What’s so bad about teenage pregnancy?

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Summary
A systematic literature review identified the most frequently cited medical consequences of teenage pregnancy as anaemia, pregnancy-induced hypertension, low birth weight, prematurity, intra-uterine growth retardation and neonatal mortality. Critical appraisal suggested that increased risks of these outcomes were predominantly caused by the social, economic, and behavioural factors that predispose some young women to pregnancy. Maternal age less than 16 years was associated with a modest (1.2-2.7 fold) increase in prematurity, low birth weight and neonatal death.

Key Words
anaemia, education, low birth weight, neonatal mortality, pregnancy-in-adolescence, pregnancy-induced hypertension, premature birth, risk factor, small for gestational age, socio-economic status, teenage pregnancy

Key message points
- Teenage pregnancy is associated with poor health for both mother and child.
- Teenage pregnancy is associated with social, economic and behavioural risk factors, which are also independent risk factors for adverse outcomes of pregnancy.
- Maternal age less than 16 years is independently associated with a 1.2-2.7 fold increase in prematurity, low birth weight and neonatal death.
- Most teenage pregnancies are low risk.

Introduction
Teenage pregnancy rates in the UK are the highest in Western Europe, and are increasing. In 1996 there were 63 pregnancies per thousand women under the age of 20, and 9.4 per thousand under the age of 16.2 Teenage pregnancy is viewed as a major social and medical problem, and is a priority target for the Government’s Social Exclusion Unit to tackle. It has been claimed that teenage mothers are more likely to suffer many adverse medical outcomes, both during and after pregnancy. These include hypertensive disorders, anaemia, cephalopelvic disproportion, increased rates of caesarean section and postnatal depression. It has also been claimed that their children are at increased risk of prematurity, intra-uterine growth retardation, congenital malformations, and death in the neonatal, post-neonatal, and infant periods. Some authors consider that it is young age itself that is responsible for the adverse outcomes associated with teenage pregnancy. However, teenage pregnancy is strongly associated with a large number of social, economic, educational and behavioural factors.

The complex interaction of these effects on teenage pregnancy is still poorly understood. Furthermore, these socioeconomic and behavioural factors associated with becoming pregnant as a teenager, are independently associated with adverse obstetric and neonatal outcomes. The relative importance of young age and these other factors in determining the outcome of teenage pregnancy has not been elucidated. These interrelationships are important for improving the health of young women and their children. Interventions aimed at reducing teenage pregnancy rates may have very little effect on the frequency of adverse medical outcomes in this population if the socio-economic and behavioural factors are not addressed.

Method
The literature on teenage pregnancy and its outcomes was reviewed. An electronic database search using Medline with the thesaurus term ‘pregnancy-in-adolescence’, and Embase using the term ‘(ado* or teen*) and (moth* or preg*)’ identified potentially relevant papers. The search was restricted to English language publications in the last 20 years. References within these publications and those recommended by experts in the field were also retrieved. Studies in a developed world setting were reviewed, including only those investigating pregnancy in women under 20 years of age, and measuring any of the following outcomes: maternal anaemia, pregnancy induced hypertension (PIH), low birth weight (LBW), prematurity, small for gestational age (SGA), and neonatal mortality. These terms were not rigidly defined at the outset to allow inclusion of the maximum number of studies. Figure 1 is a flow diagram illustrating the process of literature review.

Studies were objectively assessed using published criteria. The same set of publications was then reviewed to determine the effect of other variables on the outcomes listed above. A hierarchy of risk factors for each adverse outcome in teenagers was developed using the odds ratios (ORs) from the original studies. For uncommon events, such as the outcomes assessed here, odds ratios approximate to relative risks.

Results
Only 11 out of 5977 potentially relevant publications were considered to contain valid data for comparison (Tables 1 and 2). Only one of these studies was set in the UK. One of the studies was a meta-analysis and the others used cohort designs. Some of these studies included pre-teens (<13 years old) in their populations; here they will be considered as young teenagers. Most studies presented their results by sub-groups of age, making direct comparison difficult. Based on unadjusted figures for the study
populations, rates of neonatal mortality, premature birth, LBW, SGA and anaemia were higher in teenagers than in older women in the majority of studies. For example, Fraser et al. found LBW in 7% of teenage pregnancies, prematurity in 10% and SGA in 14%, compared with 4%, 5% and 10%, respectively, in adults. Olaussen et al. found neonatal mortality rate was 14.5 per thousand for babies of 13-15 year olds compared to 4.6 for babies of 20-24 year olds. The rate of anaemia was reported to be up to 35% in young teenagers compared with 13.8% in the adult control group. The rate of PIH was similar: 8.5% in the young teenagers compared with 7.9% in adults.

Another major problem was incorporating terminations and stillbirths. Most studies used a retrospective cohort technique, starting with the birth records of women and working backwards to look at recorded complications. If older women in poor socio-economic circumstances terminated pregnancy more often than teenagers, the adverse effect of socio-economic variables on the adult cohort would be reduced. No studies managed to control for factors such as whether pregnancies were ‘intended’ or ‘wanted’, which may be major determinants of maternal behaviour during pregnancy.

The most consistent risk factor for adverse outcome was the adequacy of antenatal care. This probably does not reflect the fact that antenatal care is particularly beneficial, but rather that those who entered late into antenatal care were those most disadvantaged in other ways. It may include those who did not realise, or sought to hide, the fact that they were pregnant and behaved more dangerously during pregnancy. Socio-economic status, maternal smoking during pregnancy, and black ethnicity were other consistent risk factors. Maternal educational attainment, body mass index at conception and metropolitan residence were weakly associated with adverse outcomes.

**Discussion**

The selection of outcomes used in this essay was based on those that were commonly reported, objective, important in terms of health, and defined in a way that was consistent between studies. Caesarean section rates and other obstetric interventions were commonly considered in publications on teenage pregnancy. However, no publications cited explicit indications for performing these interventions. Foetal malformations were too rare to allow adequate comparison. Postnatal depression, survival and development of the child, future socio-economic prospects for the mother and future health of the mother were not considered. The potential benefits of teenage pregnancy, such as the protective effect of early childbearing on breast cancer, were not considered.

No irrefutable conclusions can be drawn from studies of teenage pregnancy. There are numerous known confounding factors clouding the issue of the biological effect of young age. Statistical modelling and multiple regression analysis allow each factor to be tested for its effect on outcomes independent of other variables. Statistical methods can only control for known confounders. The results must be used with caution because the more factors analysed independently, the smaller the sub-groups on which the conclusion is based. It is not feasible to obtain sufficient numbers to perform such an analysis looking for small differences in rare outcomes. For the purposes of convenience, surrogate data such as payment status (Medicaid vs. insurance) in place of socio-economic status are used, because they are routinely available for large numbers of subjects. This may hide an effect of very low social class, or may introduce additional confounders such as eligibility for health insurance. More precise clinical data collected by hand searching of clinical notes or laboratory testing (e.g. presence of sexually transmitted infection) were only available in small studies.

Most studies had serious methodological flaws. Gestational age was frequently based solely on the last menstrual period (LMP), but it is well recognised that this is an unreliable method in teenagers because of a higher frequency of early gestational bleeding and the irregularity of menstruation shortly after menarche. It may lead to up to 50% of births being misdated in teenagers compared with 25% in adults. Few studies used additional ultrasound or newborn assessment.

Another major problem was incorporating terminations and stillbirths. Most studies used a retrospective cohort technique, starting with the birth records of women and working backwards to look at recorded complications. If older women in poor socio-economic circumstances terminated pregnancy more often than teenagers, the adverse effect of socio-economic variables on the adult cohort would be reduced. No studies managed to control for factors such as whether pregnancies were ‘intended’ or ‘wanted’, which may be major determinants of maternal behaviour during pregnancy.

The most consistent risk factor for adverse outcome was the adequacy of antenatal care. This probably does not reflect the fact that antenatal care is particularly beneficial, but rather that those who entered late into antenatal care were those most disadvantaged in other ways. It may include those who did not realise, or sought to hide, the fact that they were pregnant and behaved more dangerously during pregnancy. Socio-economic status, maternal smoking during pregnancy, and black ethnicity were other consistent risk factors. Maternal educational attainment, body mass index at conception and metropolitan residence were weakly associated with adverse outcomes.

**Premature birth**

Prematurity (birth at less than 37 completed weeks of gestation) is associated with respiratory distress syndrome, retinopathy and increased neonatal mortality rate. After adjustment for confounding factors (Table 3), four studies found that premature birth was more common in teenagers and five found that it was not significantly different from the adult controls. Most studies failed to control for many of the important confounding factors. Educational attainment
was used as a surrogate for socio-economic status in two of the largest studies, but it is a poor substitute because teenage girls are most likely to be prevented from achieving their full educational potential by pregnancy. Notwithstanding this criticism, the three largest studies all supported the increased risk of premature birth in young teenagers, and the studies disagreeing were not powerful enough to detect a small excess risk of prematurity after controlling for all the factors they assessed. This conclusion is supported by the greater risk of prematurity seen in younger teenagers compared with older teenagers in a ‘dose-response’ relationship. For 18-19 year olds there is probably no greater risk than in 20-24 year olds, but those under 17 years old are 1.2-1.5 times more likely to have a premature baby.

**Small for gestational age (SGA)**
This is a marker of intra-uterine growth retardation (IUGR), and is usually defined as birth weight below the tenth centile for gestational age. It is important because it is associated with medical problems and poor survival. The neonate is at risk of hypothermia, hypoglycaemia, hypocalcaemia and polycythaemia. Perinatal mortality increases exponentially as infant birth weight falls through each centile. After adjustment, one study reported a decrease in the number of SGA babies born to teenage mothers, four reported that there was no significant difference, and one reported more than in adults. The risk factors for SGA (Table 3) were different to those for the other outcomes, the most important being more biological than social. Prior LBW or premature birth was the most

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**Table 2** Studies considered valid for comparison

<table>
<thead>
<tr>
<th>Study</th>
<th>Setting</th>
<th>Age</th>
<th>Adjustments*</th>
<th>Prem.</th>
<th>SGA</th>
<th>LBW</th>
<th>Anaem</th>
<th>PIH</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scholl et al.</td>
<td>USA, n=887</td>
<td>12–15</td>
<td>Eth, SE, BMI, Wt, Sm, Dr.</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>–</td>
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<td>16–19</td>
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<tr>
<td>Bai et al.</td>
<td>Australia, n=7191</td>
<td>&lt;18</td>
<td>Eth, Se, Sm, Ma.</td>
<td>NS</td>
<td>–</td>
<td>NS</td>
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<tr>
<td>Amini et al.</td>
<td>USA, n=69096</td>
<td>12–15</td>
<td>Eth, SE, Pa, Yr, Ma</td>
<td>1.2 (1.07–1.4)</td>
<td>0.63</td>
<td>1.3 (1.0–1.76)</td>
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<td>NS</td>
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<td>16–19</td>
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<td>(0.83–0.93)</td>
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<tr>
<td>Scholl et al.</td>
<td>Meta-analysis</td>
<td>≤19</td>
<td>Various, Yr. and Set.</td>
<td>1.46 (1.2–1.77)</td>
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<td>NS</td>
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<tr>
<td>Olausson et al.</td>
<td>Sweden, n=320174</td>
<td>13–15</td>
<td>Yr. and Ed.</td>
<td>–</td>
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<td>2.7 (1.5–4.8)</td>
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<td>16–17</td>
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<td>1.4 (1.1–1.8)</td>
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<tr>
<td>Zuckerman et al.</td>
<td>USA, n=698</td>
<td>13–18</td>
<td>BMI, Pa, Eth, ANC, Sm, Ed, Alc, Dr.</td>
<td>NS</td>
<td>NS</td>
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<td>Sawchuck et al.</td>
<td>Gibraltar, n=295</td>
<td>≤19</td>
<td>Ma, SE</td>
<td>Excluded</td>
<td>–</td>
<td>NS</td>
<td>–</td>
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<tr>
<td>Fraser et al.</td>
<td>USA, n=134088</td>
<td>13–17</td>
<td>ANC, Ed, Ma</td>
<td>1.5 (1.0–2.2)</td>
<td>1.4 (1.0–2.0)</td>
<td>2.0 (1.2–3.1)</td>
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<td>18–19</td>
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<td>Cooper et al.</td>
<td>USA, n=127668</td>
<td>10–12</td>
<td>Eth, Ma, ANC, Pa Ed, Res.</td>
<td>1.5 (1.26–1.78)</td>
<td>1.72 (1.44–2.06)</td>
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<td>1.85 (1.16–2.94)</td>
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<td>1.36 (1.27–1.46)</td>
<td>1.32 (1.22–1.43)</td>
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<td>1.87 (1.54–2.28)</td>
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<td>1.16 (1.12–1.20)</td>
<td>1.06 (1.01–1.11)</td>
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<td>1.17 (1.03–1.33)</td>
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<tr>
<td>Konje et al.</td>
<td>UK, n= 5236</td>
<td>11–16</td>
<td>SE, Yr.</td>
<td>–</td>
<td>–</td>
<td>2.53 (2.19–2.92)</td>
<td>1.69 (1.28–2.4)</td>
<td>NS</td>
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<td>20–24</td>
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<tr>
<td>Berenson et al.</td>
<td>USA, n=551</td>
<td>12–15</td>
<td>Res, ANC, SE, Sm, Dr, STD, PNO</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>3.7 (p&lt;0.01)</td>
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<td>16–17</td>
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*Eth= ethnicity, SE = Socio-economic status, BMI = pre-pregnancy body mass index, Wt. = Weight gain during pregnancy, Sm = smoking, Dr = Illicit drug use in pregnancy, Ma = Marital status, Pa = Parity, Yr = Year of delivery, Set = Setting (developed world vs. developing), Ed = Educational attainment, ANC = adequacy of antenatal care, Alc = Alcohol, Res = Place of residence, STD = Sexually transmitted disease, PNO= Perinatal outcome. Odds Ratios (95% confidence intervals) are compared with the oldest age group in each study. NS = No significant difference.†This study only compared young teenagers with older teenagers.
imported, although most studies considered only primigravid women so this was not relevant. Smoking, ethnicity and low body mass at conception were important, but antenatal care was not so important. Cooper et al. failed to find a ‘dose response effect’ for young age on SGA. Amini et al. actually found that the youngest teenagers appeared less likely to deliver SGA babies than older teenagers and both were less likely than adults (OR 1.33 (1.23–1.50). In the former three studies, ORs for extremely low birth weight infants (below 1000 g) were consistently higher in younger infants (below 1000 g) compared to 20-22 year olds. Berenson et al. found a haematocrit less than 30 g/dl more frequently in 12-15 year olds compared to 20-22 year olds. These studies used identical definitions for anaemia in their population of 10-16 year olds than in adults. Berenson et al. found increased neonatal mortality. Olausson et al. compared predominantly older groups of teenagers with adults. None of the studies controlled for pre-pregnancy BMI, weight gain during pregnancy or smoking status, and these figures are not adjusted for gestational age. Higher rates of LBW in teenagers are compatible with the observation for SGA and prematurity. SGA is not more common but prematurity is, and it is prematurity that accounts for the excess of LBW.

Anaemia
Anaemia was badly assessed in the studies that indicated their criteria for measuring it.Konje et al. found that anaemia (Hb < 10.5 g/dl) was 2.53 (2.19-2.92) times more common in their population of 10-16 year olds than in adults. Berenson et al. found a haematocrit less than 30 g/dl more frequently in 12-15 year olds compared to 20-22 year olds. These studies used identical definitions for anaemia in young girls and adult women of different ethnicities, even though there are marked differences in the normal values in these groups. The meta-analysis by Scholl et al. compared predominantly older groups of teenagers with adults, and it showed no significant difference between teenagers and adults.

Pregnancy induced hypertension (PIH)
This complication of pregnancy is a predictor of developing pre-eclampsia in adults. Konje et al. found that the risk of PIH was 1.7 times higher in nulliparous 10-16 year olds compared with socio-economic class matched nulliparous controls, but there was no increase in the frequency of pre-eclampsia or proteinuric disorders. For their study, PIH was defined as a blood pressure of at least 90 mmHg diastolic and 140 mmHg systolic on more than one occasion. The blood pressure reference range in non-pregnant teenagers is lower than in adults. However, the effect of going to antenatal clinic as a young pregnant teenager may be sufficiently stressful to account for the elevation in blood pressure. ‘White coat hypertension’ is a well-described phenomenon. A study set in an environment designed to be supportive of teenage mothers (and less intimidating) found no increase in hypertension. There is no evidence that an increase in PIH predicts an increase in pre-eclampsia in teenagers.

Neonatal mortality
See Table 5. Two studies found no significant differences, and two found increased neonatal mortality. Olausson et al.

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**Table 3 Factors** *associated with premature birth† and SGA‡*

<table>
<thead>
<tr>
<th>Factor</th>
<th>Odds ratio (OR)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inadequate antenatal care</td>
<td>2.2 (2.1–2.3)†³⁷</td>
</tr>
<tr>
<td>Low socio-economic status</td>
<td>1.83 (1.57–2.14)†³⁷</td>
</tr>
<tr>
<td>Ethnicity (Black)</td>
<td>1.70 (1.64–1.76)⁴⁷</td>
</tr>
<tr>
<td>Smoking</td>
<td>1.46 (1.21–1.76)‡⁷</td>
</tr>
<tr>
<td>Maternal BMI at conception</td>
<td>1.41 (1.19–1.67)‡⁷</td>
</tr>
<tr>
<td>Low educational attainment</td>
<td>1.09 (1.05–1.13)‡⁷</td>
</tr>
<tr>
<td>Marital status (single)</td>
<td>1.15 (1.08–1.22)‡⁷</td>
</tr>
<tr>
<td>Low BMI at conception</td>
<td>1.20 (1.04–1.38)‡⁷</td>
</tr>
</tbody>
</table>

**SGA**
- Prior premature or LBW 10.58 (3.89–28.77)†³⁴
- Low BMI at conception 5.74 (2.18–15.08)†³⁴
- Smoking 3.07 (1.79–6.37)‡⁷
- Ethnicity (Black) 1.44 (1.37–1.52)‡⁷
- Maternal BMI at conception 1.42 (1.29–1.58)‡⁷
- Inadequate antenatal care 1.31 (1.23–1.40)‡⁷
- Low educational attainment 1.33 (1.18–1.49)‡⁷

**Table 4 Factors** *associated with low birth weight (LBW)†* 7

<table>
<thead>
<tr>
<th>Factor</th>
<th>Odds ratio</th>
</tr>
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<tbody>
<tr>
<td>Inadequate antenatal care</td>
<td>1.74 (1.65–1.83)‡³⁷</td>
</tr>
<tr>
<td>Low socio-economic status</td>
<td>1.72 (1.57–1.87)‡³⁷</td>
</tr>
<tr>
<td>Ethnicity (Black)</td>
<td>1.3 (1.0–1.7)‡⁷</td>
</tr>
<tr>
<td>Smoking</td>
<td>1.39 (1.23–1.59)‡³⁷</td>
</tr>
<tr>
<td>Maternal BMI at conception</td>
<td>1.44 (1.38–1.51)‡³⁷</td>
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<tr>
<td>Parity (multiparous)</td>
<td>1.33 (1.23–1.44)‡³⁷</td>
</tr>
<tr>
<td>Low educational attainment</td>
<td>1.3 (1–1.7)⁵⁵</td>
</tr>
<tr>
<td>Low birth weight</td>
<td>1.31 (1.19–1.45)‡³⁷</td>
</tr>
</tbody>
</table>

*In order of decreasing odds ratio. The actual ORs for each risk factor are not directly comparable; they were derived from multiple studies and no two studies included the same sets of variables in their analysis. Information on each confounder was not available for every outcome. † Birth weight < 2500g.
‡ < 10th centile for age specific weight.

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**Low birth weight (LBW)**
See Table 4. Three studies found an increase in the frequency of the more heterogeneous outcome of LBW (encompassing premature and growth-retarded babies) in teenagers, and four found no significant difference. In the former three studies, ORs for extremely low birth weight infants (below 1000 g) were consistently higher in younger than older teenagers and the odds ratios for LBW were only significant in the youngest teenagers compared to adults. These results may represent a real effect of young age because they arise from the largest studies where there were sufficient numbers of very young teenagers to allow quantification of their risks. The studies finding no difference may have lacked sufficient power to detect a small difference in the youngest teenagers.

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**Neonatal mortality**
See Table 5. Two studies found no significant differences, and two found increased neonatal mortality.
found young teenagers at increased risk of neonatal mortality but no significant difference for 18-19 year olds.27 This was almost entirely accounted for by the increased frequency of very pre-term (less than 32 weeks) births in this group. However, they only adjusted for year of delivery and educational attainment and so this provides scant evidence for the biological effect of young age upon neonatal mortality or pre-term birth. After adjustment for rather more factors, Cooper et al17 found that neonatal mortality was almost twice as common in 10-13 year olds compared with 14-15 year olds. This ‘dose-response’ effect of young age is more convincing evidence of a biological effect.

Table 5 Factors* associated with neonatal mortality†

<table>
<thead>
<tr>
<th>Factor</th>
<th>Odds ratio</th>
</tr>
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<tbody>
<tr>
<td>Prematurity</td>
<td>(not stated)</td>
</tr>
<tr>
<td>Inadequate antenatal care</td>
<td>2.70 (2.33–3.14)</td>
</tr>
<tr>
<td>Metropolitan residence</td>
<td>1.16 (1.02–1.32)</td>
</tr>
</tbody>
</table>

*In order of decreasing odds ratio. The actual ORs for each risk factor are not directly comparable; they were derived from multiple studies and no two studies included the same sets of variables in their analysis.

† Information on each confounder was not available for every outcome. 0–27 days after birth.

Conclusion
Studies of the outcomes of teenage pregnancy appear contradictory, but careful analysis reveals that they are mostly compatible, or at least differences are explicable. One problem is that the studies have often grouped ages differently making their direct comparison difficult. Another problem has been methodological over-sights, such as dating of pregnancy and definition of anaemia, that have stacked the odds against teenagers before any data are even collected. Where adverse effects of teenage pregnancy exist, they are more prominent in the younger teenagers (as would be expected) and studies that have grouped teenagers too broadly may have missed these effects with the dilution of the older teenagers who differ very little from adults. Before 16 years of age there does appear to be a very real association of teenage pregnancy with prematurity. The very premature babies account for most of the increase in low birth weight and neonatal mortality.27 The risks associated with young age (ORs ranging from 1.2-2.7) are modest compared to those for the social, behavioural and economic risk factors.

Teenagers more often go into premature labour with intact membranes than do adults,54 indicating that the underlying mechanisms may be different to those in adults. Whether this is a biological phenomenon or due to other factors predisposing to teenage pregnancy is still unclear. It makes little biological sense for young women to be able to reproduce at an age that puts their children at risk. There is now strong evidence that very premature birth (22-32 weeks) and prematurity (32-37 weeks) birth have similar risk factors, but the strength of association of social factors with very premature birth is stronger.21 Thus the most socially disadvantaged, who become pregnant youngest,32 are at disproportionately high risk of very premature babies. These very young teenagers may also be a special group: victims of abuse, prostitution or mental illness.1,2 Biological factors like low gynaecological age, low BMI at the start of pregnancy and poor weight gain during pregnancy predominate in this group. Their effect on the whole teenage group may bias results considerably when other, often older, teenagers in fact do well compared to adults. The statistical analyses used in these studies do not look to whether such a small group explains most of the adverse outcomes by itself.

A high-risk group of teenagers may be the most difficult to reach with interventions to reduce the adverse effects of teenage pregnancy.55 This would explain the results of the one trial of out-reach prenatal care, which failed to reduce rates of prematurity.33 Studies looking at primary prevention have proven that they can reduce the number of pregnant teenagers, but have not yet assessed the effect on outcomes of pregnancy in their population.33 There is scope for further research, controlling for all the important confounding factors highlighted in this discussion, to elucidate whether pregnancy at a young age really does constitute a medical problem. Research into the mechanism of premature labour in teenagers would be valuable. More importantly, trials assessing initiatives to reduce teenage pregnancy rates should also assess their impact on the outcomes of teenage pregnancy. It may be that whilst education-based programs55 achieve reduction in the absolute numbers of pregnant teenagers within an area, it is only case-management programs56 targeting the highest risk teenagers that achieve an important reduction in adverse outcomes. Future research should also look at how to change the circumstances of those who are most disadvantaged, and how this changes their risk of poor outcomes.

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References


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