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

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Abstract

We reviewed literature on adult outcomes of people born late preterm (LPT, 34-36 completed weeks) or early term (ET, 37-38). PubMed produced 9,547 articles; 53 were eligible. Of them, 12 were based on clinical cohorts, 32 on medical birth register linkages and 9 on historical birth cohorts; 48/53 on Nordic countries; 50/53 reported on LPT and 8/53 on ET. LPT+ET have increased early (<45y) 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 for stroke and all for type 2 diabetes. Most show increased risk of 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 urge authors to report findings of LPT/ET separately from those born more preterm.

Key words: Preterm, premature, birth, term, adult, cardiovascular, pulmonary, lung, fitness, 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 6 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 lifecourse 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, which can both 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 (VP;<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 of 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 standpoint of WHO long focusing on low birth weight as a perinatal indicator and 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 relatively little is known about these outcomes in adults who were born LPT or ET. While individual risks are higher 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 (SSK) in MEDLINE Database, using the search engine PubMed, between April 27th and May 7th 2018. The search produced 9,547 articles. Searches were performed using combinations of terms describing preterm birth and various health and social outcomes (see below).

SSK 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 (EK and PH) 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, please see the PRISMA flow-chart in Figure 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 (Tables 1-7) by two authors (EK and PH). 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 were 1.4 (95% 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 a 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[14] showed a hazard ratio for all-cause mortality of 1.11 (1.02, 1.20) for those born LPT. In analyses restricted to outcome-discordant maternal sibpairs, 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 BMI, waist circumference, percentage body fat and higher insulin and transaminase and uric acid concentrations, in part mediated through higher adult BMI.[15]

For office and 24-hour 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 [18] or purchases of antihypertensive medication:[19] both reported odds ratios 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.[20, 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.[22]

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 in 1924-1944 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.[23] In a subset born 1934-1944, 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 less than 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 cutoff 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 a 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 before 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 below 35 weeks compared with those born at 39-40 weeks.[29]

Seven studies based on Nordic Medical Birth Registries fulfilled search criteria (Table 3). Of them, six assessed asthma. Two studies using conscript examination showed no increased risk of asthma. [30,31] Three used purchases of asthma medication as an outcome. A Norwegian[21] and a Danish[32] study showed odds or risk ratios of 1.1 to 1.4, whereas a Swedish study showed no association, with a narrow confidence interval, 0.97 (0.90, 1.04).[33] In addition, 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[30] and another Swedish study showed that those born at 35-36 weeks had less purchases of physician-prescribed nasal corticosteroids.[35] For atopic dermatitis, no difference was seen in the Norwegian attendance benefit[34] or Danish conscript[31] studies.

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.[36]

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).[37, 38] The largest study was based on Conscript Register cardiorespiratory fitness measurement for 218,820 men. [37, 39] Maximal load on cycle ergometer was 302 (SD49) W for those born at 32-36 weeks) and 307 (SD50) W 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).[38] The study reported no significant differences between those born at 32-37 weeks and full term group, except 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, adult born LPT 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 .[40] The two other studies from the same cohort reported no differences in leisure time physical activity measured by self-report [41] or objectively measured physical activity and sedentary time[42]. The study from the Northern Ireland birth cohort assessed cardiorespiratory fitness longitudinally from adolescence to young adulthood in 791 term born participants.[43] 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 to 42 weeks .[43]

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 (0.25 SD) 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.[44]

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. [45]

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, 0.11 SD for those born 35-36 and 0.04 SD for those born ET.[46] Another study compared those born at 32-36 weeks to those born at term; mean difference corresponds to 0.12 SD, and odds ratio of subnormal performance (stanine score 1 to 3) is 1.26.[47] The third study focused on associations between intellectual ability and cardiorespiratory fitness; however, it reported an OR for above-average score of 0.94.[39]

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.[48]

3.6 Mental health

One clinical birth cohort study, 9 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).[49]

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 over 3 million people born in Sweden from 1973 onwards; this population included all participants in the two other studies. Those born late preterm had a hazard ratio of approximately 1.3 for psychotic disorders and, assessed up to 19 years, 1.3 for autism-spectrum disorders and 1.4 for ADHD. These hazard ratios were sustained also in comparisons within maternal siblings.[50].

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 hazard ratios were lower than those for preterm birth. [51]

A study based on 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.[52] 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.[52]

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.[53] 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.[54] Further, one study based on Swedish[55] and another on Norwegian prescription database[21] assessed a range of medications (Table 6).

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 among men the rate of suicides was 2-fold.[56]

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 postsecondary 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.[57] A Norwegian study at 28-37 years showed approximately 5 percentage points lower in rates of low or of graduate education.[58] Another Norwegian study at maximum 36 years compared LPT to term controls. corresponding differences were approximately 3 percentage points.[48] In both studies adjustment for socio-economic indicators attenuated the result to one third or half. A study with Swedish data showed similar or slightly lower differences; however, most differences attenuated to null when comparing preterm-born with maternal siblings. [50]

In the Helsinki 1934-1944 Birth Cohort, those born LPT were more likely to end up in manual profession, have lower education and less income. These results survived adjustment for parental socio-economic position.[59]

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 5 other were from Slovenia, Northern Ireland, Australia and United States. No study was from low or middle income countries. The Nordic region with its 0.35% share of world's population and 0.20% share of births has thus a disproportionate share in the evidence on adult outcome people born LPT or ET.

The dominance of the Nordic region in publication has a number of 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% (singletons alive at 1 year)[12] and 3.9% (all alive at 15 y).[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 of the societies can 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 sample including all degrees or prematurity, power allowing, we urge 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 obviously 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 a LPT only and studies using LPT extended to 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, while 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 lifecourse health. While 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 in particular 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.

5. 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 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.

6. 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

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Table 1. Mortality.

Citation	Setting	Design	Exposure group(s), n[1]	Controls, n	Years of birth, percent of men	Mean age at outcome assessment or end of follow-up	Main outcome(s)	Main statistical method	Adjustments[2]	Main finding / conclusion[1]
Srinivasjois J Pediatr 2017 [11]	Western Australia	Register	32-34, n=9,066 35-36, n=26,070, 37-38, n=174,146	39-41, n=412,882	1980-2010, 50.9%	6 to 30 y	All-cause mortality 6 to 30 y	Log binomial regression	Sex, race, decade, pregnancy conditions, parity, SES	HR 32-34 1.4 (1.0, 2.0) 35-36 1.1 (0.9, 1.4) 37-38 1.0 (0.9, 1.1)
Crump JAMA 2011 [12]	Sweden	Register	34-36, n=22,590	37-42, n=626,723	1973-1979, 51.4%	End 2008	All-cause mortality 18 to 36 y	Cox regression	*Birth year, sex, birth order, BWSDS, SES*	HR All-cause 1.43 (1.24, 1.64)
Crump Epidemiology 2013 [13]	Sweden	Register	37-38, n=93,645	39-42, n=536,617	1973-1979, 51.2%	End 2008	All-cause and cause-specific mortality 18 to 36 y	Cox regression	*Birth year, sex, birth order, SES*	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) Cancer 1.20 (0.95, 1.51) External 1.12 (1.01, 1.24)
Risnes PLoS One 2016 [14]	Norway	Register	34-36, n=61,082	37-41, n=1,265,248	1967-1997, 51.6%	End 2011	All-cause and cause-specific mortality after 15 y	Cox regression	Birth year, sex, parity, SES	All-cause 1.11 (1.02, 1.20) External 1.09 (0.99, 1.43) Cancer 0.86 (0.66, 1.13) Cardiovascular 1.18 (0.85-1.64) Analyses within discordant maternal sibpairs (n=29,536): no association

[1]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.
[2]Minimal adjustments in standard case, additional adjustments in *Italics*.
BWSDS, Birth weight SD score; SES, socio-economic status

Table 2. Cardiometabolic outcomes.

Citation	Setting	Design	Exposure group(s), completed weeks, n[1]	Controls, completed weeks, n	Years of birth, percent of men	Mean age at outcome assessment or end of follow-up	Main outcome(s)	Main statistical method	Adjustments[2]	Main finding / conclusion
Clinical cohort studies										
Sipola-Leppänen Am J Epidemiol 2015 [15]	Northern Finland	Birth cohort, clinical follow-up	LPT, n=242	≥37 wk, n=344	1985-1989, 49.1%	23.4 y	Metabolic syndrome and it components	Logistic and linear regression	Age, sex, source cohort; *SES, pregnancy conditions, BWSDS, parental cardiometabolic disease, adult lifestyle*	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
Sipola-Leppänen Hypertension 2015 [16]	Northern Finland	Birth cohort, clinical follow-up	LPT, n=72	≥37 wk, n=103	1985-1989 44.6%	23.2 y	Ambulatory blood pressure	Linear regression	Age, sex, sleep assessment method; *child SES, pregnancy conditions, BWSDS, adult body size lifestyle*	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, 1.4)
Matinolli Pediatr Res 2018 [17]	Northern Finland and Uusimaa, Finland	Birth cohort, clinical follow-up	LPT, n=352	≥37 wk, n=631	1985-1989, 47.6%	24.2 y	Recommended diet index from food frequency questionnaire	Linear regression	Separate analyses by sex, adjusted for age, source cohort, energy intake, *SES, pregnancy conditions, BWSDS, adult*	Mean differences in recommended diet index Women 0.06 (-0.48, 0.60) Men -0.04 (-0.62, 0.54)

									lifestyle	
Register studies										
Johansson Circulation 2005 [18]	Sweden, conscripts	Register	33-36 wk, n=12,660	37-41 wk, n=275,895	1973-1981, 100%	18.2 y-	High systolic (≥140 mmHg) or diastolic (≥90 mmHg) pressure	Logistic regression	*Age, BWSDS, parity, child SES, current body size*	High systolic: 1.21 (1.16, 1.26) High diastolic: 1.25 (1.02, 1.53)
Engeland Pharmacoepdemiol Drug Safety 2017 [21]	Norway	Register	32-24 wk, n=4,887 35-36 wk, n=12,120	≥37 wk, n=431,914	1974-1984, 56.5%	30.5 y	Purchase of medication at least twice between 30th and 31st birthday	Logistic regression	SES	RR for 32-34 and 35-36. 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, women 1.5 (0.8, 1.6), 1.1 (0.8, 1.7), men 1.6 (0.8, 3.5), 1.5 (0.9, 2.5), cardiovascular women 1.0 (0.7, 1.4), 1.3 (1.1-1.5), men 1.2 (0.9, 1.5), 1.3 (1.1, 1.5)
Crump Diabetes Care 2011 [20]	Sweden	Register	35-36 wk, n=19,025	37-42 wk, n=583,571	1973-1979, N/A	July 2005 to end 2009	Prescription of medication for diabetes	Generalised estimating equations	*Age, sex, pregnancy conditions, child SES, maternal diabetes, BWSDS*	OR for any diabetes medication 1.22 (1.08, 1.38) OR for insulin without oral diabetes medication 1.25 (1.08, 1.45)
Crump Am J Epidemiol 2011 [19]	Sweden	Register	33-34 wk, n=5,685, 35-36 wk, n=19,194 157,105	37-42 wk, n=589,573	1973-1979, N/A	July 2005 to end 2009	Prescription of antihypertensive medication	Logistic regression	*Age, sex, pregnancy conditions, child SES, maternal antihypertensive medication, BWSDS*	OR for 33-34 wk. 1.33 (1.15, 1.42) 34-35 wk 1.28 (1.15, 1.25)
Ueda Eur J Epidemiol 2014 [60]	Sweden	Register	32-36, n=62,725	37-41, n= 1,057,240	1983-1995, 51.4%	End 2010	Ischemic heart	Cox regression	Stratified by sex, *adjusted for*	Hazard ratios Cerebrovasc 1.01 (0.75, 1.35)

							disease or cerebrovascular disease from hospital discharge and death registers		*maternal characteristics, BWSDS*	Ischemic heart 1.43 (0.81, 2.52)
Zöller Pediatrics 2014 [22]	Sweden	Register	LPT, n=153,296	37-41, n=3,066,290	1973-2008, 51.4%	End 2010	Venous thromboembolism from hospital discharge and outpatient registers	Cox regression	Age, sex, birth cohort, pregnancy conditions, BWSDS, SES, family history of venous thromboembolism	HR for VTE at ≥18 y 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)
Kajantie Diabetes Care 2010 [24]	Born in 2 delivery units inf Helsinki, Finland	Birth cohort, register follow-up	<35 wk, n=247 35-36 wk, n=549	37-41 wk, n=10,711	1934-1944, 52.1%	End 2002	Special reimbursement for diabetes medication granted after 40 y	Logistic regression	Year of birth, sex, *firstborn, SES, BWSDS*	OR for diabetes <35 wk 1.68 (1.06, 2.65) 35-36 wk 0.65 (0.41, 1.05)
Kaijser Circulation 2008 [25]	Born in 4 delivery unit in Stockholm, Uppsala and Sundsvall, Sweden	Birth cohort, register follow-up	33-36 wk, n=1,945[3]	37-42 wk, n=3,221	1925-1949, N/A	End 2002	Ischaemic heart disease diagnoses from hospital discharge and death registers	Cox regression	Stratified for year of birth and sex, *adjusted for BWSDS*	HR 0.96 (0.80, 1.16)
Bonamy Am J Hypertens 2008 [26]	Born in 4 delivery units in	Birth cohort, register follow-up	33-34, n=1555[3] 35-36,	37-42, n=3174	1925-1949, N/A	End 2006	Hypertension diagnosis in hospital	Cox regression	Stratified for sex and year of birth, *BWSDS*	Hazard ratios 33-34 wk 1.32 (0.87, 1.99); 35-36 wk 1.23 (0.83, 1.83)

	Stockholm, Uppsala and Sundsvall, Sweden		n=321[3]				discharge register			
Kaijser Diabetes 2009 [27]	Born in 4 delivery units in Stockholm, Uppsala and Sundsvall, Sweden	Birth cohort, register follow-up	33-36 wk, n=1,945[3]	37-42 wk, n=3,221	1925-1949, N/A	End 2006	Diabetes diagnoses from Hospital Discharge Register	Cox regression	Year of birth, sex and SES	HR 1.29 (1.05, 1.58). Stronger when lower BWSDS
Koupil Stroke 2005 [28]	Uppsala, Sweden	Birth cohort, Register follow-up	See results	Total n 11,474 (group n N/A)	1915-1929, N/A	End 2001	Death from ischaemic heart disease or stroke	Cox regression	Age, year of birth, sex, *SES*	30-35 week: reference 36-37: CHD 0.92 (0.66, 1.28), stroke 0.72 (0.43, 1.20) 38-39: CHD 0.94 (0.70, 1.26), stroke 0.62 (0.40, 0.97), 40-41 CHD 1.01 (0.76, 1.35), stroke 0.56 (0.36, 0.88)
Kajantie Paediatr Perinat Epidemiol 2015 [23]	Born in 2 delivery units inf Helsinki, Finland	Birth cohort, register follow-up	LPT, n=1006	≥37 wk, n=17,972	1924-1944, 51.8%	End 2010	Coronary heart disease and stroke from hospital discharge and death registers	Cox regression	Stratified by sex, year of birth, *adjusted for sex, BWSDS*	Hazard ratios Coronary 0.99 (0.85, 1.14) Stroke 0.86 (0.71, 1.06)

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[2]Minimal adjustments in standard case, additional adjustments in *Italics*.
[3]The cohort was originally recruited by selecting individuals born at less than 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.
BWSDS, Birth weight SD score; SDS, socio-economic status

Table 3. Pulmonary and atopic outcomes.

Citation	Setting	Design (Register, clinical cohort, outcome-case-control)	Exposure group(s), completed weeks, n[1]	Controls, completed weeks, n	Years of birth, percent of men	Mean age at outcome assessment or end of follow-up	Main outcome(s)	Main statistical method	Adjustments[2]	Main finding / conclusion
Clinical cohort study										
Pekkanen Clin Exp All 2001 [29]	Northern Finland	Clinical birth cohort	≤35 wk, n=229, 36-38 wk, n=1,303, 39-40 wk, n=2,435		1966, 50.2%	31 y	Atopy (positive skin prick), history of doctor-diagnosed asthma	Logistic regression	Sex, pregnancy conditions, parental allergy, current BMI, SES	≤35 wk: reference 36-38: atopy 1.22 (0.87, 1.70), asthma 0.90 (0.54, 1.49) 39-40: atopy 1.42 (1.02, 1.98), asthma 0.81 (0.49, 1.33)
Register studies										
Bråbäck Clin Exp All 1998 [30]	Sweden, army recruits	Register	33-36, n=6,607	n=119,506	1973-1975, 100%	18-19 y	Allergic rhinitis or asthma by physician's examination	Logistic regression	*Sex, pregnancy conditions, older siblings*	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)
Engeland Pharmacoepdemiol Drug Safety 2017 [21]	Norway	Register	32-34 wk, n=4,887 35-36 wk, n=12,120	≥37 wk, n=431,914	1974-1984, 56.5%	30.5 y	Purchase of medication at least twice between 30th and 31st birthday	Logistic regression	SES	RR for 32-34 and 35-36. Antiasthmatics, women 1.4 (1.1, 1.8), 1.1 (0.9, 1.3), men 1.3 (1.0, 1.6), 1.2 (1.1-1.4)
Damgaard PLoS One 2015 [32]	Denmark	Register	32-36, n=31,958	37-45, n=733,787	1980-1993,[3] 51.2%	2010-2011	Purchase of prescribed asthma medication	Logistic regression	*Sex, pregnancy conditions, older siblings, SES, maternal asthma medication,*	OR for asthma at 18-24 y 1.26 (1.15, 1.38); at 25-31 y 1.14 (1.02, 1.27)

									neonatal resp morbidity	
Crump J All Clin Immunol 2011 [35]	Sweden	Register	35-36 wk, n=19,025	37-42 wk, n=583,571	1973-1979, 51.2%	7/2005-12/2009	Purchases of prescribed nasal corticosteroids or oral antihistamines	Logistic regression	*Age, sex, BWSDS, pregnancy conditions, SES, total medication prescriptions, maternal glucocorticoid/antihistamin use*	Nasal corticosteroids OR 0.94 (0.91, 0.98) Oral antihistamines OR 1.01 (0.98, 1.05)
Trønnes Pediatr Allergy Immunol 2013 [34]	Norway	Register	32-36, n=82,377	37-41, n=1,439-790	1967-2001	End 2005	Basic or attendance benefit or severe asthma or atopic dermatitis	Logistic regression	*Year of birth, pregnancy conditions, parity, maternal history, SEP*	Or for asthma 1.69 (1.56, 1.82), for atopic dermatitis 0.69 (0.90, 1.01)
Steffensen Epidemiology 2011 [31]	Denmark, conscripts	Register	34-36, n=327	≥37, n=4323	1973-~1975, 100%	8/2013-7/2014	Asthma and atopic dermatitis diagnoses at a conscript check	Logistic regression	*Birth weight, parity, pregnancy conditions.*	Asthma 1.0 (0.5, 1.7) atopic dermatitis 1.0 (0.3, 3.5)
Crump Pediatrics 2011 [33]	Sweden	Register	33-36, n=21,918	37-42, n= 579,359	1973-1979, 51.5%	7/2005-12/2007	Purchase of prescribed asthma medication	Logistic regression	Date of brith, sex, BWSDS, SES, maternal history	OR 0.97 (0.90, 1.04) for β-agonist and glucocorticoid, similar for other asthma medications
Broström Eur J Epidemiol 2013 [36]	Born in one of 4 delivery units in Stockholm, Uppsala	Birth cohort, register follow-up	33-36, n=1,945	37-42, n=3,221	1925-1949, 51.3%	End 2006	Asthma or COPD from hospital discharge or death register	Cox regression	-	Asthma 2.14 (1.08, 4.22) Any obstructive airways disease 1.23 (0.85, 1.78)

											and Sundsvall, Sweden

[1]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.
[2]Minimal adjustments in standard case, additional adjustments in *Italics*.
[3]An additional inclusion criterion was birth weight ≤2000 g in girls and ≤2100 g in boys.
BWSDS, Birth weight SD score; COPD, Chronic obstructive pulmonary disease; SES, socio-economic status

Table 4. Physical activity and fitness.

Citation	Setting	Design	Exposure group(s), completed weeks, n[1]	Controls, completed weeks, n	Years of birth, percent of men	Mean age at outcome assessment or end of follow-up	Main outcome(s)	Main statistical method	Adjustments[2]	Main finding / conclusion
Clinical cohort studies										
Ferreira J Am Heart Assoc 2017 [43]	Northern Ireland	Birth cohort study, clinical follow-up	37-38	39-40 Total n of all gestational ages 356	Since 1971, 48.1%	22 y	Cardiorespiratory fitness, submaximal cycle ergometry	Logistic and linear regression	Age, sex, cohort, BWSDS, *pregnancy conditions, body size and composition*	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)
Tikanmäki J Pediatr 2017 [41]	Northern Finland	Birth cohort study, clinical follow-up	34-36, n=210	≥37, n=311	1985-1989, 48.4%	23.4 y	Self-reported leisure time physical activity (detailed 12 m questionnaire)	Linear and logistic regression	Age, sex, source cohort, *pregnancy conditions, BWSDS, SES, current body size, smoking, asthma*	Mean difference in total volume of PA -6.5 METh/wk (-19.8, 9.1). No difference in conditioning, non-conditioning, commuting or vigorous PA
Tikanmäki Pediatr Res 2017 [42]	Northern Finland	Birth cohort study, clinical follow-up	34-36, n=108	≥37, n=178	1985-1989, 43.4%	23.3 y	Physical activity by accelerometer	Linear regression		Mean difference in total daily PA (accelerometer counts/min) 5, (-27, 38), in total PA time/day 0.8 min (-4.5, 6.1), in sedentary time 0.19% (-2.14, 2.53).
Tikanmäki Pediatrics 2016 [40]	Northern Finland	Birth cohort study, clinical follow-up	34-36, n=247	≥37, n=352	1985-1989, 48.1%	23.4 y	Cardiorepiratory fitness by step test, muscle fitness by modified push-up and	Linear regression	Age, sex, source cohort, *pregnancy conditions, BWSDS, SES, current body size, smoking,*	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 beats/min (-4, 2). No difference in self-perceived fitness.

							handgrip		*asthma, physical activity*	
Register studies										
Robič Pikel Early Hum Dev 2017 [38]	Single maternity hospital in Slovenia	Birth cohort study, follow-up by national school fitness monitoring system	32-36, n=141	37-42, n=218	1987, 56.5%	18 y	Cardiorespiratory (600 m run), anaerobic (60 m) and muscle (standing long jump, sit-up, arm hang)	ANOVA, t test	No	Graphical presentation and p values only. No consistent differences. Preterm men have less sit-ups than term men.
Svedenkrans PLoS One 2013 [37]	Sweden, Conscripts	Register	32-36, n=9,930	37-41, n=182,490	1973-1983, 100%	18 y	Exercise capacity: maximal load in cycle ergometer	General lineal model	Age, pregnancy conditions, BWSDS, pregnancy conditions, SES, current age, BMI	Mean exercise capacity 32-36 289 (95% CI of mean 287, 292); 37-41 294 (291, 296)

[1]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.
[2]Minimal adjustments in standard case, additional adjustments in *Italics*.
BWSDS, Birth weight SD score; SES, socio-economic status

Table 5. Cognitive function and intellectual disability.

Citation	Setting	Design	Exposure group(s), completed weeks, n[1]	Controls, completed weeks, n	Years of birth, percent of men	Mean age at outcome assessment or end of follow-up	Main outcome(s)	Main statistical method	Adjustments[2]	Main finding / conclusion
Clinical cohort studies										
Heinonen Dev Med Child Neurol 2018 [44]	Uusimaa, Finland	Birth cohort, clinical follow-up	34-36, n=119	37-41, n=667, most admitted to neonatal ward	1985-1986, 49.4%	25.3 y	Intellectual ability (7 WAIS-III subtests), executive functioning, attention, memory	Linear regression	Sex, age, full IQ, *pregnancy conditions, SES*	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
Heinonen Pediatrics 2015 [45]	Helsinki, Finland	Birth cohort, clinical follow-up	34-36, n=47	37-41, 872	1934-1944, 43.7%	68.1%	CERAD-NB cognitive test	Linear and logistic regression	Age, sex, *pregnancy conditions, child and adult SES*	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 scores and 2.7-fold higher odds for mild cognitive impairment.
Register studies										
Ekeus Pediatrics 2010 [46]	Sweden, conscripts	Register	33-34, n=1,088 35-36, n=39,81 37-38, n=19,146	39-41, n=94,821	1973,1976, 100%	18-19 y	Intellectual ability	*Linear regression*	Year of birth, age, conscription office, *SES, SGA, low Apgar*	Mean difference in stanine scores[3] 33-34 -0.30 (-0.41, -0.19) 35-36 -0.22 (-0.28, -0.16) 37-38 -0.07 (-0.10,-0.04)
Lundgren Pediatr Res	Sweden	Register	32-36,	37-41,	1973-1978,	18-19	Intellectual	Logistic	*BWSDS, length,*	Intellectual performance

2001 [47]			n=9,829	n=209,273	100%		ability and psychological performance by military forces test	regression	*head circumference at birth, current height*	stanine score[3] 4.88 vs 5.11, OR for subnormal performance[4] 1.26 (1.21, 1.31). Psychological performance stanine score 4.92 vs. 5.08, subnormal[4] 1.21 (1.16, 1.27)
Moster NEJM 2008 [48]	Norway	Register	34-36, n=32,187	≥37, n=853,309	1967-1983, 51.1%	End 2002	Disability benefits for mental retardation	Cox regression	Sex, year of birth, SES	Relative risk for mental retardation 1.6 (1.4, 1.8)
Svedenkrans PLoS One 2016 [39]	Sweden, conscripts	Register	32-36, n=9,927	37-41, 182,477	1973-1983, n=100%	18-19 y	Cognitive performance	Logistic regression		Mean score 2.8 vs. 2.9. OR for above-average score 0.94 (0.91, 0.98)[5]

[1]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 term are listed. An exception is exposure groups extending down to 32 weeks which have been included in the table.
[2]Minimal adjustments in standard case, additional adjustments in *Italics*.
[3]Stanine: standardized test scores with mean set at 5 and SD at 2 (one stanine score corresponds to 0.5 SD)
[4]Subnormal performance = stanine scores 1 to 3.
[5]The numbers are based on stanine scores regrouped in 6 categories.
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

Table 6. Mental health.

Citation	Setting	Design	Exposure group(s), completed weeks, n[1]	Controls, completed weeks, n	Years of birth, percent of men	Mean age at outcome assessment or end of follow-up	Main outcome(s)	Main statistical method	Adjustments[2]	Main finding / conclusion
Clinical cohort studies										
Heinonen Psychol Med 2016 [49]	Uusimaa, Finland	Birth cohort, clinical follow-up	34-36, n=106	37-41, most admitted to neonatal ward, n=617	1985-1986, 49.5%	25.3	Common mental disorders assessed by structured interview (M-CIDI)	Logistic regression	*Sex, age, pregnancy and neonatal conditions, SGA, LGA*	ORs: 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)
Register studies										
Lindström Pediatrics 2009 [51]	Sweden	Register	33-36, n=2,037 37-38, n=71,837	39-41, n=450,165	1973-1979, 51.5%	End 2002	Psychiatric and addictive disorders from hospital and death registers	Cox regression	*Age, sex, SES, parental psychiatric disorder, perinatal factors*	Any psychiatric 33-36 1.3 (1.2, 1.4), 37-38 1.1 (1.1, 1.1) Psychotic 33-36 1.3 (1.1, 1.7), 37-38 1.2 (1.0-1.3), Neuropsychiatric 33-36 2.1 (1.7, 2.4), 37-38 1.4 (1.2, 1.6), Stress-related 33-36 1.5 (1.3, 1.9), 37-38 1.0 (0.8, 1.2), Mood 33-36 1.3 (1.1, 1.5, 37-38 1.1 (1.0, 1.2), Suicide attempt 33-36 1.2 (1.0-1.4), 37-38 1.1 (1.0, 1.2), Any addictive 33-36 1.2 (1.1, 1.3), 37-38 1.1 (1.0, 1.2)
Nosarti Arch Gen Psychiatry 2012 [61]	Sweden	Register	32-36, n=47,864	37-41, n=1,022,431	1973-1985, 51.4%	End 2002	Hospital in-patient diagnoses	Cox regression	*Sex, parity, maternal age, SES, maternal psychiatric family*	HRs Nonaffective psychosis 1.8 (1.2, 2.5) Depressive disorder 1.4 (1.1, 1.7)

									history	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.
D'Onofrio JAMA Psychiatry 2013 [50]	Sweden	Register	34-36, n=114,890	37-42, n=3,146,386	1973-2008, 51.6%	37 y ADHD, autism 19 y	Hospital in- and, since 2001, outpatient diagnoses	Cox regression, additional within-sibpair comparisons	*Year of birth, sex, birth order, SES*	HRs obtained from figures (no numerical results provided) Psychotic 1.3 (1.2, 1.4) Autism 1.3 (1.2, 1.4) 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)
Mathiasen Acta Paediatr 2011 [52]	Denmark	Register	33-34, n=14,199 35-36, n=42,396 37-38, n=157,105	39-45, n= 1,104,780	1974-1996, 51.2%	End 2008	Central Psychiatric Research Register	Poisson regression	Calendar time, age, *sex, plurality, SES, parental mental health*	Rate ratios for 33-34, 35-36, 37-38 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)
Moster NEJM 2008 [48]	Norway	Register	34-36, n=32,187	≥37, n=853,309	1967-1983, 51.1%	End 2002	Schizophrenia and autism spectrum disorder from National Insurance Scheme	Cox regression	Sex, year of birth, SES	Relative risk for schizophrenia 1.3 (1.0, 1.7, autism spectrum disorder 0.8 (0.4, 1.4)
Silva Pediatrics 2014 [53]	Western Australia	Cases and controls	33-36, n=2,109	39-41, n=21,094	1981-2003, 77.1%	8/2003-12/2007	Stimulant medication	Logistic regression	Year of birth, SES	ORs for 33-36 male 1.16 (1.05, 1.28),

		identified and birth data obtained through registers	37-38, n=8,665				for ADHD			female 1.18 (0.97, 1.43) 37-38 male 1.12 (1.06, 1.18), female 1.14 (1.03, 1.27) All attenuated when further adjusted for prenatal factors.
Crump Int J Epidemiol 2010 [55]	Sweden	Register	33-34, =5,822 35-36, n=19,347	37-42, n=588,410	1973-1979, 51.4%	7/2005-12/2006	Prescription of psychotropic medication	Logistic regression	*Date of birth, sex, SES, region, maternal mental health*	OR for antipsychotics for 33-34 and 35-36 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)
Engeland Pharmacoepidemiol Drug Safety 2017 [21]	Norway	Register	32-34 wk, n=4,887 35-36 wk, n=12,120	≥37 wk, n=431,914	1974-1984, 56.5%	30.5 y	Purchase of medication at least twice between 30th and 31st birthday	Logistic regression	SES	RR for 32-34 and 35-36. Any psychotropic medication, women 1.2 (1.1, 1.4), 1.1 (1.0, 1.2), men 1.2 (1.0, 1.3), 1.1 (1.0, 1.1), antipsychotics women 1.9 (1.4, 2.5), 1.3 (1.0, 1.6) men 1.3 (1.0, 1.7), 1.2 (1.0, 1.4), anxiolytics women 1.6 (1.2, 2.0), 1.2 (1.0, 1.5), men 1.2 (1.0, 1.6), 1.1 (0.9, 1.3), hypnotics women 1.3 (1.0, 1.7), 1.2 (1.0, 1.4), men 1.3 (1.0, 1.6), 1.0 (0.9, 1.2), antidepressants women 1.5 (1.3, 1.7), 1.1 (1.0, 1.3), men 1.2 (1.0, 1.4), 1.1 (1.0, 1.2), ADHD medication women 1.2

										(0.7, 2.1), 0.9 (0.6, 1.4), men 1.0 (0.6, 1.6), 1.3 (1.0, 1.7)
Halmøy Biol Psychiatry 2012 [54]	Norway	Register	33-36, n=39,879	37-41, n=878,458	1967-1987	10/1997-4/2005	Stimulant treatment >18 y, physician's statement confirmed by a regional diagnostic committee	Multivariate relative risk models	*Year of birth, sex, pregnancy conditions, SES*	Relative risk 1.4 (1.1, 1.7) Graphical presentation showing comparison of 37-38 weeks with 39-41 weeks, relative risk 1.2 (1.0, 1.4)
Lahti Psychol Med 2015 [56]	Helsinki, Finland	Birth cohort, register follow-up	34-36, n=664	37-41, n=10,712	1934-1944, 52.3%	End 2010	Inpatient treatment with mental disorder diagnosis	Cox regression	Stratified for sex, year of birth, adjusted for SES, *BWSDS*	HRs: Any mental disorder 1.06 (0.86, 1.31) Substance use 1.14 (0.86, 1.51) Psychotic 1.34 (0.87, 2.065 Mood 0.84 (0.54, 1.23) Anxiety 0.89 (0.52, 1.53) Personality 0.85 (0.44, 1.67() Suicides 1.67 (0.89, 3.12) Suicides men 2.00 (1.03, 3.88)

[1]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 term are listed. An exception is exposure groups extending down to 32 weeks which have been included in the table.
[2]Minimal adjustments in standard case, additional adjustments in *Italics*.
BWSDS, Birth weight SD score; SES, socio-economic status

Table 7. Socio-economic outcomes

Citation	Setting	Design (Register, clinical cohort, outcome-case-control)	Exposure group(s), completed weeks, n[1]	Controls, completed weeks, n	Years of birth, percent of men	Mean age at outcome assessment or end of follow-up	Main outcome(s)	Main statistical method	Adjustments[2]	Main finding / conclusion
Clinical cohort studies										
Ulrich Acta Paediatrica 2013 [62]	Odense, Denmark	Birth cohort, survey follow-up (response rate 56%)	32–37 n=69	≥38 n=304	1972–1973 46.9%	31–32y (end 2004)	Education, social status	Logistic regression	Main results shown from univariate analyses; a*dditional adjusting for gender, social status at birth, maternal education, GA, estimates not shown*	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 (<3 years): 0.6 (0.2–1.7) Intermediate higher education/bachelor (3–4 years): 0.5 (0.2–1.3) Long higher education (>4 years): 0.5 (0.2–1.3) **Social status: (reference: lower)** Upper: 0.3 (0.1–0.9) Upper middle: 0.5 (0.2–1.0) Middle middle: 0.6 (0.2–1.4) Lower middle: 0.4 (0.2–0.9)
Nomura J Pediatric Psychology 2009 [63]	Received antenatal care and born in Johns Hopkins	Birth cohort (randomly selected, 71.4% response rate in the	33–37 n=226	>37 (? "full term") n=1,393	1960–1965, 45.3%	27–33y (end 1994)	Educational attainment: years of education, degrees/qualifications	Structural equation modelling	Mediators: Learning-related abilities (at age 7), childhood poverty	No numerical comparison of educational attainment. Structural equation modelling showed that late preterm birth associated with lower educational attainment

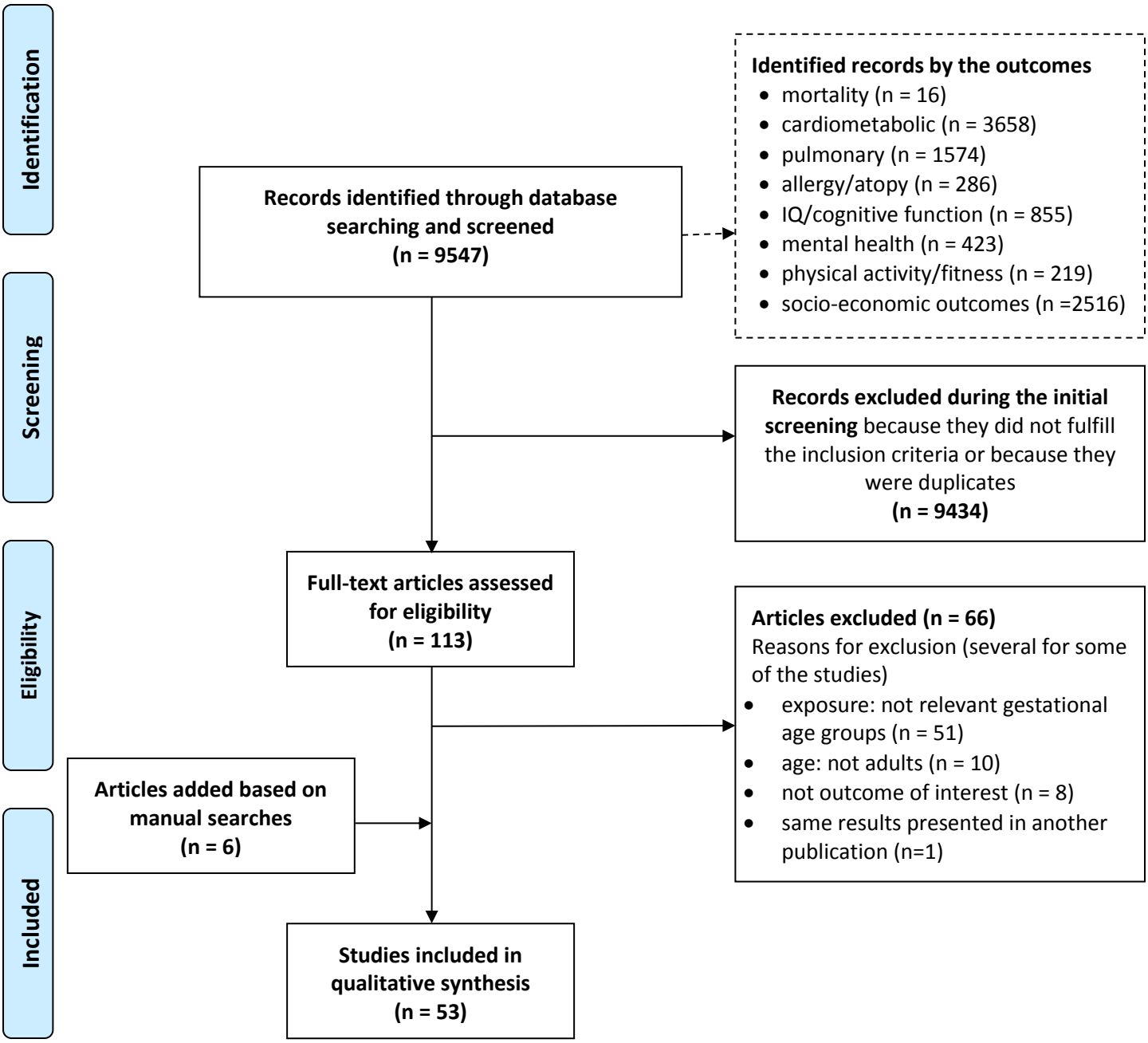
	Hospital, Baltimore, USA	follow-up), observations examinations, interviews					earned			mediated through learning-related abilities at age 7, more so in families living in poverty.
Register studies										
Lindström Pediatrics 2007 [57]	Sweden	Register	33–36 n=19,166 37–38 n=68,541	39–41 n=431,656	1973–1979 33–36:54.4% 37–38:54.3% 39–41:51.0%	23–29y (end 2002)	Postsecondary education, employment, net salary, disposable income	Logistic regression (linear for income variables)	Age, gender; *SES, single-parent household in 1985; residency, maternal age, parity, and social welfare in 1990; parental psychiatric disorders, SGA, multiple birth*	RR (95% CI) for 33-36 and 37-38: Postsecondary education 35.5%, 0.91 (0.89–0.94) 38.2% 0.98 (0.97–0.99) Controls 39.8% Employment 72.5%, 0.98 (0.97–1.00) 72.7%, 0.99 (0.98–1.00) Controls 74.1% Mean difference (Euros/year) for 33-36 and 37-38: Net salary -297, -96 Disposable income -489, -236 Net transfer -326, -138 Adjusted: attenuated by ~half
Swamy JAMA 2008 [58]	Norway	Register	33-36 all n=46,348 education analyses: n=21,912	37-42 all n= 1,053,770 education analyses: n=527,279	Education analyses: 1967-1976 51.3% (GA 33-42)	28-37y (end 2004)	Education: less than high school education; graduate education	Log link RR modeling (absolute risks AR and relative risks RR with 95% CI)	*Year of birth, maternal age, maternal education, infant sex*	RR (95% CI), ref. GA 37-42: Less than high school education GA 33-36: women: 29.9% vs 24.7%, 1.21 (1.17-1.25) men: 28.9% vs. 25.3%, 1.14 (1.11-1.17) Graduate education GA 33-36: women: 37.7% vs. 43.4% 0.87 (0.85-0.89) men: 28.8% vs. 32.6%, 0.88 (0.86-0.91) Adjusted: attenuated by ~third-half

Moster NEJM 2008 [48]	Norway	Register	34–36 n=32,945	≥37 n=858,406	1967–1983 34–36: 55.1% ≥37: 50.9%	20–36y (end 2003)	Education: completing high school/university, bachelor's degree/ postgraduate degree; unemployment; income: low/high job-related; receiving Social Security benefits	Log-binomial regression	Sex, year of birth, multiple births, single motherhood, maternal age, mother's and father's level of education, immigrant status of the parents	RR (95% CI) 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% v. 7.0%, 1.0 (0.9−1.0) Low job-related income: 20.8% vs. 20.0%, 1.1 (1.0−1.1) High job-related income: 19.9% vs. 20.0% 1.0 (0.9−1.0) Unemployed: 24.5% vs. 23.7%, 1.0 (1.0−1.0) Received Social Security benefits: 20.1% vs. 17.6%, 1.0 (1.1−1.1)
D'Onofrio JAMA Psychiatry 2013 [50]	Sweden	Register	34–36 n=114,890	37–42 n=3,146,386	1973–2008, 51.6%	1–36y (end 2009)	Failing grades, low educational attainment (<10y), higher education, social welfare benefits	Cox and logistic regression	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 comparisons)*	ORs obtained from figures (no numerical ORs provided) Failing grades 17.68% vs. 14.86%, 1.3 (1.25, 1.35) Education <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 <10 y may be lower)
Heinonen Pediatrics 2013 [59]	Finland	Register	34–36 n=486	37–41 n=8,507	1934–1944 53.0%	56–66y (end 2000)	SEP: odds of belonging to	Logistic regression	Gender, year of birth, *father's*	OR (95% CI), ref. GA 37-41: **Low:**

			lowest or highest category (also intergenerational social mobility)	*occupational category in childhood, birth order, mother's age, mother's BMI at delivery, birth weight relative to length of gestation*	Occupational status: manual worker 21.5% v. 14.8%;: 1.61 (1.26–2.05) Educational level: basic or upper secondary: 72.6% vd, 66.7% 1.31 (1.07–1.61) Income: lowest income third: 39.5%, 32.1%; 1.34 (1.11–1.62) **High:** Occupational status: senior clerical: 0.83 (0.68–1.00) Educational level: higher tertiary education: 0.85 (0.62–1.17) Income: highest income third: 0.75 (0.62–0.93) Adjustment: very little change

[1]Numbers refer to gestational age in completed postmenstrual weeks and to the number or participants. Only exposure groups relevant to late preterm or early preterm are listed. An exception are exposure groups extending down to 32 weeks which have been included in the table.
[2]Minimal adjustments in standard case, additional adjustments in *Italics*.
SES, socio-economic status.

Figure 1. PRISMA 2009 Flow Diagram



From: Moher D, Liberati A, Tetzlaff J, Altman DG, The PRISMA Group (2009). *P*referred *R*eporting *I*tems for *S*ystematic Reviews and *M*eta-*A*nalyses: The PRISMA Statement. PLoS Med 6(6): e1000097. doi:10.1371/journal.pmed1000097

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