

## Early life determinants of cardiovascular health in adulthood. The Australian Aboriginal Birth Cohort study

Pauline Sjöholm<sup>a,\*</sup>, Katja Pahkala<sup>b,c</sup>, Belinda Davison<sup>d</sup>, Markus Juonala<sup>e,f,g</sup>, Gurmeet R. Singh<sup>d,h</sup>

<sup>a</sup> Department of Medicine, University of Turku, Turku, Finland

<sup>b</sup> Research Centre of Applied and Preventive Cardiovascular Medicine, University of Turku, Turku, Finland

<sup>c</sup> Paavo Nurmi Centre, Sports & Exercise Medicine Unit, Department of Physical Activity and Health, University of Turku, Turku, Finland

<sup>d</sup> Menzies School of Health Research, Charles Darwin University, Darwin, Australia

<sup>e</sup> Department of Medicine, University of Turku, Turku, Finland

<sup>f</sup> Division of Medicine, Turku University Hospital, Turku, Finland

<sup>g</sup> Murdoch Childrens Research Institute, Parkville, Victoria, Australia

<sup>h</sup> Northern Territory Medical Program, Flinders University, Darwin, Australia

### ARTICLE INFO

#### Article history:

Received 11 April 2018

Received in revised form 28 May 2018

Accepted 8 June 2018

Available online 12 June 2018

#### Keywords:

Cardiovascular

Indigenous

Socioeconomic

Health behaviour

### ABSTRACT

**Background:** In 2010, The American Heart Association (AHA) set seven impact goals for ideal cardiovascular health (CVH): ideal blood pressure (BP), glucose, cholesterol levels and body mass index (BMI), physical activity on recommended levels, non-smoking and a healthy diet. We explored the prevalence of ideal CVH and the relationship between early life determinants and later CVH in an Aboriginal Birth Cohort in Australia.

**Methods:** The sample comprised 686 Aboriginal babies born in Darwin between 1987 and 1990. At birth, birthweight was measured and data was gathered about the families. A follow-up was conducted in adulthood. Prevalences of CVH metrics were assessed and each participant received an AHA score between 0 and 7. The relationship between socioeconomic factors at birth and later CVH was analysed.

**Results:** Ideal CVH was rare. Females had higher levels of ideal blood pressure (OR 5.87,  $P < 0.0001$ ) and lower levels of ideal physical activity (OR 0.19,  $P < 0.0001$ ). Low areal disadvantage was inversely associated with ideal levels of physical activity (OR 0.13,  $P = 0.04$ ) and ideal BP (OR 0.06,  $P = 0.04$ ). Participants living in urban environments had lower levels of ideal BP (OR 0.11,  $P = 0.03$ ). Living in large households was inversely associated with non-smoking (OR 0.22,  $P = 0.02$ ). High maternal BMI was inversely associated with ideal cholesterol (OR 0.13,  $P = 0.004$ ) and ideal BP (OR 0.20,  $P = 0.04$ ).

**Conclusions:** Several early life factors affect later CVH in this cohort. These factors could be of significance in reducing the gap in cardiovascular mortality and morbidity between the Aboriginal and the non-Aboriginal populations in Australia.

© 2018 Elsevier B.V. All rights reserved.

### 1. Introduction

Life expectancy for the Indigenous population in Australia in 2010–2012 was 10.6 years lower for males and 9.5 years lower for females when compared to the non-Indigenous population. The differences are particularly high in the Northern Territory (NT), where death rates for Indigenous Australians are 2.3 times the non-Indigenous rates. The

**Abbreviations:** ABC, Aboriginal Birth Cohort; BMI, body mass index; BP, blood pressure; CVD, cardiovascular disease; CVH, cardiovascular health; IRSEO, Indigenous Relative Socioeconomic Outcomes Index; NT, Northern Territory; OR, odds ratio; RDH, Royal Darwin Hospital.

\* Corresponding author at: Department of Medicine, University of Turku, Kiinamyllynkatu 10, FIN-20520 Turku, Finland.

E-mail address: [plsjoh@utu.fi](mailto:plsjoh@utu.fi) (P. Sjöholm).

<sup>1</sup> This author takes responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

most common cause of death among Indigenous Australians was cardiovascular disease (CVD) which explains almost one-quarter of the mortality gap [1].

The pathophysiology of CVD begins early in life and multiple factors throughout the life course affect the development of CVD. Known modifiable risk factors are tobacco smoking, sedentary lifestyle, unhealthy diet habits, psychosocial stress, excessive body fat, dyslipidemia and hypertension [2]. Associations between several in utero factors as well as environmental factors in both childhood and adulthood and later cardiovascular health have been documented. These factors include birthweight [3, 4], postnatal growth patterns [5], maternal obesity [6, 7], parental smoking [8], family socioeconomic status [8, 9] and neighborhood disadvantage [10], among others.

In 2010, the American Heart Association (AHA) introduced its impact goals for improving cardiovascular health in the US population. It defined a total of seven ideal cardiovascular health behaviours or factors

for defining and monitoring cardiovascular health. These included not smoking, being physically active, having normal weight, blood pressure, blood glucose and cholesterol levels as well as eating a healthy diet [11]. Since its release, the AHA index has been applied to several populations but never to Indigenous Australians [8, 12–14]. The Aboriginal Birth Cohort (ABC) was formed in order to better understand the reasons behind the high burden of disease of the Australian Aboriginal population and identify possibilities for early prevention. To date, the study is one of the longest running and largest Indigenous cohorts in the world. The aim of this article was to 1) describe the prevalence of ideal cardiovascular health metrics using the AHA index and its components and 2) to explore the relationship of socioeconomic factors and birthweight with cardiovascular health in adulthood in the ABC.

## 2. Methods

### 2.1. Participants

Details of the recruitment and follow-up of the ABC have been previously published in detail [15, 16]. Of all Aboriginal children born between 1987 and 1990 at the Royal Darwin Hospital, 686 of the possible 1238 were recruited into the study. There were no differences for mean birth weights or sex ratios between those recruited and those not recruited. To date, three follow-ups have been conducted: in childhood (mean age 11.4), adolescence (mean age 18.2) and most recently in early adulthood (mean age 25.4). The last follow-up took place in 2014–2016 and presented a follow-up rate of 70.9% of living participants. All procedures contributing to this work comply with the Helsinki Declaration of 1975, as revised in 2008. All participants provided written informed consent to participate in this study, and all procedures were approved by the Human Research Ethics Committee of the Northern Territory Department of Health and the Menzies School of Health Research, including the Aboriginal Ethical Sub-committee which has the power of veto.

### 2.2. AHA indicators of ideal cardiovascular health

AHA guidelines were used to construct an index of ideal cardiovascular health with possible values between 0 and 7, one point for each metric. The index was applied to the ABC in adulthood and for the total score, we excluded participants missing data on 1 or more of the metrics. Venous blood samples were taken to assess total cholesterol (TC) and plasma HbA1c. These were measured using Roche reagents (Roche Diagnostics, Basel, Switzerland) and a Hitachi 917 (Tokyo, Japan) auto-analyser. Ideal cholesterol was defined as TC < 5.17 mmol/l. Due to lacking fasting samples, glycated hemoglobin (HbA1c) was used instead of fasting plasma glucose. Ideal glucose was defined as untreated HbA1c < 5.7% according to a definition by the American Diabetes Association [17]. Ten individuals had glucose lowering medication and they were considered having non-ideal glucose status. Blood pressure was measured using an automatic oscillatory unit (Lifesigns BP Monitor, Welch Allyn, New York, USA) and ideal values were set at untreated systolic blood pressure < 120 mm Hg and untreated diastolic blood pressure < 80 mm Hg. Eight individuals had blood pressure lowering medication and they were considered having non-ideal blood pressure status. Weight was measured in light clothing while barefoot to the last complete 0.1 kg with a digital scale (TBF-521; Tanita Corporation, Arlington Heights, Illinois, USA). Height was measured with a portable stadiometer to the nearest millimetre. BMI was calculated using these measures and a value of <25 was defined as ideal. Due to a significant number of underweight participants in the cohort (BMI < 18.5, 17.3% for males and 23.7% for females), additional analyses were made where only those with a healthy BMI (18.5–25) were regarded, thus exploring the possible role of undernutrition on the results. Dietary habits, information on smoking and physical activity were obtained using a questionnaire. Due to lacking data, the AHA definition of ideal dietary habits was slightly modified for this study. Ideal dietary habits were defined through a diet score with possible values between 0 and 4. Eating at least 4 servings of fruits or vegetables per week, not consuming >1 serving of processed meats per week, eating at least 2 servings of fish weekly and not drinking >2 soft drinks per week all gave 1 point to the total score. A total diet score of 3 or 4 was defined as ideal. Participants who had never smoked or had quit >12 months earlier were classified as non-smokers. Ideal physical activity was defined as >5 h of self-reported exercise weekly.

### 2.3. Socioeconomic variables

At birth, birthweight was measured and data was gathered about the families and their living conditions. Birthweight was transformed into Z-scores and put into 5 categories. Maternal BMI was categorised as underweight (<18.5), normal (≥18.5 < 25), overweight (≥25 < 30) and obese (≥30). Families living in urban areas were classified as urban and those in remote locations as not urban. Parity of the mother at the time of birth of the participant was recorded. Household size was estimated through a questionnaire by asking the participants how many people slept in their house the night before. The number was put into four categories: 1–2, 3–5, 6–8 and 9 or more.

For areal disadvantage, the Indigenous Relative Socioeconomic Outcomes (IRSEO) index was used. It is a score calculated at the Indigenous Area level and it is based on 9 variables including 3 related to employment, 3 to education, 2 to housing and 1 to income

using information derived from the 2011 Census of Population and Housing. Each area is assigned to one of 100 percentiles, 1 for the most advantaged and 100 for the most disadvantaged [18]. Based on their reported addresses at birth, the participants were assigned an IRSEO score. The scores were categorised into four groups: least disadvantage (range 13 to 37), mid-high disadvantage (range 43 to 79), high disadvantage (range 81 to 89) and highest disadvantage (range 91 to 99).

### 2.4. Statistical analyses

Attrition analyses comparing baseline data of participants and non-participants were performed using *t*-test for continuous and  $\chi^2$ -test for categorical variables. The sex difference regarding the total AHA score was assessed with a *t*-test and the sex differences for the individual ideal cardiovascular health metrics with  $\chi^2$ -tests. Associations between socioeconomic factors and ideal cardiovascular health metrics were analysed using multivariable logistic regression. First univariate analyses adjusted for age and sex were performed. Then, multivariate models adjusted for age, gender, urban/not urban as well as categories for birthweight, maternal BMI, IRSEO score, household size and parity were analysed. Adjusted odds ratios (OR) with 95% confidence intervals (CI) were calculated for all variables. The statistical tests were performed with SAS version 9.4 (SAS Institute, Inc., Cary, NC). Statistical significance was inferred at a 2-tailed *P*-value < 0.05.

## 3. Results

The baseline characteristics of the study participants are presented in Table 1 according to sex. Among female participants, baseline household size was higher compared to males. Attrition analyses comparing baseline characteristics of follow-up study participants and non-participants were performed. Compared to non-participants, those participating in the follow-up were more often females, and they had higher IRSEO scores (supplementary material, Table 3).

### 3.1. Ideal cardiovascular health factors and behaviours

The prevalence of the individual ideal cardiovascular health metrics is presented in Fig. 1 and the distribution of the total AHA score in the cohort in Fig. 2. Only 5 participants (2.45%) met all 7 metrics for ideal cardiovascular health. One person (0.49%) met none of the metrics. As shown in Table 1, the mean total AHA scores in the cohort were  $4.7 \pm 1.3$  for males and  $3.6 \pm 1.5$  for females ( $P < 0.0001$ ). The lowest prevalences were reported for non-smoking (25.3% males vs. 31.9% females,  $P = 0.14$ ) and ideal diet (50.0% vs. 39.0%,  $P = 0.10$ ). Significant sex differences were seen in ideal physical activity (69.1% vs. 32.6%,  $P < 0.0001$ ), ideal blood pressure (56.8% vs. 86.0%,  $P < 0.0001$ ) and ideal cholesterol (69.2% vs. 80.0%,  $P = 0.01$ ). There were no significant sex differences in ideal glucose, diet, BMI or non-smoking. When analysing only participants with a BMI between 18.5 and 25, i.e. dismissing underweight participants from the analyses, there was a significant difference

**Table 1**  
Characteristics of the study participants.<sup>a</sup>

	Males	Females	P-value
N	221	246	
Birth weight, z score (N)	−0.19 ± 1.2 (201)	−0.41 ± 1.1 (237)	0.27
IRSEO score at baseline	77.0 ± 25.3(221)	78.8 ± 24.0 (246)	0.73
Parity of mother at baseline	2.8 ± 1.8 (221)	2.7 ± 1.9 (246)	0.56
Household size at baseline	5.9 ± 3.1 (197)	6.9 ± 3.6 (215)	0.006
Maternal BMI at baseline	22.0 ± 4.3 (166)	22.4 ± 4.4 (183)	0.83
Urban residence at baseline	14.9% (221)	13.8% (246)	0.73
Age at follow-up, mean ± SD	25.5 ± 1.1 (216)	25.3 ± 1.2 (243)	0.07
Ideal BMI, %	65.4% (214)	58.9% (241)	0.15
Ideal physical activity	69.1% (210)	32.6% (239)	<0.0001
Ideal diet	50.0% (106)	39.0% (123)	0.10
Non-smoking	25.3% (194)	31.9% (213)	0.14
Ideal cholesterol	69.2% (195)	80.0% (215)	0.01
Ideal blood pressure	56.8% (206)	86.0% (235)	<0.0001
Ideal glucose	84.1% (195)	81.4% (215)	0.47
Total AHA score	4.7 ± 1.3 (95)	3.6 ± 1.5 (109)	<0.0001

<sup>a</sup> Data are mean ± SD for males and females including sample number (N). BMI = body mass index. AHA = American Heart Association. IRSEO = Indigenous Relative Socioeconomic Outcomes.

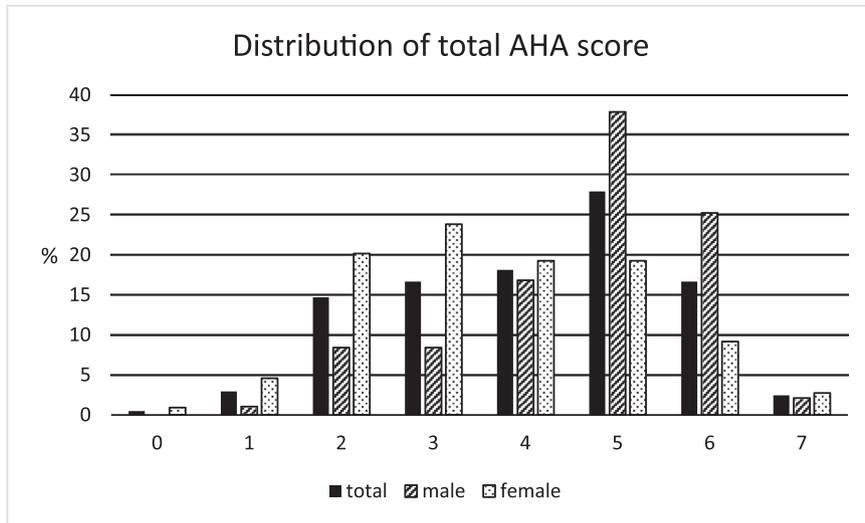


Fig. 1. Values are in percentages for the total cohort and for males and females. Mean AHA score was 4.7 for males and 3.6 for females.

between the sexes for ideal BMI: 48.1% for males and 35.3% for females ( $P = 0.005$ ). When analysing only those with a healthy BMI, the mean total AHA score was 4.5 for males and 3.4 for females ( $P < 0.0001$ ).

3.2. Socioeconomic variables and cardiovascular health in adulthood

In univariate analyses gender, areal disadvantage, urban residence and household size were associated with most AHA metrics (supplementary material, Table 4). The results of the multivariable analyses are presented in Table 2. After adjusting to all other variables, females were less likely to report ideal levels of physical activity (OR 0.19, 95% CI [0.11–0.33],  $P < 0.0001$ ) but more likely to have ideal blood pressure (OR 5.51, [2.84–10.71],  $P < 0.0001$ ). No significant association was found between birthweight and the seven cardiovascular health markers. Areal disadvantage was associated with ideal physical activity and ideal blood pressure: participants from the least disadvantaged areas had significantly lower odds for presenting ideal levels than participants

from more disadvantaged areas (OR 0.13, [0.02–0.76],  $P = 0.03$  for physical activity and OR 0.05, [0.01–0.32],  $P = 0.04$  for blood pressure). Participants from urban areas had lower odds for having ideal blood pressure (OR 0.11, [0.02–0.76],  $P = 0.03$ ) than participants from non urban areas. Being born to a family with more than six children was directly associated with ideal BMI levels (OR 3.75, [1.10–12.80],  $P = 0.04$ ). When disregarding underweight participants in the analyses, this association became non-significant (OR 1.81, [0.70–4.72],  $P = 0.17$ ). Household size was associated with smoking status: participants who slept in houses with >9 people had lower odds of being non-smokers as adults (OR 0.22, [0.06–0.76],  $P = 0.02$ ).

Compared to children of normal weight mothers, offspring of underweight mothers (BMI < 18.5) had higher odds for having ideal BMI in adulthood (OR 2.93 [1.19–7.21],  $P = 0.003$ ). When leaving underweight participants out of the analyses, this association was non-significant (OR 1.07 [0.51–2.03],  $P = 0.63$ ). Children of obese mothers (BMI > 30) had lower odds for presenting with ideal blood pressure

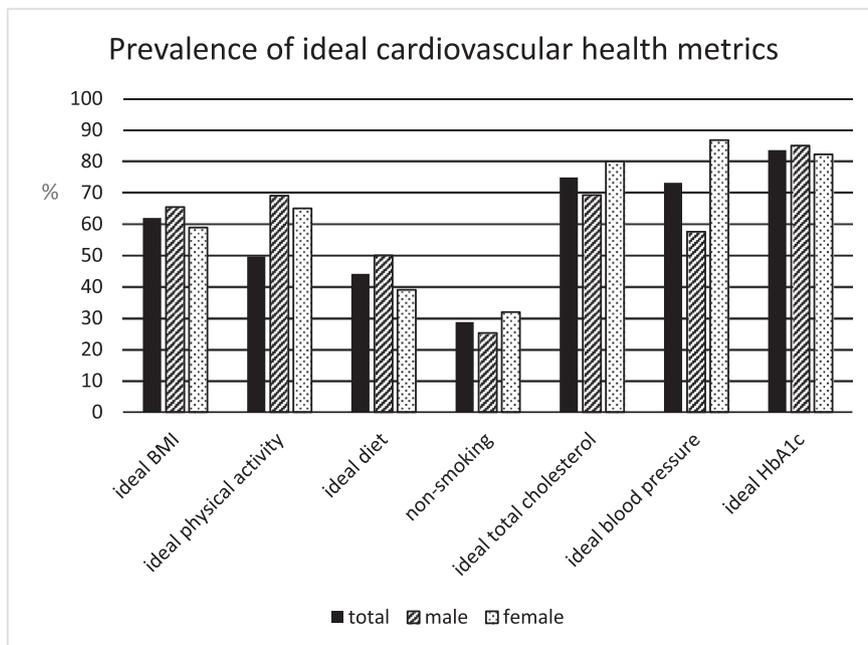


Fig. 2. Prevalences (%) for participants meeting individual health metrics in the total cohort and for males and females. BMI = body mass index.

**Table 2**  
Multivariate analyses between variables at birth and AHA index in adulthood. \*P < 0.05, \*\*P < 0.01 \*\*\*P < 0.0001. † Referent category. ‡ Adjusted for age, gender, urban/remote, categories for birthweight, areal disadvantage, household size, maternal BMI and parity of mother at the time of birth of the participant. Definitions: Least disadvantage, IRSEO score 13–37; mid-high disadvantage, IRSEO score 43–79; high disadvantage, IRSEO score 81–89; highest disadvantage, IRSEO score 91–99; number of children, number of children of mother (dead or alive) at birth of participant, underweight, BMI < 18.5; normal weight, BMI ≥ 18.5 < 25; overweight, BMI ≥ 25 < 30; obese, BMI > 30; ideal BMI < 25; ideal physical activity ≥ 5 h exercise weekly; ideal diet, at least three of following factors: ≥4 servings of fruit/vegetables weekly, ≤1 serving of processed meats weekly, ≥2 servings of fish weekly and ≤2 soft drinks weekly; ideal total cholesterol, <5.17 mmol/l; ideal blood pressure, systolic blood pressure < 120 and diastolic blood pressure < 80; ideal HbA1c < 5.15%. Sample size was not sufficient to make logistic regression analysis between HbA1c and household size.

Predictor	Ideal BMI		Ideal physical activity		Ideal diet		Non-smoking		Ideal total cholesterol		Ideal blood pressure		Ideal Hba1c	
	n(%)	AOR† (95% CI)	n(%)	AOR† (95% CI)	n(%)	AOR† (95% CI)	n(%)	AOR† (95% CI)	n(%)	AOR† (95% CI)	n(%)	AOR† (95% CI)	n(%)	AOR† (95% CI)
Sex														
Male †	140 (65.4)	1.00	145 (69.1)	1.00	53 (50.0)	1.00	49 (25.3)	1.00	135 (69.2)	1.00	117 (56.8)	1.00	164 (84.1)	1.00
Female	142 (58.9)	0.66 (0.37–1.15)	78 (32.6)	0.19 (0.11–0.33)***	48 (39.0)	0.53 (0.26–1.09)	68 (31.9)	1.41 (0.76–2.61)	172 (80.0)	1.78 (0.95–3.36)	202 (86.0)	5.51 (2.84–10.71)***	175 (81.4)	0.52 (0.26–1.04)
Birthweight (Z-score)														
<−2	23 (69.7)	0.89 (0.30–2.64)	19 (55.9)	1.54 (0.53–4.45)	9 (47.4)	1.53 (0.36–6.47)	11 (34.4)	1.76 (0.57–5.37)	23 (74.2)	0.91 (0.27–3.15)	24 (77.4)	0.1 (0.23–2.90)	26 (83.9)	1.43 (0.28–7.36)
−2 to −1	68 (76.4)	1.63 (0.77–3.45)	43 (48.3)	1.17 (0.59–2.29)	19 (38.0)	0.72 (0.29–1.78)	20 (25.6)	1.09 (0.50–2.37)	62 (78.5)	1.07 (0.47–2.48)	68 (77.3)	0.93 (0.41–2.10)	65 (82.3)	0.87 (0.37–2.07)
−1 to +1 †	146 (57.9)	1.00	115 (46.6)	1.00	52 (44.8)	1.00	65 (28.9)	1.00	173 (75.6)	1.00	184 (75.7)	1.00	190 (83.3)	1.00
+1 to +2	24 (58.5)	0.92 (0.37–2.31)	24 (60.0)	0.81 (0.33–2.03)	12 (48.0)	1.66 (0.53–5.14)	7 (19.4)	0.53 (0.15–1.86)	27 (69.2)	0.91 (0.33–2.52)	20 (51.3)	0.50 (0.19–1.32)	32 (82.1)	0.94 (0.30–2.97)
>+2	6 (46.2)	0.54 (0.12–2.46)	5 (38.5)	0.54 (0.12–2.31)	2 (40.0)	–	2 (18.2)	0.43 (0.07–2.56)	6 (54.6)	0.78 (0.14–4.28)	6 (50.0)	0.64 (0.12–3.41)	8 (72.7)	0.30 (0.05–1.80)
Areal social disadvantage														
Least disadvantage	30 (37.5)	0.09 (0.02–0.54)	27 (34.6)	0.13 (0.02–0.76)*	21 (65.6)	–	35 (44.9)	0.86 (0.18–4.18)	37 (58.7)	0.23 (0.05–1.02)	47 (60.3)	0.05 (0.01–0.32)*	56 (88.9)	0.58 (0.10–3.48)
Mid-high disadvantage	11 (45.8)	0.18 (0.03–0.44)	10 (41.7)	0.78 (0.23–2.67)	4 (40.0)	1.46 (0.25–8.58)	7 (38.9)	2.10 (0.53–8.40)	13 (61.9)	0.70 (0.17–2.96)	12 (54.6)	0.12 (0.03–0.49)	15 (71.4)	0.35 (0.08–1.53)
High disadvantage	98 (60.1)	0.48 (0.25–0.90)	80 (49.7)	0.86 (0.47–1.55)	37 (46.8)	2.24 (1.05–4.80)	33 (22.6)	0.74 (0.36–1.51)	105 (73.4)	0.90 (0.44–1.87)	107 (69.0)	0.38 (0.18–0.79)	125 (86.8)	1.86 (0.85–4.07)
Highest disadvantage †	143 (76.06)	1.00	106 (57.0)	1.00	39 (36.1)	1.00	42 (25.5)	1.00	152 (83.1)	1.00	153 (82.3)	1.00	143 (78.6)	1.00
Urban residence														
Urban †	27 (40.9)	1.00	24 (37.5)	1.00	18 (64.3)	1.00	31 (47.7)	1.00	32 (62.8)	1.00	44 (68.8)	1.00	46 (90.2)	1.00
Not urban	255 (65.6)	0.18 (0.03–1.20)	199 (51.7)	0.69 (0.10–4.84)	83 (41.3)	–	86 (25.2)	0.65 (0.11–3.74)	275 (76.6)	0.45 (0.08–2.62)	275 (72.9)	0.11 (0.02–0.76)*	293 (81.6)	0.27 (0.03–2.96)
Number of children														
1 †	101 (64.3)	1.00	78 (50.3)	1.00	38 (50.0)	1.00	42 (30.7)	1.00	103 (72.5)	1.00	111 (75.0)	1.00	118 (82.5)	1.00
2 or 3	92 (55.1)	1.00 (0.54–1.88)	84 (50.91)	0.95 (0.52–1.75)	38 (42.7)	0.92 (0.40–2.10)	43 (28.9)	0.79 (0.39–1.58)	112 (75.2)	1.83 (0.88–3.82)	114 (69.9)	1.39 (0.68–2.86)	114 (77.0)	0.56 (0.26–1.21)
4 or 5	62 (66.7)	1.37 (0.62–3.05)	47 (51.7)	0.98 (0.46–2.11)	18 (40.9)	0.80 (0.28–2.28)	26 (29.9)	0.92 (0.40–2.13)	67 (79.8)	1.74 (0.70–4.33)	65 (70.7)	1.30 (0.53–3.20)	75 (89.3)	1.77 (0.60–5.23)
6 or more	27 (71.1)	3.75 (1.10–12.80)*	14 (36.8)	0.51 (0.18–1.45)	7 (35.0)	0.38 (0.09–1.62)	6 (17.7)	0.11 (0.01–0.94)	25 (71.4)	1.65 (0.49–5.48)	29 (76.3)	2.47 (0.69–8.83)	32 (91.4)	1.54 (0.35–6.75)
Household size														
1–2	23 (63.9)	1.00	18 (51.4)	1.00	10 (58.8)	1.00	16 (48.5)	1.00	23 (74.2)	1.00	23 (67.7)	1.00	30 (96.8)	1.00
3–5	92 (60.5)	1.05 (0.35–3.16)	76 (50.7)	0.58 (0.20–1.74)	38 (49.4)	1.15 (0.30–4.49)	49 (35.0)	0.68 (0.24–1.98)	93 (71.5)	0.62 (0.17–2.18)	98 (67.6)	0.53 (0.16–1.78)	115 (88.5)	–
6–8	84 (62.7)	1.87 (0.61–5.74)	69 (52.7)	0.61 (0.20–1.85)	30 (44.1)	0.72 (0.18–2.83)	29 (24.8)	0.35 (0.11–1.07)	100 (80.0)	1.04 (0.29–3.82)	87 (66.9)	0.58 (0.17–2.01)	95 (76.0)	–
9 or more	56 (63.6)	0.96 (0.29–3.20)	45 (51.1)	0.71 (0.22–2.28)	20 (32.3)	0.67 (0.15–2.91)	12 (15.8)	0.22 (0.06–0.76)**	67 (79.8)	0.88 (0.22–3.60)	77 (87.5)	1.24 (0.30–5.10)	64 (78.1)	–
Maternal BMI														
Underweight	44 (78.6)	2.93 (1.19–7.21)**	26 (47.3)	0.83 (0.40–1.75)	9 (29.0)	0.64 (0.23–1.75)	18 (34.6)	1.45 (0.65–3.27)	38 (79.2)	1.88 (0.68–5.21)	42 (77.8)	0.79 (0.32–1.94)	42 (87.5)	1.96 (0.66–5.84)
Normal †	144 (66.4)	1.00	108 (50.2)	1.00	51 (43.2)	1.00	43 (22.9)	1.00	156 (77.2)	1.00	152 (72.0)	1.00	161 (79.7)	1.00
Overweight	26 (48.2)	0.52 (0.24–1.13)	23 (43.4)	1.10 (0.49–2.45)	9 (36.0)	0.61 (0.20–1.91)	20 (40.0)	3.04 (1.28–7.21)	36 (73.5)	0.90 (0.37–2.22)	37 (71.2)	1.21 (0.47–3.12)	40 (80.0)	0.86 (0.33–2.28)
Obese	5 (38.5)	0.46 (0.11–1.98)	9 (69.2)	4.47 (0.98–20.36)	2 (66.7)	–	3 (25.0)	1.21 (0.24–6.01)	5 (41.7)	0.13 (0.03–0.58)**	3 (25.0)	0.13 (0.03–0.62)*	10 (83.3)	0.72 (0.12–4.37)

(OR 0.13 [0.03–0.62],  $P = 0.01$ ) and cholesterol levels (OR 0.13 [0.03–0.58],  $P = 0.004$ ) in adulthood than children of normal weight mothers.

#### 4. Discussion

The present study shows that ideal cardiovascular health was rare in the ABC adult population. The most common metrics met were ideal glucose (83.6%), cholesterol (74.9%) and blood pressure (73.2%) levels. The least common metrics were related to health behaviours: non-smoking, ideal diet and ideal levels of physical activity were met by less than half of the cohort (28.8%, 44.1% and 49.7%, respectively). Sixty-two percent had an ideal BMI when defining BMI as  $<25$ . When healthy BMI was defined between 18.5 and 25, only 41% had an ideal BMI. Significant sex differences were observed in both the total score as well as regarding blood pressure and physical activity. Several early life determinants were found to independently predict future cardiovascular health. Family size and maternal BMI predict body mass index in adulthood. Areal disadvantage is associated with future blood pressure and levels of physical activity. Urban living environments were associated with non-ideal blood pressure levels. Household size was associated with smoking status in adulthood.

Previous epidemiological studies indicate that individuals who meet a larger number of ideal cardiovascular health behaviours or metrics have a lower risk of CVD mortality. The definition of ideal cardiovascular health by the AHA builds on this concept in promoting favourable health behaviours in order to reduce the burden of disease presented by cardiovascular morbidity and mortality [11]. However, several studies from other cohorts show that only few individuals meet these criteria for ideal cardiovascular health [23–26]. In an international multicohort study including 5785 participants from the i3c Consortium, ideal cardiovascular health was rare with only 1% of participants presenting with all 7 ideal cardiovascular health metrics. In the i3c, ideal glucose, ideal cholesterol and non-smoking were the metrics that had the highest prevalence (73%, 64% and 64% respectively) [12].

The adverse effects of high maternal BMI on offspring cardiovascular health is consistent with previous findings [6]. In the Generation R study, it was found that maternal obesity is associated with adverse cardiometabolic risk profiles including obesity, higher systolic blood pressure and adverse lipid levels in the offspring [19]. The association between maternal underweight, large family sizes and ideal offspring BMI is possibly related to food insecurity and malnutrition, as these differences were only evident when ideal BMI was merely defined as  $<25$  and no longer evident, when underweight participants were left out. Existing data suggests an association between household size and food insecurity in the Aboriginal population [20].

In line with our findings, both family and areal socioeconomic status have been shown to be important determinants of cardiometabolic risk factors. In the i3c consortium, data from longitudinal cohorts in Australia, Finland, and USA showed that parental education and occupation are strongly associated with the subsequent AHA cardiovascular health index among offspring in adulthood<sup>8</sup>. The association of location and cardiovascular risk profiles in Aboriginal Australians has been previously studied in the Heart of the Heart study [21]. It was found that participants from urban environments (Alice Springs) had higher blood pressure, elevated lipid levels and poorer kidney function than their remote living counterparts. Higher income was found to be associated with elevated risk of CVD in town camps but not in Alice Springs or in remote communities. Similar findings are seen in the ABC cohort, where urban residents had higher blood pressure. Participants from socially more advantaged areas according to the IRSEO score also presented with higher blood pressure and poorer levels of physical activity in this study. Similar mechanisms could lie behind these findings as the urban areas tended to score better in the IRSEO ranking.

Smoking was more common in large households ( $>9$  people sleeping in the house). Although common in all socioeconomic

groups, smoking is more common in low-income Aboriginal households [22]. This may partly explain the association.

Concerning the clinical and public health point of view, the present results provide important background information on the early life determinants of cardiometabolic health within an aboriginal community. To construct useful intervention strategies for positive health changes in this population based on these findings, it is very essential to take indigenous perspectives into account. Main tools in this process are culture-centered approach, community engagement, systems thinking, and integrated knowledge translation [27].

The strengths of the study include its longitudinal nature and well-structured follow-ups with relatively good retention rates. The study population however is relatively small causing some limitations to the interpretation of the results. Although the retention rates remained high, sample sizes for some of the described health metrics remained low, making the analyses less powerful. This was particularly evident for the total AHA score. Due to lacking data, the total score was available only for 204 participants (29.7%). Due to the modifications made to the original AHA definition of ideal cardiovascular health regarding diet and glucose levels that were needed for this paper, the results may not be directly comparable to other similar studies. Other limitations of the study include the difficult definition of SES, as the traditional variables of household income and education were not available and may not always be well-suited in the remote communities. The IRSEO score describes the areal level socioeconomic situation and does not necessarily reflect the individual SES of the participants. In a relatively small cohort, these differences may be of even larger significance. The present follow-up population may not completely represent the original birth cohort, as follow-up participants were more often females and had higher IRSEO scores compared to non-participants. Finally, the participants were still young adults during the last follow-up. After future follow-ups, cardiovascular morbidity and clinical events could be analysed for even better understanding of the clinical relevance of the cardiovascular risk profiles in this cohort.

In summary, the present study shows an association between early life predictors related to socioeconomic and family status and future cardiovascular health in the Aboriginal population. Achieving larger prevalences for individuals meeting ideal cardiovascular health metrics in the Aboriginal population in Australia could have significant effects on cardiovascular morbidity and mortality as well as reduce the healthcare costs related to these. This study shows that special attention needs to be put on health behaviours such as smoking and dietary habits as well as on gender equality in health to achieve these goals. Possible malnutrition must also be taken into account in future studies when analysing ideal BMI, as a significant number of participants showed to be underweight.

#### Conflict of interest

The authors report no relationships that could be construed as a conflict of interest.

#### Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.ijcard.2018.06.035>.

#### References

- [1] AIHW, The Health and Welfare of Australia's Aboriginal and Torres Strait Islander Peoples: 2015. Cat. No. IHW 147, AIHW, Canberra, 2015.
- [2] J. Perk, G. De Backer, H. Gohlke, et al., European guidelines on cardiovascular disease prevention in clinical practice (version 2012). The Fifth Joint Task Force of the European Society of Cardiology and other societies on cardiovascular disease prevention in clinical practice (constituted by representatives of nine societies and by invited experts), *Eur. Heart J.* 33 (13) (2012) 1635–1701.

- [3] D.J. Barker, The origins of the developmental origins theory, *J. Intern. Med.* 261 (2007) 412–417.
- [4] M.R. Skilton, N. Siitonen, P. Würtz, et al., High birth weight is associated with obesity and increased carotid wall thickness in young adults: the cardiovascular risk in young Finns study, *Arterioscler. Thromb. Vasc. Biol.* 34 (5) (2014) 1064–1068.
- [5] D.J.P. Barker, C. Osmond, T.J. Forsén, E. Kajantie, J.G. Eriksson, Trajectories of growth among children who have coronary events as adults, *N. Engl. J. Med.* 353 (17) (2005) 1802–1809.
- [6] R. Gaillard, Maternal obesity during pregnancy and cardiovascular development and disease in the offspring, *Eur. J. Epidemiol.* 30 (2015) 1141–1152.
- [7] A.J.J.M. Oostvogels, K. Stronks, T.J. Roseboom, J.A.M. van der Post, M. van Eijsden, T.G.M. Vrijkotte, Maternal prepregnancy BMI, offspring's early postnatal growth, and metabolic profile at age 5–6 years: the ABCD study, *J. Clin. Endocrinol. Metab.* 99 (10) (2014) 3845–3854.
- [8] T.T. Laitinen, K. Pahkala, A. Venn, et al., Childhood lifestyle and clinical determinants of adult ideal cardiovascular health: the Cardiovascular Risk in Young Finns Study, the Childhood Determinants of Adult Health Study, the Princeton follow-up study, *Int. J. Cardiol.* 169 (2) (2013) 126–132.
- [9] B. Galobardes, G.D. Smith, J.W. Lynch, Systematic review of the influence of childhood socioeconomic circumstances on risk for cardiovascular disease in adulthood, *Ann. Epidemiol.* 16 (2) (2006) 91–104.
- [10] C.R. Clark, M.J. Ommerborn, D.A. Hickson, et al., Neighborhood disadvantage, neighborhood safety and cardiometabolic risk factors in African Americans: biosocial associations in the Jackson Heart study, *PLoS One* 8 (2013), e63254.
- [11] D.M. Lloyd-Jones, Y. Hong, D. Labarthe, et al., Defining and setting national goals for cardiovascular health promotion and disease reduction: the American Heart Association's strategic impact goal through 2020 and beyond, *Circulation* 121 (4) (2010) 586–613.
- [12] M. Oikonen, T.T. Laitinen, C.G. Magnussen, et al., Ideal cardiovascular health in young adult populations from the United States, Finland, and Australia and its association with cIMT: the international childhood cardiovascular cohort consortium, *J. Am. Heart Assoc.* 2 (3) (2013), e000244.
- [13] E.S. Ford, K.J. Greenlund, Y. Hong, Ideal cardiovascular health and mortality from all causes and diseases of the circulatory system among adults in the United States, *Circulation* 125 (2012) 987–995.
- [14] Q. Yang, M.E. Cogswell, W.D. Flanders, et al., Trends in cardiovascular health metrics and associations with all-cause and CVD mortality among US adults, *JAMA* 307 (12) (2012) 1273–1283.
- [15] S.M. Sayers, D. Mackerras, G. Singh, I. Bucens, K. Flynn, A. Reid, An Australian Aboriginal Birth Cohort: a unique resource for a life course study of an indigenous population. A study protocol, *BMC Int. Health Hum. Rights* 3 (1) (2003) 1.
- [16] S. Sayers, G. Singh, D. Mackerras, et al., Australian Aboriginal Birth Cohort study: follow-up processes at 20 years, *BMC Int. Health Hum. Rights* 9 (2009), 23.
- [17] American Diabetes Association, 2. Classification and diagnosis of diabetes, *Diabetes Care* 40 (Suppl. 1) (2017) S11–S24.
- [18] N. Biddle, CAEPR Indigenous Population Project: 2011 Census Papers. Paper 13. Socioeconomic Outcomes, Centre for Aboriginal Economic Policy Research, The Australian National University, Canberra, 2013.
- [19] R. Gaillard, E.A.P. Steegers, L. Duijts, et al., Childhood cardiometabolic outcomes of maternal obesity during Pregnancy: Novelty and significance: the generation R study, *Hypertension* 63 (4) (2014) 683–691.
- [20] A. Markwick, Z. Ansari, M. Sullivan, J. McNeil, Social determinants and lifestyle risk factors only partially explain the higher prevalence of food insecurity among aboriginal and Torres Strait islanders in the Australian state of Victoria: a cross-sectional study, *BMC Public Health* 14 (1) (2014) 598.
- [21] A. Brown, M.J. Carrington, M. McGrady, et al., Cardiometabolic risk and disease in indigenous Australians: the heart of the heart study, *Int. J. Cardiol.* 171 (3) (2014) 377–383.
- [22] D.P. Thomas, V. Briggs, I.P.S. Anderson, J. Cunningham, The social determinants of being an indigenous non-smoker, *Aust. N. Z. J. Public Health* 32 (2) (2008) 110–116.
- [23] A.R. Folsom, H. Yatsuya, J.A. Nettleton, et al., Community prevalence of ideal cardiovascular health, by the American Heart Association definition, and relationship with cardiovascular disease incidence, *J. Am. Coll. Cardiol.* 57 (2011) 1690–1696.
- [24] J. Stamler, R. Stamler, J.D. Neaton, et al., Low risk-factor profile and long-term cardiovascular and noncardiovascular mortality and life expectancy: findings for 5 large cohorts of young adult and middle-aged men and women, *JAMA* 282 (21) (1999) 2012–2018.
- [25] M.L. Daviglius, J. Stamler, A. Pirzada, et al., Favorable cardiovascular risk profile in young women and long-term risk of cardiovascular and all-cause mortality, *JAMA* 292 (13) (2004) 1588–1592.
- [26] E.J. Benjamin, M.J. Blaha, S.E. Chiuve, et al., Heart disease and stroke statistics-2017 update: a report from the American Heart Association, *Circulation* 135 (10) (2017) e146–e603.
- [27] J. Oetzel, N. Scott, M. Hudson, et al., Implementation framework for chronic disease intervention effectiveness in Māori and other indigenous communities, *Glob. Health* 13 (1) (2017) 69.