idea that reducing cholesterol concentration has adverse effects on mood in a psychologically healthy sample. The patients treated with simvastatin, who had experienced a substantial and prolonged reduction in concentration, were no more depressed, anxious, hostile, or confused and no more likely to start psychotropic medication than those who had been on placebo and had had a minimal reduction in cholesterol concentration.

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Key messages

- Some studies have suggested that low or lowered cholesterol concentration might be associated with depression and other mood disturbances
- A randomised placebo controlled trial of simvastatin (a drug that lowers cholesterol concentration substantially) showed no associated mood disturbance over a period of nearly three years in subjects with cardiovascular disease and no pre-existing psychiatric conditions
- No effects on mood were seen in individuals with low baseline cholesterol concentrations or in older individuals, and there were no differences in use of psychotropic drugs between the treatment groups
- These results give little support to the idea that reducing cholesterol concentration has adverse effects on mood

Conflict of interest: None.

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