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Gender differences in cardiovascular disease risk: Adolescence to young adulthood

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Abstract *Background and aims:* Gender differences in cardiovascular disease (CVD) have been well documented but rarely for young adults and the extent to which gender related lifestyle differences may contribute to gender differences in CVD risk experienced by young adults have not been reported.

Methods and results: Data are from a long-running cohort study, the Mater-University of Queensland Study of Pregnancy (MUSP). We track gender differences in CVD related behaviours at 21 and 30 years (consumption of a Western Diet/Health-Oriented Diet, cigarette smoking, vigorous physical exercise, heavy alcohol consumption). At 30 years we compare males and females for CVD risk, and the extent to which lifestyle behaviours at 21 and 30 years contribute to CVD risk.

At both 21 and 30 years of age, males more frequently consume a Western Diet and less often a Health Oriented Diet. By contrast, males are also much more likely to report engaging in vigorous physical activity. On most CVD markers, males exhibit much higher levels of risk than do females at both 21 and 30 years. At 30 years of age males have about five times the odds of being at high risk of CVD. Some lifestyle behaviours contribute to this additional risk.

Conclusion: Young adult males much more frequently engage in most CVD related risk behaviours and males have a higher level of CVD risk. Gender differences in CVD risk remain high even after adjustment for CVD lifestyles, though dietary factors independently contribute to CVD risk at 30 years.

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1. Introduction

Gender differences in cardiovascular disease (CVD) have been widely discussed [1]. The incidence of female CVD lags behind men by about 10 years [2]. In older age groups, female deaths attributable to CVD may exceed rates experienced by males [3]. There has been surprisingly little research to address the basis of gender differences in young adult rates of cardiovascular disease (CVD), experienced by young adults [4–6]. Taking younger age cardiovascular deaths as a criterion, the gender differences are remarkable [7]. Data from Finland show that 4.8% of male and 0.8% of female CVD deaths were in the age group 49 years or less [3]. This difference in male and female deaths is in a context where the lifetime number of CVD deaths experienced by males and females in Finland is similar [3]. Other have also reported that the younger age groups (35–44 years) have the largest M:F differential in CVD death rates [4].

Explanations of gender differences in CVD disease are of two types. The first focus on biological/physiological differences between males and females, particularly the protective effects of premenopausal female hormones [8] and also sex differences in endothelial function [9] associated with such CVD risk factors as arterial hypertension [10]. The second type focus on the contribution of lifestyle behaviours to CVD with a recent meta-analysis of 22 studies suggesting that a healthy lifestyle may lead to a 66% reduced risk of CVD [11]. However, previous studies have rarely addressed gender differences in CVD risk behaviours and CVD risk in young adults.

We use data from a long running cohort study to examine (i) gender differences in CVD related risk behaviours at 21 and 30 years of age (ii) gender differences in CVD risk at 30 years of age (iii) the extent to which risk behaviours at 21 and 30 years of age account for gender differences in CVD risk at 30 years of age.

1.1. Lifestyles and CVD

The behaviours and lifestyles contributing to CVD generally include cigarette smoking, a diet high in saturated fats and sugars and low in vegetables and fruit, as well as physical inactivity and high levels of alcohol consumption [11,12]. For many but not all of these unhealthy lifestyle behaviours males exhibit higher rates than females [13]. Gender differences in a range of CVD related lifestyle behaviours have been reported across the life course, for example diet [14], exercise [15] and high alcohol consumption [16]. Less clear is the extent to which these differences begin in the adolescent period of the life course and persist into young adulthood.

The current paper takes data from a cohort study and compares males and females on a range of CVD risk behaviours (at ages 21 and 30 years) as well as cardiometabolic risk (blood pressure, body mass index, HDL, LDL, triglycerides and HOMA-IR) at 30 years of age.

As others have noted there are few studies which have specifically addressed gender differences in CVD risk [17] and there is a notable gap in studies of CVD focussing on the adolescent/young adult period of the life course.

2. Methods

2.1. Study population

The data for this study are taken from the Mater-University of Queensland Study of Pregnancy (MUSP) and its outcomes [18]. This is a birth cohort study with 8556 consecutive pregnant women invited to participate at their first obstetrical visit. Some 8458 mothers (98.9% participation) provided consent to participate in this longitudinal study over the period 1981–3. Some 710 mothers did not proceed to delivery (largely miscarriages), there were 59 multiple births (excluded), 41 deaths, 55 children adopted and a further 312 lost to follow-up after the birth. In total there were 7223 mothers who participated in the multiple phases of recruitment around the prenatal/postnatal period and these comprise the MUSP cohort. There were additional follow-ups at 6 months (93.0% retained), 5 years (73.5% retained) and 14 years (72.2% retained). At 21 years, 52.7% of offspring responded. Offspring were again invited to participate in the study when they were 30 years of age (in 2010–14). Some 2859 (41%) of the original sample of children participated in this follow-up but some did not complete all components of the data collection. Further, some 120 children are recorded as deceased at the most recent follow-up. This has left 2250 offspring providing data at 21 and 30 years and 1363 respondents who have provided both data with physical assessments as well as blood samples (Fig. 1). Not all respondents provided data for every variable and numbers vary somewhat in some tables. We have extensively studied the differences between those lost to follow-up and those retained in the study and the implications for attrition for our estimates of association [19]. We discuss this issue in the limitations.

Informed consent was obtained at each phase of data collection, initially from the mother (to 14 years) and then separately from the mother and the child from the 21-year follow-up and subsequently. The University of Queensland Human Research Ethics Committee or the Mater Hospital Research Ethics Committee or both, provided ethics approvals.

3. Measurements

3.1. Patterns of food consumption

Interest in food consumption and CVD can be traced to the work of Ancel Keys [20] in the 1960s, and the development of the concept of the Mediterranean diet as cardioprotective by Trichopoulou [21,22]. Patterns of food consumption reflect realities of time and place [23]. The components of diet that offer cardiovascular benefits are likely to vary depending upon the patterns of food consumption observed in particular ecological contexts.

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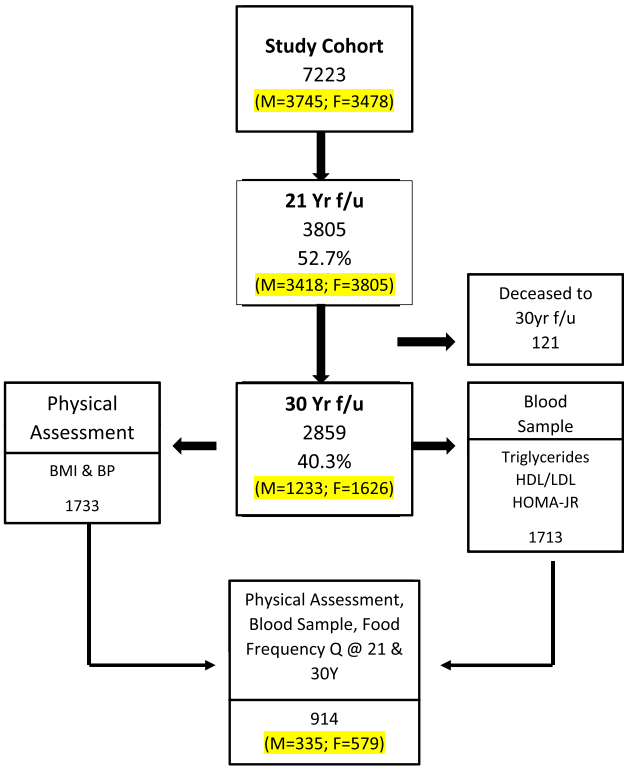

Figure 1 Sample retention to 30 years.

At the 21- and 30-year follow-ups, respondents were administered a food frequency questionnaire (FFQ) developed by the Anti-Cancer Council of Victoria, a measure based upon a FFQ originally from the US Nurses' Health Study [24]. This food frequency questionnaire was developed to estimate nutrient and energy intake, as well as the consumption of specific foods [25]. In the current study we administered a version of a self-report food frequency questionnaire used by the Australian Cancer Study/Australian Ovarian Cancer Study [26]. Reliability estimates associated with the administration of FFQs are moderate to good suggesting that both specific foods consumed and nutrient fractions are consistently reported and that the findings point to longer term patterns of food consumption [27]. Respondents are presented with a list of specific foods and asked how frequently they consume (includes per day, per week or per month) each of these foods.

Using principal components factor analysis (with orthogonal rotation) we undertook separate analyses of the food lists at the 21- and 30-year follow-ups. We set a factor loading of 0.40 or better as the basis of identifying the foods which defined factors. In the nine years between the administration of the FFQ to the 21- and 30-year samples, there were some changes in some specific foods listed in the FFQ. The first two factors identified in the factor analyses at 21 and 30 years had similar items and were labelled, consistent with previous studies, the Western Diet and the Health Oriented Diet. At 21 and 30 years, the Western Diet (see Supplementary Table 1) included pizza, hamburgers, sausages and processed meat, potatoes and chips. This is a diet high in saturated fats, sugars, red meats, and includes what has been described as hyper-palatable, ultra processed foods [28]. At 21 years, there were 14 items (Cronbach alpha reliability 0.77) in this factor, while at 30 years it comprised 19 items (Cronbach alpha reliability 0.85). The Health Oriented Diet comprised foods such as nuts, many fruits and vegetables as well as fish. At 21 years, this factor comprised 35 items (Cronbach alpha reliability 0.90) while at 30 years it comprised 30 items (Cronbach alpha reliability 0.90). These two factors broadly approximate, on the one hand, a contemporary pattern of fast-food consumption, and on the other, a diet that is of locally available fruits and vegetables.

3.2. Cigarette smoking, physical activity and alcohol consumption

At the 21- and 30-year follow-ups, respondents were asked how many cigarettes they smoked per day. Based upon their responses we created a dichotomous variable of non-smokers and smokers. At the 21- and 30-year follow-ups, respondents were also administered questions from the International Physical Activity Questionnaire (IPAQ) [29]. At 21 years, we used answers to a question about the number of times the respondent had engaged in vigorous physical activity of 20 min or more in the last week. At 30 years, we used questions which asked about physical activity at work, leisure, in domestic activities and transportation. At 30 years we have used the vigorous physical activity sub-scale of the IPAQ which is scored as minutes of activity (adjusted for intensity of activity) and is a continuous variable with higher scores reflecting higher levels of activity.

Respondents were also asked two questions about their alcohol consumption; how often they consume alcohol and how much they consume on a typical day. A composite variable was created to distinguish abstainers (reference category) from those who are light to moderate consumers (averaging up to about 3 drinks a day) and heavy drinkers, that is, those whose consumption has been implicated with an increased risk of CVD (3+ per day).

3.3. Measurement of risk of CVD

We used six measures of risk of CVD, all consistent with commonly used risk calculators, namely body mass index (BMI), blood pressure, high-density lipoproteins (HDL), low-density lipoprotein (LDL), triglycerides and HOMA-IR. All measures were taken by a trained researcher or provided by an accredited laboratory (see Supplementary Table 2 for details).

Body Mass Index (BMI) was determined using height and weight taken by a trained researcher (using a stadiometer and calibrated scales). Staff conducted measurements of participants using a Tanita Bio-Impedance machine (model BC-418) used to assess weight and calculate BMI. Participants were instructed to remove shoes and excess clothing, step onto the machine and hold

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the handles of the machine. Height was assessed using a stadiometer and entered into the Bio-Impedance machine. All calculations were directly transferred from the Bio-Impedance machine into the MUSP database. For BMI, standard cut-offs (≤24.9; 25–29.9 and 30+) were used.

Blood pressure was assessed by a trained researcher. We used the Omron blood pressure monitor (HEM-705cp) with appropriated cuffs depending on arm circumference. Respondents were asked to sit down and two blood pressure readings were taken, 5 min apart. Respondents were asked about any blood pressure medications they had taken in the last 24 h. We used criteria for the current age group and supported by the American College of Cardiology and the American Heart Association [30], namely normal (120/80 mmHg or below); elevated (above 120 mmHg in systole or above 80 mmHg in diastole or both); and high (either 140 mmHg in systole or above or 90 mmHg in diastole or above).

We have measures of cardiometabolic risk biomarkers from blood samples collected by commercial pathology laboratories. Triglyceride, High density lipoprotein (HDL) and low-density lipoprotein (LDL), insulin and glucose levels were measured with respondents asked to fast overnight and samples collected before intake of food. Cholesterol oxidase/peroxidase, lipase GK/GPO/peroxidase and phosphating state/Mg^{2+} methods were used to assess triglycerides and HDL/LDL levels using Ortho Clinical Diagnostics Vitros Analyser. Homeostatic Model Assessment (HOMA) – Insulin Resistance – was calculated by multiplying fasting insulin (ml/L) by fasting glucose (mmol/L) and dividing by 22.5. HOMA-IR scores were divided into tertiles from low to high.

3.4. Cardiovascular disease risk score

We create a CVD risk score at the 30-year data collection. We added the number of risk markers for each respondent as follows: For BMI we used standard criteria (BMI 1 = low to normal <24.9; 2 = overweight 25.0–29.9; 3 = obese 30+). For BP we used conventional cut-offs (1 = systolic <119.9 and diastolic <79.9; 2 = systolic <139.9 and diastolic <90.0; 3 = systolic ≥140.0 or diastolic ≥90.0) for HDL, LDL, triglycerides and HOMA-IR, we divided the results into tertiles with HDL levels reversed. The range of CVD risk scores was 6 (lowest risk on all six measures) to 18 (highest risk on all six measures). This measure approximates a normal distribution. We also created a 3-category composite CVD risk scores (6–9 = 1 low risk; 10–13 = 2 some risk; 14–18 = 3 highest risk).

3.5. Analytic strategy

We first present an analysis of gender differences in risk at 21 and 30 years of age using analysis of variance. We then use logistic regression and multinomial regression to present the associations between gender and the five CVD risk behaviours, without and then with adjustment for sociodemographic covariates. These analyses are repeated at the 21- and 30-year follow-ups. We then examine the extent to which lifestyle behaviours at 21 and 30 years predict CVD risk using general linear models (GLM) and comparing unadjusted and adjusted mean CVD risk scores. We adjust for gender on the basis that gender differences include biological/physiological and hormonal differences and there is a need to differentiate these from effects of lifestyle behaviours. At the 30-year follow-up, we provide an additional analysis of the association between gender and six risk factors for CVD (using multinomial logistic regression with adjustments for (1) confounding (2) lifestyle risk behaviours at 21 and 30 years).

For the relevant comparisons, female respondents are the reference category and we present the extent to which male respondents exhibit increased (or decreased) risk compared to female respondents. We present odds ratios with 95% confidence intervals.

4. Results

Table 1 provides details of gender differences for CVD risk behaviours at 21 and 30 years as well as the measures of CVD risk, the latter only at 30 years of age. Males have a higher mean score on a Western Diet (at both 21 and 30 years) and a lower mean score on a Health-Oriented Diet also at 21 and 30 years. Males engage in more vigorous physical activity at both 21 and 30 years, as well as heavy alcohol consumption. For measures of CVD risk, males have substantially higher mean levels of blood pressure, low HDL, and higher levels of BMI, LDL, triglycerides and HOMA-IR. The composite CVD risk of males is also substantially higher than the risk observed in females. While there are sociodemographic differences between male and female respondents at 21 and 30 years, these are generally small with the exception that males are more likely to be employed at 30 years than are females. The r^2 statistics provide an estimate of the relative strength of the associations in Table 1. The strongest of the associations are for blood pressure, HDL, vigorous physical activity and a Western Diet at 21 years.

Substantial gender differences in CVD related lifestyle behaviours are evident at the 21- and 30-year follow-ups (Table 2). Males are much more likely to consume a Western Diet and much less likely to consume a Health Oriented Diet at both 21 and 30 years of age (estimated relative risks vary with males about 2.5 to almost 6 times more likely to have a Western Diet). Males are substantially more likely to report heavy alcohol consumption, particularly at 30 years of age. Cigarette smoking levels appear to be relatively similar for males and females but males are much more likely to report undertaking vigorous physical activity (cardioprotective) particularly by the 30 years of age follow-up.

The associations between lifestyle behaviours and CVD risk at 30 years are presented in Table 3. These address the hypothesis that lifestyle differences at 21 and 30 years may be associated with CVD risk, even by 30 years of age. The consumption of a Western Diet and a Health Oriented Diet at 30 years both predict CVD risk at 30 years. After adjustment for gender, none of the lifestyle behaviours

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Table 1 Gender differences in CVD risk and CVD risk related behaviours at 21 and 30 years of age (analysis of variance, mean and F-ratio).

	Male (N = 335)	Female (N = 579)	F-ratio	P-value	R^2
Lifestyle behaviours at 21 years					
Western Diet[a] at 21 (14–56)	33.0	30.3	98.2	P < .001	0.097
Health Oriented Diet[b] at 21 (35–138)	72.0	74.2	7.4	P = .007	0.008
Smoking at 21 (Smoker = 2)	1.3	1.3	0.3	P = ns	0.000
Vigorous Phys Activity 21 (3[b] week = 3)	2.1	1.9	23.4	P = .001	0.025
Heavy Alcohol at 21 (Heavy = 3)	3.1	2.7	39.4	P < .001	0.042
Lifestyle behaviours at 30 years					
Western Diet[a] at 30 (22–60)	44.3	41.9	33.7	P < .001	0.037
Health Oriented Diet[b] at 30 (31–101)	63.9	68.0	35.6	P < .001	0.040
Smoking at 30 (Smoker = 2)	1.2	1.2	0.7	P = ns	0.001
Vigorous Phys Activity 30 (IPAQ[c]) (0–10,080)	2469.4	912.0	95.6	P < .001	0.105
Heavy Alcohol at 30 (Heavy = 3)	3.4	2.9	36.7	P < .001	0.040
Measures of CVD risk at 30 years					
Blood Pressure (high = 3)	2.27	1.39	333.2	P < .001	0.268
Body Mass Index (obese = 3)	1.89	1.76	5.6	P = .018	0.006
HDL (Reversed) (low = 3)	2.40	1.83	113.5	p < .001	0.111
LDL (high = 3)	2.18	1.83	42.5	P < .001	0.045
Triglycerides (high = 3)	2.09	1.86	17.0	P < .001	0.018
HOMA-IR (high = 3)	2.05	1.91	6.4	P = .012	0.007
CVD (Composite) Risk at 30 (6–18)	12.1	10.9	52.9	P < .001	0.055
Confounders at 30 years					
Employed at 30 (Employed = 2)	1.9	1.7	52.9	P < .001	0.056
Education Level at 30 (Tertiary = 2)	1.7	1.7	4.7	P = .029	0.005
Marital Status at 30 (Married = 3)	2.1	2.2	6.6	P = .010	0.007
Family Income at 30 (Highest = 2)	1.8	1.8	1.3	P = ns	0.001

[a] Western Diet; higher consumption of red meat, saturated fat, sugars.
[b] Health Oriented Diet; higher consumption of vegetables and fruit.
[c] IPAQ: International Physical Activity Questionnaire.

Table 2 Gender differences in CVD related lifestyle behaviours at 21 and 30 years (multinomial logistic regression, odds ratios, 95% CI).

	Male 21 – Unadj (Ref: Female) OR (95% CI)	Male 21 – Adj[a] (Ref: Female) OR (95% CI)	Male 30 – Unadj (Ref: Female) OR (95% CI)	Male 30 – Adj[b] (Ref: Female) OR (95% CI)
Western Diet				
Low (33%)	1	1	1	1
Mid (33%)	**2.27 (1.56,3.31)**	**2.05 (1.39,3.04)**	1.19 (0.84,1.69)	1.23 (0.85,1.79)
High (33%)	**5.91 (4.07.8.60)**	**5.40 (3.64,8.01)**	**2.39 (1.71,3.35)**	**2.57 (1.78,3.71)**
Health Oriented Diet				
Low (33%)	1	1	1	1
Mid (33%)	0.89 (0.62,1.29)	0.89 (0.60,1.31)	**0.65 (0.46,0.92)**	**0.59 (0.40,0.86)**
High (33%)	**0.62 (0.43,0.89)**	**0.64 (0.44,0.94)**	**0.37 (0.26,0.53)**	**0.36 (0.24,0.52)**
Smoking				
No	1	1	1	1
Yes	0.90 (0.66,1.22)	**0.70 (0.50,0.98)**	1.05 (0.74,1.49)	1.02 (0.70,1.50)
Vigorous Physical Activity				
Nil	1	1	1	1
Some	1.26 (0.90,1.76)	1.28 (0.90,1.82)	**1.56 (1.06,2.29)**	1.30 (0.87,1.96)
High	**2.37 (1.66,3.39)**	**2.49 (1.71,3.63)**	**4.12 (2.91,5.82)**	**3.28 (2.28,4.72)**
Alcohol				
Abstainer	1	1	1	1
Some	0.89 (0.50,1.59)	0.82 (0.45,1.48)	**1.83 (1.01,3.31)**	1.48 (0.79,2.74)
Heavy (3+ p/d)	**2.18 (1.20,3.95)**	1.73 (0.93,3.22)	**5.34 (2.66,10.72)**	**4.06 (1.96,8.42)**

Bold values denote significant values.
[a] Adjusted for respondent youth income, education level, academic performance at school and marital status (at 21 years).
[b] Adjusted for respondent employment status, education level, marital status and family income (at 30 years).

at 21 years predict CVD risk at 30 years. We have also tested for some key interactions (tables not presented), namely a Western Diet at both 21 and 30 years, a Health Oriented Diet at both 21 and 30 years and vigorous physical activity at both 21 and 30. None of these interactions approached statistical significance. Some additional possible interactions were also assessed (Health Oriented Diet and vigorous physical activity, all at 30 years) without a positive result.

Gender differences in CVD risk are substantial and remain substantial even after adjustment for CVD related lifestyle behaviours at both the 21- and 30-year follow-ups

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Table 3 Lifestyle behaviours at 21 and 30 years and CVD risk at 30 years (GLM – analysis of variance with CVD risk as dependent variable).

Variables	N	Bivariate – Unadjusted		Multivariate – Adjusted[a]	
		Means	F-Ratio	Means	F-Ratio
Western Diet 30 Y					
Low	311	10.73 (10.40,11.06)	**F = 16.5, P < .001**	11.20 (10.85, 11.54)	**F = 8.3, p < .001**
Mid	285	11.51 (11.17,11.85)		11.90 (11.56,12.25)	
High	331	12.11 (11.77,12.46)		12.13 (11.81,12.46)	
Healthy Diet 30 Y					
Low	230	12.00 (11.62,12.89)	**F = 8.9, p < .001**	12.02 (11.66,12.38)	**F = 3.6, P = .03**
Mid	299	11.50 (11.16,11.83)		11.82 (11.50,12.15)	
High	331	10.93 (10.61,11.26)		11.37 (11.03,11.71)	
Cigarette Smoking 30 Y					
No	721	11.32 (11.10,11.54)	F = 2.7, P = .10	11.65 (11.44,11.86)	F = 1.6, P = ns
Yes	170	11.74 (11.29,12.19)		11.97 (11.53,12.40)	
Vig Phys Activity 30 Y					
Nil – Low	351	11.35 (11.04,11.66)	**F = 3.5, P = .03**	11.96 (11.62,12.30)	**F = 2.9, P = .06**
Some	199	10.95 (10.53,11.36)		11.35 (10.94,11.76)	
High	255	11.68 (11.32,12.03)		11.53 (11.20,11.87)	
Heavy Alcohol 30 Y					
Abstainer	68	11.65 (10.94,12.36)	F = 1.00, p = ns	11.92 (11.12,12.72)	F = 0.7, P = ns
Light – Moderate	716	11.33 (11.11,11.55)		11.69 (11.47,11.90)	
High (3+)	103	11.71 (11.13,12.29)		11.40 (10.85,11.95)	
Western Diet 21 Y					
Low	289	10.84 (10.50,11.19)	**F = 14.5, P < .001**	11.58 (11.16,11.99)	F = 1.7, P = ns
Mid	320	11.28 (10.96,11.61)		11.58 (11.26,11.90)	
High	305	12.13 (11.80,12.46)		12.00 (11.64,12.27)	
Healthy Diet 21 Y					
Low	181	11.77 (11.34,12.21)	F = 1.6, p = ns	11.95 (11.54,12.36)	F = 2.43, P = .09
Mid	329	11.30 (10.98,11.63)		11.48 (11.17,11.79)	
High	404	11.37 (11.07,11.67)		11.89 (11.60,12.18)	
Cigarette Smoking 21 Y					
No	650	11.37 (11.13,11.60)	F = 0.09, p = ns	11.69 (11.47,11.91)	F = 0.6, p = ns
Yes	256	11.57 (11.20,11.94)		11.86 (11.50,12.22)	
Vig Phys Activity 21 Y					
No	288	11.25 (10.90,11.60)	F = 1.1, p = ns	12.44 (11.68,13.21)	F = 1.9, P = ns
1–2 p/wk	370	11.58 (11.28,11.89)		11.64 (11.39,11.90)	
3+ p/wk	249	11.36 (10.98,11.73)		11.74 (11.42,12.07)	
Heavy Alcohol 21 Y					
Abstainer	59	11.98 (11.22,12.75)	**F = 4.6, p = .01**	12.44 (11.68,13.21)	F = 1.9, P = ns
Light – Mod	564	11.19 (10.94,11.43)		11.64 (11.39,11.90)	
Heavy (3+)	283	11.76 (11.41,12.11)		11.74 (11.42,12.07)	

Bold values denote significant values.
[a] Adjusted for gender of respondent.

Table 4 Gender of respondent and CVD risk level at 30 years (multinomial logistic regression – odds ratios).

	Male Unadj	Female Unadj	Male Adj1	Female Adj1	Male Adj2	Female Adj2
Low risk	1	1	1	1	1	1
Some risk	**2.44 (1.67,3.57)**	1	**2.45 (1.64,3.64)**	1	**1.73 (1.09, 2.73)**	1
Highest risk	**5.17 (3.33,8.03)**	1	**5.44 (3.40,8.73)**	1	**4.94 (2.83,8.62)**	1

Adj1 = Adjusted for respondent employment, education, marital status and family income at 30 years.
Adj2 = Adjusted lifestyle factors, western diet (21, 30), health-oriented diet (21, 30) vigorous physical activity (21, 30) and heavy alcohol consumption (21, 30). Bold values denote significant values.

(Table 4). Males are consistently about five times more likely to be at highest risk of CVD. These differences remain very strong even after adjustment for the full range of lifestyle behaviours.

5. Discussion

At both the 21- and 30-year follow-ups, males have a cluster of lifestyle behaviours that are likely to contribute to the early age of onset of the CVDs. The lifestyle risk behaviours we examine at 21- and 30-years track over the life course; Pearson correlation coefficients for a Western Diet ($r = 0.37$, $p < .001$), Health-Oriented Diet ($r = 0.52$, $p < .001$), cigarette smoking ($r = 0.56$, $p < .001$), vigorous physical activity ($r = 0.15$, $p < .001$) and heavy alcohol consumption ($r = 0.32$, $p < .001$) are all behaviours which often persist over an extended period of the life course. While we measure these behaviours at two ages in young

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adulthood, it is likely these behaviours commenced prior to our first measurement and will persist after 30 years of age. In this context, our measurements may underestimate the impact of these behaviours on adult CVD though our findings suggest that some impacts are evident by 30 years of age.

We have previously reported an association between patterns of food consumption and cardiometabolic changes suggesting patterns of food consumption are associated with an increased risk of CVD [31] as well as elevated levels of insulin resistance. Males more often consume foods that include fat/meat/sugar and less often consume foods that include vegetables and fruits and more often report heavy alcohol consumption. These gender differences in patterns of food consumption do not explain gender differences in CVD risk at 30 years. Comparing predictors of CVD risk, our findings suggest that gender differences in biological physiological/hormonal characteristics provide a stronger prediction of CVD risk at 30 years than do lifestyle behaviours. Nevertheless, by 30 years a Western Diet pattern and a Health Oriented Diet pattern are contributing to CVD risk and it is likely that the persistence of these dietary patterns as respondents age may additionally contribute to gender differences in CVD. While males overall have a less healthy lifestyle, our findings suggest this less healthy lifestyle does not explain and consequently cannot account for the gender differences in CVD risk we have observed.

5.1. Limitations

First, there is a need to better understand the gender differences in CVD related behaviour. The behaviours involved (eg. poor diet, smoking, heavy alcohol use) may have changed greatly over time and are continuing to change. As Eagly and Wood (2013) point out, many of the differences we observe are compatible with both biological and sociocultural explanations. For example, a greater propensity for males to engage in risk taking has been widely observed across cultures (nature) but may vary greatly depending upon the type of risk taking involved. It is not yet possible to distinguish a genetic/hormonal basis for male aggression/risk taking from a sociocultural one. Males are more liable to take a variety of risks particularly associated with smoking, consuming alcohol, taking illicit drugs, driving beyond the speed limit and breaking traffic laws as well as gambling [32,33].

There is also a possibility that sample loss to follow-up may be biased and lead to misleading findings. Loss to follow-up is biased in this study with those respondents who are most economically disadvantaged and who exhibit higher levels of mental illness and high risk behaviours disproportionately likely to be lost [18]. However, concerns about possible bias contingent on attrition is tempered by two observations.

i. We have repeatedly modelled the possible effects of attrition by taking our sample at recruitment (98.9% participation) and repeating analysis of associations at recruitment and removing those subsequently lost to follow-up [19]. We find that even with high levels of attrition, associations remain materially unaffected. This finding has since been repeated using other data and analyses [34].
ii. We note that all samples are biased insofar as they are limited to time and place when the data were obtained, and involve specific measures that may vary from study to study. The generalisability of a finding is more likely a function of replication of findings in different times, places and with varying measures (see Rothman et al. for a more detailed discussion) [35].

6. Conclusions

Our findings should be interpreted in the broader health context in which CVD risk behaviours are observed. Males have higher death rates for all the main causes of death [36]. Males experience higher rates of chronic diseases than do females, have higher age specific death rates from 15 of the major causes and more frequently report some 30 behaviours which are associated with increased rates of morbidity and mortality [36–38].

Our findings suggest four directions for efforts to reduce the burden associated with cardiovascular diseases which have their origins over the early life course. First, the need for earlier efforts to reduce CVD risk behaviours even in childhood and the adolescent period. These risk factors "track" into adulthood and are integral to the progress of the CVDs. Second, the social and environmental context in which children are reared predicts many of the risk behaviours. Young males much more frequently engage in most of these risk behaviours and we suggest that this cluster of behaviours is culturally supported and constitute a component of what is perceived as a masculine lifestyle. Efforts to alter male lifestyles to, for example, discourage patterns of high fat/meat/sugar consumption and heavy alcohol consumption must confront a cultural context which supports this behaviour. Third, there is a need to develop CVD risk reduction programs which reflect associations between lower levels of education, parental marital breakdown, low income and unemployment and a wide range of risk behaviours and unhealthy lifestyles. Those with greater knowledge and better family resources adopt lower CVD risk behaviours. Targeted efforts to reduce risk will need to be creative in the context that economically and socially disadvantaged younger persons may be unable to forgo diets high in fats/meat/sugars, smoking and heavy alcohol consumption. Fourth, young males comprise a group at high risk of earlier age of onset CVD. The failure to prioritise this high risk group represents a major deficiency of existing CVD prevention efforts and demands an urgent and systemic response.

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Declaration of competing interest

There are no competing interests.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.numecd.2023.09.024.

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