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ORIGINAL ARTICLE

Poverty over the early life course and young adult cardio-metabolic risk

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Abstract

Objectives There is little known about whether exposure to family poverty at specific periods of the early life course independently contributes to coronary heart disease risk beyond the contribution of concurrent poverty.

Methods Children were recruited in early pregnancy and additional survey data obtained during the pregnancy and at the 5-, 14- and 30-year follow-ups. Fasting blood samples were also obtained at the 30-year follow-up. Analyses are multinominal logistic regressions stratified by gender and with adjustments for confounding.

Results For male offspring, family poverty at different stages of the early life course was not associated with measures of cardio-metabolic risk. For females early life course, poverty predicted obesity, homeostatic model assessment of insulin resistance (HOMA-IR) and total cholesterol/high-density lipoprotein cholesterol (TC/HDL-C), as well as concurrent family poverty associated with obesity, HOMA-IR, TC/HDL-C, HDL-C and increased systolic and diastolic blood pressure.

Conclusions Family poverty in the early life course independently predicts increased levels of cardio-metabolic risk of females. The primary finding, however, is that concurrent poverty is independently and strongly associated with increased cardio-metabolic risk levels in young adulthood.

Keywords Pregnancy · Early childhood · Adolescent period · Adulthood poverty · Cardio-metabolic risk · Gender differences

Introduction

Socioeconomic disadvantage (poverty) predicts cardiovascular disease (CVD) whether socioeconomic position is measured in childhood (Kakinami et al. 2013; Smith et al. 1998) or adulthood (Kaplan and Keil 1993), and whether CVD is measured by the presence of risk factors (Squires et al. 2000) or CVD morbidity (O'Rand and Hamil-Luker 2005) or mortality (Claussen et al. 2003). While the

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association between poverty and CVD has been well documented [see Kaplan and Keil (1993) for an early review], there remain gaps in the available literature. It is not known whether it is the age at which poverty is experienced or whether it is the cumulative period of exposure to poverty that predicts CVD (Kakinami et al. 2013). Poverty experienced during pregnancy, early childhood and adulthood is rarely distinguished in the extent to which they predict CVD. There is a need to know whether an early start to life characterised by poverty and disadvantage continues to impact on adult CVD independent of subsequent economic circumstances. No studies have addressed the possibility that poverty in the antenatal period (or early childhood) contributes to CVD risk independently of poverty at other stages in the life course. Whether exposure to poverty at various stages over the early life course predicts the biological embedding of cardiovascular disease risk well before the age that symptoms and signs of CVD become apparent (see Hertzman 2013), is also unknown.

A life course approach to CVD

From a life course perspective, adult occurring chronic disease is the end point of exposures which have occurred at previous stages of the life course (Lundberg 1993; Kuh and Ben-Shlomo 2004; Shonkoff et al. 2009). These life course exposures may reflect diverse causal pathways, e.g. sensitive periods involving foetal exposure, cumulative "weathering" following repeated exposure to adversities or indirect effects involving less healthy lifestyles exhibited by economically disadvantaged persons over an extended period of the life course (Lemelin et al. 2009).

Various life course studies of CVD have suggested that exposures associated with socioeconomic disadvantage in early life predict CVD in late adulthood (Hallqvist et al. 2004; Lynch et al. 1994; Nyström Peck 1994; Smith et al. 1998; Wannamethee et al. 1996). Barker's (Barker 2000) finding that low birthweight predicted CVD mortality, impaired glucose tolerance and non-insulin dependent diabetes stimulated a body of research addressing the early life course predictors of CVD. While poverty has been associated with impaired glucose tolerance and diabetes in studies of adults (Saydah and Lochner 2010), it is not known whether poverty during pregnancy predicts CVD when the child reaches adulthood. Exposures during pregnancy are likely to be linked to similar exposures after the child is born. It is important to distinguish the consequences of antenatal exposure to poverty from exposures at other stages of the life course.

The present study takes advantage of data provided by a long running study of early life course experiences of poverty linked to adult markers of cardio-metabolic risk. The paper specifically tests the hypothesis that antenatal and early childhood exposures to poverty differentially predict the young adult occurrence of biological changes associated with coronary heart disease (CHD) risk, that is, whether family poverty over the early life course predicts the embedding of the CHD risk by early adulthood, independent of poverty experienced at the most recent follow-up (in adulthood).

Pregnancy/early childhood exposures and CVD

There is a substantial body of research addressing the foetal and developmental origins of adult disease (Li et al. 2017). One stream of relevant research has relied upon evidence from "natural" experiments such as the Dutch (Kyle and Pichard 2006) and Chinese Famine Studies (Zhang et al. 2011). These were studies of pregnant women who experienced a starvation diet. Both these studies suggested that inadequate maternal nutrition in pregnancy predicts adult occurring cardiovascular disease. While the findings of the Leningrad Family Study were more equivocal, they have generally supported the findings derived from the Dutch and Chinese famine studies (Rotar et al. 2015). These findings have been reinforced by observational (Barker et al. 1989; Li et al. 2017) as well as laboratory studies using animal models (Li et al. 2017) and studies using population-based samples showing a consistent and specific association between low birth weight (LBW) and increased blood pressure in adults (Law and Shiell 1996). Families living in poverty are, however, unlikely to experience a rate of LBW that is comparable to the effects of a starvation diet, raising concerns about the generalisability of findings from the famine studies.

In economically developed countries, obesity disproportionately occurs in lower socioeconomic groups (Lee et al. 2009). Maternal obesity has been reported to predict childhood obesity and longer-term offspring cardio-metabolic risk (Dong et al. 2013; Drake and Reynolds 2010). In their systematic review, Senese and colleagues (2009) found an inverse association between childhood socioeconomic status (SES) and adulthood obesity in 70% (14/20) of studies in females and 27% (4/15) in males. Pollitt and colleagues (2005) found nine of eleven available observational studies reported associations between lower early life course SES and elevated body mass index (BMI) in later life.

Family poverty and sub-clinical cardiovascular disease

Research reporting an association between family poverty and indicators of sub-clinical cardiovascular disease primarily involves the use of concurrently collected measures

of family poverty (e.g. Pollitt et al. 2007; Pabayo et al. 2015).

Findings from the National Longitudinal Mortality Study found a strong association between income level and mortality due to diabetes (Saydah and Lochner 2010). Similarly data from the National Health Interview Study found that the prevalence of diabetes was twice as high in low-income families (Adams and Benson 1990). Findings from the Quebec Longitudinal Study of Child Development show that childhood poverty predicted elevated triglyceride and insulin levels (Kakinami et al. 2013). Lawlor and colleagues have also reported that adverse socioeconomic circumstances in childhood as well as adulthood predicted increased insulin resistance and other cardio-metabolic risk factors in adulthood (Lawlor and Shah 2002). Repeated studies have confirmed an association between SES and systolic (SBP) and diastolic blood pressure (DBP) (Crimmins et al. 2009; Kanjilal et al. 2006; Winkleby et al. 1992). The present study uses data on socioeconomic conditions (experiences of family poverty) recorded in early pregnancy and 5 and 14 years, as well as 30 years after the birth to predict markers of risk of cardiovascular disease 30 years after birth. The aim of the study is to identify the contribution of family poverty over the child's early life course to biological indicators of subclinical risk of cardiovascular disease when the offspring is a young adult.

Methods

Data are from the Mater-University of Queensland Study of Pregnancy (MUSP) and its outcomes (Keeping et al. 1989; Najman et al. 2015). Briefly, 8558 consecutive "public patients" attending one of the two major obstetric services in Brisbane, Australia, were invited to participate in the study. Subsequently, 7223 gave birth to a live, singleton child at the study hospital. A comparison with the population of women giving birth suggests the sample is somewhat skewed towards middle- and low-income groups. Private obstetrical patients in Australia (who were not included in the sampling frame) are fee paying and less frequently use the study hospital for routine obstetrical visits. However, babies of both public and private patients are delivered at the study hospital. Mothers were interviewed at their first clinic visit (FCV) and again at the 5- and 14-year follow-ups. In all, 72.2% of children participated in the survey at the 14-year follow-up. Figure S1 in the supplementary material provides a flowchart of sample recruitment and retention. Physical assessments and blood samples were obtained from about 1700 respondents 30 years after the recruitment of their mother to the study. Resources limits meant that respondents living in remote locations or who were more difficult to follow-up were disproportionately lost to follow-up. As Figure S1 indicates, not all respondents who provided physical assessment data also provided a blood sample. Analyses are limited to those respondents who provided both physical assessments and blood samples.

At each phase of maternal data collection (FCV, 5 and 14 years), respondents were asked the same question about their whole family income, including spouse's income. Respondents were provided with a list of categorised responses. Based upon Australian national data, the lowest 20–25% (depends upon distribution of the data) of family incomes were used to determine those living at or below the Australian poverty level. These cut-offs are consistent with the National Poverty Line in Australia (Melbourne Institute of Applied Economic and Social Research 2013). At the 30-year follow-up, the offspring were asked their own, as well as their partner's income. The amounts were totalled, and using the criteria previously applied to mothers, the income variable was dichotomised into those living at or below the poverty line.

A number of markers of cardio-metabolic risk are used in this study, all of which have been associated with the metabolic syndrome. Respondents' height was measured using a portable stadiometer. BMI was calculated (weight kg/height^2) using weight measured with the Tanita Body Composition Analyser BC-418. Obesity was defined using standard criteria provided by Cole et al. (2000).

Insulin, glucose, the ratio of total cholesterol to high-density lipoprotein cholesterol (TC/HDL-C) levels were measured in a fasting blood sample. Respondents were asked to eat by 7 pm the previous evening and fast for at least 9 h before blood sampling. Samples were collected by Mater Pathology Service, Brisbane. Respondents who lived outside the Brisbane area had their samples obtained by a participating laboratory. Homeostatic model assessment of insulin resistance (HOMA-IR) was estimated using the formula fasting insulin (mlU/L) × fasting glucose (mmol/L)/22.5 and then divided into quartiles. While there is some debate about the preferred measure of insulin resistance (Wallace et al. 2004), homeostatic model assessment (HOMA) has been generally accepted. Cholesterol oxidase/peroxidase, lipase GK/GPO/peroxidase and phosphating state/Mg^{2+} methods were used to assess serum cholesterol, triglycerides (Tg) and high-density lipoproteins (HDL) using Ortho Clinical Diagnostics Vitros Analyser. Total cholesterol: HDL ratio (TC/HDL-C) was calculated. Insulin was assessed by immuno-chemiluminescence assay (ICMA) based upon the WHO 1st IRP method using Abbott Architect Immunochemistry.

Blood pressure was measured using the OMRON HEM-703C automatic blood pressure monitor (Omron Healthcare, Lake Forest, IL). Two blood pressures were taken

5 min apart with the subject seated and at rest. Cuff sizes used were adjusted to the arm circumference of the subject.

Statistical methods

After comparing male and female respondents on markers of CHD risk at the 30-year follow-up, we use multinomial logistic regression, with poverty measured at four stages of the life course to predict indicators of CHD risk at the 30-year follow-up. All associations which are presented in Tables 1, 2, 3, 4, 5 and 6 involve adjustment for poverty at other stages of the life course as well as offspring age and marital status. With the exception of BMI, all markers of risk of CHD are presented in quartiles of risk from low to high. Respondents who reported being treated in the last year either for a diabetic condition, high blood pressure or a heart condition were excluded from the analyses. Those excluded reported receiving recent treatment for the following conditions; diabetes (ten); high blood pressure (twenty-one); irregular heart rhythm (nine) and thrombosis (five). A total of forty-five respondents were excluded because their biological markers of CVD may have been altered as a consequence of their treatment. There were no material differences between findings which included and excluded those in the sample who reported receiving treatment for a relevant condition in the previous 12 months.

Results

We initially consider gender differences in markers of coronary heart disease risk (refer Table S1 in electronic supplementary material). Male and female BMIs and Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) levels are similar at 30 years of age (Table S1 in electronic supplementary material). The female variability of BMI is greater (there appear to be more both underweight and overweight females than there are males). Males have higher TC/HDL-C mean levels, as well as higher mean DBP and SBP, but lower HDL-C. Because the mean CHD risk levels for males and females differ, the analyses which follow are stratified by gender.

Table 1 compares BMI levels by poverty experienced in pregnancy and at 5 and 14 years, as well as at 30 years. For males there is no association between poverty over the early life course and BMI at 30 years of age (adjusted analysis). For females, there are strong associations between poverty at 5 years, poverty at 30 years and obesity at 30 years. Females living in poverty early in their life course have a higher risk of obesity; in addition to concurrent poverty being related to obesity at 30 years.

The association between insulin resistance (HOMA-IR) and life course poverty is addressed in Table 2. For males, family poverty over the early life course is not associated with HOMA-IR quartile levels. For females, the associations are consistent and suggest that poverty in pregnancy, early childhood (5 years) and at the 30-year follow-up predicts the highest levels of insulin resistance. These associations are independent (and therefore cumulative) and remain after adjustment for possible confounding.

Poverty in the adolescent period is associated with the second quartile TC/HDL-C (for males), but with a wide confidence interval the lower bound of which only just achieves statistical significance (Table 3). For females, the highest quartile of TC/HDL-C scores is associated with poverty at the 5-year and 30-year follow-ups.

Table 1 Family of origin income (to 14 years) and own income (30 years) predicting respondent's body mass index (BMI) odds ratio (95% CI). From the Mater-University of Queensland Study of Pregnancy, Brisbane, Australia, 1981–2011

BMI levels in quartiles at 30 years BMI category	Family income over 14 years (maternal)				Respondent family income at 30 years
	No poverty	Poverty at FCV[a]	Poverty at 5 years	Poverty at 14 years	Poverty at 30 years
Adjusted[b]	Male (N = 482)				
Normal or below (< 24.9)	1	1	1	1	1
Overweight (25.0–29.9)	1	1.23 (0.66, 2.30)	1.02 (0.52, 2.09)	0.57 (0.28, 1.18)	0.83 (0.43, 1.57)
Obese or more (30 +)	1	1.13 (0.54, 2.35)	1.60 (0.75, 3.44)	0.89 (0.40, 1.97)	0.59 (0.26, 1.34)
Adjusted[b]	Female (N = 815)				
Normal or below (< 24.9)	1	1	1	1	1
Overweight (25.0–29.9)	1	1.19 (0.77, 1.86)	1.31 (0.75, 2.26)	0.95 (0.56, 1.61)	0.84 (0.59, 1.45)
Obese or more (30 +)	1	1.22 (0.77, 1.93)	**1.82 (1.06, 3.11)**	0.83 (0.48, 1.44)	**2.52 (1.55, 4.08)**

Bold is a statistically significant association

[a]First clinic visit

[b]Across Tables 1, 2, 3, 4, 5 and 6 models have been adjusted for poverty at all other times + adjusted for offspring age and marital status at 30 years

Table 2 Family of origin income (to 14 years) and own income (30 years) predicting respondent's HOMA-IR level (estimate of insulin resistance) odds ratio (95% CI). From the Mater-University of Queensland Study of Pregnancy, Brisbane, Australia, 1981–2011

HOMA-IR levels in quartiles at 30 years	Family income over 14 years (maternal)				Respondent family income at 30 years
HOMA-IR quartile	No poverty	Poverty at FCV[a]	Poverty at 5 years	Poverty at 14 years	Poverty at 30 years
Adjusted[b]	Male (N = 608)				
Lowest quartile (0.23–0.97)	1	1	1	1	1
Low quartile (0.98–1.38)	1	0.86 (0.45, 1.62)	0.51 (0.24, 1.07)	1.01 (0.45, 2.21)	1.24 (0.61, 2.52)
High quartile (1.39–2.04)	1	0.93 (0.51, 1.69)	0.58 (0.29, 1.14)	1.15 (0.56, 2.41)	1.19 (0.61, 2.31)
Highest quartile (2.05–58.31)	1	0.91 (0.49, 1.67)	1.20 (0.64, 2.28)	1.53 (0.76, 3.09)	1.31 (0.66, 2.58)
Adjusted[b]	Female (N = 1021)				
Lowest quartile (0.23–0.97)	1	1	1	1	1
Low quartile (0.98–1.38)	1	1.32 (0.84, 2.05)	0.98 (0.58, 1.66)	1.59 (0.94, 2.68)	0.59 (0.35, 1.02)
High quartile (1.39–2.04)	1	1.09 (0.69, 1.69)	1.05 (0.63, 1.78)	1.22 (0.72, 2.09)	1.09 (0.68, 1.77)
Highest quartile (2.05–58.31)	1	**1.72 (1.11, 2.65)**	1.36 (0.82, 2.26)	1.49 (0.89, 2.52)	**1.75 (1.10, 2.76)**

Bold is a statistically significant association

[a]First clinic visit

[b]Models have been adjusted for poverty at all other times + adjusted for offspring age and marital status at 30 years

Table 3 Family of origin income (to 14 years) and own income (30 years) predicting respondent's total cholesterol/high-density lipoprotein ratio odds ratio (95% CI). From the Mater-University of Queensland Study of Pregnancy, Brisbane, Australia, 1981–2011

Total cholesterol/HDL in quartiles at 30 years	Family income over 14 years (maternal)				Respondent family income at 30 years
TC–HDL ratio quartile	No poverty	Poverty at FCV[a]	Poverty at 5 years	Poverty at 14 years	Poverty at 30 years
Adjusted[b]	Male (N = 605)				
Lowest quartile (1.40–2.70)	1	1	1	1	1
Low quartile (2.80–3.40)	1	0.82 (0.38, 1.74)	0.43 (0.18, 1.02)	**2.96 (1.07, 8.34)**	2.06 (0.84, 5.08)
High quartile (3.50–4.20)	1	0.80 (0.38, 1.67)	0.89 (0.40, 1.99)	1.13 (0.39, 3.26)	1.34 (0.53, 3.35)
Highest quartile (4.30–13.90)	1	0.83 (0.42, 1.63)	0.74 (0.35, 1.58)	2.52 (0.96, 6.64)	1.33 (0.56, 3.15)
Adjusted[b]	Female (N = 1026)				
Lowest quartile (1.40–2.70)	1	1	1	1	1
Low quartile (2.80–3.40)	1	0.79 (0.53, 1.18)	1.00 (0.62, 1.64)	1.54 (0.97, 2.44)	1.13 (0.71, 1.78)
High quartile (3.50–4.20)	1	1.23 (0.83, 1.84)	1.53 (0.94, 2.47)	1.03 (0.63, 1.70)	1.57 (0.99, 2.47)
Highest quartile (4.30–13.90)	1	0.69 (0.43, 1.13)	**1.73 (1.00, 2.97)**	1.14 (0.65, 2.00)	**1.84 (1.10, 3.04)**

Bold is a statistically significant association

[a]First clinic visit

[b]Models have been adjusted for poverty at all other times + adjusted for offspring age and marital status at 30 years

Examining HDL cholesterol levels (Table 4), for male respondents poverty at the 5-year follow-up is lower in the second lowest HDL-C quartile group. It is relevant that the upper bound of the confidence interval is at the borderline of statistical significance. For females, concurrent poverty predicts a reduced odds of the highest quartile HDL-C levels.

In Table 5, we consider poverty over the early life course as a predictor of higher SBP at 30 years of age. For males, slightly higher (second quartile) SBP levels are associated with lower levels of poverty experienced during the adolescent period. For females, family poverty at the 30-year follow-up is associated with substantially increased levels of risk (adjusted) of higher levels of SBP.

The findings for DBP suggest that family poverty over the early life course is related to male levels of DBP, but there is a strong association of family poverty at the 30-year follow-up and increased risk of highest quartile DBP for females (Table 6).

Table 4 Family of origin income (to 14 years) and own income (30 years) predicting respondent's high-density lipoprotein cholesterol levels odds ratio (95% CI). From the Mater-University of Queensland Study of Pregnancy, Brisbane, Australia, 1981–2011

HDL[a] cholesterol levels in quartiles at 30 years HDL quartile	Family income over 14 years (maternal)				Respondent family income at 30 years Poverty at 30 years
	No poverty	Poverty at FCV[b]	Poverty at 5 years	Poverty at 14 years	
Adjusted[b]	Male (N = 606)				
Lowest quartile (0.20–1.10)	1	1	1	1	1
Low quartile (1.20–1.30)	1	0.91 (0.53, 1.52)	**0.54 (0.29, 0.98)**	1.35 (0.75, 2.45)	1.18 (0.65, 2.12)
High quartile (1.40–1.60)	1	0.98 (0.58, 1.69)	0.75 (0.43, 1.39)	1.04 (0.55, 1.96)	1.14 (0.63, 2.05)
Highest quartile (1.70–3.30)	1	0.58 (0.24, 1.31)	0.97 (0.43, 2.21)	1.15 (0.48, 2.77)	1.03 (0.43, 2.39)
Adjusted[b]	Female (N = 1027)				
Lowest quartile (0.20–1.10)	1	1	1	1	1
Low quartile (1.20–1.30)	1	0.97 (0.59, 1.58)	0.98 (0.57, 1.70)	0.74 (0.42, 1.32)	0.71 (0.43, 1.21)
High quartile (1.40–1.60)	1	0.82 (0.52, 1.36)	0.60 (0.33, 1.03)	0.86 (0.50, 1.48)	0.72 (0.45, 1.17)
Highest quartile (1.70–3.30)	1	0.96 (0.62, 1.49)	0.67 (0.37, 1.13)	1.04 (0.63, 1.76)	**0.45 (1.28, 0.72)**

Bold is a statistically significant association

[a]First clinic visit

[b]Models have been adjusted for poverty at all other times + adjusted for offspring age and marital status at 30 years

Table 5 Family of origin income (to 14 years) and own income (30 years) predicting respondent's systolic blood pressure (SBP) odds ratio (95% CI). From the Mater-University of Queensland Study of Pregnancy, Brisbane, Australia, 1981–2011

Systolic BP level in quartiles at 30 years Systolic BP quartile	Family income over 14 years (maternal)				Respondent family income at 30 years Poverty at 30 years
	No poverty	Poverty at FCV[a]	Poverty at 5 years	Poverty at 14 years	
Adjusted[b]	Male (N = 481)				
Lowest quartile (80.0–106.5)	1	1	1	1	1
Low quartile (107.0–117.5)	1	0.46 (0.12, 1.62)	1.14 (0.27, 4.45)	**0.13 (0.02, 0.69)**	0.61 (0.18, 2.00)
High quartile (118.0–129.0)	1	0.73 (0.24, 2.22)	1.37 (0.40, 4.71)	0.42 (0.13, 1.41)	0.54 (0.18, 1.64)
Highest quartile (129.5–190.0)	1	0.63 (0.21, 1.91)	0.95 (0.28, 3.20)	0.76 (0.23, 2.45)	0.50 (0.17, 1.47)
Adjusted[b]	Female (N = 791)				
Lowest quartile (80.0–106.5)	1	1	1	1	1
Low quartile (107.0–117.5)	1	1.22 (0.82, 1.82)	0.95 (0.59, 1.53)	1.01 (0.67, 1.73)	**2.09 (1.33, 3.28)**
High quartile (118.0–129.0)	1	1.43 (0.96, 2.25)	1.15 (0.68, 1.93)	0.93 (0.54, 1.61)	1.21 (0.70, 2.09)
Highest quartile (129.5–190.0)	1	1.60 (0.82, 3.15)	0.87 (0.38, 2.02)	0.50 (0.19, 1.33)	**2.86 (1.42, 5.79)**

Bold is a statistically significant association

[a]First clinic visit

[b]Models have been adjusted for poverty at all other times + adjusted for offspring age and marital status at 30 years

Discussion

We find, for males, no statistically significant associations between poverty in the early life course and risk markers for CHD (see Table S2 for summary table). Poverty in the antenatal period (both males and females) was not independently related to cardio-metabolic risk in adulthood. Our findings suggest that previous studies of such an association may reflect nuances of the samples involved (e.g. famine studies) or the selection of only one measure of poverty over the life course.

We find only limited variations in adult cardio-metabolic risk associated with family income (poverty) at the 5 year follow-up. For females (but not males), poverty at the 5 year follow-up is independently (of poverty at other life course stages) associated with adult BMI and the TC-HDL ratio in adulthood. Family income (poverty) in the early adolescent period appears to be unrelated to adult

Table 6 Family of origin income (to 14 years) and own income (30 years) predicting respondent's diastolic blood pressure (DBP) odds ratio (95% CI). From the Mater-University of Queensland Study of Pregnancy, Brisbane, Australia, 1981–2011

Diastolic BP level in quartiles at 30 years Diastolic BP quartile	Family income over 14 years (maternal)				Respondent family income at 30 years
	No poverty	Poverty at FCV[a]	Poverty at 5 years	Poverty at 14 years	Poverty at 30 years
Adjusted[b]	Male (N = 481)				
Lowest quartile (46.0–64.5)	1	1	1	1	1
Low quartile (65.0–70.5)	1	0.55 (0.25, 1.21)	1.62 (0.69, 4.04)	1.23 (0.45, 3.37)	1.25 (0.56, 2.85)
High quartile (71.0–77.5)	1	0.45 (0.21, 0.96)	1.55 (0.64, 3.75)	1.44 (0.54, 3.79)	0.94 (0.42, 2.11)
Highest quartile (78.0–122.0)	1	0.64 (0.31, 1.33)	1.63 (0.68, 3.86)	1.48 (0.58, 3.81)	1.11 (0.49, 2.47)
Adjusted[b]	Female (N = 791)				
Lowest quartile (46.0–64.5)	1	1	1	1	1
Low quartile (65.0–70.5)	1	0.99 (0.63, 1.54)	1.17 (0.69, 1.98)	0.95 (0.58, 1.57)	1.20 (0.72, 1.96)
High quartile (71.0–77.5)	1	1.22 (0.75, 1.98)	1.06 (0.59, 1.90)	1.47 (0.89, 2.42)	1.44 (0.89, 2.33)
Highest quartile (78.0–122.0)	1	1.15 (0.68, 1.94)	1.17 (0.64, 2.16)	1.64 (0.97, 2.75)	**1.90 (1.17, 3.11)**

Bold is a statistically significant association

[a]First clinic visit

[b]Models have been adjusted for poverty at all other times + adjusted for offspring age and marital status at 30 years

cardio-metabolic risk, either in male or female respondents. The most consistent set of findings concern concurrent (30 year) family poverty and increase cardio-metabolic risk, but only for female respondents.

There are two sets of findings which challenge what is currently known. The first concerns the associations that are found in females but not males; secondly why exposure to family poverty over the early life course appears to have modest impact on cardio-metabolic risk in adulthood.

There is some evidence that females might be more affected by poverty over the early life course than males (McFarland 2017). This finding needs to be considered in the context that females rarely experience adverse cardiovascular outcomes prior to the menopause (Muka et al. 2016). For the majority of poor women in this study, it will be another 20 years or more before their increased cardio-metabolic risk translates into an observable adverse outcome. There is here an opportunity to use this time period to mitigate the trajectory towards an adverse outcome. Why might females be more susceptible to the consequences of family poverty? Parents experiencing financial problems have been found to provide less consistent child rearing and less intensive monitoring of their children (Menaghan 2009). This may reflect gender differences in the rearing of children though these may be subtle (Leaper 2002). These may be expressed in such factors as the food children are given and their level of physical activity.

Despite an extensive literature suggesting a child's experiences in the first few years of life are critical to that child's future health and development (Hertzman 2013), the evidence available to support this claim is sparse. Poverty experienced in the early childhood period is associated with higher rates of child behaviour problems, both early and later in the child's life course. These early onset behaviour problems may initiate what McFarland describes as "chains of risk"; that is affected children are more likely to make decisions which have adverse consequences (McFarland 2017). For example, early onset child behaviour problems are associated with a wide range of lifestyle behaviours including tobacco, alcohol and illicit drug use (Shepherd et al. 2004). However, distinguishing these early life exposures to poverty from the impact of later life exposures has rarely been attempted and our findings suggest that if it is the later life exposures that may be more influential. Our findings suggest it is the broader behavioural and social consequences of family poverty in early adulthood that should be a focus of concern. These are likely to involve not only lifestyle factors but a range of family and work issues.

Limitations

While we find some markers of CHD associated with early life course poverty for females, this finding needs to be interpreted with caution. The number of males available for follow-up is fewer than females. The point estimates of risk rather than confidence intervals for risk suggest some similarities for males and females. This does raise the possibility that early life exposures to poverty by males fail to reach significance because of the lower sample size of

males in the study, but this is an observation which needs to be tested in a study with a larger sample of males. Another possibility is that males at the 30-year follow-up are engaged in more vigorous physical activity than females. If this were the case, it might also account for the absence of any association between early life course poverty for males. Certainly, the findings are consistent with the possibility that different lifestyle characteristics explain different findings of an association between poverty and CHD risk of male and female respondents.

The finding of an association between family poverty measured at the 5-year follow-up and markers of CHD risk at the 30-year follow-up (for females only) should also be interpreted in the context of the available measure of poverty at the 5-year follow-up. While family poverty was assessed when the child was 5 years of age, there is some stability in family poverty which suggests this measure of poverty may refer to the overall period of early childhood. We have noted that poverty in early adulthood is strongly associated with an increased level of CHD risk in early adulthood.

The finding of an association between family income and CHD risk at the 30-year follow-up (for females) is consistent with the findings of other studies (Kaplan and Keil 1993). This is effectively a cross-sectional association and suggests that current economic circumstances are the main predictor of CHD risk, independent of the economic circumstances of the family of origin of the respondent.

In this study, loss to follow-up is substantial and attrition is greatest amongst those who are the poorest (most economically disadvantaged). While loss to follow-up will affect sample means, they rarely affect estimates of association (Howe et al. 2013; Osler et al. 2008). In any event, extensive analyses of the impact of biased loss to follow-up in MUSP suggest that the estimates of association are similar in the groups retained in the study and those lost to follow-up (Saiepour et al. 2019). In these studies, we have examined numerous associations at recruitment, and then divided the sample into those subsequently retained in the study and those lost to follow-up. While the latter group is subject to biased loss to follow-up, the associations in this group are identical to the associations observed in those retained in the study. This remains the case even with 50% loss to follow-up. These findings have now been replicated (Steinhausen et al. 2020). Further, in previous papers we have used a variety of methods (multiple imputation, inverse probability weighting) to estimate the effects of biased attrition. In no instance have the imputed findings differed materially from those reported. There is also a need to reflect upon adjustment for possible confounding. We have only adjusted for poverty at other times, and respondents age and marital status. We argue that further adjustment (for example for education and say life adversities) would involve adjusting for what could be considered mediators. The issue of mediation and causal pathways is better addressed in a separate paper.

Conclusion

For many, experiences of family poverty change over the life course (Najman et al. 2018). Using repeated measures of family poverty in early pregnancy, early childhood, adolescence and adulthood, we find that experiences of poverty in the antenatal period do not appear to be related to cardio-metabolic risk when offspring are assessed at 30 years of age. Both early childhood and concurrent poverty are associated with cardio-metabolic risk in female respondents followed for 30 years. The strong associations between concurrent poverty and markers of CHD risk must raise concerns about the benefits that may be derived from early life course interventions and point to the need for interventions in early adulthood. Arguably, interventions to reduce risk of CHD need to extend over the whole life course. The associations we have observed point to such factors as diet and physical activity though the specific contribution of those and other "exposures" remains to be determined. There is also a need to test the associations we have observed in a larger sample (particularly of male children) to assess the stability of the finding.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval Data have been anonymised and irreversibly de-identified to protect the participant. For studies using pre-existing and de-identified data, formal approval from the ethics committee is not required. All phases of data collection were approved by the Human Research Ethics Committee at the University of Queensland.

Informed consent Informed consent was obtained from all participants included in the study.

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