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Randomised controlled trial of Alexander technique lessons, exercise, and massage (ATEAM) for chronic and recurrent back pain

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ABSTRACT

Objective To determine the effectiveness of lessons in the Alexander technique, massage therapy, and advice from a doctor to take exercise (exercise prescription) along with nurse delivered behavioural counselling for patients with chronic or recurrent back pain.

Design Factorial randomised trial.

Setting 64 general practices in England.

Participants 579 patients with chronic or recurrent low back pain; 144 were randomised to normal care, 147 to massage, 144 to six Alexander technique lessons, and 144 to 24 Alexander technique lessons; half of each of these groups were randomised to exercise prescription.

Interventions Normal care (control), six sessions of massage, six or 24 lessons on the Alexander technique, and prescription for exercise from a doctor with nurse delivered behavioural counselling.

Main outcome measures Roland Morris disability score (number of activities impaired by pain) and number of days in pain.

Results Exercise and lessons in the Alexander technique, but not massage, remained effective at one year (compared with control Roland disability score 8.1: massage −0.58, 95% confidence interval −1.94 to 0.77, six lessons −1.40, −2.77 to −0.03, 24 lessons −3.4, −4.76 to −2.03, and exercise −1.29, −2.25 to −0.34). Exercise after six lessons achieved 72% of the effect of 24 lessons alone (Roland disability score −2.98 and −4.14, respectively). Number of days with back pain in the past four weeks were lower after lessons (compared with control median 21 days: 24 lessons −18, six lessons −10, massage −7) and quality of life improved significantly. No significant harms were reported.

Conclusions One to one lessons in the Alexander technique from registered teachers have long term benefits for patients with chronic back pain. Six lessons followed by exercise prescription were nearly as effective as 24 lessons.

Trial registration National Research Register N0028108728.

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INTRODUCTION

Few interventions have been proved to substantially help patients with chronic back pain in the longer term. Supervised classes of mainly strengthening and stabilising exercises probably have moderate benefit.[1-5] A trial of doctors' advice to take aerobic exercise showed short term benefit for acute pain,[6] but the evidence of longer term benefit for chronic or recurrent pain and for exercise "prescriptions" is lacking.[7]

Lessons in the Alexander technique offer an individualised approach to develop skills that help people recognise, understand, and avoid poor habits affecting postural tone and neuromuscular coordination. The practice and theory of the technique, in conjunction with preliminary findings of changes in postural tone and its dynamic adaptability to changes in load and position,[8-10] support the hypothesis that it could potentially reduce back pain by limiting muscle spasm, strengthening postural muscles, improving coordination and flexibility, and decompressing the spine.

We determined the effectiveness of six or 24 lessons in the Alexander technique, massage therapy, and advice from a doctor to take exercise with nurse delivered behavioural counselling for patients with chronic or recurrent back pain.

METHODS

We recruited 64 general practices in the south and west of England; 152 local teachers and therapists agreed to participate. Each practice wrote to a random selection of patients who had attended with back pain in the past five years (see bmj.com for inclusion criteria). At the baseline appointment participants were randomised to one of eight groups (see bmj.com) using computer generated random numbers. When possible each practice was matched to two Alexander technique teachers.

The primary outcome measures were disability, measured using the Roland Morris disability questionnaire,[11 12] and number of days in pain during the past four weeks.[13] Secondary outcome measures were quality of life (SF-36)[14] and secondary measures for back pain[11]: pain and disability (Von Korff scale[13] and Deyo "troublesomeness" scale[11]), overall improvement using health transition,[15] and fear avoidance beliefs for physical activity.[16] For other measures we asked patients to agree or disagree with statements on 7 point scales from 0=strongly agree to 7=strongly disagree. We developed a back health scale (see bmj.com).[17]

We measured outcomes at baseline, three months, and one year using postal questionnaires, with two mailings to non-responders and telephone follow-up for a smaller dataset (Roland disability scale, days in pain, Von Korff scale, health transition) for those not responding. Data entry was blind to study group.

Analysis

The primary analysis was an analysis of covariance for a factorial study at one year for Roland disability score between groups and for secondary outcomes. The days in pain data were skewed so we used non-parametric (quantile) regression. We assessed interaction between Alexander technique and exercise factors before reporting the main effects. As the study was powered for only moderately large interactions we also report the individual groups for the main outcomes at one year. We assessed the statistical significance of clustering by therapist, teacher, and practice, and if these were not significant we did not allow for clustering in the models.

RESULTS

A total of 579 people were randomised and completed the baseline questionnaires, 469 (81%) completed the questionnaires at three months, and 463 (80%) the questionnaires at 12 months (see bmj.com). Responders at one year were more likely to have left full time education later and to be self employed or homemakers; response was not related to baseline Roland disability scores. Including education and employment status in the final analysis did not alter the estimates or the inferences. No significant cluster effects were found, except for enablement, which showed a practice clustering effect. Baseline characteristics were similar for all variables (see bmj.com) except for fewer women in the Alexander technique groups. Estimates were unaltered by including sex in the models.

The trial population had on average 243 (SD 131) days of pain in the previous year. Seventy nine per cent reported 90 or more days of pain in the previous year.

Little change occurred in Roland disability score or days in pain in the control group (see bmj.com).

Outcomes at one year after randomisation: mean difference compared with control group (95% confidence intervals) unless specified otherwise

Outcomes	Mean (SD) control (Alexander technique factor*)	Mean difference compared with control, P value			Mean (SD) control (exercise factor*)	Mean difference compared with control: exercise
		Massage	6 lessons	24 lessons		
Primary outcomes						
Roland disability score (n=462)†	8.07 (6.13)	−0.58 (−1.94 to 0.77), P=0.399	−1.40 (−2.77 to −0.03), P=0.045	−3.40 (−4.76 to −2.03), P<0.001	7.54 (6.25)	−1.29 (−2.25 to −0.34), P=0.008
Median (95% CI) No of days with back pain in past 4 weeks (n=435)‡	21 (18 to 25)	−7 (−12 to −2), P=0.004	−10 (−15 to −5), P<0.001	−18 (−23 to −13), P<0.001	13 (11 to 15)	−2 (−5 to 1), P=0.233
Secondary outcomes						
SF-36: quality of life physical (n=403)§	56.4 (18.5)	1.7 (−4.0 to 7.4), P=0.553	6.0 (0.30 to 11.6), P=0.039	11.3 (5.7 to 16.9), P<0.001	59.5 (18.5)	1.9 (−1.97 to 5.79), P=0.333
SF-36: quality of life mental (n=341)§	65.2 (17.4)	−0.1 (−5.5 to 5.2), P=0.956	2.0 (−3.4 to 7.5), P=0.460	4.0 (−1.4 to 9.3), P=0.145	66.5 (17.3)	0.9 (−2.8 to 4.6), P=0.636
Modified enablement scale (n=366)¶	3.80 (1.20)	1.29 (0.93 to 1.64), P<0.001	1.31 (0.95 to 1.67), P<0.001	1.80 (1.44 to 2.16), P<0.001	4.69 (1.19)	0.50 (0.24 to 0.76), P<0.001
Von Korff overall** (n=412):	3.96 (2.32)	−0.02 (−0.64 to 0.59), P=0.939	−0.60 (−1.22 to 0.007), P=0.053	−1.15 (−1.75 to −0.55), P<0.001	3.83 (2.36)	−0.59 (−1.01 to −0.17), P=0.006
Von Korff disability††	3.34 (2.24)	0.03 (−0.63 to 0.68), P=0.938	−0.57 (−1.23 to 0.08), P=0.085	−0.95 (−1.60 to −0.30), P=0.004	3.22 (2.23)	−0.59 (−1.04 to −0.14), P=0.011
Von Korff pain††	4.54 (2.19)	−0.01 (−0.65 to 0.63), P=0.981	−0.58 (−1.22 to 0.06), P=0.075	−1.30 (−1.93 to −0.67), P<0.001	4.40 (2.18)	−0.59 (−1.04 to −0.14), P=0.011
Back health transition (n=430)‡‡	3.67 (1.14)	−0.63 (−0.93 to −0.32), P<0.001	−0.55 (−0.86 to −0.24), P<0.001	−0.97 (−0.75 to −0.31), P<0.001	3.38 (2.83)	−0.53 (−0.75 to −0.31), P<0.001
Deyo troublesomeness (n=462)§§	2.94 (0.75)	0.05 (−0.16 to 0.26), P=0.627	−0.16 (−0.37 to 0.05), P=0.132	−0.34 (−0.55 to −0.12), P=0.002	2.94 (0.85)	−0.16 (−0.31 to −0.01), P=0.036
Fear avoidance for physical activity (n=350)¶¶	13.6 (5.3)	−0.23 (−1.86 to 1.39), P=0.777	−1.41 (−3.03 to 0.21), P=0.088	−2.28 (−3.90 to −0.67), P=0.006	13.2 (5.3)	−1.87 (−2.99 to −0.75), P=0.001
Back health (n=362)***	3.44 (1.45)	1.13 (0.69 to 1.56), P<0.001	1.26 (0.82 to 1.71), P<0.001	1.82 (1.38 to 2.25), P<0.001	4.15 (1.45)	0.74 (0.44 to 1.04), (P<0.001
Satisfaction with overall management (n=319)	3.17 (1.04)	0.47 (0.11 to 0.82), P=0.01	0.58 (0.22 to 0.93), P=0.001	0.70 (0.35 to 1.04), P<0.001	3.45 (1.21)	0.47 (0.22 to 0.71), P=0.001

Cronbach's α for scales: Deyo troublesomeness 0.87, Von korff 0.95, fear avoidance 0.80, enablement 0.96, back health 0.96.
*Effects in each factor are mutually controlled for other factor. Thus the control group for each factor are those that did not receive interventions for that factor. Interventions for each factor are expressed as estimated difference compared with control group, with 95% confidence intervals.
†Number of activities affected by back pain; 28=worst 0=best.
‡As a result of an administrative error at baseline, not all patients had a questionnaire containing question for days in pain at baseline, so model for days in pain does not include baseline values.
§0=worst, 100=best.
¶Mean of six items; 0=worst 6=best.
**Mean of six items; 10=worst 0=best.
††Mean of three items; 10=worst 0=best.
‡‡Back pain changed; 7=vastly worsened, 4=no change, 1=completely recovered.
§§Mean of three items; 5=worst, 1=best.
¶¶Sum of four items; 24=worst, 0=best.
***Mean of four items; 0=worst 6=best.

WHAT IS ALREADY KNOWN ON THIS TOPIC

Combined manipulation and physiotherapy-supervised exercises helps functioning moderately (1-2 activities no longer limited by back pain)

Preliminary evidence suggests that massage and lessons in the Alexander technique might help in the short term

WHAT THIS STUDY ADDS

Six sessions of massage, prescription for exercise and nurse counselling, six lessons in the Alexander technique, and 24 lessons helped with back pain and functioning at three months

Lessons in the Alexander technique still had a beneficial effect on pain and functioning after 12 months

Six lessons in the Alexander technique followed by exercise prescription are nearly as effective as 24 lessons after 12 months

Compared with the control group, significant reductions took place for all interventions at three months for Roland disability score and days in pain.

The effect of 24 lessons in the Alexander technique was greater at one year than at three months, with a 42% reduction in Roland disability score and an 86% reduction in days in pain compared with the control group (table). The effect of six lessons was maintained—a 17% reduction in Roland disability score and a 48% reduction in days in pain. Exercise still had a significant effect on Roland disability score (17% reduction) but not on days in pain. Massage no longer had an effect on Roland disability score but days in pain was reduced (by 33%). Twenty four lessons also had a significant effect on other outcomes; similar but smaller changes followed six lessons. Massage produced little change in other outcomes except perception of overall improvement in back pain (health transition), enablement, and overall satisfaction.

Good adherence (see bmj.com for definitions) was achieved by 91% (108/119) of patients in the group receiving massage, 94% (106/113) in the group receiving six Alexander technique lessons, and 81% (95/117) in the group receiving 24 lessons. Adequate adherence for exercise prescription was achieved by 76% (211/278) of patients. No meaningful change occurred in the results when selecting only patients with good adherence.

The effect of exercise combined with 24 Alexander technique lessons on Roland disability score and other outcomes was similar to the effect of 24 lessons alone (see bmj.com). The effect of six lessons followed by exercise prescription on Roland disability score and most other outcomes was almost as good (72% as effective) as 24 lessons at one year.

DISCUSSION

Twenty four lessons in the Alexander technique taught by registered teachers provides long term benefits for patients with chronic or recurrent low back pain. Both six lessons in the Alexander technique and general practitioner prescription for aerobic exercise with structured behavioural counselling by a practice nurse were helpful in the long term; classic massage provided short term benefit. Six lessons in the Alexander technique followed by exercise prescription was almost as effective as 24 lessons.

Most of the eligible patients who responded to an invitation to participate were randomised so the results should apply to most patients with chronic or recurrent back pain. The long previous duration of pain (79% had pain for >90 days) and the little change in pain and function in the control group after one year suggest that we selected a predominantly chronic, severely affected, and currently ineffectively managed population. All had attended primary care with back pain in the past.

Adherence was good for both six and 24 lessons on the Alexander technique, and for massage compared with adherence in other back pain intervention trials,[5] possibly as a result of the perceived symptomatic benefit.

Although the study was underpowered to assess significant interactions (see sample size calculation on bmj.com) the results suggest that the effect of exercise and 24 Alexander technique lessons combined is less than the sum of the two individual effects. We found no evidence of confounding or bias from losses to follow-up.

The Roland disability scale is one of the best validated self report measures for assessing the impact of back pain.[11,12] The effect of intervention on reported days in pain is unlikely to be explained by recall bias owing to the large effect size and short period of recall. Any non-differential measurement error owing to the use of reported days in pain is likely to underestimate true differences between groups.

Interventions

The previous trial for back pain was smaller and involved one teacher.[18] Our study shows enduring benefits from lessons delivered by many different teachers. That six sessions of massage were much less effective at one year than at three months whereas six lessons in the Alexander technique retained effectiveness at one year shows that the long term benefit of the lessons is unlikely to result from non-specific placebo effects of attention and touch.

Massage is helpful in the short term, which supports tentative conclusions from previous research.[19,20] Benefit in the longer term is probably less, which is supported by previous comparison with a self care booklet.[21]

Prescription from a general practitioner for home based aerobic exercise with follow-up structured counselling provided modest but useful benefits from a relatively brief intervention. Comparison with the United Kingdom back pain exercise and manipulation trial suggests the benefits are similar to a supervised exercise scheme in the short term, and potentially greater in the long term, since the effect of supervised schemes in that trial was no longer apparent by 12 months.[5] Although 24 lessons provided significant and important benefit, six lessons in the Alexander technique followed by prescription for exercise provided nearly as much benefit as 24 lessons on the Alexander technique.

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1 Van Tulder MW, Malmivarra A, Esmail R, Koes B. Exercise therapy for low back pain. *Cochrane Database Syst Rev* 2000;(2):CD000335. Review. Update in: *Cochrane Database Syst Rev* 2005;(3):CD000335.
2 Hayden J, van Tulder MW, Malmivaara A, Koes B. Meta-analysis: exercise therapy for non-specific low back pain. *Ann Intern Med* 2005;142:765-75.
3 Frost H, Klaber-Moffett J, Moser JS, Fairbank JCT. Randomised controlled trial for evaluation of fitness programme for patients with chronic low back pain. *BMJ* 1995;310:151-4.
4 Klaber-Moffett J, Torgerson D, Bell-Syer S, Jackson D, Llewelyn-Phillips H, Farrin A, et al. Randomised controlled trial of exercise for low back pain: clinical outcomes, costs and preferences. *BMJ* 1999;319:279-83.
5 UK BEAM Trial Team. United Kingdom back pain exercise and manipulation (UK BEAM) randomised trial: effectiveness of physical treatments for back pain in primary care. *BMJ* 2004;329;1377-81.
6 Little P, Roberts L, Blowers H, Garwood J, Cantrell T, Langridge J, et al. Should we give detailed advice and information booklets to patients with back pain?: a randomised controlled factorial trial of a self management booklet and doctor advice to take exercise for back pain. *Spine* 2001;26:2065-72.
7 Hillsdon M, Thorogood M, Antiss T, Morris J. Randomised controlled trials of physical activity promotion in free living populations: a review. *J Epidemiol Community Health* 1995;49:448-53.
8 Cacciatore T, Gurfinkel V, Horak F, Cordo P, Ames K. Alteration of muscle tone through conscious intervention: increased adaptability of axial and proximal tone through the Alexander technique. *Proceedings of the International Society for Posture and Gait Research* Vermont, USA, 14-18 Jul 2007;18.
9 Cacciatore T, Horak F, Henry S. Improvement in automatic postural coordination following Alexander technique lessons in a person with low back pain. *Phys Ther* 2005;85:565-78.
10 Gurfinkel V, Cacciatore T, Cordo P, Horak F, Nutt J, Skoss R. Postural muscle tone in the body axis of healthy humans. *J Neurophysiol* 2006;96:2678-87.
11 Deyo R. Outcome measures for low back pain research: a proposal for standardising use. Proceedings of the 2nd international forum for primary care research on low back pain, 30-31 May 1997, the Hague.
12 Patrick DL, Deyo RA, Atlas SJ, Singer DE, Chapin A, Keller RB. Assessing health-related quality of life in patients with sciatica. *Spine* 1995,20:1899-908.
13 Von Korff M, Ormel J, Keefe M, Dworkin S. Grading the severity of chronic pain. *Pain* 1992;50:133-49.
14 Ware J, Sherbourne C. The SF36 health status survey 1: conceptual framework and item selection. *Med Care* 1996;30:473-83.
15 Beurskens A, de Vet H, Koke A. Responsiveness of functional status in low back pain: a comparison of different instruments. *Pain* 1996;65:71-6.
16 Waddell G, Newton M, Henderson I, Somerville D, Main C. A fear-avoidance beliefs questionnaire (FABQ) and the role of fear avoidance beliefs in chronic low back pain and disability. *Pain* 1993;52:157-68.
17 Howie J, Heaney D, Maxwell M, Walker J, Freeman G, Rai H. Quality at general practice consultations: cross sectional survey. *BMJ* 1999;319:738-43.
18 Ernst E, Canter P. The Alexander technique: a systematic review of controlled clinical trials. *Forschende Komplementärmedizin und Klassische Naturheilkunde/Research in Complementary and Classical Natural Medicine* 2003;10:325-9.
19 Furlan A, Brosseau M, Imamura M, Ivin E. Massage for low-back pain. *Cochrane Database Syst Rev* 2002;(2):CD001929.
20 Ernst E. Massage therapy for low back pain: a systematic review. *J Pain Symptom Manage* 1999;17:65-9.
21 Cherkin D, Eisenberg D, Sherman K, Barlow W, Kaptchuk T, Street J, et al. Randomized trial comparing traditional Chinese medical acupuncture, therapeutic massage, and self-care education for chronic low back pain. *Arch Intern Med* 2001;161:1081-8.

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Self reported receipt of care consistent with 32 quality indicators: national population survey of adults aged 50 or more in England

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ABSTRACT

Objective To assess the receipt of effective healthcare interventions in England by adults aged 50 or more with serious health conditions.
Design National structured survey questionnaire with face to face interviews covering medical panel endorsed quality of care indicators for both publicly and privately provided care.
Setting Private households across England.
Participants 8688 participants in the English longitudinal study of ageing, of whom 4417 reported diagnoses of one or more of 13 conditions.
Main outcome measures Percentage of indicated interventions received by eligible participants for 32 clinical indicators and seven questions on patient centred care, and aggregate scores.
Results Participants were eligible for 19 082 items of indicated care. Receipt of indicated care varied substantially by condition. The percentage of indicated care received by eligible participants was highest for ischaemic heart disease (83%, 95% confidence interval 80% to 86%), followed by hearing problems (79%, 77% to 81%), pain management (78%, 73% to 83%), diabetes (74%, 72% to 76%), smoking cessation (74%, 71% to 76%), hypertension (72%, 69% to 76%), stroke (65%, 54% to 76%), depression (64%, 57% to 70%), patient centred care (58%, 57% to 60%), poor vision (58%, 54% to 63%), osteoporosis (53%, 49% to 57%), urinary incontinence (51%, 47% to 54%), falls management (44%, 37% to 51%), osteoarthritis (29%, 26% to 32%), and overall (62%, 62% to 63%). Substantially more indicated care was received for general medical (74%, 73% to 76%) than for geriatric conditions (57%, 55% to 58%), and for conditions included in the general practice pay for performance contract (75%, 73% to 76%) than excluded from it (58%, 56% to 59%).
Conclusions Shortfalls in receipt of basic recommended care by adults aged 50 or more with common health conditions in England were most noticeable in areas associated with disability and frailty, but few areas were

exempt. Efforts to improve care have substantial scope to achieve better health outcomes and particularly need to include chronic conditions that affect quality of life of older people.

INTRODUCTION

Data on the quality of primary care in the United Kingdom have been reported since 2004 through a scheme to pay general practices according to their performance on the basis of quality indicators in several chronic conditions.[1 2] We assessed the delivery across England of effective healthcare interventions for a wide range of conditions to those aged 50 or more, in whom most common major health conditions occur.

METHODS

We collected information on quality of care from the 2004-5 wave of the English longitudinal study of ageing. The sample, selected to be representative of adults aged 50 or more from private residences in England,[3] was drawn from households that had responded to the health survey for England in 1998, 1999, and 2001. Interviewers collected data in participants' homes.

Questions from the English longitudinal study of ageing on quality of care were derived from quality indicators developed for the study on assessing care of vulnerable elders, to assess quality of care for conditions chosen according to several factors, including the potential for quality improvement.[4] Quality indicators for the conditions that had been assessed in the vulnerable elders study using interviews were rated for validity in England by an expert panel of clinicians, using an adapted method.[5 6]

Thirty two indicators for 13 medical conditions were included in the longitudinal study: stroke, depression, diabetes mellitus, falls management, hearing problems, hypertension, ischaemic heart disease, osteoarthritis, osteoporosis, pain management, smoking cessation, urinary incontinence, and cataract. We assessed quality mainly for incident conditions to reduce recall bias. The exceptions when we used prevalent conditions were diabetes, hearing problems, osteoporosis, cataract, and smoking.

We classified the quality indicators into one of three domains of care: screening and prevention, diagnosis, and treatment and follow-up. Each of the 13 conditions was classified into one of two clinical categories, general medical or geriatric care, and according to whether the condition was included in the UK general practice pay for performance contract 2004-5 (see bmj.com).[1] Conditions classified within geriatric care were falls, osteoarthritis, urinary incontinence, cataract, hearing problems, and osteoporosis. We adapted seven additional questions on the quality of patient centred care from three established survey instruments (see bmj.com).

We adapted survey questions from the vulnerable elders interview for quality indicators that were similar to those of the longitudinal study. Questions aimed to determine whether participants had received indicated care for eligible conditions (see bmj.com).

Statistical analysis

The quality score for each indicator was the number of times the indicator was achieved divided by the number of times it was triggered, expressed as a percentage (we excluded "don't know" responses). The data were weighted for non-response and to adjust for respondents' age and sex distribution to that of the 2001 census for the non-institutionalised population.

RESULTS

Overall, 8688 of 10 770 people (80.7%) interviewed for wave 1 of the English longitudinal study of ageing in 2002 completed an interview during 2004-5. In total,

Table 1 | Achievement rates aggregated by condition, adjusted for weighted data, multistage sampling, and multiple indicators per participant

Condition	No of quality indicators	No of times quality indicators achieved	No of times quality indicators eligible	% Quality indicators achieved (95% CI)
Ischaemic heart disease	5	442	530	83.0 (79.7 to 86.4)
Hearing problems	2	1366	1728	78.9 (76.7 to 81.1)
Pain management	1	209	268	77.7 (72.6 to 82.8)
Diabetes	5	1729	2324	74.1 (72.2 to 76.0)
Smoking	1	976	1318	73.5 (71.1 to 76.0)
Hypertension	1	414	571	72.4 (68.6 to 76.2)
Stroke	1	51	78	64.9 (53.8 to 75.9)
Depression	3	168	264	63.6 (57.3 to 69.8)
Cataract	1	339	594	58.4 (54.3 to 62.6)
Osteoporosis	2	399	742	53.3 (49.3 to 57.2)
Urinary incontinence	4	668	1301	50.7 (47.2 to 54.2)
Falls management	2	124	284	43.5 (36.5 to 50.6)
Osteoarthritis	4	288	993	29.0 (26.0 to 31.9)
Overall clinical care	32	7173	10 995	65.2 (64.2 to 66.2)
Patient centred care	7	4738	8087	58.4 (56.9 to 59.8)
Overall, including patient centred care	39	11 911	19 082	62.3 (61.5 to 63.2)

4417 of the wave 2 participants (50.8%) reported having one or more of 13 conditions (see bmj.com). The median number of conditions per participant was 1 (range 0-7), with a median of 1 (range 0-14) quality indicator per participant. Participants with at least one condition each had a median of 2 (range 1-14) quality indicators.

Some quality indicators applied to incident conditions. Incidence varied from 0.9% reporting stroke to 6.6% reporting a new diagnosis of hypertension since the previous survey wave (see bmj.com). Other indicators applied to prevalent conditions. Prevalence varied from 6.7% for osteoporosis to 15.2% for current smokers.

Indicated care was achieved on 11 911 of 19 082 opportunities for care to be delivered: after adjustment the overall achievement rate was 62.3% (95% confidence interval 61.5% to 63.2%); table 1). The quality of care achieved varied substantially by condition, from 83.0% in ischaemic heart disease to 29.0% in osteoarthritis (table 1). "Don't know" responses were given by either no participants or one participant for just over half of the indicators and by less than 5% of eligible participants for all indicators except two: 15 participants (19%) when asked about antihypertensive drugs for stroke and 47 (7%) when asked about annual measurements for glycated haemoglobin level.

Achievement rates for individual quality indicators also varied within a condition (see example on bmj.com). Achievement was substantially higher for general medical conditions (74.2%) than for geriatric conditions (56.5%) and was higher for conditions included in the general practice contract than excluded (74.6% *v* 57.7%; table 2). Achievement was also substantially higher for screening and preventive care than for treatment and follow-up care (79.8% *v* 64.1%), which in turn was higher than diagnostic care (60.0%).

Three quality indicators for diabetes and three for ischaemic heart disease in the English longitudinal study of ageing were similar to indicators used in the UK general practice contract, and achievement was comparable (see bmj.com).

Table 2 | Achievement rates aggregated by condition category and domain, adjusted for weighted data, multistage sampling, and multiple indicators per participant

Variable	No of quality indicators	No of times quality indicators achieved	No of times quality indicators eligible	% Quality indicators achieved (95% CI)
Condition category (clinical):				
General medical	17	3989	5353	74.2 (72.9 to 75.5)
Geriatric	15	3184	5642	56.5 (54.9 to 58.1)
Condition category (policy):				
Included in contract*	13	3612	4821	74.6 (73.2 to 75.9)
Excluded from contract*	19	3561	6174	57.7 (56.3 to 59.2)
Domain:				
Screening and prevention	5	1099	1377	79.8 (77.5 to 82.2)
Diagnosis	6	1387	2299	60.0 (57.7 to 62.2)
Treatment and follow-up	21	4687	7319	64.1 (62.9 to 65.3)

*UK general practice pay for performance contract 2004-5.[1]

DISCUSSION

Quality of care received by adults aged 50 or more living in private households in England and with a diagnosis of at least one of 13 medical conditions varied substantially by condition. Participants reported better quality of care for general medical conditions than for geriatric conditions and better quality of care for conditions included in the UK general practice pay for performance contract than those excluded.

Strengths of the study include the large number of participants and range of conditions, sampled across England. The English longitudinal study of ageing includes the independent sector as well as the National Health Service, which is important for interventions commonly provided privately.

This study has several limitations. Thirty two quality indicators and seven questions on patient centred care inevitably give an incomplete picture of quality. The quality indicators refer to processes of care rather than to outcomes because of the difficulty attributing individuals' health outcomes to the quality of their care.[7]

Because of the design of the longitudinal study the cumulative proportion of responders from the original population sample decreases with each survey wave, and was below half for the second wave analysed in this paper. The data were weighted to correct for non-response, although the age and sex distributions of the weighted and unweighted samples were similar. Weights cannot account for all differences between responders and non-responders, however, and important differences may exist that we have no information about.

The questions were designed for the assessment of quality at population level and some people might have been classified as eligible for the quality indicator yet were not clinically suitable for the intervention. We attempted to target the questions as accurately as possible to the eligible population. Two related sources of potential inaccuracy result from the survey application of quality indicators. Firstly, the translation of quality indicators into a survey resulted in the text of the survey questions not being identical to the indicator text. Secondly, some minor differences were present between the populations specified by quality indicators and the survey populations who were asked the corresponding questions.

Creating a summary score has benefits and drawbacks.[8] The purpose in this paper of reporting a summary score was to put these results in the context of other papers[2,9,10] that report summary scores using equal weighting per indicator. It is plausible that some indicators are more difficult to achieve than others and that some are more important than others. No single accepted method exists for weighting quality indicators for difficulty or importance, so we weighted all indicators equally. One effect of equal weighting is that conditions affecting more participants than others contributed more to the overall score.

The feasibility of measuring quality of care by self report was one of the selection criteria for conditions and

quality of care questions for the English longitudinal study of ageing. Concordance between survey self reports and medical records is good for diagnoses of some chronic conditions and process of care measures.[11] Previous work has shown that self reports tend to score the same or higher than medical records. Don't know responses were given by more than 5% of respondents for antihypertensive drugs for stroke and annual measurement of glycated haemoglobin levels. The higher rates may mean that these two indicators are less accurate than others.

No one way exists to measure all of quality. Each method measures some portion of quality and has strengths and limitations. In this study a strength was that the English longitudinal study of ageing measured more indicators of quality of care than most UK studies and on a large number of people. The high levels of agreement with similar indicators in the general practice contract 2004-5 provide further validation.

These results are similar to the findings from US studies, which used data extracted from medical records as well as telephone interviews. A US nationally representative survey found that participants received 55% of recommended care in 1998-2000,[10] and the assessing care of vulnerable elders study, also found a summary quality score of 55% for adults aged 65 or more, with worse care for geriatric conditions.[9] The assessing care of vulnerable elders study differed from the English longitudinal study of ageing in that its population was vulnerable elders and the number of quality indicators surveyed was higher. The variation in quality between indicators is similar to that found in US studies.[9 10 12] Similarities suggest that compromised quality of care is a property of health systems inter nationally.[13]

Implications

The shortfall in achievement of quality indicators reported here has serious implications. For example, a study determined that if those at high risk of coronary heart disease received better advice on lifestyle, an estimated 4410 events might be prevented.[14]

Quality for geriatric conditions was relatively poor and none were included in the general practice contract. Inclusion of geriatric conditions in future payment for performance schemes might improve quality. The general medical conditions included stroke, hypertension, diabetes, and ischaemic heart disease, all of which have been subject to major national guidelines such as national service frameworks, and have been the focus of quality improvement activities in primary care trusts.[15] Arguably barriers to implementing evidence based practice for geriatric conditions are greater, than for the high mortality conditions that are the focus of much medical practice,[16 17] and the clinical skills required for these conditions may be less well taught to doctors.[9]

Conclusion

We found a large gap between recommended care and care that is actually received. In England, performance monitoring through the general practice pay for performance contract has been linked with improved care for included conditions.[18 19] Making information on performance available for a wider range of conditions is an essential component of quality improvement. Including more conditions that affect the quality of life of older people in future revisions of the general practice contract is one way to do this and has the advantage that the infrastructure is already largely in place.

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1 General Practitioners Committee BMA, NHS Confederation. *Investing in general practice: the new general medical services contract*. London: NHS Confederation, 2003.
2 Doran T, Fullwood C, Gravelle H, Reeves D, Kontopantelis E, Hiroeh U, et al. Pay-for-performance programs in family practices in the United Kingdom. *N Engl J Med* 2006;355:375-84.
3 Banks J, Breeze E, Lessof C, Nazroo J. *Retirement, health and relationships of the older population in England: the 2004 English longitudinal study of ageing (wave two)*. London: Institute for Fiscal Studies, 2006.
4 Wenger NS, Shekelle PG, Davidoff F, Mulrow C. Quality indicators for assessing care of vulnerable elders. *Ann Intern Med* (suppl) 2001;135:S641-758.
5 Steel N, Melzer D, Shekelle PG, Wenger NS, Forsyth D, McWilliams BC. Developing quality indicators for older adults: transfer from the USA to the UK is feasible. *Qual Saf Health Care* 2004;13:260-4.
6 Brook RH, Chassin MR, Fink A, Solomon DH, Kosecoff J, Park RE. A method for the detailed assessment of the appropriateness of medical technologies. *Int J Technol Assess Health Care* 1986;2:53-63.
7 Lilford RJ, Brown CA, Nicholl J. Use of process measures to monitor the quality of clinical practice. *BMJ* 2007;335:648-50.
8 Reeves D, Campbell SM, Adams J, Shekelle PG, Kontopantelis E, Roland MO. Combining multiple indicators of clinical quality: an evaluation of different analytic approaches. *Med Care* 2007;45:489-96.
9 Wenger NS, Solomon DH, Roth CP, MacLean CH, Saliba D, Kamberg CJ, et al. The quality of medical care provided to vulnerable community-dwelling older patients. *Ann Intern Med* 2003;139:740-7.
10 McGlynn EA, Asch SM, Adams J, Keesey J, Hicks J, DeCristofaro A, et al. The quality of health care delivered to adults in the United States. *N Engl J Med* 2003;348:2635-45.

WHAT IS ALREADY KNOWN ON THIS TOPIC

Studies of single conditions have suggested shortfalls in quality of care in England

Adults in the United States might receive only about 55% of recommended care

WHAT THIS STUDY ADDS

Quality of health care for adults aged 50 or more with common health conditions in England varied substantially by condition

Quality scores ranged from 83% for ischaemic heart disease to 29% for osteoarthritis

The greatest scope for improvement is in chronic conditions that affect the quality of life of older people

11 Tisnado DM, Adams JL, Liu H, Damberg CL, Chen WP, Hu FA, et al. What is the concordance between the medical record and patient self-report as data sources for ambulatory care? *Med Care* 2006;44:132-40.
12 Jha AK, Perlin JB, Kizer KW, Dudley RA. Effect of the transformation of the veterans affairs health care system on the quality of care. *N Engl J Med* 2003;348:2218-27.
13 Institute of Medicine.Committee on Health Care in America. *Crossing the quality chasm: a new health system for the 21st century*. Washington DC: National Academy Press, 2001.
14 Gemmell I, Heller RF, Payne K, Edwards R, Roland M, Durrington P. Potential population impact of the UK government strategy for reducing the burden of coronary heart disease in England: comparing primary and secondary prevention strategies. *Qual Saf Health Care* 2006;15:339-43.
15 Wilkin D, Coleman A, Dowling B, Smith K. *The national tracker survey of primary care groups and trusts 2001/2002: taking responsibility?* Manchester: National Primary Care Research and Development Centre, 2002.
16 Inouye SK, Studenski S, Tinetti ME, Kuchel GA. Geriatric syndromes: clinical research, and policy implications of a core geriatric concept. *J Am Geriatr Soc* 2007;55:780-91.
17 Tinetti ME, Gordon C, Sogolow E, Lapin P, Bradley EH. Fall-risk evaluation and management: challenges in adopting geriatric care practices. *Gerontologist* 2006;46:717-25.
18 Campbell S, Reeves D, Kontopantelis E, Middleton E, Sibbald B, Roland M. Quality of primary care in England with the introduction of pay for performance. *N Engl J Med* 2007;357:181-90.
19 Steel N, Maisey S, Clark A, Fleetcroft R, Howe A. Quality of clinical primary care and targeted incentive payments: an observational study. *Br J Gen Pract* 2007;57:449-54.

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Effect of integration of supplemental nutrition with public health programmes in pregnancy and early childhood on cardiovascular risk in rural Indian adolescents: long term follow-up of Hyderabad nutrition trial

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ABSTRACT

Objective To determine whether integration of nutritional supplementation with other public health programmes in early life reduces the risk of cardiovascular disease in undernourished populations.

Design Approximately 15 years' follow-up of participants born within an earlier controlled, community trial of nutritional supplementation integrated with other public health programmes.

Setting 29 villages (15 intervention, 14 control) near Hyderabad city, south India.

Participants 1165 adolescents aged 13-18 years.

Intervention Balanced protein-calorie supplementation (2.51 MJ, 20 g protein) offered daily to pregnant women and preschool children aged under 6 years, coupled with integrated delivery of vertical public health programmes.

Main outcome measures Height, adiposity, blood pressures, lipids, insulin resistance (homoeostasis model assessment (HOMA) score), and arterial stiffness (augmentation index).

Results The participants from the intervention villages were 14 mm (95% confidence interval 4 to 23; P=0.007) taller than controls but had similar body composition. The participants from the intervention villages had more favourable measures of insulin resistance and arterial stiffness: 20% (3% to 39%; P=0.02) lower HOMA score and 3.3% (1% to 5.7%; P=0.008) lower augmentation index. No strong evidence existed for differences in blood pressures and serum lipids.

Conclusions In this undernourished population, integrated delivery of supplemental nutrition with other public health programmes in pregnancy and early childhood was associated with a more favourable profile of cardiovascular disease risk factors in adolescence. This pragmatic study provides the most robust evidence to date on this important hypothesis for which classic trials are unlikely. Improved maternal and child nutrition may have a role in reducing the burden of cardiovascular disease in low income and middle income countries.

INTRODUCTION

The proposition that inadequate diet in early life may result in heightened sensitivity to lifestyle related risk factors is of some importance to the unfolding cardiovascular disease epidemic in low income and middle income countries, where undernutrition and urbanisation now often coexist.[1] The evidence in support of this hypothesis is largely circumstantial.[2-5] Direct evidence of a relation between inadequate diet in early life and later risk of cardiovascular disease is almost non-existent. We examined the prevalence of risk factors for cardiovascular disease in adolescents born within an earlier community trial of nutritional supplementation offered to pregnant women and young children.[6]

METHODS

Integrated Child Development Services is a national community based programme aimed at improving the health, nutrition, and development of children in India.[7,8] The centrepiece of this programme is the provision of free food: a cereal based meal prepared variably from locally available ingredients for pregnant/lactating women and children up to 6 years. The supplement has to be collected daily by the woman (or her children) from the Integrated Child Development Services centre (run by a local woman trained for this

programme), but they are not obliged to eat it there. The programme is complemented by health, hygiene, and nutrition education for the mothers and delivery of other national programmes (immunisation, anaemia control, and basic health care) which are available universally.[7-9]

Initial trial design (1987-90)

Using the opportunity afforded by the stepwise expansion of this programme during the 1980s and 1990s, the National Institute of Nutrition in India ran a trial to assess the impact of food supplementation in pregnancy on the birth weight of offspring. A cluster of villages with a total population of 30 000 was chosen from each of the two adjacent administrative areas, one of which already had the Integrated Child Development Services programme in place (intervention arm), whereas the other was awaiting implementation (control arm). Villages were chosen geographically for random selection and 15 villages from the intervention arm and 14 villages from the control arm were recruited to the study.

A 12 member team of investigators resided full time in the field for the duration of the study. The trial included all births in the area between 1 January 1987 and 31 December 1990. The field team collected data on the outcome of the pregnancy and weighed the newborn within 48 hours.

The supplement given in this trial was "upma," a local preparation made from corn-soya blend (120 g) and soybean oil (16 g), providing 2.51 MJ and 20 g protein to the women and half this amount to the children. The other universal programmes—immunisation, anaemia control in pregnancy through distribution of iron and folic acid tablets, and the provision of basic health care—existed to a similar extent in both the intervention and control areas, although their uptake may be presumed to have been higher in the intervention area.

Follow-up survey (2003-5)

We designed this follow-up study to establish the status of women and their offspring who took part in the trial and to clinically examine offspring who were still resident in the area. We did two parallel surveys on a village to village basis—the first to identify the study participants and the second to do clinical examinations. In the identification survey, which preceded the clinical survey by three months, we first identified women who took part in the baseline trial and interviewed them to identify all children born during the initial trial period (1987-90). We invited only those children who could be successfully matched to the previous records to have clinical examinations.

Measurements

For the clinical survey we measured height, weight, skinfold thickness at four sites (biceps, triceps, subscapular, and suprailiac), and blood pressure. We measured the augmentation index (a measure of global arterial stiffness) over the radial artery. The characteristic pressure waveform produced by blood flow in the arteries changes as the arteries get stiffer with age or under conditions that lead to their premature stiffening (such as atherosclerosis).[10] The augmentation index (difference between the first and second peaks of the

Multivariable association* between supplemental nutrition and cardiovascular disease risk factors at follow-up in Hyderabad nutrition trial

	β coefficient (95% CI); P value			
Measure	Model 1†	Model 2‡	Model 3§	Model 4¶
Height (mm)	−14.1 (−23.3 to −4.9); 0.004	−13.6 (−23.1 to −4.1); 0.007	NA	NA
Fat mass index (kg/m^2)	0.01 (−0.15 to 0.16); 0.9	0.04 (−0.10 to 0.18); 0.5	0.06 (−0.07 to 0.19); 0.4	NA
Fat-free mass index (kg/m^2)	0.23 (0.03 to 0.43); 0.029	0.04 (−0.10 to 0.18); 0.5	0.28 (0.11 to 0.46); 0.003	NA
Central-peripheral skinfold ratio	−0.02 (−0.06 to 0.03); 0.5	−0.02 (−0.08 to 0.03); 0.4	−0.01 (−0.07 to 0.05); 0.7	NA
Systolic blood pressure (mm Hg)	0.86 (−0.72 to 2.45); 0.2	0.59 (−1.11 to 2.29); 0.4	1.10 (−0.68 to 2.87); 0.2	0.64 (−0.99 to 2.27); 0.4
Diastolic blood pressure (mm Hg)	0.21 (−0.81 to 1.23); 0.7	0.08 (−0.90 to 1.07); 0.8	0.21 (−0.77 to 1.19); 0.6	0.11 (−0.85 to 1.07); 0.8
Augmentation index (%)	3.29 (0.74 to 5.84); 0.013	3.30 (0.96 to 5.65); 0.008	2.84 (0.39 to 5.30); 0.025	2.96 (0.55 to 5.38); 0.018
Total cholesterol (mmol/l)	0.01 (−0.11 to 0.12); 0.9	−0.02 (−0.11 to 0.08); 0.7	−0.02 (−0.12 to 0.07); 0.6	−0.03 (−0.13 to 0.06); 0.5
LDL cholesterol (mmol/l)	−0.01 (−0.12 to 0.09); 0.8	−0.03 (−0.13 to 0.07); 0.6	−0.03 (−0.13 to 0.07); 0.5	−0.04 (−0.14 to 0.06); 0.4
HDL cholesterol (mmol/l)	0.01 (−0.05 to 0.06); 0.7	−0.00 (−0.06 to 0.05); 1.0	−0.00 (−0.06 to 0.05); 0.9	−0.00 (−0.06 to 0.05); 0.9
Triglycerides (mmol/l)**	0.02 (−0.03 to 0.07); 0.4	0.03 (−0.03 to 0.08); 0.3	0.02 (−0.03 to 0.08); 0.4	0.02 (−0.03 to 0.07); 0.4
Glucose (mmol/l)	0.04 (−0.22 to 0.29); 0.8	0.03 (−0.21 to 0.26); 0.8	0.03 (−0.21 to 0.26); 0.8	0.04 (−0.20 to 0.28); 0.8
Insulin (mU/l)**	0.18 (0.04 to 0.32); 0.016	0.18 (0.03 to 0.34); 0.02	0.19 (0.04 to 0.35); 0.016	0.19 (0.03 to 0.35); 0.016
HOMA score**	0.18 (0.04 to 0.32); 0.014	0.18 (0.03 to 0.33); 0.021	0.19 (0.04 to 0.35); 0.016	0.19 (0.03 to 0.34); 0.02

HDL=high density lipoprotein; HOMA=homoeostasis model assessment; LDL=low density lipoprotein; NA=not applicable.
Sample size: n=1120 for height, fat mass index, fat-free mass index, and central-peripheral skinfold ratio; n=1118 for systolic and diastolic blood pressure; n=862 for augmentation index; n=1050 for total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides; n=1008 for glucose and insulin; n=1003 for HOMA score.
*Linear regression models with robust standard errors.
†Adjusted for age, sex, pubertal stage (early puberty, middle puberty, late puberty, and post-puberty), room temperature (blood pressure only), and heart rate (augmentation index only).
‡Adjusted for variables in model 1 plus current socioeconomic circumstances (household's standard of living index (high, medium, low) and village urbanisation (village population <2000, 2000-5000, >5000)).
§Adjusted for variables in model 2 plus height (mm).
¶Adjusted for variables in model 3 plus body composition (fat mass index, fat free mass index, and central-peripheral skinfold ratio).
**Differences (between means) are on log scale.

central arterial waveform, expressed as a percentage of pulse pressure) is negative in healthy young adults but becomes increasingly positive as arteries stiffen. We classified sexual maturation into four stages, measured socioeconomic position with the standard of living index used in India, and assessed urbanisation of the villages by population size (people). We collected fasting blood samples (at least eight hours) and did assays for glucose, triglycerides, total cholesterol, and high density lipoprotein cholesterol on the same day and estimated insulin concentrations within four to six weeks. We produced detailed protocols and used them regularly to standardise the work and ensure quality control of the fieldwork team (see bmj.com).

Statistical analyses

We used the log of the sum of four skinfolds to calculate the percentage of body fat and then converted this into fat mass index (kg/m^2) and fat-free mass (kg/m^2) by using body weight and height measurements. We assessed central adiposity by the ratio of central (subscapular plus suprailiac) to peripheral (biceps plus triceps) skinfolds. We estimated low density lipoprotein cholesterol from triglycerides, total cholesterol, and high density lipoprotein cholesterol, and calculated insulin resistance by homoeostasis model assessment (HOMA), excluding those with fasting glucose ≥7 mmol/l.[11]

We used linear regression models to investigate association of supplemental nutrition with cardiovascular disease risk factors. We used area of birth as proxy (irrespective of whether the participant took the supplement or not). We fitted four predefined models to adjust incrementally for the main domains of potential confounding or intermediary variables (see table and bmj.com for details of modelling.)

We examined interaction between the intervention and the sex of the participant, as evidence exists of preferential feeding of male children in this setting, as well as possible sex differential effects of early undernutrition on risk of cardiovascular disease. Sample size calculations done before the start of the study suggested that the anticipated sample (estimated as 1268 overall) was adequate to detect important differences in most outcomes.

RESULTS

Of the 4338 pregnancies recorded in the trial, birth weights were available for 2964 (68%) children. The mean birth weight of children born in the intervention area (2655 (SD 424) g) was higher than that of controls (2594 (SD 430) g); the mean difference was 61 g (95% confidence interval 18 to 104; P=0.007). From the identification survey, 2601 children were eligible for follow-up (born between 1987 and 1990 and still alive in 2003). From these we invited only those with existing information in the trial dataset (n=1492; 57%) to have a clinical examination. A total of 1165 children participated in the clinics: 654 (82%) in the intervention area and 511 (74%) in the control area, representing 45% of all eligible births from the area at the time. Children who took part in the clinics were slightly older and more likely to be males and students than those who were eligible but did not participate (see bmj.com). Complete data were available for 1120 (96%) children for at least one outcome, with slightly fewer for blood pressure (1118; 96%), lipid profile (1050; 90%), glucose-insulin (1008; 87%), and arterial stiffness (862; 74%).

The distributions of key exposures were largely similar across the two areas. The distribution of the standard of living index was consistent with the peri-urban situation of these villages. Only two children reported smoking tobacco or consuming alcohol, and all except two were breast fed, so we did not consider these variables in further analyses. The main outcome measures were body size and composition, cardiovascular physiology, lipid profile, and glucose homoeostasis. Body composition data confirm the short, lean habitus of this population. The mean values for indices of insulin resistance (insulin and HOMA score) and, to a smaller extent, arterial stiffness (although data are limited) were higher than those generally reported from high income countries, suggesting a pro-atherogenic profile in this undernourished population. Village level clustering of outcome measures was less than 0.1, except for fasting blood glucose (0.14). All results are based on models with robust standard errors, with village as the level of cluster.

Children in the intervention and control arms were not different in their sexual maturation. The table shows the results of multivariably adjusted associations between supplemental nutrition and risk factors for cardiovascular disease. Children in the intervention area were on average 14 (95% confidence interval 4 to 23) mm taller than the control children but similar in body composition. The augmentation index showed an association with supplemental nutrition. Children in the control arm had higher insulin concentration and HOMA score, robust to adjustments for height and body composition (identical results for insulin and HOMA due to high correlation). The regression coefficients shown for insulin and HOMA score are differences between the means on the log scale; the HOMA score value for model 2 equates to 20% (95% confidence interval 3% to 39%) higher values in the control children (on the original scale).

DISCUSSION

This is the first intervention study to show that modest improvements in the protein-calorie intake of pregnant women and young children may result in a more favourable cardiovascular disease risk factor profile among populations with prevalent undernutrition. These findings may indicate potential life course pathways underlying the causes of cardiovascular diseases in general.[12] These observations, if replicated, have important implications for the primary prevention of cardiovascular diseases in low income and middle income countries, as the intervention tested was pragmatic, cheap, and relatively easy to implement.

WHAT IS ALREADY KNOWN ON THIS TOPIC

Observational studies suggest that early undernutrition predisposes to cardiovascular disease in later life, but robust evidence from intervention trials is lacking

WHAT THIS STUDY ADDS

Integration of supplemental nutrition with public health programmes in pregnancy and early childhood was associated with a reduction in cardiovascular risk

Improved maternal and child nutrition may have a role in reducing the burden of cardiovascular disease in low income and middle income countries

Comparison with previous research

The importance of balanced protein-calorie malnutrition in early life has been studied in two natural experiments based on the starvation experience of populations in the second world war (Dutch and Leningrad studies) and one small randomised controlled trial of supplemental nutrition from Guatemala.[13-16] Whereas the Dutch study reported associations with adiposity and dyslipidaemia (early gestation starvation) and with abnormal glucose homoeostasis (late gestation starvation), these findings were not replicated in the Leningrad study.[14 15] The Guatemala trial, at follow-up at age 24 years, found no association of supplement type with adiposity or blood pressure but an association of higher energy supplement with lower fasting glucose in women only. One potential reason for the negative findings could be that these studies were underpowered, or the exposure may have been inadequate for programming of long term physiological changes.

Strengths and limitations

The main strength of this study is its setting: a nutritional intervention in a population with high background levels of chronic undernutrition, so we could realistically expect to find programming effects of inadequate diet, if they exist. The controlled design of the original evaluation would have reduced the chances of confounding; furthermore, the crucial confounders were either completely absent (maternal smoking) or severely restricted (socioeconomic heterogeneity and urbanisation). The age group (adolescence) was also ideal for studying exposures in early life, as the participants were old enough to show variations in risk factors but young enough that any associations seen were not distorted by any differences in adult lifestyle.

The study also has some important limitations, chief among them the potential for bias owing to non-randomisation of villages in the baseline study, losses to follow-up, and lack of data on current diet and patterns of physical activity (see bmj.com). Finally, despite the relative automation of most of the outcome measures, the possibility of bias arising from the lack of blinding of the fieldworkers cannot be ruled out.

Potential mechanisms

Insulin resistance is believed to be central to many of the changes attributed to the thrifty phenotype.[4 17] If the fetus develops insulin resistance in times of undernutrition, this may improve the short term chances of survival but predispose to cardiovascular disease on persistence into adult life. Arterial stiffness is a relatively novel risk factor for atherosclerosis.[18] Whether it is a risk marker for atherosclerosis or an independent risk factor for cardiovascular disease is unclear. Insulin is a risk factor for arterial stiffness and may be the link between early undernutrition, arterial stiffness, and hypertension.[19 20]

The relative importance of nutrition in various stages, sources of nutrition, or indeed the rate of growth in early versus late postnatal life cannot be delineated from this study. The other public health programmes were available in both intervention and control areas but may have had greater uptake in the intervention area owing to their integration with the nutritional component. Their contribution to cardiovascular disease risk, if any, is more likely to have been mediated through improvements in the nutritional status.

Public health implications

The Anglo-Cardiff collaborative study suggested that a 3% lower augmentation index equates to a three year less aged vascular phenotype.[21] However, extreme caution in interpretation is warranted because of differences in study setting and design. Similarly, extrapolating the data from a meta-analysis of studies on insulin concentrations and risk of cardiovascular disease also suggests a potential 8% reduction in cardiovascular disease risk of the supplemented children.[22] Although these improvements are substantial in themselves, even more important is the potential for these risk factors to interact with the behavioural risk factors in adulthood, resulting in a major amplification in the risk difference.[2-23]

This study provides realistic estimates of the benefit that can be expected from supplemental nutrition given in the context of "holistic" programmes to tackle undernutrition in children. Such programmes already exist around much of the developing world but are being questioned because of the epidemics of obesity and cardiovascular disease.[24 25] Our results provide some reassurance about the longer term benefits of improving maternal and childhood nutrition in undernourished populations. At this stage in the participants' life course, this intervention does not seem to increase the risk of obesity and may even decrease the future risk of cardiovascular disease.

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1 Fall CH. Non-industrialised countries and affluence. *Br Med Bull* 2001;60:33-50.
2 Barker DJP. *Mothers, babies and health in later life*. Edinburgh: Churchill Livingstone, 1998.
3 Godfrey KM, Barker DJ. Fetal nutrition and adult disease. *Am J Clin Nutr* 2000;71:1344-52S.
4 Armitage JA, Khan IY, Taylor PD, Nathanielsz PW, Poston L. Developmental programming of the metabolic syndrome by maternal nutritional imbalance: how strong is the evidence from experimental models in mammals? *J Physiol* 2004;561:355-77.
5 Waterland RA, Garza C. Potential mechanisms of metabolic imprinting that lead to chronic disease. *Am J Clin Nutr* 1999;69:179-97.
6 The Director. *Annual report 1990-91*. Hyderabad: National Institute of Nutrition, 1991:8-9.
7 Kapil U, Tandon BN. ICDS scheme—current status, monitoring, research and evaluation system. *Indian J Public Health* 1990;34:41-7.
8 Tandon BN. A coordinated approach to children's health in India. *Lancet* 1981;1:650-3.
9 Tandon BN. Nutritional interventions through primary health care: impact of the ICDS projects in India. *Bull World Health Organ* 1989;67:77-80.
10 Oliver JJ, Webb DJ. Noninvasive assessment of arterial stiffness and risk of atherosclerotic events. *Arterioscler Thromb Vasc Biol* 2003;23:554-66.
11 Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia* 1985;28:412-9.
12 Ben-Shlomo Y, Kuh D. A life course approach to chronic disease epidemiology: conceptual models, empirical challenges and interdisciplinary perspectives. *Int J Epidemiol* 2002;31:285-93.
13 Conlisk AJ, Barnhart HX, Martorell R, Grajeda R, Stein AD. Maternal and child nutritional supplementation are inversely associated with fasting plasma glucose concentration in young Guatemalan adults. *J Nutr* 2004;134:890-7.
14 Painter RC, Roseboom TJ, Bleker OP. Prenatal exposure to the Dutch famine and disease in later life: an overview. *Reprod Toxicol* 2005;20:345-52.
15 Stanner SA, Yudkin JS. Fetal programming and the Leningrad siege study. *Twin Res* 2001;4:287-92.
16 Webb AL, Conlisk AJ, Barnhart HX, Martorell R, Grajeda R, Stein AD. Maternal and childhood nutrition and later blood pressure levels in young Guatemalan adults. *Int J Epidemiol* 2005;34:898-904.
17 Hales CN, Barker DJ. The thrifty phenotype hypothesis. *Br Med Bull* 2001;60:5-20.
18 Arnett DK, Evans GW, Riley WA. Arterial stiffness: a new cardiovascular risk factor? *Am J Epidemiol* 1994;140:669-82.
19 Cockcroft JR, Webb DJ, Wilkinson IB. Arterial stiffness, hypertension and diabetes mellitus. *J Hum Hypertens* 2000;14:377-80.
20 Westerbacka J, Yki-Jarvinen H. Arterial stiffness and insulin resistance. *Semin Vasc Med* 2002;2:157-64.
21 McEniery CM, Yasmin R, Hall IR, Qasem A, Wilkinson IB, Cockcroft JR. Normal vascular aging: differential effects on wave reflection and aortic pulse wave velocity: the Anglo-Cardiff collaborative trial (ACCT). *J Am Coll Cardiol* 2005;46:1753-60.
22 Ruige JB, Assendelft WJ, Dekker JM, Kostense PJ, Heine RJ, Bouter LM. Insulin and risk of cardiovascular disease: a meta-analysis. *Circulation* 1998;97:996-1001.
23 Bateson P, Barker D, Clutton-Brock T, Deb D, D'Udine B, Foley RA, et al. Developmental plasticity and human health. *Nature* 2004;430:419-21.
24 Bhutta ZA, Darmstadt GL, Hasan BS, Haws RA. Community-based interventions for improving perinatal and neonatal health outcomes in developing countries: a review of the evidence. *Pediatrics* 2005;115:519-617.
25 Oken E, Gillman MW. Fetal origins of obesity. *Obes Res* 2003;11:496-506.

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Physical control and coordination in childhood and adult obesity: longitudinal birth cohort study

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ABSTRACT

Objective To identify whether measures of childhood physical control and coordination as markers of neurological function are associated with obesity in adults.

Design Longitudinal birth cohort study.

Setting National child development study in Great Britain.

Participants 11 042 people born during one week in 1958.

Main outcome measure Obesity at age 33 years defined as body mass index ≥30.

Results Among 7990 cohort members at age 7 years, teachers reported that poor hand control, poor coordination, and clumsiness "certainly applied" more often among those who would be obese adults, producing adjusted odds ratios of 1.57 (95% confidence interval 1.13 to 2.20; P=0.008) for poor hand control, 2.30 (1.52 to 3.46; P<0.001) for poor coordination, and 3.91 (2.61 to 5.87; P<0.001) for clumsiness. Among 6875 participants who had doctor administered assessments with continuous scores at age 11 years, poorer function was associated with later obesity, indicated by adjusted odds ratios (change in risk per unit increase in score) of 0.88 (0.81 to 0.96; P=0.003) for copying designs, 0.84 (0.78 to 0.91; P<0.001) for marking squares, and 1.14 (1.06 to 1.24; P<0.001) for picking up matches (a higher score indicates poor function in this test). Further adjustment for contemporaneous body mass index at age 7 or 11 years did not eliminate statistical significance for any of the associations.

Conclusion Some aspects of poorer neurological function associated with adult obesity may have their origins in childhood.

INTRODUCTION

Obesity in adults is associated with cognitive impairment and dementia,[1-3] and this is assumed to be a consequence of obesity or obesity related processes such as secretion of bioactive hormonal compounds, altered insulin signalling, diabetes, hypertension, and other cardiovascular disease processes.[4-9] However, some relevant processes begin in earlier life, potentially with different mechanisms.

Obese adults and those with adult onset type 2 diabetes mellitus may already have lower levels of cognitive function in childhood, consistent with a subtle developmental impairment.[10 11] Therefore, pathways to impaired cognitive function in these groups may begin much earlier in life than was previously thought, reflecting early biological processes relevant to neurological function and cognition. An alternative explanation is that tests of cognitive function are influenced directly by social and cultural factors,[12 13] rather than indicating a neurological mechanism, and these social influences also increase future risk of obesity.

We investigated whether tests of physical control and coordination in children are associated with obesity in adults, by using measures less subject to confounding by social factors than many tests of cognition.

METHODS

The national child development study is following everyone born between 3 and 9 March 1958 and living in Great Britain, with data collection sweeps at various ages throughout childhood and in adult life.[14] A total of 11 042 people had information on body mass index at age 33; among these, 7990 also had information for assessments and potential confounding factors at age 7 and 6875 had such information at age 11 and were free from major disabilities and in mainstream schools.

At age 7, classroom teachers (who also supervised physical education classes) were asked to identify poor ability in hand control, coordination, and overall clumsiness, scored as certainly, somewhat, no, and unsure. The Bristol social adjustment guide is a test in which class teachers recorded descriptions of behaviour that applied to the child among 150 items.[15] A local authority medical officer measured weight and height and recorded information on mental retardation and all significant disabilities and chronic illnesses.

At age 11 years, local authority medical officers measured height and weight, as well as administering a series of functional assessments, of which we selected three for these analyses as indicating hand control and coordination: copying a simple design (accuracy scored from 0 to 12); marking squares on paper within one minute (maximum 200); time in seconds that it took to pick up 20 matches (maximum 99 s). General ability (cognitive function) tests with a range of 0-76 were administered at school.[16]

At age 33, trained interviewers measured height and weight (the latest measured rather than self reported records). Obesity was defined as body mass index (weight (kg)/(height (m)^2) of 30 or over.

Statistical analysis

We used logistic regression to estimate associations with obesity at age 33 as the dependent variables in six

Table 1 | Teachers' assessments at age 7 years and obesity at age 33 years

	Obesity (%) at age 33 years		Unadjusted		Adjusted*		Adjusted†	
Assessments	No (n=7078)	Yes (n=912)	Odds ratio (95% CI)	P value	Odds ratio (95% CI)	P value	Odds ratio (95% CI)	P value
Poor hand control:								
Certainly	244 (3.4)	52 (5.7)	1.83 (1.34 to 2.49)	<0.001	1.57 (1.13 to 2.20)	0.008	1.48 (1.04 to 2.10)	0.030
Somewhat	998 (14.1)	177 (19.4)	1.52 (1.27 to 1.82)	<0.001	1.38 (1.14 to 1.67)	0.001	1.33 (1.09 to 1.63)	0.005
No	5824 (82.3)	679 (74.5)	Reference		Reference		Reference	
Unsure	12 (0.2)	4 (0.4)	2.86 (0.92 to 8.89)	0.070	2.69 (0.85 to 8.48)	0.091	1.99 (0.60 to 6.58)	0.275
Poor coordination:								
Certainly	123 (1.7)	38 (4.2)	2.58 (1.78 to 3.74)	<0.001	2.30 (1.52 to 3.46)	<0.001	1.57 (1.01 to 2.45)	0.047
Somewhat	671 (9.5)	118 (12.9)	1.47 (1.19 to 1.81)	<0.001	1.37 (1.10 to 1.71)	0.005	1.16 (0.92 to 1.47)	0.223
No	6249 (88.3)	748 (82.0)	Reference		Reference		Reference	
Unsure	35 (0.5)	8 (0.9)	1.91 (0.88 to 4.13)	0.100	1.75 (0.80 to 3.83)	0.159	1.50 (0.67 to 3.38)	0.329
Clumsy:								
Certainly	95 (1.3)	43 (4.7)	4.05 (2.80 to 5.86)	<0.001	3.91 (2.61 to 5.87)	<0.001	2.04 (1.31 to 3.20)	0.002
Somewhat	659 (9.3)	162 (17.8)	2.20 (1.82 to 2.66)	<0.001	2.24 (1.82 to 2.76)	<0.001	1.51 (1.21 to 1.89)	<0.001
No	6291 (88.9)	703 (77.1)	Reference		Reference		Reference	
Unsure	33 (0.5)	4 (0.4)	1.09 (0.38 to 3.07)	0.878	1.04 (0.36 to 2.97)	0.947	1.00 (0.34 to 2.93)	0.996

*Adjusted for social class, sex, mother's age, birth weight standardised for gestational age, ethnic origin, maternal smoking during pregnancy, Bristol social adjustment guide score, and four chronic disease variables associated with either later obesity or motor function (general motor disability, mental retardation, epilepsy, and other central nervous system conditions).
†Adjusted for all above measures and also for body mass index at age 7.

Table 2 | Assessments done during medical examination at age 11 years and risk of obesity at age 33 years

	Unadjusted		Adjusted*		Adjusted†	
Assessment	Odds ratio (95% CI)	P value	Odds ratio (95% CI)	P value	Odds ratio (95% CI)	P value
Copying designs	0.84 (0.77 to 0.91)	<0.001	0.88 (0.81 to 0.96)	0.003	0.88 (0.81 to 0.97)	0.006
Marking squares	0.83 (0.77 to 0.90)	<0.001	0.84 (0.78 to 0.91)	<0.001	0.82 (0.75 to 0.89)	<0.001
Picking up matches	1.17 (1.08 to 1.26)	<0.001	1.14 (1.06 to 1.24)	<0.001	1.09 (1.00 to 1.18)	0.050

Independent measures are continuous test scores; modest magnitude of odds ratios reflects change in obesity risk associated with one unit change in test score.
*Adjusted for social class, sex, mother's age, birth weight standardised for gestational age, ethnic origin, maternal smoking during pregnancy, pubertal development, and four chronic disease variables associated with either later obesity or motor function (general motor disability, mental retardation, epilepsy, and other central nervous system conditions).

separate models for each of the measures of physical control and coordination at ages 7 and 11 years. See bmj.com for a full description of the methods.

RESULTS

Lower social class was associated with increased risk of obesity at age 33. Table 1 shows the associations of teacher assessed hand control, coordination, and clumsiness at age 7 with obesity at age 33. All three measures showed a graded and statistically significant association of poorer control and coordination with obesity at age 33, and the associations were independent of the markers of childhood circumstances and behaviour. Further adjustment for body mass index at age 7 did not eliminate statistical significance.

Table 2 shows the association of doctor administered tests of hand control and coordination at age 11 with obesity at age 33. The tests scores are continuous measures, so no reference category exists; the odds ratios are modest in magnitude, as they represent the increase in risk of obesity associated with a one unit change in the score. The significantly lower scores (worse performance) for copying designs and marking squares associated with obesity are illustrated by odds ratios below 1.00. In contrast, a higher score in the picking up matches test indicates worse performance (a longer time); this was significantly associated with obesity, indicated by odds ratios above 1.00. Adjustment for childhood characteristics and pubertal development did not notably alter the associations or their statistical significance. Further adjustment for body mass index at age 11 did not eliminate statistical significance.

DISCUSSION

All the investigated measures of physical control and coordination at ages 7 and 11 were associated with obesity in adults, independent of multiple markers of social and material conditions in childhood. Even adjustment for contemporaneous childhood body mass index did little to alter the associations, as it was not strongly associated with the tests, except picking up matches. This indicates that childhood body mass is not responsible for the differences in function seen. To ensure that this was not due to poor estimation of childhood body mass index, we also modelled an alternative estimate of childhood body mass index that may be a better indicator of childhood body mass.[17] Arguably, including childhood body mass index is an over-adjustment that may mask important associations, as many (although far from all) obese adults tend to be heavier children.[18] Some early life exposures or characteristics associated with obesity in adults may influence development of physical control and coordination long before the onset of obesity.

Use of tests of motor competence

A disadvantage of this study is that it could not identify specific measures of neurological function; instead it used markers of physical control and coordination likely to be relevant. Unlike earlier studies of cognitive ability,[10 11] the functional assessments used here are less susceptible to confounding by immediate social factors, as poor performance in cognitive function tests may reflect a lack of knowledge and experience rather than indicating true cognition.[12] Thus, the study provides evidence of poorer motor competence indicating neurological function in childhood among those who will be obese adults. Given the social gradient of obesity, social factors are likely to be of primary importance in explaining its causes and also the associated increased risk of poorer physical control and coordination.

Childhood body mass and cognitive function

Inclusion of childhood body mass index and the general (cognitive) ability score in the models clearly represents over-adjustment but also provides useful information. Adjustment for childhood body mass index not only indicates that body mass does not seem to directly influence physical control and coordination but also represents an indirect adjustment for a variety of factors, such as physical activity, that are plausible confounding factors. However, body mass index may be too crude a measure to identify specific types of adipose tissue in childhood that indicate physical activity or produce bioactive compounds.[2 5 19 20] The effect of adjustment for general ability indicates associations with cognitive function in copying designs. Interestingly, associations between obesity in adults

WHAT IS ALREADY KNOWN ON THIS TOPIC

Poorer cognitive function in childhood is associated with obesity and type 2 diabetes in adults

Whether this is mediated through neurological function or confounding by social factors that limit performance in cognitive function tests is unclear

WHAT THIS STUDY ADDS

Poorer physical control and coordination in childhood is associated with obesity in adults

Some aspects of poorer neurological function associated with obesity in adults may have their origins in childhood

and the other tests of physical control and coordination are more independent of cognitive function, so exclusive mediation through persistently lower cognitive ability is unlikely. The varying effects of adjustment for cognitive function and childhood body mass index depending on the test suggest heterogeneity in the childhood characteristics measured by these tests.

Potential limitations and the importance of a life course approach

The reported associations are independent of several well recognised measures of socioeconomic and personal characteristics, as well as additional markers such as birth weight and maternal smoking during pregnancy, which may be relevant to risk of obesity but also signal a variety of associated cultural and economic exposures.[21-23] Many other environmental or individual characteristics could explain the associations. These might be considered as confounding factors and are important targets for future research to assist in our understanding of how risks for obesity and associated complications accumulate across the life course. Clearly, diet and exercise may be important, as continuity in patterns of physical activity are likely to influence physical control and coordination as well as risk of obesity. Exercise may influence physical control and coordination, or people with lower motor competence may be less likely to exercise, thus increasing risk of obesity. Some aspects of personality (possibly also influenced by earlier environmental exposures) could be potentially relevant to behaviour influencing physical control and coordination as well as risk of obesity. Rather than being explained by a single factor, an accumulation throughout life of many associated cultural, personal, and economic exposures is likely to underlie the risks for obesity and some elements of associated neurological function.

Conclusion

This study cannot identify the specific biological processes linking poorer physical control and coordination in childhood with later obesity. However, it suggests that some of the processes associated with poorer neurological function in obese adults have their origins in childhood.

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1 Whitmer RA, Gunderson EP, Barrett-Connor E, Quesenberry CP Jr, Yaffe K. Obesity in middle age and future risk of dementia: a 27 year longitudinal population based study. *BMJ* 2005;330:1360.
2 Knecht S, Ellger T, Levine JA. Obesity in neurobiology. *Prog Neurobiol* 2008;84:85-103.
3 Gustafson D. Adiposity indices and dementia. *Lancet Neurol* 2006;5:713-20.
4 Graf C, Koch B, Kretschmann-Kandel E, Falkowski G, Christ H, Coburger S, et al. Correlation between BMI, leisure habits and motor abilities in childhood (CHILT-project). *Int J Obes Relat Metab Disord* 2004;28:22-6.
5 Yaffe K, Kanaya A, Lindquist K, Simonsick EM, Harris T, Shorr RI, et al. The metabolic syndrome, inflammation, and risk of cognitive decline. *JAMA* 2004;292:2237-42.
6 Craft S. Insulin resistance and Alzheimer's disease pathogenesis: potential mechanisms and implications for treatment. *Curr Alzheimer Res* 2007;4:147-52.
7 Strachan MW, Price JF, Frier BM. Diabetes, cognitive impairment, and dementia. *BMJ* 2008;336:6.
8 Reitz C, Tang MX, Manly J, Mayeux R, Luchsinger JA. Hypertension and the risk of mild cognitive impairment. *Arch Neurol* 2007;64:1734-40.
9 Rosengren A, Skoog I, Gustafson D, Wilhelmsen L. Body mass index, other cardiovascular risk factors, and hospitalization for dementia. *Arch Intern Med* 2005;165:321-6.
10 Chandola T, Deary IJ, Blane D, Batty GD. Childhood IQ in relation to obesity and weight gain in adult life: the national child development (1958) study. *Int J Obes* 2006;30:1422-32.
11 Olsson GM, Hulting AL, Montgomery SM. Cognitive function in children and subsequent type 2 diabetes mellitus. *Diabetes Care* 2008;31:514-6.
12 Jefferis BJ, Power C, Hertzman C. Birth weight, childhood socioeconomic environment, and cognitive development in the 1958 British birth cohort study. *BMJ* 2002;325:305.
13 Tong S, Baghurst P, Vimpani G, McMichael A. Socioeconomic position, maternal IQ, home environment, and cognitive development. *J Pediatr* 2007;151:284-8.
14 Ferri E. *Life at 33: the fifth follow-up of the national child development study*. London: National Children's Bureau, 1993.
15 Stott GH. *The social adjustment of children: manual to the Bristol social adjustment guides*. London: Hodder and Stoughton, 1987.
16 Douglas JWB. *The home and the school*. London: MacGibbon and Kee, 1964.
17 Power C, Lake JK, Cole TJ. Measurement and long-term health risks of child and adolescent fatness. *Int J Obes* 1997;21:507-26.
18 Viner RM, Cole TJ. Adult socioeconomic, education, social and psychological outcomes of childhood obesity: a national birth cohort study. *BMJ* 2005;330:1354-8.
19 Hausman DB, DiGirolamo M, Bartness TJ, Hausman GJ, Martin RJ. The biology of white adipocyte proliferation. *Obes Rev* 2001;2:239-54.
20 Yki-Järvinen H, Westerbacka J. Vascular actions of insulin in obesity. *Int J Obes Relat Metab Disord* 2000;24:S25-8.
21 Montgomery SM, Ekbom A. Smoking during pregnancy and diabetes mellitus in a British longitudinal birth cohort. *BMJ* 2002;324:26-7.
22 Toschke AM, Montgomery SM, Pfeiffer U, von Kries R. Early intrauterine exposure to tobacco-inhaled products and obesity. *Am J Epidemiol* 2003;158:1068-74.
23 Breslau N, Paneth N, Lucia VC, Paneth-Pollak R. Maternal smoking during pregnancy and offspring IQ. *Int J Epidemiol* 2005;34:1047-53.

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