Seminars in Fetal and Neonatal Medicine 24 (2019) 66–83

Contents lists available at ScienceDirect

Seminars in Fetal and Neonatal Medicine

journal homepage: www.elsevier.com/locate/siny

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

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ARTICLE INFO

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

ABSTRACT

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.

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

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https://doi.org/10.1016/j.siny.2018.11.001

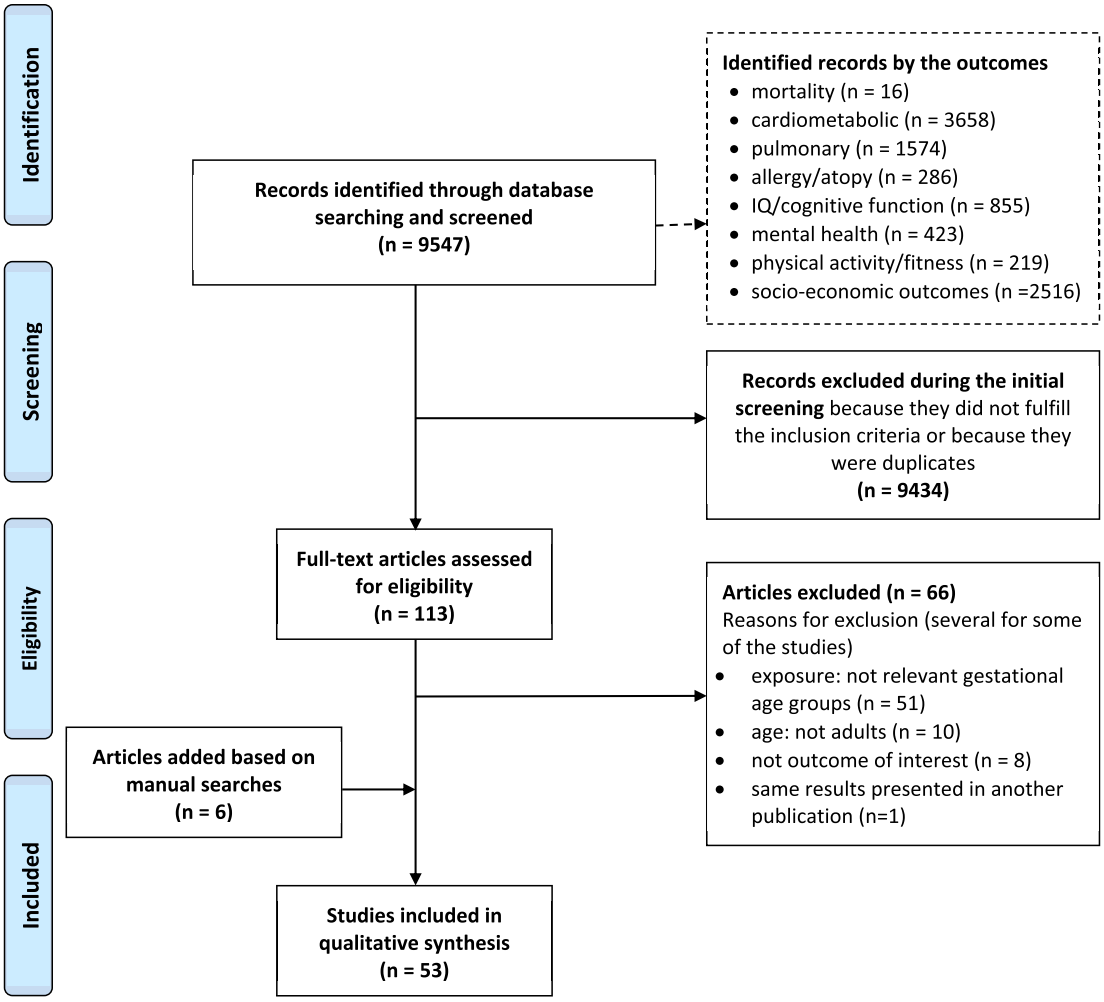

Fig. 1. PRISMA 2009 flow diagram.

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 1.1–1.4, 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 [34,35].

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

Table 1
Mortality.

First author [ref.]	Setting	Design	Exposure group (s)[a]	Controls	Years of birth, percent of men	Mean age at outcome assessment or end of follow-up	Main outcome(s)	Main statistical method	Adjustments	Main finding/conclusion[a]
Srinivasjois [11]	Western Australia	Register	32–34 weeks, n = 9066 35–36 weeks, n = 26,070 37–38 weeks, n = 174,146	39–41, n = 412,882	1980–2010, 50.9%	6–30 years	All-cause mortality 6–30 years	Log binomial regression	Sex[b], race[b], decade[b], pregnancy conditions[b], parity[b], SES[b]	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)
Crump [12]	Sweden	Register	34–36, n = 22,590	37–42, n = 626,723	1973–1979, 51.4%	End 2008	All-cause mortality 18–36 years	Cox regression	Birth year[c], sex[c], birth order[c], BWSDS[c], SES[c]	HR: all-cause 1.43 (1.24, 1.64)
Crump [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 18–36 years	Cox regression	Birth year[c], sex[c], birth order[c], SES[c]	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 [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 years	Cox regression	Birth year[b], sex[b], parity[b], SES[b]	HR: 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

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

[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 2
Cardiometabolic outcomes.

First author [ref.]	Setting	Design	Exposure group (s), completed weeks[a]	Controls, completed weeks	Years of birth, percent of men	Mean age at outcome assessment or end of follow-up	Main outcome(s)	Main statistical method	Adjustments	Main finding/conclusion
Clinical cohort studies										
Sipola-Leppänen [15]	Northern Finland	Birth cohort, clinical follow-up	LPT, $n = 242$	≥37 weeks, $n = 344$	1985–1989, 49.1%	23.4 years	Metabolic syndrome and its components	Logistic and linear regression	Age[b], sex[b], source cohort[b]; SES[c], pregnancy conditions[c], BWSDS[c], parental cardiometabolic disease[c], adult lifestyle[c]	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 [16]	Northern Finland	Birth cohort, clinical follow-up	LPT, $n = 72$	≥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 body size[c], lifestyle[c]	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 [17]	Northern Finland and Uusimaa, Finland	Birth cohort, clinical follow-up	LPT, $n = 352$	≥37 weeks, $n = 631$	1985–1989, 47.6%	24.2 years	Recommended diet index from food frequency questionnaire	Linear regression	Separate analyses by sex, adjusted for age[b], source cohort[b], energy intake[b], SES[c], pregnancy conditions[c], BWSDS[c], 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, $n = 12,660$	37–41 weeks, $n = 275,895$	1973–1981, 100%	18.2 years	High systolic (≥140 mmHg) or diastolic (≥90 mmHg) pressure	Logistic regression	Age[c], BWSDS[c], 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, $n = 4887$ 35–36 weeks, $n = 12,120$	≥37 weeks, $n = 431,914$	1974–1984, 56.5%	30.5 years	Purchase of medication at least twice between 30th and 31st birthday	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, 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 [20]	Sweden	Register	35–36 weeks, $n = 19,025$	37–42 weeks, $n = 583,571$	1973–1979, N/A	July 2005 to end 2009	Prescription of medication for diabetes	Generalised estimating equations	Age[c], sex[c], pregnancy conditions[c], child SES[c], maternal diabetes[c], BWSDS[c]	OR for any diabetes medication 1.22 (1.08, 1.38) OR for insulin without oral diabetes medication 1.25 (1.08, 1.45)

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Table 2 (*continued*)

First author [ref.]	Setting	Design	Exposure group (s), completed weeks[a]	Controls, completed weeks	Years of birth, percent of men	Mean age at outcome assessment or end of follow-up	Main outcome(s)	Main statistical method	Adjustments	Main finding/conclusion
Crump [21]	Sweden	Register	33–34 weeks, n = 5685; 35–36 weeks, n = 19,194 157,105	37–42 weeks, n = 589,573	1973–1979, N/A	July 2005 to end 2009	Prescription of antihypertensive medication	Logistic regression	Age[c], sex[c], pregnancy conditions[c], child SES[c], maternal antihypertensive medication[c], BWSDS[c]	OR for 33–34 weeks, 1.33 (1.15, 1.42) 34–35 weeks, 1.28 (1.15, 1.25)
Ueda [22]	Sweden	Register	32–36, n = 62,725	37–41, n = 1,057,240	1983–1995, 51.4%	End 2010	Ischemic heart disease or cerebrovascular disease from hospital discharge and death registers	Cox regression	Stratified by sex[b], adjusted for maternal characteristics[c], BWSDS[c]	Hazard ratios Cerebrovasc 1.01 (0.75, 1.35) Ischemic heart 1.43 (0.81, 2.52)
Zöller [23]	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[b], sex[b], birth cohort[b], pregnancy conditions[b], BWSDS[b], SES[b], family history of venous thromboembolism[b]	HR for VTE at ≥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)
Kajantie [24]	Born in two delivery units in Helsinki, Finland	Birth cohort, register follow-up	< 35 weeks, n = 247 35–36 weeks, n = 549	37–41 weeks, n = 10,711	1934–1944, 52.1%	End 2002	Special reimbursement for diabetes medication granted after 40 y	Logistic regression	Year of birth[c], sex[c], firstborn[c], SES[c], BWSDS[c]	OR for diabetes < 35 weeks: 1.68 (1.06, 2.65) 35–36 weeks: 0.65 (0.41, 1.05)
Kaijser [25]	Born in four delivery units in Stockholm, Uppsala and Sundsvall, Sweden	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 and death registers	Cox regression	Stratified for year of birth and sex, adjusted for BWSDS[c]	HR 0.96 (0.80, 1.16)
Bonamy [26]	Born in four delivery units in Stockholm, Uppsala and Sundsvall, Sweden	Birth cohort, register follow-up	33–34, n = 1555[d]; 35–36, n = 321[d]	37–42, n = 3174	1925–1949, N/A	End 2006	Hypertension diagnosis in hospital discharge register	Cox regression	Stratified for sex and year of birth, BWSDS[c]	Hazard ratios 33–34 weeks: 1.32 (0.87, 1.99); 35–36 weeks: 1.23 (0.83, 1.83)
Kaijser [27]	Born in four delivery units in Stockholm, Uppsala and Sundsvall, Sweden	Birth cohort, register follow-up	33–36 weeks, n = 1945[d]	37–42 weeks, n = 3221	1925–1949, N/A	End 2006	Diabetes diagnoses from Hospital Discharge Register	Cox regression	Year of birth[b], sex[b] and SES[b]	HR 1.29 (1.05, 1.58). Stronger when lower BWSDS

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Table 2 (*continued*)

First author [ref.]	Setting	Design	Exposure group (s), completed weeks[a]	Controls, completed weeks	Years of birth, percent of men	Mean age at outcome assessment or end of follow-up	Main outcome(s)	Main statistical method	Adjustments	Main finding/conclusion
Koupil [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[b], year of birth[b], sex[b], SES[c]	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) 40–41 weeks: CHD 1.01 (0.76, 1.35), stroke 0.56 (0.36, 0.88)
Kajantie [23]	Born in two delivery units in Helsinki, Finland	Birth cohort, register follow-up	LPT, *n* = 1006	≥37 weeks, *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[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 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.

[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.

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

Table 3
Pulmonary and atopic outcomes.

First author [ref.]	Setting	Design (register, clinical cohort, outcome case–control)	Exposure group(s), completed weeks[a]	Controls, completed weeks	Years of birth, percent of men	Mean age at outcome assessment or end of follow-up	Main outcome(s)	Main statistical method	Adjustments	Main finding/conclusion
Clinical cohort study										
Pekkanen [30]	Northern Finland	Clinical birth cohort	≤35, *n* = 229 36–38, *n* = 1303 39–40, *n* = 2435		1966, 50.2%	31 years	Atopy (positive skin prick), history of doctor-diagnosed asthma	Logistic regression	Sex[b], pregnancy conditions[b], parental allergy[b], current BMI[b], SES[b]	≤35 weeks: reference 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)
Register studies										
Bråbäck [31]	Sweden, army recruits	Register	33–36, *n* = 6607	37–41, *n* = 119,506	1973–1975, 100%	18–19 years	Allergic rhinitis or asthma by physician's examination	Logistic regression	Sex[c], pregnancy conditions[c], older siblings[c]	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 [19]	Norway	Register	32–34, *n* = 4887 35–36, *n* = 12,120	≥37 weeks, *n* = 431,914	1974–1984, 56.5%	30.5 years	Purchase of medication at least twice between 30th and 31st birthday	Logistic regression	SES[b]	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 (1.0, 1.6), 1.2 (1.1–1.4)
Damgaard [32]	Denmark	Register	32–36, *n* = 31,958	37–45, *n* = 733,787	1980–1993[d], 51.2%	2010–2011	Purchase of prescribed asthma medication	Logistic regression	Sex[c], pregnancy conditions[c], older siblings[c], SES, maternal asthma medication[c], neonatal respiratory morbidity[c]	OR for asthma at 18–24 years 1.26 (1.15, 1.38); at 25–31 years 1.14 (1.02, 1.27)
Crump [33]	Sweden	Register	35–36, *n* = 19,025	37–42 weeks, *n* = 583,571	1973–1979, 51.2%	Jul 2005 to Dec 2009	Purchases of prescribed nasal corticosteroids or oral antihistamines	Logistic regression	Age[c], sex[c], BWSDS[c], pregnancy conditions[c], SES[c], total medication prescriptions[c], maternal glucocorticoid/antihistamin use[c]	Nasal corticosteroids OR 0.94 (0.91, 0.98) Oral antihistamines OR 1.01 (0.98, 1.05)
Trønnes [34]	Norway	Register	32–36, *n* = 82,377	37–41, *n* = 1439–790	1967–2001	End 2005	Basic or attendance benefit or severe asthma or atopic dermatitis	Logistic regression	Year of birth[c], pregnancy conditions[c], parity[c], maternal history[c], SEP[c]	Or for asthma 1.69 (1.56, 1.82), for atopic dermatitis 0.69 (0.90, 1.01)
Steffensen [35]	Denmark, conscripts	Register	34–36, *n* = 327	≥37, *n* = 4323	1973–~1975, 100%	Aug 2013 to Jul 2014	Asthma and atopic dermatitis diagnoses at a conscript check	Logistic regression	Birth weight[c], parity[c], pregnancy conditions[c]	Asthma 1.0 (0.5, 1.7) atopic dermatitis 1.0 (0.3, 3.5)
Crump [36]	Sweden	Register	33–36, *n* = 21,918	37–42, *n* = 579,359	1973–1979, 51.5%	Jul 2005 to Dec 2007	Purchase of prescribed asthma medication	Logistic regression	Date of birth[b], sex[b], BWSDS[b], SES[b], maternal history[b]	OR 0.97 (0.90, 1.04) for β-agonist and glucocorticoid, similar for other asthma medications
Broström [37]	Born in one of four delivery units in Stockholm, Uppsala and Sundsvall, Sweden	Birth cohort, register follow-up	33–36, *n* = 1945	37–42, *n* = 3221	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)

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

[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.

[d] An additional inclusion criterion was birth weight ≤2000 g in girls and ≤2100 g in boys.

Table 4
Physical activity and fitness.

Citation	Setting	Design	Exposure group(s), completed weeks[a]	Controls, completed weeks	Years of birth, percent of men	Mean age at outcome assessment or end of follow-up	Main outcome(s)	Main statistical method	Adjustments	Main finding/conclusion
Clinical cohort studies										
Ferreira [38]	Northern Ireland	Birth cohort study, clinical follow-up	37–38	39–40 Total no. of all gestational ages = 356	Since 1971, 48.1%	22 years	Cardiorespiratory fitness, submaximal cycle ergometry	Logistic and linear regression	Age[b], sex[b], cohort[b], BWSDS[b], pregnancy conditions[c], body size and composition[c]	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 [39]	Northern Finland	Birth cohort study, clinical follow-up	34–36, $n = 210$	≥37, $n = 311$	1985–1989, 48.4%	23.4 years	Self-reported leisure time physical activity (detailed 12 m questionnaire)	Linear and logistic regression	Age[b], sex[b], source cohort[b], pregnancy conditions[c], BWSDS[c], SES[c], current body size[c], smoking[c], asthma[c]	Mean difference in total volume of PA −6.5 METh/weeks (−19.8, 9.1). No difference in conditioning, non-conditioning, commuting or vigorous PA
Tikanmäki [40]	Northern Finland	Birth cohort study, clinical follow-up	34–36, $n = 108$	≥37, $n = 178$	1985–1989, 43.4%	23.3 years	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 [41]	Northern Finland	Birth cohort study, clinical follow-up	34–36, $n = 247$	≥37, $n = 352$	1985–1989, 48.1%	23.4 years	Cardiorepiratory fitness by step test, muscle fitness by modified push-up and handgrip	Linear regression	Age[b], sex[b], source cohort[b], pregnancy conditions[c], BWSDS[c], SES[c], current body size[c], smoking[c], asthma[c], physical activity[c]	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.
Register studies										
Robič Pikel [42]	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 years	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 [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[b], pregnancy conditions[b], BWSDS[b], pregnancy conditions[b], SES[b], current age[b], BMI[b]	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 5
Cognitive function and intellectual disability.

Citation	Setting	Design	Exposure group (s), completed weeks[a]	Controls, completed weeks	Years of birth, percent of men	Mean age at outcome assessment or end of follow-up	Main outcome(s)	Main statistical method	Adjustments	Main finding/conclusion
Clinical cohort studies										
Heinonen [45]	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 years	Intellectual ability (7 WAIS-III subtests), executive functioning, attention, memory	Linear regression	Sex[b], age[b], full IQ[b], pregnancy conditions[c], SES[c]	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 [46]	Helsinki, Finland	Birth cohort, clinical follow-up	34–36, n = 47	37–41, 872	1934–1944, 43.7%	68.1 years	CERAD-NB cognitive test	Linear and logistic regression	Age[b], sex[b], pregnancy conditions[c], child and adult SES[c]	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 [47]	Sweden, conscripts	Register	33–34, n = 1088 35–36, n = 39,81 37–38, n = 19,146	39–41, n = 94,821	1973,1976, 100%	18–19 years	Intellectual ability	Linear regression	Year of birth, age[b], conscription office[b], SES[c], SGA[c], low Apgar[c]	Mean difference in stanine scores[d] 33–34 weeks: −0.30 (−0.41, −0.19) 35–36 weeks: −0.22 (−0.28, −0.16) 37–38 weeks: −0.07 (−0.10, −0.04)
Lundgren [48]	Sweden	Register	32–36, n = 9829	37–41, n = 209,273	1973–1978, 100%	18–19 years	Intellectual ability and psychological performance by military forces test	Logistic regression	BWSDS[c], length[c], head circumference at birth[c], current height[c]	Intellectual performance stanine score[d] 4.88 vs 5.11, OR for subnormal performance[e] 1.26 (1.21, 1.31). Psychological performance stanine score 4.92 vs 5.08, subnormal[e] 1.21 (1.16, 1.27)
Moster [49]	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[b], year of birth[b], SES[b]	Relative risk for mental retardation 1.6 (1.4, 1.8)
Svedenkrans [44]	Sweden, conscripts	Register	32–36, n = 9927	37–41, 182,477	1973–1983, n = 100%	18–19 years	Cognitive performance	Logistic regression		Mean score 2.8 vs 2.9. OR for above-average score 0.94 (0.91, 0.98)[f]

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.

[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.

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

[e] Subnormal performance = stanine scores 1 to 3.

[f] The numbers are based on stanine scores regrouped in six categories.

Table 6
Mental health.

Citation	Setting	Design	Exposure group(s), completed weeks[a]	Controls, completed weeks	Years of birth, percent of men	Mean age at outcome assessment or end of follow-up	Main outcome(s)	Main statistical method	Adjustments	Main finding/conclusion
Clinical cohort studies										
Heinonen [50]	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[b], age[b], pregnancy and neonatal conditions[b], SGA[b], LGA[b]	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)
Register studies										
Lindström [51]	Sweden	Register	33–36, *n* = 2037 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[b], sex[b], SES[b], parental psychiatric disorder[b], perinatal factors[b]	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) 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)
Nosarti [52]	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[b], parity[b] maternal age[b], SES[b], maternal psychiatric family history[b]	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.
D'Onofrio [53]	Sweden	Register	34–36, *n* = 114,890	37–42, *n* = 3,146,386	1973–2008, 51.6%	37 years ADHD, autism 19 years	Hospital in- and, since 2001, outpatient diagnoses	Cox regression, additional within-sib-pair comparisons	Year of birth[b], sex[b], birth order[b], SES[b]	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)

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Table 6 (*continued*)

Citation	Setting	Design	Exposure group(s), completed weeks[a]	Controls, completed weeks	Years of birth, percent of men	Mean age at outcome assessment or end of follow-up	Main outcome(s)	Main statistical method	Adjustments	Main finding/conclusion
Mathiasen [54]	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[c], age[c], sex[b], plurality[b], SES[b], parental mental health[b]	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)
Moster [49]	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[c], year of birth[c], SES[c]	Relative risk for: schizophrenia 1.3 (1.0, 1.7) autism spectrum disorder 0.8 (0.4, 1.4)
Silva [55]	Western Australia	Cases and controls identified and birth data obtained through registers	33–36, n = 2109 37–38, n = 8665	39–41, n = 21,094	1981–2003, 77.1%	Aug 2003–Dec 2007	Stimulant medication for ADHD	Logistic regression	Year of birth[c], SES[c]	ORs for 33–36 weeks: male 1.16 (1.05, 1.28), female 1.18 (0.97, 1.43) 37–38 weeks: male 1.12 (1.06, 1.18), female 1.14 (1.03, 1.27) All attenuated when further adjusted for prenatal factors.
Crump [56]	Sweden	Register	33–34, = 5822 35–36, n = 19,347	37–42, n = 588,410	1973–1979, 51.4%	Jul 2005–Dec 2006	Prescription of psychotropic medication	Logistic regression	Date of birth[b], sex[b], SES[b], region[b], maternal mental health[b]	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)
Engeland [19]	Norway	Register	32–34 weeks, n = 4887 35–36 weeks, n = 12,120	≥37 weeks, n = 431,914	1974–1984, 56.5%	30.5 years	Purchase of medication at least twice between 30th and 31st birthdays	Logistic regression	SES[c]	RR for 32–34 and 35–36 weeks: 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)

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Table 6 (*continued*)

Citation	Setting	Design	Exposure group(s), completed weeks[a]	Controls, completed weeks	Years of birth, percent of men	Mean age at outcome assessment or end of follow-up	Main outcome(s)	Main statistical method	Adjustments	Main finding/conclusion
Halmøy [57]	Norway	Register	33–36, n = 39,879	37–41, n = 878,458	1967–1987	Oct 1997–Apr 2005	Stimulant treatment > 18 years, physician's statement confirmed by a regional diagnostic committee	Multivariate relative risk models	Year of birth, sex[b], pregnancy conditions[b], SES[b]	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 [58]	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[c], year of birth[c], adjusted for SES[c], BWSDS[b]	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)

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

[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] Additional adjustments.

[c] Minimal adjustments.

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% (singletons alive at 1 year)

Table 7
Socio-economic outcomes.

First author [ref.]	Setting	Design (Register, clinical cohort, outcome case–control)	Exposure group(s), completed weeks[a]	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	Main finding/conclusion
Clinical cohort studies										
Ulrich [59]	Odense, Denmark	Birth cohort, survey follow-up (response rate 56%)	32–37 *n* = 69	≥38 *n* = 304	1972–1973 46.9%	31–32 years (end 2004)	Education, social status	Logistic regression	Main results shown from univariate analyses; additional adjusting for gender[b], social status at birth[b], maternal education[b], GA[b], 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 [60]	Received antenatal care and born in Johns Hopkins Hospital, Baltimore, USA	Birth cohort (randomly selected, 71.4% response rate in the follow-up), observations examinations, interviews	33–37 *n* = 226	> 37 (? "full term") *n* = 1393	1960–1965, 45.3%	27–33 years (end 1994)	Educational attainment: years of education, degrees/qualifications earned	Structural equation modelling	Mediators: Learning-related abilities (at age 7 years)[c], childhood poverty[a]	No numerical comparison of educational attainment. 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.
Register studies										
Lindström [61]	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%	23–29 years (end 2002)	Postsecondary education, employment, net salary, disposable income	Logistic regression (linear for income variables)	Age[c], gender[c]; SES[b], single-parent household in 1985[b]; residency[b], maternal age[b], parity[b], and social welfare in 1990[b]; parental psychiatric disorders[b], SGA[b], multiple birth[b]	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 (€/year) for 33–36 and 37–38: Net salary −297, −96 Disposable income −489, −236 Net transfer −326, −138 Adjusted: attenuated by ~ half

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Table 7 (*continued*)

First author [ref.]	Setting	Design (Register, clinical cohort, outcome case–control)	Exposure group(s), completed weeks[a]	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	Main finding/conclusion
Swamy [62]	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)	28–37 years (end 2004)	Education: less than high school education; graduate education	Log link RR modelling (absolute risks AR and relative risks RR with 95% CI)	Year of birth[b], maternal age[b], maternal education[b], infant sex[b]	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 about third–half
Moster [49]	Norway	Register	34–36 *n* = 32,945	≥37 *n* = 858,406	1967–1983 34–36: 55.1% ≥37: 50.9%	20–36 years (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[c], year of birth[c], multiple births[c], single motherhood[c], maternal age[c], mother's and father's level of education[c], immigrant status of the parents[c]	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% vs 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 [53]	Sweden	Register	34–36 *n* = 114,890	37–42 *n* = 3,146,386	1973–2008, 51.6%	1–36 years (end 2009)	Failing grades, low educational attainment (< 10y), higher education, social welfare benefits	Cox and logistic regression	Sex[c], birth order[c], year of birth[c]; maternal/paternal: age at the child's birth[b], highest level of education completed in 2008[b], lifetime history of any criminal conviction[b]; fixed-effects model (siblings, also cousin comparisons)[b]	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)

(*continued on next page*)

Table 7 (*continued*)

First author [ref.]	Setting	Design (Register, clinical cohort, outcome case–control)	Exposure group(s), completed weeks[a]	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	Main finding/conclusion
Heinonen [63]	Finland	Register	34–36 *n* = 486	37–41 *n* = 8507	1934–1944 53.0%	56–66 years (end 2000)	SEP: odds of belonging to lowest or highest category (also intergenerational social mobility)	Logistic regression	Gender[c], year of birth[c], father's occupational category in childhood[b], birth order[b], mother's age[b], mother's BMI at delivery[b], birth weight relative to length of gestation	OR (95% CI), ref. GA 37–41: Low: Occupational status: manual worker 21.5% vs 14.8%; 1.61 (1.26–2.05) Educational level: basic or upper secondary: 72.6% vs 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

SES, socio-economic status.

[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] Additional adjustments.

[c] Minimal adjustments.

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.

4.1. 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 socioeconomic 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.

4.2. 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.

Conflicts of interest

None declared.

Funding sources

None.

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