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Adult outcomes of being born late preterm or early term – what do we know?

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SUMMARY

The literature on adult outcomes of people born late preterm (LPT, 34–36 completed weeks) or early term (ET, 37–38 weeks) was reviewed. In PubMed, 9547 articles were identified; 53 were eligible. Of these, 12 were based on clinical cohorts, 32 on medical birth register linkages, and nine on historical birth cohorts; 48 out of 53 on Nordic countries; 50 out of 53 reported on LPT and eight out of 53 reported on ET. LPT plus ET have increased early (<45 years) adult all-cause mortality. Despite increased cardiometabolic risk factors and slightly lower cardiorespiratory fitness in LPT, no studies showed increased risk for coronary heart disease, some showed increased risk for stroke, and all showed increased risk for type 2 diabetes. Most show increased risk for asthma and decreased allergic rhinitis. LPT have slightly lower cognitive abilities and higher rates of several mental disorders; ET have intermediate values. LPT and ET adults have slightly lower education, occupational status, and income. We recommend that authors report findings of LPT/ET separately from those born more preterm.

Keywords:
Preterm
Cardiovascular
Pulmonary
Physical activity
Neurocognitive
Psychiatric
Socio-economic

1. Introduction

The long-term outcomes of late preterm (LPT; generally defined as birth between 34 and 36 completed postmenstrual weeks, that is up to 36 weeks and six days) or early term birth (ET; between 37 and 38 completed weeks) have recently raised much interest. This interest comes from two directions. First, neonatal follow-up programs that have extended to adult life raise the question whether and to what extent findings characteristic of adults born smallest and most immature are present in the much larger groups of adults born LPT or ET. Second, traditional life-course studies have used low birth weight as a marker of early adversity. From this perspective, it is natural to ask to what extent the findings are a consequence of preterm or ET birth and to what extent a consequence of slow fetal growth, both of which can result in low birth weight.

Neonatal follow-up programs are generally based in high-income countries and often run by neonatologists and allied clinical professionals. They originate from the rapid developments in neonatal intensive care from the 1970s onwards, which have substantially increased survival of those born very preterm (VPT; <32 weeks) or at very low birth weight (VLBW; <1500 g). The first infants who experienced these improvements are soon entering middle age. Research shows that most of them are healthy and live normal lives, but on average they are characterized by a number of risk factors. These include higher levels of risk factors for cardiometabolic disease, lower pulmonary airflow, lower cognitive abilities, and a behavioral phenotype characterized by inattention and difficulties in social relationships. These findings are summarized in a number of recent reviews [1–8].

Many of the early epidemiological studies on what today is known as the ‘developmental origins of health and disease theory’ started from describing the association of birth weight with a range of adult outcomes [9]. Low birth weight was largely perceived as a proxy of slow fetal growth. Determination of gestational age by last menstrual period was originally considered too inaccurate and thus received little attention. This paralleled the longstanding focus of the World Health Organization on low birth weight as a perinatal
indicator, only in the 2000s emphasizing the distinction of preterm birth, small for gestational age, or a combination thereof [10].

From both perspectives, it is clear that little is known about these outcomes in adults who were born LPT or ET. While individual risks are greater in adults born VPT/VLBW, even lesser risks in the much larger number of LPT/ET adults may cause a substantial public health burden.

Our aim was to review the current literature on adult outcomes of LPT/ET infants. We chose to focus on key areas where previous studies suggest increased risks in VP/VLBW: adult mortality, cardiometabolic disease and risk factors, pulmonary and atopic outcomes, physical activity and fitness, cognitive functioning, mental health, and socio-economic outcomes such as education and occupation.

2. Methods

Comprehensive literature searches were carried out by one author (S.S.K.) in Medline database, using the search engine PubMed, between April 27th and May 7th, 2018. The search produced 9547 articles. Searches were performed using combinations of terms describing preterm birth and various health and social outcomes (see below).

S.S.K. also conducted the initial screening of the titles, using the following inclusion criteria: the exposure was gestational age in categories of late preterm (34–36 completed weeks) and early term (37–38 completed weeks) (also papers including moderately preterm – starting from 32+0 – were accepted if they could not be separated from the late preterm group); the outcome was examined among adults (mean age ≥18 years); and the outcome was either mortality, cardiometabolic, pulmonary, allergy/atopy, physical activity or fitness, cognitive function, mental health, or socio-economic outcomes. The studies also had to have used quantitative analysis methods, providing estimates for (longitudinal) associations between the exposure and outcomes of interest. No study population size restrictions were applied. Included studies had to be published as original research articles with full text available. No language or publication year restrictions were used. If more than one study reported the same findings on the same cohort, only the study including the primary publication was included in the review.

Next, two other authors (E.K. and P.H.) assessed the 113 remaining articles for eligibility, systematically the abstracts and, if necessary, full texts against the above criteria. Six articles were also added based on screening the reference lists of the included articles. In the end, 53 original articles were selected for the final review. The selected studies were published between 1998 and 2018. For more detail, see the PRISMA flow chart in Fig. 1.
Key characteristics of the studies selected for the review, as well as available structured data on outcomes associated with later preterm and early term birth, were extracted from the studies and entered into literature tables by two authors (E.K. and P.H.). A qualitative synthesis of the included studies was performed.

Search terms (asterisk used for truncated terms):
- Exposure: premature birth, premature infant, born premature, preterm, early term
- In all searches: adult*
- Outcomes:
  - Mortality: adult mortality, adult death, trends in mortality, all-cause mortality
  - Cardiometabolic: cardiovasc*, cardiometab*, stroke, diabetes, coronary heart disease, metabolic syndrome, hypertension, glucose
  - Pulmonary: pulmonar*, lung, asthma, airways
  - Allergy/atopy: allergy, allergic, atopy, atopic
  - Physical activity and fitness: physical activity, fitness*, exercise
  - Cognitive function: IQ, intelligence, learning, executive function, neurocogn*, attention, memory, processing speed
  - Mental health: psychopathology, psychiatric, mental health
  - Socio-economic outcomes: education*, occupation*, socio-economic*, unemploy*, employ*

3. Results

3.1. Mortality

Four register studies on adult mortality up to 45 years fulfilled the inclusion criteria (Table 1).

A Western Australian study assessed all-cause mortality between 6 and 30 years of age. Hazard ratios (HRs) were 1.4 (95% confidence interval (CI): 1.0, 2.0) for those born at 32–34 weeks, 1.1 (0.9, 1.4) for those born at 35–36 weeks and 1.0 (0.9, 1.1) for those born ET [11].

Two Swedish studies had maximum follow-up until 36 years of age. One reported HR of 1.43 (1.24, 1.64) for adult all-cause mortality for LPT compared with those born at 37–41 weeks. Cause-specific mortality was only assessed with gestational age as continuous variable [12]. The other study compared ET with those born at 39–41 weeks. HR for all-cause mortality was 1.20 (1.10, 1.30) with multiple contributing causes of death [13].

A Norwegian study with maximum follow-up until 45 years showed a hazard ratio for all-cause mortality of 1.11 (1.02, 1.20) for those born LPT [14]. In analyses restricted
to outcome-discordant maternal sib-pairs, this association was not sustained. Neither was there any association with cause-specific mortality.

3.2. Cardiometabolic outcomes

Three clinical cohort studies fulfilled inclusion criteria, all based on the ESTER Study in Northern Finland (Table 2). Young adults born LPT had 2.5-fold odds for metabolic syndrome and higher odds for obesity, hypertension and fatty liver biochemical index. Regarding components of metabolic syndrome, LPT adults had higher body mass index (BMI), waist circumference, percentage body fat, and higher insulin, transaminase and uric acid concentrations, in part mediated through higher adult BMI [15].

For office and 24 h mean systolic pressures, the differences of 1.7 and 2.7 mmHg did not reach statistical significance [15,16]. No difference was seen in nutrient intake and healthy nutrition index in adults born LPT and at term [17].

Eleven studies that assessed outcomes through healthcare registers fulfilled search criteria (Table 2). Six of them were based on Nordic Medical Birth Registers on births from 1973 onwards, and five on Nordic birth cohorts that have retrospectively collected early-life records of people born between the 1910s and 1940s.

Two of the medical birth register studies assessed hypertension, based on blood pressure measurements in military conscripts or purchases of antihypertensive medication: both reported odds ratios (ORs) of 1.2 or 1.3. A further two assessed diabetes (mostly type 1) by purchase of medication, with odds ratios at 1.2 to 1.4 [18–21]. One Swedish study with maximal follow-up to 38 years assessed cerebrovascular or ischaemic heart disease as outcomes and showed no differences in those born at 32–37 weeks and those born at term. Another Swedish study showed increased rates of venous thromboembolism, in particular pulmonary embolism [23].

As many cardiometabolic disease end-points occur later in life, birth cohorts that have retrospectively collected pregnancy records provide valuable information. The Helsinki Birth Cohort of 20,345 people born in one of two public delivery hospitals in Helsinki between 1924 and 1944 includes data on last menstrual period; the cohort includes only survivors (people alive in Finland in 1971), of whom 5.3% were born LPT and 0.7% before 34 weeks. In that cohort, people born LPT had no increased risk of coronary heart disease or stroke [29]. In a subset born between 1934 and 1944, those born before 35 weeks have an increased risk of type 2 diabetes [24].

A Swedish cohort focused on preterm birth and low birth weight by including all people born at <35 weeks or 2100 g (boys) or 2000 g (girls) in four delivery hospitals and a
random sample of all other births in these hospitals as reference; however, the analyses used the standard 37-week cut-off to define preterm birth, with further subgroupings. In that cohort, no increased risk for coronary heart disease [25] or hypertension [26] in hospital discharge registers is observed. For diabetes, those born between 33 and 36 weeks had HR of 1.29 (1.05, 1.58) [27].

The Uppsala 1915–1929 birth cohort includes data on last menstrual period: compared with those born at term, those born at <35 weeks were more likely to die from stroke but not from CHD [28].

No study compared adults born early term with the remaining adults born at term.

3.3. Pulmonary and atopic outcomes

The search produced one clinical cohort study, based on 31-year assessment of Northern Finland Birth Cohort 1966. That study showed similar rates of asthma history and lower rates of atopy (skin prick tests) in those born at <35 weeks compared with those born at 39–40 weeks [30].

Seven studies based on Nordic Medical Birth Registries fulfilled search criteria (Table 3). Of these, six assessed asthma. Two studies using conscript examination showed no increased risk of asthma [31,35]. Three used purchases of asthma medication as an outcome. A Norwegian and a Danish study showed ORs or risk ratios of 1.1 to 1.4, whereas a Swedish study showed no association, with a narrow 95% CI of 0.97 (0.90, 1.04) [19,32,36]. Another Norwegian study assessed basic or attendance benefit, indicating only severe cases. OR for LPT asthma for the 32–36 weeks group was 1.69 (1.56, 1.82) [34].

As to allergic rhinitis, a Swedish army recruit study found a decreased risk for those born at 33–36 weeks and another Swedish study showed that those born at 35–36 weeks had less purchases of physician-prescribed nasal corticosteroids [31,33]. For atopic dermatitis, no difference was seen in the Norwegian attendance benefit or Danish conscript studies [35,34].

One study from the Swedish 1925–1949 Birth Cohort found that those born at 33–36 weeks had increased rates of hospital diagnosis of asthma. This association was due to higher rates among women, who also had higher rates of any obstructive airways disease diagnosis [37].

No study compared adults born early term with the remaining adults born at term.
3.4. Physical activity and fitness

Two register studies, both on fitness, fulfilled the inclusion criteria (Table 4) [42,43]. The largest study was based on Conscript Register cardiorespiratory fitness measurement for 218,820 men [43,44]. Maximal load on cycle ergometer was 302 W (standard deviation (SD): 49 for those born at 32–36 weeks) and 307 W (SD: 50) for the term-born group). The difference corresponded to 0.1 SD. The other study included 396 participants and used data from a national system for systematic monitoring of physical growth, exercise capacity and agility (the SLOfit) [42]. The study reported no significant differences between those born at 32–37 weeks and full-term group, except that moderately preterm boys had poorer trunk strength (unadjusted analyses).

Four clinical cohort studies of young adults from two birth cohorts of Northern Ireland and Northern Finland were identified. In one of the three studies from the Finnish ESTER Preterm Birth Study, LPT-born adults had lower muscular fitness (performed fewer modified push-ups) than term-born controls, but there were no differences in cardiorespiratory fitness, measured by submaximal step test [41]. The two other studies from the same cohort reported no differences in leisure-time physical activity measured by self-report or objectively measured physical activity and sedentary time [39,40]. The study from the Northern Ireland birth cohort assessed cardiorespiratory fitness longitudinally from adolescence to young adulthood in 791 term-born participants [38]. Those born ET had a risk ratio of 1.57 (95% CI: 1.14–2.16) for poor cardiorespiratory fitness compared with individuals born at 39–42 weeks [38].

3.5. Cognitive function and intellectual disability

The search criteria were fulfilled by two birth cohort studies and four medical birth register studies (Table 5).

Among young adults of the Arvo Ylppö Longitudinal Study, those born LPT had 3.71 IQ points (SD: 0.25) lower full IQ estimate. This was largely due to lower scores among those born LPT small for gestational age. There was no difference in tests measuring executive functioning, attention, and memory [45].

A study in the Helsinki 1934–1944 Birth Cohort showed no difference in a CERAD-NB neuropsychological test among the whole cohort. However, among those who had attained less than tertiary adult education, those born LPT had a 2.7-fold odds for mild cognitive impairment [46].

Studies using military conscript data are based on Swedish data and partly overlapping cohorts. One study used those born at 39–41 weeks as controls and reported
mean differences (converted from stanine to SD scores) of 0.15 SD for those born 33–34 weeks, 0.11 SD for those born 35–36 weeks, and 0.04 SD for those born ET [47]. Another study compared those born at 32–36 weeks to those born at term; mean difference corresponded to 0.12 SD, and OR of subnormal performance (stanine score 1–3) was 1.26 [48]. The third study focused on associations between intellectual ability and cardiorespiratory fitness; however, it reported an OR for above-average score of 0.94 [44].

According to a Norwegian register study, those born late preterm had a 1.6-fold risk of mental retardation compared with those born at term [49].

3.6. Mental health

One clinical birth cohort study, nine medical birth register studies, and one historical cohort study fulfilled the criteria (Table 6).

Young adults of the Arvo Ylppö Longitudinal Study underwent structured interview (M-CIDI) to assess common mental disorders. Odds ratio for any common mental disorder in those born LPT was 1.11 (0.67, 1.84) [50].

Three Swedish register studies used hospital discharge register diagnoses as a main source in partly overlapping populations. The most comprehensive of these was based on more than three million people born in Sweden from 1973 onwards; this population included all participants in the two other studies. Those born late preterm had an HR of ~1.3 for psychotic disorders and, assessed up to 19 years, 1.3 for autism-spectrum disorders and 1.4 for attention deficit/hyperactivity disorder (ADHD). These HRs were sustained also in comparisons within maternal siblings [53].

Lindström et al. also assessed early term birth as an exposure. It was associated with slightly increased risks of any psychiatric (HR: 1.1), psychotic (1.2), neuropsychiatric (1.4) and mood disorders (1.1), suicide attempt (1.1), and any addictive disorder. These HRs were lower than those for preterm birth [51].

A study based on the Danish Central Psychiatric Research Register showed rate ratios of 1.25 for all psychiatric diseases for those born at 33–34 weeks and 1.19 for those born at 35–36 weeks [54]. A Norwegian study assessed outcomes severely affecting working capacity from the National Insurance Scheme and found relative risk of 1.3 (1.0, 1.7) for schizophrenia but no increased risk for autism spectrum disorders (0.8; 0.4, 1.4); however, 0.05% of individuals had such a diagnosis [54].

Four studies assessed medication as an outcome. A Western Australian retrospective case–control study identified people who used stimulant medication for ADHD and controls who did not, and linked data with birth registry data. ORs for stimulant
medication for those born at 33–36 weeks were 1.16 for men and 1.18 for women; for those born at 37–38 weeks, they were 1.12 and 1.14 [55]. Another study on entitlement for stimulant treatment was based on Norwegian data. Those born at 33–36 weeks had a relative risk of 1.4 and those at 37–38 weeks 1.2 [57]. Further, one study based on Swedish and another on Norwegian prescription database assessed a range of medications (Table 6) [19,56].

A study in the Helsinki 1934–1944 Birth Cohort found no difference in inpatient treatment on a range of psychiatric diagnoses between those born late preterm and those born at term, except that among men the rate of suicides was two-fold [58].

3.7. Socio-economic outcomes

Two clinical cohort studies, four medical birth registry studies and one retrospective birth cohort study assessed socio-economic outcomes (Table 7).

In a Danish birth cohort at 31–32 years, those born between 32 and 37 weeks reported similar education, but they were less likely to have upper-level socio-economic position. A US birth cohort study included no numerical LPT–control comparison (Table 7).

The medical birth register studies used varying exposures and outcomes. A Swedish study compared those born at 33–36 weeks and those born ET with controls born at 39–41 weeks: percentages of post-secondary education, assessed at 23–29 years, were 35.5%, 38.2%, and 39.8% and of employment 72.5%, 72.7%, and 74.1%. Both exposure groups, students excluded, had lower net salary and disposable income than controls; differences in disposable income were larger, indicating lower transfer from society [61]. A Norwegian study at 28–37 years showed approximately five percentage points lower in rates of low or of graduate education [62]. Another Norwegian study at maximum 36 years compared LPT to term controls. Corresponding differences were approximately three percentage points [49]. In both studies, adjustment for socio-economic indicators attenuated the result to one-third or one-half. A study with Swedish data showed similar or slightly smaller differences; however, most differences attenuated to null when comparing preterm-born with maternal siblings [53].

In the Helsinki 1934–1944 Birth Cohort, those born LPT were more likely to end up in a manual profession, have lower education and less income. These results obtained despite adjustment for parental socio-economic position [63].

4. Discussion

Altogether 53 publications fulfilled the criteria of our search. We have compared the publications outcome by outcome in the results section. In the discussion, we focus on methodological issues and limitations plus implications of the findings.
Of the 53 reviewed publications, 48 were based on Nordic populations; the remaining five were from Slovenia, Northern Ireland, Australia, and USA. No study was from low- or middle-income countries. The Nordic region, with its 0.35% share of the world’s population and 0.20% share of births, thus has a disproportionate share in the evidence on adult outcomes of people born LPT or ET.

The dominance of the Nordic region in publication has several consequences. In a worldwide scale, they represent low preterm birth frequency countries. Accordingly, among referred register studies using standard definitions, rates of LPT are 3.3% (singleton alive at 1 year) and 3.9% (all alive at 15 years) [12,14]. ET frequency is at 13.8% (of liveborn singletons) [13]. Nordic countries are high-income societies characterized by low levels of inequality and universal healthcare including free-of-charge antenatal and child healthcare and inclusive education. These characteristics may be thought to reduce the consequences of LPT or ET births and thus the results may represent conservative estimates.

In the evaluation of abstract and full text papers, the most common reason for exclusion was not meeting the gestational age criteria. Many of these papers included all infants born preterm in the same exposure group. This makes it impossible to distinguish to what extent the findings represent those born LPT or ET. Thus, the findings of these now-excluded studies could be explained by those born very preterm, who, in population-based samples, constitute a small group that is generally expected to have larger effects. Further, some papers used gestational age as a continuous exposure variable, again leaving the contribution of those born LPT or ET unknown. In studies reporting on samples including all degrees or prematurity, power allowing, we recommend authors to report findings of individuals born LPT or ET separately from those born more preterm. Findings in these groups may have grossly different implications than in those born very preterm.

Because of the wide variation in exposure group definitions, we relaxed our gestational age criteria to include those born moderately preterm, from 32 weeks onwards, if they could not be separated from the LPT group; otherwise we would have needed to exclude much essential literature. This also leaves the possibility that the findings could be attributable to those born at 32–33 weeks rather than those born LPT. However, comparisons of effect sizes in studies using LPT only and studies using LPT extended to the 32 weeks group are generally consistent with a linear dose–response relationship between gestational age at birth and many of the outcomes.

We do not have space to discuss the individual outcomes in detail. By and large, the results are consistent with what is known of “adult preterm phenotype” from studies of
adults born very preterm or with very low birth weight. This includes higher all-cause mortality for several causes of death, higher levels of cardiometabolic risk factors (although evidence of manifest cardiometabolic disease, with the exception of type 2 diabetes, remains uncertain), probably higher rates of asthma and lower rates of atopic disease, lower physical fitness (with little or no evidence of lower physical activity), lower cognitive abilities, higher rates of several mental health disorders, and slightly lower educational attainment, occupational status, and income. For many outcomes, there seems to be a dose–response relationship with earlier gestational age at birth. Accordingly, in LPT and ET adults, whereas the increases in risk remain small on an individual level, in these large groups they may result in relatively high population-attributable fractions. In addition, as discussed above, most studies come from Nordic welfare societies likely to buffer the effects of LPT/ET birth and may thus represent conservative estimates. Also of note, because of space limitations, some relevant outcomes could not be included, such as starting a family and reproduction.

As to clinical implications, our results highlight the long-term importance of LPT/ET birth on life-course health. Although it may be too early for concrete implications in pre- and neonatal care, information on perinatal events such as gestational age or birth weight should be included when obtaining a full medical history in adult patients.

For future studies it would be important to diversify the populations studied and especially to study LPT/ET outcomes in low- and middle-income settings. Many longitudinal studies obtain data on gestational age so that in many cases an additional analysis of LPT/ET individuals would be sufficient. However, Nordic countries are overwhelmingly represented for a reason: the possibility for register linkage in these countries creates unique possibilities for further study, for example, to identify additional risk and protective factors. Moreover, differences in seemingly similar outcome between the Nordic countries, such as those for all-cause mortality, call for comparisons between Nordic countries.

**Practice points**

- Adults born late preterm or early term may be at an increased risk of common non-communicable and mental health disorders, have on average lower cognitive abilities, and attain slightly lower socio-economic position than those born at term.
- Many of the risks are relatively small on an individual level, but because late preterm and early term birth is common, they may bring about a significant population-attributable risk.
- For diseases that manifest later in life, such as cardiovascular disease, evidence is scanty and inconsistent.
A full medical history of adults should include perinatal factors such as gestational age at birth.

Research directions
- Low- and middle-income settings.
- Risk of manifest late-life disorders including cardiometabolic and other non-communicable disease.
- Risk and protective factors.
- Follow-up studies that include adults born across the range of gestational ages should, power allowing, report separately findings for the large groups of individuals born late preterm or early term.

Conflict of interest statement
None declared.

Funding sources
None.

References


<table>
<thead>
<tr>
<th>First author [ref.]</th>
<th>Setting</th>
<th>Design</th>
<th>Exposure group(s)</th>
<th>Controls</th>
<th>Years of birth, percent of men</th>
<th>Mean age at outcome assessment or end of follow-up</th>
<th>Main outcome(s)</th>
<th>Main statistical method</th>
<th>Adjustments</th>
<th>Main finding/conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Srinivasjois [11]</td>
<td>Western Australia Register</td>
<td>32–34 weeks, n = 9066, 35–36 weeks, n = 26,070, 37–38 weeks, n = 174,146</td>
<td>39–41, n = 412,882</td>
<td>1980–2010, 50.9%</td>
<td>6–30 years</td>
<td>All-cause mortality 6–30 years</td>
<td>Log binomial regression</td>
<td>Sex\textsuperscript{b}, race\textsuperscript{b}, decade, pregnancy conditions, parity, SES</td>
<td>HR: 32–34 weeks: 1.4 (1.0, 2.0) 35–36 weeks: 1.1 (0.9, 1.4) 37–38 weeks: 1.0 (0.9, 1.1)</td>
<td></td>
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<tr>
<td>Crump [12]</td>
<td>Sweden Register</td>
<td>34–36, n = 22,590, 37–42, n = 626,723</td>
<td>37–42, n = 626,723</td>
<td>1973–1979, 51.4%</td>
<td>End 2008</td>
<td>All-cause mortality 18–36 years</td>
<td>Cox regression</td>
<td>Birth year, sex, birth order, BWSDS, SES</td>
<td>HR: all-cause 1.43 (1.24, 1.64)</td>
<td></td>
</tr>
<tr>
<td>Crump [13]</td>
<td>Sweden Register</td>
<td>37–38, n = 93,645, 39–42, n = 536,617</td>
<td>39–42, n = 536,617</td>
<td>1973–1979, 51.2%</td>
<td>End 2008</td>
<td>All-cause and cause-specific mortality 18–36 years</td>
<td>Cox regression</td>
<td>Birth year, sex, birth order, SES</td>
<td>HR: All-cause 1.20 (1.10, 1.30) Anomalies 2.43 (1.49, 3.95) Endocrine 2.07 (1.30, 3.29) Respiratory 1.70 (0.89, 3.23) Cardiovascular 1.40 (1.02, 1.93) Neurological 1.27 (0.83, 1.94)</td>
<td></td>
</tr>
</tbody>
</table>
Cancer 1.20 (0.95, 1.51)
External 1.12 (1.01, 1.24)

Risnes [14] Norway Register 34–36, n = 61,082
51.6%

All-cause Cox
Cancer Cardiovascular
HR: All-cause 1.11 (1.02, 1.20)
External 1.09 (0.99, 1.43)
Cancer 0.86 (0.66, 1.13)
Analyses within discordant maternal sibpairs (n = 29,536):
no association
HR:

BWSDS, birth weight SD score; SES, socio-economic status; HR, hazard ratio.

Numbers refer to gestational age in completed postmenstrual weeks and to the number of participants. Only exposure groups relevant to adult outcomes of late preterm or early preterm are listed. An exception are exposure groups extending down to 32 weeks which have been included in the table.

Minimal adjustments.

Additional adjustments.
**Table 2. Cardiometabolic outcomes.**

<table>
<thead>
<tr>
<th>First author [ref.]</th>
<th>Setting</th>
<th>Design</th>
<th>Exposure group(s), completed weeks</th>
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</thead>
<tbody>
<tr>
<td>Sipola-Leppänen [15]</td>
<td>Northern Finland</td>
<td>Birth cohort, clinical follow-up</td>
<td>LPT, ( n = 242 )</td>
<td>( \geq 37 ) weeks, ( n = 344 )</td>
<td>1985–1989, 49.1%</td>
<td>23.4 years</td>
<td>Metabolic syndrome and its components</td>
<td>Logistic and linear regression</td>
<td>Age(^b), sex(^b), source cohort(^b); SES(^c), pregnancy conditions(^c), BWSDS(^c), parental cardiometabolic disease(^c), adult lifestyle(^c)</td>
<td>Odds ratios: metabolic sdr 2.5 (1.2, 5.3) fatty liver 8.6 (1.0, 72.8) mean differences BMI 2.9 (0.1, 5.8) waist 3.3 cm (1.3, 5.3) lean mass 1.3 kg (–0.9, 3.5) percentage fat 8.0% (2.4, 13.8). Higher fasting insulin, ALT, AST, uric acid, no difference in glucose, CRP or other markers</td>
</tr>
</tbody>
</table>
| Sipola-Leppänen [16] | Northern Finland | Birth cohort, clinical follow-up | LPT, \( n = 72 \) | \( \geq 37 \) weeks, \( n = 103 \) | 1985–1989, 44.6% | 23.2 years | Ambulatory blood pressure | Linear regression | Age\(^b\), sex\(^b\), sleep assessment method\(^b\); child SES\(^c\), pregnancy conditions\(^c\), BWSDS\(^c\), adult | Mean differences: systolic mean 2.7 (–0.5, 5.8) diastolic mean 0.9 (–1.3, 3.1) systolic variability 0.5 (–0.3, 1.4) diastolic variability 0.8 (0.1,
Matinolli [17] Northern Finland and Uusimaa, Finland
Birth cohort, clinical follow-up
LPT, \( n = 352 \) and \( n = 631 \), \( \geq 37 \) weeks, 1985–1989, 47.6% of \( n = 631 \) with \( \geq 37 \) weeks, 24.2 years
Recommended diet index from food frequency questionnaire
Linear regression analyses by sex\(^b\), adjusted for age\(^b\), source cohort\(^b\), energy intake\(^b\), SES, pregnancy conditions\(^c\), BWSDS, adult lifestyle\(^c\)
Mean differences in recommended diet index: women 0.06 (–0.48, 0.60), men –0.04 (–0.62, 0.54)

Register studies
Johansson [18] Sweden, conscripts Register
33–36 weeks, \( n = 12,660 \), \( \geq 37 – 41 \) weeks, \( n = 275,895 \), 1973–1981, 100%
High systolic (\( \geq 140 \) mmHg) or diastolic (\( \geq 90 \) mmHg) pressure
Logistic regression Age\(^c\), BWSDS, parity\(^c\), child SES\(^c\), current body size\(^c\)
High systolic: 1.21 (1.16, 1.26), high diastolic: 1.25 (1.02, 1.53)

Engeland [19] Norway Register
32–24 weeks, \( n = 4887 \), \( \geq 37 \) weeks, \( n = 431,914 \), 1974–1984, 56.5% of \( n = 431,914 \), \( \geq 37 \) weeks, 30.5 years
Purchase of medication at least twice between 30\(^{th}\) and 31\(^{st}\) years
Logistic regression SES\(^b\)
RR for 32–34 and 35–36 weeks:
Insulin, women 1.6 (1.0–2.5), 1.2 (0.9, 1.7); insulin, men 1.3 (0.9, 1.9), 1.4 (1.1, 1.7); Other diabetes medication,
<table>
<thead>
<tr>
<th>Study</th>
<th>Country</th>
<th>Register</th>
<th>Gestation Period</th>
<th>Duration</th>
<th>Method of Analysis</th>
<th>Covariates</th>
<th>OR for any diabetes medication</th>
<th>OR for insulin without oral diabetes medication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crump [20]</td>
<td>Sweden</td>
<td>Register</td>
<td>35–36 weeks, n = 19,025; 35–36 weeks, n = 583,571</td>
<td>1973–1979, N/A to end 2009</td>
<td>Logistic regression</td>
<td>Age, sex, pregnancy conditions, child SES, maternal diabetes, BWSDS</td>
<td>1.22 (1.08, 1.38)</td>
<td>1.25 (1.08, 1.45)</td>
</tr>
<tr>
<td>Crump [21]</td>
<td>Sweden</td>
<td>Register</td>
<td>33–34 weeks, n = 5685; 35–36 weeks, n = 589,573</td>
<td>1973–1979, N/A to end 2009</td>
<td>Logistic regression</td>
<td>Age, sex, pregnancy conditions, child SES, maternal antihypertensive medication, BWSDS</td>
<td>1.33 (1.15, 1.42)</td>
<td>1.28 (1.15, 1.25)</td>
</tr>
<tr>
<td>Ueda [22]</td>
<td>Sweden</td>
<td>Register</td>
<td>32–36, n = 62,725; 37–41, n = 1,057,240</td>
<td>1983–1995, 51.4% to end 2010</td>
<td>Cox regression</td>
<td>Stratified by sex, adjusted for maternal characteristics</td>
<td>Ischemic heart disease or cerebrovascular disease from Cerebrovasc 1.01 (0.75, 1.35)</td>
<td>Ischemic heart 1.43 (0.81, 2.52)</td>
</tr>
</tbody>
</table>
hospital discharge and death registers

Zöller [23] Sweden Register LPT, \( n = 153,296 \)
37–41, \( n = 3,066,290 \)
1973–2008, End 2010 Venous Cox regression thomboembolism from hospital discharge and outpatient registers

37–41, \( n = 3,066,290 \) 51.4%

End 2010 Venous Cox regression thomboembolism from hospital discharge and outpatient registers

Kajantie [24] Born in two delivery units in Helsinki, Finland Birth cohort, register follow-up <35 weeks, \( n = 247 \)
37–41 weeks, \( n = 10,711 \) 1934–1944, 52.1%
End 2002 Special Logistic regression reimbursement for diabetes medication granted after 40 y

<Kaijser [25] Born in four delivery units in Stockholm, Uppsala Birth cohort, register follow-up 33–36 weeks, \( n = 1945^d \)
37–42 weeks, \( n = 3221 \) 1925–1949, N/A End 2002 Ischaemic heart disease diagnoses from hospital discharge Cox regression

HR for VTE ≥18 years, 1.24 (1.10, 1.40)
Pulmonary embolism 1.29 (1.04, 1.59)
Deep vein thrombosis 1.15 (0.98, 1.34)
Other VTE 1.12 (0.88, 1.44)

Logistic regression

Year of birth, sex, firstborn, SES, BWSDS

OR for diabetes

<35 weeks: 1.68 (1.06, 2.65)
35–36 weeks: 0.65 (0.41, 1.05)

HR 0.96 (0.80, 1.16)
<table>
<thead>
<tr>
<th>Study</th>
<th>Location</th>
<th>Birth Cohort</th>
<th>Follow-up Period</th>
<th>Year of Birth</th>
<th>Sex</th>
<th>Hypertension Diagnosis</th>
<th>Cox Regression</th>
<th>Stratified For</th>
<th>Hazard Ratios</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bonamy [26]</td>
<td>and Sundsvall, Sweden</td>
<td>33–34, ( n = 1555 )</td>
<td>35–36, ( n = 3174 )</td>
<td>1925–1949</td>
<td>End 2006</td>
<td>Hypertension in hospital discharge register</td>
<td>Cox regression</td>
<td>Sex and year of birth, BWSDS</td>
<td>33–34 weeks: 1.32 (0.87, 1.99); 35–36 weeks: 1.23 (0.83, 1.83)</td>
</tr>
<tr>
<td>Kajser [27]</td>
<td>and Sundsvall, Sweden</td>
<td>33–36 weeks, ( n = 1945 )</td>
<td>37–42 weeks, ( n = 3221 )</td>
<td>1925–1949</td>
<td>End 2006</td>
<td>Diabetes diagnoses from Hospital Discharge Register</td>
<td>Cox regression</td>
<td>Year of birth, sex, SES</td>
<td>HR 1.29 (1.05, 1.58). Stronger when lower BWSDS</td>
</tr>
<tr>
<td>Koupil [28]</td>
<td>Uppsala, Sweden</td>
<td>See Results</td>
<td>Total ( n = 11,474 ) (group ( n = 1915–1929, \ N/A ))</td>
<td>1915–1929</td>
<td>End 2001</td>
<td>Death from ischaemic heart disease or stroke</td>
<td>Cox regression</td>
<td>Age, year of birth, sex, SES</td>
<td>30–35 weeks: reference; 36–37 weeks: CHD 0.92 (0.66, 1.28), stroke 0.72 (0.43, 1.20); 38–39 weeks: CHD 0.94 (0.70, 1.26), stroke 0.62 (0.40, 0.97)</td>
</tr>
</tbody>
</table>
Kajantie [23] Born in two delivery units in Helsinki, Finland
Birth cohort, LPT, $n =$ 1006
Birth weight ≥37 weeks, $n =$ 17,972
1924–1944, End 2010
≥37 weeks, Var 1924–1944, $n =$ 1006

Coronary heart disease and stroke
Cox regression
Stratified by sex$^b$, year of birth$^b$, adjusted for sex$^c$, BWSDS$^c$
Hazard ratios:

- Coronary: 0.99 (0.85, 1.14)
- Stroke: 0.86 (0.71, 1.06)

BWSDS, birth weight SD score; SES, socio-economic status.

$^a$Numbers refer to gestational age in completed postmenstrual weeks and to the number of participants. Only exposure groups relevant to adult outcomes of late preterm or early preterm are listed. An exception are exposure groups extending down to 32 weeks which have been included in the table.

$^b$Minimal adjustments.

$^c$Additional adjustments.

$^d$The cohort was originally recruited by selecting individuals born at <35 weeks or with a birth weight of ≤2000 g in girls and ≤2100 g in boys and as a reference population a random sample of all remaining births.
### Table 3. Pulmonary and atopic outcomes.

<table>
<thead>
<tr>
<th>First author [ref.]</th>
<th>Setting</th>
<th>Design (register, clinical cohort, outcome case–control)</th>
<th>Exposure group(s), completed weeks</th>
<th>Controls, completed weeks</th>
<th>Years of birth, percent of men</th>
<th>Mean age at outcome assessment or end of follow-up</th>
<th>Main outcome(s)</th>
<th>Main statistical method</th>
<th>Adjustments</th>
<th>Main finding/conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical cohort study</strong></td>
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<tr>
<td>Pekkanen [30]</td>
<td>Northern Finland</td>
<td>Clinical birth cohort</td>
<td>≤35, n = 229</td>
<td>1966, 50.2%</td>
<td>31 years</td>
<td>Atopy (positive skin prick), history of doctor-diagnosed asthma</td>
<td>Logistic regression</td>
<td>Sex¹, pregnancy conditions², parental allergy³, current BMI³, SES³</td>
<td>≤35 weeks: reference</td>
<td>36–38 weeks: atopy 1.22 (0.87, 1.70), asthma 0.90 (0.54, 1.49) 39–40 weeks: atopy 1.42 (1.02, 1.98), asthma 0.81 (0.49, 1.33)</td>
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<td></td>
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<td>36–38, n = 1303</td>
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<td>39–40, n = 2435</td>
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<td><strong>Register studies</strong></td>
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<tr>
<td>Bråbäck [31]</td>
<td>Sweden, army recruits</td>
<td>Register</td>
<td>33–36, n = 6607</td>
<td>1973–1975, 100% years</td>
<td>Allergic rhinitis or asthma by physician’s examination</td>
<td>Logistic regression</td>
<td>Sex¹, pregnancy conditions², older siblings³</td>
<td>Allergic rhinitis 0.85 (0.78, 0.93), asthma + rhinitis 1.16 (0.96, 1.39), asthma without rhinitis 1.06 (0.93, 1.21)</td>
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<td>n = 119,506</td>
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<td></td>
<td>≥37 weeks, n = 4887</td>
<td>1974–1984, 56.5% years</td>
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<td>Purchase of medication at least twice</td>
<td>Logistic regression</td>
<td>SES³</td>
<td>RR for 32–34 and 35–36. Anti-asthmatics, women 1.4 (1.1, 1.8), 1.1 (0.9, 1.3), men 1.3</td>
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<td>35–36, n = 431,914</td>
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<tr>
<td>Study</td>
<td>Country</td>
<td>Register</td>
<td>Birth Cohort</td>
<td>Year of Follow-Up</td>
<td>Analysis</td>
<td>Exposure</td>
<td>Predictors</td>
<td>OR (95% CI)</td>
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<tr>
<td>Damgaard [32]</td>
<td>Denmark</td>
<td>Register</td>
<td>32–36, n = 31,958; 37–45, n = 733,787</td>
<td>1980–1993&lt;sup&gt;4&lt;/sup&gt;, 2010–2011</td>
<td>Logistic regression</td>
<td>Purchase of prescribed asthma medication</td>
<td>Sex&lt;sup&gt;c&lt;/sup&gt;, pregnancy conditions&lt;sup&gt;c&lt;/sup&gt;, older siblings&lt;sup&gt;c&lt;/sup&gt;, SES, maternal asthma medication&lt;sup&gt;c&lt;/sup&gt;, neonatal respiratory morbidity&lt;sup&gt;c&lt;/sup&gt;</td>
<td>(1.0, 1.6), 1.2 (1.1–1.4)</td>
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<tr>
<td>Crump [33]</td>
<td>Sweden</td>
<td>Register</td>
<td>35–36, n = 19,025; 37–42 weeks, n = 583,571</td>
<td>1973–1979, Jul 2005 to Dec 2009</td>
<td>Logistic regression</td>
<td>Purchases of prescribed nasal corticosteroids or oral antihistamines</td>
<td>Age&lt;sup&gt;c&lt;/sup&gt;, sex&lt;sup&gt;c&lt;/sup&gt;, BWSDS&lt;sup&gt;c&lt;/sup&gt;, pregnancy conditions&lt;sup&gt;c&lt;/sup&gt;, SES&lt;sup&gt;c&lt;/sup&gt;, total medication prescriptions&lt;sup&gt;c&lt;/sup&gt;, maternal glucocorticoid/antihistamine use&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Nasal corticosteroids OR 0.94 (0.91, 0.98); Oral antihistamines OR 1.01 (0.98, 1.05)</td>
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<tr>
<td>Trønnes [34]</td>
<td>Norway</td>
<td>Register</td>
<td>32–36, n = 82,377; 37–41, n = 1439–790</td>
<td>1967–2001, End 2005</td>
<td>Logistic regression</td>
<td>Basic or attendance benefit or severe</td>
<td>Year of birth, pregnancy conditions&lt;sup&gt;c&lt;/sup&gt;, parity&lt;sup&gt;c&lt;/sup&gt;, parental asthma history&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Or for asthma 1.69 (1.56, 1.82), for atopic dermatitis 0.69 (0.90, 1.01)</td>
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<tr>
<td>Study</td>
<td>Country</td>
<td>Data Source</td>
<td>Gestational Age</td>
<td>Sample Size</td>
<td>Exposures</td>
<td>Outcomes</td>
<td>Methods</td>
<td>Additional Information</td>
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<tr>
<td>Broström [37]</td>
<td>Sweden</td>
<td>Birth cohort, register follow-up</td>
<td>33–36, n = 1945; 37–42, n = 3221</td>
<td>1925–1949, 51.3%</td>
<td>Date of birth, sex, BWSDS, SES, maternal history</td>
<td>Asthma or COPD from hospital discharge or death register</td>
<td>Cox regression</td>
<td>End 2006</td>
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</tr>
</tbody>
</table>

BWSDS, birth weight SD score; COPD, chronic obstructive pulmonary disease; SES, socio-economic status.

Numbers refer to gestational age in completed postmenstrual weeks and to the number of participants. Only exposure groups relevant to adult outcomes of late preterm or early preterm are listed. An exception are exposure groups extending down to 32 weeks which have been included in the table.
Minimal adjustments.

Additional adjustments.

An additional inclusion criterion was birth weight ≤2000 g in girls and ≤2100 g in boys.
<table>
<thead>
<tr>
<th>Citation</th>
<th>Setting</th>
<th>Design</th>
<th>Exposure group(s), completed weeks&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Controls, completed weeks</th>
<th>Years of birth, percent of men</th>
<th>Mean age at outcome assessment or end of follow-up</th>
<th>Main outcome(s)</th>
<th>Main statistical method</th>
<th>Adjustments</th>
<th>Main finding/conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical cohort studies</td>
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<tr>
<td>Ferreira [38]</td>
<td>Northern Ireland</td>
<td>Birth cohort study, clinical follow-up</td>
<td>37–38, 39–40</td>
<td>Since 1971, 48.1%</td>
<td>22 years</td>
<td>Cardiorespiratory fitness, submaximal cycle ergometry</td>
<td>Logistic and linear regression</td>
<td>Age&lt;sup&gt;b&lt;/sup&gt;, sex&lt;sup&gt;b&lt;/sup&gt;, cohort&lt;sup&gt;b&lt;/sup&gt;, BWSDS&lt;sup&gt;c&lt;/sup&gt;, pregnancy conditions&lt;sup&gt;c&lt;/sup&gt;, body size and composition&lt;sup&gt;c&lt;/sup&gt;</td>
<td>RR for poor fitness 1.57 (1.14, 1.26), mean increase in fitness per week 0.46 mL/kg/min (0.14, 0.79)</td>
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<tr>
<td>Tikkanmäki [39]</td>
<td>Northern Finland</td>
<td>Birth cohort study, clinical follow-up</td>
<td>34–36, n = 210, ≥37, n = 311</td>
<td>1985–1989, 48.4%</td>
<td>23.4 years</td>
<td>Self-reported leisure time physical activity (detailed 12 m questionnaires)</td>
<td>Linear and logistic regression</td>
<td>Age&lt;sup&gt;b&lt;/sup&gt;, sex&lt;sup&gt;b&lt;/sup&gt;, source cohort&lt;sup&gt;b&lt;/sup&gt;, pregnancy conditions&lt;sup&gt;c&lt;/sup&gt;, BWSDS&lt;sup&gt;c&lt;/sup&gt;, SES&lt;sup&gt;c&lt;/sup&gt;, current body size&lt;sup&gt;c&lt;/sup&gt;, smoking&lt;sup&gt;c&lt;/sup&gt;, asthma&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Mean difference in total volume of PA –6.5 MET/h/weeks (–19.8, 9.1). No difference in conditioning, non-conditioning, commuting or vigorous PA</td>
<td></td>
</tr>
<tr>
<td>Tikkanmäki [40]</td>
<td>Northern Finland</td>
<td>Birth cohort study,</td>
<td>34–36, n = 108, ≥37, n = 178</td>
<td>1985–1989, 43.4%</td>
<td>23.3 years</td>
<td>Physical activity by linear regression</td>
<td></td>
<td></td>
<td>Mean difference in total daily PA (accelerometer counts/min)</td>
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<tr>
<td>Study</td>
<td>Country</td>
<td>Cohort Details</td>
<td>Follow-up Period</td>
<td>Sample Size</td>
<td>Fitness Measures</td>
<td>Analysis Methods</td>
<td>Results</td>
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<tr>
<td>Tikannäki [41]</td>
<td>Northern Finland</td>
<td>Birth cohort study, clinical follow-up</td>
<td>1985–1989, ≥37</td>
<td>34–36, n = 247, ≥37, n = 352</td>
<td>Clinical follow-up</td>
<td>Accelerometer</td>
<td>5, (–27, 38), in total PA time/day 0.8 min (–4.5, 6.1), in sedentary time 0.19% (–2.14, 2.53).</td>
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<tr>
<td>Robič Pikel [42]</td>
<td>Single maternity hospital in Slovenia</td>
<td>Birth cohort study, follow-up by national school fitness monitoring system</td>
<td>1987, 37–42</td>
<td>32–36, n = 141, 37–42, n = 218</td>
<td>Cardiorespiratory fitness (600 m run), anaerobic (60 m) and muscle fitness (standing long jump, sit-up, arm hang)</td>
<td>Linear regression, ANOVA, t-test</td>
<td>Mean difference in modified push-up –0.8 (–1.4, –0.3), handgrip –9.1 N (–28.1, 9.9), heart rate after step test 1 beat/min (–4, 2). No difference in self-perceived fitness.</td>
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</table>
Svedenkrans [43] Sweden, conscripts Register 32–36, \( n = 9930 \) 37–41, \( n = 182,490 \) 1973–1983, 100% 18 years Exercise capacity: maximal load in cycle ergometer General lineal model Age, pregnancy conditions, BWSDS, pregnancy conditions, SES, current age, BMI

| Mean exercise capacity 32–36 years: 289 (95% CI of mean 287, 292); 37–41 years: 294 (291, 296) |

BWSDS, birth weight SD score; SES, socio-economic status; BMI, body mass index.

*a*Numbers refer to gestational age in completed postmenstrual weeks and to the number or participants. Only exposure groups relevant to adult outcomes of late preterm or early preterm are listed. An exception are exposure groups extending down to 32 weeks which have been included in the table.

*b*Minimal adjustments.

*c*Additional adjustments.
<table>
<thead>
<tr>
<th>Citation</th>
<th>Setting</th>
<th>Design</th>
<th>Exposure group(s), completed weeks&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Controls, completed weeks</th>
<th>Years of birth, percent of men</th>
<th>Mean age at outcome assessment or end of follow-up</th>
<th>Main outcome(s)</th>
<th>Main statistical method</th>
<th>Adjustments</th>
<th>Main finding/conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heinonen [45]</td>
<td>Uusimaa, Finland</td>
<td>Birth cohort, clinical follow-up</td>
<td>34–36, n = 119</td>
<td>37–41, n = 667, most admitted to neonatal ward</td>
<td>1985–1986, 49.4%</td>
<td>25.3 years</td>
<td>Intellectual ability (7 WAIS-III subtests), executive functioning, attention, memory</td>
<td>Linear regression</td>
<td>Sex&lt;sup&gt;b&lt;/sup&gt;, age&lt;sup&gt;b&lt;/sup&gt;, full IQ&lt;sup&gt;b&lt;/sup&gt;, pregnancy conditions&lt;sup&gt;c&lt;/sup&gt;, SES</td>
<td>Mean difference in full IQ –3.71 (–0.71, –6.72), verbal IQ –3.11, performance IQ –3.03. Differences attributed by those born preterm SGA. No difference in executive function, attention or memory tests</td>
</tr>
<tr>
<td>Heinonen [46]</td>
<td>Helsinki, Finland</td>
<td>Birth cohort, clinical follow-up</td>
<td>34–36, n = 47</td>
<td>37–41, 872</td>
<td>1934–1944, 43.7%</td>
<td>68.1 years</td>
<td>CERAD-NB cognitive test</td>
<td>Linear and logistic regression</td>
<td>Age&lt;sup&gt;e&lt;/sup&gt;, sex&lt;sup&gt;e&lt;/sup&gt;, pregnancy conditions&lt;sup&gt;e&lt;/sup&gt;, child and adult SES&lt;sup&gt;e&lt;/sup&gt;</td>
<td>No difference in risk of mild cognitive impairment or in CERAD subtests except lower word list recognition. Interaction with adult education: when analysis was restricted to those with non-tertiary education, those born late preterm had lower subtest</td>
</tr>
</tbody>
</table>
Register studies

<table>
<thead>
<tr>
<th>Study</th>
<th>Location</th>
<th>Register</th>
<th>Year of birth</th>
<th>Sample Size</th>
<th>Cognitive performance</th>
<th>Other Predictors</th>
<th>Odds Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ekeus [47]</td>
<td>Sweden, conscripts</td>
<td>Register</td>
<td>1973–1976, 18–19 years</td>
<td>33–34, n = 1088, 35–36, n = 39,81, 37–38, n = 19,146</td>
<td>Intellectual ability</td>
<td>Linear regression</td>
<td>Mean difference in stanine scores(^d)</td>
</tr>
<tr>
<td>Lundgren [48]</td>
<td>Sweden</td>
<td>Register</td>
<td>1973–1978, 18–19 years</td>
<td>32–36, n = 9829, 37–41, n = 209,273</td>
<td>Intellectual ability and psychological performance by military forces test</td>
<td>Logistic regression</td>
<td>Intellectual performance stanine score(^d)</td>
</tr>
<tr>
<td>Moster [49]</td>
<td>Norway</td>
<td>Register</td>
<td>End 2002</td>
<td>34–36, n = 32,187, ≥37, n = 853,309</td>
<td>Disability benefits for mental retardation</td>
<td>Cox regression</td>
<td>Relative risk for mental retardation</td>
</tr>
<tr>
<td>Swedenkrans [44]</td>
<td>Sweden, conscripts</td>
<td>Register</td>
<td>1973–1983, 18–19 years</td>
<td>32–36, n = 9927, 37–41, n = 100%</td>
<td>Cognitive performance</td>
<td>Logistic regression</td>
<td>Mean score 2.8 vs 2.9, OR for above-average score 0.94 (0.91, 0.98)</td>
</tr>
</tbody>
</table>
BWSDS, Birth weight SD score; CERAD-NB, Consortium to Establish a Registry for Alzheimer’s Disease Neuropsychological Battery, SES, socio-economic status; WAIS, Wechsler Adult Intelligence Scale.

*Numbers refer to gestational age in completed postmenstrual weeks and to the number of participants. Only exposure groups relevant to adult outcomes of late preterm or early preterm are listed. An exception are exposure groups extending down to 32 weeks which have been included in the table.*

*Minimal adjustments.*

*Additional adjustments.*

*Stanine: standardized test scores with mean set at 5 and SD at 2 (one stanine score corresponds to 0.5 SD).*

*Subnormal performance = stanine scores 1 to 3.*

*The numbers are based on stanine scores regrouped in six categories.*
### Table 6. Mental health.

<table>
<thead>
<tr>
<th>Citation</th>
<th>Setting</th>
<th>Design</th>
<th>Exposure group(s), completed weeks&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Controls, completed weeks</th>
<th>Years of birth, percent of men</th>
<th>Mean age at outcome assessment or end of follow-up</th>
<th>Main outcome(s)</th>
<th>Main statistical method</th>
<th>Adjustments</th>
<th>Main finding/conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical cohort studies</strong></td>
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<tr>
<td>Heinonen [50] Uusimaa, Finland Birth cohort, clinical follow-up</td>
<td></td>
<td></td>
<td>34–36, n = 106</td>
<td>37–41, most admitted to neonatal ward, n = 617</td>
<td>1985–1986, 49.5%</td>
<td>25.3</td>
<td>Common mental disorders assessed by structured interview (M-CIDI)</td>
<td>Logistic regression</td>
<td>Sex&lt;sup&gt;b&lt;/sup&gt;, age&lt;sup&gt;b&lt;/sup&gt;, pregnancy and neonatal conditions&lt;sup&gt;b&lt;/sup&gt;, SGA&lt;sup&gt;b&lt;/sup&gt;, LGA&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Odds ratios: Any common mental disorder 1.11 (0.67, 1.84) Mood 1.11 (0.54, 2.25) Anxiety 1.00 (0.40, 2.50) Substance use 1.31 (0.74, 2.32)</td>
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<tr>
<td><strong>Register studies</strong></td>
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<tr>
<td>Lindström [51] Sweden Register</td>
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<td></td>
<td>33–36, n = 2037 37–38, n = 71,837</td>
<td>39–41, n = 450,165</td>
<td>1973–1979, 51.5%</td>
<td>End 2002</td>
<td>Psychiatric and addictive disorders from hospital and death registers</td>
<td>Cox regression</td>
<td>Age&lt;sup&gt;b&lt;/sup&gt;, sex&lt;sup&gt;b&lt;/sup&gt;, SES&lt;sup&gt;b&lt;/sup&gt;, parental psychiatric disorder&lt;sup&gt;b&lt;/sup&gt;, perinatal factors&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Any psychiatric: 33–36 weeks: 1.3 (1.2, 1.4) 37–38 weeks: 1.1 (1.1, 1.1) Psychotic: 33–36 weeks: 1.3 (1.1, 1.7) 37–38 weeks: 1.2 (1.0–1.3) Neuropsychiatric: 33–36 weeks: 2.1 (1.7, 2.4) 37–38 weeks: 1.4 (1.2, 1.6)</td>
</tr>
<tr>
<td>Study</td>
<td>Country</td>
<td>Register</td>
<td>Years</td>
<td>Sample Size</td>
<td>Hospital end year</td>
<td>Analysis Method</td>
<td>HRs</td>
<td>Additional Information</td>
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<tr>
<td>Nosarti [52]</td>
<td>Sweden</td>
<td>Register</td>
<td>32–36, $n = 47,864$</td>
<td>37–41, $n = 1,022,431$</td>
<td>1973–1985, End 2002</td>
<td>Cox regression, sex$^a$, parity$^a$, maternal age$^b$, maternal psychiatric family history$^b$</td>
<td>HRs: nonaffective psychosis 1.8 (1.2, 2.5) depressive disorder 1.4 (1.1, 1.7) bipolar disorder 2.6 (1.6, 4.4) eating disorders 1.4 (0.8, 2.3) drug dependency 1.3 (1.1, 1.6) alcohol dependency 1.4 (1.2, 1.7); adjusted similar.</td>
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<tr>
<td>D’Onofrio [53]</td>
<td>Sweden</td>
<td>Register</td>
<td>34–36, $n = 114,890$</td>
<td>37–42, $n = 3,146,386$</td>
<td>1973–2008, 37 years</td>
<td>Cox regression, sex$^b$, birth order$^b$, SES$^b$</td>
<td>HRs obtained from figures (no numerical results provided): psychotic 1.3 (1.2, 1.4) autism 1.3 (1.2, 1.4)</td>
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</tbody>
</table>

Stress-related:
- 33–36 weeks: 1.5 (1.3, 1.9)
- 37–38 weeks: 1.0 (0.8, 1.2)

Mood:
- 33–36 weeks: 1.3 (1.1, 1.5)
- 37–38 weeks: 1.1 (1.0, 1.2)

Suicide attempt:
- 33–36 weeks: 1.2 (1.0–1.4)
- 37–38 weeks: 1.1 (1.0, 1.2)

Any addictive:
- 33–36 weeks: 1.2 (1.1, 1.3)
- 37–38 weeks: 1.1 (1.0, 1.2)
<table>
<thead>
<tr>
<th>Study</th>
<th>Country</th>
<th>Source</th>
<th>Study Period</th>
<th>Number</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mathiasen [54]</td>
<td>Denmark</td>
<td>Register</td>
<td>1974–1996, End 2008</td>
<td>33–34, n = 14,199, 39–45, n = 1,104,780, 35–36, n = 42,396, 37–38, n = 157,105</td>
<td>ADHD 1.4 (1.35, 1.45). The above remain in comparisons within maternal sibships: suicide attempt 1.3 (1.2, 1.4), nullified in within-sibship comparison; substance use 1.05 (1.0, 1.1)</td>
</tr>
<tr>
<td>Moster [49]</td>
<td>Norway</td>
<td>Register</td>
<td>1967–1983, End 2002</td>
<td>34–36, n = 32,187, 37–38, n = 853,309</td>
<td>Rate ratios for 33–34, 35–36, 37–38 weeks for all psychiatric 1.25 (1.20, 1.33), 1.19 (1.12, 1.24), 1.11 (1.10, 1.13); for any psychotropic medication 1.25 (1.06, 1.48), 1.11 (1.00, 1.23), 0.87 (0.82, 0.92)</td>
</tr>
</tbody>
</table>
identified and birth data obtained through registers for ADHD

Crump [56] Sweden Register

33–34, 37–42, n = 5822
35–36, n = 588,410
36–37 weeks: male 1.12 (1.06, 1.18), female 1.14 (1.03, 1.27) All attenuated when further adjusted for prenatal factors.

Engeland [19] Norway Register

32–34, ≥37 weeks, n = 4887
35–36 weeks, n = 12,120

Date of birthb, sexb, SESb, regionb, maternal mental healthb

OR 33–34 and 35–36 weeks for: antipsychotics, 1.41 (1.14, 1.75), 1.36 (1.20, 1.54) antidepressants 1.05 (0.95, 1.16), 1.08 (1.02, 1.14) hypnotics/sedatives 1.29 (1.14, 1.46), 1.20 (1.12, 1.29) anxiolytics 1.29 (1.13, 1.47), 1.13 (1.05, 1.22) psychostimulant 1.43 (0.84, 2.43), 1.11 (0.79, 2.54) any psychotropic 1.17 (1.09, 1.27), 1.12 (0.17, 1.17)
<table>
<thead>
<tr>
<th>Study</th>
<th>Location</th>
<th>Data Source</th>
<th>Duration</th>
<th>Sample Size</th>
<th>Diagnostic Criteria</th>
<th>Methodology</th>
<th>Analysis</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Halmøy [57]</td>
<td>Norway</td>
<td>Register</td>
<td>1967–1987, Oct 1997–Apr 2005</td>
<td>39,879, 878,458</td>
<td>Stimulant treatment &gt;18 years, physician’s statement confirmed by a regional diagnostic committee</td>
<td>Multivariate relative risk models</td>
<td>Year of birth, sex, pregnancy conditions, SES</td>
<td>Relative risk 1.4 (1.1, 1.7)</td>
</tr>
<tr>
<td>Lahti [58]</td>
<td>Helsinki, Finland</td>
<td>Birth cohort, register follow-up</td>
<td>1934–1944, End 2010</td>
<td>664, 10,712</td>
<td>Inpatient treatment with mental disorder</td>
<td>Cox regression</td>
<td>Stratified for sex, year of birth, adjusted for SES</td>
<td>HRs: any mental disorder 1.06 (0.86, 1.31), substance use 1.14 (0.86, 1.51)</td>
</tr>
<tr>
<td>diagnosis</td>
<td>BWSDS&lt;sup&gt;b&lt;/sup&gt;</td>
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<tr>
<td>psychotic</td>
<td>1.34 (0.87, 2.06)</td>
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<tr>
<td>mood</td>
<td>0.84 (0.54, 1.23)</td>
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<tr>
<td>anxiety</td>
<td>0.89 (0.52, 1.53)</td>
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<tr>
<td>personality</td>
<td>0.85 (0.44, 1.67)</td>
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<tr>
<td>suicides</td>
<td>1.67 (0.89, 3.12)</td>
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<tr>
<td>suicides men</td>
<td>2.00 (1.03, 3.88)</td>
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</tbody>
</table>

BWSDS, birth weight SD score; SES, socio-economic status

<sup>a</sup>Numbers refer to gestational age in completed postmenstrual weeks and to the number or participants. Only exposure groups relevant to adult outcomes of late preterm or early preterm are listed. An exception are exposure groups extending down to 32 weeks which have been included in the table.

<sup>b</sup>Additional adjustments.

<sup>c</sup>Minimal adjustments.
<table>
<thead>
<tr>
<th>First author [ref.]</th>
<th>Setting</th>
<th>Design (Register, clinical cohort, outcome case–control)</th>
<th>Exposure group(s), completed weeks (^\text{a})</th>
<th>Controls, completed weeks, (n)</th>
<th>Years of birth, percent of men</th>
<th>Mean age at outcome assessment or end of follow-up</th>
<th>Main outcome(s)</th>
<th>Main statistical method</th>
<th>Adjustments</th>
<th>Main finding/conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical cohort studies</td>
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<tr>
<td>Ulrich [59] Odense, Denmark</td>
<td>Birth cohort, survey follow-up (response rate 56%)</td>
<td>32–37 (n = 69) ≥38 (n = 304)</td>
<td>1972–1973 years 46.9%</td>
<td>31–32 years (end 2004)</td>
<td>Education, social status</td>
<td>Logistic regression</td>
<td>Main results shown from univariate analyses; additional adjusting for gender(^b), social status at birth(^b), maternal education(^b), GA(^b), estimates not shown</td>
<td>Univariate OR (95% CI), premature vs mature: Education: None: 1 Occupational training in elementary school age: 0.7 (0.2–1.7) Short higher education (&lt;3 years): 0.6 (0.2–1.7) Intermediate higher education/bachelor (3–4 years): 0.5 (0.2–1.3) Long higher education (&gt;4 years): 0.5 (0.2–1.3) Social status: (reference: lower) Upper: 0.3 (0.1–0.9)</td>
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<tr>
<td>Name</td>
<td>Country</td>
<td>Study Type</td>
<td>Birth cohort</td>
<td>Education attainment</td>
<td>Structural equation modelling</td>
<td>Mediators:</td>
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<tr>
<td>Nomura [60]</td>
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<td>33–37</td>
<td>&gt;37 (? “full term”)</td>
<td>27–33 years (end 1994)</td>
<td>Learning-related abilities (at age 7 years)&lt;sup&gt;a&lt;/sup&gt;, childhood poverty&lt;sup&gt;a&lt;/sup&gt;</td>
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<td></td>
<td>n = 226</td>
<td>1960–1965, 45.3%</td>
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<td>No numerical comparison of educational attainment.</td>
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<td>n = 1,393</td>
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<td>Structural equation modelling showed that late preterm birth associated with lower educational attainment mediated through learning-related abilities at age 7 years, more so in families living in poverty.</td>
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<tr>
<td>Linström [61]</td>
<td>Sweden</td>
<td>Register</td>
<td>33–36</td>
<td>39–41</td>
<td>23–29 years (end 2002)</td>
<td>RR (95% CI) for 33–36 and 37–38:</td>
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<td>n = 19,166</td>
<td>1973–1979, 54.4%</td>
<td></td>
<td>Postsecondary education 35.5%, 0.91 (0.89–0.94)</td>
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<td>n = 431,656</td>
<td>33–36: 54.4%</td>
<td></td>
<td>37–38: 38.2% 0.98 (0.97–0.99) Controls 39.8%</td>
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<td></td>
<td>n = 68,541</td>
<td>39–41: 54.3%</td>
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<td>Employment 72.5%, 0.98 (0.97–1.00) Controls 74.1%</td>
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<td></td>
<td>51.0%</td>
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<td>Mean difference (€/year) for 33–36 and 37–38:</td>
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- Upper middle: 0.5 (0.2–1.0)
- Middle middle: 0.6 (0.2–1.4)
- Lower middle: 0.4 (0.2–0.9)
<table>
<thead>
<tr>
<th>Author</th>
<th>Country</th>
<th>Register Interval</th>
<th>Education</th>
<th>GA 33-36</th>
<th>GA 37-42</th>
<th>Year of birth, maternal age, infant sex, multiple birth</th>
<th>Net salary, Disposable income, Net transfer, Adjusted</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moster [49]</td>
<td>Norway</td>
<td>all analyses: education n = 32,945</td>
<td>1967–1983 years (end 2003)</td>
<td>Education: completing high school/university, bachelor’s degree</td>
<td>Log-binomial regression Sex, year of birth, multiple births, single motherhood, maternal age, mother’s and father’s level of</td>
<td>RR (95% CI)</td>
<td>Completed high school: 72.3% vs. 75.4%, 1.0 (1.0–1.0) Bachelor’s degree: 31.5% vs 34.7%, 1.0 (1.0–1.0) Postgraduate degree: 6.1% vs 7.0%, 1.0 (0.9–1.0)</td>
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</table>
postgraduate degree; unemployment; income: low/high job-related; receiving Social Security benefits

<table>
<thead>
<tr>
<th>D’Onofrio [53]</th>
<th>Sweden</th>
<th>Register 34–36</th>
<th>37–42</th>
<th>n = 3,146,386</th>
<th>1973–2008, 1−36 years (end 2009)</th>
<th>1–36 years</th>
<th>Cox and logistic regression</th>
<th>Sex, birth order, year of birth; maternal/paternal age at the child’s birth; highest level of education completed in 2008, lifetime history of any criminal conviction; fixed-effects model (siblings, also cousin)</th>
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<tr>
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<td>Failed grades, low educational attainment (&lt;10 y), higher education, social welfare benefits</td>
<td>34–36</td>
<td>Failing grades, low educational attainment (&lt;10 y), higher education, social welfare benefits</td>
<td>ORs obtained from figures (no numerical ORs provided) Failing grades 17.68% vs 14.86%, 1.3 (1.25, 1.35) Education &lt;10 y 31.10% vs 28.77%, 1.1 (1.05, 1.15) Higher education 27.95% vs. 31.95%, 0.9 (0.75, 0.85) Comparisons between maternal siblings: associations no longer present (education &lt;10 y may be lower)</td>
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<td>114,890</td>
<td>51.6%</td>
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<tr>
<td>Heinonen [63]</td>
<td>Finland</td>
<td>Register</td>
<td>34–36</td>
<td>37–41</td>
<td>1934–1944</td>
<td>56–66</td>
<td>SEPs: odds of belonging to lowest or highest category (also intergenerational social mobility)</td>
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<tr>
<td>n = 486</td>
<td>n = 8507</td>
<td>53.0%</td>
<td>56–66 years (end 2000)</td>
<td>Logistic regression</td>
<td>Gender, year of birth, father’s occupational category in childhood, birth order, mother’s age, mother’s BMI at delivery, birth weight relative to length of gestation</td>
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<tr>
<td>OR (95% CI), ref. GA 37–41:</td>
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<td>Low: Occupational status: manual worker 21.5% vs 14.8%; 1.61 (1.26–2.05)</td>
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<td>Educational level: basic or upper secondary: 72.6% vs 66.7%; 1.31 (1.07–1.61)</td>
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<td>Income: lowest income third: 39.5%, 32.1%; 1.34 (1.11–1.62)</td>
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<td>High: Occupational status: senior clerical: 0.83 (0.68–1.00)</td>
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<td>Educational level: higher tertiary education: 0.85 (0.62–1.17)</td>
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<td>Income: highest income third: 0.75 (0.62–0.93)</td>
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<td>Adjustment: very little change</td>
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</table>

SES, socio-economic status.

*a*Numbers refer to gestational age in completed postmenstrual weeks and to the number of participants. Only exposure groups relevant to adult outcomes of late preterm or early preterm are listed. An exception are exposure groups extending down to 32 weeks which have been included in the table.

*b*Additional adjustments. *c*Minimal adjustments.
Figure 1. PRISMA 2009 Flow Diagram


For more information, visit www.prisma-statement.org.