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Comparison of three lifecourse models of poverty in predicting cardiovascular disease risk in youth

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ABSTRACT

Objective: Childhood poverty heightens the risk of adulthood cardiovascular disease (CVD), but the underlying pathways are poorly understood. Three lifecourse models have been proposed but have never been tested among youth. We assessed the longitudinal association of childhood poverty with CVD risk factors in 10-year-old youth according to the timing, accumulation, and mobility models.
Methods: The Québec Longitudinal Study of Child Development birth cohort was established in 1998 (n = 2120). Poverty was defined as annual income below the low-income thresholds defined by Statistics Canada. Multiple imputation was used for missing data. Multivariable linear regression models adjusted for gender, pubertal stage, parental education, maternal age, whether the household was a single parent household, whether the child was overweight or obese, the child's physical activity in the past week, and family history.
Results: Approximately 40% experienced poverty at least once, 16% throughout childhood, and 25% intermittently. Poverty was associated with significantly elevated triglycerides and insulin according to the timing and accumulation models, although the timing model was superior for predicting insulin and the accumulation model was superior for predicting triglycerides.
Conclusions: Early and prolonged exposure to poverty significantly increases CVD risk among 10-year-old youth.

The link between early childhood poverty and adulthood mortality from cardiovascular disease (CVD) has been well-documented: This association holds even after controlling for current income and socioeconomic status in adulthood [1,2]. However, the mechanisms through which childhood poverty affects CVD risk is less clear, with speculation that the association may depend on when, or for how long poverty persists during childhood. Three lifecourse models have been proposed to pinpoint which aspect of poverty may directly influence health: The timing model proposes that only poverty during a critical developmental period is detrimental to health [3,4], the accumulation model proposes that the effects of poverty are additive [5,6] and the mobility model proposes that it is the instability from intermittent poverty that negatively affects health [7].

Few studies have examined the association between poverty and CVD risk during childhood. These studies have been primarily cross-sectional [8–10]. Thus, we assessed the longitudinal association of poverty on CVD risk factors in an ongoing, prospective cohort study. To the best of our knowledge, the 3 conceptual models have never been tested to predict CVD risk in childhood. Because CVD risk factors have been shown to track well from childhood to adulthood [11,12], improving our understanding of the deleterious effects of poverty on CVD risk factors among youth is an important public health issue.

Methods

The Québec Longitudinal Study of Child Development (QLSCD) is a longitudinal birth cohort. Details have been previously described and only briefly presented here [13,14]. A representative sample of 5-month-old singleton children born in 1998 was selected from the Ministère de la Santé et des Services Sociaux's master birth registry, using a multistage cluster random sampling strategy. Babies with

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special needs and families living in Aboriginal territories or remote regions were excluded; the resulting sampling frame included 96.6% of the target population.

Data collection

Annual data collection at the participant's home included interviewer-administered, pretested questionnaires starting at 5 months of age. The study was approved by the ethics committees of the Institut de la Statistique du Québec, Centre Hospitalier Universitaire (CHU) Ste-Justine, and the Université de Montréal. Parents and youth provided signed informed consent and assent, respectively.

Cardiometabolic procedures

In 2008, when the children were approximately 10 years old, fasting plasma measurements of total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, triglycerides (TG), insulin, glucose, body mass index (kg/m^2), and systolic and diastolic blood pressure (SBP and DBP, respectively) were obtained and were the dependent variables in this study. SBP and DBP were measured on the right arm with an oscillometric instrument (BpTRU, model BPM-100, VSM MedTech Ltd, Vancouver, Canada) and a cuff size based on arm circumference [15]. The child was in a seated position after a 5-minute rest and $\geq$30 minutes after a light meal. Blood pressure was measured in quadruplicate in 1-minute intervals, and the mean of the last 3 measures was used. For anthropometric measurements, participants wore light indoor clothing and no shoes. Weight was measured with a calibrated spring scale to the nearest 0.1 kg and height was measured with a standard measuring tape to the nearest 0.1 cm. Measurements were done in duplicate; if they differed by $\geq$0.2 kg or $\geq$0.5 cm, a third measurement was taken. The average of the 2 closest measurements was used. body mass index was classified as overweight or obese as defined by the U.S. Centers for Disease Control and Prevention age- and gender-specific growth curves [16].

Trained nurses collected blood samples after participants had fasted overnight. Samples were immediately placed on ice, centrifuged within 30 minutes, aliquoted, and frozen on dry ice for transportation to CHU Sainte-Justine within 24 hours where they were stored at -80°C until analysis. Plasma glucose and lipid concentrations were determined on a Synchron LX20 with Beckman Instruments reagents. Plasma insulin was measured with the ultrasensitive Access immunoassay system (Beckman Coulter, Inc., Brea, CA), which has no cross-reactivity with proinsulin or C-peptide. Analyses were performed in batch at a single site (CHU Sainte-Justine Clinical Biochemistry laboratory) twice a month. Low-density lipoprotein cholesterol was calculated based on the Friedewald equation [17].

Income

Annual household income in the previous 12 months was measured in nine of the ten data collection cycles (when the child was approximately 5, 17, 29, 41, 61, 74, 86, 98, and 122 months). Annual household income was compared with the low-income cutoffs computed by Statistics Canada, which adjusts for household size and the geographic region. Households were grouped into 2 categories based on their annual income: *Adequate*, if the income was equal to or above the cutoff, and *poor*, if the income was below the cutoff [18,19].

Conceptual models

Poverty exposure was grouped into 3 time periods: Exposure between the ages of 0 and 2, exposure between the ages of 3 and 6, and exposure between the ages of 7 and 10. This categorization was necessary to test the competing lifecourse models utilizing the structured approach developed by Mishra et al. [20], which is further described below. Poverty was then operationalized in the following ways in accordance with the lifecourse models.

Timing model

No exposure (reference group) was compared with exposure during 1 of 3 time periods: Ages 0–2, ages 3–6, or ages 7–10. In accordance with the model's assumptions that duration of exposure is not a critical component, exposure to poverty was binary [3,4].

Accumulation model

No exposure (reference group) was compared with the number of time periods of exposure (0–3).

Mobility model

Children who were exposed to poverty in only 1 or 2 time periods were the risk group. In accordance with the mobility model's assumptions, participants from stable households (either with no exposure to poverty in all 3 time periods, or consistent exposure to poverty in all 3 time periods) were the reference group [7]. We tested a model of any mobility, as well as a mobility of an increasing likelihood of poverty.

Covariates

Pubertal stage (pre- or pubertal) at age 10 was self-reported: Participants were shown gender-specific drawings depicting stages of adolescent development and were asked to indicate which best corresponded with their current stage of development. These drawings were based on the Tanner stage photographs [21,22] and have been validated among youth [23]. Whether the child was overweight or obese was defined according to the U.S. Centers for Disease Control and Prevention growth curves [16], as previously described. Physical activity was assessed in self-administered questionnaires adapted from the Sallis et al. [24] checklist to reflect common activities in Québec youth. Maternal characteristics, such as maternal age, as well as parental history of diabetes, hypertension, or hypercholesterolemia, and household characteristics, such as highest education of both biologic parents, and whether the household was a 2-parent or a single parent household were reported by the person that knows the child best (>95% of the time was the mother). These covariates were selected for the multivariable models owing to their reported association in the literature or their association with cardiometabolic risk or poverty in bivariate analyses.

Data analysis

All analyses were performed with SAS 9.2 (SAS Institute, Inc., Cary, NC). Follow-up of the cohort was high until the children were approximately 5 years of age (91% retention rate). However, in a longitudinal study design, missing data are inevitable and by the age of 10, retention was 63% (n = 1334). Missing data analytic procedures were explored for 2 reasons: (1) We detected evidence of differential attrition (lower income households were more likely to be lost to follow-up), and (2) although approximately only half of the retained sample at age 10 were visited by a nurse and provided blood samples, there were no differences between the 603 with blood samples and the 1334 retained in the study at the age of 10. Multiple imputation is increasingly being used to handle missing data in longitudinal health science studies and has been shown to produce estimates that are less biased than those produced using the last observation carried forward, or complete cases, especially in circumstances of differential attrition [25,26].

Table 1
Characteristics of the analytic population

	Girls (n = 1040)*	Boys (n = 1080)
Anthropometric characteristics		
Age in months, mean (SD)	121.71 (3.1)	121.99 (3.1)
Height, mean (SD)	142.64 (6.9)	142.08 (7.1)
Weight, mean (SD)	38.46 (9.5)	38.15 (9.9)
BMI percentile, mean (SD)	61.64 (29.6)	63.13 (31.9)
BMI categories		
Normal	71.0%	63.3%
Overweight	17.4%	20.9%
Obese	11.6%	15.8%
Tanner stage: Pubertal	76.0%	78.9%
Underweight at birth	3.2%	3.3%
Lipid, metabolic and blood pressure profile, mean (SD)		
Total cholesterol (mmol/L)	4.3 (0.7)	4.2 (0.7)
High-density lipoprotein cholesterol (mmol/L)	1.4 (0.3)	1.4 (0.3)
Low-density lipoprotein cholesterol (mmol/L)	2.5 (0.7)	2.5 (0.7)
Triglycerides (mmol/L)	0.8 (0.4)	0.7 (0.3)
Insulin (pmol/L)	44.2 (26.9)	37.7 (25.9)
Glucose (mmol/L)	4.9 (0.4)	5.0 (0.4)
Systolic blood pressure (mmHg)	95.7 (10.2)	96.7 (10.8)
Diastolic blood pressure (mmHg)	60.8 (9.5)	62.4 (10.5)
Income characteristics		
Timing model		
Poverty between the ages of 0 and 2	30.7%	31.5%
Poverty between the ages of 3 and 6	28.5%	28.3%
Poverty between the ages of 7 and 10	26.3%	25.8%
Accumulation model		
Never experienced poverty	58.1%	58.7%
Poverty for 1 period	14.7%	13.3%
Poverty for 2 periods	10.8%	11.5%
Poverty for 3 periods	16.4%	16.4%
Mobility model	15.6%	16.6%
Any mobility (downward or upward)	25.5%	24.9%
Downward mobility	4.2%	3.5%

BMI = body mass index; SD = standard deviation.
* Percent after imputation of 50 datasets unless otherwise indicated.

An unbounded Markov Chain Monte Carlo method of imputation was used [25,27]. To improve model fit and convergence, the multiple imputation model included all the covariates previously mentioned in addition to the following auxiliary variables measured when the child was 10 years of age unless otherwise indicated: Whether the household was a 2-parent or a single parent household at baseline and at age 5, whether the mother was an immigrant, mother's employment status, duration of child's sleep, number of hours the child watches TV in an average week, birth order of the child, as well as measured height and weight when the child was 6, 7, and 8 years of age. The proportion of data that were multiply imputed ranged from <10% to 73% (Appendix A). Although stationary distributions and independence between imputations were found for the majority of the imputed variables by ignoring the first 200 iterations and including 50–200 iterations between imputations, to be conservative we increased these to 1000. Model convergence was met, and the autocorrelation and time-series plots indicated no systematic trends. Fifty datasets were imputed, and categorical variables were rounded based on their individualized thresholds as recommended in the literature [25]. The point estimates and standard errors from the imputed datasets were combined for univariate and multivariable analyses to produce single estimates and standard errors [28].

The conceptual models were assessed in separate linear regression models adjusted for age in addition to the previously described covariates. The SBP and DBP models also adjusted for height, and the body mass index *Z*-score models (which did not adjust for current weight status of the child) additionally adjusted for whether the mother was overweight or obese, and whether the child was born with a low birth-weight (<2500 g). Subanalyses revealed that current household income was not an independent predictor of cardiometabolic risk in multivariable analyses, and additionally adjusting for current household income did not affect results (data not shown), and was thus not included in the final model owing to a concern of overadjustment.

To test competing multivariable lifecourse models, we used the structural approach developed by Mishra et al. [20], which enables competing models to be concurrently compared. This approach considers each lifecourse model nested within a saturated model encompassing all main effects, and all possible interactions and tested with generalized *F*-statistics. Lifecourse models that were substanitally different from the saturated model were identified as inferior as this suggested meaningful information from the saturated model was lost. Thus, the best lifecourse model was one that was not different from the saturated model. Only multivariable models with poverty risk estimates that were significant were tested against the saturated model.

Results

At the time of the blood sample, the mean age of participants was 121 months, with approximately 51% of them being male (Table 1). Demographic and cardiometabolic characteristics were similar between the participants with full cardiometabolic data ("complete case" n = 603), youth that were still actively participating in data collection (n = 1334), and the full sample that was initially enrolled into the study (n = 2120; Appendix B). The proportion of youth that experienced poverty at birth in nonimputed data was 24.8% in the full sample but only 20% using the active or complete case sample, providing further evidence of differential study attrition. Approximately 40% of the sample had experienced

Table 2
Unadjusted beta estimates for the association between childhood poverty and cardiometabolic risk factors

	Total cholesterol (SE)	HDL (SE)	LDL (SE)	TG (SE)	Insulin (SE)	Glucose (SE)	BMI Z (SE)	SBP (SE)	DBP (SE)
Timing									
Exposure age 0–2†	−0.03 (0.07)	−0.05 (0.02)*	−0.03 (0.06)	0.10 (0.03)**	7.59 (2.21)**	0.003 (0.03)	0.27 (0.20)	1.20 (0.7)	0.46 (0.7)
Exposure age 3–6†	−0.02 (0.07)	−0.06 (0.03)	−0.02 (0.06)	0.11 (0.03)**	6.87 (2.13)**	−0.003 (0.03)	0.23 (0.22)	1.65 (0.8)*	0.74 (0.7)
Exposure age 7–10†	−0.03 (0.07)	−0.05 (0.03)	−0.02 (0.06)	0.08 (0.03)**	4.70 (2.17)*	−0.01 (0.03)	0.12 (0.23)	1.73 (0.8)*	0.80 (0.8)
Accumulation†	−0.01 (0.03)	−0.02 (0.01)*	−0.01 (0.02)	0.04 (0.01)**	2.95 (0.86)**	−0.001 (0.01)	0.10 (0.09)	0.70 (0.3)*	0.30 (0.3)
Mobility									
Any mobility‡	0.02 (0.05)	−0.02 (0.02)	0.03 (0.05)	0.02 (0.02)	−1.17 (1.84)	0.01 (0.03)	0.05 (0.21)	0.19 (0.6)	−0.04 (0.6)
Downward mobility§	0.02 (0.13)	0.01 (0.05)	0.04 (0.12)	−0.04 (0.06)	−5.54 (4.08)	−0.02 (0.06)	−0.22 (0.51)	−0.66 (1.6)	−0.40 (1.5)

BMI = body mass index; DBP = diastolic blood pressure; HDL = high-density lipoprotein cholesterol; LDL = low-density lipoprotein cholesterol; SBP = systolic blood pressure; SE = standard error; TG = triglycerides.
*$P < .05$; **$P < .01$; ***$P < .001$.
† Reference group of no exposure.
‡ Reference group of no mobility.
§ Reference group of no mobility or upward mobility.

Table 3
Adjusted beta estimates for the association between childhood poverty and cardiometabolic risk factors

	Total cholesterol (95% CI)	HDL (95% CI)	LDL (95% CI)	TG (95% CI)†	Insulin (95% CI)‡	Glucose (95% CI)	BMI Z (95% CI)	SBP (95% CI)	DBP (95% CI)
Timing									
Exposure age 0–2§,‖	−0.03 (−0.17, 0.11)	−0.03 (−0.08, 0.01)	−0.04 (−0.16, 0.08)	0.09 (0.02, 0.16)**	5.48 (1.26, 9.71)*	−0.002 (−0.07, 0.07)	0.24 (−0.16, 0.64)	0.41 (−1.13, 1.96)	0.12 (−1.29, 1.53)
Exposure age 3–6§,‖	−0.03 (−0.17, 0.12)	−0.04 (−0.10, 0.02)	−0.03 (−0.15, 0.09)	0.10 (0.03, 0.16)**	4.53 (0.49, 8.57)*	−0.007 (−0.08, 0.06)	0.19 (−0.27, 0.64)	0.94 (−0.66, 2.54)	0.43 (−1.09, 1.95)
Exposure age 7–10§,‖	−0.04 (−0.19, 0.10)	−0.03 (−0.09, 0.03)	−0.04 (−0.16, 0.08)	0.07 (0.003, 0.13)*	2.15 (−2.38, 6.69)	−0.01 (−0.08, 0.06)	0.05 (−0.42, 0.53)	1.12 (−0.49, 2.73)	0.51 (−1.01, 2.03)
Accumulation§,‖	−0.01 (−0.08, 0.05)	−0.02 (−0.04, 0.01)	−0.02 (−0.07, 0.03)	0.04 (0.01, 0.07)**	1.97 (0.26, 3.68)*	−0.003 (−0.03, 0.03)	0.08 (−0.10, 0.26)	0.38 (−0.27, 1.03)	0.16 (−0.43, 0.75)
Mobility									
Any mobility§,¶	0.03 (−0.07, 0.13)	−0.01 (−0.05, 0.03)	0.03 (−0.06, 0.12)	0.02 (−0.03, 0.07)	0.53 (−2.92, 3.98)	0.02 (−0.04, 0.07)	0.02 (−0.39, 0.43)	−0.17 (−1.46, 1.12)	−0.14 (−1.37, 1.09)
Downward mobility§,#	0.01 (−0.24, 0.27)	0.02 (−0.08, 0.12)	0.02 (−0.20, 0.25)	−0.06 (−0.18, 0.06)	−6.11 (−13.90, 1.69)	−0.008 (−0.13, 0.12)	−0.27 (−1.27, 0.73)	−0.70 (−3.89, 2.48)	−0.42 (−3.49, 2.65)

BMI = body mass index; CI = confidence interval; DBP = diastolic blood pressure; HDL = high-density lipoprotein cholesterol; LDL = low-density lipoprotein cholesterol; SBP = systolic blood pressure; TG = triglycerides.
*$P < .05$; **$P < .01$; ***$P < .001$.
† Only the accumulation model performed as well as the saturated model.
‡ Only the timing age 0–2 model performed as well as the saturated model.
§ Adjusted for the following age 10 covariates: Gender, maturity stage, whether ≥1 parent had at least a high school education, mother's age, whether the household was a single parent household, whether the child was overweight or obese, average number of days the child exercised ≥15 minutes at a time in an average week, and family history. SBP and DBP models also adjusted for height, and BMI *Z*-score models (which did not adjust for whether the child was overweight or obese), additionally adjusted for whether the mother was overweight or obese, and if the child was low-weight at birth. Family history was defined as history of hypercholesterolemia (total cholesterol, HDL, LDL, and TG models), diabetes (insulin, glucose models), or hypertension (systolic blood pressure, diastolic blood pressure models).
‖ Reference group of no exposure.
¶ Reference group of no mobility.
# Reference group of no mobility or upward mobility.

poverty during ≥1 period, 16% had experienced poverty in all 3 time periods, and 25% were either in a household with an increasing or decreasing likelihood of poverty.

Unadjusted linear regressions are presented (Table 2). Exposure to poverty during any time period was associated with elevated TG and insulin, as well as elevated SBP or lower high-density lipoprotein cholesterol. Prolonged exposure according to the accumulation model was associated with elevated TG, insulin, and SBP, and lower HDL. Inconsistent exposure to poverty according to the mobility models was not associated with cardiometabolic risk factors.

After adjusting for previously defined covariates, children who experienced poverty between the ages of 0 and 2 according to the timing model had 0.09 mmol/L higher TG ($P < .01$) and 5.48 pmol/L higher insulin ($P < .05$) at age 10 compared with children with no experiences of poverty (Table 3). Children with exposure between the ages of 3 and 6 had 0.10 mmol/L higher TG ($P < .01$) and 4.53 pmol/L higher insulin ($P < .01$). In accordance with the accumulation model, prolonged exposure to poverty per time period of exposure was associated with 0.04 mmol/L higher TG ($P < .01$) and 1.97 pmol/L higher insulin ($P < .05$). The mobility models were not associated with any cardiometabolic risk factors. Parental education and current income was not associated with any cardiometabolic risk factors in multivariable models.

Comparison of models

In the multivariable models TG and insulin were associated with the conceptualizations of poverty according to the timing and accumulation models and were compared with the saturated model. The other cardiometabolic risk factors were not associated with poverty and were not further assessed. Compared with the saturated model, the *F*-statistics indicated that TG was best predicted by the accumulation model, and insulin was best predicted by the timing (between the ages of 0 and 2) model.

Discussion

Although lifecourse models were originally conceptualized to examine childhood exposures on adult health, the growing literature supports their use in youth [29]. Consistent with the literature among adults, the 10-year-old participants from poor households exhibited worse TG and insulin levels than children from higher income households according to the timing and accumulation models [30,31].

Although the results were generally consistent between the timing and accumulation models, the model comparisons revealed that the accumulation model was superior for predicting TG and the early period from the timing model was superior for predicting insulin. Multiple mechanisms have been proposed to explain the increased cardiometabolic risk among children from poor households: Poorer access to sports and recreation facilities, fewer fruits or vegetables, and poorer walkability near their homes [32–34], as well as biologic mechanisms such as increased stress levels, and different phenotypic expressions of health owing to early life experiences to impoverished nutritional conditions [35]. Although these mechanisms are similar to those associated with other socioeconomic status constructs, such as education or occupation, these constructs have been shown to be distinctive from one another [36,37], with higher income in particular translating into better access to these healthier behaviors and lifestyles that decrease cardiovascular risk [32–34]. Although the results were not affected by adjusting for physical activity, we cannot adjust for diet,

or investigate these other hypothesized mechanisms. It is not clear why the superior lifecourse models differed between cardiometabolic risk factors. However, it is important to keep in mind that poverty according to both the timing and accumulation models were associated with elevated TG and insulin. Further studies on the underlying biologic mechanisms should be explored.

Because many of the children met multiple definitions of poverty, the 3 conceptual models are not mutually exclusive and it is difficult to isolate the exposure to investigate each specific lifecourse theory individually. Nevertheless, because unique information from each model can be gleaned to build a more comprehensive picture of the underlying association, Rosvall et al. [38], argue for the importance of further investigation and comparison of these models and how they relate to health. The analytic approach conceptualized by Mishra et al. [20] allows for direct comparisons of lifecourse theories to one another, and more important, may be able to disentangle the effects of exposure from 1 lifecourse model from the others. For example, in the multivariable models predicting insulin levels, the timing period between the ages of 0 and 2 was the best model, suggesting that the additional information obtained from the other lifecourse models were not informative of insulin levels above and beyond what could be gleaned from exposure between 0 and 2 years of age. Further studies are warranted to further disentangle these associations. In particular, it would be of interest to improve our understanding across the full range of income and how it relates to cardiometabolic risk.

The results of this study should be interpreted with the following limitations in mind. Although the cohort was a representative sample of singleton births in 1998 in Québec, owing to study attrition over the past 10 years, active participants are no longer representative. Thus, to account for this differential attrition, we used multiple imputation on a significant portion of biologic data. However, conclusions drawn from ignoring missing data and using only complete cases have been shown to be more biased than using multiple imputation for even $\leq$75% missing data, particularly when study attrition is owing to socioeconomic factors [39,40]. In sensitivity analyses, there were no differences in the results or conclusions when imputing data for all participants from baseline ($n = 2120$), or imputing data for only active participants ($n = 1334$; data not shown), increasing our confidence in parameter estimates presented here. Although the literature suggests associations between obesity and poverty in the transition between childhood and adulthood is not affected by race [41], we were unable to assess ethnic differences owing to a predominately Caucasian cohort. Last, the structured approach developed by Mishra et al. [20] requires a saturated model with all main effects and possible interactions. Because we had 9 years of data collection, a saturated model with all 9 main effects and possible interactions would have been very computationally intensive. However, because we recognize the strengths of utilizing Mishra et al.'s methods, which were designed to specifically compare lifecourse models to one another, we chose to instead group the data into 3 time periods. Sensitivity analyses indicated other permutations of timing periods did not affect overall results.

Conclusion

Early or prolonged exposure to poverty increases CVD risk factors among elementary school children. Results are cautionary reminders that health disparities owing to economic inequalities begin at an early age and addressing these inequalities are critical to public health.

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Appendix A

Responses at different waves of data collection

Child characteristics	n
Age	
Age at study enrollment	2120
Age at age 5	1759
Age at age 10	1334
Birthweight at study enrollment	2119
Sleep duration the previous night at age 10	797
Physical activity at age 10	964
Anthropometric measurements	
Height at age 7	1174
Weight at age 7	1171
Height at age 8	1486
Weight at age 8	1483
Height at age 9	1468
Weight at age 9	1466
Height at age 10	976
Weight at age 10	975
Pubertal status at age 10	973
Cardiometabolic risk measurements at age 10	
Total cholesterol	622
High-density lipoprotein cholesterol	622
Low-density lipoprotein cholesterol	620
Triglycerides	621
Insulin	621
Glucose	621
Systolic blood pressure	967
Diastolic blood pressure	967
Household characteristics	
Income	
Income at study enrollment	2082
Income at age 2	2015
Income at age 3	1966
Income at age 5	1913
Income at age 6	1736
Income at age 7	1480
Income at age 8	1512
Income at age 9	1438
Income at age 10	1327
Family household	
Single parent household at study enrollment	2112
Single parent household at age 5	1753
Single parent household at age 10	1328
Whether $\geq$1 parent had a high school education at study enrollment	2012
Maternal characteristics	
Immigration status at study enrollment	2118

Appendix B

Characteristics of complete case, active participants, and the complete cohort

	Complete case (n = 603)	Active participants (n = 1334)	Complete cohort* (n = 2120)
Anthropometric characteristics			
Age in months, mean (SD)	121.8 (3.0)	121.8 (3.1)	121.8 (3.1)
Height, mean (SD)	142.2 (7.3)	142.2 (7.0)	142.0 (7.0)
Weight, mean (SD)	37.3 (8.4)	38.1 (9.6)	38.1 (9.7)
Body mass index percentile, mean (SD)	51.5 (29.1)	61.5 (29.5)	62.2 (31.0)
Tanner stage: Pubertal	80.3%	78.3%	77.9%
Underweight at birth	3.5%	3.0%	3.3%
Lipid, metabolic and blood pressure profile, mean (SD)			
Total cholesterol (mmol/L)	4.2 (0.7)	4.2 (0.7)	4.2 (0.7)
High-density lipoprotein cholesterol (mmol/L)	1.4 (0.3)	1.4 (0.3)	1.4 (0.3)
Low-density lipoprotein cholesterol (mmol/L)	2.5 (0.6)	2.5 (0.7)	2.5 (0.7)
Triglycerides (mmol/L)	0.7 (0.3)	0.8 (0.4)	0.7 (0.4)
Insulin (pmol/L)	37.3 (24.9)	39.4 (26.2)	39.3 (26.6)
Glucose (mmol/L)	5.0 (0.4)	5.0 (0.4)	5.0 (0.4)
Systolic blood pressure (mmHg)	94.4 (8.8)	95.6 (10.5)	95.2 (10.5)
Diastolic blood pressure (mmHg)	60.3 (8.9)	61.3 (10.0)	61.0 (9.9)
Income characteristics			
Poverty at study enrollment (data not imputed)	20.2%	19.8%	24.8%
Timing model			
Poverty between the ages of 0 and 2	25.0%	26.2%	31.5%
Poverty between the ages of 3 and 6	21.7%	22.6%	28.4%
Poverty between the ages of 7 and 10	19.3%	19.8%	26.1%
Accumulation model			
Never experienced poverty	65.5%	64.8%	58.4%
Poverty for 1 period	12.5%	12.6%	14.0%
Poverty for 2 periods	12.2%	11.9%	11.2%
Poverty for 3 periods	9.7%	10.7%	16.4%
Mobility model			
Any mobility (downward or upward)	24.7%	24.5%	25.2%
Downward mobility	2.5%	2.8%	3.8%

SD = standard deviation.

* Percent after imputation of 50 datasets unless otherwise indicated.