Teenage motherhood and risk of premature death: long-term follow-up in the ONS Longitudinal Study

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Background. Teenage motherhood is relatively common in the UK, but little is known about related health inequalities in this population. We estimated cause-specific mortality risks over three decades in a nationally representative cohort.

Method. We examined premature mortality in a 1.1% sample of all women who were teenagers in England and Wales during the 1970s, 1980s and 1990s using data from the Office for National Statistics Longitudinal Study (ONS LS). Our primary outcome was suicide. Long-term follow-up to 31 December 2006, to a potential maximum age of 49 years, was achieved through near-complete routine linkage to national mortality records. We created a time-dependent exposure variable, with relative risks estimated according to age when women first experienced motherhood *versus* a reference group of those currently without children.

Results. Women who were teenage mothers were around 30% more likely to die prematurely by any cause and almost 60% more likely to die unnaturally, whereas first-time motherhood at mature age conferred lower risk compared to women without children. Teenage motherhood was associated with a more than doubled risk of suicide [mortality rate ratio (MRR) 2.23, 95% confidence interval (CI) 1.30–3.83], and elevated risks of fatal cancer of the cervix and lung were also found. Changing the reference category to first-time mothers at 20 years and above also revealed a significant elevation in risk of accidental death.

Conclusions. The complex psychosocial needs of these women require greater attention from clinicians, public health professionals, social services and policymakers. Their elevated risk of poor health outcomes may persist well beyond the actual teenage motherhood years.

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Introduction

The UK has the highest rate of teenage motherhood among western European countries (Moffitt *et al.* 2002; Department of Health, 2010). Strong links with health inequality, poverty and social exclusion prompted the government to highlight the importance of this issue by establishing a multi-agency Teenage Pregnancy Strategy (Department for Children, Schools and Families, 2010). In essence this is a preventive initiative, but little is known about what happens to these mothers following the birth of their children, in particular their health outcomes including premature mortality. The only population-based investigation of this topic to date was conducted in Sweden

* Address for correspondence : Dr R. T. Webb, Mental Health and Neurodegeneration Research Group, Jean McFarlane Building, The University of Manchester, Oxford Road, Manchester M13 9PL, UK. (Otterblad-Olausson *et al.* 2004). This national registry study examined cause-specific mortality outcomes during 1990–1995 among approximately 61 000 women who became teenage mothers compared to approximately 400 000 women who became first-time mothers at 20–29 years. With adjustment for family size and socio-economic status, significantly elevated risks were observed for both suicide and homicide. Such information highlights an important health inequality that may be overlooked in the context of this well-known social phenomenon. Understanding more about poor health outcomes in this group is likely to facilitate more effective planning of health-care services, and delivery of better mental health and primary care services.

Rates of teenage childbearing are considerably higher in the UK than in Sweden. Social scientists have shown that the likelihood of adverse outcome for this group may vary greatly between countries according to differences in prevalence of teenage motherhood

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and its acceptability to the wider community (Robson & Berthoud, 2003). Teen mothers in the UK may have very different characteristics and associated outcomes than those in Scandinavia. Therefore, we aimed to examine risks of suicide and other types of premature death using data from the Office for National Statistics Longitudinal Study (ONS LS). This freely available national resource provided long-term historical follow-up data from the 1970s until the end of 2006. We hypothesized that, compared with teenage girls and women without children, those who were teenage mothers would have elevated risk of premature mortality, that women who became mothers for the first time in their twenties and thirties would be at lower risk, and that effect sizes would be larger for unnatural than for natural deaths.

Method

Description of the study cohort, exposure classification and potential confounders

This investigation was conducted entirely with data extracted from the ONS LS, which contains linked census and vital event data for a 1.1% representative sample of approximately 0.5 million people from the entire population of England and Wales at each 10-year national census (www.ons.gov.uk/about/ who-we-are/our-services/longitudinal-study/index. html). The sample is selected at random by entering into the cohort every person born on one of four index dates each year. Data from the four consecutive national censuses from 1971 onwards, and also national birth, death and emigration/immigration registration systems, are linked routinely to the ONS LS. Tracing of cohort members is done primarily through the National Health Service (NHS) Central Register. Independent searches of mortality registration records for deaths to persons born on an ONS LS sampling date are also conducted to increase linkage completeness, which was around 96% for deaths during the 1970s and is almost complete from the 1980s onwards. Mother-infant linkage through birth registration records is estimated to be around 92% and 94% complete during the 1970s and 1980s ONS LS member cohorts respectively.

For this study we identified all women who were estimated to have reached age 13 years during the 1970s, 1980s and 1990s and who were sampled members of the ONS LS at the 1971, 1981 and 1991 censuses respectively. The study data sets were analysed as three discrete cohorts, although there was overlap between them, in that some members of the 1970s cohort were also members of the 1980s cohort, and likewise for the 1980s *versus* 1990s cohorts. A generic study exit date of 31 December 2006 was applied for all three cohorts, and a person-years count for each woman was calculated up to that date, or date of emigration or death, whichever came first. We conducted our primary analyses on the 1970s cohort. This enabled long-term follow-up with a potential maximum 37-year observation period (an age range of 13–49 years), and it also maximized statistical power and precision. Additional analyses of suicide risk for the 1980s and 1990s cohorts were conducted for comparative purposes. To enable meaningful comparison of effects across all three cohorts, we assessed risk at age <35 years in these analyses.

Maternal status was treated as a time-dependent variable by stratifying each woman's person-years contribution over time; the methodological details of this approach are explained in the 'Statistical analyses' section. The reference group for relative risk estimation consisted of teenage girls and mature women who had not yet experienced motherhood, the exposure group of primary interest was women who became teenage mothers, and for comparative purposes we also examined mortality risks in women who were first-time mothers at ages 20-29 years and at 30 years and older. This particular reference group was chosen because it made the largest contribution to the total person-years denominator (over 50%), and also because we wanted to examine a likely protective effect of first becoming a mother at mature age. We conducted additional analyses (Table 4) directly comparing risks in those who first became mothers in their teens compared with in their twenties and older.

The estimates were adjusted for age group (13–24, 25–34, \geq 35 years) and calendar year period in decades (1970s, 1980s, 1990s, 2000s) as time-dependent variables, and some were further adjusted for socioeconomic status using electoral ward-level Carstairs scores at the 1971 Census. This is a widely used composite ecological measure of material deprivation based on four small area-level variables: male unemployment, overcrowding, lack of car ownership, and low occupational social class (Morris & Carstairs, 1991; McLeod, 2001). Relative area-level deprivation was assigned to all ONS LS members, categorized into quintiles of equal population size, according to the distribution of Carstairs scores across all wards in England and Wales. We opted to examine only the 1971 Carstairs quintiles because all women in the 1970s cohort were present at that time point. Applying Carstairs values from later censuses in 1981, 1991 or 2001 would have been problematic because women were subsequently lost from the 1970s cohort due to premature death or emigration. For example, 14.8% of all the unnatural deaths to cohort members occurred

| Table 1. IC | D codes | used to | delineate | specific | causes of | premature | mortality |
|-------------|---------|---------|-----------|----------|-----------|-----------|-----------|
| | | | | | | | |

| Unnatural causes: ICD-8 and ICD-9 E800–E999; ICD-10 V01–Y98 Suicide (including open verdict): ICD-8 and ICD-9 E950–E959, E980–E989 (excluding E988.8); ICD-10 X60–X84, Y10–Y34 (excluding Y33.9), Y87.0, Y87.2 Accident: ICD-8 and ICD-9 E800–E949; ICD-10 V01–X59, Y85–Y86 Homicide: ICD-8 and ICD-9 E960–E969, E988.8; ICD-10 X85–Y09, Y33.9, Y87.1 |
|---|
| Natural causes: all codes except for ICD-8 and ICD-9 E800–E999; ICD-10 V01–Y98 |
| Cancers – all sites: ICD-8 and ICD-9 140–239; ICD-10 C00–D48 |
| Lung cancer: ICD-8 and ICD-9 162; ICD-10 C33–C34 |
| Breast cancer: ICD-8 and ICD-9 174; ICD-10 C50 |
| Cervical cancer: ICD-8 and ICD-9 180; ICD-10: C53 |
| Heart disease : ICD-8 and ICD-9 410–414 ; ICD-10 I20–I25 |
| Respiratory diseases: ICD-8 and ICD-9 460–519; ICD-10 J00–J99 |
| Alcohol-related deaths ^a : ICD-8 291, 303, 305, 425, 571, 860; ICD-9 (1980–1992) 291, 303, 305, 425, 571, 860; ICD-9 (1993–2000) 291–291.9, 303–303.9, 305.0, 425.5, 571–571.8, 860; ICD-10: F10–F10.9, K70–70.9, K73–K74.6, X45–X45.9 |

^a With the exception of alcohol-related deaths, each specific cause was categorized according to the underlying cause assigned on the death certificate. Contributory causes were also included in the event count for alcohol-related deaths, although the contributory cause codes were unavailable for December 1986 to January 1993.

during the 1970s, and another 33.6% of these deaths happened in the next decade. A recent validation exercise comparing Carstairs data over time has shown that, although areas do change somewhat in their level of deprivation, the relative position of areas stays similar over time (Norman, 2010). Furthermore, applying the later Carstairs values would have required much finer stratification of the aggregated person-years data, to such a level that would have prevented their release by the ONS LS. To address the fact that the 1971 Carstairs data become increasingly outdated as the follow-up time lengthened, we conducted two sets of adjusted analyses, one for the entire follow-up period up to 31 December 2006 and the other for follow-up only until 31 December 1989.

Premature mortality outcomes

We selected suicide as our primary outcome because of its strong influence on premature mortality by years of potential life lost (YPLL; Gardner & Sanborn, 1990; Gunnell & Middleton, 2003). In line with widely accepted UK convention, we examined suicide and open verdicts as a coalesced category (Linsley *et al.* 2001), which is referred to as 'suicide' for the remainder of the paper. We also expected to observe higher risk of death due to homicide, heart disease, respiratory diseases, and cancer of the cervix and lung, as shown previously in Sweden (Otterblad-Olausson *et al.* 2004). We used International Classification of Disease (ICD) codes, 8th, 9th and 10th revisions, to categorize specific causes of death (WHO, 1967, 1977, 1992), as shown in Table 1.

Statistical analyses

Statistical analyses were conducted using Stata version 10 (StataCorp, 2008). We examined maternal status as a time-dependent explanatory variable so that a woman's exposure status switched from the reference group (no children) to first-time mother as a teenager, or at 20–29 years or at \geq 30 years, at the birth of her first child. Each woman's person-years count started at age 13 years. We stratified the person-years and cause-specific death counts according to the timedependent maternal status exposure variable, the time-dependent variables age group and decade, and the time-fixed variable 1971 Carstairs quintile (Clayton & Hills, 1993). These data were then aggregated and analysed by Poisson regression modelling (Gardner et al. 1995) to generate mortality rate ratios (MRRs) as our measure of relative risk. The fit of these models was assessed in the equivalent negative binomial model (using the $\alpha = 0$ test), and none of the reported models were overdispersed. The rarity of certain cause-specific mortality outcomes meant that we encountered some sparse event data. For MRRs based on an event count of less than 5, we applied exact Poisson confidence intervals (CIs); for all other effect estimates we used asymptotic Wald approximations to generate the CI. Multivariate models were generated to assess the confounding effect of area-level deprivation using quintiles of ward-level Carstairs index. This was fitted as a categorical variable only, to avoid making any assumptions about the pattern of association between this variable and the premature mortality outcomes.

Table 2. Mortality rate ratios (MRRs) for all deaths, and for all unnatural and all natural deaths, before age 50 years: 1970s ONS LS cohort

| Type of death | No. of children (ref.) | Teenage motherhood | First-time motherhood at 20–29 years | First-time motherhood at ≥30 years |
|----------------------------|---------------------------|-----------------------|--|--|
| Person-years at risk | 1 021 417 | 159716 | 603 274 | 129 188 |
| (% of total person-years) | (53.4) | (8.3) | (31.5) | (6.8) |
| All deaths | | | | |
| No. of deaths | 490 | 150 | 414 | 81 |
| Rate per 100 000 | 48.0 | 93.9 | 68.6 | 62.7 |
| MRR (95% CI) ^a | 1.00 | 1.29 (1.07–1.56) | 0.82 (0.71–0.95) | 0.57 (0.45-0.73) |
| Unnatural deaths | | | | |
| No. of deaths | 132 | 37 | 53 | 7 |
| Rate per 100 000 | 12.9 | 23.2 | 8.8 | 5.4 |
| MRR (95% CI) ^a | 1.00 | 1.58 (1.08-2.31) | 0.58 (0.40-0.82) | 0.32 (0.14-0.70) |
| Natural deaths | | | | |
| No. of deaths | 358 | 113 | 361 | 74 |
| Rate per 100 000 | 35.0 | 70.8 | 59.8 | 57.3 |
| MRR (95 % CI) ^a | 1.00 | 1.22 (0.98-1.52) | 0.88 (0.75-1.03) | 0.62 (0.48-0.81) |

CI, Confidence interval; ONS LS, Office for National Statistics Longitudinal Study.

^a MRRs adjusted for age group ($<25, 25-34, \ge 35$ years) and decade as time-dependent variables; statistically significant effects highlighted in bold.

Source: ONS LS.

Results

In the ONS LS cohort of girls reaching age 13 years during the 1970s, there were over 1.9 million personyears of follow-up to the end of 2006 and 1135 deaths from all causes (906 natural deaths and 229 unnatural deaths). With the reference group being teenagers and women without children (53.4% of the total denominator), those who became teenage mothers (8.3%) were almost 30% more likely to die from any cause and almost 60% more likely to die from an unnatural cause (Table 2). A smaller increase in risk of natural death was also indicated, although this effect was nonsignificant (p = 0.07). Women who become first-time mothers at age 20-29 years had significantly lower mortality risks from all causes and by unnatural causes, compared with the reference group, and this apparent protective effect was even stronger among women experiencing motherhood for the first time at age \geq 30 years. Adjusted for time-dependent age group and decade of follow-up, the MRRs suggest that these older first-time mothers were far less likely to die than their peers without children, with the protective effect being especially strong against unnatural death. We examined age group (13-34 v. 35-49 years) as a potential effect modifier (results not shown in the tables). For these analyses we collapsed the first-time mothers aged 20–29 and \geq 30 years into a single category because of the sparse data for unnatural deaths. The interaction terms indicated no evidence of variation by age group in relative risks for all unnatural (χ^2 = 2.9, 2 df, *p* = 0.23) and all natural deaths (χ^2 = 0.7, 2 df, *p* = 0.71). However, these results did show that, at age < 35 years (MRR 0.46, 95% CI 0.26–0.81) and at 35–49 years (MRR 0.53, 95% CI 0.33–0.84), the risk of these mature first-time mothers dying unnaturally was roughly half that seen in women who did not have children.

We also examined specific causes of death (Table 3). We found significantly higher risk of death by suicide and by cancer of the cervix and lung among the women who first became mothers as teenagers. There was a trend towards higher risk of alcohol-related death, although this effect was non-significant (p=0.14). The event data were too sparse to examine whether risk of dying by homicide varied significantly according to time-dependent maternal status. Compared to women without children, those who first became mothers in their twenties and at older age had a significantly lower risk of accidental death. To enable better comparison across the three ONS LS cohorts (1970s, 1980s, 1990s), we examined suicide risk at age <35 years. These results are presented in Table 4. A consistent, approximate threefold increase in risk **Table 3.** Mortality rate ratios (MRRs) for specific causes of death before age 50 years:

 1970s ONS LS cohort

| Specific cause of death | No. of children (ref.) | Teenage motherhood | First-time motherhood at ≥20 years |
|----------------------------|---------------------------|-----------------------|---------------------------------------|
| | () | | |
| Suicide | | 21 | 22 |
| No. of deaths | 44 | 21 | 33 |
| Rate per 100 000 | 4.3 | 13.1 | 4.5 |
| MRR (95% CI) ^a | 1.00 | 2.23 (1.30–3.83) | 0.67 (0.41–1.10) |
| Accident | | | |
| No. of deaths | 81 | 14 | 25 |
| Rate per 100 000 | 7.9 | 8.8 | 3.4 |
| MRR (95% CI) ^a | 1.00 | 1.07 (0.60–1.94) | 0.41 (0.25–0.69) |
| Homicide | | | |
| No. of deaths | 7 | - | - |
| Rate per 100 000 | 0.7 | - | - |
| MRR (95 % CI) ^b | 1.00 | 1.83 (0.19–9.60) | 0.40 (0.04–2.09) |
| Cancers – all sites | | | |
| No. of deaths | 155 | 57 | 231 |
| Rate per 100 000 | 15.2 | 35.7 | 31.5 |
| MRR (95% CI) ^a | 1.00 | 1.32 (0.96-1.79) | 0.91 (0.73-1.13) |
| Cancer of the cervix | | . , | |
| No. of deaths | 17 | 12 | 18 |
| Rate per 100 000 | 1.7 | 7.5 | 2.5 |
| MRR (95% CI) ^a | 1.00 | 2.26 (1.07-4.77) | 0.59 (0.30–1.16) |
| | 1.00 | | 0.00 (0.00 1.10) |
| Cancer of the lung | 10 | 10 | 10 |
| No. of deaths | 10 | 10 6.3 | 19 2.6 |
| Rate per 100 000 | 1.0 | | |
| MRR (95% CI) ^a | 1.00 | 2.87 (1.18–6.98) | 0.86 (0.39–1.88) |
| Cancer of the breast | | | |
| No. of deaths | 35 | 10 | 89 |
| Rate per 100 000 | 3.4 | 6.3 | 12.2 |
| MRR (95% CI) ^a | 1.00 | 0.81 (0.40–1.64) | 1.17 (0.79–1.74) |
| Cancer – other sites | | | |
| No. of deaths | 93 | 25 | 105 |
| Rate per 100 000 | 9.1 | 15.7 | 14.3 |
| MRR (95% CI) ^a | 1.00 | 1.14 (0.72–1.79) | 0.85 (0.62–1.16) |
| Heart disease | | | |
| No. of deaths | 46 | 16 | 71 |
| Rate per 100 000 | 4.5 | 10.0 | 9.7 |
| MRR (95 % CI) ^a | 1.00 | 1.25 (0.70-2.24) | 0.93 (0.62-1.39) |
| Respiratory diseases | | | |
| No. of deaths | 24 | 7 | 19 |
| Rate per 100 000 | 2.3 | 4.4 | 2.6 |
| MRR (95% CI) ^a | 1.00 | 1.50 (0.62–3.60) | 0.79 (0.40–1.59) |
| | | | |
| Alcohol-related death | 24 | 10 | 16 |
| No. of deaths | 24 | 13 | 46 |
| Rate per 100 000 | 2.3 | 8.1 | 6.3 |
| MRR (95% CI) ^a | 1.00 | 1.63 (0.82–3.24) | 0.93 (0.56–1.55) |

CI, Confidence interval; ONS LS, Office for National Statistics Longitudinal Study. ^a Except for homicide, MRRs adjusted for age group (<25, 25–34, \geq 35 years) and

decade as time-dependent variables; statistically significant effects highlighted in bold. ^b Homicide MRRs unadjusted due to sparse data; exact Poisson CIs calculated; event cell counts <5: numerators and rates suppressed in line with ONS LS regulations.

Source : ONS LS.

Table 4. Mortality rate ratios (MRRs) for suicide before age 35 years: 1970s, 1980s and 1990s ONS LS cohorts

| Specific cause of death | No. of children (ref.) | Teenage motherhood |
|----------------------------|---------------------------|--------------------------------|
| 1970s cohort | | |
| Person-years at risk | 851 586 | 92 760 |
| (% of total person-years) | (68.1) | (7.4) |
| No. of deaths | 28 | 12 |
| Rate per 100 000 | 3.3 | 12.9 |
| MRR (95% CI) ^a | 1.00 | 3.19 (1.59-6.42) |
| 1980s cohort | | |
| Person-years at risk | 840 125 | 82 168 |
| (% of total person-years) | (72.1) | (7.1) |
| No. of deaths | 28 | 9 |
| Rate per 100 000 | 3.3 | 11.0 |
| MRR (95% CI) ^a | 1.00 | 2.92 (1.35-6.30) |
| 1990s cohort | | |
| Person-years at risk | 561 533 | 42 796 |
| (% of total person-years) | (83.5) | (6.4) |
| No. of deaths | 14 | 4 |
| Rate per 100 000 | 2.5 | 9.3 |
| MRR (95 % CI) ^a | 1.00 | 3.73 (0.88–12.14) ^k |

CI, Confidence interval; ONS LS, Office for National Statistics Longitudinal Study. ^a MRRs adjusted for age group (<25, 25–34 years) and decade as time-dependent variables; statistically significant effects highlighted in bold.

^b Exact Poisson CI calculated due to sparse event data. *Source*: ONS LS.

Table 5. Mortality rate ratios (MRRs) re-estimated with first-time motherhood at ≥ 20 years as the reference : 1970s ONS LS cohort

| Specific cause of death | Teenage motherhood MRR (95% CI) ^a | | |
|-------------------------|---|--|--|
| | 3.35 (1.93–5.80) | | |
| Accident | | | |
| 1 icenteriti | 2.59 (1.34–5.01) | | |
| Homicide ^b | 4.59 (0.33-63.27) | | |
| Cancer – all sites | 1.45 (1.08-1.93) | | |
| Cancer of the cervix | 3.82 (1.84-7.94) | | |
| Cancer of the lung | 3.35 (1.55-7.20) | | |
| Cancer of the breast | 0.69 (0.36-1.33) | | |
| Cancer – other sites | 1.34 (0.86-2.07) | | |
| Heart disease | 1.35 (0.78-2.32) | | |
| Respiratory diseases | 1.88 (0.79-4.51) | | |
| Alcohol-related deaths | 1.76 (0.95–3.25) | | |

CI, Confidence interval; ONS LS, Office for National Statistics Longitudinal Study.

^a Except for homicide, MRRs adjusted for age group (<25, 25–34, \geq 35 years) and decade as time-dependent variables; statistically significant effects highlighted in bold.

^b Homicide MRR unadjusted due to sparse event data; exact Poisson CI calculated.

Source: ONS LS.

was seen among women who became mothers as teenagers compared to women without children for each cohort. We also recalculated the MRRs for death at any age between 13 and 49 years among women who became teenage mothers, this time using firsttime motherhood at age ≥ 20 years as an alternative reference category (Table 5). For these analyses we observed significantly greater relative risks for death by suicide, accident, cancer at all sites, cervical cancer and lung cancer. The impact of using this different reference category on the observed relative risk was greatest in relation to accidental death; compared to women with no children there was no evidence of higher risk in the teenage mother group (MRR 1.07, 95% CI 0.60-1.94; Table 3), but compared to women who first became mothers at ≥ 20 years, a two- to threefold higher risk was seen (MRR 2.59, 95% C I -1.34 to 5.01; Table 5).

Finally, we adjusted the estimates for all deaths and for all natural and all unnatural deaths by Carstairs deprivation quintile, as at the 1971 Census. The quintiles showed a clear gradient of increasing prevalence of teenage motherhood by greater level of area-level deprivation, ranging from 6.0% of the total personyears denominator in the least deprived quintile to **Table 6.** Mortality rate ratios (MRRs) for teenage motherhood adjusted for ward-level

 Carstairs deprivation quintile at 1971 Census: 1970s ONS LS cohort

| MRR (95% CI) ^a | Adjusted MRR (95 % CI) ^b |
|---------------------------|--|
| | |
| 1.30 (1.08-1.57) | 1.29 (1.06–1.56) |
| 1.40 (0.97-2.02) | 1.41 (0.97–2.03) |
| | |
| 1.60 (1.09-2.34) | 1.58 (1.08-2.32) |
| 1.70 (0.95-3.04) | 1.69 (0.94–3.02) |
| | |
| 1.23 (0.99-1.53) | 1.21 (0.98-1.51) |
| 1.25 (0.78–2.01) | 1.27 (0.79–2.04) |
| | 1.30 (1.08–1.57) 1.40 (0.97–2.02) 1.60 (1.09–2.34) 1.70 (0.95–3.04) 1.23 (0.99–1.53) |

CI, Confidence interval; ONS LS, Office for National Statistics Longitudinal Study. ^a MRRs adjusted for age group (<25, 25–34, \geq 35 years) and decade as

time-dependent variables.

^b MRRs adjusted for age and decade and also for 1971 Carstairs deprivation quintile.

Source: ONS LS.

10.8% in the most deprived. However, when we fitted Carstairs quintile as a categorical variable in the Poisson models, the MRR estimates for effects linked with teenage motherhood were materially unaltered, as shown in Table 6. This was seen across the whole follow-up period, and also when we restricted the follow-up period to the 1970s and 1980s. In this restricted analysis, almost half of all unnatural deaths, 22.5% of all natural deaths and 27.8% of all deaths from any cause occurred on or before 31 December 1989.

Discussion

We present novel population-based evidence from England and Wales indicating that women who became mothers during their teen years in the 1970s were almost 30% more likely to die from any cause before reaching age 50 years than women without children, and were almost 60% more likely to die unnaturally. By contrast, mature first-time mothers seemed to be protected against premature death, and this was especially true for unnatural deaths and for those who became first-time mothers in their thirties. We observed a strong protective effect against unnatural death below 35 years of age and also at age 35-49 years among women who were beyond their teen years when they became mothers for the first time. Teenage motherhood was linked with a greater than twofold increased risk of suicide at any time before age 50, and elevated risks of fatal cervical and lung cancer were also found. Switching the reference category to first-time motherhood at age 20 years or older also revealed a significant elevation in risk of accidental death among women who became teenage mothers *versus* those who first became mothers at mature age. Restricting the analyses to death below 35 years indicated a threefold higher risk of suicide across all three subsequent cohorts of teenage mothers: 1970s, 1980s and 1990s. Finally, we found no evidence that higher risk of premature death linked with teenage motherhood was confounded by arealevel deprivation effects.

As the UK has particularly high rates of teenage motherhood, the ONS LS provides an ideal population base for examining adverse health outcomes in this group. The major strength of our study was that we could investigate vital event data in a representative sample of all teenage girls in England and Wales across three consecutive decades, with complete longterm follow-up of cause-specific premature mortality up to age 50 years without any significant selection or attrition biases. However, there were also some important limitations. First, sparse data restricted examination of the rarest cause-specific mortality outcome: homicide. Second, our measure of socio-economic status was area based, and thus subject to ecological error. It was also time-fixed at the 1971 Census, thereby introducing further misclassification for long-term follow-up. However, we sought to minimize the latter problem by restricting our adjusted analyses to the earlier period of follow-up (i.e. the 1970s and 1980s).

Third, estimation of reaching age 13 years was based on incrementing subjects' declared age at the preceding census. Thus, some girls will have entered our study up to almost 12 months after their thirteenth birthday should one of the ONS LS index dates fall in May. However, the degree of error introduced by this approximation is likely to be small and unsystematic, thereby creating an immaterial degree of random error with slight attenuation of the observed effect sizes, rather than a significant systematic bias (Copeland *et al.* 1977).

Our results generally concur with those reported by the only other population-based study, from the Swedish national registers (Otterblad-Olausson et al. 2004), which showed approximately twofold increased risks of suicide and accidental death for teenage mothers compared with women who first became mothers in their twenties, in addition to higher risks of fatal cervical and lung cancer. These investigators also reported an imprecise, but statistically significant, 10-fold increased risk of homicide. Our MRR estimates for homicide are also imprecise and, with such sparse event data, are not close to significance. Few other relevant reports have been published on this topic. A study in Georgia, USA, investigated risk of fatal injuries in mothers aged 15-19 years during the first postnatal year (Dietz et al. 1998). It reported a significantly higher risk of homicide (MRR 2.6) and a reduced risk of accidental motor vehicle deaths (MMR 0.4) in these teenagers in the postpartum period, compared with teenagers who were not mothers. A similar study, using national data for England and Wales during 1973-1984 (Appleby, 1991), showed a much reduced suicide risk for the first postnatal year among mothers aged 15-44 years [standardized mortality ratio (SMR) 0.17] compared with the female general population, with a somewhat weaker protective effect of recent motherhood seen at 15-19 years (SMR 0.36). Our findings indicate that this apparent postnatal protective effect of maternity does not persist in the longer term among teenage mothers. Having adjusted for age and decade at death, teenage mothers had a twofold higher risk of killing themselves before reaching their fiftieth birthday and a threefold higher risk of doing so before age 35, compared to women without children.

That we failed to show any confounding effect by area-level deprivation requires further comment. This was an unexpected finding given that fairly strong confounding of this association by individual-level socio-economic status has been reported from Sweden (Otterblad-Olausson *et al.* 2004). Furthermore, living in a deprived area has been shown to be a strong predictor of premature mortality in later years by studies conducted using the ONS LS over shorter follow-up

periods than in our investigation (Sloggett & Joshi, 1998; Norman et al. 2005). We were concerned that the 1971 Carstairs data were increasingly outdated when assessed across the whole follow-up period up to the end of 2006. However, our separate adjusted analysis, with follow-up restricted to the end of 1989, also indicated no evidence of any confounding effect. This indicates that deprivation measured at ward level does not in fact confound this particular association. Recent evidence from Gloucestershire, England, shows that women's perceptions of their own neighbourhood more strongly predict incidence of teenage motherhood than ward-level deprivation measures (Johns, 2010). Applying ward-level indices of deprivation may not accurately reflect the degree of social disadvantage experienced by individual women, especially if teenage mothers are highly concentrated in small pockets of deprivation that lie within ward populations that are not deprived as a whole. This notion concurs with earlier research on deprivation and premature mortality conducted by Sloggett & Joshi (1994). They reported that area-level deprivation effects were entirely explained by individual-level variables, with disadvantaged individuals seeming to receive no protection from risk of earlier death by living in areas that were generally not deprived. Thus, we conclude that socio-economic status measured at the individual level should be assessed in any future investigations this topic.

The long-term effects of teenage motherhood on elevated mortality risk have also been shown in a comparative study of the populations of England and Wales and Austria (Dolhammer, 2000), indicating a significantly higher risk of death from all causes at 50-85 years in England and Wales (MRR 1.26) and at 50-94 years in Austria (MRR 1.09). In the same report, having no children was associated with significantly higher risk than being a mother (England and Wales: MRR 1.15; Austria: MRR 1.15). Results reported from a similar study of the England and Wales population alone agreed with those from our study by indicating a protective effect of motherhood per se (Grundy & Tomassini, 2005). Our findings are novel in showing an even more pronounced protective effects against unnatural compared with natural deaths, and for motherhood first experienced at age 30 years and older compared with at 20-29 years. Evidence from the USA suggests that a mother experiencing suicidal ideation may be protected from making suicidal acts by three types of 'child-related concerns' (Linehan et al. 1983): (i) that suicide would directly harm her children; (ii) that it would be wrong to leave her children in someone else's care; and (iii) a desire to see her children grow up. It seems plausible that such influences would also confer a lower prevalence of risk-taking behaviour among mothers, thereby reducing their risk of premature accidental death.

Why such protective influences fail in the context of teenage motherhood is yet to be understood. Increased risk for these outcomes is likely to be determined largely by a high prevalence of psychopathology and related psychosocial factors. For example, analysis of data from the 1958 British Birth Cohort revealed an increased risk of psychiatric morbidity in adulthood among women who became mothers as teenagers (Maughan & Lindelow, 1997). However, it is unclear whether or not these factors tend to occur as a direct consequence of the economic and social difficulties frequently encountered following childbirth at young age, including lone parenting, restricted educational and career opportunities, subsequent low income and other forms of social isolation and exclusion (Hobcroft & Kiernan, 2001; Otterblad-Olausson et al. 2001). Evidence from the UK, the USA, Australia and New Zealand indicates that girls with antecedent mental illness (Kovacs et al. 1994; Kessler et al. 1997) or socioeconomic problems (Kiernan, 1997; Maughan & Lindelow, 1997; Williams et al. 1997; Quinlivan et al. 2004) are much more likely to become teenage mothers than their peers. However, we do not know whether these same women would have had a similarly high risk of unnatural premature death if they had not become mothers in their teen years. In other words, are these women at high risk directly because of their young maternal age, or is poor outcome merely a reflection of their own family background predisposing them to experiencing teenage motherhood? Studies of this nature have been conducted among children of teenage mothers (Geronimus et al. 1994; Turely, 2003), but the outcomes of teenage mothers themselves are yet to be examined in this way. The varied mechanisms that explain higher risk of premature unnatural death in these women may act conversely to protect women who first experience motherhood at mature age. Irrespective of their age on becoming mothers, lone mothers have been shown to have much higher rates of psychiatric illness according to national survey or registry data from New Zealand, Sweden, the UK and Australia (Sarfati & Scott, 2001; Ringbäck Weitoft et al. 2002; Targosz et al. 2003; Butterworth, 2004). The overlap in social adversity factors between lone and teen mothers may therefore explain some of the elevated risk of premature death that we found in women who became mothers as teenagers.

Higher risks of fatal lung and cervical cancer among those who experienced teenage motherhood, and the lower risks seen in first-time mothers at mature age, are likely to be explained by variation in prevalence of risk factors specific to these cancer types. Thus, smoking is widely known to be the leading cause of

lung cancer, and teenage mothers have high baseline rates of smoking and low rates of quitting subsequently, according to longitudinal evidence from New Zealand (McGhee & Williams, 1996). Smoking also predicts cervical cancer, and the other main risk factors, including poor uptake for screening, age at first intercourse and number of sexual partners, may also have high prevalence among teenage mothers, given that young maternal age at first birth predicts higher risk of disease onset (Bjørge & Øystein, 1996). Even after childbirth, the greater levels of psychosocial adversity that teenage mothers generally encounter may make it harder for them to modify unhealthy lifestyles to improve their chances of experiencing better physical health outcomes in later life. This could explain why the protective effects we observe in the majority of women who become mothers seem not to operate among those who become mothers as teenagers. It was perhaps surprising that we did not find a significantly increased risk of death due to alcoholrelated deaths. Studying these deaths using routinely recorded mortality data is problematic because of inconsistency of coding and probable under-reporting, and thus a high level of misclassification and subsequent attenuation of effect size is plausible (Breakwell et al. 2007).

This is the first UK longitudinal study of premature death up to age 50 years in mothers who had their first child as a teenager. The psychosocial needs of this vulnerable group require greater attention from policymakers, commissioners and health service planners. Mental health and primary care clinicians, in addition to public health and social services department, need to fully recognize the risk of health inequality that teenage motherhood presents. The difficulties these women encounter, and their associated health risks, persist well beyond the years when they experience being a teenage mother. Before costly preventive strategies can be developed at the population level, further research is needed to identify the complex and time-dependent causal mechanisms that explain these poor outcomes. In particular, it will be important to examine these outcomes among the sisters of teenage mothers, thereby adjusting for antecedent factors in the family and its environment. It is clear that we need to understand the degree to which these poor health outcomes are explained by teenage childbearing per se, as opposed to wider family background influences.

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Declaration of Interest

None.

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