









«ΣΥΝΕΠΕΙΕΣ ΤΗΣ ΠΡΟΩΡΟΤΗΤΑΣ ΣΤΗΝ ΕΝΗΛΙΚΟ ΖΩΗ»

Μακροχρόνιες καρδιαγγειακές διαταραχές της προωρότητας

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Λάρισα, 01/06/2022

Προωρότητα

Key facts

- An estimated 13.4 million babies were born preterm in 2020 (before 37 completed weeks of gestation) (1).
- Preterm birth complications are the leading cause of death among children under 5 years of age, responsible for approximately 900 000 deaths in 2019 (2).
- Three-quarters of these deaths could be prevented with current, cost-effective interventions.
- Across countries, the rate of preterm birth ranges from 4–16% of babies born in 2020.

Overview

Preterm is defined as babies born alive before 37 weeks of pregnancy are completed. There are subcategories of preterm birth, based on gestational age:

- extremely preterm (less than 28 weeks)
- very preterm (28 to less than 32 weeks)
- moderate to late preterm (32 to 37 weeks).

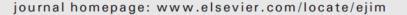
Babies may be born preterm because of spontaneous preterm labour or because there is a medical indication to plan an induction of labour or caesarean birth early.





Contents lists available at SciVerse ScienceDirect

European Journal of Internal Medicine





Review article

Consequences of prematurity on adult morbidities

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ARTICLE INFO

Article history: Received 24 December 2012 Received in revised form 7 January 2013 Accepted 10 January 2013

Keywords: Prematurity Long term health Programming

Available online xxxx

ABSTRACT

Early life events play a key role in the development of adult diseases. Survival is promoted by the developmental adaptation to environment although advantage in the short term implies a long-term cost with regard to the development of adult morbidities. This is particularly true for the infant born prematurely as preterm birth is accompanied by a disruption of the normal organogenesis of multiple organ systems.

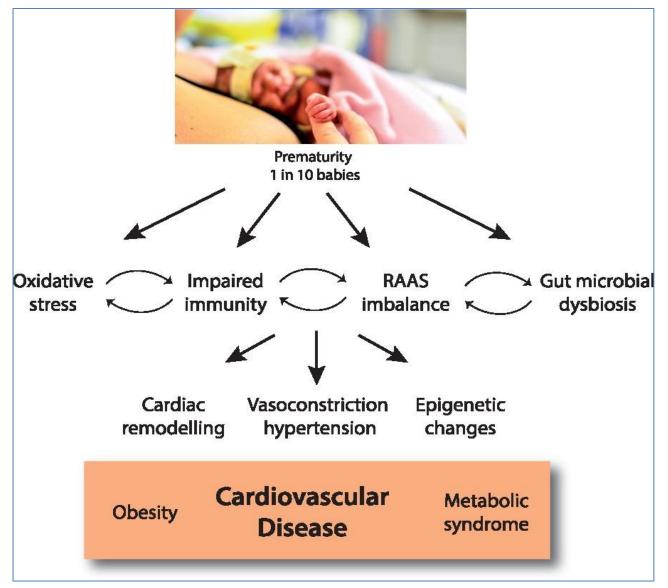
This review will examine the effect of prematurity on the development of cardiovascular, kidney, respiratory and metabolic diseases in later life in addition to the neurodevelopment disabilities associated with preterm birth.

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Παθοφυσιολογία

- Οξειδωτικό στρες
- Φλεγμονή
- Δυσλιπιδαιμία
- Ανάπτυξη μετά τη γέννηση και μεταβολικό σύνδρομο
- Ο ρόλος του συστήματος ρενίνης- αγγειοτενσίνης- αλδοστερόνης
- Γενετική και επιγενετική
- Μικροβίωμα και αθηροσκλήρωση

Παθοφυσιολογικοί μηχανισμοί που συνδέουν την πρόωρη γέννηση και την καρδιαγγειακή νοσηρότητα



Epub 2021 Nov 22.

The association between late preterm birth and cardiometabolic conditions across the life course: A systematic review and meta-analysis

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Affiliations + expand

PMID: 34806197 DOI: 10.1111/ppe.12831
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Abstract

Background: The effect of being born late preterm (34-36 weeks gestation) on cardiometabolic outcomes across the life course is unclear.

Objectives: To systematically review the association between being born late preterm (spontaneous or indicated), compared to the term and cardiometabolic outcomes in children and adults.

Data sources: EMBASE(Ovid), MEDLINE(Ovid), CINAHL.

Study selection and data extraction: Observational studies up to July 2021 were included. Study characteristics, gestational age, cardiometabolic outcomes, risk ratios (RRs), odds ratios (ORs), hazard ratios (HRs), mean differences and 95% confidence intervals (CIs) were extracted.

Synthesis: We pooled converted RRs using random-effects meta-analyses for diabetes, hypertension, ischemic heart disease (IHD) and body mass index (BMI) with subgroups for children and adults. The risk of bias was assessed using the Newcastle-Ottawa scale and certainty of the evidence was assessed using the grading of recommendations, assessment, development and evaluation (GRADE) approach.

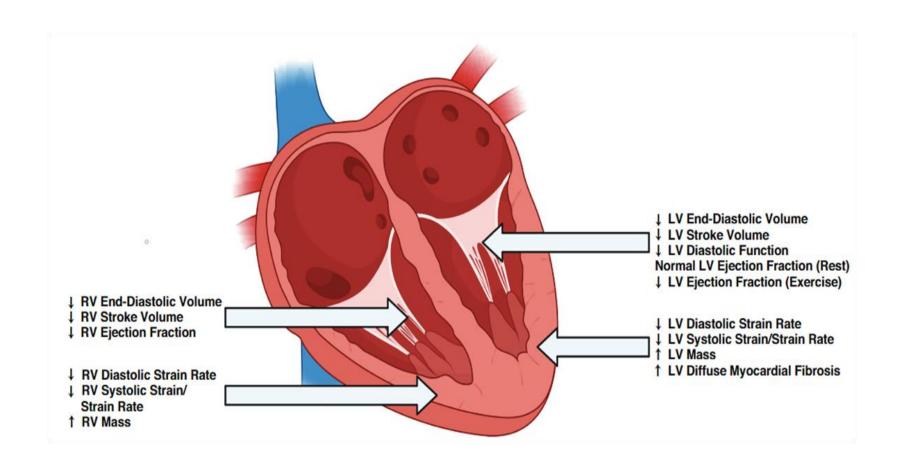
Results: Forty-one studies were included (41,203,468 total participants; median: 5.0% late preterm). Late preterm birth was associated with increased diabetes (RR 1.24, 95% CI 1.17, 1.32; nine studies; n = 6,056,511; incidence 0.9%; I² 51%; low certainty) and hypertension (RR 1.21, 95% CI 1.13, 1.30; 11 studies; n = 3,983,141; incidence 3.4%; I² 64%; low certainty) in children and adults combined. Late preterm birth was associated with decreased BMI z-scores in children (standard mean difference -0.38; 95% CI -0.67, -0.09; five studies; n = 32,602; proportion late preterm 8.3%; I² 96%; very low certainty). There was insufficient evidence that late preterm birth was associated with increased IHD risk in adults (HR 1.20, 95% CI 0.89, 1.62; four studies; n = 2,706,806; incidence 0.3%; I² 87%; very low certainty).

Conclusions: Late preterm birth was associated with an increased risk of diabetes and hypertension. The certainty of the evidence was low or very low. Inconsistencies in late preterm and term definitions, confounding variables and outcome age limited the comparability of studies.

Relation between late preterm birth and incidence of hypertension (late preterm birth vs. term birth) in children <18 years and adults ≥18 year

	Number (%)	of Outcome	Risk Ratio		Risk Ratio
Study or Subgroup	Unexposed	Exposed	IV, Random, 95% CI	Weight	IV, Random, 95% CI
Children (<18 years)					
Berard 2012	N/R	N/R	1.03 [0.56, 1.88]	1.3%	
Bergmann 2017	9.9	11.1	1.12 [0.97, 1.29]	12.7%	 -
Guttier 2019	N/R	N/R	1.83 [0.93, 3.60]	1.0%	-
Park 2019	39.1	26.8	0.69 [0.40, 1.16]	1.6%	
Sipola-Leppanen 2014 Subtotal (95% CI)	3.9	6.6	1.68 [0.98, 2.88] 1.16 [0.88, 1.52]	1.6% 18.2%	•
Heterogeneity: Tau² =	0.04; df = 4; I ²	= 47%			
Adults (≥18 years)					
Bonamy 2008	13.1	14.4	1.10 [0.96, 1.27]	12.7%	 -
Crump 2011b	1.5	1.8	1.24 [1.12, 1.37]	17.3%	-
Crump 2020b	1.4	1.8	1.28 [1.23, 1.33]	24.8%	
Johansson 2005	20.2	23.5	1.19 [1.15, 1.24]	24.8%	
Sipola-Leppanen 2015	a N/R	N/R	4.30 [1.92, 9.61]	0.7%	
Sipola-Leppanen 2015	b N/R	N/R	1.53 [0.87, 2.69]	1.5%	+
Subtotal (95% CI)			1.23 [1.14, 1.32]	81.8%	♦
Heterogeneity: Tau² = 0	0.00; df = 5; I ²	= 73%	-		
Total (95% CI)			1.21 [1.13, 1.30]	100.0%	•
Heterogeneity: Tau² = 0	0.00; df = 10; I	² = 64%			0.1 0.2 0.5 1 2 5 1 Favours Term Favours Late Preterm

Observed cardiac structural and functional changes in young adults born preterm



Pregnancy Complications and Risk of Cardiovascular Disease Later in Life: A Nationwide Cohort Study

Elin Täufer Cederlöf ¹, Maria Lundgren ², Bertil Lindahl ^{1 3}, Christina Christersson ¹

Affiliations + expand

PMID: 35014876 DOI: 10.1161/JAHA.121.023079

Free article

Abstract

Background The aim of this study was to investigate the associations between pregnancy complications and cardiovascular mortality and hospitalizations of cardiovascular disease (CVD) after adjustment for major confounding. Methods and Results In a nationwide register-based cohort study, women with singleton births between 1973 and 2014 were included from the Swedish Medical Birth Register. Outcomes of mortality and hospitalizations of CVD were collected from the Cause of Death Register and the National Inpatient Register. The cohort was followed from the date of the first delivery until death or end of follow-up, whichever occurred first. The pregnancy complications studied were preeclampsia or eclampsia, gestational hypertension, gestational diabetes, preterm birth, small for gestational age, and stillbirth. Among the 2 134 239 women (mean age at first pregnancy, 27.0 [SD, 5.1] and mean parity 1.96 [SD, 0.9]), 19.1% (N=407 597) had 1 of the studied pregnancy complications. All pregnancy complications were associated with all-cause and cardiovascular mortality and hospitalization for CVD (ischemic heart disease, ischemic stroke, and peripheral artery disease) after adjustment for major confounding in a Cox proportional hazard regression model. The adjusted hazard ratio for cardiovascular mortality was 1.84 (95% CI, 1.38-2.44) for preterm birth and 3.14 (95% CI, 1.81-5.44) for stillbirth. Conclusions In this large cohort study, pregnancy complications were associated with all-cause mortality, cardiovascular mortality, and hospitalizations for CVD, also after adjusting for confounding, including overweight, smoking, and comorbidities. The study highlights that less established pregnancy complications such as preterm birth and stillbirth are also associated with cardiovascular mortality and CVD.

Keywords: cardiovascular disease; ischemic heart disease; ischemic stroke; peripheral artery disease; pregnancy complications.

> J Am Heart Assoc. 2022 Jan 18;11(2):e023079. doi: 10.1161/JAHA.121.023079. Epub 2022 Jan 11.

Pregnancy Complications and Risk of Cardiovascular Disease Later in Life: A Nationwide Cohort Study

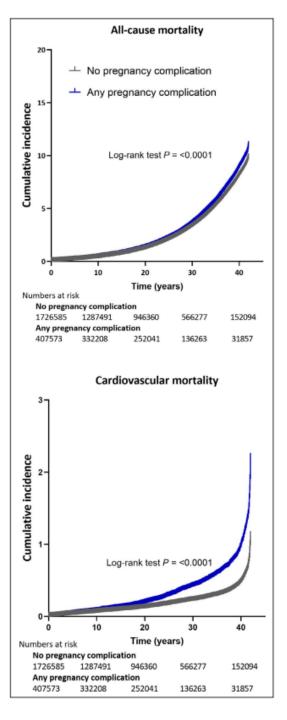
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Elin Täufer Cederlöf <sup>1</sup>, Maria Lundgren <sup>2</sup>, Bertil Lindahl <sup>1</sup> <sup>3</sup>, Christina Christersson <sup>1</sup>
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Affiliations + expand

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Free article

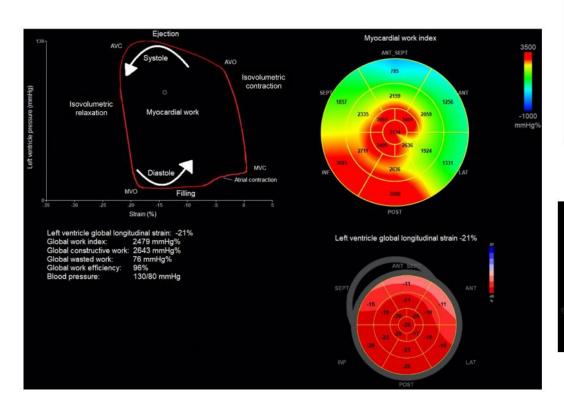
 The adjusted hazard ratio for cardiovascular mortality was 1.84 (95% CI, 1.38–2.44) for preterm birth

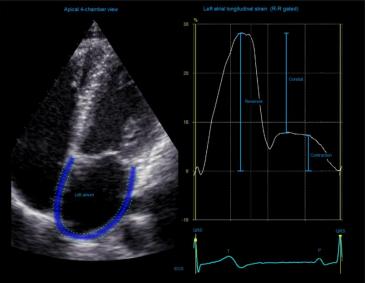


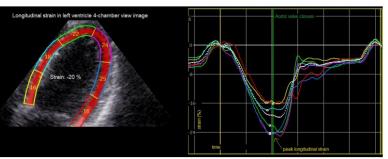
RESEARCH Open Access

Myocardial function including estimates of myocardial work in young adults born very preterm or with extremely low birthweight - a cohort study

Britt Engan^{1,2*}, Tom R. Omdal², Gottfried Greve^{1,2}, Maria Vollsaeter^{1,3} and Elisabeth Leirgul²







ORIGINAL ARTICLES

Does Preterm Birth Influence Cardiovascular Risk in Early Adulthood?

Gerthe F. Kerkhof, MSc, Petra E. Breukhoven, MD, Ralph W. J. Leunissen, MD, PhD, Ruben H. Willemsen, MD, PhD, and Anita C. S. Hokken-Koelega, MD, PhD

Objective To investigate the effect of preterm birth on risk factors for cardiovascular disease (CVD), independent of birth size.

Study design Observational study using data of 406 healthy participants aged 18-24 years, from the PROgramming factors for Growth And Metabolism and Prematurity and Small for Gestational Age studies. Associations between gestational age (GA), systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse pressure (PP), blood pressure variability, heart rate (HR), pulse wave velocity, and carotid intima media thickness (cIMT) were studied. To study the differential effects of preterm birth and small birth size for gestational age, these parameters were also analyzed in subgroups born either preterm or term: young adults born small for gestational age with short or normal adult stature, and young adults born appropriate for gestational age with normal adult stature.

Results Subjects born preterm (GA <36 weeks) had higher unadjusted SBP, PP, SBP and DBP variability, and HR, but a lower DBP than subjects born term. GA was inversely associated with SBP, PP, blood pressure variability, and HR, and positively associated with DBP, also after adjustment for confounders. There was no effect of GA on pulse wave velocity and cIMT, a marker of atherosclerosis. Of all the CVD risk factors measured, higher PP affected cIMT the most.

Conclusions Young adults born preterm might have a higher risk for CVD than those born term. (*J Pediatr* 2012;161:390-6).

Patent Ductus Arteriosus of the Preterm Infant

Shannon E G Hamrick ^{1 2}, Hannes Sallmon ³, Allison T Rose ¹, Diego Porras ⁴, Elaine L Shelton ⁵, Jeff Reese ⁵, Georg Hansmann ⁶

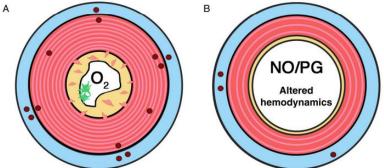
Affiliations + expand

PMID: 33093140 PMCID: PMC7605084 DOI: 10.1542/peds.2020-1209

Free PMC article

Abstract

Postnatal ductal closure is stimulated by rising oxygen tension and withdrawal of vasodilatory mediators (prostaglandins, nitric oxide, adenosine) and by vasoconstrictors (endothelin-1, catecholamines, contractile prostanoids), ion channels, calcium flux, platelets, morphologic maturity, and a favorable genetic predisposition. A persistently patent ductus arteriosus (PDA) in preterm infants can have clinical consequences. Decreasing pulmonary vascular resistance, especially in extremely low gestational age newborns, increases left-to-right shunting through the ductus and increases pulmonary blood flow further, leading to interstitial pulmonary edema and volume load to the left heart. Potential consequences of left-to-right shunting via a hemodynamically significant patent ductus arteriosus (hsPDA) include increased risk for prolonged ventilation, bronchopulmonary dysplasia, necrotizing enterocolitis or focal intestinal perforation, intraventricular hemorrhage, and death. In the last decade, there has been a trend toward less aggressive treatment of PDA in preterm infants. However, there is a subgroup of infants who will likely benefit from intervention, be it pharmacologic, interventional, or surgical: (1) prophylactic intravenous indomethacin in highly selected extremely low gestational age newborns with PDA (<26 + 0/7 weeks' gestation, <750 g birth weight), (2) early targeted therapy of PDA in selected preterm infants at particular high risk for PDAassociated complications, and (3) PDA ligation, catheter intervention, or oral paracetamol may be considered as rescue options for hsPDA closure. The impact of catheter-based closure of hsPDA on clinical outcomes should be determined in future prospective studies. Finally, we provide a novel treatment algorithm for PDA in preterm infants that integrates the several treatment modalities in a staged approach.



Factors Promoting Postnatal DA Closure	Factors Promoting Preterm DA Patency				
Molec	ular Factors				
Increased O ₂ tension Decreased vasodilating prostaglandins Activation of cytochrome P450 Increased endothelin-1 levels Production of isoprostanes (8-iso-PGF2ct) Inhibition of potassium channels (K _{ATP} , Kv. BK _{Ca}) Activation of transient receptor potential channels Decrease in intracellular cAMP and/or cGMP levels Angiotensin II Bradykinin Acetylcholine Norepinephrine Activation of RhoA, RhoB, Rock1, and Rock2	Hypoxia Increased nitric oxide signaling Increased prostaglandin signaling				
Physiol	ogic Factors				
Decreased pulmonary vascular resistance Increased systemic vascular resistance	Prolonged bidirectional or right-to-left blood flow Low-velocity blood flow				
Struct	ural Factors				
Mature contractile smooth muscle cells Prominent intimal cushions Vasa vasorum Zone of ischemia and/or necrosis Platelet adherence to lumen	Thin layer or immature smooth muscle Insufficient intimal cushion development Thrombocytopenia or platelet dysfunction				



POPULATION STUDY ARTICLE

Electrocardiographic features at rest and during exercise in young adults born preterm below 30 weeks of gestation

Anne-Sophie Gervais¹, Adrien Flahault¹, Tevy Chan¹, Camille Bastien-Tardif¹, Amy Al-Simaani¹, Anik Cloutier¹, Thuy Mai Luu¹, Sylvia Abadir^{1,2} and Anne-Monique Nuyt¹

BACKGROUND: Preterm birth has adverse consequences on the cardiovascular system. Whether premature birth is associated with conduction and repolarisation abnormalities past childhood and into adulthood still needs to be demonstrated.

METHODS: We analyzed the ECG of young adults $(23.9 \pm 3.1 \text{ years})$ born term ($\geq 37 \text{ weeks}$, n = 53) and preterm (< 30 weeks, n = 49) at rest, peak exercise and 3 min into recovery during an exercise test on a cycle ergometer. We measured PR, QRS and QT intervals, calculated the corrected QT (QTc), and determined blood calcium, magnesium, potassium and fasting glucose.

RESULTS: Mean gestational age was 39.7 ± 1.1 and 27.3 ± 1.3 weeks for the term and the preterm groups, respectively. Apart from an increased heart rate at rest in individuals born preterm, no significant difference was found between both groups for any other ECG parameters at rest. None of the participants had a severely prolonged QTc (>500 ms) at rest; exercise revealed severely prolonged QTc in two participants including one in the preterm group. The use of QT-prolonging medications did not influence ECG parameters in either groups.

CONCLUSIONS: We observed no significant difference in electrocardiographic measurements between young adults born preterm and term. Current results do not support avoidance of QT-prolonging medications in individuals born preterm.

Pediatric Research _#################; https://doi.org/10.1038/s41390-020-0814-9

IMPACT:

- Preterm birth is associated with adverse cardiovascular consequences in early adulthood, but controversial evidence exists regarding differences in electrocardiographic features between young individuals born term and preterm.
- This study aims to assess the differences in electrocardiographic features between young adults born term and preterm, at rest and during exercise training.
- In contrast with previously published data, we observed no significant difference in electrocardiographic measurements between young adults born preterm and term.
- Our study does not support that preterm birth itself exposes young adults to a higher risk of QT prolongation.
- Current results do not support avoidance of QT-prolonging medications in individuals born preterm.

Table 2. Electrocardiog	raphy meas	urements.		
Outcome measurements	Preterm	Term	<i>p</i> -value	Adjusted p-value ^{\$}
At rest	n = 49	n = 53	_	_
HR, bpm	98 ± 22	90 ± 17	0.032	0.001
QTc, ms	408 ± 34	409 ± 31	0.899	0.794
QTc mild increase*	4 (10)	4 (8)	1	_
QTc severe increase ^{&}	0 (0)	0 (0)	1	_
QRS, ms	85 ± 10	84 ± 13	0.684	0.600
PR, ms	141 ± 27	149 ± 26	0.128	0.064
At peak exercise	n = 38	n = 41	_	_
HR, bpm	180 ± 14	178 ± 13	0.631	0.335
QTc, ms	387 ± 34	387 ± 23	0.906	0.847
QTc mild increase*	0 (0)	0 (0)	1	_
QTc severe increase ^{&}	0 (0)	0 (0)	1	_
QRS, ms	75 ± 12	75 ± 10	0.773	0.988
PR, ms	104 ± 18	106 ± 15	0.683	0.634
At 3 min of recovery	n = 35	n = 43	_	_
HR, bpm	142 ± 16	138 ± 17	0.28	0.031
QTc	418 ± 35	409 ± 34	0.298	0.219
QTc mild increase*	3 (9)	1 (2)	0.31	_
QTc severe increase ^{&}	1 (3)	1 (2)	1	_
QRS, ms	83 ± 11	79 ± 14	0.271	0.169
PR, ms	131 ± 18	133 ± 16	0.658	0.254

Data are presented as mean \pm SD or n (%). p-Value calculated using Student's t-test or the Fisher exact test. HR heart rate, QTc QT corrected with Bazett's formula. *QTc mild increase: QTc > 450 ms (men) or > 460 ms (females) but <500 ms; ⁸QTc severe increase: QTc > 500 ms; ⁵Adjusted p-value: adjusted for age, sex, BMI and current tobacco smoking using a multivariate linear model.

Preterm Birth as a Risk Factor for Metabolic Syndrome and Cardiovascular Disease in Adult Life: A Systematic Review and Meta-Analysis

Panagiota Markopoulou, MD, MSc¹, Eleni Papanikolaou, PhD², Antonis Analytis, PhD³, Emmanouil Zoumakis, PhD¹, and Tania Siahanidou, MD, PhD¹

Objective To determine if preterm birth is associated with components of the metabolic syndrome in adult life. **Study design** A structured literature search was performed using PubMed. All comparative studies reported metabolic and cardiovascular outcomes in adults (≥18 years of age) born preterm (<37 weeks of gestation) compared with adults born at term (37-42 weeks of gestation) and published through March 2018 were included. The major outcomes assessed were body mass index, waist circumference, waist-to-hip ratio, fat mass, systolic blood pressure (SBP), diastolic blood pressure (DBP), 24-hour SBP, 24-hour DBP, endothelium-dependent brachial artery flow-mediated dilation, carotid intima-media thickness, pulse wave velocity, fasting glucose and insulin, Homeostasis Model Assessment-Estimated Insulin Resistance Index, and lipid profiles. Quality appraisal was performed using a modified version of the Newcastle-Ottawa scale. A meta-analysis was performed for comparable studies which reported sufficient data.

Results Forty-three studies were included, including a combined total of 18 295 preterm and 294 063 term-born adults. Prematurity was associated with significantly higher fat mass (P = .03), SBP (P < .0001), DBP (P < .0001), 24-hour SBP (P < .001), and 24-hour DBP (P < .001). Furthermore, preterm-born adults presented higher values of fasting glucose (P = .01), insulin (P = .002), Homeostasis Model Assessment-Estimated Insulin Resistance Index (P = .05), and total cholesterol levels (P = .05) in comparison with adults born at term, in random effect models. No statistically significant difference was found between preterm and term-born adults for the other outcomes studied.

Conclusions Preterm birth is strongly associated with a number of components of the metabolic syndrome and cardiovascular disease in adult life. (*J Pediatr 2019;* ■:1-12).

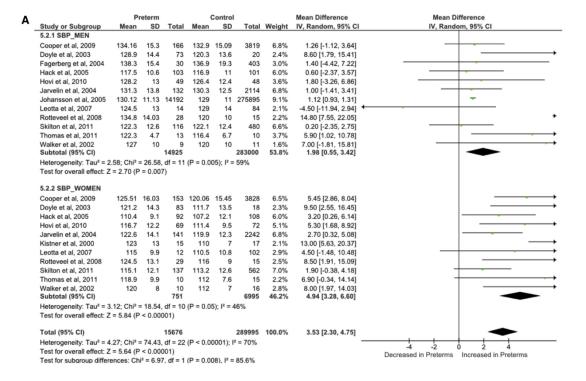
Table II. Results of meta-analyses of study outcomes: Associations between preterm birth (gestational age of <37 weeks) and components of the metabolic syndrome/cardiovascular risk factors in adult life

Outcomes	No. of studies	No. of participants	Pooled mean difference (95% CI)	FE/RE <i>P</i> value	Heterogeneity (I ² P value)	Egger test (<i>P</i> value)
ВМІ	22	P: 16 185 T: 287 296	RE: 0.04 kg/m ² (-0.22 to 0.31) FE: -0.02 kg/m ² (-0.07 to 0.04)	RE: <i>P</i> = .75 FE: <i>P</i> = .53	l ² : 35%; <i>P</i> = .05	.42
Waist circumference	7	P: 609 T: 2174	RE: 0.84 cm (-0.3 to 1.99) FE: 0.84 cm (-0.3 to 1.99)	RE: <i>P</i> = .15 FE: <i>P</i> = .15	I^2 : 0%; $P = .86$.32
WHR	4	P: 157 T: 181	RE: 0.01 (-0.02 to 0.05)	RE: $P = .52$	I^2 : 85%; $P = .0002$.70
Percent FM	9	P: 602 T: 656	RE: 1.46% (0.13 to 2.79) FE: 1.22% (0.31 to 2.14)	RE: $P = .03$ FE: $P = .009$	I^2 : 45%; $P = .07$.23
SBP	20	P: 16 520 T: 291 446	RE: 4.22 mm Hg (2.98 to 5.45)	RE: <i>P</i> < .0001	I ² : 82%; <i>P</i> < .00001	<.001
DBP	19	P: 16 201 T: 291 283	RE: 2.27 mm Hg (1.22 to 3.31)	RE: <i>P</i> < .0001	I ² : 86%; <i>P</i> < .00001	.001
24-Hour SBP	6	P: 569 T: 458	RE: 4.62 mm Hg (2.04 to 7.21)	RE: <i>P</i> = .0005	I ² : 77%; <i>P</i> < .001	.68
24-Hour DBP	6	P: 569 T: 458	RE: 1.69 mm Hg (0.89 to 2.48) FE: 1.69 mm Hg (0.89 to 2.48)	RE: <i>P</i> < .0001 FE: <i>P</i> < .0001	I^2 : 0%; $P = .62$.31
FMD*	4	P: 448 T: 1180	RE: -7% (-30 to 15)	RE: <i>P</i> = .53	I ² : 94%; <i>P</i> < .0001	.85
cIMT	4	P: 579 T: 1391	RE: 0.04 mm (-0.01 to 0.08)	RE: <i>P</i> = .1	I ² : 97%; <i>P</i> < .0001	.19
PWV	2	P: 234 T: 281	RE: 0.05 m/s (-0.12 to 0.22)	RE: <i>P</i> = .56	I^2 : 0%; $P = .33$	_
Glucose	10	P: 650 T: 2069	RE: 0.07 mmol/L (0.02 to 0.13) FE: 0.06 mmol/L (0.02 to 0.1)	RE: <i>P</i> = .01 FE: <i>P</i> = .008	I^2 : 26%; $P = .2$.31
Insulin*	8	P: 578 T: 2022	RE: 16% (6 to 26)	RE: <i>P</i> = .002	I ² : 82%; <i>P</i> < .001	.18
HOMA-IR*	4	P: 178 T: 154	RE: 24% (0 to 47)	RE: <i>P</i> = .05	I^2 : 55%; $P = .08$.76
Total cholesterol	8	P: 432 T: 740	RE: 0.17 mmol/L (0.00 to 0.34) FE: 0.17 mmol/L (0.04 to 0.29)	RE: <i>P</i> = .05 FE: <i>P</i> = .008	I ² : 40%; <i>P</i> = .11	.89
HDL	10	P: 615 T: 2400	RE: 0.03 mmol/L (-0.01 to 0.08) FE: 0.03 mmol/L (0.00 to 0.07)	RE: <i>P</i> = .13 FE: <i>P</i> = .07	I ² : 16%; <i>P</i> = .13	.52
LDL	7	P: 489 T: 1982	RE: 0.06 mmol/L (-0.04 to 0.16) FE: 0.06 mmol/L (-0.04 to 0.16)	RE: <i>P</i> = .21 FE: <i>P</i> = .21	I^2 : 0%; $P = .74$.93
TG*	10	P: 890 T: 2568	RE: 6% (-5 to 17)	RE: $P = .26$	l ² : 76%; <i>P</i> < .001	.44

	Pr	eterm		С	ontrol			Mean Difference	Mean Difference
Study or Subgroup	Mean	SD	Total	Mean	SD	Total	Weight	IV, Random, 95% CI	IV, Random, 95% CI
Fagerberg et al, 2004	6.5	1.3	35	5.9	1	361	10.3%	0.60 [0.16, 1.04]	
Hovi et al, 2011	4.6	0.8	92	4.6	0.8	68	19.4%	0.00 [-0.25, 0.25]	
Irving et al, 2000	4.3	0.9	34	4	0.9	27	9.9%	0.30 [-0.15, 0.75]	-
Kistner et al, 2004	4.5	0.7	15	4.7	0.9	17	7.4%	-0.20 [-0.76, 0.36]	-
Lewandowski et al, 2013	4.89	1.05	102	4.51	1.07	132	18.0%	0.38 [0.11, 0.65]	-
Mathai et al, 2013	4.39	1.23	31	4.65	1.14	21	5.7%	-0.26 [-0.91, 0.39]	•
Morrison et al, 2017	4.77	0.96	100	4.67	0.87	89	18.8%	0.10 [-0.16, 0.36]	-
Thomas et al, 2011	4.5	0.69	23	4.3	0.84	25	10.6%	0.20 [-0.23, 0.63]	-
Total (95% CI)			432			740	100.0%	0.17 [-0.00, 0.34]	-
Heterogeneity: Tau ² = 0.02	; Chi² =	11.63,	df = 7	(P = 0.1)	11); l ² :	= 40%			
Test for overall effect: Z =	1.96 (P =	= 0.05)		•					-0.5 -0.25 0 0.25 0.5 Decreased in Preterms Increased in Preterms

	Pr	eterm		С	ontrol			Mean Difference	Mean Difference
Study or Subgroup	Mean	SD	Total	Mean	SD	Total	Weight	IV, Fixed, 95% CI	IV, Fixed, 95% CI
Fagerberg et al, 2004	6.5	1.3	35	5.9	1	361	7.9%	0.60 [0.16, 1.04]	
Hovi et al, 2011	4.6	0.8	92	4.6	0.8	68	24.5%	0.00 [-0.25, 0.25]	
Irving et al, 2000	4.3	0.9	34	4	0.9	27	7.5%	0.30 [-0.15, 0.75]	•
Kistner et al, 2004	4.5	0.7	15	4.7	0.9	17	5.0%	-0.20 [-0.76, 0.36]	-
Lewandowski et al, 2013	4.89	1.05	102	4.51	1.07	132	20.6%	0.38 [0.11, 0.65]	-
Mathai et al, 2013	4.39	1.23	31	4.65	1.14	21	3.6%	-0.26 [-0.91, 0.39]	-
Morrison et al, 2017	4.77	0.96	100	4.67	0.87	89	22.7%	0.10 [-0.16, 0.36]	-
Thomas et al, 2011	4.5	0.69	23	4.3	0.84	25	8.2%	0.20 [-0.23, 0.63]	-
Total (95% CI)			432			740	100.0%	0.17 [0.04, 0.29]	•
Heterogeneity: Chi ² = 11.6	3, df = 7	(P = 0)	.11); l ²	= 40%					05 005 05
Test for overall effect: Z = 2	2.64 (P	0.008	3)						-0.5 -0.25 0 0.25 0.5 Decreased in Preterms Increased in Preterms

Forest plots showing the unadjusted pooled association between premature birth (gestational age of <37 weeks) and total cholesterol (random and fixed effect model). IV, inverse variance



В		P	reterm			Contro	1		Mean Difference	Mean Difference
о,	Study or Subgroup	Mean	SD	Total	Mean	SD	Total	Weight	IV, Random, 95% CI	IV, Random, 95% CI
	6.2.1 DBP_MEN									
	Cooper et al, 2009	82.99	10.95	166	82.09	10.46	3819	7.0%	0.90 [-0.80, 2.60]	
	Doyle et al, 2003	73.9	9.5	73	70.2	7.1	20	3.8%	3.70 [-0.10, 7.50]	
	Fagerberg et al, 2004	83.9	9.1	30	82.4	10	403	4.3%	1.50 [-1.90, 4.90]	
	Hack et al, 2005	73.7	8.6	103	73.1	8.6	101	5.9%	0.60 [-1.76, 2.96]	
	Hovi et al, 2010	79.8	8	49	76.9	8.1	48	4.6%	2.90 [-0.30, 6.10]	-
	Jarvelin et al, 2004	80.3	13.1	132	80.3	11.3	2114	6.0%	0.00 [-2.29, 2.29]	
	Johansson et al, 2005	67	10	14192	67	10	275895	8.8%	0.00 [-0.17, 0.17]	†
	Leotta et al, 2007	72	9.9	14	73.4	8.8	84	2.4%	-1.40 [-6.92, 4.12]	· · ·
	Rotteveel et al, 2008	70.7	8	28	64	5	15	3.7%	6.70 [2.80, 10.60]	
	Skilton et al, 2011	72.8	10.8	116	73.8	10.8	480	6.2%	-1.00 [-3.19, 1.19]	
	Thomas et al, 2011	78.3	6.3	13	71.9	4.8	10	3.1%	6.40 [1.86, 10.94]	
	Subtotal (95% CI)			14916			282989	55.7%	1.29 [0.13, 2.45]	-
	Heterogeneity: Tau ² = 1				0 (P = 0)	.001); I	² = 65%			
	Test for overall effect: 2	= 2.19 (P = 0.03	3)						
	6.2.2 DBP_WOMEN									
	Cooper et al, 2009		11.05		75.46	10.2	3828	6.9%	4.07 [2.29, 5.85]	
	Doyle et al, 2003	71.2	9.6	83	66	6.9	18	3.8%	5.20 [1.40, 9.00]	
	Hack et al, 2005	72.5	8.5	92	72.1	8.9	108	5.8%	0.40 [-2.02, 2.82]	
	Hovi et al, 2010	77.9	9.1	69	74.1	8	72	5.1%	3.80 [0.97, 6.63]	
	Jarvelin et al, 2004	75.7	10.4	141	74.7	10.9	2242	6.9%	1.00 [-0.77, 2.77]	
	Kistner et al, 2000	69	8	15	64	7	17	2.5%	5.00 [-0.24, 10.24]	
	Leotta et al, 2007	72.1	9.9	12	69.2	11	102	2.1%	2.90 [-3.09, 8.89]	
	Rotteveel et al, 2008	72.5	8	29	66	8	15	2.7%	6.50 [1.51, 11.49]	
	Skilton et al, 2011	71.2	10.2	137	69.6	10.1	562	6.6%	1.60 [-0.30, 3.50]	
	Thomas et al, 2011	74.4	7.5	10 741	69	8.5	15 6979	1.9% 44.3%	5.40 [-0.93, 11.73]	
	Subtotal (95% CI)							44.3%	2.89 [1.61, 4.16]	
	Heterogeneity: Tau ² = 1				(P = 0.0)	06); I ² =	45%			
	Test for overall effect: 2	= 4.45 (P < 0.00	0001)						
	Total (95% CI)			15657			289968	100.0%	2.13 [1.14, 3.11]	•
	Heterogeneity: Tau ² = 2	2.88; Chi ²	= 78.13	3, df = 2	0 (P < 0	.00001); I ² = 74%	6		
	Test for overall effect: Z	= 4.22 (P < 0.00	001)						-4 -2 0 2 4 Decreased in Preterms Increased in Preterms
	Test for subgroup differ	ences: C	hi ² = 3.2	29, df =	1 (P = 0	.07), I ²	= 69.6%			Decreased in Fleterins Increased in Fleterins

the unadjusted pooled association between premature birth (gestational age of <37 weeks) and A, SBP, B, DBP, in men and women.

Published in final edited form as:

Child Care Health Dev. 2019 January; 45(1): 71-78. doi:10.1111/cch.12616.

Prematurity and Cardiovascular Risk at Early Adulthood

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Abstract

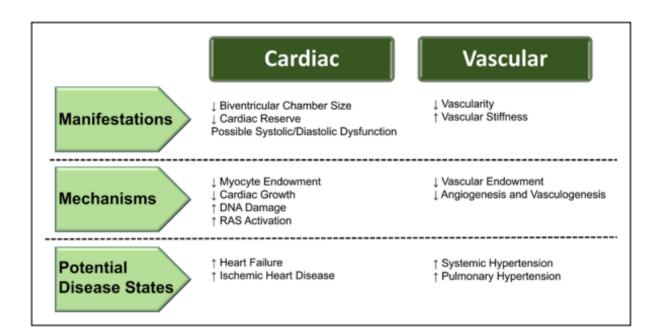
Background: Theories of early stress exposure and allostatic load offer a lifespan perspective to adult health after prematurity based on these early stressors affecting endocrine and metabolic systems. In this study, we examine cardiovascular and metabolic risk by comparing two groups of preterm infants who experienced a full spectrum of neonatal illness and a term-born group at age 23.

Methods: Of the 215 infants recruited at birth, (84%) participated at age 23. The cohort included 45 full term (FT), 24 healthy preterm (HPT), and 111 sick preterm (SPT) infants. Socioeconomic status (SES) was equivalent across groups. Cardiovascular and metabolic outcomes were: blood pressure (BP), fasting glucose and lipid profiles, weight, waist-hip ratio (WHR), and body mass index (BMI). Clinical and sub-clinical ranges were compared across neonatal groups and gender.

Results: At age 23, the HPT and SPT groups had higher systolic BP compared to the FT group. The SPT group had lower weight compared to the FT and HPT groups.

No group differences were found on diastolic BP, glucose, total cholesterol, HDL, LDL, triglycerides, BMI or WHR. Preterm males had more systolic hypertension and low HDL than FT males. Former preterm males and females had high WHR ratios and BMI at 23 years. Subclinical pre-hypertensive rates were highest for the HPT female group, followed by the SPT females. Only one (4.2%) HPT adult male was clinically diabetic.

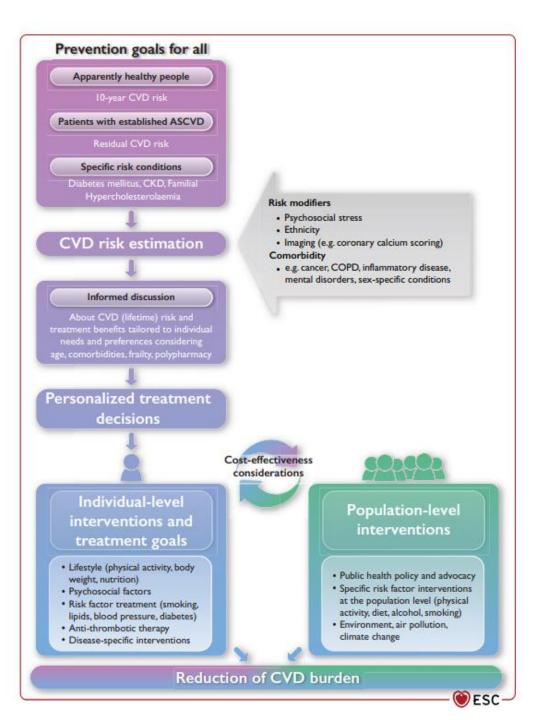
Conclusions: As young adults, HPT and SPT infants had early indicators of cardiovascular risk, but no indicators of metabolic risk. There is utility in using clinical and sub-clinical ranges to identify early cardiovascular risk in early adulthood.



Category	Definition	Disease Risk
Gestational age categories	weeks*days	
Extremely preterm birth	<28+0	
Very preterm birth	28+0 to 31+6	
Moderate to late preterm birth	32+0 to 36+6	
Term birth	>37+0	•

Πρόληψη...





Διατροφή

Recommendations for nutrition and alcohol

Recommendations	Class ^a	Level ^b
A healthy diet is recommended as a cornerstone of CVD prevention in all individuals. 401,402	1	A



Table 8 Healthy diet characteristics

Adopt a more plant- and less animal-based food pattern

Saturated fatty acids should account for <10% of total energy intake, through replacement by PUFAs, MUFAs, and carbohydrates from whole grains

Trans unsaturated fatty acids should be minimized as far as possible, with none from processed foods

<5 g total salt intake per day

30-45 g of fibre of per day, preferably from wholegrains

 \geq 200 g of fruit per day (\geq 2-3 servings)

 \geq 200 g of vegetables per day (\geq 2-3 servings)

Red meat should be reduced to a maximum of 350 - 500 g a week, in particular processed meat should be minimized

Fish is recommended 1-2 times per week, in particular fatty fish

30 g unsalted nuts per day

Consumption of alcohol should be limited to a maximum of 100 g per week

Sugar-sweetened beverages, such as soft drinks and fruit juices, must be discouraged

MUFA = monounsaturated fatty acid; PUFA = polyunsaturated fatty acid.

Σωματική άσκηση – Διακοπή καπνίσματος

Recommendations for physical activity

Recommendations	Classa	Level ^b
It is recommended for adults of all ages to strive for at least 150 - 300 min a week of moderate-intensity or 75 - 150 min a week of vigorous-intensity aerobic PA, or an equivalent combination thereof, to reduce all-cause mortality, CV mortality, and morbidity. 371,372	ı.	A
It is recommended that adults who cannot per- form 150 min of moderate-intensity PA a week should stay as active as their abilities and health condition allow. ^{373,374}	ı	В
It is recommended to reduce sedentary time to engage in at least light activity throughout the day to reduce all-cause and CV mortality and morbidity. 375–377	T.	В
Performing resistance exercise, in addition to aerobic activity, is recommended on 2 or more days per week to reduce all-cause mortality. 378,379	T.	В
Lifestyle interventions, such as group or individ- ual education, behaviour-change techniques, tel- ephone counselling, and use of consumer-based wearable activity trackers, should be considered to increase PA participation. ^{380–382}	lla	В

Recommendations for smoking intervention strategies

Recommendations	Classa	Level ^b
All smoking of tobacco should be stopped, as tobacco use is strongly and independently causal of ASCVD. 487,488	1	A
In smokers, offering follow-up support, nicotine replacement therapy, varenicline, and bupropion individually or in combination should be considered. 489–494	lla	A
Smoking cessation is recommended regardless of weight gain, as weight gain does not lessen the ASCVD benefits of cessation. 495	1	В





AHA's My Life Check – Life's Simple 7



Circulation. 2020;141:e139-e596.











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