RESEARCH

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Income inequality, mortality, and self rated health: meta-analysis of multilevel studies

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EDITORIAL by Pickett and Wilkinson

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ABSTRACT

Objective To provide quantitative evaluations on the association between income inequality and health.
Design Random effects meta-analyses, calculating the overall relative risk for subsequent mortality among prospective cohort studies and the overall odds ratio for poor self rated health among cross sectional studies.
Data sources PubMed, the ISI Web of Science, and the National Bureau for Economic Research database.
Review methods Peer reviewed papers with multilevel data.
Results The meta-analysis included 59 509 857 subjects in nine cohort studies and 1 280 211 subjects in 19 cross sectional studies. The overall cohort relative risk and cross sectional odds ratio (95% confidence intervals) per 0.05 unit increase in Gini coefficient, a measure of income inequality, was 1.08 (1.06 to 1.10) and 1.04 (1.02 to 1.06), respectively. Meta-regressions showed stronger associations between income inequality and the health outcomes among studies with higher Gini (≥0.3), conducted with data after 1990, with longer duration of follow-up (>7 years), and incorporating time lags between income inequality and outcomes. By contrast, analyses accounting for unmeasured regional characteristics showed a weaker association between income inequality and health.
Conclusions The results suggest a modest adverse effect of income inequality on health, although the population impact might be larger if the association is truly causal. The results also support the threshold effect hypothesis, which posits the existence of a threshold of income inequality beyond which adverse impacts on health begin to emerge. The findings need to be interpreted with caution given the heterogeneity between studies, as well as the attenuation of the risk estimates in analyses that attempted to control for the unmeasured characteristics of areas with high levels of income inequality.

WHAT IS ALREADY KNOWN ON THIS TOPIC

Dozens of studies have examined the association between income inequality and population health, but consensus remains elusive because of inconsistent findings

Researchers have suggested several factors—such as a threshold effect of income inequality on health—that could account for heterogeneity between studies

WHAT THIS STUDY ADDS

Our meta-analysis found that income inequality was associated with a modest excess risk of premature mortality and poor self rated health

The studies reviewed were highly heterogeneous, one potential explanation being the existence of a threshold effect of income inequality (Gini ≥0.3) on population health

If the inequality-mortality relation is truly causal then the population attributable fraction suggests that upwards of 14 million deaths (9.6%) could be averted in 30 OECD countries by levelling the Gini coefficient below the threshold value of 0.3

INTRODUCTION

Empirical studies have attempted to link income inequality with poor health, but recent systematic reviews have failed to reach a consensus because of mixed findings. Many developed countries have experienced a surge in income inequality during the era of globalisation,[1] and if economic inequality is truly damaging to health, then even a "modest" association can amount to a considerable population burden.

Income inequality could damage health through two pathways. Firstly, a highly unequal society implies that a substantial segment of the population is impoverished, and poverty is bad for health. Secondly, income inequality is thought to affect the health of not just the poor, but the better off in society as well. The so called spillover effects of inequality have in turn been attributed to the psychosocial stress resulting from invidious social comparisons,[2 3] as well as the erosion of social cohesion.[4]

We sought to provide quantitative evaluations of the income inequality hypothesis by conducting a meta-analysis of prospective cohort studies and cross sectional studies on the association of income inequality with mortality and self rated health. We also evaluated the potential factors explaining the differences between studies.

METHODS

Study selection

We included cohort studies on the association between income inequality and mortality or cross sectional studies on the association between income inequality and self reported health. To be included studies had to use multilevel data—at least two levels including one or more region variable(s); address sample clustering caused by multilevel data structure; and adjust for age, sex, and individual socioeconomic status.

We searched papers published between January 1995 and July 2008, using PubMed, ISI Web of Science, and the National Bureau of Economic Research database using the following keywords: "inequalit(y/ies)", "income", "Gini", "mortality", "death", and "health".

Data extraction

Two investigators independently extracted information from included studies. We resolved discrepancies

Cohort study	Weight (%)	Relative risk (95% CI)
Denmark, CCHS/CPS 1976-8[w1]		
Male	5.45	1.01 (0.99 to 1.02)
Female	5.17	1.01 (0.98 to 1.03)
Finland, Census 1990[w2]	5.09	1.02 (0.99 to 1.04)
Norway, Census 1980-2002[w3]		
Male age 30-39	5.06	1.17 (1.14 to 1.20)
Male age 40-49	5.28	1.13 (1.10 to 1.15)
Male age 50-59	5.45	1.10 (1.08 to 1.11)
Male age 60-69	5.55	1.07 (1.07 to 1.08)
Male age 70-79	5.58	1.06 (1.05 to 1.06)
Female age 30-39	4.46	1.20 (1.15 to 1.25)
Female age 40-49	5.00	1.16 (1.13 to 1.20)
Female age 50-59	5.30	1.12 (1.10 to 1.14)
Female age 60-69	5.48	1.11 (1.10 to 1.12)
Female age 70-79	5.56	1.06 (1.05 to 1.07)
New Zealand, Census 1991[w4]		
Male	2.49	1.10 (1.01 to 1.20)
Female	2.46	1.04 (0.95 to 1.13)
Sweden, Census 1990[w5]	4.13	1.02 (0.98 to 1.07)
Sweden, SLC 1980-6[w6]	0.42	1.17 (0.89 to 1.53)
US, NHEFS 1971-5[w7 w8]	1.50	1.10 (0.97 to 1.25)
US, NHIS 1987-94[w9]	5.57	1.01 (1.01 to 1.02)
US, NLMS 1979-85[w10]		
Male age 25-64	3.65	1.19 (1.13 to 1.26)
Female age 25-64	3.70	1.07 (1.01 to 1.13)
Male age ≥65	3.44	1.02 (0.96 to 1.08)
Female age ≥65	4.20	0.99 (0.95 to 1.04)
Combined	100.00	1.08 (1.06 to 1.10)
I^2 = 96% (95% CI 95% to 97%), heterogeneity P=0.000		

Cross sectional study	Weight (%)	Odds ratio (95% CI)
Canada, OHS 1996-7[w13]	11.12	1.02 (1.00 to 1.03)
Chile, NSCS 2000[w14]	11.14	1.02 (1.00 to 1.03)
China, CHNS 1991/1993/1997[w15]	3.42	1.16 (1.08 to 1.25)
Japan, AGES 2003[w16]	2.48	1.16 (1.06 to 1.27)
Japan, LCPHW 1995[w17]	11.23	1.00 (0.99 to 1.02)
Scotland, SHS 1999-2000[w18]	9.39	0.97 (0.95 to 1.00)
UK, BHPS 1991[w19]	3.93	1.06 (0.99 to 1.13)
US, BRFSS 1993-4[w20]	11.91	1.03 (1.03 to 1.04)
US, BRFSS 2000[w21]	1.59	1.22 (1.08 to 1.37)
US, CPS 1995/1997[w22 w23]	1.42	1.39 (1.23 to 1.58)
US, CTS 1996[w24]	12.08	1.01 (1.01 to 1.02)
US, NMIHS 1988[w25]	7.81	0.99 (0.96 to 1.02)
East Euro 1996/1998[w26]	1.49	1.15 (1.02 to 1.30)
Middle/East Euro 2004[w27]	2.44	1.06 (0.97 to 1.16)
WHO, CHBSAC 1997-8[w28]		
Male	3.79	1.11 (1.03 to 1.18)
Female	4.76	1.12 (1.06 to 1.18)
Combined	100.00	1.04 (1.02 to 1.06)
I^2 = 88% (95% CI 79% to 91%), heterogeneity P=0.000		

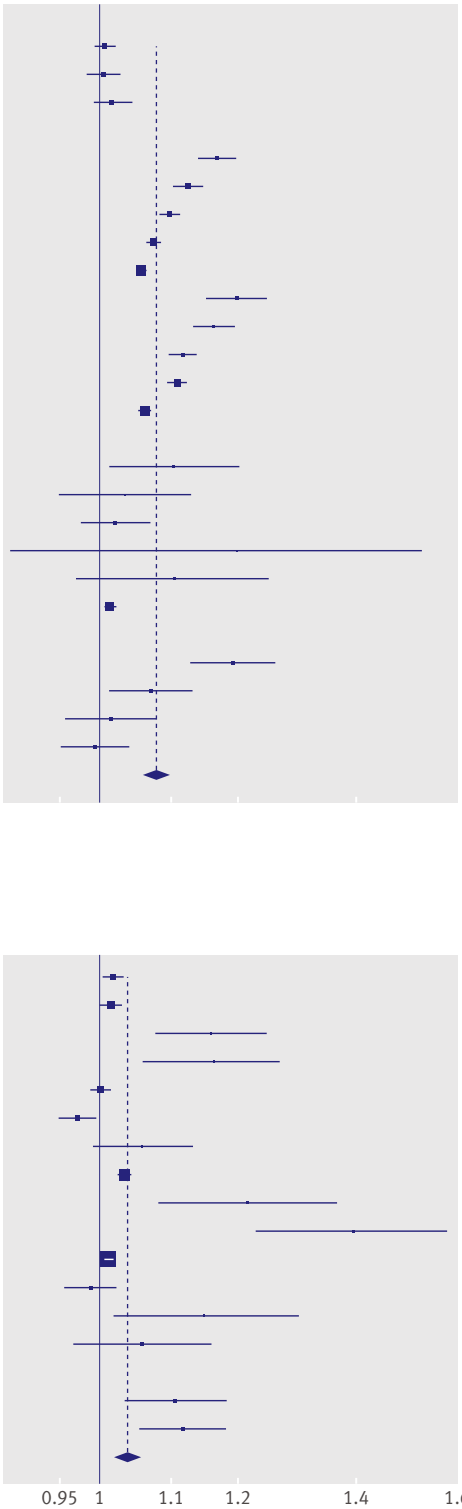

Fig 1 | **Result of primary meta-analysis of cohort and cross sectional studies: relative risks for subsequent mortality and odds ratios for poor self rated health per 0.05 unit increase in Gini coefficient. Combined relative risks and odds ratios based on weights for individual studies calculated with random effects models with restricted maximum likelihood estimate**

Country	Gini	Excess deaths	Fraction of estimated 1.5 million averted deaths (%)
Reference countries	0.225 to 0.293	0	0
Canada	0.301	103	0.007
Ireland	0.304	8	0.0065
Australia	0.305	219	0.01
Japan	0.314	23 163	1.5
South Korea	0.316	4861	0.3
UK	0.326	11 818	0.8
Spain	0.329	6971	0.5
New Zealand	0.337	78	0.005
Greece	0.345	674	0.04
Italy	0.347	16 513	1.1
Portugal	0.356	938	0.01
US	0.357	883 914	57
Poland	0.367	22 849	1
Turkey	0.439	144 723	9
Mexico	0.480	425 759	28
Total		1 542 591 (9.6% of total OECD population)	100

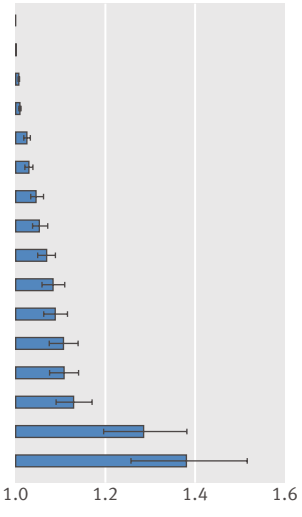

Fig 2 | Relative risks for subsequent mortality by 30 OECD member countries and estimated number of deaths avoided by levelling Gini to <0.3

between the two investigators. We contacted authors to obtain missing information.

Standardisation of income inequality measures and effect size

Some studies used other measures of income inequality; as alternative measures are all highly correlated (Pearson's r >0.94),[5] we transformed all measures to Gini coefficients. We standardised specifications of effect estimates so that they represented effects per 0.05 unit increase in Gini (about equivalent to 2.0-2.5 SD of the US state Gini).[6]

Statistical analysis

We estimated the overall relative risk for subsequent mortality among cohort studies and the overall odds ratio for poor self rated health among cross sectional studies per 0.05 unit increase in Gini coefficient. We evaluated heterogeneity.[7 8]

Using a meta-regression approach we evaluated potential factors hypothesised to account for the heterogeneity between studies—that is, potential thresholds of the Gini coefficient,[4] study region,[4 9] the length of follow-up, the incorporation of time lags between income inequality and health outcomes,[10-12] the age range of the subjects,[w10 13] and whether the study was between countries versus within one country. Additional potential sources of heterogeneity evaluated included data period, alternative income inequality measures, and adjustment for area income.

In a sensitivity analysis we examined alternative sets of models—for example, those controlling for unmeasured regional characteristics through fixed effects. A meta-analysis substituting three models[w3 w6 w10] with their region adjusted alternatives further evaluated the effect of adjusting for unmeasured regional characteristics. We used funnel plots to detect publication bias. Finally, we estimated the potential national impacts of income inequalities on mortality in every OECD country based on thresholds suggested.

Gini coefficient

The Gini coefficient is formally defined as half of the arithmetic average of the absolute differences between all pairs of incomes within the sample, with the total then being normalised on mean income. If incomes are distributed completely equally, the value of the Gini will be zero. If one person has all the income (complete inequality) the Gini will assume a value of 1.

RESULTS

From the 2839 potentially relevant articles, we excluded 2679 because they were outside the scope of this review. Among the 160 remaining papers, 54 articles had multilevel data on income inequality and mortality or self rated health. We excluded a further 26 papers. Finally, nine cohort and 19 cross sectional data matched our inclusion criteria, covering 59 509 857 cohort and 1 280 211 cross sectional individuals. The studies included 10 countries: Canada, Chile, China, Denmark, Finland, Japan, Norway, New Zealand, the United Kingdom, and the US.

The overall cohort relative risk (95% confidence interval) for mortality adjusted for sociodemographic characteristics (including individual socioeconomic status) was 1.08 (1.06 to 1.10) per 0.05 unit increase in Gini (fig 1). The overall cross sectional odds ratio for poor self rated health was 1.04 (1.02 to 1.06) in binary logistic regressions (fig 1) and 1.08 (1.01 to 1.14) in ordinal regressions (see fig A on bmj.com). The effect sizes among studies were heterogeneous (P<0.001 for heterogeneity for all meta-analyses).

Meta-regression analyses showed a significantly higher cohort relative risk among studies with higher average Ginis, later baseline data (>1990), and adjustment for area income compared with their counterparts; while the length of follow-up (>7 years) showed a marginally higher relative risk (see bmj.com). The overall cohort relative risk for studies with average Gini of 0.30 or higher was 1.01 (1.07 to 1.12), while the relative risk was 1.02 (0.97 to 1.07) for those lower than 0.30. Heterogeneity between studies was not explained by the choice of income inequality measure (Gini or median share), adjustment for other contextual factors, whether the study was done in the US or not, or age range (<60 v ≥60). Cross sectional meta-regressions showed similar trends in terms of average Gini, incorporation of time lag, and study regions. In addition, between country studies showed significantly higher overall odds ratios (1.11) than within country studies (1.02).

In our sensitivity analyses, none of the inclusions and exclusions of specific studies (see table A on bmj.com) nor one by one exclusions of each study (data not shown) materially changed the results of the primary meta-analyses. One exception is the alternative meta-analysis replacing three models[w3 w6 w10] with those

adjusted for regions, which attenuated the overall relative risk from 1.08 (1.06 to 1.10) to 1.02 (1.0 to 1.04).

We did not find a significant publication bias among cohort studies (Begg's P=0.60), although there was a suggestion of publication bias among the cross sectional studies (P=0.03) (see fig B on bmj.com). When we removed the three smallest cross sectional studies[w21-23 w26] the bias was not significant (P=0.13).

We predicted the potential excess risks of premature mortality for each OECD country. The excess risks for selected countries were 3% in Japan, 11% in the US, and 38% in Mexico compared with the countries having Ginis lower than 0.3 (fig 2).

DISCUSSION

Principal findings

Our meta-analysis of cohort studies including around 60 million participants found that people living in regions with high income inequality have an excess risk for premature mortality independently of their socioeconomic status, age, and sex. A similar conclusion was supported by our meta-analysis of cross sectional studies with poor self rated health as the outcome. The estimated excess mortality risk was 8% per 0.05 unit increase in the Gini coefficient. If the inequality-mortality relation is truly causal then the population attributable fraction suggests that upwards of 14 million deaths (9.6%) could be averted in 30 OECD countries by levelling the Gini coefficient below the threshold value of 0.3.[14]

Sources of heterogeneity between studies

The combined cohort relative risk and cross sectional odds ratio should be interpreted with caution, given the substantial heterogeneity detected between studies. Several local factors seem to account for this heterogeneity, including the possibility of a "threshold" effect of income inequality on health (with Gini ≥0.3 indicating a more consistent association with adverse health effects), the time period in which the analyses were carried out (with studies after 1990 indicating a more consistent association), and the length of follow-up in the cohort studies.

Among the cross sectional studies, between country studies showed a significantly stronger association between income inequality and self rated health than within country studies. This observation is consistent with the conclusion of a recent systematic review suggesting that studies with smaller reference groups are less likely to show an association with health because the spatial scale does not reflect the social stratification of societies.[15]

Study limitations

Several limitations need to be borne in mind in interpreting our findings. First, all meta-analyses of observational studies are prone to biases in the original studies.[16] Secondly, five cross sectional analyses did not report the necessary information to permit us to include them in the meta-analysis.[11 17-20] Their omission might have influenced our conclusions. Thirdly, the Gini coefficient is an overall summary measure of income distribution that is insensitive to the shape of the distribution.

Conclusion

Although our study suggests that there is an association between higher income inequality and worse health outcomes, further investigations are needed because of the lack of empirical evidence from many parts of the world. Factors accounting for the heterogeneity between studies warrant further study.

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Competing interest: None declared.

Ethical approval: Not required.

Data sharing: No additional data available.

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Teenage pregnancy and social disadvantage: systematic review integrating controlled trials and qualitative studies

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ABSTRACT

Objectives To determine the impact on teenage pregnancy of interventions that address the social disadvantage associated with early parenthood and to assess the appropriateness of such interventions for young people in the United Kingdom.

Design Systematic review, including a statistical meta-analysis of controlled trials on interventions for early parenthood and a thematic synthesis of qualitative studies that investigated the views on early parenthood of young people living in the UK.

Data sources 12 electronic bibliographic databases, five key journals, reference lists of relevant studies, study authors, and experts in the field.

Review methods Two independent reviewers assessed the methodological quality of studies and abstracted data.

Results 10 controlled trials and five qualitative studies were included. Controlled trials evaluated either early childhood interventions or youth development programmes. The overall pooled effect size showed that teenage pregnancy rates were 39% lower among individuals receiving an intervention than in those receiving standard practice or no intervention (relative risk 0.61; 95% confidence interval 0.48 to 0.77). Three main themes associated with early parenthood emerged from the qualitative studies: dislike of school; poor material circumstances and unhappy childhood; and low expectations for the future. Comparison of these factors related to teenage pregnancy with the content of the programmes used in the controlled trials indicated that both early childhood interventions and youth development programmes are appropriate strategies for reducing unintended teenage pregnancies. The programmes aim to promote engagement with school through learning support, ameliorate unhappy childhood through guidance and social support, and raise aspirations through career development and work experience. However, none of these approaches directly tackles all the societal, community, and family level factors that influence young people's routes to early parenthood.

Conclusions A small but reliable evidence base supports the effectiveness and appropriateness of early childhood interventions and youth development programmes for reducing unintended teenage pregnancy. Combining the findings from both controlled trials and qualitative studies provides a strong evidence base for informing effective public policy.

WHAT IS ALREADY KNOWN ON THIS TOPIC

Evidence suggests that sex education and better sexual health services do not reduce teenage pregnancy rates

A number of controlled trials have tested the effects of interventions that target the social disadvantage associated with early pregnancy and parenthood, and a number of qualitative studies have considered young people's views of the factors associated with teenage pregnancy

No systematic review has brought these quantitative trials and qualitative studies together to determine intervention effectiveness and appropriateness

WHAT THIS STUDY ADDS

Early childhood interventions and youth development programmes that combine individual level and structural level measures to tackle social disadvantage can lower teenage pregnancy rates

Such interventions are likely to be appropriate for children and young people in the UK because they improve enjoyment of school, raise expectations and ambitions for the future, and ameliorate the effect of an unhappy childhood in poor material circumstances

A policy move to invest in interventions that target social disadvantage should complement rather than replace high quality sex education and contraceptive services

INTRODUCTION

Traditional approaches to reducing teenage pregnancy rates—such as sex education and better sexual health services—are not effective on their own.[1 2] This has increased interest in interventions that target the social disadvantage associated with early pregnancy and parenthood.

The objectives of this study were to determine, on the basis of evidence in qualitative and quantitative research, the impact on teenage conceptions of interventions that address social disadvantage and to assess the appropriateness of such interventions for young people in the UK.

METHODS

We undertook a three part systematic review of the research evidence on social disadvantage and pregnancy in young people by using an innovative method for integrating qualitative and quantitative research.[3 4] The first part of the review focused on quantitative controlled trials, the second on qualitative research, and in the third part we integrated the two sets of findings to assess the extent to which existing interventions address the needs and concerns of young people.

The inclusion of qualitative research in systematic reviews facilitates the incorporation of "real life" experiences into evidence based policy making. Although we included trials conducted in any country, we drew only on qualitative studies conducted in the UK.

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Search strategy

Our literature searches covered seven major databases and five specialist registers. See bmj.com.

We included randomised and non-randomised controlled trials that evaluated interventions designed to target social disadvantage and that reported teenage conceptions or births as an outcome measure. We included any qualitative study published between 1994 and 2004 that focused on teenage pregnancy and social disadvantage among young people aged less than 20 years old living in the UK.

Relevant interventions were those that aimed to improve young people's life opportunities and financial circumstances—for example, through educational or income support. Relevant interventions could be targeted at children, young people, or their families.

Quality assessment

We assessed the extent to which controlled trials had minimised bias and error in their findings. "Sound" trials were those that reported data on each outcome measure indicated in the study aims; used a control or comparison group; and provided pre and post-intervention data for all individuals in each group. The criteria we used to assess the methodological quality of the qualitative studies were built on those suggested in the literature on qualitative research. Each study was assessed according to 12 criteria designed to aid judgment on the extent to which study findings were an accurate representation of young people's perspectives and experiences. See bmj.com.

Data synthesis

The data synthesis was conducted in three stages.[3] Firstly, we used statistical meta-analysis techniques to assess the effectiveness of the interventions in the trials. Relative risk (RR) was used to calculate both individual study and combined effect sizes. Secondly, we conducted a thematic synthesis of the findings from the qualitative studies, following established principles. Thirdly, we constructed a methodological and conceptual matrix to integrate the findings of the two syntheses.

RESULTS

Study characteristics and quality

Ten controlled trials[w1-w10] and five qualitative studies[w11-w15] met our inclusion criteria. Six controlled trials were judged to be of sufficient methodological quality to provide reliable evidence about the impact of interventions on teenage pregnancy rates.[w1-w3 w6 w7 w9] All controlled trials were conducted in the US and targeted disadvantaged groups of children and young people. See bmj.com.

Each of the methodologically sound controlled trials evaluated one of two intervention types: (*a*) an early childhood intervention, or (*b*) a youth development programme. Three studies evaluated early childhood interventions that aimed to promote cognitive and social development through preschool education, parent training, and social skills training.[w2 w3 w7] A further three studies evaluated youth development programmes that aimed to promote self esteem, positive aspirations, and a sense of purpose through vocational, educational, volunteering, and life skills work.[w1 w6 w10]

All five qualitative studies were judged to be of medium or high quality.[w11-w15] Four studies focused on, or included, the views of young parents,[w11 w12 w14 w15] and two included the views of young fathers as well as young mothers.[w14 w15] See bmj.com.

Comparison of themes arising from studies of young people's views with interventions assessed in "sound" trials

Themes and potential measures to address them	Coverage in "sound" trials
Dislike of school	
Involve young people in decision making about the curriculum; rules and regulations; and design and layout of the school, and other aspects of school culture	None identified
Support young people starting at new schools	None identified
Equip young people with the skills to form positive relationships with other young people	Allen et al, 1997[w1]; Hawkins et al, 1999[w7]
Equip young people with the skills to resolve conflicts	Hawkins et al, 1999[w7]
Introduce anti-bullying strategies	None identified
Introduce training for secondary school teachers to provide emotional support for young people	None identified
Introduce learning support interventions	Hahn et al, 1994[w6]; Philliber et al, 2001[w10]
Increase parental involvement during secondary school	Hawkins et al, 1999[w7]
Low expectations for the future	
Improve work experience opportunities	Allen et al, 1997[w1]; Hahn et al, 1994[w6]; Philliber et al, 2001[w10]
Protect young people against bad experiences of work (for example, by introducing minimum wage, better regulation, and legislation)	None identified
Actively involve young people in careers development	Allen et al, 1997[w1]; Hahn et al, 1994[w6]; Philliber et al, 2001[w10]
Provide activities out of school to improve self esteem and positive outlook	Hahn et al, 1994[w6]; Philliber et al, 2001[w10]
Create more employment opportunities in disadvantaged communities	None identified
Raise awareness of training, employment, and careers opportunities	Allen et al, 1997[w1]; Hahn et al, 1994[w6]; Philliber et al, 2001[w10]
Unhappy childhood and poor material circumstances	
Introduce interventions to prevent domestic violence	None identified
Support children and young people experiencing family breakdown and conflict (for example, with counselling services)	Allen et al, 1997[w1]; Hahn et al, 1994[w6]; Philliber et al, 2001[w10]
Train parents in conflict resolution	Hawkins et al, 1999[w7]
Improve the continuity and quality of care for children and young people in the care of the social services	None identified
Introduce housing interventions (for example, by investing in new housing and housing repairs)	None identified

Quantitative studies of the effects of interventions on teenage pregnancy rates

Of the six controlled trials, four measured pregnancy rates reported by young women,[w1 w2 w7 w10] three measured partner pregnancy rates reported by young men,[w1 w7 w10] and two measured birth rates reported by young men and young women separately[w3] or together.[w6] Tests revealed no statistical heterogeneity between the studies, suggesting that it would be appropriate to pool the effect sizes. However, effect sizes for youth development interventions and early childhood education interventions were pooled separately.

The pooled effect size from the first meta-analysis showed that early childhood interventions and youth development programmes reduced teenage pregnancy rates among young women (RR 0.61, 95% CI 0.48 to 0.77). The pooled effect size from the second meta-analysis showed that young men who had received an early childhood or youth development intervention reported fewer partner pregnancies than those who had not, but this result was not statistically significant (RR 0.59, 95% CI 0.34 to 1.02). See forest plots on bmj.com.

Qualitative studies of the views and experiences of young people

Three major themes relating to teenage pregnancy emerged from the findings of the five qualitative studies: dislike of school; poor material circumstances and unhappy childhood; and low expectations and aspirations for the future.

Dislike of school was a key aspect of young parents' accounts of their lives before becoming parents and of young people identified as "at risk" of becoming teenage parents (for example, "Still be at school? I'd rather have a baby than that. I just didn't like school, it was hard, it was horrible"[w14]). The reasons young people gave for disliking school varied such as bullying by teachers and peers (for example, "I got bullied so I just stopped going"[w12]). Young parents reported unhappiness, rather than poverty in itself, as the most significant aspect of their childhood experiences that related to becoming a parent, although unhappiness went hand in hand with adversity and material disadvantage in their accounts. Common experiences included family conflict and breakdown, sometimes caused by violence, which could lead to living in care. Young parents noted how they had to "grow up faster" in order to survive, and also reported a lack of confidence, low self esteem, and high anxiety levels.[w11] However, not all the teenage mothers who participated in these studies had grown up unhappy or experienced personal adversity.

There were differences in the expectations and aspirations of young people who had, or wanted to have, a baby early in life and young people who had or wanted to have a baby later in life. Both young mothers and young fathers believed that few opportunities were open to them apart from poorly paid, temporary work in jobs that they disliked (for example, "There are so many jobs out there that I didn't even know existed . . . I probably could have done something but I just didn't even think of these high paid jobs I could have done"[w14]).

Do current interventions address the needs and concerns reported by young people?

The themes in our synthesis of qualitative studies suggest areas that should be addressed in preventive interventions, but measures to target these areas have not all been soundly evaluated for their effect on teenage pregnancy rates (table).

DISCUSSION

Summary of principal findings

The evidence from the controlled trials showed that early childhood interventions and youth development programmes can significantly lower teenage pregnancy rates. Both types of intervention target the social determinants of early parenthood. Preschool education and support appear to exert a long term positive influence on the risk of teenage pregnancy, as well as on other outcomes associated with social and economic disadvantage such as unemployment and criminal behaviour.[5] Programmes of social support, educational support, and skills training delivered to young people have a much more immediate impact. Our review of qualitative studies indicated that happiness, enjoying school, and positive expectations for the future can all help to delay early parenthood.

Our findings are especially important in the light of evidence that sex education and sexual health services are not on their own effective strategies for encouraging teenagers to defer parenthood.[1] However, important gaps exist in the evidence on how effectively current early childhood interventions and youth development programmes address the themes from the qualitative synthesis. Structural and systemic issues such as housing, employment opportunities, community networks, bullying, and domestic violence were all important issues in young people's accounts, but these factors have yet to be addressed in appropriate interventions and evaluated as wider determinants of teenage pregnancy.

Comparison with other studies

Dislike of school, an unhappy childhood, and a lack of opportunities for jobs and education have all emerged as explanatory factors in large scale national and international epidemiological analyses.[6-12] Our analysis of qualitative research provides additional insight into how factors that increase the risk of teenage pregnancy may operate.

We found few trials conducted in the UK, which raises questions about the generalisability of this evidence and shows that further trials are needed in the UK and elsewhere. In particular, trials are needed that deliver interventions in normal community settings and schools, rather than out of mainstream schools, as in a recent youth development programme in the UK.[13]

Strengths and limitations of the study

The strengths of our review include the comprehensiveness of our searches, the exclusion of methodologically weak studies, the rigorous synthesis methods used, and the inclusion of qualitative research alongside controlled trials. Including only studies that evaluated interventions relative to control conditions over the same period of time avoids missing temporal differences between groups.

The small numbers of studies we found are a limitation, as is the dominance of controlled trials conducted in the US. We are only aware of one relevant study published since the searches for this review were carried out.[13] Whether this study would meet the quality criteria for our review is unclear, but it should be considered in any update.

Conclusion and policy implications

A policy move to invest in youth programmes should complement rather than replace high quality sex education and contraceptive services, and should aim to improve enjoyment of school, raise expectations and ambitions for the future, and provide young people with relevant social support and skills.

Contributors: See bmj.com

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Competing interests: None declared.

Data sharing: Technical appendix available at http://eppi.ioe.ac.uk/cms/Default.aspx?tabid=674.

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Sign of the cross

There we stood in room 17 of the neurosurgical ward—ENT specialist registrar poised with tracheal dilator, ENT senior house officer with head light, and staff nurse armed with suction—surrounding a teenage patient with cerebral palsy on the bed for routine change of his long term tracheostomy tube. Suddenly the patient's eyes began to roll, and his limbs began to jerk. Nurses were called for, and the ENT team started looking edgy in that "please someone else lead this emergency, it's not our field" sort of way.

And then the most extraordinary thing happened. The patient's mother rose from her bedside chair, calm as a millpond. Leaning over her son, she murmured his name reassuringly and pressed a button on a stopwatch. She then picked up a small rectangular object and firmly traced a large sign of the cross on his left hemi-thorax. He continued to fit. Seconds later another nurse walked in and, with similar equanimity, traced the symbol of christianity on to the patient's chest. At one minute twenty seconds his fitting subsided.

There we still stood in room 17—relieved yes, but utterly baffled. Was this hocus-pocus, had ward 35 turned uber holy, or had we been sleeping when this particular form of seizure control was taught at medical school?

It turns out the latter was true. What the mother and nurse were so expertly demonstrating was an evidence based treatment for intractable epilepsy. Vagus nerve stimulation is a treatment for epilepsy where a small generator is implanted under the skin below the left collar bone and connected to a lead with three coils at one end. These coils are wrapped around the vagus nerve in the left side of the neck in a small operation. The device stimulates the vagus nerve at intervals to reduce the frequency and intensity of seizures.

The generator sends impulses from the vagus nerve in the neck to the brain and delivers therapy in two ways. Firstly, a doctor programmes a daily "dose" of automatically delivered intermittent stimulation. Additionally, when a patient, family member, or care giver senses a seizure coming on, they can pass a magnet over the area in the chest where the generator is implanted to activate an extra, on-demand stimulation.

Fascinating stuff. Effective too.

Amen.

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Patient consent obtained.

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pico

Effect of tailored practice and patient care plans on secondary prevention of heart disease in general practice: cluster randomised controlled trial

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STUDY QUESTION What is the effectiveness of a complex intervention with tailored plans for practices and patients on outcomes for patients with coronary heart disease?

SUMMARY ANSWER Hospital admissions were significantly reduced after an 18 month intervention but no other benefits were shown, possibly because of a ceiling effect related to improved management of coronary heart disease.

WHAT IS KNOWN AND WHAT THIS PAPER ADDS Structured programmes in primary care lead to improved provision of secondary prevention for patients with established heart disease, but expected returns may not be achieved when baseline management levels are high. Current efforts at secondary prevention in primary care should be maintained but future focus may be at the population level and on those patients with additional absolute risk or who are less likely to be receiving optimal therapy.

Design

Practices were randomly selected until 16 had been recruited in each of two centres in the Republic of Ireland and one centre in Northern Ireland. The practices prepared lists of all patients with heart disease, and potential participants were randomly selected and invited in sequence until 20 in each practice agreed to participate. The intervention involved tailored practice care (such as individual practice care plans and practice based training in prescribing and behavioural change) and tailored patient care (such as motivational interviewing and goal identification and regular review using a patient held booklet).

Participants and setting

Our study population comprised 903 patients with heart disease from 48 general practices in two different healthcare systems.

Primary outcome(s)

Our main outcome measure was the proportion of patients above target levels for blood pressure and total cholesterol concentration and being admitted to hospital after 18 months.

Main results and the role of chance

All practices completed the study and 838 of 903 (92.8%) patients participated in follow-up. At 18 months there were no significant differences between intervention and control groups in the numbers of patients above the recommended limits: systolic blood pressure, intervention 98/360 (27.2%) *v* control, 133/405 (32.8%), odds ratio 1.51 (95% confidence interval 0.99 to 2.30; P=0.06); diastolic blood pressure, intervention 32/360 (8.9%) *v* control, 40/405 (9.9%), 1.40 (0.75 to 2.64; P=0.29); and total cholesterol concentration, intervention 52/342 (15.2%) *v* control, 64/391 (16.4%), 1.13 (0.63 to 2.03; P=0.65). The number of patients admitted to hospital over the 18 month study period significantly decreased in the intervention group compared with the control group: 107/415 (25.8%) *v* 148/435 (34.0%), 1.56 (1.53 to 2.60; P=0.03).

Harms

The two groups showed no differences in physical or mental status, as measured by the SF-12.

Bias, confounding, and other reasons for caution

Selection bias might have favoured "good" practices and "compliant" patients, with the result that baseline performance was high with little scope for improvement. The study may be underpowered for determination of blood pressure and cholesterol outcomes. Data collection was not blinded. Analysis of hospital admissions may have been affected by different data collection periods at baseline and follow-up. Economic evaluation of the intervention is required.

Generalisability to other populations

Patient randomisation occurred subsequent to baseline data collection, which should enhance the trial's internal validity. The low numbers of practice nurses in the Republic of Ireland made many practices there ineligible for inclusion. The successful delivery of the trial in two healthcare systems indicates that the intervention can be implemented in different settings.

Study funding/potential competing interests

The study was funded by the Health Research Board and Irish Heart Foundation.

Trial registration number

Current Controlled Trials ISRCTN24081411.

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COMPARISON OF CONTINUOUS OUTCOMES ADJUSTED FOR CLUSTERING, BASELINE DIFFERENCES, AND PRESPECIFIED COVARIATES AT 18 MONTH FOLLOW-UP

Variables	Follow-up		Mean difference (95% CI)
	Intervention (n=444)	Control (n=459)	
Systolic blood pressure (mm Hg)	133.8 (17.0)	137.9 (19.3)	3.31 (-1.02 to7.63)
Diastolic blood pressure (mm Hg)	77.4 (10.1)	78.6 (10.4)	0.17 (-2.16 to 2.51)
Total cholesterol concentration (mmol/l)	4.2 (0.9)	4.2 (0.9)	0.13 (-0.03 to 0.30)
No of hospital admissions per patient	0.4 (0.7)	0.5 (1.0)	-0.15 (-0.01 to -0.29)
No of cardiovascular hospital admissions per patient	0.14 (0.5)	0.23 (0.7)	-0.11 (-0.21 to -0.01)

Values are means (standard deviations) unless stated otherwise

pico

Slow walking speed and cardiovascular death in well functioning older adults: prospective cohort study

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EDITORIAL by Harwood and Wilkinson

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STUDY QUESTION What is the relation between slow walking speed over a short distance and mortality, overall and according to main causes of death, in older people living in the community?

SUMMARY ANSWER In older people, a slow walking speed over a short distance is associated with an increased risk of death, in particular of cardiovascular mortality.

WHAT IS KNOWN AND WHAT THIS PAPER ADDS Decreased walking speed has been shown to be associated with several adverse health related events, including death. Participants with walking speed in the lower third had about a threefold increased risk of cardiovascular mortality, though no relation with mortality from cancer or other causes of death was observed.

Participants and setting

Participants aged 65-85 and living in the community were recruited from 1999 to 2001 from the Dijon centre (France) of the Three-City (3C) study.

Design, size, and duration

As part of a prospective cohort study, 3208 participants without a major cause of reduced mobility (Parkinson's disease, dementia, recent hip fracture, disabling stroke) and without coronary artery disease were followed for a mean duration of 5.1 years. Maximum walking speed was measured at baseline over six metres. The main outcome measure was mortality, overall, and according to the main causes of death.

Main results and the role of chance

Mean age of the participants at baseline was 73.2; 65% were women. During follow-up, 209 deaths occurred (99 from cancer, 59 from cardiovascular disease, 51 from other causes). Mortality (per 1000 person years) was 19.2 in those with a walking speed in the lowest third (≤1.50 m/s in men, ≤1.35 m/s in women) and 9.5 in those who walked faster. Participants in the lowest third of walking speed had an increased risk of all cause and cardiovascular mortality, while there was no association with mortality from cancer or other causes of death. Hazard ratios were adjusted for a range of baseline variables.

WALKING SPEED IN OLDER PEOPLE AND RISK OF DEATH

Cause of death	Adjusted hazard ratio (95% CI)	P value
All cause	1.44 (1.03 to 1.99)	0.02
Cancer	1.03 (0.65 to 1.70)	0.83
Cardiovascular	2.92 (1.46 to 5.84)	0.002
Other causes	1.41 (0.74 to 2.67)	0.30

Bias, confounding, and other reasons for caution

High reproducibility of the walking speed measure was ensured by using photoelectric cells. Cause of death was determined by a committee blinded to the measure of walking speed, based on medical records and information provided by treating physicians. Few participants were lost to follow-up (n=8).

Generalisability to other populations

The 3C cohort was volunteer based and is not representative of the general population aged over 65. Although this is likely to lead to an underestimation of the incidence of events such as deaths among older people, the relation between a baseline characteristic (walking speed) and incidence of an event (death) during follow-up is unlikely to be biased because of the negligible number of losses to follow-up.

Study funding/potential competing interests

The 3C study is conducted under a partnership agreement between INSERM, Bordeaux II University, and Sanofi-Synthélabo, and is supported by FRM, CNAMTS, DGS, HAS, INPES, MGEN, Conseils Régionaux de Bourgogne, Fondation de France, Ministère de la Recherche, Institut de la Longévité, Conseil Général de la Côte d'or. JD was supported by a PhD scholarship from the FRM. The authors were independent from the funders in all aspects of the study design, analysis of data, and writing of the manuscript.

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Trends in Down's syndrome live births and antenatal diagnoses in England and Wales from 1989 to 2008: analysis of data from the National Down Syndrome Cytogenetic Register

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STUDY QUESTION How have changes in maternal age and advances in screening affected numbers of Down's syndrome live births and antenatal diagnoses in England and Wales between 1989 and 2008?

SUMMARY ANSWER Despite the population numbers of births in 1989/90 and 2007/8 being similar, antenatal and postnatal diagnoses of Down's syndrome increased by 71%. In the absence of antenatal screening and subsequent terminations, the number of Down's syndrome births would have been expected to increase by 48%. However, numbers of live births with Down's syndrome fell by 1% because of an increase in antenatal screening and subsequent terminations.

WHAT IS KNOWN AND WHAT THIS PAPER ADDS Older mothers are at increased risk of having babies with Down's syndrome. The number of Down's syndrome live births has remained fairly constant since 1989, as improvements in antenatal screening have offset an increase Down's syndrome resulting from rising maternal age.

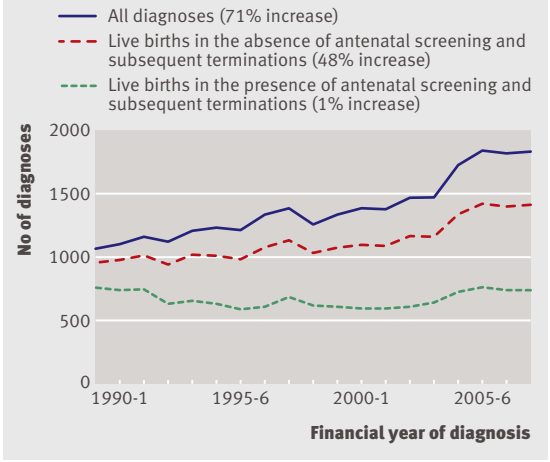
Participants and setting

The National Down Syndrome Cytogenetic Register holds details of 26 488 antenatal and postnatal diagnoses of Down's syndrome made by all cytogenetic laboratories in England and Wales since 1989.

Design

Longitudinal analysis of register data.

DOWN'S SYNDROME DIAGNOSES AND LIVE BIRTHS



Primary outcome(s)

Number of live born babies with Down's syndrome.

Main results and the role of chance

The figure compares total numbers of Down's syndrome diagnoses (top line, rising from 1075 in 1989/90 to 1843 in 2007/8) with estimated numbers of Down's syndrome live births that would have occurred in the absence of antenatal diagnoses and selective termination (middle line, rising from 959 in 1989/90 to 1422 in 2007/8). The two lines differ because the top line includes pregnancies detected antenatally and terminated that, had the pregnancy continued, would have miscarried naturally and not resulted in a live birth. The bottom line shows estimated numbers of live born babies with Down's syndrome in the presence of antenatal diagnoses and selective termination (falling from 752 to 743, 1.10 to 1.08 per 1000 births). The difference between the bottom two lines is attributable to antenatal screening and subsequent terminations, the effects of which have clearly increased over time. The rise in the number of live births expected in the absence of screening and subsequent terminations is due to a true increase in the incidence of Down's syndrome, which can be attributed to the increase in maternal age.

Bias, confounding, and other reasons for caution

The National Down Syndrome Cytogenetic Register is a unique resource that has ascertained more than 93% of diagnoses of Down's syndrome in all of England and Wales for 19 years. Its main weakness is the necessity to estimate the number of recent live births because of largely administrative delays in receiving pregnancy outcomes after an antenatal diagnosis.

Generalisability to other populations

Other countries have reported similar trends in Down's syndrome diagnoses, screening, and subsequent live births.

Study funding/potential competing interests

The NHS Fetal Anomaly Screening Programme funded the National Down Syndrome Cytogenetic Register to collect the data until March 2009. The funders had no role in the analysis or writing of this paper.

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