DIETARY INTAKE IN RELATION TO METABOLIC SYNDROME AND ASSOCIATED RISKS IN CANADIAN ADULTS AND ADOLESCENTS

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ABSTRACT

Type 2 diabetes and cardiovascular disease (CVD), major public health concerns in Canada and worldwide, are the main outcomes of Metabolic Syndrome (MetS). MetS is a clustering of five chronic disease risk factors, including abdominal obesity, dyslipidemia (elevated triglycerides and reduced high-density lipoprotein cholesterol level), hypertension, and elevated fasting plasma glucose. Abdominal obesity and insulin resistance are the main factors contributing to development of MetS and thus CVD and diabetes. Further, dyslipidemia (specifically the total cholesterol/HDL-C ratio) is necessary to be monitored in people with these diseases or risk factors. The Canadian Health Measures Survey (CHMS) has direct health measurements that provide the opportunity to identify risk factors associated with chronic disease. Further, dietary intake has been assessed in CHMS, which can be utilized to evaluate the association with diseases and risk factors controlling for potential covariates.

To examine the association between dietary intake with MetS and associated risks and diseases in Canadian adults and adolescents, data from CHMS cycle1, 2007-2009 (n=5604, aged 6-79 y) were used in which dietary intake was assessed using a semi-quantitative food frequency questionnaire. Frequency of food intake (times/day) and the distribution of individuals with and without disease or risk factors by socio-demographic characteristic was determined. Further, the association between dietary intake and each disease or risk factor was evaluated using logistic models. Data manipulation, cleaning, and creation of new variables were done using IBM SPSS statistics for windows, version 20. All statistical analyses were conducted by STATA/SE 11, StataCorp. As per Statistics Canada's recommendation, data were weighted and bootstrapped using specific commands in STATA in order to be representative of the Canadian population.

Physically active Canadians had significantly more milk and dairy product, fresh fruit and vegetable, and fruit and vegetable juice intake compared to inactive Canadians. Low-income households had significantly greater intakes of sugar-sweetened beverages (SSBs) and lower amounts of fresh fruit and vegetables than high-income households. Individuals with the lowest level of education had greatest potato and lowest fruit and vegetable juice intake. The prevalence of abdominal obesity (12-79 y), elevated TC/HDL-C ratio (20-79 y), MetS (12-79 y), and diabetes (20-79 y) was 35.7%, 20.5%, 18.3%, and 7.5%, respectively. The overall estimated percent risk of CVD over 10 years in adults aged 30-74 y was 8.66%. Abdominal obesity had the

greatest contribution (~90%) to development of MetS in each age group compared to other components of MetS. Also, abdominal obesity was a strong significant predictor for development of other risk factors and chronic diseases including elevated TC/HDL-C ratio, diabetes, and CVD (OR: 6.12, CI: 2.68-13.96; OR: 2.25, CI: 1.24-4.06, OR: 1.4, CI: 1.06-1.87, respectively). High consumption of diet soft drink was associated with increased risk of abdominal obesity, Mets, diabetes, and elevated TC/HDL-C ratio. High consumption of fruit and vegetables and cereal was associated with decreased risk of CVD over 10 years whereas potato intake increased the risk. Intake of nuts appeared to be associated with reduction in the risk of MetS among Canadians. Dietary fat intake of one to less than 5 times/week was associated with increased risk of abdominal obesity in individuals over 40 years of age compared to 12-19 y individuals. Further, "ever drinking" alcohol compared to "never drinking" alcohol was associated with decreased risk of diabetes and elevated TC/HDL-C ratio. Low level of education contributed to development of abdominal obesity. Thus, abdominally obese individuals with specifically elevated TC/HDL-C ratio are at risk of developing MetS, CVD, and diabetes. They need to be educated with specific focus on physical activity, and greater intake of fruit and vegetables, nuts, and lower intake of dietary fat, diet soft drinks, and potato. Additionally, considering the social determinants which might contribute to increase the risk of chronic diseases or risk factors such as income, housing, accessibility to facilities in neighborhoods, school programs, or community programs is essential.

ACKNOWLEDGEMENTS

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LIST OF ABBREVIATIONS

AHA American Heart Association

ARIC Atherosclerosis Risk in Communities

Blood Pressure BP

BMI Body Mass Index

NCDV Newly Created Derived Variables

CV Coefficient of Variation

CCS Canadian Cardiovascular Society

CDC Centres for Disease Control and prevention

CI Confidence Interval

CMR CardioMetabolic Risk

CHD Coronary Heart Disease

CRP C-Reactive Protein

CVD Cardiovascular Disease

CHMS Canadian Health Measure Survey

CCHS Canadian Community Health Survey

DXA Dual Energy X-Ray Absorptiometry

EGIR European Group for the study of Insulin Resistance

FFQ Food Frequency Questionnaire

FRS Framingham Risk Score

HDL-C High-Density Lipoprotein Cholesterol

HFCS High Fructose Corn Syrup

HbA1$_c$ Glycated Hemoglobin

HR category High Risk category

IDF International Diabetes Federation

IFG Impaired Fasting Glucose

IGT Impaired Glucose Tolerance

IR category Intermediate Risk category

LDL-C Low Density Lipoprotein Cholesterol

LFS Labour Force Survey

LR category Low Risk category

MetS Metabolic Syndrome

MEC Mobile Examination Centre

mFRS modified Framingham Risk Score

mCAFT modified Canadian Aerobic Fitness Test

NHLBI National Heart, Lung, and Blood Institute

NHANSE III Third National Health and Nutrition Examination Survey

NCEP National Cholesterol Education Program-Third Adult Treatment Panel

NHS Nurses' Health Study

OR Odds Ratio

SSBs Sugar-Sweetened Beverages

Tchol Total cholesterol

tFRS traditional Framingham Risk Score

TGs Triglycerides

T2DM Type 2 Diabetes Mellitus

TC/HDL-C ratio Total Cholesterol/ High-Density Lipoprotein Cholesterol ratio

US United States

WHO World Health Organization

WHR Waist-Hip-Ratio

WHO/ISH World Health Organization/International Society of Hypertension

WC Waist Circumference

CHAPTER 1

INTRODUCTION

1.1 Background Information and Rationale

Type 2 diabetes and cardiovascular disease (CVD) are the main outcomes of Metabolic Syndrome (MetS) (Brien & Katzmarzyk, 2006; Cameron et al., 2008). Metabolic syndrome (MetS) is a combination of five risk factors, including abdominal obesity, dyslipidemia (elevated triglycerides (TG) and reduced high-density lipoprotein cholesterol (HDL-C) level), hypertension, and elevated fasting plasma glucose (FPG)(Grundy, 2008). Patients with MetS have twice the risk of CVD and five times the risk of diabetes (Alberti, Zimmet, & Shaw, 2006; Grundy, 2008). The prevalence of MetS in Canadian adults increased from 14.4% (17.5% of men and 11.2% of women) in 1986/92 (Brien & Katzmarzyk, 2006) to 19.1% (17.8% of men and 20.5% of women) in 2011 (Riediger & Clara, 2011). Women hold greater proportion of the increment in the prevalence probably due to greater prevalence of abdominal obesity in Canadian women (Setayeshgar, Whiting, & Vatanparast, 2013a). From 1994 to 2005 the prevalence of heart disease in Canada increased by 19% and 2% among men and women, respectively (Lee et al., 2009). Also, a 70% increase (72% for females and 69% for males) in the prevalence of type 2 diabetes occurred over 10 years from 1999 to 2009 (Public Health Agency of Canada, 2011). Abdominal obesity and insulin resistance need to be prevented or monitored to control the increasing trend in MetS and its consequences (diabetes and CVD). Furthermore, dyslipidemia needs to be monitored in people with diabetes, abdominal obesity, and a strong family history of premature CVD. Current evidence suggests using Total Cholesterol/HDL-C (TC/HDL-C) ratio rather than just measuring LDL-C (Jiang et al., 2004) for the best assessment of disease risk.

In the pathogenesis of MetS, insulin resistance and central obesity stand out as potential causative factors. In Canada, obesity among adults has increased from 13.8% to 23.1% between 1978/79 and 2004 (Tjepkema, 2006). Among children and youth in 2004, the prevalence of obesity alone was 2.5 times higher than 1978/79 (Shields, 2006). The estimated prevalence of abdominal obesity in Canada increased from 1.8% in 1981 to 12.8% in 2007/09 among adolescents and from 11.4% to 35.7% in adults (Janssen, Shields, Craig, & Tremblay, 2011). To

measure the abdominal obesity as a surrogate of insulin resistance, waist circumference measurement, a convenient and practical approach, is suggested (Janssen, Katzmarzyk, & Ross, 2004). In the presence of abdominal obesity while the BMI is still below 30 kg/m^2, the severity of the disease risk increases. Therefore, to manage MetS and its outcomes, prevention strategies could be mostly confined to monitor abdominal obesity.

Physical activity and diet management are the two fundamental approaches to prevent or treat abdominal obesity and therefore MetS and the outcomes. Dietary intake has been linked to individual components of MetS (Lutsey, Steffen, & Stevens, 2008; Malik et al., 2010; McKeown et al., 2004; van Meijl, Vrolix, & Mensink, 2008). Evidence indicates that high fiber consumption is associated with lower risk of obesity (Laitinen, Pietiläinen, Wadsworth, Sovio, & Järvelin, 2004; P. Newby et al., 2003), MetS (McKeown, et al., 2004; Sahyoun, Jacques, Zhang, Juan, & McKeown, 2006), type 2 diabetes (Montonen, Knekt, Järvinen, Aromaa, & Reunanen, 2003; Schulze, Liu, et al., 2004; Stevens, Ahn, Houston, Steffan, & Couper, 2002; Weickert et al., 2006), and CVD (Jacobs & Gallaher, 2004; Mellen, Walsh, & Herrington, 2008). The positive association between sugar-sweetened beverages and risk of obesity (Bermudez & Gao, 2010; Mozaffarian, Hao, Rimm, Willett, & Hu, 2011; Vartanian, Schwartz, & Brownell, 2007) and type 2 diabetes (de Koning, Malik, Rimm, Willett, & Hu, 2011; Malik, Popkin, Bray, Després, & Hu, 2010; Palmer et al., 2008) is well-documented. However, the contribution of different specified food groups or beverages in the risk of abdominal obesity, elevated TC/HDL-C ratio, MetS, type 2 diabetes and CVD at the population level in Canada is still unknown.

The Canadian Health Measures Survey (CHMS) conducted by Statistics Canada in partnership with Health Canada and the Public Health Agency of Canada provides the opportunity to identify specific factors associated with chronic diseases or risk factors. Dietary intake has been assessed in CHMS data which can be utilized to evaluate the association with diseases and risk factors controlling for potential covariates.

1.2 Objectives and hypothesis of studies

The overall purpose of this research was to explore the association between dietary intake and five health outcomes including abdominal obesity, elevated TC/HDL-C ratio, diabetes, metabolic syndrome, and cardiovascular disease.

1.2.1 Study 1 (outlined in chapter 4)

Overview of the study

Study 1 focused on dietary intake of Canadians aged 6 to 79 y and the association of diet with cardiovascular disease risk factors and diabetes in Canadian adults. The objective was to determine the dietary intake by different socio-demographic characteristics as well as the dietary intake of individuals with and without three health outcomes including abdominal obesity, elevated TC/HDL-C ratio, and diabetes. Additionally the association between dietary intake and the three health outcomes considering possible covariates using multivariable logistic model was evaluated.

Objectives

1. To determine the intake of each food group in Canadians aged 6-79 y as well as the dietary intake by different socio-demographic characteristics.
2. To determine the status of cardiovascular risk factors including abdominal obesity and elevated TC/HDL-C ratio as well as diabetes and the status of these health outcomes by dietary intake.
3. To determine the association between each health outcome with dietary intake considering potential covariates.

Hypothesis

1. It was hypothesized that dietary intake of Canadians is worse in inactive individuals or individuals with lower socio-economic status compared to active individuals or individuals with higher socio-economic status.

2. It was hypothesized that increased risk of abdominal obesity, elevated TC/HDL-C ratio, and diabetes are associated with unhealthy food and beverage intake adjusting for potential covariates.

1.2.2 Study 2 (outlined in chapter 5)

Overview of the study

Study 2 focused on metabolic syndrome among Canadians aged 12-79 y. Abdominal obesity is the main component and cause of metabolic syndrome and individuals with MetS are five times greater at risk of developing diabetes. The prevalence of MetS by socio-demographic characteristics was evaluated. Also, the dietary intake of individuals with and without MetS was analyzed. The association between dietary intake and MetS controlling for potential covariates was evaluated using multivariable logistic model.

Objectives

1. To determine the prevalence of MetS and the status of MetS by dietary intake.
2. To determine dietary intake of patients with Mets having diagnosed diabetes compared to individuals with MetS having no diagnosis of diabetes
3. To determine the association between MetS and dietary intake considering potential covariates

Hypothesis

1. It was hypothesized that dietary intake of Canadians is worse in individuals with MetS compared to individuals without MetS.
2. It was hypothesized that participants reporting having been diagnosed with diabetes adhere to a special diet.
3. It was hypothesized that increased risk of MetS is associated with unhealthy food and beverage intake adjusting for potential covariates.

1.2.3   Study 3 (Outlined in chapter 6)

Overview of the study

Risk of developing cardiovascular disease doubles in individuals with MetS. The main objective of study 3 was to estimate the 10-year risk of CVD in Canadians aged 30-74 y specifically considering individuals diagnosed with MetS. The dietary intake according to three levels of CVD risk (low, medium, high) was evaluated.

Objectives

1. To determine the 10-year risk of CVD in Canadian adults and potential changes at population level when individuals with MetS are taken into account.
2. To determine 10-year risk of CVD in Canadian adults by different socio-demographic characteristics.
3. To determine the dietary intakes of Canadians in risk categories of 10-year risk of CVD

Hypothesis

1. It was hypothesized that the overall 10-year risk of CVD increased while considering individuals with MetS.
2. It was hypothesized that the individuals with high risk for 10-year risk of CVD have unhealthy dietary intake compared to individuals with low risk of CVD.

1.2.4   Study 4 (outlined in chapter 7)

Overview of the study

In study 4 as opposed to study 3 a non-fasted sub-sample was used. Study 4 concerned the 10-year risk of CVD in Canadian aged 30-74 y considering two modifications suggested by Canadian Cardiovascular Society (CCS): 1- calculating cardiovascular age rather than actual age; 2- considering the individuals with the history of premature cardiovascular events in the first degree relative. The first objective was to compare the change in overall 10-year risk of CVD when 2 modifications were implemented. The second objective was to evaluate the distribution

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of individuals in three categories of risks. The last objective was to determine the association between dietary intake and risk of 10-year risk of CVD ≥10% controlling for potential covariates using multivariable logistic regression.

Objectives

1. To determine whether the estimate of the percent of 10-year risk of CVD will change after implementing the two modifications and which modification has stronger contribution
2. To determine the distribution of the Canadians adult in three categories of 10-year risk of CVD.
3. To determine the association between diet and the predicted 10-year risk of CVD while controlling for the potential covariates.

Hypothesis

1. It was hypothesized that estimate of 10-year risk of CVD is higher while applying two modifications compared to the estimate of 10-year risk of CVD without modifications.
2. It was hypothesized that having 10-year risk of CVD over 10% is associated with unhealthy food and beverage intake adjusting for potential covariates.

Of the four studies presented in this thesis, two have been published (Studies 2 and 3) and the third (Study 4) has been accepted for publication. Prior to presenting these studies, a Literature Review (chapter 2) and a General Methodology (chapter 3) are provided.

CHAPTER 2

LITERATURE REVIEW

2.1   Obesity

Obesity is defined as excess body fat accumulation with pathological consequences (Hardman & Stensel, 2009). Unhealthy lifestyles such as inactivity and poor diet are the fundamental contributors to obesity. Obesity is related to insulin resistance. Insulin resistance and obesity are considered as the main causes to develop MetS (Alberti et al., 2006). Obesity is increasing rapidly in many developing and developed countries throughout the world. Based on a report from WHO, 35% of adults around the world aged 20 and over were overweight in 2008, and 11% were obese (World Health Organization, 2008). The obesity rate in North American countries is among the highest in the world. In Canada from 2003 to 2011, obesity among adult men ($\geq$ 18 y) rose from 16.0% to 19.8%, and from 14.5% to 16.8% among women (Statistics Canada, 2011b). In the US the prevalence of obesity increased significantly among men from 1999-2000 to 2009-2010 (27.5% to 35.5%); whereas, among women, 33.4% were obese in 1999–2000 with no significant change in 2009–2010 (35.8%) (Ogden, Carroll, Kit, & Flegal, 2012). The increasing trend in both countries is considered a tremendous public health concern.

Obesity is associated with many chronic conditions including Metabolic Syndrome (MetS), type 2 diabetes, CVD, and some types of cancer such as colon, breast, esophagus, pancreas, gallbladder, and prostate (Nakano et al., 2010). Obesity increases cardiovascular disease risk including hypertension, type 2 diabetes, and dyslipidemia through several mechanisms. Dyslipidemia related to central obesity is characterized by elevated triglyceride levels, low high-density lipoprotein (HDL-C) levels, increased small dense, low-density lipoprotein (LDL-C) particles, and increased apolipoprotein B-100 (apoB) concentration (Miller, Nori-Janosz, Lillystone, Yanez, & McCullough, 2005). These abnormal lipoprotein levels are associated with insulin resistance and result in MetS. In fact, central obesity and insulin resistance are the two main causes of metabolic syndrome (Alberti, et al., 2006). Metabolic syndrome also is related to other chronic disorders such as diabetes and cardiovascular diseases.

2.1.1   Assessment of obesity in adults

The excess body fat in obesity can be measured by a variety of methods such as skinfold measurements, underwater weighing, bioelectrical impedance analysis, air-displacement plethysmography, and dual-energy X-ray absorptiometry (Nelms, Sucher, Lacey, & Long Roth, 2011). However, these methods are not practically or economically available in clinical settings or at the population level. Accurately measuring weight and height is an easy and rapid method to determine whether a person is obese. Body Mass Index (BMI) is obtained by dividing weight (in kilograms) by height (in meters) squared. Obesity and overweight are defined when BMI is $\geq$ 30 kg/m^2 and 25 to 29.9 kg/m^2, respectively (Nelms, et al., 2011). BMI measurement correlates well with measurements obtained from underwater weighing and DXA (Nelms, et al., 2011). However, BMI does not differentiate between muscle and fat in some situations including high muscularity (Hardman & Stensel, 2009), large skeletal mass, presence of edema, muscle wasting and osteoporosis (Nelms, et al., 2011).

Evidence from studies using computed tomography and magnetic resonance imaging has supported the role of central obesity rather than general obesity as the major contributor to the metabolic complications (Després et al., 2008; Jensen, 2006). The complications include type 2 diabetes, hypertension, dyslipidemia, coronary heart disease, and MetS (Nelms, et al., 2011). Central or abdominal obesity also might be present even when BMI is within a healthy range. To quantify adipose tissue within the abdominal region, impractical methods include magnetic resonance imaging (MRI) or computed tomography. Measuring waist circumference is a more practical approach (Janssen, et al., 2004). Waist circumference is a feasible and accessible indicator of abdominal obesity and is more precise than MRI or computed tomography when different cut-points for various ethnicities are considered (Alberti et al., 2009).

Abdominal obesity often is associated with increased insulin resistance which is the main cause of MetS. However, measuring insulin sensitivity is expensive and impractical, particularly at the population level. Thus, waist circumference as a convenient and practical approach is suggested. To define abdominal obesity for MetS, the International Diabetes Federation (IDF) has ethno-specific cut-offs as shown in (**Table 2-1**) (Alberti, et al., 2009).

To provide a better indication of relative disease risk, the combination of BMI and waist circumference can be used (**Table 2-2**). Epidemiological studies indicate that the risk of chronic diseases in adults with BMI between 25.0 kg/m^2 and 34.9 kg/m^2 increases when waist circumference exceeds 102 cm in males and 88 cm in females (**Table 2-2**).A waist circumference in excess of these values is regarded as increased, high, very high, or extremely high risk depending on the value obtained from BMI (**Table 2-2**) (Nelms, et al., 2011).

Table 2-1 Current recommended waist circumference thresholds for abdominal obesity by International Diabetes Organization

Population	Organization (reference)	Recommended waist circumference threshold for abdominal obesity	
		Men	Women
Europid	IDF[1]	≥ 94 cm	≥ 80 cm
Caucasian	WHO[2]	≥ 102 cm	≥ 88 cm
United States	AHA/NHLBI (ATPIII)[3]*	≥ 102 cm	≥ 88 cm
Canada	Health Canada	≥ 102 cm	≥ 88 cm
European	European Cardiovascular Societies	≥ 102 cm	≥ 88 cm
Asian (including Japanese)	IDF	≥ 90 cm	≥ 80 cm
Asian	WHO	≥ 90 cm	≥ 80 cm
Japanese	Japanese Obesity Society	≥ 85 cm	≥ 90 cm
China	Cooperative Task Force	≥ 85 cm	≥ 80 cm
Middle East, Mediterranean	IDF	≥ 94 cm	≥ 80 cm
Sub-Saharan African	IDF	≥ 94 cm	≥ 80 cm
Ethnic Central and South American	IDF	≥ 90 cm	≥ 80 cm

Adopted from Alberti et al., 2009
* Recent AHA/NHLBI guidelines for metabolic syndrome recognize an increased risk for CVD and diabetes at waist-circumference thresholds of ≥ 94 cm in men and ≥ 80 cm in women and identify these as optional cut points for individuals or population with increased insulin resistance.
[1] International Diabetes Federation
[2] World Health Organization
[3] American Heart Association/National Heart, Long, and Blood Institute (Adult Treatment Panel III)

Table 2-2 Classification of overweight and obesity by body mass index, waist circumference, and associated disease risk

Weight	BMI (kg/m^2)	Obesity class	Disease risk (relative to normal weight and waist circumference)	
			Men ≤ 102 cm Women ≤ 88 cm	Men > 102 cm Women > 88 cm
Underweight	< 18.5		-	-
Normal	18.5 – 24.9		-	-
Overweight	25 – 29.9		Increased	High
Obese	30 – 34.9	l	High	Very high
	35 – 39.9	ll	Very high	Very high
	≥ 40	lll	Extremely high	Extremely high

Adopted from National Heart, Lung, and Blood Institute, 2000

2.1.2 Abdominal obesity in adolescents

Metabolic syndrome in children is defined after age 10. One approach to define abdominal obesity in children is the suggestion by the International Diabetes Federation (IDF) of using ≥90th percentile cutoff in youth between the ages of 16 to 19 y (Zimmet et al., 2007). Age- and sex-specific waist circumference percentile values (cm) are available for Canadian youth 11–18 y of age from the 1981 Canada Fitness Survey (Katzmarzyk, 2004). Another approach to define abdominal obesity was developed using Third National Health and Nutrition Examination Survey (NHANES III, 1988 to 1994) and the 1999 to 2000 and 2001 to 2002 NHANES surveys among US adolescents aged 12 to 19 y (Jolliffe & Janssen, 2007). In this approach age- and sex-specific cut-points were developed for all components of the MetS excluding glucose levels. Therefore, for every 1-year from 12 to 19, there would be specific cut-points for abdominal obesity and other components of MetS (**Table 2-3** & **Table 2-4**). Each cut-point could be used for all adolescents within a 1-year age range.

Table 2-3 Age specific metabolic syndrome cut-points and corresponding percentiles for males

Age* (y)	WC (cm)		BP (mm Hg)		HDL-C (mmol/L)§ (26th)	TG (mmol/L)§ (89th)	Glucose (mmol/L)§
	NCEP ATP† (92nd)	IDF ‡ (83rd)	SBP (92nd)	DBP (97th)			
12	94.2	85.1	121	76	1.13	1.44	5.6
13	96.2	87.0	123	78	1.10	1.48	5.6
14	98.0	88.9	125	79	1.07	1.52	5.6
15	99.5	90.5	126	81	1.04	1.56	5.6
16	100.6	91.8	128	82	1.03	1.59	5.6
17	101.4	92.7	128	83	1.03	1.62	5.6
18	101.8	93.4	129	84	1.03	1.65	5.6
19	102.0	93.8	130	85	1.03	1.68	5.6
20	102.0	94.0	130	85	1.03	1.70	5.6

Adopted from Jolliffe & Janssen, 2007

*MetS cut-point values represent the midpoint of a 1-year increment (i.e., the values for age 12 years represent the values at 12.5 years) and can be used for individuals within the 1-year age range (i.e.,12.0 to 12.9 years). †MetS defined as 3 of the 5 criteria. ‡MetS defined as having elevated WC and 2 of the remaining 4 criteria. Note that elevated WC can be assumed if body mass index values above thresholds presented in. §To convert HDL-C in mmol/l to mg/dl, multiply by 38.67. To convert TG in mmol/l to mg/dl, multiply by 88.5. To convert glucose in mmol/l to mg/dl, multiply by 18.03.ATP _ Adult Treatment Panel; BP _ blood pressure; IDF _ International Diabetes Federation; NCEP _ National Cholesterol Education Program; SBP_ Systolic Blood Pressure; DBP_Diastolic Blood Pressure; TG_Triglyceride.

Table 2-4 Age specific metabolic syndrome cut-points and corresponding percentiles for females

Age* (y)	WC (cm)		BP (mm Hg)		HDL-C (mmol/L)§ (26th)	TG (mmol/L)§ (89th)	Glucose (mmol/l)§
	NCEP ATP† (92nd)	IDF ‡ (83rd)	SBP (92nd)	DBP (97th)			
12	79.5	72.5	121	80	1.25	1.60	5.6
13	81.3	74.2	123	82	1.25	1.53	5.6
14	82.9	75.7	125	83	1.26	1.46	5.6
15	84.2	76.8	126	84	1.26	1.44	5.6
16	85.2	77.7	128	84	1.27	1.46	5.6
17	86.2	78.5	128	85	1.27	1.53	5.6
18	87.0	79.2	129	85	1.28	1.61	5.6
19	87.7	79.8	130	85	1.29	1.68	5.6
20	88.0	80.0	130	85	1.30	1.70	5.6

Adopted from Jolliffe & Janssen, 2007

*MetS cut-point values represent the midpoint of a 1-year increment (i.e., the values for age 12 years represent the values at 12.5 years) and can be used for individuals within the 1-year age range (i.e.,12.0 to 12.9 years). †MetS defined as 3 of the 5 criteria. ‡MetS defined as having elevated WC and 2 of the remaining 4 criteria. Note that elevated WC can be assumed if body mass index values above thresholds presented in. §To convert HDL-C in mmol/l to mg/dl, multiply by 38.67. To convert TG in mmol/l to mg/dl, multiply by 88.5. To convert glucose in mmol/l to mg/dl, multiply by 18.03.ATP _ Adult Treatment Panel; BP _ blood pressure; IDF _ International Diabetes Federation; NCEP _ National Cholesterol Education Program; SBP_ Systolic Blood Pressure; DBP_Diastolic Blood Pressure; TG_Triglyceride.

2.1.3 Food intake and obesity

Weight gain leading to obesity occurs when energy intake chronically exceeds energy expenditure. Energy intakes have increased in recent decades due to an increase in portion sizes offered to people as they eat out. Since eating out has turned to a normal habit, energy intake level from fast food has increased compared to energy expenditure in the body (Hensrud, 2004). According to the data from the Canadian Community Health Survey (CCHS) among Canadian adults, quantity (total energy intake) had a more significant effect than quality (the balance of nutrients consumed) in developing obesity (Langlois, Garriguet, & Findlay, 2009). The only exception was the association between higher fiber intake and lower rates of obesity among Canadian men (Langlois, et al., 2009). The other components of dietary intake which are more in the center of attention in relation to obesity are usage of dietary fat and sugar-sweetened beverages (SSBs) which have been reviewed in the current thesis. SSBs is evaluated in the "beverage consumption and obesity" section.

Dietary fiber intake and obesity

Evidence suggests a beneficial role of high-fiber diets in weight control (Howarth, Saltzman, & Roberts, 2001). Since diets rich in fiber are generally low in fat, particularly saturated fat, greater consumption of high-fiber products are recommended by both Institute of Medicine and Food and Nutrition Board (Institute of Medicine Food and Nutrition Board, 2002). According to Dietary Reference Intake (DRI) the recommended consumption for dietary fiber is 14 g per 1,000 kcal, or 25 g/day for adult women and 38 g/day for adult men to protect against cardiovascular disease (Institute of Medicine Food and Nutrition Board, 2002). An inverse association between dietary fiber intake and weight gain in all different ages and sex groups was observed in all studies reviewed in **Table 2-5**.

A number of potential mechanisms explain the role of fiber in preventing weight gain. In general, a fiber-rich diet reduces calorie density compared to a high-fat diet. It contributes to early an early satiety as well as gastric distention resulting from greater bulk compared to a high-fat diet. Moreover, fiber-rich food ingestion could prolong the feeling of fullness due to an increase in the gastric emptying time (Alfieri, Pomerleau, Grace, & Anderson, 1995; Kimm,

1995). A high-fiber diet also modulates insulin response to carbohydrate. This effect results in reduced satiety since insulin is considered to be an appetite stimulant (Kimm, 1995; Ludwig et al., 1999). Thus, to prevent weight gain or to lose weight, obese or overweight individuals definitely need to take the advantage of including fiber in their diet through fruit and vegetables and/or whole-grain products.

Table 2-5Evidence for an Association between Fiber intake and obesity

Authors	Population	Obesity definition	Results
Alfieri et al., 1995	150 Canadian volunteers from a large cross-sectional study examining circadian rhythms	1) Normal weight (20.0 ≤ BMI ≤ 27.0) 2) Mildly to moderately obese (27.1 ≤ BMI ≤ 39.9) and 3) Severely obese (BMI ≥ 40.0)	- Normal weight subjects consumed higher intakes of total dietary fiber but lower intakes of fat than moderately or severely obese groups - Fiber intake was inversely associated with BMI even after adjusting for sex, age, education level, and income
Kimm, 1995	Review paper on children		- A high-carbohydrate diet accompanied by a high fiber content and conversely with lower fat seemed to be associated with a reduced risk of childhood obesity
Ludwig et al., 1999	2909 healthy black and white adults, 18 to 30 years of age enrolled in the Coronary Artery Risk Development In Young Adults (CARDIA) cohort study	Body weight, waist to hip ratio	- Body weight was inversely associated with fiber and carbohydrate and positively associated with protein intake in whites adjusted for lifestyle behaviors - Fiber consumption predicted insulin level, weight gain, and other CVD risk factors more strongly than did total or saturated fat consumption
Liu et	Nurses' Health Study	Weight	- Over the entire follow-up period, an

al., 2003	(NHS) from 1984 to 1996 including 74 091 US female nurses, aged 38–63 y	changes reported	increased intake of dietary fiber and whole grains was associated with significantly less weight gain - Adjustment for total energy intake had little effect on these results - An increase of 12 g in dietary fiber intake was associated with~3.5 kg less weight gain during 12 y
Newby et al., 2003	459 healthy men and women participating in the ongoing Baltimore Longitudinal Study (BLSA), aged 27-88 y	Annual changes in BMI	-There was a significantly greater annual increase in BMI among subjects in the meat-and-potatoes pattern (β = 0.25; 95% CI: 0.07, 0.43) and in waist circumference among subjects in the white-bread pattern (β = 0.90 cm; 95% CI: 0.12, 1.68) than among subjects in the healthy cluster (high fiber diet)
Laitinen et al., 2004	2841 men and 2930 women from The Northern Finland 1966 Birth Cohort at birth, 14, and 31 y	waist/hip-ratio $\geq$90th percentile	- The proportions of men with abdominal obesity were higher among those who did not have a daily or almost daily intake of foods rich in fiber - Among women, only infrequent consumption of fruit and fresh vegetables and salads was associated with abdominal obesity.

Dietary fat intake and obesity

Out of the four macronutrients (carbohydrate, protein, fat, and alcohol) related to energy intake, percentage of energy intake from dietary fat has been a particular focus with regards to obesity. According to NHANES data from 1971-1975 and 2005-2006, the percentage of energy from protein intakes decreased from 16.5% to 15.7% (Austin, Ogden, & Hill, 2011). Obese individuals might be able to improve their perceptions of satiety if they obtain enough percentage of energy from protein (Leidy, Carnell, Mattes, & Campbell, 2007).

There are limitations in the literature explaining definitive advantages of low-fat diets over high-fat diets in preventing obesity. A significant positive association between fat

consumption and the proportion of the population who are overweight or obese was found in several studies (Astrup, 2005; Gillis, Kennedy, Gillis, & Bar-Or, 2002; Paeratakul, Popkin, Keyou, Adair, & Stevens, 1998). The association between dietary fat intake and obesity is more significant when physical activity is limited (Bray, Paeratakul, & Popkin, 2004). It appeared that adaptation to high fat diet is slower especially when the individuals are not physically active compared to low fat diet (Lissner & Heitmann, 1995). Adaptation to a diet can be estimated through the ratio of carbohydrate to fat oxidation which is called respiratory quotient. Thus, weight loss by reduction in dietary fat intake may be more effective when physical activity increases (Bray, et al., 2004; Donahoo et al., 2008; Lissner & Heitmann, 1995; Saltzman, Dallal, & Roberts, 1997).

A high-fat diet is highly palatable, and this can lead to overconsumption of tasty foods with loss of portion control or lack of satiating power. Moreover, high-fat foods have greater energy density which can result in overconsumption of energy, regardless of fat content (Rolls, 2000; Saltzman, et al., 1997) . Long term studies indicated that portion control and physical activity, are likely to have a greater impact on weight than use of fat-modified products or reduction in dietary fat intake (Wylie-Rosett, 2002). In fact, the direct impact of percent energy intake from fat on gaining weight is inconclusive.

2.1.4 Beverage consumption and obesity

Today beverages account for a substantial share of daily calories around the world, and Canada is no exception (Garriguet, 2008a). Although, there are beverages without calories, people tend to drink high-calorie beverages such as sugar-sweetened beverages and fruit drinks. Beverages energy contents notably come from different types of sugar. The effect of beverages on obesity can be evaluated through different categories including sugar-sweetened, nutrient-based, alcoholic, and non-caloric beverages. Non-caloric drinks include plain coffee, tea, water, and diet soft drinks. Diet soft drinks and their association with obesity are still a controversy because they have no calories. Epidemiologic studies of artificial sweetener use in children have generally shown a positive association with weight gain (Brown, Banate, & Rother, 2010). In longer-term studies of those with normal weights, the use of artificial sweeteners resulted in a compensatory increase in energy at a later time (Benton, 2005).

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<u>Sugar-sweetened beverages (SSBs)</u>

A notable percentage of daily calories come from SSBs in Canadian children and adolescents (Garriguet, 2008a, 2008b). SSBs include carbonated (fizzy) or non-carbonated (still) drinks including soft drinks, fruit punch, fruit drinks, lemonade, sweetened powder drinks, or any other non-artificially sweetened beverages. Calories from SSBs may take individuals over recommended levels for calorie consumption due to high added sugars (Garriguet, 2008a) which could result in gaining weight.

A positive association between SSBs consumption and obesity has been observed. A positive associations was observed in a review study of three prospective studies with a total of 120,877 non-obese American adults who were followed for 20 years (Mozaffarian, et al., 2011). On the basis of increased daily servings of individual dietary components, increased daily SSBs intake of one serving was associated with an average of 1.0-pound weight gain in a 4-year interval controlling for potential confounders such as socio-demographic characteristics. The positive association has also been indicated in a meta-analysis from 12 cross-sectional studies (Vartanian, et al., 2007), in a 4-year cohort study among women in the Nurses' Health Study II (Schulze, Manson, et al., 2004), a Mediterranean population-based study of Spanish adults (Balcells et al., 2011), and in a cross-sectional study among American adults (Bermudez & Gao, 2010). In the Mediterranean study, an increment of 100 ml per day in soft drinks consumption was associated with an increment of 0.213 kg/m^2 in BMI (Balcells, et al., 2011). The positive association between SSBs consumption and obesity remained significant even adjusting for energy intake and socio-demographic factors in all studies.

Increased marketing and greater portion sizes are likely responsible for consuming greater SSBs in the last decade (Pereira, 2006). Increased portion sizes of soft drinks may result in overconsumption of sugar. Also, according to experimental and observational studies SSBs are less satiating than solid food and the energy in SSBs is not well compensated for by reductions in the intake of solid food (Brown, Dulloo, Yepuri, & Montani, 2008; Pereira, 2006; Popkin et al., 2006; Woodward-Lopez, Kao, & Ritchie, 2011). Consumption of high fructose corn syrup (HFCS), which is used as a sweetener to flavour SSBs in North America, may be related to increased body weight and fat mass or even to cardiometabolic diseases compared to

consumption of artificial sweetener (Bray, 2010; Malik, Schulze, & Hu, 2006; Raben, Vasilaras, Moller, & Astrup, 2002). The dramatic increase in HFCS consumption in beverages has been parallel to the obesity epidemic (Bray, 2010). A possible mechanism linking HFCS consumption to obesity is formation of fructose-1-phosphate from fructose in the liver. Fructose-1-phosphate becomes a substrate for producing triglycerides (Brown, et al., 2008; Havel, 2005). Accumulation of triglyceride in liver and elevation of systemic free fatty acid and very low-density lipoprotein (VLDL) could result in insulin resistance in liver and peripheral organs (R. J. Johnson et al., 2009). Fructose may also decrease insulin sensitivity indirectly through obesity-associated mechanism. Overall, increased intake of sugar or HFCS could result in obesity. Thus insulin resistance or HFCS could also directly cause insulin resistance.

Despite the availability of many studies in favor of the positive association between SSBs and obesity there are studies supported by industries which rarely conclude that sweetened beverages (or HFCS) are associated with increased risk of obesity. White (2008) concluded that the HFCS-obesity hypothesis is supported neither in the United States nor worldwide (White, 2008). This author is a consultant to the food and beverage industry in nutritive sweeteners, including HFCS and sucrose. He only emphasized the biologic virtue of HFCS and tried to prove that HFCS has the same sugar composition as other "benign" fructose-glucose sweeteners with no adverse effect. He has not mentioned the effect of increased energy intake from SSBs and incomplete compensation in having less solid food. Moreover, he failed to consider the direct metabolic effect of HFCS on decreasing insulin sensitivity and increasing triglycerides. Forshee et al. (2007) in a study supported by Tate and Lyle had a similar finding with HFCS. Tate and Lyle is a global provider of distinctive, high quality ingredients and solutions to the food, beverage and other industries. The authors concluded that HFCS does not appear to contribute to being overweight and obesity any differently than do other energy sources (Forshee et al., 2007). Similar to White, Forshee et al. also concluded that HFCS would not affect satiety or absorption and metabolism of fructose any differently than would sucrose. Although this conclusion might be correct, it has very little to do with obesity. While high content of sugar in SSBs is not the only contributor to increase trend of obesity, enlarged portion sizes and intention in drinking soft drinks along with sedentary lifestyle cannot be ignored.

Milk (nutrient-based beverages)

The association between milk and weight gain is controversial. An inverse association between milk and weight gain has been reported in some epidemiologic, clinical, and rodent studies (Zemel, Richards, Milstead, & Campbell, 2005). In a 3-year longitudinal study among 53 white children (2 - 96 months) higher intakes of calcium, monounsaturated fat, and dairy products were associated with lower body fat (Carruth & Skinner, 2001). An inverse association between milk and weight gain was seen among 18 to 30 y American adults (Pereira et al., 2002) and 9-14 y girls in Hawaii (Huang & McCrory, 2005). However, in another 3-year longitudinal study among 196 non-obese pre-menarcheal girls 8–12 y dairy food consumption was not associated with BMI z-score or percentage body fat during adolescence (Phillips et al., 2003). Different contributors might be responsible for lack of consistent findings in studies including age, sex, types of milk consumed, and confounders such as physical activity, dietary pattern and lifestyle indicators.

Types of milk in terms of fat content and amount of milk consumed indicated different impacts on weight gain. In a cohort study from 1996 to 1999 among US children aged 9 to 14 y, children who reported higher total milk intake (≥3 serving/day) experienced larger weight gains than those who consumed between 1 and 2 serving/day. Also, children who drank more 1% and skim milk had larger weight gain than those who drank smaller amounts of 1% and skim milk (Berkey, Rockett, Willett, & Colditz, 2005). In the latter study, dietary calcium intake was positively correlated with weight gain but dietary fat was not, although the impact of calcium dropped when adjusted for energy. It is possible that the effect of elevated BMI is attributable to the more energy obtained from drinking more milk or to the weight-gain induced by whey protein or estrone, an estrogenic hormone in milk (Berkey, et al., 2005). According to the NHANES 1999-2004 data in individuals 18 y and over, whole milk was weakly and negatively associated with the risk of central obesity, whereas low-fat milk had the opposite effect. However, they suspect that the weak positive association between low-fat milk intake and central obesity was due to modified behaviors among obese individuals (Beydoun et al., 2008). Regarding nutrients in milk, higher magnesium intake was associated with a greater reduction in the prevalence of obesity and central obesity than increment in calcium (Beydoun, et al., 2008). Therefore, the inverse association between milk consumption and obesity possibly happens when

it is consumed no more than three servings per day regardless of the types of milk through the virtue of low glycemic index and compounds such as calcium, magnesium, vitamin D, and proteins.

Several factors in milk have been investigated for their possible impact on weight loss. Bioactive compounds of milk or the dietary pattern of low glycemic index of drinking milk appeared to be responsible for the inverse association between consuming milk and obesity (Pereira, et al., 2002). Milk has some compounds modulating fat accumulation, such as vitamin D and angiotensin-converting enzyme inhibitory peptides. The latter is responsible for inhibiting angiotensin II production which induces adipocyte lipogenesis (Barba, Troiano, Russo, Venezia, & Siani, 2005). Calcium and magnesium contents of milk have been inversely associated with weight gain and body fat (Beydoun, et al., 2008; Pfeuffer & Schrezenmeir, 2007). The inverse association between calcium and magnesium with obesity and central obesity may be attributable to the ability of these nutrients in forming soaps with fatty acids in the intestine and thus reducing the digestible energy content of the diet. Therefore, the beneficial metabolic alterations on lowering fat accumulation by milk consumption cannot be ignored.

Alcoholic beverages

Alcohol is the second most energy-dense macronutrient after fat and it has appetite-enhancing property which results in weight gain (Lukasiewicz et al., 2005). In a prospective study of 7608 men aged 40–59 y in 24 British towns, heavy alcohol intake (≥ 30 g or 2 drinks/d) was associated with weight gain and obesity, irrespective of the type of alcohol consumed (Wannamethee & Shaper, 2003). A cross-sectional study of 1481 women aged 35–60 y and 1210 men aged 45–60 y revealed a J-shaped relationship between total alcohol consumption and waist-to-hip ratio (WHR) in both sexes and between total alcohol consumption and BMI in men only (Lukasiewicz, et al., 2005). The J-shaped relationship was also observed for wine consumption. Individuals who consumed < 100g/day alcohol compared to those who were non-consumers or high drinkers had the lowest WHR. Spirits consumption was also associated with greater BMI while no association was seen between beer and BMI or WHR. An eight-year prospective study of 49,324 women 27 to 44 y examining the risk of weight gain ≥ 5 kg related to alcohol consumption revealed that heavy consumers (> 3 drinks/day) had higher odds ratio

compared to light to moderate drinkers. Further, light to moderate consumers had significantly lower odds ratio than non-consumers (Wannamethee, Field, Colditz, & Rimm, 2004).

The question why moderate consumption is related to lower weight compared to the non-consumption or high consumption of alcohol has been addressed by researchers. Suter et al. (1997) speculate moderate alcohol drinkers unlike heavy alcohol drinkers usually add alcohol to their daily energy intake rather than substituting it for food (Suter, Hasler, & Vetter, 1997). Thus, the negative association between moderate alcohol consumption and weight gain in some studies may be due to unmeasured confounders such as smoking, physical activity, and socioeconomic status or it may reflect the physiologic effect of alcohol on increased basal energy expenditure and inefficient energy use (Wannamethee, et al., 2004). It is suggested that moderate alcohol consumption could increase weight in overweight individuals (Crouse & Grundy, 1984; Wannamethee, et al., 2004). Overweight and obese individuals who are consuming a high fat diet, may gain weight more easily by drinking alcohol than lean consumers due to the effect of alcohol on inhibiting lipid oxidation (Wannamethee, et al., 2004). Therefore, the negative association between moderate consumption of alcohol and weight gain might be observed more in normal weight individuals.

The effect of moderate consumption of alcohol or type of alcohol may influence abdominal obesity as well as general obesity. Consuming more than three drinks of alcohol (>30 g ethanol) in Spanish individuals aged 25-74 y was significantly associated with increased risk of abdominal obesity controlling for diet and lifestyle confounders (Schröder et al., 2007). In a population-based cohort of 807 men, high intake of alcohol was positively associated with waist circumference in men in the lowest tertile of BMI. The association mainly was driven by liquor intake rather than by beer or wine intake (Riserus & Ingelsson, 2007). In co-twin case-control (monozygotic twin) analysis, moderate alcohol drinking (12-17.9 g/day) accounted for 300 g less central abdominal fat than abstinence or light drinking independent of genetic and other environmental factors (Greenfield et al., 2003). Gene environment interaction analysis indicated that this association was limited to subjects at high genetic risk of abdominal obesity. Heavy drink consumption of alcohol ($\geq$ 18 g/day) was related to increased fat deposition specifically in the central body unlike moderate consumption (Greenfield, et al., 2003). Heavy alcohol consumption might have a stronger association with abdominal obesity rather than general

obesity through non-caloric mechanisms, such as alterations of steroid hormones (e.g. decrease in testosterone) or lipid mobilizing hormones that target central fat storage (Riserus & Ingelsson, 2007).

Fruit juice (nutrient-based beverages)

Fruit juice is one beverage with greater consumption among Canadian children than in Canadian adults. In Canada boys aged 1 to 3 y drink the highest and men aged 71 y and older drink the lowest amount of fruit juice (Garriguet, 2008a, 2008b). The energy content of juice comes from the major carbohydrates which are sucrose, glucose, fructose, and sorbitol. Consumption of fruit juice especially by children is problematic because of its high sugar content and low levels of fiber (Wojcicki & Heyman, 2012). In a sample of 2801 children aged 1-4 y in New York, those who were overweight had the higher risk of adiposity by fruit juice consumption (Faith, Dennison, Edmunds, & Stratton, 2006). A cross-sectional study of 1999-2002 NHANES among 2-11 y children revealed that drinking fruit juice resulted in higher consumption of energy, carbohydrates, vitamins C and B6, potassium, riboflavin, magnesium, iron, and folate and significantly lower intakes of total fat, saturated fatty acids, and added sugar (Nicklas, O'Neil, & Kleinman, 2008). In this study children consumed fruit juice less than the maximum recommended by American Academy of Pediatrics (118 to 177 ml/day) and no association was found between fruit juice consumption and weight gain. Similar results are reported in 12-18 y adolescents and the consumption of fruit juice was not associated with decreased intake of milk, meat, or grains (O'Neil, Nicklas, & Kleinman, 2010). Reviewing studies by O'Neil (2008) indicated no systemic association between 100% fruit juice consumption and overweight in children and adolescents (C. E. O'Neil & Nicklas, 2008). Thus, when fruit juice is consumed no greater than the recommended level, there is insufficient evidence to prove the positive association between fruit juice intake and obesity. The Institute of Medicine (IOM) recently suggested following the American Academy of Pediatrics and the American Heart Association recommendation of no more than four to six ounces per day (0.5 to ¾ cups) in children aged one to six years (Institute of Medicine, 2010).

2.2 Total Cholesterol/HDL Cholesterol Ratio

Monitoring lipid profile is specifically important for men over 40 years of age and menopausal women or women over 50 years of age. Further, lipids need to be monitored in people with diabetes, atherosclerosis or in individuals with risk factors such as smoking, abdominal obesity, hypertension, and a strong family history of premature cardiovascular disease (Statistics Canada, 2010). Treatment of dyslipidemia is essential for patients who most likely benefit from primary prevention for CVD (Anderson et al., 2013).

Although LDL-C is a very common and strong independent predictor of coronary heart disease especially in individuals with diabetes (Howard et al., 2000), current evidence suggests using TC/HDL-C ratio rather than just measuring LDL-C (Jiang, et al., 2004; Ridker, Rifai, Cook, Bradwin, & Buring, 2005). Cholesterol ratio is obtained by dividing HDL-C level into the total cholesterol and is a sensitive and specific index of CVD risk (Genest, Frohlich, Fodor, & McPherson, 2003). According to the two prospective cohort studies among healthy US women and diabetic men, TC/HDL-C ratio was as good as or better than apolipoprotein fractions among healthy women and was the best predictor of CVD among diabetic men (Jiang, et al., 2004; Ridker, et al., 2005). The detailed information of the two papers is described in (**Table 2-6**).

Table 2-6 Studies supporting the use of TC/HDL-C rather than LDL-C

Study Design & Population	Duration of Study	Objective
Prospective cohort study of 15 632 initially healthy US women aged 45 years or older	Over 10 years	To directly compare the clinical utility of different lipid profile as predictors of future cardiovascular events in women
Prospective cohort study of 746 diabetic men in the Health Professionals' Follow-up Study aged 46–81	6 years	To evaluate the role of non-HDL-C and apolipoprotein B, in comparison with LDL-C as predictors of cardiovascular disease

2.2.1 Criterion for TC/HDL-C ratio assessment

According to dyslipidemia guidelines suggested by the Canadian Cardiovascular Society, treatment is initiated in individuals with intermediate 10-year risk of CVD and TC/HDL-C ratio > 5.0 (Genest et al., 2009). Further, according to American Heart Association the goal is to keep the ratio below 5:1 (American Heart Association, 2011). The criterion > 5 was considered in the current thesis indicating the elevated risk for the TC/HDL-C ratio.

2.2.2 Dietary intake and risk of elevated TC/HDL-C ratio

The majority of the studies have focused on the intake and independent atherogenic lipid profiles rather than TC/HDL-C ratio. Elevated TC/HDL-C ratio is more responsive to lifestyle modifications such as increase in physical activity and weight reduction and improvements in glycemic control and dietary fat intake than an isolated LDL-C elevation (Mancini, Hegele, & Leiter, 2013).

A meta-analysis of 60 studies evaluating the effect of dietary fatty acids and carbohydrates on the serum TC/HDL-C ratio and on serum lipids revealed interesting findings (Mensink, Zock, Kester, & Katan, 2003). Such study suggested that replacement of saturated fatty acid (SF) with carbohydrate does not improve the TC/HDL-C ratio (Mensink, et al., 2003; F. M. Sacks & Katan, 2002) while replacement of SF with *cis* unsaturated fatty acid decreased the TC/HDL-C ratio (Mensink, et al., 2003). *Cis* unsaturated fatty acid can be found in canola, soybean, sunflower, and olive oils. Further, the largest cholesterol-raising effect of SF such as lauric acid, found in tropical oils such as coconut and palm kernel, was reflected in HDL-C cholesterol. As a result, lauric acid had a more favorable effect on TC/HDL-C ratio than any other fatty acid, either saturated or unsaturated (Mensink, et al., 2003).

Diet high in sucrose, glucose, and fructose (> 20% of energy) can elevate plasma triglycerides, and very low-density lipoproteins especially in men (Johnson et al., 2009). The effect of fructose is more detrimental than glucose as fasting apolipoprotein B, LDL-C, small dense LDL-C, oxidized LDL-C, and postprandial concentrations of remnant-like particle–triglyceride and cholesterol significantly increased during fructose but not glucose consumption (Stanhope et al., 2009). Several mechanisms suggested by which fructose can increase fasting

and postprandial triglyceride levels. These include increased de novo lipogenesis in the liver, elevated hepatic triglyceride synthesis, and secretion of very-low-density lipoproteins, as well as reduced lipoprotein lipase activity at the adipocyte, which lowers the rate of peripheral triglyceride clearance (Johnson, et al., 2009). Thus, initial treatment for those with elevated TC/HDL-C ratio should consist of intensifying lifestyle modification strategies and improving glycemic and dietary fat control.

2.3   Metabolic Syndrome

Metabolic syndrome (MetS) is a clustering of five chronic disease risk factors, including abdominal obesity, dyslipidemia (elevated triglycerides (TG) and reduced high-density lipoprotein cholesterol (HDLC) level), hypertension, and elevated fasting plasma glucose (FPG) (Grundy, 2008). MetS is considered to be the main contributor to cardiovascular diseases (CVD) and diabetes (Brien & Katzmarzyk, 2006; Cameron, et al., 2008).With MetS, the risk of CVD doubles and the risk of diabetes increases fivefold (Alberti, et al., 2006; Grundy, 2008). Increasing obesity and sedentary life style are related to a rise in the prevalence of MetS worldwide (Alberti, et al., 2009). The prevalence of MetS among 18-64 y Canadians based on the nationally representative Canadian Heart Health Survey over two decades ago (1986 to 1992) using Adult Treatment Panel III (ATP III) criteria was 14.4% (Grundy, 2008). The recent prevalence using Canadian Health Measure Survey data (CHMS, 2007-2009) in adults over 18 years of age, according to the latest criteria increased to 19.1% (Riediger & Clara, 2011). There is no study available on the prevalence of MetS among Canadian adolescents. Identifying the risk factors in adolescents, which may resist into adulthood, could help to prevent chronic diseases in future. The increasing trend in prevalence of MetS is an emerging public health and a clinical concern not only in Canada but also all around the globe.

2.3.1   The pathogenesis of Metabolic Syndrome

The two interrelated factors insulin resistance and obesity especially abdominal obesity are the major causative factors for MetS (Alberti, et al., 2006). Moreover, genetic factors such as adiponectin gene and environmental factors and physical inactivity play important causal roles in the presence of insulin resistance, and MetS (Grundy, 2008; Kadowaki et al., 2006). There are

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some other minor factors which contribute to MetS such as ageing, pro-inflammatory state and hormonal dysregulation. Yet, their effect compared to obesity is minor and they have varied influence across different ethnicities (Alberti, et al., 2006; P. Anderson et al., 2001). In a prospective follow-up study among 22 528 men and 24 684 women aged 25–64 y, obese subjects (BMI $\geq$30 kg/m^2) had significantly higher cardiovascular and total mortality rate than the normal weight (18.5$\leq$BMI<25 kg/m^2) subjects (Hu et al., 2005). In addition, obesity contributes to hyperglycemia, hypertension, elevated serum triglycerides (TGs), low HDL-C, insulin resistance (Tunstall-Pedoe, Kuulasmaa, Mähönen, Tolonen, & Ruokokoski, 1999) and type 2 diabetes (Kahn & Flier, 2000; Maggio & Pi-Sunyer, 2003; Zimmet, Alberti, & Shaw, 2001). Therefore, obesity is associated with all components and even the outcomes of MetS (type 2 diabetes and cardiovascular diseases).

Abdominal obesity is more appropriate than general obesity to define MetS due to its relation to metabolic complication. Body Mass Index, which indicates general obesity, is not a reliable criterion in diagnosing MetS in individuals, unless abdominal obesity is also considered (Alberti, et al., 2006). Waist circumference is considered to be a practical and convenient approach to define abdominal obesity for MetS. In a study among 218 healthy men and women intra-abdominal fat (IAF) was independently associated with all of the MetS criteria (Carr & Brunzell, 2004). Three scenarios have been proposed by Despres and Lemieux regarding the link between visceral adipose tissue and metabolic abnormalities. First, exposing the liver to high concentration of free fatty acids by hyperlipolytic state of the visceral fat leads to hyperinsulinemia, glucose intolerance, and hypertriglyceridemia. Second, visceral adipose tissue is a source of adipokines and inflammatory cytokines leading to insulin resistance, pro-inflammatory, pro-thrombotic, and pro-hypertensive state of visceral obesity. Insulin resistance, abdominal obesity, and adipokines are interrelated. They all result in inflammation, impaired fibrinolysis, elevated blood pressure, atherogenic dyslipidemia, and dysglycemia (Després, et al., 2008). The third scenario speculates that excess visceral adiposity suppresses the subcutaneous adipose tissue to act as a protective metabolic sink leads to accumulation of fat at undesired sites such as liver, heart, skeletal muscle, and pancreas (Després & Lemieux, 2006). Thus, abdominal obesity measured conveniently through waist circumference would be an appropriate indicator of metabolic complications.

2.3.2 Definitions of Metabolic Syndrome in adults

To diagnose MetS in any individual a defined standard criteria is needed. Three definitions for MetS have been widely used including: the World Health Organization (WHO), The European Group for the study of Insulin Resistance (EGIR) and the National Cholesterol Education Program-Third Adult Treatment Panel (NCEP ATP III) (**Table 2-7**). Obesity, insulin resistance, dyslipidemia and hypertension are the main components for all three definitions. However, each may have different cut-points for components or even different components to identify their clusters (Alberti, et al., 2006).

In the WHO definition, published in 1999, the major contributor is insulin resistance or its likely surrogate such as impaired glucose regulation, impaired glucose tolerance (IGT), or diabetes. In the presence of two or more of the other criteria MetS was diagnosed (Alberti, et al., 2006). Following the WHO definition, EGIR proposed a modified definition for non-diabetic individuals which are simple to use in epidemiologic studies since it did not need a euglycaemic clamp to quantify insulin secretion and resistance to measure insulin sensitivity. The EGIR definition proposed the use of fasting insulin levels to estimate insulin resistance and impaired fasting glucose (IFG) as a surrogate for IGT. Therefore, the main contributor was insulin resistance which was defined as hyperinsulinaemia- i.e., the top 25% of fasting insulin values among the non-diabetic population (Alberti, et al., 2006; Balkau & Charles, 1999). If two or more of the other criteria existed, MetS was diagnosed. The last criteria, ATP III, were established in 2001 in order to prevent coronary heart disease (CHD) and cover the limitation of the earlier definitions. It differs from the previous two definitions in two ways: 1) it did not include insulin resistance measures as a component and 2) it was not 'glucose-centric' (Adult Treatment Panel, 2001; Alberti, et al., 2006). MetS was diagnosed in any person with three or more of the five risk factors (**Table 2-7**).

Table 2-7 Metabolic syndrome definition

Criteria	WHO (1999)	EGIR (1999)	NCEP ATP III (2001)
Fasting plasma glucose		≥ 6.1 mmol/l (110 mg/dl) but non-diabetic	≥ 6.1 mmol/l (110 mg/dl)[a]
Blood pressure	≥ 140/90 mm Hg raised	≥ 140/90 mmHg or treatment	≥ 130/ ≥ 85 mmHg
Triglycerides	≥ 1.7 mmol/l (150 mg/ dl) *and/or*	> 2.0 mmol/l (178 mg/ dl) or treatment *and/or*	≥ 1.7 mmol/l (150 mg/ dl)
HDL-cholesterol	Men: < 0.9 mmol/l (35 mg/ dl) Women: < 1.0 mmol/l (39 mg/ dl)	< 1.0 mmol/l (39 mg/ dl) or treatment	Men: < 1.03 mmol/l (40 mg/ dl) Women: < 1.29 mmol/l (50 mg/ dl)
Obesity	Men: waist–hip ratio > 0.90 Women: waist–hip ratio > 0.85 and/or BMI > 30 kg/m2	Men: waist circumference ≥ 94 cm Women: waist circumference ≥ 80 cm	Men: waist circumference > 102 cm Women: waist circumference > 88 cm
Microalbuminuria	Urinary albumin excretion rate ≥ 20 μg/min or albumin:creatinine ratio ≥ 30 mg/g		

Adopted from Alberti et al., 2006
[a]The 2001 definition identified fasting plasma glucose of ≥ 6.1 mmol/l (110 mg/dl) as elevated. This was modified in 2004 to be ≥5.6 mmol/l (100 mg/dl)

Due to differences in defining components across the three sets of criteria, inconsistent prevalence of MetS has been reported. In the Australian study among the non-diabetic population only 9.2% of individuals had the same defining components across all three sets of criteria (Dunstan et al., 2005). A criteria were proposed by the International Diabetes Federation (IDF) for clinical practice as well as epidemiologic studies in May 2004. IDF held an expert workshop to produce a simple diagnostic tool to use in clinical practice and in research. It used the 2001 ATP III definition as a starting point. Insulin resistance measurement was not essential to the new definition. According to IDF criteria to diagnose MetS, a person must have abdominal obesity plus any two of four additional factors which are summarized in **Table 2-8**. If BMI was

more than 30 kg/m^2 then central obesity could be considered without measuring waist circumference (Alberti, et al., 2006).

The earlier criteria to diagnose MetS, defined by WHO or EGIR are not used anymore due to difficulties in measuring insulin resistance or the 'glucose-centric' virtue of the criteria. In the most recent and widely-accepted definition, the ATP III (2001) and IDF (2004) criteria were combined in 2005 by IDF in collaboration with American Heart Association/National Heart, Lung, and Blood Institute (AHA/NHLBI). Both sides agreed that abdominal obesity should not be a prerequisite for diagnosis but that it is 1 of 5 criteria, so that the presence of any 3 of 5 risk factors constitutes a diagnosis of MetS (Alberti, et al., 2009). According to this latest definition, IDF suggested using different cut-offs for waist measurement according to different ethnic groups to define abdominal obesity (**Table 2-1**), other cut-offs are the same (**Table 2-8**).

Table 2-8 International Diabetes Federation definition of metabolic syndrome

Abdominal obesity	Cut-offs were presented in **Table** 2-1
Raised triglycerides	≥ 1.7 mmol/l (150 mg/dl)
	or specific treatment for this lipid abnormality
Reduced	< 1.03 mmol/l (40 mg/dl) in males
HDLcholesterol	< 1.29 mmol/l (50 mg/dl) in females
	or specific treatment for this lipid abnormality
Raised blood pressure	Systolic: ≥ 130 mmHg or
	Diastolic: ≥ 85 mmHg
	or treatment of previously diagnosed hypertension
Raised fasting plasma	Fasting plasma glucose ≥ 5.6 mmol/l (100 mg/dl)
glucose	or previously diagnosed type 2 diabetes
	If > 5.6 mmol/l or 100 mg/dl, oral glucose tolerance test is strongly
	recommended but is not necessary to define presence of the syndrome

Adopted from Alberti et al., 2006

2.3.3 Definitions in children and adolescents

In children below the age of 10 y, MetS is not diagnosed because the majority of children do not present health consequences of MetS. However, they may demonstrate the risk for metabolic syndrome. The IDF adult criteria have been used for adolescents aged ≥ 16 y (Zimmet, et al., 2007). In adolescents according to the criteria established by IDF for 10-16 y, the only

difference from adult is found in waist circumference which is $\geq 90^{th}$ percentile and one (rather than a sex-specific) cut-off is used for High Density Lipoprotein cholesterol (HDL-C) (**Table 2-9**). The purpose of this definition for children and adolescents is for managing type 2 diabetes in this age group (Alberti et al., 2004) and is for providing a unified tool for diagnosing MetS (Zimmet, et al., 2007).

The percentiles for different components which are defined in some studies (Cook, Weitzman, Auinger, Nguyen, & Dietz, 2003), have been arbitrary chosen and have no health basis (Jolliffe & Janssen, 2007). Therefore, using cut-points that are on the basis of health risk, such as the ones used in ATP III and IDF criteria, seems more appropriate. For this purpose, age- and sex-specific cut-points criteria for adolescents have been developed using National Health and Nutrition Examination Survey (NHANES III), 1999 to 2000, and 2001 to 2002 data (Jolliffe & Janssen, 2007). These age and sex-specific cut-points are available for adolescents 12-20 y (**Table 2-3** & **Table 2-4**). Age- and sex-specific growth curves were developed by linking each MetS component values to the corresponding adult ATP and IDF cut-point (Jolliffe & Janssen, 2007). Each cut-point reflects the midpoint of a given year (i.e., for age 12 the cut-points represents 12.5y) from 12 to 20 y and could be used for all adolescents within 1-year age range (**Table 2-3** & **Table 2-4**).

Table 2-9International Diabetes Federation definition of at risk group and metabolic syndrome in children and adolescents

Criteria	6 - < 10[a]y	10 - < 16y	16 + (adult criteria)
Obesity (waist circumference)	≥90th percentile	≥90th percentile or adult cutoff if lower	WC ≥ 94cm for Europid males and ≥ 80cm for Europid females, with ethnic-specific values for other groups*)
Triglycerides		≥1.7 mmol/L (≥150 mg/dL)	≥1.7 mmol/L (≥150 mg/dL) or specific treatment for high triglycerides
HDL-C		<1.03 mmol/L (<40 mg/dL)	<1.03mmol/L (<40 mg/dL) in males and <1.29mmol/L (<50 mg/dL) in females, or specific treatment for low HDL
Blood pressure		Systolic BP ≥130 or diastolic BP ≥85 mm Hg	Systolic BP ≥130 or diastolic BP ≥85 mm Hg or treatment of previously diagnosed hypertension
Glucose		FPG ≥5.6 mmol/L (100 mg/dL)** or known T2DM	FPG ≥5.6 mmol/L (100 mg/dL)** or known T2DM

Adopted from Zimmet et al., 2007
[a]Metabolic syndrome cannot be diagnosed, but further measurements should be made if there is a family history of metabolic syndrome, T2DM, dyslipidemia, cardiovascular disease, hypertension and/or obesity.
*For those of South and South-East Asian, Japanese, and ethnic South and Central American origin, the cutoffs should be≥90 cm for men, and ≥80 cm for women.
**For clinical purposes, but not for diagnosing the MetS, if FPG 5.6-6.9 mmol/L (100-125 mg/dl) and not known to have diabetes, an oral glucose tolerance test should be performed

2.3.4   Clinical outcome of Metabolic Syndrome

As discussed previously, MetS is considered to be the main contributor to cardiovascular diseases (CVD) and diabetes (Brien & Katzmarzyk, 2006; Cameron, et al., 2008). Regardless of ethnic diversity, with MetS the risk of CVD doubles (Grundy, 2008) and the risk of diabetes increases five-fold (Alberti, et al., 2006). Non-diabetic people with MetS are also at high risk in developing type 2 diabetes (Grundy, 2008). In a 3-year cohort study of US adults 14.9% and 17.9% of those with four or more components of MetS developed CVD and diabetes respectively

(Klein, Klein, & Lee, 2002). In another cohort study among US adult, individuals with 1 to 2 MetS risk factors were at increased risk for mortality from CHD and CVD (Malik et al., 2004). The increased risk has also been observed in other populations. According to Lakka et al. (2002) the greater risks for CVD death is suggested for those middle-aged men with MetS in Finland (Lakka et al., 2002). Higher CVD and overall mortality in adults was associated with MetS in Sweden (Isomaa et al., 2001). Since MetS has CVD risk factors, predicting relative risk of CVD by MetS is expected (Alberti, et al., 2006). The factor which contributes to increased risk of CVD and diabetes could be inflammatory biomarker such as C-reactive protein (CRP). Patients with the MetS and CRP levels of ≥3 mg/L had the highest risk of CVD events and diabetes (Haffner, 2006). Thus, prevention and management strategies for MetS simultaneously could prevent CVD events and diabetes.

2.3.5   Food intake and Metabolic Syndrome

Dietary intake has been linked to individual components of MetS (Lutsey, et al., 2008; Malik, Popkin, Bray, Després, & Hu, 2010; McKeown, et al., 2004; van Meijl, et al., 2008) although, the protective or non-protective effect of each food group is not consistent throughout studies. The Dietary Guidelines for Americans (DGA) concentrates on chronic diseases such as metabolic syndrome, CVD, diabetes, hypertension, cancer, and osteoporosis. New DGA recommend consuming more of certain foods and nutrients such as fruits, vegetables, whole grains, fat-free and low-fat dairy products and seafood. In contrast, DGA recommends consuming fewer foods with sodium (salt), saturated fats, trans fats, cholesterol, added sugars, and refined grains (Dietary Guidelines for Americans US, 2010). Consumption of a balanced dietary pattern of all food groups considering the DGA guideline is necessary for prevention and management of these chronic conditions.

Dietary fibers

Foods have been shown to influence different components of MetS. Dietary fibers have a favorable effect on blood pressure, body weight, visceral adiposity, insulin sensitivity, and diabetes (Anderson et al., 2009). Soluble fibers can sustain significant hypo-cholesterolemic effects for long-term periods and thus reflects the best-characterized effects of fiber consumption

on risks for CVD. High levels of dietary fiber intake are associated with a significant reduction in the prevalence of diabetes due to reduction in postprandial glycemia and insulinemia and increase in insulin sensitivity (Weickert, et al., 2006). In a prospective study conducted between 1981 to 1984 among 535 healthy persons ≥ 60 y, by increasing quartile categories of whole-grain intake, glucose concentrations and BMI decreased as opposed to refined-grain foods in which fasting glucose concentration and systolic blood pressure increased by elevating the quartile categories of intake (Sahyoun, et al., 2006). Additionally, compared with the individuals in the lowest quartile of whole-grain or refined-grain intake, those who consumed more were at a significantly lower or greater risk of having MetS, respectively, even after controlling for several risk factors . Similarly according to the fifth examination (1991–1995) of the Framingham Offspring Study among individuals ≥ 26 y, cereal fiber and whole-grain intakes were inversely associated with the MetS after adjustment for life-style characteristics, energy intake, and physical activity (McKeown, et al., 2004). The inverse association between whole-grain intake and MetS was largely explained by cereal fiber since the association no longer remained significant between whole-grain intake and the risk of MetS after adjusting for cereal fiber. The favorable impact of fiber from cereal or whole-grain products on MetS or its component needs to be taken into account.

Data on a favorable association between intake of fruit and vegetables and the incidence of MetS are inconclusive. During 12.9 years of follow-up among 28,082 US women ≥ 39 y, higher intake of all fruits but not all vegetables was significantly associated with reduced risk of hypertension after adjustment for lifestyle and dietary factors. However, adding BMI to the models eliminated all associations (Lichtenstein et al., 2006). Also, fruit and vegetable consumption was associated with a better lipid profile. There was no association between incidence of MetS and consumption of fruits and vegetables according to prospective data from Atherosclerosis Risk in Communities (ARIC) (Lutsey, et al., 2008). Similarly in a cross-sectional study by Mckeown et al (2004) fiber from fruit, vegetable, and legumes were not inversely associated with the prevalence of MetS as opposed to the fiber from cereal (McKeown, et al., 2004). Among 482 Tehrani females aged 40-60 y, compared with those in the lowest quintile of fruit and vegetable intakes, individuals in the highest category had lower odds of having MetS, even after controlling for CRP concentrations, lifestyle, and dietary confounders (Azadbakht &

Esmaillzadeh, 2009). Consumption of diets high in fruit and vegetables was associated with lower blood pressure (Appel et al., 2006). Fruit and vegetables might have different favorable impact on MetS and its components in different population with different amount of consumption. Further, the impact might be more influential in populations with low prevalence of obesity.

The possible inverse association between dietary fiber and MetS is explained through the biological properties of fibers. Soluble fibers mostly present in fruit have beneficial impact on the components of MetS including glucose levels and lipid profile due to their contribution in reducing the stomach emptying and absorption of cholesterol, respectively. Insoluble fibers mostly present in cereals might have more beneficial effect on the MetS and its components than soluble fibers by decreasing BMI and glucose concentration. Adjustment for life-style confounders and even for fruit, vegetable and dairy product did not change the negative association between cereal fibers and MetS (McKeown, et al., 2004; Sahyoun, et al., 2006). However, the beneficial impact of fruit lowered after adjustment for BMI (Lichtenstein, et al., 2006). Therefore, it appears that in the investigation of the impact of fibers on MetS and its components the source of fiber is necessary to consider including cereals and fruit and vegetables.

Meat consumption

There is not enough evidence on the intake of red meat and increased risk of MetS due to detrimental effects of saturated fat content. The Atherosclerosis Risk in Communities (ARIC) study revealed that Americans in the highest quintile of meat consumption (red or white) had 26% greater risk of developing MetS compared to individuals in the lowest quintile of meat consumption (Lutsey, et al., 2008). It was suggested that high content of saturated fat in meat which is adversely associated with cholesterol, blood pressure, obesity, and diabetes risk might be responsible. Among 482 Tehrani female teachers aged 40–60 y, red meat was directly associated with increased risk of MetS adjusting for potential confounders, BMI, and CRP. MetS and its components (high serum triacylglycerol concentrations, low serum HDL-C, and high systolic blood pressure) were more prevalent among those in the upper quintile of meat consumption than among those in the lowest quintile (Azadbakht & Esmaillzadeh, 2009). Since

CRP-related inflammation has been reported to be involved in the pathogenesis of MetS, they proposed that the association between red meat and MetS might be explained by the effect of CRP. However, there was no association between the intake of meat or fish and MetS in men or in women of a 9-year follow-up Epidemiological Study on the insulin resistance syndrome in France (Mennen et al., 2000). The method of assessing dietary intake was different in the studies. In the first two studies food frequency questionnaire (FFQ) was used as opposed to the latter study in which only 2 questions were asked ("How much meat do you eat per day?; How many times do you eat fish per week?"). Moreover, there is not enough evidence on the impact of different types of meat in terms of protein content (red meat and white meat) on MetS and its components. More investigation is warranted to fill the gap in the understanding of the association between meat consumption and MetS.

Studies evaluated the impact of fish consumption on the risk of MetS or its components, focus on the omega-3 (n-3) fatty acid content of fish. According to a population-based prospective cohort study (4 years) which included 3,504 male and female Koreans aged 40 to 69 y, the incidence of MetS was 57% lower in men who ate fish daily when compared with those who ate fish less than once a week (Baik, Abbott, Curb, & Shin, 2010). Similarly, the incidence of MetS was 47% lower in men in the top decile of n-3 fatty acid intake when compared with those in the bottom decile. The n-3 fatty acid/fish pattern was associated with lower odds of MetS (OR: 0.54, 95% CI: 0.34, 0.86) in a cross-sectional analysis of 1207 Puerto Rican adults aged 45–75 y (Noel, Newby, Ordovas, & Tucker, 2010). In another cross-sectional population based survey in France among middle aged men, the negative association between MetS and tertiles for fish intake remained significant after multivariate adjustment (Ruidavets et al., 2007). Diets including fish reduce triglycerides and total and LDL cholesterol and increase HDL cholesterol (Mori et al., 1994). Additionally, increased n-3 fatty acid intake alters the composition of n-3 and n-6 fatty acids in cell membranes in favor of n-3 fatty acids. This leads to production of eicosanoids that have a minimal potency in platelet aggregation, vasoconstriction, and inflammation (Galli & Simopoulos 1989). Fish consumption has its protective impact in developing MetS through n-3 content. However, whether n-3 supplements could be a substitution for fish consumption needs further investigation.

2.3.6 Dietary pattern and Metabolic Syndrome

Different dietary patterns are characterized by a variety of food groups. A traditional and healthy dietary pattern called the Mediterranean Diet (MedDiet) is characterized by a high consumption of legumes, fruit and vegetables, grains and olive oil, a moderate consumption of wine and dairy products and a low consumption of red and processed meat, cream and pastries. Western dietary pattern which is not recommended is usually characterized as high intake of red meat, refined grains, high fat dairy products, sweets and dessert whereas prudent dietary pattern is considered as the healthy pattern including high fiber and low fat products. Every ethnic group has their own dietary pattern. Therefore, it is necessary to explore the variety of dietary patterns and their impact on the risk of MetS and its components (**Table 2-10**).

Table 2-10 The impact of different dietary patterns on metabolic syndrome and its determinants.

Study	Population/country	Dietary pattern investigated	Result
Prospective studies			
Sonnenberg et al., 2005	1615 Framingham Offspring-Spouse Study (FOS) women age 18 to 76y in the US	<u>Heart Healthier:</u> higher consumption of dietary fiber, F&V, and low-fat milk and low intakes of total and saturated fat. <u>Lighter Eating:</u> lowest intake of refined grains, soft margarines and oils, and sweets and animal fats. <u>Wine and Moderate Eating dietary pattern:</u> high intakes of alcohol, particularly wine, cholesterol-rich foods, high-fat dairy, and snack foods but low consumption of sweetened beverages and comparatively low dietary lipid intakes <u>Higher fat dietary pattern:</u> lowest intakes of fruits and low-fat milk and the high levels of refined grains, soft margarines and oils, sweets and animal fats, and saturated fat <u>The female Empty Calorie pattern:</u> highest intake of total fat, calories, and sweetened beverages and lowest intakes of dietary fiber and vegetables.	-MetS prevalence was highest in those with Empty Calorie eating patterns in both obese and non-obese women. -Women with Wine and Moderate Eating patterns had lower overall MetS rates, lower dyslipidemia and higher rates of hypertension. -Heart Healthier and Lighter Eating women had comparatively high rates of elevated BP, high TGs, and abdominal obesity -Women with the Higher Fat dietary pattern had more problematic HDL levels but relatively lower (albeit high) rates of hypertension and abdominal obesity.
Kimokoti et al., 2012	1146 Framingham Offspring-Spouse Study (FOS) women age 25	<u>Heart Healthier</u> <u>Lighter Eating</u>	-In multivariate-adjusted models, the Higher fat and Wine clusters had

		Wine and Moderate Eating dietary pattern: Women with higher fat dietary pattern The female Empty Calorie pattern	lower odds for abdominal obesity compared with the Empty Calorie cluster. -None of the clusters were associated with MetS or other MetS components.
to 77y in the US			
Lutsey et al.,2008	Prospective data from Atherosclerosis Risk in Communities (ARIC) study with three-year intervals reexamination. n=9514 participants aged 45-64y in the US	Western dietary pattern: high intakes of refined grains, processed meat, fried foods, and red meat Prudent dietary pattern: greater consumption of cruciferous and carotenoid F&V, fish, and poultry	-Higher scores for the Western dietary pattern were associated with greater risk of developing MetS, even after adjustment for behavioral characteristics. -Participants in the highest quintile of Western dietary pattern scores had an 18% greater risk of developing MetS than those in the lowest quintile. -Consumption of a prudent dietary pattern was not associated with incidence of MetS.
Rumawas et al.,2009	2730 participants of the Framingham Heart Study Offspring Cohort followed up for 7 years in the US	Mediterranean-style dietary pattern score (MSDPS)	-Those in the highest quintile category of MSDPS had the lowest cumulative incidence of MetS. -Participants with a higher MSDPS had significantly lower waist circumference (WC), FPG, or TG and higher HDL-C at the follow-up examination than those with lower scores, after multiple adjustment

Tortosa et al., 2007	Seguimiento Universidad de Navarra (SUN) dynamic cohort, composed of 3,497 Spanish university graduates followed up for 6 years in Spain	MedDiet	-The inverse relationship between adherence to MedDiet and the cumulative incidence of the MetS was observed
Cross-sectional studies			
Esmailzadeh et al.,2007	583 sample of individuals, representative of Tehrani female aged 40–60 y in Iran	Healthy dietary pattern: high in fruits, tomatoes, poultry, legumes, cruciferous and green leafy vegetables, other vegetables, tea, fruit juices, and whole grains
Western dietary pattern: high in refined grains, red meat, butter, processed meat, high-fat dairy products, sweets and desserts, pizza, potatoes, eggs, hydrogenated fats, and soft drinks and low in other vegetables and low-fat dairy products.
Traditional dietary pattern: refined grains, potatoes, tea, whole-grains, hydrogenated fats, legumes, and broth	- Even after additional control for BMI, the inverse association of the healthy dietary pattern score AND…
- The positive association of western dietary pattern score with MetS remained significant
Panagiotakos et al., 2007	1,514 men (aged 18 to 87y) and 1,528 women (aged 18 to 89y) from the Attica region in Greece	Component 1, healthful food pattern: low-fat products such as fish, F&V, legumes, cereals,
component 2, high glycemic index and high-fat pattern: red or white meat and	- Component 1 was associated with 13% lower likelihood of having the MetS, whereas component 6 was associated with 26% higher likelihood of having the MetS.

		meat products, and potatoes Component 3: consumption of pasta Component 4: intake of dairy products and eggs Component 5: consumption of sweets Component 6: consumption of alcohol	- Component 1 was inversely associated with WC, systolic BP, TG and positively associated with HDL-C levels. - Component 2 and 6 was positively correlated with WC and inversely correlated with HDL-C levels
Babio et al., 2009	808 high cardiovascular risk participants of the Reus PREDIMED Centre conducted in Spain	MedDiet	-There has been an inverse association between adherence to the MedDiet and the prevalence of the MetS even after multiple adjustment
Deshmukh-Taskar et al., 2009	Bogalusa Heart Study (1995–1996), USA. Young adults (19–39 years; $n= 995$; 61 % females/39 % males)	Western Dietary Pattern (WDP): refined grains, French fries, high-fat dairy products, dishes with cheese, red meats, processed meats, eggs, snacks, sweets and desserts, sweetened beverages and condiments Prudent Dietary Pattern (PDP): whole grains, legumes, vegetables, tomatoes, fruits, 100 % fruit juices, low fat dairy products, poultry, clear soups and low-fat salad dressings.	-WC and the occurrence of MetS were inversely associated with the PDP. -Serum TG was negatively associated with both PDP and WDP. -After adjusting for BMI in addition to other covariates, serum HDL-C was inversely associated with the WDP. -The overall occurrence of MetS did not differ by the two dietary intakes.
Dibello et al., 2009	American Samoan (n = 723) and Samoan (n = 785) adults (≥18 y)	The neo-traditional dietary pattern: high intake of local foods, including crab/lobster, coconut products, and taro, and low intake of processed foods,	- Neo-traditional dietary pattern was associated with decreased WC in both population and with increased HDL-C levels among American

		including potato chips and soda	Samoa.
		The modern pattern: high intake of processed foods such as rice, potato chips, cake, and pancakes and low intake of local foods	-The modern dietary pattern was associated with increased prevalence of MetS and increased levels of TG in both Samoa and American Samoa.
Denova-Gutierrez et al., 2010	Health Workers Cohort Study. n=5240 (1489 men and 3751 women) in Mexico	Western dietary pattern: high intake of pastries, refined cereals, corn tortillas, and soft drinks and low consumption of whole cereals, seafood, and dairy products	-After controlling for age and sex, participants in the highest tertile of the Western dietary pattern score had higher odds of MetS.
		Prudent dietary pattern: high intakes of processed vegetable juices, potatoes, fresh fruits, fresh vegetables, and legumes	-The prudent dietary pattern was not associated with MetS.
		High-protein/-fat dietary pattern: greater consumption of red meat, processed meat, margarine (saturated fats), and eggs.	-After more adjustment the western dietary pattern was associated with higher prevalence of high FPG, high serum TG, low serum HDL-C, WC, and high BP.

Among all the papers reviewed in **Table** 2-10 almost all studies on MedDiet were in support of a significant inverse association between MetS and Med Diet (Babio et al., 2009; Dietary Guidelines for Americans US, 2010; Rumawas, Meigs, Dwyer, McKeown, & Jacques, 2009; Tortosa et al., 2007). Regardless of MedDiet, the significant association between dietary intake and MetS reviewed in **Table 2-10** was mostly observed for unhealthy dietary habits and MetS such as Western dietary intake (Denova-Gutiérrez et al., 2010; Esmaillzadeh et al., 2007; Lutsey, et al., 2008), high-protein/-fat dietary pattern (Denova-Gutiérrez, et al., 2010), empty calorie pattern (Sonnenberg et al., 2005), consumption of alcohol (Panagiotakos, Pitsavos, Skoumas, & Stefanadis, 2007), and the modern pattern (DiBello et al., 2009). High intake of sugar in beverages as well as refined grains and low intake of fruit and vegetables are the dominant indicators of Western dietary habits. In only two cross-sectional studies significant negative association between healthy eating habits and occurrence of MetS was observed (Esmaillzadeh, et al., 2007; Panagiotakos, et al., 2007). No prospective study indicated such negative association between healthy eating and the incidence of MetS which necessitates further investigation. Unlike MedDiet which is a traditional dietary pattern with the same food group content in any community, healthy dietary pattern studied in **Table** 2-10 were different in terms of food groups included. There might be a requirement to reach a healthy dietary pattern by reviewing studies. The foods which were included evenly in healthy dietary patterns were fruit and vegetables, legumes and low fat dairy products which all are disappeared in Western dietary pattern. MedDiet contained all healthy food ingredients, and perhaps could be the best healthy dietary pattern for individuals with MetS or any chronic condition.

2.3.7   Beverage consumption and metabolic syndrome

Sweetened beverages (regular soda and diet soda) and metabolic syndrome

Evidence suggests a positive association between consumption of sweetened beverages and metabolic disorders such as obesity (Malik, et al., 2006), MetS (Denova-Gutiérrez, et al., 2010), type 2 diabetes (Schulze, Manson, et al., 2004), CVD, and hypertension (Johnson et al., 2007). A cross-sectional study of 5240 Mexican individuals aged 20 to 70 y showed that consuming more than two servings of sweetened beverages daily was associated with two times

greater risk of MetS than not consuming sweetened beverages (Denova-Gutiérrez, et al., 2010). A similar result was observed in Dhingra's study in which individuals consuming ≥1 soft drink/d had a higher prevalence of MetS (Dhingra et al., 2007). In a recent population-based study among Costa Ricans substituting one serving of homemade fruit juice for one serving of regular soda or instant fruit juice was associated with 30% and 29% lower risk of MetS, respectively (Mattei, Malik, Hu, & Campos, 2012). Consumption of ≥ 1 serving/day of instant fruit juice or ≥ 1 serving/day of SSBs were associated with greater risk of MetS compared to those who never consumed instant fruit juice or SSBs after adjustment for energy intake and BMI. Therefore, it is probable that even having one serving of SSB/d increases the risk of developing MetS.

The effect of sugar-sweetened beverages (SSBs) on MetS or its components is mainly related to the high content of sugar. Sugar-sweetened beverages contribute to weight gain due to rapidly absorbable carbohydrates such as sucrose (50% glucose and 50% fructose) and high-fructose corn syrup (most often 45% glucose and 55% fructose) (Malik, et al., 2006). Sugar-sweetened beverages also lead to incomplete compensation for total energy intake at subsequent meals; that is, obtaining energy from SSBs does not necessarily result in having less food in the subsequent meal. Additionally, SSBs may increase the risk of metabolic syndrome and type 2 diabetes by raising dietary glycemic load, which in turn leads to insulin resistance, β-cell dysfunction, and inflammation (Schulze, Manson, et al., 2004). Metabolic syndrome is correlated with elevated serum uric acid raised by fructose in the form of table sugar or high-fructose corn syrup in sweetened beverages (Nakagawa, Tuttle, Short, & Johnson, 2005). Uric acid inhibits nitric oxide bioavailability which is necessary for insulin to stimulate glucose uptake. Therefore, fructose-induced hyperuricemia may have a pathogenic role in MetS by developing insulin resistance. More investigations are needed to study the effect of lowering uric acid on features of the MetS and to address potential pathophysiologic effects of uric acid (Heinig & Johnson, 2006).Therefore, SSBs could predispose the individuals to develop MetS indirectly through gaining weight or directly by increasing insulin resistance.

Not all studies have been in favor of the positive association between regular sweetened beverages and prevalence of MetS. In the Atherosclerosis Risk in Communities (ARIC) cohort study of 9514 participants from four US communities, sweetened beverages were associated with incidence of MetS after baseline adjustments including age, sex, race, education, and total

calories (HR= 1.17). However, the association became non-significant with additional adjustments of behavioral characteristics and food intake (Lutsey, et al., 2008). In a Multi-Ethnic Study of Atherosclerosis (MESA) among Caucasian, African American, Hispanic, and Chinese adults, data showed no significant associations between sugar-sweetened soda consumption and risk of either MetS or type 2 diabetes (Nettleton et al., 2009). According to a population-based study in three counties in Iran, no association between SSB consumption and MetS was found before or after adjustment for potential confounders including BMI (Khosravi-Boroujeni, Sarrafzadegan, et al., 2012). To investigate the effect of SSBs and MetS controlling the potential confounders especially BMI is warranted. Also, the effect might be more evident for the components of MetS rather than MetS. Another assumption might be explained through the intention of some patients with MetS to consume less SSBs and replace it with diet soft drinks.

In the ARIC study, diet soda intake was strongly associated with increased MetS risk (Lutsey, et al., 2008). As diet soda was higher among diabetics than non-diabetics; therefore, it was possible that reverse causality or residual confounding may explain this finding. Similarly in the MESA study, compared with non-consumers, the risk of MetS was 36% greater in those consuming $\geq$1 serving of diet soda which did not remain significant after adjustment for adiposity (waist circumference and/or BMI). However, a 67% elevated risk of type 2 diabetes due to drinking diet soda remained significant after adjustment (Nettleton, et al., 2009). Artificial sweeteners in diet sodas impair the ability of the body to predict the caloric content of foods and may lead to higher food intake and increased body weight (Dhingra, et al., 2007). Moreover, it is likely that people with diagnosed disease such as diabetes adhere to a special diet (Fitzgerald, Damio, Segura-Perez, & Perez-Escamilla, 2008) such as drinking diet soda rather than regular soft drinks. Diabetics are reported to pay more attention to the food labels especially to the sugar information, than those without diabetes. In the Multiethnic Cohort (MEC) study on average diabetics consumed significantly 2.6 times more diet soft drinks than individuals without diabetes; whereas, the consumption of regular soft drinks was less than half of what individuals without diabetes drank (Nöthlings et al., 2011). Moreover, Nettleton found that individuals consuming $\geq$1 daily serving of diet soda had a significantly greater risk of developing elevated waist circumference ($\geq$102 cm if male and$\geq$88 cm if female) or elevated fasting glucose ($\geq$100 mg/dl) during follow-up (Nettleton, et al., 2009). This is probably due to lack of energy

compensation by drinking diet soda. Thus, excluding individuals who have already been diagnosed as diabetic in studies may give a more precise evaluation of the association between SSBs or diet soft drink with MetS.

<u>Milk</u>

Although the majority of the existing literature suggests a negative association between dairy consumption and MetS, the data still remain inconclusive. There are studies which found inverse relationships between dairy intake and risk of developing, MetS, type 2 diabetes (Fumeron et al., 2011) and cardiovascular disease (Elwood et al., 2008). In a 9-year follow up of Atherosclerosis Risk in Communities (ARIC) study of 9514 participants from four US communities, individuals with the highest quintile of dairy consumption were found to be at 13% lower risk of developing MetS (Lutsey, et al., 2008). Another 9-year follow-up study in France indicated that all dairy products were inversely associated with incidence of MetS. However, cheese and dietary calcium density, as opposed to other dairy product, were not inversely associated with impaired fasting glucose or type 2 diabetes (Fumeron, et al., 2011). According to a recent review on the association between dairy intake and MetS, 5 out of 10 cross-sectional and 2 out of 3 prospective studies found an inverse association (Crichton, Bryan, Buckley, & Murphy, 2011). Using NHANES 1999–2004 data (8970 women, 8091 men aged ≥18 y), a significant inverse association between consumption of dairy products particularly, milk and yogurt was seen with obesity, central obesity, and MetS (Beydoun, et al., 2008). Research revealed that drinking milk lowers the risk of developing MetS by 62% in men residing in the UK. (Elwood, Pickering, & Fehily, 2007). A study of children in Argentina (Hirschler, Oestreicher, Beccaria, Hidalgo, & Maccallini, 2009) found milk consumption to be inversely associated with MetS components: waist circumference, insulin resistance, and blood pressure. The inverse association between dairy products and MetS needs to be distinguished between different dairy products and provide more detailed information on the quantity of intake. Various types of milk products may influence MetS or its components differently.

The beneficial impacts of milk components on chronic conditions such as MetS and cardiovascular diseases outweigh the adverse effects of the high saturated fat content of milk. In contrast to earlier studies where the high fat intake from dairy foods was implicated in CVD risk,

recent analyses show no adverse relationships (Elwood, et al., 2008). Milk consumption has beneficial impact on blood pressure. Observational data have shown the relationship between inadequate dairy product consumption with increased risk of arterial pressure consistently among age/sex and ethnic groups and across the range of blood pressures in adults, infants, elderly and in pregnant women (Jorde & Bønaa, 2000; Miller, DiRienzo, Reusser, & McCarron, 2000). Dairy proteins are precursors of angiotensin-I converting enzyme-inhibitory peptides, which may lower blood pressure (van Meijl, et al., 2008). Milk consumption lowers cholesterol levels, likely due to its calcium and protein content (Pfeuffer & Schrezenmeir, 2007). Calcium binds to intestinal fatty acids or bile acids and helps to remove them from the body, or it changes intracellular calcium metabolism by suppressing calciotropic hormones such as parathyroid hormone and 1, 25-hydroxyvitamin D (calcitriol). Increased dietary calcium results in decreased fatty acid synthesis by reduction in vitamin D activity and decreased insulin secretion by the pancreas. Consequently, lipogenesis is reduced and lipolysis is enhanced, resulting in a net fat loss (Huang & McCrory, 2005; van Meijl, et al., 2008). Milk decreases glycemic response compared with mixed meals due to its whey content which is insulinotropic (Pfeuffer & Schrezenmeir, 2007). Also, whey protein has the ability to inhibit fat deposition (Huang & McCrory, 2005). Milk is considered as the main dietary source of vitamin D in Canada (Vatanparast, Calvo, Green, & Whiting, 2010). Studies reported that inadequate vitamin D levels were associated with risk for diabetes (Mitri, Muraru, & Pittas, 2011), hypertension (Awad, Alappat, & Valerio, 2012), and cardiovascular disease (Parker et al., 2010). In summary, milk consumption, regardless of its fat content, may have beneficial impact related to metabolic complications due to the availability of different components such as calcium, magnesium, whey, or vitamin D.

Alcoholic Beverages

Both protective and detrimental effects have been reported from alcohol drinking on MetS (Fan et al., 2008). The effect depends on the type and amount of consumption. Analysis on data from 8,125 participants of the NHANES III revealed that mild to moderate alcohol consumption particularly wine and beer was associated with a lower prevalence of MetS especially among whites, with a favorable influence on lipids, waist circumference, and fasting insulin (Freiberg, Cabral, Heeren, Vasan, & Ellison, 2004). In a study among 7962 Korean

adults, an increasing dose-response relationship was found between alcohol consumption and MetS (Yoon, Oh, Baik, Park, & Kim, 2004). Different findings may be attributable to the varied alcohol consumption patterns in different populations (Fan, et al., 2008). Among the US population using NHANES (1999-2002) data, drinking alcohol more than the recommended level ($\geq$ 3 drinks/d for men or $\geq$ 2 drinks/d for women) and having one or more binge drinking episode (number of days having five or more drinks in the past 12 months) were significantly associated with increased risk of MetS compared with current drinkers (participants who consumed 12 alcoholic drinks or more during the past 12 months) (Fan, et al., 2008). The protective impact of alcohol is attributed to the moderate consumption of alcohol.

The J-shaped or the protective effect of moderate consumption of alcohol on MetS seems to be similar to the effect of alcohol on MetS components. Moderate consumption contributes to increase HDL-C level (Fan, et al., 2008; Yoon, et al., 2004). The level of HDL-C was significantly higher in all alcohol consumption groups compared with non-drinkers of 4341people residing in the Seoul and Kyung-gi regions (Lee, Jung, Park, Rhee, & Kim, 2005). Also, light to moderate consumption may enhance insulin sensitivity (Goude, Fagerberg, & Hulthe, 2002). Heavy consumption of alcohol is related to an increase in other components which are risk factors for cardiovascular disorders such as blood pressure (Lee, et al., 2005) as well as triglycerides (Fan, et al., 2008; Yoon, et al., 2004). Therefore, the beneficial impact of the moderate consumption is evident for MetS and its components, specifically HDL-C.

2.4 Diabetes

Diabetes is a lifelong condition in which either the body does not produce enough insulin (type 1 diabetes), or the body cannot use the insulin (type 2 diabetes). Nine out of ten people with diabetes have type 2 (Health Canada, 2012). The number of people with type 2 diabetes is increasing due to rise in obesity prevalence and sedentary life-style worldwide. About 285 million of people worldwide have diabetes and it is expected to reach 438 million in 2030 (Canadian Diabetes Association, 2009). In 2012, 29.1 million (9.3%) Americans (American Diabetes Association, 2014) and 1.9 million (6.5%) Canadians aged 12 or older (Statistics Canada, 2013a) had diabetes. Aboriginal people are at three to five times higher risk of diabetes compared to the general population. Approximately 80% of new Canadians are from the

population with higher risk of type 2 diabetes: Hispanic, Asian, South Asian or African descent (Canadian Diabetes Association, 2009). In 2010, approximately 12 billion dollars was spent on diabetes in Canada. This value is expected to reach 16.9 billion in 2020 (Canadian Diabetes Association, 2009). The increasing trend in the prevalence and cost of diabetes, necessitates the associated organizations to establish effective diabetes prevention and control strategies.

2.4.1   Risk Factors, outcome and treatment of diabetes

Genetics has an important role in manifesting both type 1 and type 2 diabetes. Obesity or central body adiposity in particular increases the risk of type 2 diabetes. Unhealthy diet, physical inactivity regardless of body weight, high-birth weight or low-birth weight babies, poor placental growth, and obesity related insulin resistance are other factors that might increase risk of type 2 diabetes (Canadian Diabetes Association, 2009; Nelms, et al., 2011). Major chronic complications of diabetes are CVD, nephropathy, neuropathy, amputation, and retinopathy. Cardiovascular disease is the major complication of diabetes in both men and women. In fact, some argued "diabetes is a cardiovascular disease" (Grundy, 1999). Factors that are responsible for developing CVD in diabetic adults are obesity, physical inactivity, heredity, sex, and advancing age. These factors worsen the major risk factors such as dyslipidemia, hypertension, and glucose tolerance (Grundy, 1999). Management and treatment of diabetes include medical therapy, physical activity both aerobic and resistance training for improving insulin sensitivity and dietary management (Nelms, et al., 2011). Health-promotion and disease-prevention strategies for the entire population are necessary to minimize the risk of developing diabetes. Also, should type 2 diabetes be developed, different strategies should be followed to manage it effectively.

2.4.2   Food intake and risk of diabetes

Dietary control plays a critical role in managing, treating, or preventing diabetes. IDF suggests diabetics avoid foods high in sugars and saturated fats, and limit alcohol consumption. Diabetics are recommended to include more fiber from different sources in their diet. In the 8-y follow-up study of 91 249 female nurses in the United States, high intakes of cereal and fruit fiber were associated with a lower risk of diabetes after adjustments for BMI, alcohol

consumption, smoking, family history of diabetes, and other covariates. Among the different sources of fiber, cereal fiber was the most strongly associated with decreased risk of diabetes (Schulze, Liu, et al., 2004). According to the Finnish Mobile Clinic Health Examination Survey, a cohort study of 2286 men and 2030 women aged 40–69 y, the observed negative association between whole-grain consumption and type 2 diabetes persisted after adjustment for potential confounding factors related to lifestyle (Montonen, et al., 2003). The association was independent of the consumption of fruit and vegetables. The ARIC study during 9 years follow-up after adjustment for age, BMI, education, smoking status, physical activity, sex, and field center, found no statistically significant associations of intake of total dietary fiber, fruit fiber, legume fiber, glycemic index, or glycemic load with the incidence of diabetes (Stevens, et al., 2002). However, cereal fiber intake was inversely associated with risk of diabetes in whites but not in African-Americans due to small sample size. Intake of fiber especially cereal is important in diabetes regimen due to its effect on lowering BMI and glucose concentration.

There is no conclusive evidence on the influence of high dietary fat intake and increased risk of diabetes. According to the Health Professionals Follow-Up, the increased risk of type 2 diabetes was associated with greater intake of saturated fat and total fat (van Dam, Willett, Rimm, Stampfer, & Hu, 2002). However, the association was attenuated after adjustment for other lifestyle factors including intake of cereal fiber, magnesium and even disappeared after controlling for BMI. Intakes of oleic acid, *trans*-fat, long-chain n-3 fat, and α-linolenic acid were not associated with diabetes risk after multivariate adjustment. In the cohort of the Nurses' Health Study among US women no association was found between total or saturated fat and risk of type 2 diabetes (Salmeron et al., 2001). Yet, *trans*-fatty acids increased and polyunsaturated fatty acids reduced the risk. In a study among Hispanic and non-Hispanic white adults, high fat, low carbohydrate diet significantly predicted type 2 diabetes risk among individuals with similar baseline levels of obesity and impaired glucose tolerance (Marshall, Hoag, Shetterly, & Hamman, 1994). No definitive evidence is available on the positive association between dietary fat intake and risk of diabetes. Further research is required considering different types of dietary fat and comparing healthy or pre-diabetic individuals with diabetic ones.

2.4.3 Beverage consumption and risk of diabetes

Sugar-Sweetened Beverages and Risk of Diabetes

Prospective cohort studies have found a strong association between SSBs and type 2 diabetes. In the Health Professionals Follow-Up Study (HPFS) in men, the Hazard Ratio (HR) for SSBs consumption after adjustment for confounders was 1.24 whereas artificially sweetened beverages were not significantly associated with type 2 diabetes after multivariate-adjusted analysis (de Koning, et al., 2011). Nurses' Health Study II revealed that consuming more than one can of SSB or fruit punch per day was associated with increased risk of type 2 diabetes even after controlling the confounders in women (Schulze, Manson, et al., 2004). Another prospective follow-up study among African American women found higher incidence of type 2 diabetes associated with both soft drinks and fruit drinks (Palmer, et al., 2008). Drinking two or more cans of soft drinks or fruit drinks per day increased the incidence of diabetes 24% and 31%, respectively, compared with drinking less than 1 can of soft drink per day. However, orange and grapefruit juice or diet soft drink consumption was not associated with the incidence of diabetes. Fruit drink consumption was associated with the incidence of type 2 diabetes independent of body weight while the association of soft drinks was mediated by body weight (Palmer, et al., 2008). In addition, based on a meta-analysis of 11 prospective cohort studies, individuals in the highest quintile of SSBs consumption (most often 1–2 servings/day) had a 26% greater risk of developing type 2 diabetes than those in the lowest quintile (none or <1 serving/month) after adjustment for potential confounding variables such as diet and lifestyle factors (Malik, Popkin, Bray, Després, Willett, et al., 2010) . Evidence indicates that even drinking one can of SSB per day could possibly increase the risk of developing type 2 diabetes.

Several possible mechanisms are suggested to explain the association between SSBs and higher risk of type 2 diabetes. It seems that weight gain is responsible for half of this association. In addition, HFCS contribute to increase insulin secretion and decrease leptin release and thus reduces satiety. It appears that rapidly absorbable carbohydrate in the form of refined sugar in SSBs is the main factor related to the adverse impact of SSBs on type 2 diabetes (Malik, Popkin, Bray, Després, Willett, et al., 2010; Palmer, et al., 2008).

<u>Coffee/Tea and Risk of Diabetes</u>

Coffee (caffeinated and decaffeinated) and tea have been reported to have inverse association with the risk of type 2 diabetes (Huxley et al., 2009; Salazar-Martinez et al., 2004; Tuomilehto, Hu, Bidel, Lindström, & Jousilahti, 2004). In a meta-analysis of prospective studies high intakes of coffee, decaffeinated coffee, and tea were associated with reduced risk of diabetes (Huxley, et al., 2009). Drinking three to four cups of coffee per day was associated with an approximate 25% lower risk of diabetes than drinking none or two or fewer cups per day. Individuals who drank more than three to four cups of decaffeinated coffee or tea per day had an approximate one-third and one-fifth lower risk of diabetes than those consuming no decaffeinated coffee or tea, respectively. However, the potential effect of confounding on the relationship was not examined (Huxley, et al., 2009). In two cohort studies in the US an inverse relationship between coffee consumption and risk of diabetes has been observed (Iso, Date, Wakai, Fukui, & Tamakoshi, 2006; Paynter et al., 2006). In both women and men in the Atherosclerosis Risk in Communities (ARIC) Study (1987–1999), who drank more than 4 cups (0.95 liter) of coffee per day a significant inverse association was observed as well as a significant relative hazard of 0.66 and 0.69 compared with those who drank none, respectively (Paynter, et al., 2006). A prospective cohort study, using data from the First National Health and Nutrition Examination Survey found a negative association between coffee consumption and developing diabetes particularly among ≤ 60 y individuals who had previously lost weight and consumed ground-caffeinated coffee, ground-decaffeinated coffee, regular tea and caffeine itself (Greenberg, Axen, Schnoll, & Boozer, 2005). The Nurses' Health Study and Health Professionals' Follow-up Study among 41 934 men from 1986 to1998 and 84 276 women from 1980 to 1998, revealed an inverse association between coffee intake and type 2 diabetes after adjustment for age, body mass index, and other risk factors. The association for decaffeinated coffee was modest but there was no association for tea consumption (Salazar-Martinez, et al., 2004). No significant association was found between green tea and black tea consumption with a risk of diabetes in a cohort study in Takayama (Oba et al., 2010). The inverse association between coffee consumption and diabetes was stronger than the effect of tea. Not all studies agree on the association between tea and diabetes.

Observing the inverse association of decaffeinated coffee on diabetes led researches to focus on the effect of other components of tea and coffee. Epidemiological studies found that acute consumption of caffeine could have an adverse health effect. However, glucose intolerance and decreased insulin sensitivity caused by acute consumption can be improved by chronic consumption. Non-caffeine components may be responsible for enhanced glucose tolerance and insulin sensitivity such as chlorogenic acid, lignin, and magnesium (Greenberg, et al., 2005; Huxley, et al., 2009) (**Figure 2-1**). The cohort study in Takayama city investigating the effect of caffeine on the risk of diabetes concluded that the beneficial effect of coffee consumption exists independent of its caffeine content (Oba, et al., 2010). Caffeine intake from coffee, green tea and other caffeinated beverages and chocolate snacks was not associated with the risk of diabetes (Oba, et al., 2010). The lack of such effect for instant-caffeinated coffee may support the assumption of non-caffeine impact of components due to the removal of such constituents in the process of manufacturing. However, it is possible that caffeine may reduce the risk of diabetes by inducing weight loss through the effect of thermogenesis and stimulation of lipid oxidation (Greenberg, et al., 2005) (**Figure 2-1**).
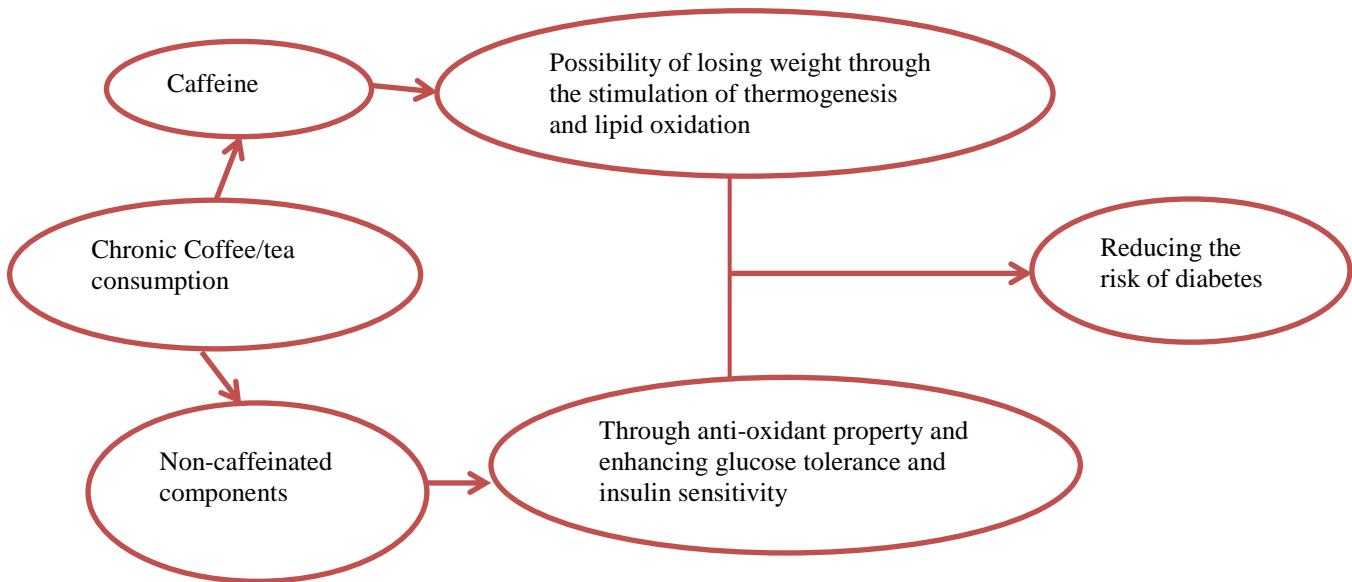
Figure 2-1 How does coffee help to reduce the risk of diabetes?

Alcoholic Beverages and Risk of Diabetes

Alcohol consumption is an important and modifiable risk factor for type 2 diabetes. Studies have found nonlinear (U-shaped) association between alcohol consumption and risk of type 2 diabetes (Beulens et al., 2005; Wannamethee & Shaper, 2003). For women who participated in the European Prospective Study Into Cancer and Nutrition a linear negative association was found between moderate alcohol consumption and the risk of type 2 diabetes. However, type of beverage did not bring about any difference. In addition, life-time alcohol consumption has shown a U-shaped relationship with the risk of type 2 diabetes (Beulens, et al., 2005). In another 4-year prospective study, wine consumption was inversely associated with the risk of type 2 diabetes among women. Among men the highest intake was associated with a reduced risk compared to lowest intake (Hodge, English, O'dea, & Giles, 2006). Consuming 210g alcohol over three or fewer days had an elevated odds ratio, while the same total amount over four or more days was not associated with increased risk. Therefore, frequency of consumption appeared to be more important than total volume. In a 32-year longitudinal study in Sweden, BMI as a covariate attenuated the relationship between alcohol consumption and risk of type 2 diabetes. When socioeconomic status and physical activity were included in the model, wine was not related to the reduced risk of type 2 diabetes anymore (Lapidus et al., 2005). Therefore, the association between alcohol intake and the risk of diabetes may be exaggerated if adjustment is not made for potential confounders.

2.5   Cardiovascular disease (CVD)

Cardiovascular disease (CVD) is a group of disorders related to heart and blood vessels including coronary heart disease, cerebrovascular disease, peripheral arterial disease, rheumatic heart disease, congenital heart disease, deep vein thrombosis and pulmonary embolism (World Health Organization, 2011). CVD is the largest killer of the world which claimed 17.1 million lives in 2004 and, it is expected, 23.6 million in 2030. CVD is the leading cause of death among Canadians too (Public Health Agency of Canada, 2010). In 2005 about 1.3 million Canadians had heart disease, a 19% and 2% increase from 1994 among men and women, respectively (D. Lee et al., 2009). In 2008 CVD accounted for 29% of all deaths in Canada, 28.0% in males and 29.7% in females (Heart and Stroke Foundation, 2012). Behavioral risk factors are liable for 80% of

coronary heart disease and cerebrovascular disease including tobacco use, physical inactivity, and unhealthy diet (World Health Organization, 2011). Unhealthy diet and physical inactivity manifest in individuals as increased blood pressure, increased blood glucose, increased blood lipids, overweight and obesity (intermediate risk factors). All these risk factors predispose individuals to develop CVD. Although some risk factors cannot be changed such as family history, sex or age, heart disease can be prevented by following a heart-healthy lifestyle including increasing physical activity, stopping smoking, and having a healthy dietary habit.

2.5.1 Risk prediction charts

Although CVD deaths decreased by 27% from 1994 to 2004 in the US (Nelms, et al., 2011) and 25% over the past 10 years in Canada (Heart and Stroke Foundation, 2012) as a result of progression in treatment and prevention, the burden of CVD cost is still huge for the governments of these countries. According to the American Heart Association and the National Heart, Lung, and Blood Institute (NHLBI) the cost of CVD and heart disease in the US is calculated at about $475.3 billion in 2009 (Vogenberg & Fendrick, 2010). Over $20.9 billion is spent each year in Canada for physician services, hospital expenses, wages and decreased productivity related to heart disease and stroke (Heart and Stroke Foundation, 2012).Therefore, identifying individuals who are at high risk for CVD would lessen the cost and death caused by CVD. The World Health Organization/International Society of Hypertension (WHO/ISH) generated charts to estimate the cardiovascular risk of people who have not yet manifested CVD clinically (Mendis et al., 2007). Individuals diagnosed with CVD need intensive lifestyle intervention and appropriate drug therapy (World Health Organization, 2007a).

The risk prediction charts are derived from the WHO Comparative Risk Assessment study. They give 10-year risk of a fatal or non-fatal myocardial infarction and stroke by sex, age, smoking, systolic blood pressure, presence or absence of diabetes and total cholesterol for 14 WHO epidemiological sub-regions (Mendis, et al., 2007). The relative risk of risk factor profiles along with the population-level estimate of absolute risk was obtained from low- and middle-income countries (LMIC). There are two sets of charts for settings which are or are not able to measure blood cholesterol for individuals over 40 y. Another approach to predict 10-year risk of CVD is Framingham Risk Score (FRS). The FRS, developed through the Framingham Heart

Study from 1971 to 1974, is the most commonly recommended assessment tool for evaluating 10-year risk of CVD in the US (Sheridan, Pignone, & Mulrow, 2003). The National Cholesterol Education Program (Adult Treatment Panel-III) recommends using FRS to assess 10-year risk of CVD (Wilson, D'Agostino, et al., 1998). The FRS has been validated for the Canadian population by a committee of clinicians and researchers (Sheridan, et al., 2003). Using any of the risk assessment tools depends on the situation in which the study is going through. Obtaining 10-year risk of CVD using WHO/ISH charts are not easy at the population level especially when data have already been collected; however, FRS is useable at both individual and population levels.

Assessment of CVD using FRS

Separate score sheets were developed for each sex according to age, blood pressure, total cholesterol (T-Chol), low density lipoprotein cholesterol (LDL-C), and high density lipoprotein cholesterol (HDL-C) categories. Using either LDL-C or T-Chol is optional in assessing CVD risk (Sheridan, et al., 2003) depends on the availability of either of them. The points assigned for each risk factor are based on the value for the β-coefficient of the proportional hazards regressions (Wilson, D'Agostino, et al., 1998). Further, the presence or absence of diabetes and smoking status (smoker versus non-smoker) need to be considered. All points are added up at the end and specific percent of 10-year risk of CVD will be assigned for each specific point achieved (**Appendices 1 & 2**).

Cardiometabolic risk

MetS is considered to be the main contributor to CVD and diabetes (Brien & Katzmarzyk, 2006; Grundy, 2008). Regardless of ethnic diversity, the risk of CVD doubles with MetS; also, the risk of diabetes increases five-fold (Dekker et al., 2005; Galassi, Reynolds, & He, 2006; Gami et al., 2007; Stern, Williams, González-Villalpando, Hunt, & Haffner, 2004). However, an increase in relative risk of CVD cannot be used to evaluate absolute risk. Furthermore, reported relative risk of CVD and its association with MetS are not comparable between studies as not all studies take into consideration potential confounders. The new approach on assessing "cardiometabolic risk (CMR)" or "global cardiometabolic risk" considers

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the factors that go beyond the traditional risk factors (Leiter et al., 2011a, 2011b). In 2009, the Canadian Cardiometabolic Risk Working Group suggested that CMR represents the comprehensive catalogue of factors related to CVD and type 2 diabetes (Leiter, et al., 2011a, 2011b). Therefore, in evaluating CMR, both MetS and 10-year risk of CVD are considered. The calculation of FRS followed by the evaluation of the presence or the absence of MetS helps identify individuals whose risk might be underestimated. Therefore, in order to evaluate CMR, both the risk factors of MetS and the risk factors used to calculate 10-year risk of CVD are considered. This novel approach, CMR, has been used to identify the risk of CVD at the individual level (Leiter, et al., 2011a, 2011b); however, to my knowledge no nationally representative study has used this method to evaluate the risks at the population level. Moreover, it is not understood if calculating CMR could possibly change the prevalence of the 10-year risk of CVD at a population level. This issue was evaluated in my second paper published in International Journal of Hypertension (Setayeshgar, Whiting, & Vatanparast, 2013b).

Modifying Framingham Risk Score

In 2012, the Canadian Cardiovascular Society (CCS) modified FRS to increase the appropriate use of evidence-based CVD risk assessment in the management of dyslipidemia as a substantial factor in reducing risk in the Canadian population. Two modifications were suggested (Anderson, et al., 2013). Firstly the percent of 10-year risk of CVD was doubled in subjects aged 30-59 y without diabetes with the history of premature CVD present in a first-degree relative before 55 y for men and 65 y for women. Secondly, since a 10-year risk does not entirely account the risk in younger individuals; CCS recommends calculating "cardiovascular age" for each individual (Anderson, et al., 2013).

For the second modification, calculating the cardiovascular age, CCS suggested two charts for males and females. Blood pressure, total cholesterol/HDL ratio, smoking, and diabetes are the indicators needed to obtain the cardiovascular age for each individual. However, the charts are applicable at the individual level. Cooney et al suggested a formula to achieve the cardiovascular age applicable at the population level (Cooney et al., 2012). The generic equation to calculate risk age, from a Cox function, where age is calculated as a risk factor is as follows:

$$Risk\ Age = [(\beta chol \times chol) + (\beta sBP \times sBP) + (\beta smok \times currsmoking) +$$
$$(\beta age \times age) - (\beta chol \times 4) - (\beta sBP \times 120)] \div \beta age$$

Where chol is cholesterol and sBP is systolic blood pressure. The β is achieved by running a regression analysis with the percent of 10-year risk of CVD as the dependent variable and total cholesterol, sBP, smoking (0=non-smokers, 1=smoker), and age (30-74 y) as independent variables. After obtaining the risk age and implementing the modified ages for each individual, new 10-year risk of CVD or mFRS is achieved using two modifications.

2.5.2 Food intake and risk of CVD

The role of diet is crucial in the development and prevention of cardiovascular disease. Diet is one of the key variables that will impact all other cardiovascular disease risk factors. WHO suggests three elements which increase the risk of heart attack and stroke: tobacco use, an unhealthy diet, and physical inactivity. The recommendation for reducing the incidence of CVD regarding dietary factors is eating five or more portions of fruit and vegetables each day, reducing intake of fat (particularly saturated fatty acids no more than one-third of fat intake) less than 35% of total energy requirement, reducing salt intake to 6 g/d and eating one portion of oily fish per week (Kelly & Stanner, 2003). The evidence-based impact of each food group on CVD needs to be evaluated.

Fruit and vegetable intake and CVD risk

Dietary habit with high intake of fruit and vegetables are associated with a lower risk of obesity (Bes-Rastrollo, Martinez-Gonzalez, Sanchez-Villegas, de la Fuente Arrillaga, & Martinez, 2006; He et al., 2004), hypertension (Pfeuffer & Schrezenmeir, 2007; Wang, Shen, Goldman, & Bibbins-Domingo, 2012), and type 2 diabetes (Schulze, Liu, et al., 2004; Stevens, et al., 2002) which are all risk factors for CVD. Also, the direct inverse association between fruit and vegetable consumption and risk of CVD (not the risk factors) appears to be supported by evidence from epidemiologic studies (Bazzano, Serdula, & Liu, 2003; Dauchet, Amouyel, Hercberg, & Dallongeville, 2006). According to a meta-analysis of nine cohort studies, fruit and vegetable consumption was inversely associated with the occurrence of Coronary Heart Disease, CHD (Dauchet, et al., 2006). The relative risks (RRs) of CHD for each increment of 1 portion

per day of fruit and vegetables varied between 0.79 and 0.97. The corresponding RRs for fruit were between 0.81 and 0.95 and for vegetable were between 0.60 and 0.98. In general, the risk of CHD was decreased by 4% for each additional portion per day of fruit and vegetables and by 7% for fruit consumption (Dauchet, et al., 2006). The majority of articles in a review indicated dose-response relationships between intake of fruit and vegetable and CVD risk (Bazzano, et al., 2003). Fruit and vegetable intake and their beneficial impact to reduce the risk of CVD and its risk factors is supported by many studies especially when it is included in a healthy dietary pattern.

Fruit and vegetables are always included in healthy dietary patterns. High intakes of fruit and vegetables usually are associated with a prudent dietary pattern and inversely related to the consumption of western pattern (Heidemann et al., 2008; Kerver, Yang, Bianchi, & Song, 2003). The prudent diet was associated with a 28% lower risk of cardiovascular mortality among American women (Heidemann, et al., 2008). Additionally, the western pattern in US adults was associated positively with cardiovascular biomarkers including serum C-peptide, serum insulin, and glycated hemoglobin and inversely with red blood cell folate concentrations after adjustment for confounding variables (Kerver, et al., 2003).The potential compounds in fruit and vegetables responsible for CVD prevention are fibers, potassium, and folate. Dietary fiber delays the absorption of carbohydrate and therefore decreases the glycemic responses. Potassium lowers the blood pressure and dietary intake of folate is inversely associated with plasma homocysteine (Bazzano, et al., 2003). Fruit and vegetables included in healthy dietary pattern play an important role to reduce the risk of CVD through their high content of such nutrients.

Whole-grain intake and risk of CVD

A growing body of research shows that returning to whole-grains and other less-processed sources of carbohydrates and cutting back on refined grains improves health. In a meta-analysis of six observational studies reflecting cardiovascular events, consumption of 2.5 servings of whole-grains was associated with a 21% lower risk of incident CVD compared to intake of 0.2 servings per day, after adjustment for cardiovascular risk factors (Mellen, et al., 2008). In a review of 17 articles a range of 20% to 40% reduction in long term risk of CVD was observed in habitual consumers of whole grains compared to those who rarely eat these foods

(Jacobs & Gallaher, 2004). Fiber was not the only constituents responsible for the impact of whole-grains. It was a synergy of the whole-grain constituents including fiber and phytochemicals. Whole-grain foods improve insulin sensitivity as well as reduce both fasting blood glucose and urinary glucose excretion. Whole-grain foods are recommended for prevention of CVD because they contain many cardio-protective compounds including dietary fiber, trace minerals, phytoestrogens, and antioxidants.

Saturated fat and risk of CVD

Although the adverse contribution of saturated fat (SF) to the risk of CVD is high, recent studies have produced conflicting results. Whether SF is a risk factor for CVD is a question with controversial views. Hoenselaar in his recent review concluded that there is discrepancy between scientific literature and dietary advice around SF and CVD risk or mortality (Hoenselaar, 2012). In Hoenselaar's review, dietary recommendations around reducing intake of SF were assessed. The recommendations were established by three advisory committees including the U.S. Department of Agriculture (USDA), The Institute of Medicine (IOM), and the European Food Safety Authority (EFSA). Hoenselaar argues that results and conclusions about SF intake in relation to CVD, from leading advisory committees, do not reflect the available scientific literature. Meta-analyses available on SF and CVD are presented in **Table 2-11**.

Table 2-11 Studies investigating the effect of saturated fat on cardiovascular disease

Study	Study design and population	Results
Siri-Tarino et al., 2010	Mata-analysis of 21 cohort studies	- Saturated fat intake was not associated with an elevated risk of CHD, stroke, or CVD as a composite outcome - The RRs[3] were 1.07 for risk of CHD[4], 0.81 for risk of stroke, and 1.00 for overall CVD risk.
Mozaffarian et al.,2010	Meta-analysis of 8 RCT[1] investigating the effects of increased PUFA[2] consumption, as a replacement for SF, on CHD endpoints	- Increasing PUFA consumption as a replacement for SF reduced the occurrence of CHD events by 19%; each 5% greater energy from PUFA consumption reduced CHD risk by 10%.
Ramsden et al., 2010	Meta-analyses of mixed n-3/n-6 and n-6 specific PUFA RCT	- Mixed n-3/n-6 PUFA RCT significantly reduced the risks of non-fatal MI[5] by 27% and non-fatal MI + CHD death by 22 %. - n-6 specific PUFA diets increased risk of all CHD endpoints, with the increased risk of death from all causes approaching statistical significance
Skeaff & Miller, 2009	Meta-analysis of cohort and RCT studies	Cohort studies: - Intake of SF was not significantly associated with CHD mortality, with a RR of 1.14, for those in the highest (14-18%) compared with the lowest (7-11%) category of SF intake. - Moreover, there was no significant association with CHD death (RR 1.11) per 5% total energy increment in SF intake. RCT: - The RR of fatal CHD was not reduced by either the low-fat diets or the high Polyunsaturated/Saturated (P/S) diets - High P/S diets reduced the risk of total CHD events (RR 0.83), whereas the low fat diets did not affect CHD events.

[1] Randomized Control Trial
[2] Polyunsaturated Fatty Acid
[3] Ralative Risk
[4] Coronary Heart Disease
[5] Myocardial infarction

Meta-analyses included in **Table 2-11** revealed that SF intake alone would not be responsible for the increased risk of CVD. It appears that the ratio of PUFA consumption in a dietary pattern plays the more important role. Intake of SF increases levels of low-density lipoprotein (LDL) cholesterol (Mensink, et al., 2003) which is positively associated with CVD (Prospective Studies Collaboration, 2007). This finding is the reason of directing recommendations towards reduction in the consumption of SF. Current evidence is not strong enough to conclude that all types of SF could possibly increase the risk of CVD. In a recent cohort of Multi-Ethnic Study of Atherosclerosis, MESA among 5909 adults aged 45–84 y, each 5-unit increase in the percentage of energy from dairy SF was associated with 38% lower risk of CVD (de Oliveira Otto et al., 2012). However, the risk increased 48% for each +5% of total energy from meat SF. No associations were observed between plant or butter SF and CVD risk. It appears that there should be more caution in SF recommendation to individuals at risk of CVD. Decreasing the intake of SF while increasing the intake of PUFA might be a better recommendation. Additionally, in the PUFA recommendation the proper ratio of n-3 fatty acid to n-6 needs to be considered.

Salt intake and CVD risk

In some people too much sodium causes blood pressure to rise (Cappuccio, 2007). In a meta-analysis of 14 cohorts (Strazzullo, D'Elia, Kandala, & Cappuccio, 2009), higher salt intake was associated with greater risk of stroke (RRs=1.23). Similar findings were found for total CVD (RRs = 1.17). In other words, a difference of 5 g a day in habitual salt intake was associated with a 23% difference in the rate of stroke and 17% difference in the rate of total CVD. For both stroke and CVD outcomes, separate analyses of the male and female cohorts indicated consistent association and no significant difference between the sexes. Reduction in salt consumption could lower the risk of CVD at the population level. In the United States to quantify the effect of population-wide reduction in dietary salt of up to 3 g per day on the future of CVD, CHD Policy Model (a computer-simulation, state-transition model on future CVD) was built (Bibbins-Domingo et al., 2010). The postulated dietary reduction of 3 g of salt per day was assumed to benefit the entire U.S. population and yielded substantial reductions in morbidity, mortality, and health care costs. The annual number of new cases of CHD decreased by 60,000 to 120,000, stroke by 32,000 to 66,000, and myocardial infarction by 54,000 to 99,000.

Established strategies for reducing salt consumption might have benefit in reducing the development of cardiovascular events in populations.

2.5.3   Beverage intake and risk of CVD

Sugar-Sweetened Beverages and CVD

Data are available from different types of studies and in different populations examining the association between SSBs consumption with CVD risk factors including obesity (Balcells, et al., 2011; Bermudez & Gao, 2010; Malik & Hu, 2012; Mozaffarian, et al., 2011; Schulze, Manson, et al., 2004; Vartanian, et al., 2007) MetS (Chen, Pan, Malik, & Hu, 2012; Denova-Gutiérrez, et al., 2010; Dhingra, et al., 2007), type 2 diabetes (de Koning, et al., 2011; Malik & Hu, 2012; Malik, Popkin, Bray, Després, Willett, et al., 2010; Mattei, et al., 2012; Palmer, et al., 2008; Schulze, Manson, et al., 2004), and CVD events (Chen, et al., 2012) with most of them in favor of positive association. In 2006 the American Heart Association (AHA) recommended minimizing intake of beverages and foods with added sugars (Johnson, et al., 2009). In the Framingham Heart Study, consumption of ≥1 can soft drink per day was associated with increased odds of developing MetS, obesity, increased waist circumference, higher blood pressure, hypertriglyceridemia, and low high-density lipoprotein cholesterol which are all cardiometabolic risk factors (Dhingra, et al., 2007). In the follow up Nurses' Health Study (NHS), the relative risk (RR) of CHD by consuming ≥2 servings/d of sugar sweetened beverages (SSBs) compared with <1 serving/mo was 1.35 after adjustment for Alternate Healthy Eating Index, AHEI (Fung et al., 2009). The association was moderately attenuated (RR= 1.21) after further adjustment for diabetes, BMI, and total energy intake. It appeared that excess calorie intakes and obesity mediated the association. According to the most recent investigation on children aged three to 11 participating in NHANES, 1999 to 2004, each additional SSB serving per day was significantly associated with a 0.01 mg/dL increase in CRP levels, a 0.27 cm increase in waist circumference, and a 0.57 mg/dL decrease in HDL-C levels after adjustment for energy intake and lifestyle characteristics (Kosova, Auinger, & Bremer, 2013). The associations were driven largely by the nine to 11-year-old age group because they were the largest consumer of SSBs in these younger children. The current evidence suggests cardio-metabolic markers might be influenced directly or indirectly by SSBs consumption.

No direct study is available on the association between SSBs consumption in children and CVD risk. Yet, strong associations between childhood obesity and early dyslipidemia, hypertension, and insulin resistance are observed. The exhibition of such cardiometabolic risk in children may persist into adulthood and contribute to development of CVD which needs special attention (Raghuveer, 2010).

Milk and CVD

Milk consumption has been identified as having beneficial impact on blood pressure which is one of the important risk factors for CVD. Observational data have shown the relationship between inadequate dairy product consumption with increased risk of arterial pressure consistently among age/sex groups, and ethnic groups and across the range of blood pressures in adults, infants, older individuals and in pregnant women (Miller, et al., 2000). In a cross-sectional study in Norway after adjusting for covariates such as body mass index, alcohol, coffee consumption, physical activity, cigarette smoking, and vitamin D intake, there was a significant linear decrease in systolic and diastolic blood pressure with increasing dairy calcium intake in both sexes (Jorde & Bønaa, 2000). In randomized controlled trials, a marked heterogeneity was seen in the blood pressure response to increasing calcium intake (Miller, et al., 2000). This effect has been explained through three different hypotheses: 1) the baseline calcium intake: subjects who had low intake of dietary calcium showed a stronger blood pressure effect from increased calcium intake than those whose intake was sufficient; 2) increased calcium intake from food sources which brings other minerals, had greater impact on blood pressure than only calcium supplements intake; 3) groups at high risk of hypertension (African-Americans, salt-sensitive persons and pregnant women) were particularly sensitive to increased mineral intake and therefore experienced stronger beneficial effects of increasing calcium intake (Miller, et al., 2000). The inverse association between stroke and calcium consumption in two prospective studies was stronger for dairy calcium than for nondairy calcium (Abbott et al., 1996; H. Iso et al., 1999). It is therefore possible that milk peptides may also have an additional role to lower blood pressure or to decrease the risk of stroke (Jauhiainen & Korpela, 2007). Thus, milk consumption due to its contribution in lowering cholesterol and blood pressure and by its virtue of low glycemic index would be beneficial to decrease the risk of CVD.

It would seem that by virtue of high concentration of saturated fatty acid in milk, the risk of CVD would be increased. However, benefits of elevated concentration of magnesium and calcium in milk outweigh the adverse effect of saturated fatty acids (Pfeuffer & Schrezenmeir, 2007). Calcium and protein content of milk contribute to lower cholesterol. Additionally, consumption of milk decreases glycemic response compared with mixed meals due to its whey content which is insulinotropic.

Alcoholic Beverages and CVD

Prospective studies have reported reduced rate of death from coronary heart disease among moderate drinkers compared with nondrinkers (Murray et al., 2002; Wollin & Jones, 2001). The Framingham Study revealed a negative association between moderate ethanol consumption and coronary heart disease mortality in males. The same result was obtained for women but it was not significant due to underreporting by women. In fact, the relation between alcohol consumption and risk of CVD was J-shaped (Wollin & Jones, 2001). Binge drinking which is defined as consumption of five or more drinks at one sitting is associated with increased risk of coronary heart disease in both men and women. This finding has been confirmed in a longitudinal study in Canada (Murray, et al., 2002). All types of alcoholic beverages do not provide the same protective effect against CVD. The benefits from alcohol consumption mostly come out of the consumption of red wine (Wollin & Jones, 2001). In a meta-analysis of 26 studies, in 10 studies wine had J-shape association with CVD risks. The same result was evident for beer but the relationship was weaker, probably due to smaller number of studies (Di Castelnuovo, Rotondo, Iacoviello, Donati, & de Gaetano, 2002). In the cohort of Copenhagen City Heart Study after adjusting for confounding factors, weekly consumers of wine had lower risk of stroke. The intake of neither beer nor spirits was associated with stroke risk (Truelsen, Grønbæk, Schnohr, & Boysen, 1998). Alcohol content and phenolic compounds in red wine are responsible for the protective effect against CVD. Phenolic compounds reduce plasma peroxide, lipid peroxide, LDL oxidation, and thrombosis whereas alcohol components decrease platelet aggregation induced by fibrinogen and collagen. All of these effects contribute to reduce the risk of CVD (Wollin & Jones, 2001)(Wollin & Jones, 2001). The J-shaped impact of moderate alcohol consumption on CVD is similar to the effect on the other chronic complications

including MetS and type 2 diabetes. The role of wine is more notable on decreasing the risk of CVD.

Tea and CVD

Since tea is the second most common beverage consumed in the world, even a small effect of tea on individuals would have a large effect in public health. Tea due to its polyphenolic flavonoid content has a protective antioxidant effect against low density lipoprotein (LDL) (Peters, Poole, & Arab, 2001). A meta-analysis of 10 cohort studies and seven case-control studies revealed that tea consumption of three cups per day reduces the incidence rate of myocardial infarction by 11%. This is due to its effect on improving endothelium-dependent vasodilation (Duffy et al., 2001) via the activation of the endothelial nitric oxide synthase (eNOS) in flavonoids. A population-based, prospective cohort study (1995-2005) among 40530 Japanese adults aged 40 to 79 y found that green tea consumption is associated with reduced mortality due to all causes and due to cardiovascular disease (especially in women) but not with reduced mortality due to cancer. Polyphenols in green tea especially epigallocatechin-3-gallate, might have the protective effect (Kuriyama et al., 2006). Acute and chronic protective effects can be obtained by taking 150 mg and 500 mg flavonoids, respectively. This amount is equivalent to 1 to 3.5 cups of brewed tea because each 235 ml of brewed tea contains 172 mg flavonoids (Kris-Etherton & Keen, 2002). Black and green tea (Leung et al., 2001), due to their antioxidant content inhibit LDL oxidation and thus reduce the risk of CVD.

Coffee and CVD

Epidemiological studies have found that coffee reduces the biomarkers of oxidative stress via polyphenols, including chlorogenic acids volatile aroma compounds and heterocyclic compounds, including pyrroles, oxazoles, furans, thiazoles, thiophenes, imidazoles, and pyrazines (Andersen, Jacobs, Carlsen, & Blomhoff, 2006). The 15-year Iowa Women's Health Study cohort comprised 41 836 women aged 55–69 y revealed that death rates from CVD showed a U-shaped association with coffee intake and the least risk was seen for 1-3 cups/day. In a review moderate coffee consumption (< 300ml/day) reduced the risk of CVD about 31% compared to no consumption. Yet, high consumption of boiled coffee (> 600ml/day)

significantly increased the risk (Bonita, Mandarano, Shuta, & Vinson, 2007). Boiled coffee (non-filtered) provided 30% to 40% greater risk for myocardial infarction compared to filtered coffee. Boiled coffee has a higher concentration of coffee oils which are hyper-cholesterolemic. In preparation of boiled coffee higher temperature is used and a longer contact time between the coffee grounds and water is taken which produces coffee oils. Decaffeinated coffee was not associated with CHD in four case–control and three cohort studies (Bonita, et al., 2007). Thus, moderate consumption of filtered caffeinated coffee may have beneficial impact for CVD risk.

2.6   Conclusion

The interrelationship between diet, dyslipidemia, obesity, MetS, CVD, and type 2 diabetes was evaluated in the current review. Uncontrolled obesity specifically abdominal obesity is the main cause for MetS and MetS could result in CVD or type 2 diabetes. Patients with such diseases or risk factors usually have elevated TC/HDL-C ratio. One of the most important approaches to manage and prevent such conditions is having a healthy and balanced diet. In this review the impact of dietary intake on each disease or risk factor was assessed based on available evidence. Since the diseases or risk factors are interrelated there is a possibility of the similar impact of dietary intake on such diseases or risk factors.

Obesity occurs when energy intake (quantity of consumption) chronically exceeds energy expenditure. Increased fiber intake due to its potential characteristics in prolonging the feeling of fullness, increasing in the digestibility of protein and carbohydrate, and modulating insulin response to carbohydrate could contribute to prevent weight gain. The favorable impact of fiber on MetS and diabetes is mostly evident for cereal fibers. Fruit and vegetable fibers might be more influential in the population with low prevalence of obesity.

The direct impact of increased dietary fat intake on weight gain or on the risk of type 2 diabetes is inconclusive. The palatability and therefore overconsumption of high-fat food might be responsible to gain weight. However, in making dietary fat recommendations more caution is needed. Evidence does not show that SF consumption is a risk factor for CVD, although dietary advice is based on the adverse association between SF and CVD risk. Reduction in SF intake might be beneficial when PUFA consumption is increased and the ratio of n-3 fatty acid to n-6

fatty acid is considered. Substituting SF with *Cis* unsaturated fatty acid significantly decreases the TC/HDL-C ratio. In fact, paying more attention to the amount and type of PUFA consumption in the diet is required.

Dietary patterns which are characterised by specific varieties of food groups have special impact on MetS risk. A significant inverse association was observed between MedDiet, as a traditional and healthy dietary pattern, and MetS. Unlike MedDiet, western dietary pattern was associated with increased risk of MetS. Regardless of MedDiet, the negative association between other healthy dietary patterns and risk of MetS is not definitive in all studies and needs further investigation. Dietary patterns containing high intake of fruit and vegetable, legumes, whole-grains, and low fat dairy products or dietary habit with low intake of refined grains, high fat dairy products, sweets and dessert, and red meat are suggested to reduce the risk of MetS.

Beverages account for a substantial share of daily calories in all age groups. Increased consumption of sugar-sweetened beverages has occurred recently due to increased marketing and greater portion sizes. Overconsumption of sugar in the form of beverages is less satiating than solid food and the energy in SSBs is not well compensated in the subsequent meal. Therefore, increased intake of SSBs is positively associated with obesity prevalence. Additionally, even one serving of SSBs/day may increase the risk of MetS and type 2 diabetes through increasing dietary glycemic load and thus insulin resistance. High consumption of SSBs also might increase blood pressure, triglyceride, and decrease HDL-C which are all cardio-metabolic biomarkers. Milk consumption due to its contribution in lowering cholesterol and blood pressure and by virtue of low glycemic index is beneficial to decrease the risk of MetS, type 2 diabetes, and CVD. However, the inverse association between milk and weight gain is still controversial. Other confounders may interact with the association such as physical activity, age, sex, lifestyle indicators, and dietary pattern. Moderate consumption of alcohol is associated with reduced risk of MetS, type 2 diabetes, and CVD. Moderate consumption of alcohol especially wine seems to increase HDL-C levels and insulin sensitivity. The effect of moderate consumption of alcohol on gaining weight due to the impact of alcohol on supressing lipid oxidation is controversial. It appears that the risk of weight gain might increase in overweight or obese individuals who are moderate drinkers but not in normal weight individuals. The polyphenol component of tea and coffee make them a healthy beverage for individuals at risk of type 2 diabetes and CVD.

Caffeine content of coffee may also reduce the risk of diabetes and CVD by inducing weight loss through the effect of thermogenesis and stimulation of lipid oxidation. Thus, the specific effect of any types of beverages on the risk of chronic diseases or risk factors needs to be considered due to the recent increment in beverage consumption.

The lack of the dietary assessment for chronic diseases is observed in evaluating different dietary patterns or traditional dietary patterns among different ethnicities. Research on overall dietary patterns is required to improve the understanding of the numerous modifiable determinants of disease risk that may guide to develop preventative strategies. Specifically, healthy dietary pattern needs to be investigated rather than unhealthy dietary pattern. Among the food groups, enough evidence is not available around any types of meat consumption such as red meat versus white meat. Furthermore, dietary fat consumption especially saturated fat needs to be re-evaluated to make a more efficient recommendation for individuals at risk of diseases. The more research available on dietary intake related to chronic diseases and some risk factors the more appropriate recommendations can be made for having a healthy diet.

CHAPTER 3

GENERAL METHODOLOGY

This chapter includes general methodological information related to all chapters involving studies in this thesis. The present study was conducted using data from Canadian Health Measures Survey (CHMS) cycles 1 (2007-2009).

3.1   Canadian Health Measures Survey (CHMS)

The Canadian Health Measures Survey or CHMS includes two main parts: the first part is an in-home general health interview and the second part is a visit to a mobile examination center (MEC) for direct health measures which was conducted one day to six weeks after the first part.

3.1.1   Data Collection

The well-trained individuals working for CHMS are divided into three sub-teams: field team, MEC team, and head office staff. Each of these three sub-teams is responsible for specific portions of the survey. The CHMS field team consists of Statistics Canada household interviewers and an interviewer manager. The CHMS MEC team is a group of health professionals responsible for physical measurements and a site manager who monitored the operation of the MEC. The CHMS staff at the head office monitor data collection response rates and data quality and visited sites periodically and controlled staff performance (Giroux, 2007).

The household questionnaire content was obtained from external expert and questions taken from other surveys such as Canadian Community Health Survey (CCHS). Each MEC consists of two trailers, the administrative trailer and the clinic trailer. Computer assisted interviewing (CAI) is used to capture the responses for the household, physical measurements and laboratory components of the CHMS.

3.1.2   Participants

A multistage sampling strategy was used in the CHMS. Labor Force Survey (LFS) sampling frame was used and 257 collection sites were created for MEC. The clusters are small

geographic units that contain approximately 200 dwellings. A collection site is a geographic area with a population of at least 10,000 and a maximum respondent travel distance of 100 kilometers (50 kilometers in urban areas and 100 kilometers in rural areas). The collection sites were classified into five regions based on Statistics Canada's standard regional boundaries (SRB). The regions were British Columbia, the Prairies (Alberta, Manitoba and Saskatchewan), Ontario, Quebec and the Atlantic provinces (Newfoundland and Labrador, Prince Edward Island, Nova Scotia and New Brunswick). However, due to the logistical and cost constraints associated with the use of MEC the number of collection sites for cycle 1 was restricted to 15. (Giroux, 2007; Statistics Canada, 2007).

In cycle 1 the health indicators of approximately 5600 individuals aged 6 to 79 y were measured with roughly 500 females and 500 males in each of the following age groups strata: 6 to 11 y, 12 to 19 y, 20 to 39 y, 40 to 59 y, and 60 to 79 y. Dwellings having at least one member from the specified age group were chosen and each dwelling was selected by simple random sampling. When the goal was 6-11 y age group two people were selected in the dwelling. However, for 12 to 79 y age groups one person in each dwelling was selected. The target population was indiviuals living at home and residing in the ten provinces, three territories. Individuals who were living in aboriginal settlements in the provinces, institutional residents, and full-time members of the Canadian Forces were excluded. In addition, persons living in some remote areas or areas with a low population density were excluded. Finally, CHMS cycle 1 covered 96.3% of Canadian population aged 6 to 79 y from New Brunswick, Quebec, Ontario, Alberta and British Columbia (Giroux, 2007; Statistics Canada, 2007).

3.1.3 Data Processing

Processing of the household interview data and clinical measurement began with validating data at the record level and then at the individual variable level, followed by detailed top-down editing. The processing of laboratory results began with significant file manipulation and then the data were validated while collecting the data and at the individual variable level, and then several new variables were derived.

Pre-coded answers were provided for all suitable variables. The long answers were coded into one of the existing listed categories if the long answers duplicated a listed category. If not, the response was coded as "other". Most editing of the data was conducted at the time of the interview by the computer-assisted interviewing (CAI) application. In addition, data underwent a series of adjustments at head-office. As a result, the final data were completed and contained reserve codes for responses of "less than limit of detection", "don't know", "refused", "not stated" and "not applicable."

Three types of derived variables were created: collapsing data from one variable into groups; combining the data from one or more survey questions for a single respondent; or combining data from more than one respondent. Three other types of derived variables in the cycle 1 CHMS data file include converting responses given in various time unit (days, weeks, months or a year) into one type of units such as yearly; calculation of body mass index (BMI); and the creation of the Health Utility Index which is based on the responses from a series of questions (Giroux, 2007; Statistics Canada, 2007).

3.1.4 Weighting

Weight adjustment is the estimation that each participant in a probability of the sample represents a number of people that are outside the sample. In CHMS the selection weights for collection sites are multiplied by the selection weights for dwellings (households), adjusted for non-response. Following the conversion of household weights into person weights, the latter are adjusted for non-response at the interview stage and MEC stage, and with several other adjustments, this weight becomes the final person weight. The variable which is obtained after calibration is called WGH_FULL for the full sample or WGT_FAST for the fasted sub-sample dataset (Giroux, 2007; Statistics Canada, 2007).

3.1.5 Data Quality

The response rate before adjustment for 1 or 2 persons in each household was 84.9%. Quality assurance measures before data collection and quality control during and after data collection were applied to control the quality of the data.

Non-sampling error in CHMS was classified into "partial non-response" and "total non-response". Partial non-response, which was related to those respondents who did not answer to one or more questions, was predominantly small in CHMS cycle 1. For minimizing the effect of total non-response error the weights of persons who responded were corrected to compensate for those who did not respond.

Sampling error measurement in CHMS is based on the standard deviation of the estimates drawn from the survey results. Standard deviation is divided by the estimates obtained and the resulting measurement is expressed as a percentage of the estimate and is called the coefficient of variation (CV). If the CV was between 0% and 16.6% the estimates were accepted, if it was between 16.6% and 33.3% the estimates were marginally accepted, and if it was more than 33.3% the estimates were not accepted (Giroux, 2007; Statistics Canada, 2007).

3.1.6 CHMS measurements

CHMS gathered information related to nutrition, smoking habits, alcohol use, medical history, current health status, sexual behavior, lifestyle and physical activity, the environment and housing characteristics, as well as demographic and socioeconomic variables. In addition, the survey has collected blood and urine samples to test for chronic and infectious diseases, nutrition and environment markers. However, the focus is on the variables related to health and nutrition.

3.1.7 Validation of questionnaire responses

Different activities were done to validate the household questionnaire including 1) the interviewers' remarks were reviewed and adjusted to the data, 2) questions with the high response rates to the "other", "specify", "don't know", and "refusal" were monitored according to expected rates from other Statistics Canada health surveys. These data were also monitored between CHMS collection sites to identify site-specific problems, 3) validation was also performed on the questions which were similar to the questions from the other Statistics Canada health surveys, namely cycles 3.1 and 4.1 of the Canadian Community Health Survey (Giroux, 2007; Statistics Canada, 2007).

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3.2 Analytical Approaches

In this section the preparation of data is explained. Further, the type of variables used in this research will be examined. Advanced statistical analysis on how to get the best fitted model or how to generalize the data to the Canadian population will be explained.

3.2.1 Data Cleaning and Processing

Data manipulation, cleaning, grouping and creating variables of interest were done to obtain the final dataset. The interest variables were classified into two groups. The first group is the "Existing Variables" which are present as raw or derived variable in CHMS data. The variables of the second group were created called "Newly Created Derived Variables (NCDV)" which were created using the raw variables available in the CHMS dataset (**Table 3-1**).

Table 3-1 CHMS variables applied in the current study

Existing Variables
-Age (years)
-Sex
-Socio-economic status (SES) including *education* and *income*
-Physical activity (self-reported)
-Food and beverage consumption including *dietary fat, grains, fruit and vegetables, meat and fish, milk and dairy product, potato, sugar-sweetened beverages, diet soft drink, fruit and vegetable juice, and alcohol intake*

Newly Created Derived Variables (NCDV)
-Ethnicity created using all ethnicities available in CHMS. The final variable included *white (White category included white Canadian, American and European) versus non-white (Non-White category included the rest of the ethnicities)* due to small sample size in non-white category
-Smoking habits derived from *type of smoker and number of years since stopping smoking completely*
-Cardiovascular age created using a *specific formula*
Outcome variables:
-Adult and adolescent central obesity (abdominal obesity) derived from *waist circumference*
-Elevated TC/HDL-C ratio derived from *total cholesterol and HDL-C values*
- Metabolic syndrome derived from *waist circumference or percentile cut-off, triglycerides, High Density Lipoprotein (HDL), blood pressure, fasting plasma glucose or previously diagnosed type 2 diabetes*
-Diabetes risk derived from *Glycated hemoglobin (HbA1$_c$)*
-10-year risk of cardiovascular disease using Framingham Risk Score (FRS) derived from *presence or absence of diabetes, sex, smoker or non-smoker, age, systolic blood pressure, total blood cholesterol (mmol/l) or Low Density Lipoprotein Cholesterol (LDL-C), and High Density Lipoprotein Cholesterol (HDL-C)*
-Cardiometabolic Risk derived from *10-year risk of CVD and MetS*
-Modified 10-year risk of CVD derived from the *traditional FRS and considering the history of premature CVD event in first degree relatives in individuals 30-59 y without diabetes as well as considering "cardiovascular age"*

3.2.2   Existing Variables

The variables below were used in all studies of the corresponding thesis.

<u>Age and sex</u>

All age/sex groups, 6-79 y, were of interest in order to investigate the dietary intake of Canadians. However, the age categories used to evaluate the chronic diseases or risk factors were 12 – 79 y: 12 – 19 y, 20 – 39 y, 40 – 59 y, and 60-79 y.

<u>Socio-Economic Status</u>

Socio-economic status including education for 15 – 79 y subjects and household income for 12 – 79 y subjects were investigated.

There are 2 derived variables for education in CHMS data. One variable has 4 levels and the other has 10 levels. These variables indicate the highest level of education acquired by any member of the household. **Table 3-2** indicates the categories of each variable. The 10-level education variable which has been used and tested in Steele's article (2007) for CCHS data may determine a better estimate than 4-level education (Steele, Dewa, Lin, & Lee, 2007). However, 4-level education variable was considered in the studies of this thesis to avoid small sample sizes for some categories.

Table 3-2 Levels of education investigated in CHMS

4-level education variable	10-level education variable
- Less than secondary school graduation - Secondary school graduation, no post-secondary education - Some post-secondary education - Post-secondary degree/diploma	- Grade 8 or lower - Grade 9-10 - Grade 11-13 - Secondary school graduate, no post-secondary education - Some post-secondary education - Trade certificate or diploma from a vocational school or apprenticeship training - Non-university certificate or diploma from a community college, CEGEP, school of nursing, etc. - University certificate below bachelor's level - Bachelor's degree - University degree or certificate above bachelor's degree

There are 3 categorical derived variables for income in CHMS data. These variables classify the total household income into two, four, and five categories derived from the total household income and the number of people living in the household introduced in the National Population Health Survey (NPHS) cycle 1. **Table 3-3** shows the categories of each variable. Reducing multiple categories to only two categories, results in a loss of information (Humphries & Van Doorslaer, 2000). Therefore, four-category variable was used because it indicated better understanding in income and gave better frequency in cross-tabulation.

Table 3-3 Categories of income investigated in CHMS

Two-category income	Four-category income	Five-category income
- Low income - Middle or high income	- Lowest income grouping - Lower middle income grouping - Upper middle income grouping - Highest income grouping	- Lowest income grouping - Lower middle income grouping - Middle income grouping - Upper middle income grouping - Highest income grouping

Physical Activity

There are two measurements for physical activity in CHMS: self-reported physical activity which was collected through household questionnaire and directly measured physical activity by Actical Accelerometer. The Actical is an omnidirectional accelerometer which is sensitive to movement in all directions and is a valid and reliable measure of physical activity in children and adolescents (Puyau, Adolph, Vohra, Zakeri, & Butte, 2004).

In the NHANES 2003-2004 physical activity level for both self-report and accelerometer were qualitatively consistent; however, the count and duration of physical activity outcomes declines dramatically with accelerometer especially during adolescence. They actually over-reported their physical activity through self-report measurement (Troiano et al., 2008). Colley (2011), who has worked with CHMS data, found inconsistency between self-report and accelerometer-measured physical activity among Canadians (Colley et al., 2011a, 2011b). Colley

suggested the relationships between physical activity and health needs to be re-examined by using directly measured physical activity in adults (Colley, et al., 2011a). In addition, among children and adolescents inconsistency between self-reported and directly measured physical activity has been identified (Colley, et al., 2011b). However, in this thesis no differences were observed in using self-reported or measured physical activity in the models. Additionally, self-reported physical activity was more practical due to creation of three categories of leisure time physical activity (inactive, moderately active, and active). Physical activity was measured in CHMS using a questionnaire which calculated the total daily leisure time energy expenditure (EE) values (kcal/kg/day). Respondents were subsequently categorized into "active" (EE $\geq$ 3), "moderate" ($1.5 \leq EE < 3$), or "inactive" ($0 \leq EE < 1.5$) physical activity.

Dietary intake

The food group consumption was evaluated (**Table 3-4**) to observe the dietary intake of Canadians as well as the association between chronic diseases and risk factors with dietary intake. The respondents were asked about the frequency of meat and fish, milk and dairy product, grains, fruit and vegetables, dietary fat consumption, and beverages. The intake was reported as the daily frequency of consumption and for the regression associations the intake was categorized into quartiles. The original questions can be found elsewhere (Statistics Canada, 2013b). In CHMS grains and fruit and vegetables are in the same category. Further, regular soft drinks, sport drinks, fruit drinks, diet soft drinks, and fruit/vegetable juice are all in a group of "water and soft drinks". Alcohol consumption variables were collected differently compared to other beverages. They were chosen based on the Freiberg's (2004) results on the NHANES III data who examined the relationship between alcohol consumption and the prevalence of metabolic syndrome (Freiberg, et al., 2004). "Ever drinker" versus "never drinker" was the most appropriate variable that could be derived from data. This variable was used as a covariate in logistic models. If categories of grains and meat became significant in the model they were broken down to cereal and others for grain group and to nuts, legumes, fish, and others for meat group. The socio-demographic characteristics include age, sex, physical activity, education, income, and ethnicity which are explained in this chapter.

Table 3-4 Dietary intake classification and definitions by CHMS

Intake Category	Types of Food
Beverage Categories	
Sugar-Sweetened Beverages	The number of times per day the respondent drinks regular soft drinks, sport drinks such as Gatorade or Powerade, or fruit flavoured drinks such as Sunny Delight, fruit punch, or Kool-Aid
Fruit & Vegetable Juice	The number of times per day the respondent drinks fruit juices such as apple, orange, or grape fruit juice whether or not they are made from concentrate. Fruit and vegetable juice is 100% pure juice.
Alcoholic Drinks	Ever drinkers versus never drinkers derived from two variables in CHMS dataset: frequency of drinking alcohol and type of drinker
Non-Caloric Beverages	The number of times per day the respondent drinks diet soft drinks
Food Group Categories	
Meat & Fish	The number of times per day the respondent eats red meat, organs, hotdogs, sausage or bacon, seafood, eggs, beans, nuts
Grains	The number of times per day the respondent eats hot/cold cereal, white bread, brown bread, any kind of rice, any kind of pasta
Fruit & Vegetable	The number of times per day the respondent eats fruit and vegetables
Potato	The number of times per day the respondent eats french fries, hash brown, boiled, backed, and mashed potato excluding sweet potato
Milk & Dairy Product	The number of times per day the respondent has milk or enriched milk substitutes or uses them on cereal including 3.25%, 2%, 1%, 0.5%, skim or non-fat (including powder milk), flavoured milk beverages (such as chocolate milk and flavoured milks such as Oh Henry), rice (enriched), soya (enriched), cottage cheese, yogurt, and ice cream.
Dietary Fat	The number of times per day the respondent eats regular-fat salad dressing or mayonnaise, regular-fat potato chips, tortilla chips, and corn chips

3.2.3 Newly Created Derived Variables (NCDV)

NCDV are the following variables which were created using raw variables existing in CHMS data.

Ethnicity

After categorizing the subjects into different ethnicities available in the CHMS data, the sample size in each ethnicity other than white was very small. Thus, only 2 categories of ethnicities were created: white versus non-white. White category consisted of American, Canadian and European and the rest were considered non-white. This variable was used in all of the analyses.

Smoking habits

Smoking habit was mostly used as a confounder in evaluating the association between dietary intake and chronic diseases or risk factors. Additionally, in order to create the 10-year risk of CVD recognizing smokers was needed. To achieve the final binomial variable of smoker versus non-smoker, two variables were used (**Table 3-5**). In the first variable "current daily smoker" and "occasional smoker" were considered as smokers. The second variable was needed because the abstinence of smoking for less than a year does not exclude the individual of being smoker (World Health Organization, 2007b). Therefore, the individuals who stopped smoking in less than a year were also considered as smokers.

Table 3-5 Smoking habits estimates in CHMS

Variable	Definition
Type of smoker	*This variable indicates the type of smoker the respondent is, based on his/her smoking habits including* Current Daily smoker Occasional smoker (former daily smoker) Occasional smoker (never a daily smoker or has smoked less than 100 cigarettes lifetime) Non-smoker (former daily smoker) Non-smoker (former occasional smoker, at least 100 cigarettes in lifetime) Never smoked (at least 100 cigarettes)
Number of years since stopping smoking completely	*This variable indicates the approximate number of years since former daily smokers completely quit smoking.*

<u>Identifying abdominally obese adults and adolescents</u>

Since abdominal obesity is the main risk factor for chronic diseases, it was utilized in all the studies of the thesis including metabolic syndrome, diabetes, TC/HDL-C ratio, and CVD. Abdominal obesity was derived using waist circumferences. As the International Diabetes Federation (IDF) recommended, different cut-offs were implemented for different ethnicities to define abdominal obesity (**Table 3-6**). In adolescents 12-19 y, the age and sex-specific cut-offs for waist circumference generated by Jolliffe and Janssen, 2007 was used (**Table 2-3** & **Table 2-4**)

Table 3-6 Waist circumference threshold for abdominal obesity (Alberti, et al., 2009)

Population	Men	women
	Centimeter	**Centimeter**
White (Canadian, American, and European)	≥ 102	≥ 88
Middle east, Mediterranean, Sub-Saharan African	≥ 94	≥ 80
Asian	≥ 90	≥ 80
Chinese	≥ 85	≥ 80
Japanese	≥ 85	≥ 90

3.2.4   Advanced statistical analysis

Five main outcome variables including abdominal obesity, elevated TC/HDL-C ratio, MetS, diabetes, and cardiovascular disease were considered to investigate the association with dietary intake controlling for potential covariates. All of the outcome variables were assessed as binary variables (having a disease=1 versus not having a disease=0) in logistic regression models. To build the best model for each disease or risk factor six stages were implemented: 1- univariate analysis (Dhand, 2010); 2- test of multicollinearity (Williams, 2014); 3- multivariate analysis and testing nested model (Briun, 2007; Tardanico, 2008); 4- test for interaction and confounders (Buis, 2010; Knol, van der Tweel, Grobbee, Numans, & Geerlings, 2007; UCLA Statistical Consulting Group, 2007a); 5- obtaining the best model; and 6- specification error (UCLA Statistical Consulting Group, 2007b).

Building the best model using logistic regression and STATA

There are stages for building model by regression which have to be taken into account. Jumping straight to multivariate analysis without conducting the previous stages, can result in erroneous outputs. Moreover, there are stages after multivariate testing which are important in order to obtain the best model.

Stage 1: Univariate Analysis

Selection of the economical subset of the explanatory variables that account for maximum variability in the outcome is an objective of multivariate analysis (University of Sydney, 2010). After selecting relevant variables, univariate analysis was conducted to shortlist the variables. Statistically non-significant variables were excluded. A p-value of 0.2 was accepted as significant. More traditional values such as 0.05 can fail in identifying important variables (Bendel & Afifi, 1977; Mickey & Greenland, 1989). Statistically significant variables and confounders remained in the model regardless of being significant or not significant.

Stage 2: Test of Multicollinearity

Multicollinearity happens when two explanatory variables are highly correlated with each other. The greater the multicollinearity is, the greater the standard errors and the wider confidence intervals are obtained. In addition, the t-statistics become very small (Williams, 2014).

The most widely-used diagnostic for multicollinearity is Variance inflation factor (Vif). The Vif may be calculated for each predictor by doing a linear regression of that predictor on all the other predictors, and then obtaining the R^2 from that regression. The Vif is just $1/(1-R^2)$. "Vif indicates how much the variance of a coefficient is "inflated" because of the multicollinearity". $1-R^2$ is referred to as *Tolerance of the variables*. A tolerance close to 1 indicates little multicollinearity, whereas a value close to zero shows high multicollinearity. A rule of thumb is that Vifs of 10 or higher (tolerance of 0.1 or less) is a threat for multicollinearity

(Williams, 2014). However, according to Paul Allison, professor of sociology at the University of Pennsylvania there is still concern when Vif is over 2.5 (Allison, 2012).

In order to run multicollinearity in STATA "regress" command should be used first. After "regress" command "vif" and "vce, corr" commands are implemented to obtain tables showing vif, tolerance, and correlations (Williams, 2014). Furthermore, a program called "Collin" can be used to detect the multicollinearity in STATA (UCLA Statistical Consulting Group, 2007b). All these commands except vce, corr are useful when the data are not weighted.

Stage 3: Multivariate Analysis and testing nested model

After finding non-significant variables by univariate analysis and predictors with multicollinearity, the remaining variables were entered into a logistic model.

The likelihood ratio (lr) test, Wald test, and Langrange multiplier (score test) are used to assess the difference between nested models. The nested model is a model in which there are some limitations in terms of the presence of predictors. If the sample is un-weighted lr test is used to assess nested models; if not, the Wald test should be used (Tardanico, 2008; UCLA Stata Frequently Asked Questions, 2007). The advantage of the Wald test is that it requires only one model be estimated whereas in lr test at least 2 models are evaluated simultaneously (UCLA Stata Frequently Asked Questions, 2007). The Wald test considers that the specific predictors are simultaneously equal to zero. If they are, this suggests that removing those variables from the model will not reduce the fit of that model.

The first step in doing the Wald test is to run the full model in STATA using "SVY: logit outcome variable variable1 …… variable n" command. In The second step the Wald test can be run using "test" command. In order to test whether the coefficient of specific variables are simultaneously equal to zero those variables should be added after the "test" command. To test the coefficient of one variable it could be added after "test" command alone. The first output is the null hypothesis. The chi-squared value is generated by the Wald test as well as the P-value associated with the chi-squared. Based on a p-value < 0.05 the null hypothesis is rejected indicating that the coefficients for the specific variables are not simultaneously equal to zero.

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Therefore, the presence of those variables improves the model and they need to remain in the model.

Stage 4: Test for interaction and confounders

"Interaction occurs when the effect of variable A on an outcome is different across strata of variable B. In fact interaction involves exploring differences in differences" (UCLA Statistical Consulting Group, 2007a). Interpreting interaction is possible through two ways: interpreting interaction on an additive scale and interaction on a multiplicative scale (Buis, 2010; Knol, et al., 2007). If the combined effect of A and B is larger (or smaller) than the sum of the individual effect of A and B, the interaction is departure from additivity. The estimate of how much increase or decrease is expected for the dependent variable for a unit change in an independent variable is called the marginal effect on an additive scale. On the other hand, the ratio or the marginal effect by which the dependent variable changes for a unit change in independent variable is presented on a multiplicative scale. In logistic regression the regression coefficient on the interaction indicates departure from multiplicativity. Since it is more common to look at the multiplicative interaction in studies, in the current study the focus is on this type of interaction in logistic models. Also, the interactions will be interpreted in terms of odds ratio because odds ratio compared to probability remains the same regardless of where the covariate constant is held.

Categorical by categorical variable interaction (CRM Portal, 2006; UCLA Statistical Consulting Group, 2007a)

This procedure can be performed for binary variables as well as categorical variables with more than 2 categories using STATA. This form of interaction was used for the thesis as all significant interacted variables were categorical. In order to interpret the interaction on a multiplicative scale the conclusion can be made directly by looking at the p-value, odds ratio estimate, and regression coefficient estimate obtained from the combined effect of A and B. However, the corresponding values are the odds ratio or coefficient for B when A is zero or corresponds to any category of A which has been considered as a reference group. In order to obtain the values of odds ratio for variable B when the variable A equals one or for the other

categories of A, "margin" command in STATA should be used: margins, over (A B) expression (exp (xb())) noatlegend. The option "expression (exp (xb()))" insures that we are looking at the results in the odds ratio metric. Then the odds ratio for each level of B can be computed using the margins in the table. When the interaction is significant in the logistic model, the table below is generated by using "margin" command after "logit" command in STATA.

Example of a table generated by STATA using margins command adopted from (UCLA Statistical Consulting Group, 2007a)

margins, over (A B) expression (exp (xb())) noatlegend
Predictive margins   Number of obs: 200
Mode1 VCE: OIM
Expression: exp (xb())
Over: A B

	Margin	Delta-method Std. Err.	Z	P> $\mid$ Z $\mid$	(95% Conf. Interval)	
A B						
0 0	0.13	0.07	1.77	0.076	-0.01	0.27
0 1	1.42	0.51	2.76	0.006	0.41	2.43
1 0	2.60	1.13	2.3	0.022	0.38	4.83
1 1	3.67	1.31	2.8	0.005	1.10	6.24

Odds ratio at A=0: 1.42/0.13 = 10.92

Odds ratio at A=1: 3.67/2.60 = 1.41

Interpretation: when A=0 the odds of the outcome being one are 10.92 times greater for B=1 then for B=0 (The value of 10.92 can be read directly from the logistic table generated by STATA). For A=1 the ratio of the two odds, which can be generated using margin table, is only 1.41.

In order to plot the graph showing the interaction between two variables the predicted probability for each subject was created using "predict …" command after logit or logistic in STATA. In the … space any name for the created "predict" variable can be used. After creating the variable, SPSS was used to plot the graph using the error bar option. The graphs are observed in the **Appendix 4**.

In the absence of interaction, the effect of confounders is tested. Confounding variables (covariates) are the third variables which distort the association between dependent variable and

variables of interest significantly (Frank, 2000). Confounders are associated with both the putative risk factor and the outcome. Some pre-set percentage of change in the estimate between the crude and adjusted coefficient is defined to identify the confounders. Such percentages of change are identified by the researchers and they need to be problem-dependent. Epidemiologists such as Mickey and Greenland have generally agreed that change-in-estimate (CE) criterion indicates important confounding (Mickey & Greenland, 1989; Tong & Lu, 2001). In this method in case of presenting a confounder, the crude odds ratio differs from the adjusted odds ratio by 10% or more.

Stage 5. Obtaining the best model

After recognizing the significant variables, variables with interaction, and confounding variables, the best model with the specific variables can be built.

Stage 6: Specification Error

The STATA command "linktest" can be used to detect a specification error, and it is issued after the "logit" or "logistic" command. The idea behind "linktest" is that if the model is properly built, any additional predictors that are statistically significant cannot be found except by chance. After the regression command, "linktest" indicates the linear predicted value (_hat) and linear predicted value squared (_hatsq) as the predictors to rebuild the model. The variable _hat should be a statistically significant predictor, since it is the predicted value from the model and it indicates that meaningful predictors have been chosen. Whereas, if the model is properly specified, variable _hatsq shouldn't have much predictive power except by chance and it shouldn't be statistically significant. Therefore, if _hatsq is significant, then the "linktest" is significant. This usually means that either we have omitted relevant variable(s) or our model is not correctly specified. To remove this error the first thing to do to is to see if we have included all of the relevant variables. Secondly, we might overlook the possible interactions among some of the predictor variables (UCLA Statistical Consulting Group, 2007b). Therefore, we need to re-test the model by adding specific variables.

3.2.5   Generalizability & Degrees of freedom

To be able to generalize the results for Canadians at national level specific measures as recommended by Statistics Canada was taken into account (weighting, bootstrapping). Weighting was explained in section 3.1.4. For weighting data in STATA svy[pw=wgt_full] command was implemented. For calculating a precise variance or coefficient of variation, in the absence of simple mathematical formula re-sampling methods should be used. In resampling the generated inferences are based upon repeated sampling within the same sample (Yu, 2003). There are four methods for resampling including randomization exact test, Cross-validation, Jackknife, and Bootstrap.The bootstrap method is the one recommended for analysing CHMS data (Statistics Canada, 2007). The bootstrap creates a large number of datasets with replacement from the original sample and computes the estimate on each of these datasets. The purpose of bootstrapping is deriving robust estimates of standard errors and confidence intervals of a population parameter like a mean, median, proportion, odds ratio, correlation coefficient or regression coefficient. In STATA software, the following command was used to conduct bootstrapping: svyset[pw=wgt_full], bsrweight (bsw1-bsw500) vce(bootstrap) dof(11) mse.

Another factor that was necessary to consider in data analyses using CHMS data was the degrees of freedom due to its specific sampling design. Degrees-of-freedom is a generic term to reflect the amount of information used to estimate variances and covariances (Statistics Canada, 2007). In a multistage cluster sampling design such as in CHMS, the degrees of freedom are based on the number of primary sampling units (PSUs) and strata, rather than the sample size. An approximation often used for calculating degrees of freedom is (# of PSUs - # of strata). For the CHMS, the collection sites (15) are the PSUs. For the purpose of variance estimation, the one Atlantic region PSUs is collapsed in with the 4 Quebec region PSUs, thus leaving 4 strata and degrees of freedom equal to $(15 - 4) = 11$. Therefore, for estimating confidence intervals or p-values, or testing hypotheses, the computation will be done under the assumption that the z-statistic has a t-distribution with 11 degrees of freedom.

Various statistical software was used in the study. IBM SPSS statistics for windows, version 20 (Armonk, NY) was used for data manipulation, summarizing, categorizing, and creating new variables. Stat/Transfer data conversion software used to convert the format of data

across statistical packages. STATA/SE 11, StataCorp was used for all statistical analyses. Alpha was set at the level of 0.05 for almost all analyses.

CHAPTER 4

STUDY 1. Dietary intake of Canadian children and adults and the association between dietary intake and cardiovascular risk factors and diabetes

Assessing dietary intake of Canadians aged 6 to 79 y was the first study in the series of four studies in this thesis in order to have a general overview of intake among Canadian. The second main objective of the entire thesis was to evaluate the association between metabolic syndrome and the associated risk factors or diseases with dietary intake. In the first study three health outcomes were investigated abdominal obesity, elevated TC/HDL-C ratio, and diabetes. All three health outcomes are interrelated as abdominal obesity is the main cause for the two other health outcomes and diabetics usually suffer from adverse lipid profile.

Two abstracts from the first study were published in the Applied Physiology, Nutrition, and Metabolism.

Setayeshgar, S., Whiting, S. J., Vatanparast, H. (2013). Is dietary intake associated with abdominal obesity in Canadian adults and adolescents? Applied Physiology, Nutrition, and Metabolism, 38(4), 472.

Setayeshgar, S., Whiting, S. J., Vatanparast, H. (2012). Abdominal obesity and high consumption of sugar-sweetened beverages are associated with the risk of elevated atherogenic plasma lipids among Canadian women. Applied Physiology, Nutrition, and Metabolism, 37(3), 589.

4.1 Introduction

Identifying food intake patterns at a population level provides valuable insight toward nutritional health status. One important reason is to evaluate dietary predictors of the prevalent chronic diseases. According to the 2004 Canadian Community Health Survey (CCHS) data, most of Canadians in all age groups did not meet the minimum recommendation of 5 servings for fruit and vegetables per day (Garriguet, 2007). Despite a decrease in dietary fat intake from calories from 40% to 31% between 1970-1972 and 2004, there were 28% of Canadians aged 31 to 50 y who obtained more than 35% of their total calories from fat in 2004. The individuals in this age group are more vulnerable to CVD due to having other risk factors such as high blood pressure.

The first objective of this chapter is to identify the dietary intake by different socio-demographic characteristics. Characterizing the change in dietary pattern at a population level indicates how health recommendations and policies impact dietary intake. Due to limited data on dietary intake in CHMS, the analysis was directed to the food intake by different socio-demographic characteristics.

The second objective of this chapter is to evaluate the association between dietary intake and cardiovascular risk factors (abdominal obesity, elevated TC/HDL-C ratio) and diabetes controlling for socio-demographic characteristics. Results indicated an association between dietary intake and all these health outcomes. Fiber intake is inversely associated with risk of abdominal obesity or diabetes (Carter, Gray, Troughton, Khunti, & Davies, 2010; He, et al., 2004; Schulze, Liu, et al., 2004; Weickert, et al., 2006). Replacement of saturated fatty acid (SF) with *cis* unsaturated fatty acid could decrease the level of TC/HDL-C ratio (Mensink, et al., 2003). A critical examination of this issue requires a representative sample of Canadian population since dietary pattern is different in various population groups. In addition, the prevalence of abdominal obesity, elevated TC/HDL-C ratio, and diabetes by different socio-demographic characteristics have been determined in the current study. In addition, it was hypothesized that unhealthy dietary intake is in association with increased risk of all or any of the three health outcomes.

4.2   Materials and Methods

4.2.1 Study population

Data from the Canadian Health Measures Survey (CHMS), cycle 1, 2007 - 2009, conducted by Statistics Canada in partnership with Health Canada and the Public Health Agency of Canada, was used. Statistics Canada is a member of the industry portfolio producing statistics that help Canadians better understand their country, its population, resources, economy, society and culture. In addition to conducting a census every five years, it conducts about 350 active surveys on virtually all aspects of Canadian life. Among them the CHMS is a nationally representative survey collecting health indicators from a sample of approximately 5,500 Canadians aged 6 to 79 y (representative of 96.3% Canadians through a multi-stage sampling strategy). The survey consists of two stages: the first is self-reported data collection through interviews, and the second consists of taking direct physical measurements at Mobile Examination Centers (MEC). Individuals living on reserves or in other aboriginal settlements in the provinces, those remote areas, institutional residents, and full-time members of the Canadian Forces were excluded from the survey. The sampling weights, provided by Statistics Canada, were calculated by multiplying the selection weights for collection sites and for dwellings (obtained from 2006 census), adjusted for non-response (Giroux, 2007). The final individual population weight was obtained after converting the household weights followed by adjustment for non-response at the interview stage and the MEC stage. For the purpose of the study, the dietary intake of all individuals aged 6 to 79 y were evaluated. Individuals under the age of 12 and pregnant women were excluded for the study of abdominal obesity. Additionally, individuals under the age of 20 and pregnant women were excluded for the study of elevated TC/HDL-C ratio and diabetes. The total number of subjects studied for dietary intake, abdominal obesity, elevated TC/HDL-C, and diabetes were 5604, 4445, 3430, and 3390, respectively.

4.2.1   Classification for dietary intake

Food and beverages categories used in the study are shown in **Table 3-4**. The respondents were asked about the frequency of meat and fish, milk and dairy product, grains, fruit and vegetable, dietary fat, and beverage consumption. The intake is reported as the daily

frequency of consumption (times/day) and for the regression associations the intake categorized into quartiles.

4.2.2 Identifying abdominally obese adults and adolescents

To identify abdominally obese adults cut-points suggested by IDF were implemented (**Table 3-6**) and for adolescents the cut points suggested by Jolliffe and Janssen (2007) were used (**Table 2-3** & **Table 2-**4). More explanation can be found in the "General Methodology" Chapter.

4.2.3 Identifying individuals with elevated TC/HDL-C ratio

In order to obtain elevated TC/HDL-C ratio, HDL-C variable and total cholesterol variable were needed which were both available in the full sample of the CHMS. Therefore, the TC was divided by HDL. A ratio of 5 or higher was coded as "1" and below 5 coded as "0".

4.2.4 Identifying Diabetics

In the CHMS household questionnaire, the respondents were asked about the presence and the type of diabetes. The corresponding variable was considered as self-reported diabetes or diagnosed diabetes. To identify individuals with undiagnosed diabetes, glycated hemoglobin (HbA1c) $\geq 6.5\%$ was used as the criterion (International Expert Committee, 2009). To obtain the total number of diabetics, cases of self-reported diabetes were added to the individuals with undiagnosed diabetes. Persons with type 1 diabetes or gestational diabetes were excluded from the analyses.

4.2.5 Statistical analysis

4.2.5.1 Descriptive analysis

The CHMS full sample (n=5604) was divided into 10 age/sex groups (6-11 y, 12-19 y, 20-39 y, 40-59 y, and 60-79 y). Regarding the dietary intake, the average daily consumption of defined food groups (frequency of consumption/day) among the Canadian population as well as the mean intake by different socio-demographic characteristics was evaluated. To determine significant difference in intake by various characteristics, 95% confidence intervals (CI) were

compared. Having no overlap in 95% CI of the estimates was considered significant statistical difference at 0.05. The distribution of Canadians with and without having a specific health outcome was assessed by various socio-demographic characteristics. Similarly no overlap in 95% CI of the estimates was considered statistically significantly different at 0.05. To determine significant differences in dietary intake in individuals with and without abdominal obesity, elevated TC/HDL-C, and diabetes independent sample *t*-test was used.

4.2.5.2   Analytical analysis

The association between dietary intake and abdominal obesity, elevated TC/HDL-C, and diabetes was evaluated using a multivariate logistic model. Details on how to obtain the best model were explained in "general methodology". All of the outcome variables were assessed as binary variables (having a disease=1 versus not having a disease=0) in logistic regression models. IBM SPSS statistics for windows, version 20 (Armonk, NY) was used for data manipulation, summarizing, categorizing, and creating new variables. STATA/SE 11, StataCorp was used for all statistical analyses. Alpha was set at the level of 0.05 for all analyses unless otherwise indicated.

4.3   Results

4.3.1   Descriptive Dietary Intake

Mean dietary intakes in each food group in the full sample and by sex and age as well as some other socio-demographic characteristics including ethnicity, income, education, and physical activity are presented here, **Table 4-1** & **Table 4-2**. Summary intake of nine food groups was evaluated including meat and fish consumption, milk and dairy product consumption, grains consumption, fruit and vegetable consumption, potato consumption, dietary fat consumption, sugar sweetened beverages (SSBs) consumption, diet soft drink consumption, fruit and vegetable juice consumption. The intake has been reported by daily frequency of consumption.

Table 4-1 Mean intake, standard error (SE) and 95% confidence interval of food consumption (times/day)   by socio-demographic characteristics (population size = 29,235,445).

Socio-demographic characteristics	Meat & Fish Mean±SE[†] (95% CI)	Milk & Dairy Mean±SE (95% CI)	Grains Mean±SE (95% CI)	Fruit & Vegetable Mean±SE (95% CI)	Potato Mean±SE (95% CI)	Fat Mean±SE (95% CI)
All individuals	1.64±0.05 (1.53-1.75)	1.75±0.05 (1.63-1.86)	3.49±0.16 (3.14-3.84)	3.29±0.07 (3.12-3.46)	0.42±0.02 (0.37-0.46)	0.45±0.01 (0.42-0.49)
Sex						
Male	1.74±0.07 (1.59-1.89)	1.69±0.06 (1.56-1.82)	3.72±0.23 (3.22-4.22)	2.97±0.08 (2.79-3.16)[*]	0.45±0.02 (0.40-0.50)	0.47±0.02 (0.43-0.51)
Female	1.54±0.06 (1.40-1.68)	1.80±0.06 (1.66-1.94)	3.26±0.15 (2.93-3.59)	3.61±0.09 (3.40-3.82)[*]	0.38±0.02 (0.34-0.43)	0.45±0.02 (0.41-0.49)
Age groups[1]						
6-11y	1.37±0.05 (1.26-1.48)[*]	2.93±0.06 (2.79-3.08)[*]	4.84±0.53 (3.67-6.01)[*]	3.30±0.08 (3.10-3.49)	0.40±0.02 (0.37-0.45)	0.44±0.02 (0.39-0.49)[*]
12-19 y	1.63±0.15 (1.31-1.96)	2.22±0.08 (2.04-2.41)[*]	3.88±0.25 (3.33-4.44)	3.05±0.14 (2.75-3.36)	0.46±0.02 (0.42-0.49)	0.59±0.02 (0.54-0.64)[*]
20-39 y	1.61±0.05 (1.50-1.73)[*]	1.68±0.09 (1.47-1.88)[*]	3.15±0.23 (2.64-3.67)[*]	3.15±0.07 (3.00-3.30)	0.39±0.02 (0.33-0.45)	0.48±0.03 (0.42-0.54)[*]
40-59 y	1.64±0.06 (1.50-1.78)[*]	1.47±0.06 (1.34-1.61)[*]	2.96±0.15 (2.61-3.30)[*]	3.35±0.12 (3.09-3.62)	0.38±0.02 (0.34-0.44)	0.44±0.02 (0.39-0.48)[*]
60-79 y	1.81±0.12 (1.53-2.08)[*]	1.56±0.07 (1.40-1.72)[*]	4.29±0.33 (3.57-5.01)	3.58±0.14 (3.28-3.88)	0.50±0.03 (0.43-0.58)	0.37±0.02 (0.33-0.42)[*]
Ethnicity						
White individuals	1.58±0.04 (1.49-1.67)[*]	1.80±0.04 (1.70-1.89)[*]	3.66±0.16 (3.30-4.02)	3.34±0.09 (3.14-3.55)	0.44±0.02 (0.39-0.48)[*]	0.47±0.02 (0.44-0.50)
Non-white individuals	1.94±0.11 (1.69-2.19)[*]	1.49±0.08 (1.30-1.67)[*]	2.67±0.20 (2.22-3.12)	3.03±0.08 (2.86-3.21)	0.31±0.02 (0.25-0.36)[*]	0.38±0.04 (0.30-0.46)

Income[2]

Low income	1.92±0.22	1.68±0.07	4.29±0.52	2.64±0.16	0.48±0.03	0.48±0.05
	(1.44-2.41)	(1.52-1.85)	(3.14-5.43)	(2.29-2.99)*	(0.42-0.55)	(0.36-0.59)
Lower middle income	1.67±0.10	1.78±0.11	3.88±0.35	3.07±0.10	0.44±0.03	0.44±0.02
	(1.44-1.90)	(1.54-2.03)	(3.12-4.65)	(2.84-3.30)	(0.36-0.52)	(0.39-0.50)
Upper middle income	1.59±0.06	1.72±0.07	3.47±0.18	3.31±0.12	0.41±0.02	0.43±0.02
	(1.48-1.68)	(1.55-1.88)	(3.06-3.89)	(3.04-3.57)*	(0.37-0.46)	(0.39-0.46)
High income	1.58±0.04	1.74±0.04	3.12±0.22	3.46±0.09	0.39±0.02	0.47±0.02
	(1.45-1.72)	(1.65-1.84)	(2.64-3.61)	(3.25-3.67)*	(0.34-0.44)	(0.43-0.50)

Education

< secondary school graduation	1.81±0.26	1.44±0.13	4.82±0.66	3.14±0.36	0.54±0.04	0.41±0.03
	(1.23-2.39)	(1.15-1.73)	(3.35-6.28)	(2.34-3.94)	(0.45-0.63)	(0.34-0.48)
Secondary school graduation	1.51±0.07	1.68±0.08	3.29±0.34	2.94±0.07	0.46±0.02	0.43±0.02
	(1.34-1.67)	(1.51-1.87)	(2.54-4.03)	(2.77-3.10)	(0.40-0.51)	(0.40-0.47)
Some post-secondary	1.56± 0.11	1.65± 0.08	3.84± 0.29	2.85± 0.11	0.47± 0.03	0.49± 0.03
	(1.32-1.80)	(1.46-1.84)	(3.19-4.49)	(2.59-3.10)	(0.40-0.54)*	(0.42-0.56)
Post-secondary graduation	1.64±0.05	1.79±0.05	3.34±0.12	3.41±0.08	0.39±0.02	0.45±0.01
	(1.52-1.77)	(1.67-1.91)	(3.07-3.62)	(3.22-3.59)	(0.35-0.43)*	(0.42-0.49)

Physical activity

Active	1.63±0.03	1.91±0.07	3.35±0.24	3.79±0.10	0.39±0.02	0.45±0.02
	(1.56-1.70)	(1.74-2.09)*	(2.81-3.89)	(3.55-4.04)*	(0.35-0.43)	(0.41-0.50)
Moderately active	1.62±0.05	1.72±0.05	3.26±0.32	3.57±0.12	0.38±0.02	0.45±0.02
	(1.52-1.73)	(1.60-1.84	(2.56-3.95)	(3.29-3.85)	(0.35-0.42)	(0.41-0.48)
Inactive	1.69±0.08	1.49±0.07	3.45±0.20	2.93±0.08	0.44±0.03	0.46±0.02
	(1.50-1.88)	(1.33-1.65)*	(3.01-3.89)	(2.75-3.11)*	(0.38-0.51)	(0.41-0.52)

* Statistically significant

[†] Standard Error

[1] 6-11 y individuals had lower intake of meat and fish compared with 20-39 y, 40-59 y, and 60-79 y individuals. Children 6-11 y and adolescents 12-19 y significantly had greater milk and dairy consumption compared with other age groups. 6-11 y individuals had significantly greater intake of grains compared with 20-39 y and 40-59 y individuals. Adolescents 12-19 y had significantly greater intake of dietary fat compared with other age groups.

[2] Upper middle income and high income households had greater fruit and vegetable significantly compared with low income households

4.3.1.1 Meat and fish consumption

The mean dietary intake across various socio-demographic characteristics was evaluated. Age groups and ethnicity indicated significant difference in meat consumption. Meat and fish intake was significantly lower among 6-11 y individuals compared with 20-39 y, 40-59 y, and 60-79 y. Furthermore, non-white individuals had significantly more meat and fish consumption compared to white individuals

4.3.1.2 Milk and dairy product consumption

Milk consumption was significantly greater among 6-11 y and 12-19 y individuals compared with 20-39 y, 40-59 y, and 60-79 y. White Canadians had more milk and dairy product consumption compared to non-white individuals. Furthermore, physically active Canadians had greater consumption of milk and dairy product compared to their inactive counterparts.

4.3.1.3 Grains consumption

The frequency of grains consumption among 6-11 y individuals was higher compared with 20-39 y and 40-59 y.

4.3.1.4 Fruit and vegetable consumption

Females had significantly greater amount of fruit and vegetable intake compared to males. Individuals from upper middle and high income families had significantly greater amount of fruit and vegetable intake compared to individuals from low income families. Moreover, active and moderately active individuals had more fruit and vegetable intake in comparison with inactive individuals.

4.3.1.5 Potato consumption

White individuals consumed significantly greater amount of potatoes compared to their non-white counterpart. Individuals from low-educated families had a greater amount of potato consumption compared to individuals from high-educated families.

4.3.1.6 Dietary fat consumption

Adolescents or 12-19 y individuals consumed the greatest amount of dietary fat and this was significantly greater compared to other age groups.

Table 4-2 Mean intake, standard error (SE) and 95% confidence interval of beverage consumption (times/day) by socio-demographic characteristics (population size = 29,235,445).

Socio-demographic characteristics	Sugar-Sweetened Beverages	Diet Soft Drinks	Fruit & Vegetable Drinks
	Mean±SE (95% CI)	Mean±SE (95% CI)	Mean±SE (95% CI)
All individuals	0.49±0.02 (0.44-0.45)	0.17±0.01 (0.14-0.20)	0.76±0.02 (0.71-0.81)
Sex			
Male	0.63±0.03 (0.56-0.70)[*]	0.17±0.01 (0.14-0.20)	0.81±0.03 (0.75-0.88)
Female	0.35±0.03 (0.30-0.42)[*]	0.17±0.03 (0.12-0.22)[1]	0.73±0.03 (0.65-0.77)
Age groups[3]			
6-11 y	0.63±0.04 (0.55-0.72)[*]	0.09±0.06 (-0.04-0.23)[2]	0.98±0.03 (0.91-1.07)[*]
12-19 y	0.85±0.05 (0.75-0.95)[*]	0.10±0.01 (0.06-0.13)	0.97±0.06 (0.84-1.10)[*]
20-39 y	0.64±0.05 (0.52-0.76)[*]	0.18±0.02 (0.13-0.24)	0.75±0.04 (0.66-0.85)[*]
40-59 y	0.36±0.03 (0.29-0.43)[*]	0.19±0.02 (0.15-0.24)	0.69±0.03 (0.63-0.75)[*]
60-79 y	0.19±0.02 (0.14-0.25)[*]	0.12-0.22 (0.12-0.22)	0.68±0.02 (0.63-0.74)[*]
Ethnicity			
White individuals	0.49±0.02 (0.44-0.55)	0.16±0.01 (0.14-0.18)	0.79±0.03 (0.73-0.85)
Non-white individuals	0.48±0.03 (0.40-0.56)	0.20±0.07 (0.05-0.35)[2]	0.63±0.04 (0.54-0.71)
Income[4]			
Low income	0.76±0.07 (0.61-0.91)[*]	0.28±0.12 (0.00-0.56)[1]	0.84±0.08 (0.67-1.01)
Lower middle income	0.52±0.04 (0.42-0.62)	0.24±0.08 (0.05-0.43)[1]	0.75±0.07 (0.59-0.91)

Upper middle income	0.51±0.04	0.14±0.01	0.76±0.04
	(0.43-0.60)[*]	(0.10-0.17)	(0.68-0.85)
High income	0.43±0.03	0.17±0.01	0.76±0.03
	(0.35-0.51)[*]	(0.14-0.19)	(0.68-0.84)
Education			
< secondary school	0.64±0.15	0.10±0.02	0.62±0.04
graduation	(0.30-0.98)	(0.05-0.14)[1]	(0.52-0.71)[*]
Secondary school graduation	0.53±0.05	0.15±0.03	0.72±0.06
	(0.42-0.64)	(0.07-0.20)[1]	(0.58-0.87)
Some post-secondary	0.75± 0.08	0.11± 0.02	0.79± 0.05
	(0.57-0.93)[*]	(0.06-0.15)[1]	(0.67-0.91)
Post-secondary graduation	0.45±0.02	0.18±0.01	0.78±0.03
	(0.40-0.50)[*]	(0.15-0.22)	(0.72-0.84)[*]
Physical activity			
Active	0.45±0.03	0.18±0.03	0.90±0.06
	(0.39-0.52)	(0.11-0.24)[1]	(0.77-1.02)
Moderately active	0.44±0.02	0.15±0.02	0.80±0.04
	(0.38-0.49)	(0.11-0.19)	(0.71-0.88)
Inactive	0.52±0.03	0.18±0.01	0.65±0.03
	(0.44-0.59)	(0.15-0.22)	(0.58-0.72)

[*] Statistically significant
[1] Data with a coefficient of variation from 16.6% to 33.3%.
[2] Data with a coefficient of variation >33.3%. The user is advised that > 33.3% for coefficient of variation does not meet Statistics Canada's quality standards for this statistical program.
[3] Adolescents 12-19 y had significantly greater intake of SSBs and 60-79 y had significantly lower intake compared to other age groups. Children 6-11 y and adolescents 12-19 y had significantly greater fruit and vegetable juice intake compared to other age groups.
[4] Low income households consumed greater SSBs compared to upper middle income and high income households.

4.3.1.7   Sugar sweetened beverages (SSBs) consumption

Canadian men drank a significantly greater amount of SSBs compared to Canadian females. Adolescents (12-19 y) consumed the greatest amount of SSBs and this was statistically significant compared to almost all other age groups. Also, 40-59 y and 60-79 y individuals drank a significantly lower amount of SSBs compared to other age groups. Individuals from the low income category consumed a greater number of SSBs compared to individuals from upper-middle income and high income categories. Furthermore, the presence of a member with some post-secondary education in the family increased SSBs consumption significantly compared to the presence of a member with post-secondary graduation in the family.

4.3.1.8　Diet soft drink consumption

No significant difference was observed by socio-demographic characteristics.

4.3.1.9　Fruit and vegetable juice consumption

Both 6-11 y and 12-19 y groups drank significantly greater fruit and vegetable juice compared to 40-59 y and 60-79 y. White individuals consumed more fruit and vegetable juice compared to their non-white counterparts. Moreover, individuals from families with at least one member with less than secondary school graduation consumed a significantly lower amount of fruit and vegetable juice compared to individuals from families with at least one member with post-secondary graduation. Active Canadians significantly drank more fruit and vegetable juice compared to inactive Canadians.

4.3.2　Abdominal Obesity

4.3.2.1　Descriptive abdominal obesity

The sample (n=4445) used to investigate abdominal obesity represents 26,483,831 Canadians aged 12-79 y. The estimated overall prevalence of abdominal obesity for this group was 35.8%±1.8. The prevalence was significantly greater among Canadian women compared to men (**Table 4-3**). The ascendant increase in the prevalence of abdominal obesity by increase in age is observed in **Table 4-3**. The prevalence of abdominal obesity was significantly lower among Canadian households with postsecondary graduation compared to less than secondary education. With respect to income categories, the prevalence in the highest income category was significantly lower than the lower-middle income category. The prevalence of abdominal obesity decreased with increasing level of physical activity (**Table 4-3**).

Dietary consumption among Canadians aged 12 to 79 y with and without abdominal obesity is presented in **Table 4-4**. Canadians with abdominal obesity consumed significantly greater dairy products and diet soft drinks but less dietary fat and fruit and vegetable juice compared to Canadians without abdominal obesity.

Table 4-3 Weighted estimates of the prevalence of abdominal obesity by socio-demographic characteristics of Canadians aged 12 to 79 y (population size= 26,483,831)

Characteristics	Abdominal Obesity, estimated prevalence±SE[1]	Confidence Intervals
Sex		
Male	30.8±2.0[*]	26.28-32.29
Female	40.7±2.2[*]	35.89-45.52
Age group[3]		
12 – 19y	12.4±2.2[*2]	7.64-17.26
20 – 39y	28.2±2.1[*]	23.69-32.81
40 – 59y	40.4±2.4[*]	35.03-45.80
60 – 79y	56.2±2.8[*]	49.97-62.48
Education level		
< secondary school graduation	54.4±4.5[*]	44.49-64.30
Secondary school graduation	43.1±3.2	36.12-50.05
Some postsecondary	39.2±5.0	28.19-50.25
Postsecondary graduation	32.6±1.7[*]	28.73-36.52
Income level		
Lowest income	38.0±4.0	29.10-46.92
Lower-middle income	47.1±3.5[*]	39.31-54.97
Upper-middle income	35.9±1.9	31.71-40.16
Highest income	32.6±2.3[*]	27.40-37.73
Physical activity[4]		
Inactive	43.3±2.3[*]	38.30-48.32
Moderately active	33.2±2.3[*]	28.20-38.30
Active	21.9±2.5[*]	16.38-27.42
Alcohol		
Never drink	31.4±2.9	25.06-37.82
Ever drink	36.2±2.0	31.70-40.77
Ethnicity		
Non-white	34.7±4.6	24.56-44.80
White	36.0±1.7	32.18-39.90
Smoking		
Non-smokers	36.9±2.0	32.47-41.38
Smokers	30.9±2.1	26.29-35.60

[*] Statistically significant
[1] SE= Standard Error
[2] Data with a coefficient of variation from 16.6% to 33.3%.
[3] The prevalence of abdominal obesity increased with advancing age
[4] The prevalence of abdominal obesity increased with increase in the level of physical activity

Table 4-4 Dietary consumption among Canadians aged 12 to 79 y with and without abdominal obesity (population size=26,483,831)

Food and beverages times/day[1]	Individuals with abdominal obesity, Mean±SE[2] (CI[3])	Individuals without abdominal obesity, Mean±SE[2] (CI[3])
Meat and Fish		
-Red meat, organs, hotdogs, sausage or bacon, seas foods, eggs, beans, and nuts	1.68±0.07 (1.53-1.84)	1.74±0.11 (1.49-2.00)
Grains, Fruit & Vegetable		
-Hot/cold cereal, white bread, brown bread, rice, pasta (grains)	3.42±0.27 (2.82-4.01)	3.03±0.16 (2.67-3.39)
-Fruit and Vegetable	3.17±0.09 (2.95-3.39)	3.36±0.09 (3.16-3.55)
Milk & Dairy Products		
-Milk, cottage cheese, and yogurt or ice cream	1.71±0.05[*] (1.60-1.82)	1.54±0.07[*] (1.39-1.70)
Dietary Fat		
-Regular-fat salad dressing or mayonnaise and regular-fat potato chips, tortilla chips or corn chips	0.40±0.03[*] (0.33-0.47)	0.43±0.02[*] (0.37-0.49)
Water & Soft Drinks		
-Regular soft drink, sport drink, and fruit drink (sugar-sweetened beverages)	0.42±0.04 (0.33-0.51)	0.52±0.04 (0.43-0.60)
- Diet soft drink	0.21±0.01[*] (0.18-0.24)	0.15±0.01[*] (0.13-0.19)
-Fruit and vegetable juice	0.65±0.02[*] (0.60-0.70)	0.70±0.03[*] (0.63-0.77)

[*] Intake is significantly different between individuals with and without abdominal obesity based on independent sample t-test.
[1] Frequency of consumption
[2] SE= Standard Error
[3] Confidence Intervals

4.3.2.2 Association between abdominal obesity and dietary intake

4.3.2.2.1 Univariate analysis

Table 1 and **Table 2** in **appendix 3** indicate the univariate association between abdominal obesity and each independent predictor. Socio-demographic variables and food groups were included in **Table 1** and **Table 2**, respectively.

Among socio-demographic characteristics, women, older age, low level of education, low income, low level of physical activity, and smoking were significantly associated with risk of abdominal obesity based on the assumption of the p-value <0.2 for univariate analysis (**Table 1, appendix 3**).

Since "sex" was considered as a significant predictor in favor of women (OR: 0.65; p-value<0.01; CI: 0.53-0.79), the analysis was conducted separately for males and females. The association between socio-demographic variables and abdominal obesity for males and females were similar to the full sample (**Table 1, appendix 3**).

All food groups investigated were significantly associated with the risk of abdominal obesity based on the assumption of the p-value <0.2 for univariate analysis (**Table 2, appendix 3**). However, the significant association was not seen in all quartiles of intake in each food group. Compared to the first quartile of intake, consuming more meat and fish, grains, fruit and vegetable, dietary fat, milk and dairy products, SSBs, and fruit and vegetable juice were associated with reduced risk of abdominal obesity. However, diet soft drink intake was associated with increased risk of abdominal obesity. The results for potato consumption were different for different levels of intake.

The trend in the association between food groups and abdominal obesity for males were similar to the overall population (**Table 2, appendix 3**). However, the intake in meat and fish, grains, fruit and vegetable, dairy product, and fruit and vegetable juice were no longer associated with abdominal obesity in females.

4.3.2.2.2 Multivariate analysis

The mean Vifs for the full sample, male and female samples were equal to 1.92, 1.79, and 1.69, respectively, which was less than 10 as the threat for multicollinearity. Moreover, it was less than what Paul Allison considers (Vif=2.5) as the threat for multicollinearity (Allison, 2012). **Table 4-5** indicates the best model of the association between abdominal obesity and independent indicators after implementing specification error test. Additionally, a sex-specific model was conducted and results are presented in **Table 4-5**.

Table 4-5 Logistic regression analysis indicating the association between abdominal obesity and the dietary intake after adjusting for the potential covariates[§] (population size: all=26,483,831, male=13,171,095, female=13,312,736)

Predictor Variables	Odds Ratio(95% Confidence Interval) P-value		
Age Groups	**All**	**Male**	**Female**
12-19 y[1]			
20-39 y	2.32(1.09-4.91) 0.031[*]	2.25(0.79-6.40) 0.116	1.96(1.00-3.85) 0.048[*]
40-59 y	2.47(1.49-4.07) 0.002[*]	4.52(1.82-11.22) 0.004[*]	1.84(0.97-3.52) 0.059
60-79 y	4.70(2.68-8.25) <0.001[*]	8.57(3.31-22.17) <0.001[*]	3.92(1.97-7.81) 0.001[*]
Physical activity			
Active[1]			
Moderately active	1.69(1.08-2.63) 0.024[*]	1.58(1.04-2.39) 0.034[*]	1.61(0.88-2.96) 0.108
Inactive	2.50(1.78-3.52) <0.001[*]	2.49(1.62-3.82) 0.001[*]	2.35(1.54-3.59) 0.001[*]
Education			
< secondary school graduation[1]			
Secondary school graduation	0.83(0.47-1.46) 0.496	N/A[3]	N/A
Some postsecondary	0.90(0.45-1.79) 0.748	N/A	N/A
Postsecondary graduation	0.60(0.37-0.92) 0.023[*]	N/A	N/A
Smoking[2]	0.77(0.62-0.97) 0.030[*]	N/A	N/A
Dietary Fat			
1st Q [1]			
2nd Q	0.55(0.31-0.98) 0.047[*]	N/A	0.61(0.28-1.30) 0.181
3rd Q	0.43(0.19-0.95) 0.040[*]	N/A	0.30(0.09-0.99) 0.050[*]
4th Q	0.65(0.28-1.51) 0.285	N/A	0.49(0.13-1.83) 0.259
Diet Soft Drink[3]	1.46(1.15-1.85) 0.005[*]	1.55(1.04-2.31) 0.034[*]	N/A

Fruit & Vegetable Juice			
1st Q [1]			
2nd Q	N/A	0.79(0.49-1.25) 0.285	N/A
3rd Q	N/A	0.52(0.36-0.74) 0.002[*]	N/A
4th Q	N/A	0.70(0.45-1.08) 0.101	N/A
Dietary fat # age group			
2nd Q#20-39 y	0.88(0.29-2.57) 0.797	N/A	0.67(0.18-2.48) 0.514
3rd Q#20-39 y	1.17(0.42-3.25) 0.740	N/A	1.54(0.40-5.91) 0.487
4th Q#20-39 y	0.85(0.30-2.44) 0.751	N/A	1.19(0.33-4.33) 0.771
2nd Q#40-59 y	2.37(1.14-4.94) 0.025[*]	N/A	2.60(1.09-6.20) 0.034[*]
3rd Q#40-59 y	2.19(0.94-5.14) 0.067	N/A	2.66(0.61-11.66) 0.172
4th Q#40-59 y	1.58(0.59-4.22) 0.325	N/A	2.08(0.42-10.25) 0.332
2nd Q#60-79 y	1.60(0.66-3.89) 0.269	N/A	1.67(0.59-4.73) 0.302
3rd Q#60-79 y	2.74(1.14-6.60) 0.028[*]	N/A	3.79(1.15-12.44) 0.031[*]
4th Q#60-79 y	1.30(0.50-3.38) 0.556	N/A	1.56(0.36-6.69) 0.515

According to the "linktest" the p-value was <0.001 and 0.402 for variable _hat and variable _hatsq, respectively, indicate that the model fitted well for all population. Similarly, sex-specific p-values for _hat and variable _hatsq were <0.001 and 0.133 for males and <0.001 and 0.584 for females, respectively

§ Covariates include age, sex, physical activity, ethnicity, education, income, smoking, and alcohol intake.

[*] Statistically significant from the reference group

[1] Reference group

[2] Smokers=1(reference group) versus non-smokers=0

[3] Diet soft drink categorized into 2 categories (0 times/day and > 0 times/day).

[3] Not Applicable

Men were 34% less at risk of having abdominal obesity than women (OR: 0.66; p-value= 0.004; CI: 0.52- 0.85). According to **Table 4-5,** the risk of abdominal obesity in moderately active and inactive individuals was greater in comparison with active individuals. Individuals from the households with some post-secondary graduation significantly had less risk of (23%) abdominal obesity compared to individuals from less than secondary school graduation. Smokers

were less at risk of having abdominal obesity than non-smokers. Consuming diet soft drink was associated with increased risk of abdominal obesity by 1.5 times compared to not consuming.

A similar trend in associations was observed in males and females (**Table 4-5**). Regarding fruit and vegetable juice intake in males, consumption in the amount of 3rd quartile was associated with decreased risk of abdominal obesity by 48% compared to the 1st quartile of intake. It appears that there might be a U-shaped association between fruit and vegetable juice intake and abdominal obesity among men. In order to have a better sense of how much intake is categorized in each quartile, the intake is identified based on the frequency of consumption per week (**Table 4-5**). If each time of intake is considered as one serving of intake which is 125 ml or half a cup, a reverse association is observed between the intake of 2 to 3.5 cups of juice per week among males with abdominal obesity.

An interaction was observed between dietary fat intake and age groups in the full sample and in females. The effect of dietary fat on abdominal obesity is defined while considering age groups and it also revealed a U-shaped association. According to **Table 4-5** there was a significant interaction between the fat intake and age group 3 (**Figure 1 & 2, Appendix 4**). In the second quartile of fat consumption, the risk of having abdominal obesity in age group 3 (40-59 y) is 6.25 and 5.07 times the risk than age group 1 (12-19 y) in all and females, respectively. Moreover, in the 3rd quartile of fat consumption, the risk of having abdominal obesity in age group 4 (60-79 y) is 14.83 and 16.83 times the risk than age group 1 (12-19 y) in all and females, respectively. The frequency of dietary fat intake has been converted to the serving of intake per week in **Table 4-6**.

Table 4-6 The conversion of the quartile of dietary fat and fruit and vegetable juice intake into weekly frequency of consumption

Quartile of Intake	Fat[1]		Fruit & Vegetable Juice[2]
	All (times/week)	Female (times/week)	Male (times/week)
1st quartile	Intake < 1	Intake < 1	Intake≤ 1
2nd Quartile	1≤ intake≤2.5	1≤intake<2.5	1< intake<4
3rd Quartile	2.5< intake<5	2.5≤ intake<5	4≤ intake≤7
4th Quartile	Intake ≥ 5	Intake ≥ 5	Intake > 7

[1] 1 tablespoon of salad dressing or mayonnaise or 15-20 chips was considered as one serving
[2] One serving is equivalent to ½ cup of 100% fruit or vegetable juice

4.3.2.3   Summary

The estimated overall prevalence of abdominal obesity in 12-79 y Canadians was 35.7% with the prevalence significantly greater in women (40.7 vs. 30.7). Controlling for the potential confounders, the risk of abdominal obesity was explained strongly by age, physical activity, education, smoking, dietary fat intake, and diet soft drink intake in Canadians 12-79 y. Households in which at least one member was highly educated had less risk of abdominal obesity compared to the households without highly educated individuals. The risk dropped in smokers versus non-smokers. Dietary fat intake in the 2nd quartile among 20-39 y individuals or dietary fat intake in the 3rd quartile among 60-79 y individuals was associated with increased risk of abdominal obesity.

4.3.3   Total Cholesterol/HDL Cholesterol Ratio

4.3.3.1 Descriptive TC/HDL-C Ratio

The sample (n=3430) used to investigate elevated TC/HDL-C ratio represents 23,135,787 Canadians aged 20-79 y. The estimated overall prevalence of elevated ratio for this population was 20.5%±1.7. The prevalence was significantly greater among Canadian men than women (**Table 4-7**). The greatest prevalence was observed among middle-age individuals which was greater than for young individuals 20-39 y. With respect to other socio-demographic variables no statistically significant difference was indicated.

Dietary consumption among Canadians aged 20 to 79 y with and without elevated TC/HDL-C ratio is shown in **Table 4-8**. Canadians with elevated TC/HDL-C ratio consumed significantly greater dietary fat and sugar-sweetened beverages but less fruit and vegetable compared to Canadians with normal TC/HDL-C ratio.

Table 4-7 Weighted estimates of the prevalence of elevated TC/HDL-C ratio by socio-demographic characteristics of Canadians aged 20 to 79 y (population size= 23,135,787)

Characteristics	TC/HDL-C ratio, estimated prevalence±SE[1]	Confidence Intervals
Sex		
Male	29.1±2.1[*]	25.01-34.34
Female	11.7±1.8[*]	8.20-16.45
Age group		
20 – 39y	15.4±2.1[*]	11.35-20.48
40 – 59y	25.3±2.2[*]	20.83-30.37
60 – 79y	20.1±2.0	16.28-24.59
Education level		
< secondary school graduation	25.2±4.1	17.25-35.40
Secondary school graduation	23.4±3.1	17.27-30.99
Some postsecondary	18.3±3.1	12.36-26.21
Postsecondary graduation	19.9±1.9	15.90-24.58
Income level		
Lowest income	18.4±4.8	10.04-31.30
Lower-middle income	19.8±3.1	13.91-27.50
Upper-middle income	18.9±2.3	14.35-24.63
Highest income	22.1±2.1	17.79-27.08
Physical activity		
Inactive	21.4±23	16.84-26.82
Moderately active	20.8±2.4	15.88-26.71
Active	17.6±1.7	14.12-21.82
Alcohol		
Never drink	31.4±2.9	25.06-37.82
Ever drink	36.2±2.0	31.70-40.77
Ethnicity		
Non-white	20.5±2.9	14.79-27.70
White	20.5±1.8	16.65-24.93
Smoking		
Non-smokers	19.2±1.9	15.26-23.99
Smokers	25.4±1.9	21.26-29.94

[*] Statistically significant
[1] SE= Standard Error

Table 4-8 Dietary consumption among Canadians aged 20 to 79 y with and without elevated TC/HDL-C ratio (population size=23,135,787)

Food and beverages times/day[1]	Individuals with elevated ratio Mean±SE[2] (CI[3])	Individuals without elevated ratio, Mean±SE[2] (CI[3])
Meat and Fish		
-Red meat, organs, hotdogs, sausage or bacon, seas foods, eggs, beans, and nuts	1.66±0.15 (1.32-2.01)	1.78±0.11 (1.54-2.02)
Grains, Fruit & Vegetable		
-Hot/cold cereal, white bread, brown bread, rice, pasta (grains)	3.17±0.16 (2.81-3.53)	3.31±0.16 (2.94-3.66)
-Fruit and Vegetable	3.07±0.10[*] (2.85-3.30)	3.39±0.08[*] (3.21-3.57)
Milk & Dairy Products		
-Milk, cottage cheese, and yogurt or ice cream	1.55±0.06 (1.42-1.68)	1.57±0.06 (1.44-1.70)
Dietary Fat		
-Regular-fat salad dressing or mayonnaise and regular-fat potato chips, tortilla chips or corn chips	0.47±0.03[*] (0.39-0.55)	0.38±0.02[*] (0.32-0.43)
Water & Soft Drinks		
-Regular soft drink, sport drink, and fruit drink (sugar-sweetened beverages)	0.33±0.04[*] (0.26-0.40)	0.24±0.01[*] (0.20-0.28)
- Diet soft drink	0.21±0.02 (0.16-0.26)	0.18±0.01 (0.15-0.21)
-Fruit and vegetable juice	0.78±0.07 (0.61-0.95)	0.70±0.02 (0.64-0.75)

[*] Intake is statistically significant between individuals with and without elevated TC/HDL-C ratio based on the independent sample t-test
[1] Frequency of consumption
[2] SE= Standard Error
[3] Confidence Intervals

4.3.3.1   Association between elevated TC/HDL-C ratio and dietary intake

4.3.3.1.1   Univariate analysis

Table 3 and **Table 4** in **Appendix 3** indicate the univariate association between elevated TC/HDL-C ratio and each independent predictor. Socio-demographic variables and food groups were included in **Table 3** and **Table 4,** respectively. Among socio-demographic characteristics, men, older age, low level of education, low level of physical activity, smoking, and never

drinking alcohol were significantly associated with risk of elevated TC/HDL-C ratio based on the assumption of the p-value <0.2 for univariate analysis (**Table 3, Appendix 3**).

As "sex" was considered as a significant predictor in favor of men (OR: 3.15; p-value <0.001; CI: 2.27-4.36), sex-specific analysis was conducted. The associations between socio-demographic variables and elevated TC/HDL-C ratio for males and females were similar to that for all population (**Table 3, Appendix 3**).

The association between food groups and elevated TC/HDL-C ratio by considering the assumption of the p-value <0.2 for univariate analysis are shown in (**Table 4, Appendix 3**). Compared to the first quartile, consuming greater amount of meat and fish, fruit and vegetable, and milk and dairy products were associated with reduced risk of elevated TC/HDL-C ratio. However, compared to the first quartile, greater intake of dietary fat, SSBs, and diet soft drink was associated with increased risk of elevated TC/HDL-C ratio.

In women, consuming meat and fish in the amount of 3rd and 4th quartile were associated with decreased risk of elevated TC/HDL-C ratio by 29% and 55% compared to the first quartile, respectively. Fruit and vegetable intake and diet soft drink consumption were no longer significant among women. Intake of SSBs was not significant among men.

4.3.3.1.2 Multivariate analysis

The mean Vif for the full sample, male sample, and female sample was equal to 1.59, 1.40, and 1.51, respectively, which was less than 2.5 as the threat for multicollinearity. **Table 4-9** indicates the best model of the association between elevated total/HDL cholesterol ratio and independent indicators after implementing specification error test. Additionally, a sex-specific model was conducted and has been presented in **Table 4-9.**

Table 4-9 Logistic regression analysis indicating the association between elevated TC/HDL-C ratio and the dietary intake after adjusting for the potential covariates[§] (population size: all=23,135,787, male=11,440,786, female=11,695,001)

Predictor Variables	Odds Ratio(95% Confidence Interval)		
	P-value		
Age group	All	Male	Female
20-39 y[1]			
40-59 y	1.91(1.00-3.64)	1.68(0.97-2.92)	2.08(1.10-3.93)
	0.048[*]	0.063	0.027[*]
60-79 y	2.08(1.33-3.27)	1.38(0.89-2.12)	1.46(0.79-2.67)
	0.004[*]	0.126	0.199
Abdominal Obesity[2]	6.12(2.68-13.96)	3.09(1.35-7.09)	2.79(1.14-6.87)
	0.001[*]	0.012[*]	0.029[*]
Smoking[3]	2.13(1.45-3.11)	N/A[6]	2.68(1.64-4.23)
	0.001[*]		0.001[*]
Milk & Dairy Products			
1st Q[1]			
2nd Q	1.27(0.80-2.00)	N/A	N/A
	0.272		
3rd Q	2.01(1.29-3.15)	N/A	N/A
	0.005[*]		
4th Q	1.66(0.97-2.85)	N/A	N/A
	0.060		
Dietary Fat			
1st Q[1]			
2nd Q	N/A	N/A	0.37(0.13-1.03)
			0.057
3rd Q	N/A	N/A	0.53(0.19-1.44)
			0.189
4th Q	N/A	N/A	1.59(0.49-5.10)
			0.397
Alcohol intake[4]	0.43(0.22-0.84)	N/A	0.37(0.17-0.83)
	0.018[*]		0.016[*]
Diet Soft Drink[5]	1.37(1.03-1.82)	1.62(1.11-2.37)	N/A
	0.031[*]	0.016[*]	
Abdominal Obesity # age group			
Obese#40-59 y	0.82(0.39-1.69)	0.82(0.36-1.87)	N/A
	0.559	0.614	
Obese#60-79 y	0.44(0.26-0.73)	0.52(0.30-0.93)	N/A
	0.005[*]	0.031[*]	

Smoking # age group			
Smoker#40-59 y	0.78(0.39-1.63) 0.507	N/A	N/A
Smoker#60-79 y	0.50(0.29-0.87) 0.019[*]	N/A	N/A
Abdominal Obesity # Milk & Dairy Products			
Obese#2nd Q	0.54(0.29-0.99) 0.049[*]	N/A	N/A
Obese#3rd Q	0.64(0.29-1.39) 0.238	N/A	N/A
Obese#4th Q	0.50(0.25-0.94) 0.036[*]	N/A	N/A
Abdominal Obesity # Dietary Fat			
Obese#2nd Q	N/A	N/A	3.09(0.90-10.58) 0.069
Obese#3rd Q	N/A	N/A	3.78(1.30-10.93) 0.019[*]
Obese#4th Q	N/A	N/A	0.98(0.24-4.02) 0.979
Physical Activity			
Active[1]			
Moderately active	1.25(0.86-1.82) 0.206	1.20(0.66-2.20) 0.500	1.12(0.48-2.59) 0.767
Inactive	1.19(0.79-1.80) 0.360	1.18(0.64-2.14) 0.555	0.89(0.44-1.80) 0.725

According to the "linktest" the p-value was 0.001 and 0.220 for variable _hat and variable _hatsq, respectively, indicate that the model fitted well for all population. Similarly, sex-specific p-values for _hat and variable _hatsq were 0.007 and 0.384 for males and <0.001 and 0.184 for females, respectively.
[§] Covariates include age, sex, physical activity, ethnicity, education, income, smoking, abdominal obesity, and alcohol intake.
[*] Statistically significant from the reference from
[1] Reference group
[2] Abdominal obesity =1 (reference group versus abdominal obesity=0.
[3] Smokers=1(reference group) versus non-smokers=0.
[4] Ever drinker =1 (reference group) versus never drinker=0.
[5] Diet soft drink categorized into 2 categories (0 times/day and > 0 times/day).
[6] Not Applicable

Being male was associated with risk of elevated TC/HDL-C (OR: 4.10; p-value< 0.001; CI: 2.94- 5.72). According to **Table 4-9**, consuming diet soft drinks was associated with increased risk of elevated TC/HDL-C compared to not consuming in the full sample and among men. However, ever drinking alcohol was associated with reduced risk of elevated TC/HDL-C ratio by 57% and 63% in the full sample and women, respectively, compared with never drinking alcohol.

An interaction was observed between abdominal obesity and age groups in the whole population and among females. Moreover, an interaction was observed between abdominal obesity and dairy product and between smoking and age group in the whole population. There was an interaction between abdominal obesity and dietary fat among females (**Table 4-9**).

Abdominally obese individuals had greater risk of elevated TC/HDL-C ratio than non-abdominally-obese individuals. There was a significant interaction between age group 60-79 y compared to age group 20-39 y and abdominal obesity on the risk of elevated TC/HDL-C ratio (**Figure 3 & 4, Appendix 4**). In the absence of abdominal obesity, the risk of having elevated ratio in 60-79 y individuals was 1.05 times the risk compared to 20-39 y individuals. In the presence of abdominal obesity, the risk of having elevated ratio by 60-79 y individuals was 55% less than 20-39 y individuals.

There was also an interaction between age group and abdominal obesity (**Figure 4, Appendix 4**). In the absence of abdominal obesity the risk of having elevated ratio in age group 60-79 y was 1.35 times greater than age group 20-39 y. In the presence of abdominal obesity the risk of having elevated ratio by 60-79 y individuals was 29% less than 20-39 y individuals.

Smoking was associated with 2 times more risk of elevated TC/HDL-C ratio than non-smoking. In the entire Canadian population a significant interaction was observed between smoking and age group 60-79 y compared to age group 20-39 y (**Figure 5, Appendix 4**). In non-smokers, the risk of having elevated ratio in age group 60-79 y was 10% less than age group 20-39 y. In smokers, the risk of having elevated ratio in age group 60-79 y was 55% less than age group 20-39 y.

Furthermore, in the entire Canadian population, a significant interaction was observed between milk and dairy intake in the 4th and 2nd quartile (compared to first quartile) and abdominal obesity (**Figure 6, Appendix 4**). In the absence of abdominal obesity, the risk of having elevated ratio in individuals having dairy in the amount of 4th quartile was 1.63 times the risk in the individuals having dairy products in the first quartile. In the presence of abdominal obesity, the risk of having elevated ratio in individuals having dairy in the amount of 4th quartile was about 20% less than individuals having dairy in the first quartile.

Among females there was an interaction between dietary fat and abdominal obesity (**Figure 7, Appendix 4**). In the absence of abdominal obesity the risk of having elevated ratio in females having dietary fat in the 4th quartile is 47% less than female in the first quartile. In the presence of abdominal obesity the risk of having elevated ratio in females having dietary fat in the 4th quartile was about 2 times those in the first quartile.

In order to have an estimation of intake in each quartile, the quartiles of dairy and dietary fat intake were converted to the frequency of weekly intake (**Table 4-10**). Each time of intake may be considered as one serving of consumption for each food group according to Canada's food guide.

Table 4-10 The conversion of the quartile of dietary fat and dairy intake into weekly frequency of consumption

Quartile of Intake	Milk and dairy product intake[1] (times/week)	Dietary fat intake among women[2] (times/week)
1st Q	Intake ≤ 5	Intake ≤ 0.5
2nd Q	5< intake≤9	0.5<intake≤2
3rd Q	9< intake<15	2.5≤ intake≤4
4th Q	Intake ≥ 15	Intake > 4

[1] One serving of milk and dairy is equivalent to 250 ml of milk, 50 grams of cheese, or 175 grams of yogurt
[2] One serving fat is equivalent to 1 tablespoon of salad dressing or mayonnaise or 15-20 chips

4.3.3.2 Summary

The estimated overall prevalence of elevated TC/HDL-C ratio in Canadians of age 20-79 y was 20.5% with the prevalence significantly in favor of men (29.5 vs. 11.7%). Controlling for the potential covariates, the risk of elevated TC/HDL-C ratio was explained statistically significant by abdominal obesity, age, sex, smoking, alcohol intake, milk and dairy product consumption, and diet soft drink in Canadians 20-79 y. Canadian males and older individuals, specifically middle age individuals, were highly at risk as well as abdominally obese Canadians. Milk and dairy product consumption in the 3rd quartile increased the risk of elevated TC/HDL-C ratio compared with 1st quartile, however, the effect was different according to the presence of abdominal obesity. Consuming diet soft drinks versus not consuming diet soft drinks increased the risk of elevated TC/HDL-C ratio. Furthermore, smoking and ever drinking alcohol were associated with reduced risk of elevated TC/HDL-C ratio.

4.3.4 Diabetes

4.3.4.1 Descriptive diabetes

The sample (n=3390) used to investigate diabetes represents 22,674,040 Canadians aged 20-79 y. The estimated overall prevalence of diabetes for this group was 7.6%±1.1 with no significant difference between males and females (**Table 4-11**). The prevalence of diabetes was greatest in individuals aged 60-79 y. The prevalence of diabetes was significantly lower among Canadian households with some postsecondary education or postsecondary graduation compared to both less than secondary education and secondary school graduation. Diabetic Canadians had a greater prevalence of abdominal obesity compared to non-diabetic individuals. Moreover, the prevalence of diabetes was significantly lower in individuals who have ever had alcohol compared to individuals who have never had alcohol.

Dietary consumption among Canadians aged 20 to 79 y with and without diabetes is presented in **Table 4-12**. Canadians with diabetes consumed a greater amount of diet soft drinks but less fruit and vegetable juice and SSBs compared to Canadians without diabetes.

Table 4-11Weighted estimates of the prevalence of identified diabetes by socio-demographic characteristics of Canadians aged 20 to 79 y (population size= 22,674,040)

Characteristics	Diabetes, estimated prevalence± SE[1]	Confidence Intervals
Sex		
Male	8.1 ± 1.4^2	5.55-11.81
Female	7.0 ± 1.0	5.00-9.70
Age group[3]		
20 – 39y	$1.0\pm0.3^{*2}$	0.51-2.19
40 – 59y	$8.3\pm1.5^{*2}$	5.50-12.48
60 – 79y	$17.7\pm2.1^{*}$	13.63-22.74
Education level[4]		
< secondary school graduation	$18.3\pm2.9^{*}$	12.72-25.60
Secondary school graduation	$9.0\pm2.5^{*2}$	22.03-40.26
Some postsecondary	$5.7\pm1.7^{*2}$	2.94-10.88
Postsecondary graduation	$6.5\pm1.2^{*2}$	4.33-9.75
Income level		
Lowest income	12.4 ± 3.6^2	9.44-22.57
Lower-middle income	11.8 ± 1.9	8.17-16.89
Upper-middle income	7.2 ± 1.2	5.01-10.32
Highest income	6.3 ± 1.2^2	4.15-9.50
Physical activity		
Inactive	9.5 ± 1.4	6.86-12.94
Moderately active	6.0 ± 0.9	4.16-8.54
Active	4.4 ± 1.2^2	2.39-7.95
Abdominal obesity		
Abdominal obese	$13.1\pm1.4^{*}$	10.34-16.44
Not abdominal obese	$3.9\pm1.0^{*}$	2.13-6.99
Alcohol		
Never drink	$14.7\pm2.3^{*}$	10.38-20.50
Ever drink	$6.4\pm1.0^{*}$	4.48-8.97
Ethnicity		
Non-white	8.9 ± 2.7^2	4.38-17.16
White	7.3 ± 1.0	5.37-9.92
Smoking		
Non-smokers	8.1 ± 1.2	5.79-11.12
Smokers	5.9 ± 1.2^2	3.73-9.34

* Statistically significant
[1] SE= Standard Error
[2] Data with a coefficient of variation from 16.6% to 33.3%.
[3] The prevalence of diabetes increased with advancing age.
[4] The prevalence of diabetes was significantly lower in the two high levels of education compared to two low levels of education.

Table 4-12 Dietary consumption among Canadians aged 20 to 79 y with and without diabetes (population size=22,674,040)

Food and beverages times/day[1]	Individuals with Diabetes, Mean ±SE[2] (CI[3])	Individuals without Diabetes, Mean±SE[2] (CI[3])
Meat and Fish		
-Red meat, organs, hotdogs, sausage or bacon, seas foods, eggs, beans, and nuts	1.72±0.25 (1.17-2.28)	1.67±0.05 (1.55-1.79)
Grains, Fruit & Vegetable		
-Hot/cold cereal, white bread, brown bread, rice, pasta (grains)	3.93±0.58 (2.65-5.20)	3.24±0.13 (2.96±3.52)
-Fruit and Vegetable	3.24±0.09 (3.04-3.44)	3.34±0.09 (3.14-3.53)
Milk & Dairy Products		
-Milk, cottage cheese, and yogurt or ice cream	1.40±0.07 (1.25-1.56)	1.58±0.05 (1.46-1.71)
Dietary Fat		
-Regular-fat salad dressing or mayonnaise and regular-fat potato chips, tortilla chips or corn chips	0.36±0.04 (0.27-0.50)	0.44±0.02 (0.41-0.50)
Water & Soft Drinks		
-Regular soft drink, sport drink, and fruit drink (sugar-sweetened beverages)	0.22±0.05[*] (0.11-0.33)	0.45±0.03[*] (0.39±0.51)
- Diet soft drink	0.39±0.06[*] (0.27-0.52)	0.17±0.01[*] (0.15-0.20)
-Fruit and vegetable juice	0.58±0.05[*] (0.48-0.69)	0.73±0.03[*] (0.67-0.79)

[*] Intake is statistically significant between individuals with and without diabetes based on the independent sample t-test
[1] Frequency of consumption
[2] SE= Standard Error
[3] Confidence Intervals

4.3.4.2 Association between diabetes and dietary intake

4.3.4.2.1 Univariate analysis

Table 5 and **Table 6** in **Appendix 3** indicate the univariate association between diabetes and each independent predictor. Socio-demographic variables and food groups are included in **Table 5** and **Table 6** respectively.

Among socio-demographic characteristics, older age, low level of education, low income, low level of physical activity, smoking, and alcohol intake were significantly associated with risk of diabetes based on the assumption of the p-value <0.2 for univariate analysis (**Table 5, Appendix 3**).

Based on the assumption of the p-value <0.2 for univariate analysis (**Table 6, Appendix 3**), compared to the first quartile, consuming more dietary fat, milk and dairy products, SSBs, and fruit and vegetable juice were associated with reduced risk of diabetes. However, compared to the first quartile, the risk of diabetes was associated with increased consumption of diet soft drink.

4.3.4.2.2 Multivariate analysis

The mean Vif was equal to 1.84 which was less than 2.5 as the threat for multicollinearity. **Table 4-13** indicates the best model of the association between diabetes and independent indicators after implementing specification error test.

Table 4-13 Logistic regression analysis indicating the association between diabetes and the dietary intake after adjusting for potential covariates[§] (population size=22,674,040)

Predictor Variables	Odds Ratio	P-value	95% Confidence Interval
Age Groups			
20-39 y[1]			
40-59 y	6.82	<0.001[*]	4.09-11.35
60-79 y	14.62	<0.001[*]	9.00-23.74
Abdominal Obesity[2]	2.25	0.012[*]	1.24-4.06
Physical activity			
Active[1]			
Moderately active	1.26	0.210	0.86-1.86
Inactive	2.39	0.001[*]	1.60-3.56
Education			
Less than secondary school graduation[1]			
Secondary school graduation	0.58	0.206	0.25-1.40
Some postsecondary	0.48	0.168	0.16-1.43
Postsecondary graduation	0.63	0.219	0.29-1.37
Alcohol intake[3]	0.49	0.007[*]	0.31-0.79
Sugar Sweetened Beverages			
1st quartile[1]			
2nd quartile	0.53	0.064	0.27-1.04
3rd quartile	0.75	0.303	0.42-1.34
4th quartile	0.46	0.042[*]	0.22-0.97
Diet Soft Drink[4]	2.72	<0.001[*]	1.89-3.92

According to the "linktest" the p-value was 0.013 and 0.145 for variable _hat and variable _hatsq, respectively, indicate that the model fitted well.
[*] Statistically significant from the reference group
[§] Covariates include age, sex, physical activity, ethnicity, education, income, smoking, abdominal obesity and alcohol intake.
[1]Reference group
[2]Abdominal obese=1 (reference group) versus not abdominal obese=0
[3] Ever drinking alcohol=1(reference group) versus never drinking alcohol=0.
[4] Diet soft drink categorized into 2 categories (0 times/day and >0 times/day).

According to the **Table 4-13** compared to age 20-39 y, the reference group, having

diabetes was associated with greater risk by age groups 40-59 y, and 60-79 y, respectively. All

variables remained significant after adding abdominal obesity into the model. The risk of

diabetes was greater in abdominally obese individual compared to non-abdominally obese

counterpart. Having diabetes among inactive individuals was associated greater risk in

comparison with active individuals. Moreover, the risk dropped by 51% in individuals who have

ever had alcohol. Consuming SSBs in the amount of 4th quartile appeared to decrease the risk of

diabetes by 54% compared to the first quartile. In contrast, consumption of diet soft drink was associated greater risk of diabetes.

Table 4-14 indicates the frequency of SSBs intake in each quartile. Each time of intake may be considered as one can serving.

No interaction was observed among the variables. However, education was a confounder for SSBs intake and alcohol intake. Therefore, it remained in the final model although it was not significant. The percent of change in the estimate between the crude and adjusted coefficients (data not shown) are observed below which are all greater than 10% as the pre-set percentage of change.

Formula: │ [crude coefficient − adjusted coefficient]/crude coefficient │ X100

SSBs consumption:

2nd quartile: │ [-0.75 − -0.64]/-0.75 │ X100 = 14.66

3rd quartile: │ [-0.38 − -0.28]/-0.38 │ X100 = 26.31

4th quartile: │ [-0.92 − -0.77]/-0.92 │ X100 = 16.30

Alcohol intake: │ [-0.86 − -0.70]/-0.86 │ X100 = 18.60

Table 4-14 The conversion of the quartile of sugar-sweetened beverages intake into weekly frequency of consumption

Quartile of Intake	Sugar-sweetened beverages consumption[1] (times/week)
$1^{st} Q$	Intake = 0
$2^{nd} Q$	0< intake<0.5
$3^{rd} Q$	0.5≤ intake<3
$4^{th} Q$	Intake ≥ 3

[1] 1 serving = 1 can (355ml) of SSB

4.3.4.3 Summary

The estimated overall prevalence of diabetes in Canadians aged 20-79 y was 7.5% with no significant difference between males and females. Controlling for the potential confounders,

the risk of diabetes was explained strongly by age, physical activity, abdominal obesity, SSBs consumption, diet soft drink intake in Canadians 20-79 y. Older ages were at higher risk as well as inactive individuals. Furthermore, the risk dropped in ever drinker of alcohol versus never drinkers. SSBs intake in the 4th quartile decreased the risk of disease compared to consumption in the 1st quartile. Additionally, diet soft drink consumption was associated with an increased risk of diabetes.

4.4 Discussion

4.4.1 Dietary intake

The mean frequencies of food and beverage intake among Canadians aged 6-79 y are shown in **Figure 4-1- Figure 4-4**. The most frequently reported food groups consumed by Canadians were grain products (**Figure 4-1**) and fruit and vegetables. According to beverages categories, fruit and vegetable juice had the greatest contribution which was significantly greater than SSBs and diet soft drink (**Figure 4-2**).
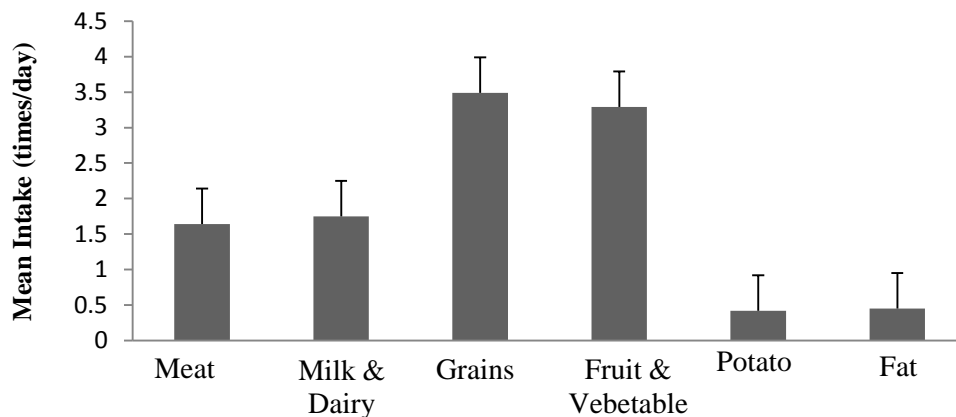
Figure 4-1 Mean frequency of some food groups intake (times/day) among Canadian aged 6-79 y (population size = 29,235,445)
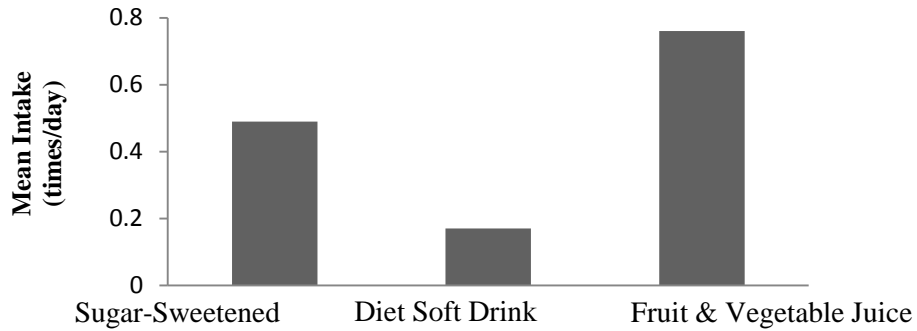
Figure 4-2 Mean frequency of some beverages intake (times/day) among Canadian aged 6-79 y (population size = 29,235,445).

Different age groups had significantly different meat and fish, milk and dairy, grains, fat, SSBs, and fruit and vegetable juice intake. Higher intake of dietary fat and sugary beverages including SSBs and fruit and vegetable juice are a concern in adolescents (**Figure 4-3**). Children and adolescents had significantly greater amount of milk and dairy compared to other age groups and children had greater grains consumption compared to 20-59 y individuals (**Figure 4-4**).
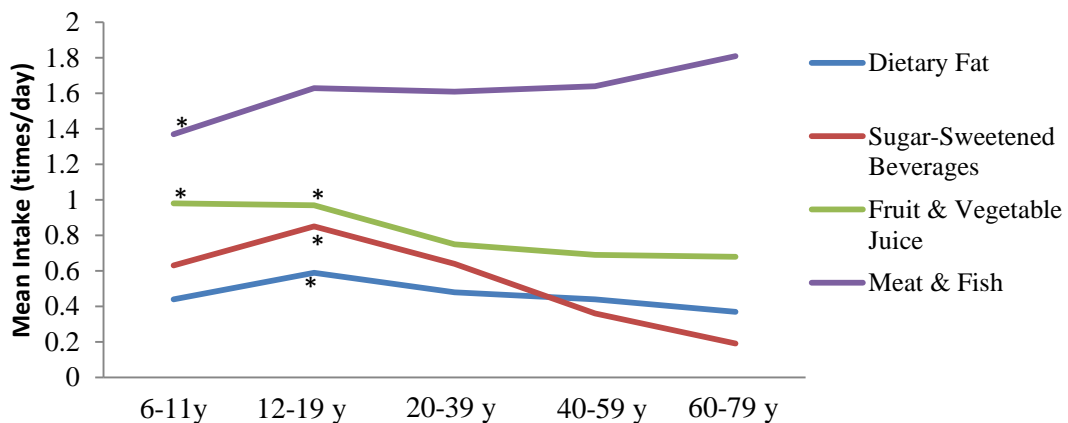
Figure 4-3 Mean frequency of some food groups intake (times/day) among Canadian by age group (population size = 29,235,445). *Indicates the significant difference from the other age groups*.
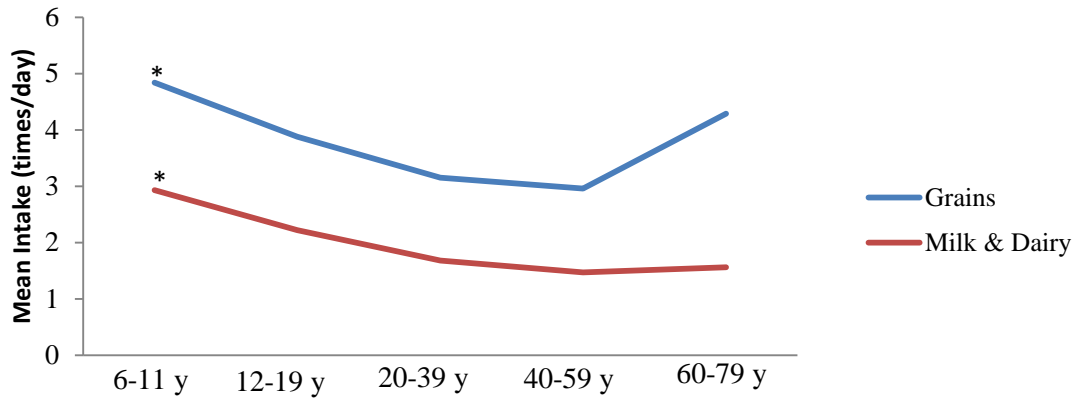
Figure 4-4 Mean frequency of some food groups intake (times/day) among Canadian by age group (population size = 29,235,445). *Indicates the significant difference from the other age groups.*

It appeared that adolescents had a diet high in sugary beverages and fat, although they also had high intake of milk and dairy products. CCHS data revealed that the patterns of beverage intake in Canadian children and adolescents are mainly SSBs. It contributed between 2% and 18% of total energy intake in individuals aged 6-18 y (Danyliw, 2010). Even in Canadian adults, SSBs (fruit drinks and soft drinks) intake was over 50% of total energy intake from beverages (Nikpartow, Danyliw, Whiting, Lim, & Vatanparast, 2012). According to the National Health and Nutrition Examination Survey, American adolescents (12-19 y) had greatest intake of total SSBs (Han & Powell, 2013). The SSBs category was the same as the categories used in the current study including regular soft drink, fruit drinks, and sport/energy drink; however, they used a 24-hour dietary recall (Han & Powell, 2013). They observed that the intake of total SSBs decreased from 1999-2000 to 2007-2008 among adolescents as opposed to children. However, sports/energy drink consumption tripled (4% to 12%) among adolescents. Sports drinks and energy drinks similar to sodas and fruit drinks are the top source of added sugars in the American diet, containing 46% of added sugar intake (Story & Klein, 2012). Thus, to better understand SSB consumption especially among Canadian adolescents, longitudinal data providing trends of each type of SSB consumption is required.

Physically active Canadians had greater intake of milk and dairy, fruit and vegetables, and fruit and vegetable juice compared to inactive individuals. Active individuals may need to be

educated regarding high sugar content of fruit and vegetable juice. People may believe that 100% fruit and vegetable juice is healthy just because it is natural, yet they do not know for example one cup orange juice is approximately the sugar of two to four medium oranges. Similar to the current study, the prevalence of consuming fruits and vegetables five or more times per day was associated with increased level of leisure-time physical activity in American adults (Serdula et al., 2004).

Lower-income individuals had lower intake of fruit and vegetable but higher intake of SSBs. SSBs have low price along with mass marketing which indicates why low-income households tend to buy SSBs more than higher income households (Wang, Coxson, Shen, Goldman, & Bibbins-Domingo, 2012). It has been well established that low-income populations have lower access to fresh fruit and vegetables especially in North America (Hendrickson, Smith, & Eikenberry, 2006; Herman, Harrison, & Jenks, 2006). Six major barriers to fruit and vegetable consumption have been found according to a study in North Carolina: cost, transportation, quality, variety, changing food environment, and changing societal norms on food (Haynes-Maslow, Parsons, Wheeler, & Leone, 2013). Cost is a very important factor as evident when subsidies directed to fresh fruits and vegetables were provided to low-income women in Los-Angeles. The subsidies was almost fully used by this population and a wide variety of fresh fruits and vegetables were purchased (Herman, et al., 2006). Further, residents of four low-income food desert communities in Minnesota expressed concern that healthy food choices were not affordable within their communities and people in their community suffered from food insecurity (Hendrickson, et al., 2006). According to CCHS, among the socio-economic (SES) characteristics, high level of education was observed in lower proportion in the SSBs cluster in men compared to other beverage clusters (Nikpartow, et al., 2012). Thus, income or education is a social determinant that needs to be included in the analysis when SSBs and fruit and vegetable intake are evaluated.

Highly-educated individuals had greater fruit and vegetable juice and lower potato intake than lower-educated individuals. A population-based study in the United States observed that nutrition education of eating five or more servings of fruits and vegetables per day is effective for increasing the intake of fruits and vegetables because few adults are aware of this recommendation and such knowledge is strongly associated with increased intake (Krebs-Smith

et al., 1995). Also, highly educated members in a household could have a positive effect on other members to have a healthy diet. In a systematic review a positive association was observed between parental education and adolescent's fruit, fruit juice, and vegetable intake (Pearson, Biddle, & Gorely, 2009). Thus, providing appropriate nutrition education, such as reducing SSB intake or increasing fruit and vegetable intake should be targeted to specific sex, age and socio-demographic groups.

4.4.2   Abdominal Obesity

4.4.2.1   Descriptive abdominal obesity

The prevalence of abdominal obesity was 35.7% among Canadians aged 12-79 y (n=2173) with a higher prevalence rate in women (40.7% v.s. 30.7%). The elevated estimate in the prevalence of abdominal obesity in Canadian women was similar to other population. The prevalence of abdominal obesity in Spanish individuals aged 18 years and older was 35.5% (31.7% in men and 39.2% in women) (Gutiérrez-Fisac et al., 2012). Also, in Portuguese adults aged 18 years and older the prevalence of abdominal obesity was higher in adult females (37.9%) compared to males (19.3%) (Sardinha et al., 2012). A Report show that the prevalence for the Chinese population is 37.6% (31.1% for men and 43.9% for women,) (Wang et al., 2012). For English adults between 1993 and 2008 abdominal obesity rose from 19.2% to 35.7% in men, and from 23.8% to 43.9% in women (Howel, 2012). Among US men in 2008, the age-adjusted abdominal obesity was 43.7% in men versus 61.8% in women (Ford, Li, Zhao, & Tsai, 2010). In all populations the prevalence rate increased with advancing age.

4.4.2.2   Predictors of abdominal obesity

After adjusting the logistic model the risk of abdominal obesity was associated with older age, low education, inactivity, non-smoking, and high diet soft drink consumption. There was an interaction between age group and dietary fat intake. The detrimental effect of dietary fat on abdominal obesity was significant among 60-79 y individuals.

Certain diseases are more common in lower socio-economic classes such as CVD. This is matched by high prevalence of CVD risk factors including smoking, obesity, physical inactivity, hypertension and hypercholesterolemia (Meier & Ackermann-Liebrich, 2005). The increase

association between odds of abdominal obesity and low-educated individuals, both men and women, was observed in other population-based studies (Li, Ford, McGuire, & Mokdad, 2007; Sardinha, et al., 2012; Wang. et al., 2012). Although the education measurement may be different in studies, the trend is similar.

The inverse association between physical activity and waist circumference has been confirmed in other studies (Kim & Lee, 2009; Sorensen, Smolander, Louhevaara, Korhonen, & Oja, 2000; Van Pelt et al., 1998). Moderate to high intensity exercise of at least eight weeks is effective to reduce abdominal fat. According to a systematic review, aerobic exercise is an effective intervention, however, resistance or interval training are also advantageous especially in older obese individuals with CVD, arthritis, osteoporosis, or mobility disorders (Kay & Fiatarone Singh, 2006). These older individuals may not tolerate moderate to high intensity aerobic training.

The association between abdominal obesity and physical activity may be mediated by food and beverage intake. In a cross-sectional study among Australian adults the association between sedentary activity such as TV viewing and abdominal obesity attenuated after adjustment for food and beverage consumption (Cleland, Schmidt, Dwyer, & Venn, 2008). Another review suggested that increase in physical activity is not related to a corresponding reduction in abdominal obesity in a dose-response manner (Ross & Janssen, 2001). Thus, the intensity and duration of physical activity as well as dietary intake needs to be monitored in order to have an effective management of abdominal obesity. The 2008 physical activity guidelines for Americans suggests two types of physical activity per week to improve health: aerobic and muscle-strengthening (U.S Department of Health and Human Services, 2008).

Smokers had less risk of abdominal obesity compared to non-smokers. This observation is not surprising as it has been well found that smokers generally have a lower BMI than non-smokers and smoking cessation is often associated with weight gain (Filozof, Pinilla, & Fernández-Cruz, 2004; Froom, Melamed, & Benbassat, 1998; Stamford, Matter, Fell, & Papanek, 1986). In the short term, nicotine increases energy expenditure and reduces appetite. Smoking a single cigarette could generate a 3% rise in energy expenditure within 30 min (Dallosso & James, 1983). However, the effect changes by weight status and degree of physical

activity. Risky behaviors such as low degree of physical activity and poor diet are usually associated with smokers especially heavy smokers (Chiolero, Faeh, Paccaud, & Cornuz, 2008). In an observational study in South Korea, a U-shaped association was found between visceral fat and total smoking (Kim et al., 2012). However, the association turned to dose-dependent in current/past smokers. Although smoking had no impact on BMI, smoking could alter the fat distribution among current/past smokers (Kim, et al., 2012). Smoking increases insulin resistance which is associated with central fat accumulation and developing MetS, type 2 diabetes, and CVD. According to the 1998 Korea National Health and Nutrition Examination Survey, current smoking amounts had a significant dose-dependent association with MetS (Oh et al., 2005). When the analyses were confined to current smokers, there was a positive dose-dependent association between current smoking and abdominal obesity. Further, high triglycerides and low HDL-C had a significant dose-dependent association with total pack-years (Oh, et al., 2005). Thus, smokers with low BMI may have metabolically adverse fat distribution resulting from insulin resistance, elevated triglyceride, or reduced HDL-C. Subjects in the current study may be the ones with lower BMI but there is no guarantee they are necessarily healthy in terms of fat distribution.

There was no strong association between dietary intake and having abdominal obesity after multiple adjustments. Diet soft drink consumption was associated with increased risk of abdominal obesity in the full sample and among men. This finding was also seen for other chronic diseases or risk factors in the current thesis including MetS, diabetes, and elevated TC/HDL-C ratio. Individuals with abdominal obesity like individuals with MetS, diabetes, or elevated TC/HDL-C ratio were drinking a greater amount of diet soft drink compared with individuals without abdominal obesity. These individuals are possibly following a special diet as they are aware about their metabolic disorder.

Individuals with abdominal obesity had a significantly lower amount of dietary fat compared to non-abdominally obese individuals probably due to adherence to a special diet. According to the logistic model dietary fat intake indicated a U-shaped association with abdominal obesity and the ORs indicate reduction in the risk of having abdominal obesity (**Table 4-5**). Intake in the second quartile and third quartile (one to less than 5 times/wk) decreased the risk of abdominal obesity compared to the first quartile by 45% and 57%, respectively. Among

females with dietary fat intake in the third quartile, there was a 70% reduction in risk of abdominal obesity compared to those in the first quartile. However, the impact of dietary fat intake was not independent of age. Interaction terms between age and dietary fat intake were significant predictors in the model. Considering the second quartile of dietary fat intake, a higher OR for abdominal obesity was observed for individuals 40-59 y compared to 12-19 y individuals. Considering the third quartile of dietary fat intake, a higher OR for abdominal obesity was observed for individuals 60-79 y compared with 12-19 y individuals. Dietary fat intake of one to less than 5 times/wk was associated with increased risk of abdominal obesity in individuals over 40 years of age. Further explanation regarding dietary fat intake effects is found in chapter 5 of the thesis.

Individuals with abdominal obesity in the current study had lower fruit and vegetable juice intake compared to non-abdominally obese individuals. ORs for fruit and vegetable juice intake in men (**Table 4-5**) indicated a U-shaped association with abdominal obesity. Consumption of 4 to 7 times/wk was associated with decreased risk of abdominal obesity. Studies on fruit and vegetable juice intake are mainly limited to children. Several studies indicated no association between fruit juice intake and anthropometrics in children (Alexy, Sichert-Hellert, Kersting, Manz, & Schoch, 1999; Newby et al., 2004; Skinner & Carruth, 2001). Due to high sugar content of 100% fruit and vegetable juice and concern about gaining weight, the Canadian Diabetes Association recommends having fruit and vegetables more often than juice (Gougeon, Aylward, Nichol, Quinn, & Whitham, 2008). This might have an effect on the amount of juice intake by individuals who are aware of their obesity. Also, individuals with high consumption of fruit juice might be those who are trying to follow a healthy lifestyle. Research on fruit and vegetable juice and obesity still needs further investigation in which factors are controlled properly.

4.4.3   Total/HDL Cholesterol Ratio

4.4.3.1   Descriptive total/HDL Cholesterol Ratio

According to a meta-analysis from 61 prospective cohort studies mostly in Western Europe or North America, the most informative lipid measurement as a predictor of ischemic heart disease mortality was the ratio total/HDL cholesterol rather than lower HDL-C and higher

non-HDL cholesterol and was more than twice as informative as total cholesterol (Prospective Studies Collaboration, 2007).

The prevalence of elevated TC/HDL-C ratio was 20.5% among Canadians aged 20-79 y (n=3430) with a higher prevalence rate in men than women (29.5% vs. 11.7%, respectively). Middle-aged individuals (40-59 y) had the greatest prevalence (25.3%) which was significantly greater compared to 20-39 y Canadians (15.3%). According to the published meta-analysis, there was a weak positive association of stroke mortality with the TC/HDL-C ratio at ages 40-69 y (HR:0·86 [0·74–0·99]) per 1.33 higher TC/HDL-C ratio, but no evidence of an association at older ages (70-89 y)(Prospective Studies Collaboration, 2007). Additionally, in the logistic model middle-aged Canadian women (40-59 y) were at significantly greater risk of having elevated TC/HDL-C ratio compared to 20-39 y Canadian women. The association was not significant for 60-79 y individuals. Thus, elevated TC/HDL-C ratio might be more serious in middle-aged Canadians. This finding was no longer significant among men possibly due to lower sample size in men and women (**Table 4-7**).

4.4.3.2   Predictors of total/HDL Cholesterol Ratio

In the final model abdominal obesity remained as a strong predictor for elevated TC/HDL-C ratio in both men and women. Smokers especially women were at greater risk of having elevated TC/HDL-C ratio. Ever drinking alcohol was associated with reduction in the risk of having elevated TC/HDL-C ratio especially in men. Diet soft drink consumption was associated with increased risk of having elevated TC/HDL-C ratio.

Obesity characterised by intra-abdominal visceral fat is associated with dyslipidemia, cardiovascular disease, and diabetes mediated by insulin resistance (Hamdy, Porramatikul, & Al-Ozairi, 2006; Nieves et al., 2003). Insulin resistance results in glucose intolerance, low HDL-C, elevated triglyceride, and hypertension (Hamdy, et al., 2006). Abdominal obesity, measured through waist circumference, was a strong predictor of elevated TC/HDL-C ratio among Canadian men and women.

Interaction terms between age and abdominal obesity were significant predictors in the model. In individuals without abdominal obesity, OR was greater for elevated TC/HDL-C ratio

in 60-79 y individuals compared to 20-39 y. In individuals with abdominal obesity, the odds ratio was lower for elevated TC/HDL-C ratio in 60-79 y individuals compared to 20-39 y. Thus, presence of abdominal obesity appeared more harmful for younger Canadian individuals to develop elevated TC/HDL-C ratio. The reason might be due to the presence of other risk factors affecting this ratio that were more frequent in older individuals, such as high blood pressure or fasting plasma glucose (**Figure 5-1**).

Interaction terms between milk and dairy product intake and abdominal obesity were significant predictors in the model. It appeared that milk and dairy product consumption could be protective in abdominally obese individuals to reduce the risk of having elevated TC/HDL-C ratio in the amount of 2nd and 4th quartile ($5 <$ intake≤ 9 and Intake ≥ 15 times/wk, respectively). In a review of the evidence investigating the relationship between high fat dairy foods and cardiovascular health, the majority of observational studies have failed to find an association between the intake of dairy products and increased risk of CVD. Saturated fat did not alter the TC/HDL-C ratio because saturated fat increased total cholesterol and LDL-C, but also reduced triglyceride and increased HDL-C (Huth & Park, 2012). Further prospective research using 24-h dietary recall to obtain the usual intake among Canadians might be required. Combing data from cycle 1 and 2 of CHMS may also give a more comprehensive picture of the association between milk and dairy products with TC/HDL-C ratio.

Smoking was associated with a two-fold risk of having elevated TC/HDL-C ratio in the full sample and in women. Cigarette smoking is a substantial risk factor for coronary atherosclerosis (Yasue et al., 2006). Evidence has indicated that smoking contributes to increased triglycerides and reduced HDL-C (Mammas et al., 2003; Oh, et al., 2005) and increased TC/HDL-C ratio (Mammas, et al., 2003). Also, smoking results in reduction in HDL-C level and increase in triglycerides, remnant-like particle cholesterol, and apolipoprotein-B but no change in the levels of LDL-C (Yasue, et al., 2006). The detrimental impact of smoking on lipid profile is expected, however, the effect did not remain significant among Canadian men indicating the importance of other factors in men such as abdominal obesity. Alternatively, the lack of association may be due to the small sample size in CHMS. Also, the odds of having elevated TC/HDL-C ratio was greater among young smoking individuals (20-39 y) probably due to a

higher prevalence of smoking among younger individuals or the presence of other risk factors in older individuals.

Although data on alcohol intake was weak, a protective effect of alcohol intake compared with never drinking alcohol on TC/HDL-C ratio was found in the full sample and women. Light to moderate consumption of alcohol is associated with reduced risk of multiple cardiovascular events (Ronksley, Brien, Turner, Mukamal, & Ghali, 2011). The mechanism of the protective effect of moderate alcohol consumption on CVD is mainly explained through increasing HDL-C (Gaziano et al., 1993; Gordon, Ernst, Fisher, & Rifkind, 1981). According to a meta-analysis of the moderate alcohol intake and lower risk of coronary heart disease an approximate dose of 30 g alcohol/day elevated HDL-C level by 3.99 mg/dl, apolipoprotein A1 by 8.82 mg/dl and triglyceride by 5.69 mg/dl (Rimm, Williams, Fosher, Criqui, & Stampfer, 1999). The association between moderate consumption and increase in triglyceride was weak as the intake of 30 g of alcohol a day reduced the risk of coronary heart disease by 24.7% owing to changes in these markers (Rimm, et al., 1999).

Diet soft drink increased the odds of having elevated TC/HDL-C ratio in the full sample and among men. More explanation regarding diet soft drink will be provided in the MetS section.

4.4.4   Diabetes

4.4.4.1   Predictors of diabetes

After adjusting the logistic model and testing all food group intake and socio-demographic characteristics, older age, abdominal obesity, physical inactivity, never drinking alcohol, low sugar-sweetened beverages (SSBs) intake, and low dietary fat intake were associated with the risk of diabetes.

Abdominal adiposity, a growing clinical and public health problem, was included as a covariate in the models instead of BMI. The greater impact of waist circumference compared with BMI on diabetes mellitus is consistent in studies (Balkau et al., 2007; Carey et al., 1997; Wang, Rimm, Stampfer, Willett, & Hu, 2005). Abdominal obesity directly or indirectly contributes to development of diabetes. Indirectly abdominal obesity is the main component contributing to MetS (Setayeshgar, Whiting, & Vatanparast, 2012) and MetS is considered to be

the main contributor to CVD and diabetes (Brien & Katzmarzyk, 2006; Cameron, et al., 2008). With MetS, the risk of CVD doubles and the risk of diabetes increases fivefold (Alberti, et al., 2006; Grundy, 2008). Directly, abdominal obesity is a risk factor for CVD, dyslipidemia (reduced HDL-C, elevated triglyceride, or elevated LDL-C), hypertension, stroke, and type 2 diabetes (Balkau, et al., 2007; Carr & Brunzell, 2004). Visceral fat is associated with inflammation, unfavorable adipokine profiles, insulin resistance and adverse glucose homeostasis (Fontana, Eagon, Trujillo, Scherer, & Klein, 2007; Shoelson, Lee, & Goldfine, 2006).

Exercise, diet, and medication are the cornerstones in managing diabetes. The risk of having diabetes in the Canadian population was about 2.4 times greater in inactive individuals ($0 \leq$ Energy Expenditure< 1.5 kcal/kg/d) compared with active individuals (EE ≥ 3). According to the Canadian Diabetes Association, "adding more physical activity each day is one of the most important things a diabetic can do to help manage diabetes and improve health" (Canadian Diabetes Association, 2014). The Canadian Diabetes Association recommends 150 min/wk of moderate- to vigorous-intensity aerobic activity and three sessions of resistance activity. More explanation on the type and duration of physical activity to manage diabetes is found in a consensus statement from the American Diabetes Association (Sigal, Kenny, Wasserman, Castaneda-Sceppa, & White, 2006). Exercise helps to reduce the risk of diabetes by reducing weight, decreasing the need for insulin, increasing insulin sensitivity, and thus by helping to improve glucose tolerance (Alberti & Gries, 1988; Brankston, Mitchell, Ryan, & Okun, 2004; Church et al., 2004).

Numerous studies have evaluated the adverse effect of SSBs on type 2 diabetes (Hu & Malik, 2010; Malik, Popkin, Bray, Després, & Hu, 2010; Malik, Popkin, Bray, Després, Willett, et al., 2010; Palmer, et al., 2008; Schulze, Manson, et al., 2004). Weight gain caused by excess calorie intake from SSBs would be the main reason to increase the risk of type 2 diabetes (Malik, et al., 2006). However, according to a recent meta-analysis investigating dietary sugars and cardiometabolic risk, dietary sugars adversely influenced blood pressure and serum lipids independent of the effects on body weight (Te Morenga, Howatson, Jones, & Mann, 2014). Reduced risk of diabetes by having SSBs in the fourth quartile is probably due to the adherence of individuals with diabetes to some recommendations. Patients may try to have less regular soft

drink or fruit and vegetable juice as their intake was significantly lower than individuals without diabetes. Further, they have a greater intake of diet soft drinks which are part of the recommendations.

Ever drinking alcohol compared with not consuming alcohol decreased the risk of having diabetes by about 50% in the current study. Findings from published studies suggest a U-shaped relationship between alcohol and type 2 diabetes (Wei, Gibbons, Mitchell, Kampert, & Blair, 2000). According to two meta-analyses of prospective cohort studies, moderate alcohol consumption (6-48 g/day) reduced the risk of diabetes by 30% (Carlsson, Hammar, & Grill, 2005; Koppes, Dekker, Hendriks, Bouter, & Heine, 2005). However, the risk of type 2 diabetes in heavy drinkers (≥48 g/day) was equal to that in non-consumers (1.04 [0.84–1.29]) (Koppes, et al., 2005). Moderate consumption is recognized to increase HDL-C concentration whereas high consumption elevates body weight, triglyceride, and blood pressure. Another plausible mechanism is through increasing insulin sensitivity due to a U-shaped association between insulin concentration and moderate consumption of alcohol (Koppes, et al., 2005). Heavy consumption of alcohol decreases glucose uptake due to inhibition of insulin secretion or increase in insulin resistance which results in reduction in glucose tolerance in heavy consumers (Wei, et al., 2000). Hence, mild to moderate consumption might be protective for type 2 diabetes. Further research is needed on the type of alcohol.

4.5 Strengths & Limitations

Using CHMS data as a nationally representative dataset and examining such health outcomes at population level is unique. However, several limitations are present as in other population based studies. The cross-sectional nature of the data confined our results to indicate associations, not causal relationships. Also, dietary assessment in CHMS data was based on the semi-quantitative food frequency questionnaire therefore information on diet consumption pertained to the frequency of consumption, and not the amounts consumed.

4.6 Conclusion

Adolescents had the greatest intake of dietary fat and SSBs compared to other age groups. Physically active Canadians had higher milk and dairy, fruit and vegetable, and fruit and

vegetable juice intake compared to inactive Canadians. Higher income had a positive impact on having fruit and vegetables, whereas, education had a positive impact on fruit and vegetable juice intake. Further, highly educated individuals had lower potato intake as opposed to SSBs consumption which was greatest among individuals with some post-secondary education.

It appeared that abdominally obese Canadians might be aware about their metabolic complications and thus tried to follow the recommendations of having less dietary fat and regular soft drinks, and more milk and dairy products. Moderate consumption of dietary fat might be protective for abdominal obesity; however, the detrimental effect of fat was evident in Canadians 40 years of age or older. Diet soft drink consumption appeared to be associated with risk of all three health outcomes especially among Canadian men, yet this observation might be due to greater consumption of diet soft drink among individuals with a known disease. Additionally, consuming milk and dairy products (>5 times/wk) in abdominally obese individuals resulted in having less risk of elevated TC/HDL-C ratio. Being young (20-39 y) and smoking were associated with the risk of elevated TC/HDL-C ratio whereas ever drinking alcohol was associated with decreased risk of elevated TC/HDL-C ratio and diabetes.

CHAPTER 5

STUDY 2. Metabolic Syndrome in Canadian Adults and Adolescents: Prevalence and Associated Dietary Intake

Results from study 1 indicated abdominal obesity was a major risk factor for diabetes and elevated TC/HDL-C ratio. Abdominal obesity is also a major component of metabolic syndrome (MetS). In study 2 the status of MetS was investigated in Canadian adults and adolescents as well as the association with dietary intake controlling for potential covariates.

The condensed version of this chapter (descriptive results) was published in International Scholarly Research Network Obesity Journal

5.1   Introduction

Mortality and morbidity due to cardiovascular disease (CVD) and diabetes are a major public health concern in Canada and worldwide. Metabolic syndrome (MetS) is a clustering of five chronic disease risk factors: abdominal obesity; dyslipidemia (elevated triglycerides (TG); reduced high-density lipoprotein cholesterol (HDL-C) level); hypertension; and elevated fasting blood glucose (FPG)(Grundy, 2008). MetS is considered to be the main contributor to CVD and diabetes (Brien & Katzmarzyk, 2006; Cameron, et al., 2008). With MetS, the risk of CVD doubles and the risk of diabetes increases five-fold (Alberti, et al., 2006; Grundy, 2008). Over the past 17 years (1992 - 2009), the prevalence of MetS in Canadian adults has increased from 14.4% (Brien & Katzmarzyk, 2006) to 19.1% (Riediger & Clara, 2011).

Although dietary intake has been linked to individual components of MetS (Lutsey, et al., 2008; Malik, Popkin, Bray, Després, & Hu, 2010; McKeown, et al., 2004; van Meijl, et al., 2008), dietary behaviour among Canadians with and without MetS has not been compared. The prevalence of MetS among Canadian adults, using different criteria, has recently been reported (Riediger & Clara, 2011). However, the prevalence of MetS in adolescents, as well as defining ethno-specific abdominal obesity in adults, using the most recent criteria, has yet to be investigated. The most prevalent form of the constellation of metabolic abnormalities is found in patients with abdominal obesity (Després, et al., 2008).

The aims of this study was to evaluate the association between dietary intake and MetS considering potential covariates. Additionally, the prevalence of MetS in Canadians (12 – 79 y) as well as the dietary intake of Canadians with and without MetS was determined. As healthy dietary behavior is an important factor in self-management of chronic diseases, it is likely that people diagnosed with conditions such as diabetes adhere to a special diet (Fitzgerald, et al., 2008). Therefore, the dietary intake of Canadians with MetS diagnosed with diabetes was compared to those with MetS with no such diagnosis.

5.2 Materials and Methods

5.2.1 Study population

For the purpose of the study, individuals under the age of 12, non-fasting participants, and pregnant women were excluded. The final un-weighted number of respondents was 2,173.

5.2.2 Metabolic Syndrome definition

For adults (20-79 y), the most recent unified definition was applied. The criteria were established in 2005 by the International Diabetes Federation (IDF) in collaboration with American Heart Association/National Heart, Lung, and Blood Institute (AHA/NHLBI)(Alberti, et al., 2009). The presence of at least three out of the five metabolic risk factors constitutes a diagnosis of MetS: abdominal obesity (**Table 2-1**), elevated TG level ($\geq$1.7 mmol/L), reduced HDL-C level (<1.0 mmol/L in males; <1.3 mmol/L in females), elevated Blood Pressure (BP) (Systolic $\geq$ 130 and/or diastolic $\geq$ 85 mm Hg), and elevated FPG level ($\geq$ 5.6 mmol/L). Individuals who have already been diagnosed as hypertensive, diabetic or those who were using antihypertensive drugs were also included. Abdominal obesity was defined using ethno-specific cut points which are highly recommended by the International Diabetes Federation (IDF) (**Table 2-1**).

In adolescents (12-19 y), age- and sex-specific cut points for each component (except for FPG) developed using the Third National Health and Nutrition Examination Survey (NHANES, 1999 to 2002), was used to identify MetS (Jolliffe & Janssen, 2007). Age- and sex-specific growth curves were created with the Lambda Mu Sigma method and each MetS component growth curve was linked to the corresponding adult ATP and IDF cut point. Each cut point reflects the midpoint of a given year (i.e., for age 12 the cut points represents 12.5 y) from 12 to 19 y and could be used for all adolescents within 1-year age range (**Table 2-3** & **Table 2-4**). In this study, the overall prevalence of MetS was reported using Adult Treatment Panel (ATP III) cut points for abdominal obesity in adolescents due to the lack of any significant difference in the prevalence of MetS using either ATP or IDF criteria.

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5.2.3   Dietary assessment

In CHMS, usual dietary intake was collected through a semi-quantitative food-frequency questionnaire. Dietary intake was collected based on the frequency of daily, weekly, monthly, or yearly consumption. Food groups in CHMS were explained in "general methodology". Validation of questionnaire responses were conducted by experts at the end of each interview completed. A respondent's case file was reviewed and adjusted using notes and remarks made by interviewers. For the purpose of the current study all dietary consumption data were converted into a daily frequency of consumption (times/day).

5.2.4   Diabetes

Having been already diagnosed with chronic diseases such as diabetes might have an impact on dietary intake and such individuals might adhere to a special diet. Therefore, the dietary intake in Canadians identified with MetS and having diagnosed diabetes was compared with Canadians identified with MetS having no diagnosis of diabetes. Data on self-reported diabetes, which was specified as being diagnosed by health professionals, are available in CHMS. This indicator was used to identify the difference in dietary intake between individuals with MetS and diagnosed diabetes, and individuals with MetS who are not diagnosed with diabetes. The latter group consisted of two different subgroups: i) individuals with MetS who had glycated hemoglobin (HbA1c) levels $\geq 6.5\%$ (criterion for diabetes) yet without the knowledge of their disease state, and ii) individuals with MetS and without diabetes, i.e., glycated hemoglobin (HbA1c) levels $< 6.5\%$. In 2008, an international committee with members of the American Diabetes Association, the European Association for the Study of Diabetes, and the International Diabetes Federation suggested using HbA1c (The International Expert Committee, 2009) as an accurate and precise measure for chronic glycemic levels and well-correlated criteria with the risk of diabetes complications in both adults and adolescents.

5.2.5   Statistical analysis

The distribution of the Canadians with and without MetS was assessed as well as the distribution by various socio-demographic characteristics. Similarly no overlap in 95% CI of the

estimates was considered as a statistically significant difference at 0.05. To differentiate the dietary intake of individuals with and without MetS independent sample-t test was used.

The association between dietary intake and MetS was evaluated using a multivariable logistic model. Details on how to get the best model was explained in "general methodology". The outcome variables (MetS) were assessed as binary variables (having MetS=1 versus not having MetS=0) in logistic regression models. IBM SPSS statistics for Windows, version 20 (Armonk, NY) was used for data manipulation, summarizing, categorizing, and creating new variables. STATA/SE 11, StataCorp was used for all statistical analyses. Alpha was set at the level of 0.05 for almost all analyses.

5.3 Results

5.3.1 Descriptive Metabolic Syndrome (MetS)

The sample used to investigate MetS (n=2173) represents 27,043,753 Canadians aged 12-79 y. The prevalence of identified MetS by socio-demographic characteristics is identified in **Table 5-1**. The estimated overall prevalence of MetS for this range of age was 18.31%±1.5 with no significant difference between males and females. The prevalence of MetS was greater in individuals aged 40-59 y and 60-79 y compared with either 12-19 y or 20-39 y. The prevalence of MetS was significantly lower among Canadian households with some postsecondary education or postsecondary graduation compared to both less than secondary education and secondary school graduation. With respect to income categories, individuals from lower middle income category had the most prevalence which was significantly greater compared to the highest income classification. Moreover, the prevalence of MetS was significantly lower among active Canadians compared to moderately active and inactive counterparts.

Table 5-1 Weighted estimates of the prevalence of identified metabolic syndrome by socio-demographic characteristics of Canadians aged 12 to 79 y (population size= 27,043,753)

Characteristics	MetS, estimated prevalence±SE[1]	Confidence Intervals
Sex		
Male	17.2±1.8	13.26-21.25
Female	19.3±1.7	15.59-23.08
Age group[4]		
12 – 19 y	3.5±1.5[*2]	0.20-6.89
20 – 39 y	7.8±1.4[*3]	4.74-10.97
40 – 59 y	21.6±2.5[*]	16.17-27.12
60 – 79 y	40.9±3.5[*]	33.05-48.77
Education level[5]		
< secondary school graduation	39.7±6.9[3*]	24.49-55.06
Secondary school graduation	31.1±4.1[*]	22.03-40.26
Some postsecondary	10.7±3.3[3*]	3. 36-18.16
Postsecondary graduation	14.6±1.4[*]	11.53-17.66
Income level		
Lowest income	19.9±4.6[3]	9.69-30.13
Lower-middle income	29.7±4.3[*]	20.16-39.33
Upper-middle income	20.3±2.2	15.51-25.21
Highest income	14.1±1.5[*]	10.69-17.47
Physical activity[6]		
Inactive	23.0±2.0[*]	18.45-27.55
Moderately active	17.1±1.6[*]	13.55-20.72
Active	8.7±1.5[3*]	5.27-12.29
Alcohol		
Never drink	17.6±4.5[3]	7.57-27.68
Ever drink	18.3±1.5	15.10-21.65
Ethnicity		
Non-white	21.2±3.8[3]	12.70-29.70
White	17.7±1.3	14.84-20.59
Smoking		
Non-smokers	18.4±1.6	14.87-22.04
Smokers	17.7±1.6	14.12-21.41

[*] Statistically significant

[1] SE= Standard Error

[2] Data with a coefficient of variation >33.3%. The user is advised that 3.5% do not meet Statistics Canada's quality standards for this statistical program.

[3] Data with a coefficient of variation from 16.6% to 33.3%.

[4] The prevalence of MetS in individuals 40-59 y and 60-79 y was significantly greater than both 6-11 y and 12-19 y individuals.

[5] The prevalence of MetS was significantly lower in individuals with some postsecondary education or graduation compared with individuals with less than secondary or secondary school graduation

[6] The prevalence of MetS was significantly lower in inactive individuals compared with active or moderately active individuals

Figure 5-1 illustrates the age-specific distribution of MetS components among individuals identified with MetS. Abdominal obesity was the most frequent component of MetS in almost all the age groups (~86% to ~95%). The prevalence nevertheless decreased by advancing age due to high prevalence of the other components in older individuals. In adolescents, reduced HDL and abdominal obesity were almost equally the most prevalent components (94.9% and 96.5%, respectively). Among 60-79 y individuals, elevated blood pressure and abdominal obesity had similar prevalence (~86%). Elevated triglyceride and fasting plasma glucose were more frequent in 20-39 y (83%) and 60-79 y (60.5%) individuals, respectively.
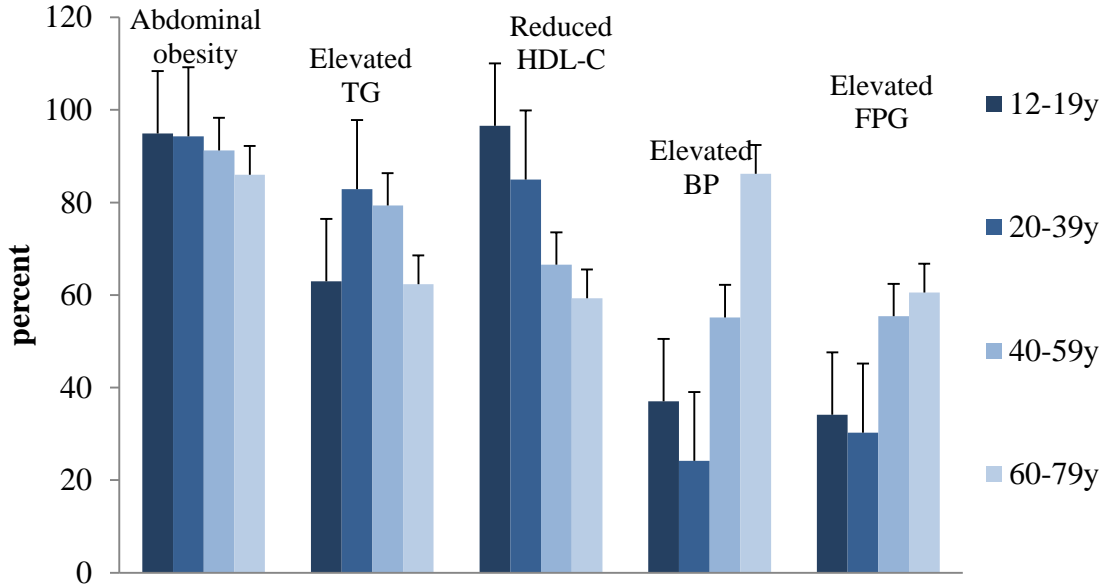
Figure 5-1 Age-specific prevalence of the metabolic syndrome components among individuals identified with metabolic syndrome aged 12 to 79 y (population size=27,043,753). TG = triglyceride, BP = blood pressure, FPG = fasting plasma glucose

Dietary consumption among Canadians aged 12 to 79 y with and without MetS is shown in **Table 5-2**. Canadians with MetS consumed significantly less dairy products, dietary fat, sugar-sweetened beverages, and more diet soft drinks compared to Canadians without MetS. Since the SSBs and dietary fat consumption was lower and diet soft drink intake was greater

among individuals with MetS compared to individuals without MetS, the dietary habits of individuals with known chronic diseases such as diabetes were evaluated. It might be possible that individuals with known diabetes alter their dietary intake and decrease SSBs and fat consumption or increase diet soft drink consumption. Based on the independent sample t-test Canadians with MetS and diagnosed diabetes significantly consumed less fruit and vegetable juice (**Table 5-2**) compared to individuals with no diagnosis of diabetes. Although it was not statistically significant, patients with diagnosed diabetes had more diet soft drinks and less SSBs.

Table 5-2 Dietary consumption among Canadians aged 12 to 79 y with and without metabolic syndrome (MetS, population size=27,043,753)

Food and beverages times/day[1]	Individuals with MetS, Mean±SE[2] (CI[3]) n=426	Individuals without MetS, Mean±SE[2] (CI[3]) n=1746	Individuals with MetS & diagnosed diabetes, Mean±SE[2] (CI[3]) n=71	Individuals with MetS & no diagnosis of diabetes, Mean±SE[2] (CI[3]) n=355
Meat and Fish				
-Red meat, organs, hotdogs, sausage or bacon, seas foods, eggs, beans, and nuts	1.55±0.16 (1.19-1.91)	1.92±0.17 (1.54-2.31)	1.27±0.06 (1.13-1.42)	1.60±0.20 (1.15-2.05)
Grains, Fruit & Vegetable				
-Hot/cold cereal, white bread, brown bread, rice, pasta (grains)	3.6±0.31 (2.91-4.30)	3.18±0.21 (2.72-3.64)	4.62±0.90 (2.63-6.62)	3.41±1.34 (2.65-4.17)
-Fruit and Vegetable	3.61±0.17 (3.2 -3.99)	3.71±0.08 (3.52-3.90)	3.64±0.25 (3.08-4.20)	3.60±0.18 (3.18-4.01)
Milk & Dairy Products				
-Milk, cottage cheese, and yogurt or ice cream	1.38±0.07[*] (1.21-1.54)	1.69±0.05[*] (1.56-1.82)	1.55±0.10 (1.33-1.77)	1.34±0.08 (1.16-1.52)
Dietary Fat				
-Regular-fat salad dressing or mayonnaise and regular-fat potato chips, tortilla chips or corn chips	0.36±0.03[*] (0.29-0.43)	0.48±0.02[*] (0.44-0.53)	0.39±0.14 (0.07-0.71)	0.36±0.02 (0.31-0.40)
Water & Soft Drinks				
-Regular soft drink, sport	0.37±0.04[*]	0.53±0.04[*]	0.20±0.07	0.40±0.05

drink, and fruit drink *(sugar-sweetened beverages)*	(0.27-0.46)	(0.44-0.62)	(0.04-0.35)	(0.28-0.52)
- *Diet soft drink*	0.22±0.03[*]	0.14±0.02[*]	0.47±0.13	0.17±0.03
	(0.16-0.28)	(0.10-0.18)	(0.18-0.76)	(0.09-0.26)
- *Fruit and vegetable juice*	0.70±0.06	0.74±0.04	0.50±0.07[*]	0.74±0.07[*]
	(0.55-0.85)	(0.65-0.82)	(0.34-0.65)	(0.57-0.91)

[*] Intake is statistically significant between individuals with and without MetS or between individuals with MetS and diagnosed diabetes or undiagnosed diabetes based on the independent sample t-test
[1] Frequency of consumption
[2] SE= Standard Error
[3] Confidence Intervals

5.3.2 Association between metabolic syndrome and dietary intake

5.3.2.1 Univariate analysis

The association between MetS and dietary intake was assessed using logistic regression. The outcome was MetS (having MetS=1 and not having MetS=0) and the independent predictors were different food groups and other covariates (socio-demographic characteristics). In order to reach the best model, the steps explained in chapter 3 were implemented. **Table 7** and **Table 8** in **Appendix 3** indicate the univariate association between MetS and each independent predictor. Socio-demographic variables and food groups were included in **Table 7** and **Table 8**, respectively.

Among socio-demographic characteristics, the significant predictors of MetS were older age, low level of education, low income, and low level of physical activity.

All the food groups investigated were significantly associated with the risk of MetS based on the assumption of the p-value <0.2 for univariate analysis (**Table 8, Appendix 3**). However, the significant association was not seen in all quartiles of intake in each food group. Compared to the first quartile, consuming more meat and fish, grains, fat, milk and dairy products, SSBs, and fruit and vegetable juice were associated with reduced risk of MetS. However, compared to the first quartile consuming more fruit and vegetable and diet soft drink were associated with the increased risk of MetS.

5.3.2.2 Multivariate analysis

In the next step all significant univariate analysis were entered into a logistic model and multicollinearity was evaluated. The mean Vif was equal to 1.99 which was less than 2.5 as the threat for multicollinearity. **Table 5-3** indicates the best model of the association between MetS and independent indicators after implementing specification error test.

Table 5-3 Logistic regression analysis indicating the association between metabolic syndrome and the dietary intake after adjusting for the potential covariates[§] (population size=27,043,753)

Food groups	Odds Ratio	P-value	95% Confidence Interval
Age Groups			
12-19 y[1]			
20-39 y	1.69	0.364	0.50-5.77
40-59 y	5.98	0.006[*]	1.88-19.06
60-79 y	17.78	<0.001[*]	6.09-51.85
Physical activity			
Active[1]			
Moderately active	2.52	0.004[*]	1.44-4.39
Inactive	3.38	<0.001[*]	2.04-5.60
Dietary Fat			
1st Q[1]			
2nd Q	0.53	0.048[*]	0.28-0.99
3rd Q	0.63	0.090	0.36-1.09
4th Q	0.61	0.091	0.33-1.10
Nut consumption			
1st Q[1]			
2nd Q	0.77	0.276	0.47-1.26
3rd Q	0.75	0.236	0.46-1.24
4th Q	0.36	0.001[*]	0.22-0.60
Diet Soft Drink[2]	1.37	0.046[*]	1.01-1.87

According to the "linktest" the p-value was <0.05 and 0.413 for variable _hat and variable _hatsq, respectively, indicate that the model fitted well.

[§] Covariates include age, sex, physical activity, ethnicity, education, income, smoking, and alcohol intake.
[*] Statistically significant from the reference group
[1]Reference group
[2] Diet soft drink used as a continuous variable

After adjusting for the potential covariates, age, physical activity, dietary fat intake, nut consumption, and diet soft drink remained statistically significant in the model. Being young, leisure time physical activity, having nut intake in the amount of the fourth quartile, having fat intake in the amount of the second quartile, and having less diet soft drink were associated with reduced risk of MetS.

No interaction or confounder was observed among the variables. Meat and fish intake was statistically significant in the univariate analysis; therefore, each component of the corresponding food group was evaluated individually. Meat and fish group is various in terms of healthy and unhealthy components such as red meat, organs, hotdogs, sausage or bacon, sea

foods, eggs, beans, and nuts. Nut consumption indicated significant association and was included in the model.

In order to have a sensible estimation of intake in each quartile, the quartiles of dietary fat and nut consumption were converted to the frequency of weekly intake (**Table 5-4**). Each time of intake was considered as one serving of consumption according to Canada's food guide.

Table 5-4 The conversion of the quartile of dietary fat and dairy intake into weekly frequency of consumption

Quartile of Intake	Nut intake (times/week)[1]	Dietary fat intake (times/week)[2]
1st Q	Intake ≤ 0.2	Intake < 6
2nd Q	$0.2 <$ intake < 1	$6 \leq$ intake ≤ 11
3rd Q	$1 \leq$ intake < 3	$11 <$ intake ≤ 19
4th Q	Intake ≥ 3	Intake > 19

[1] One serving is equivalent to 60 ml or ¼ cup
[2] One serving is equivalent to 1 tablespoon of salad dressing or mayonnaise or 15-20 chips

5.3.2.3 Summary

The estimated overall prevalence of MetS in Canadians aged 12-79 y was18.3% with no significant difference between males and females. Controlling for the potential covariates, the risk of MetS was explained strongly by age, physical activity, dietary fat intake, nut consumption, and dietary soft drink intake in Canadians 12-79 y. Older age were highly at risk as well as inactive individuals. Consumption of nuts in the 4th quartile was associated with decreased risk of MetS compared to the first quartile. Additionally, every additional time having diet soft drink each day was associated with risk of MetS.

5.4 Discussion

5.4.1 Descriptive metabolic syndrome

The prevalence of MetS was 18.3% among Canadians aged 12-79 y (n=2173) with a higher prevalence rate in older Canadians and with no significant difference between sexes. The prevalence of MetS was lower among physically active Canadians than inactive or moderately active individuals or among Canadians with higher socio-economic status (SES) compared to individuals with lower SES. Abdominal obesity was identified using different ethno-specific cut points for Canadian adults. Considering Canadians identified with MetS, abdominal obesity in all

age groups was the most prevalent component. In adolescents, reduced HDL-C was equally the most prevalent component of MetS along with abdominal obesity.

A similar trend in the prevalence of MetS by different age groups was observed among the US population according to NHANES data (Ford, Giles, & Dietz, 2002; Ford, H. W. Kohl, A. H. Mokdad, & U. A. Ajani, 2005b) wherein, prevalence increased from 6.7% in individuals aged 20- 29 y to ~43% in 60 and over. The greater prevalence of MetS in older adults is not surprising as this group showed higher prevalence of most MetS defining components.

The majority of MetS studies have focused on adults, however identifying MetS risks in adolescents is important in preventing the early establishment of diseases related to MetS. The reason for not including adolescents in such studies is due to the lack of consistent defined criteria of MetS (Alberti, et al., 2009). Using the age- and sex-specific criteria, the prevalence of MetS in American adolescents was 7.6% (Jolliffe & Janssen, 2007) compared to 3.5% in the current study. The greater prevalence in American adolescents is expected to double because the obesity prevalence is twice among American adolescents (18.1%) (Ogden, Carroll, Curtin, Lamb, & Flegal, 2010) compared to Canadian adolescents (9.4%) (Public Health Agency of Canada, 2011).

Including adolescents in the data decreased the previously reported (Riediger & Clara, 2011) overall prevalence of 19.6% (the prevalence in adults over 18 y) to 18.31% due to lower prevalence of MetS in adolescents. Using the same criteria and the same data, the estimated prevalence of MetS in Canadian adults over 18 y was similar to Riediger and Clara's (Riediger & Clara, 2011) report (19.6% vs. 19.1%, respectively) with a 0.5% increase in the current study. Using ethno-specific cut points increased the prevalence of abdominal obesity in each age group by 1 to 4% among adults. Therefore, the 0.5% difference in the prevalence for adults at the population level might be due to using ethno-specific defined abdominal obesity.

Abdominal obesity was the most frequent component of MetS in almost all age groups identified with MetS. Additionally, abdominal obesity was the most prevalent metabolic abnormality not only in individuals identified with MetS but also in the whole population (35.7%) which is consistent with US population data (Ford, et al., 2002; E. S. Ford, W. H. Giles, & A. H. Mokdad, 2004). There is enough evidence supporting the notion that abdominal obesity

is predictive of metabolic risk factors (Després, et al., 2008). It has been previously observed that abdominal obesity is associated with reduced HDL-C and LDL-C size, increased LDL-C number (Pascot et al., 2001; Sam et al., 2008), triglyceride (Pascot, et al., 2001), and insulin resistance (Ascaso et al., 2003; Nieves, et al., 2003). In adolescents, reduced HDL-C was the most prevalent component (96%) similar to American adolescents (Duncan, Li, & Zhou, 2004). One reason could be due to the biological changes in which mean HDL-C levels drop especially among boys over 12 y during puberty in most ethnic groups (Morrison et al., 1979; Porkka, Viikari, & Åkerblom, 1991). Abdominal obesity and reduced HDL-C were the most prevalent components in development of MetS in adolescents. This could help to characterize a strategy to decrease the prevalence of MetS in adulthood.

One of the defining components of MetS in some MetS cases (n=71) was the existence of diabetes. Among MetS cases, although individuals with diabetes consumed greater dairy products, the consumption was not statistically significant from the individuals without diabetes probably due to the small number of MetS cases with diabetes. The most specific dietary recommendations for individuals with diabetes are focused on carbohydrate or sugar, dietary fat, and cholesterol intake (Nöthlings, et al., 2011). Evidence suggests dietary fat quality influences insulin sensitivity and its associated metabolic abnormalities (Nöthlings, et al., 2011). Individuals with MetS consumed less dietary fat compared to individuals without MetS. However, among MetS cases, diabetic individuals did not consume significantly less dietary fat than individuals with non-diabetics. Such finding is comparable to the results from the Multiethnic Cohort Study (MEC) (Nöthlings, et al., 2011) conducted in five ethnic groups (n=13 776). Saturated fat consumption was not significantly different between diabetics and individuals without diabetes. Individuals with MetS had greater diet soft drink and lower regular soft drink intake which might be due to the same trend of intake among diabetic and non-diabetic individuals. There is evidence that diet soft drinks are usually consumed more by individuals who are following other healthy behaviors (Dhingra, et al., 2007). The MEC study (Nöthlings, et al., 2011) observed that diabetic individuals on average consumed 2.6 times more diet soft drinks than individuals without diabetes, whereas the consumption of regular soft drinks was less than half of what individuals without diabetes drank (Nöthlings, et al., 2011). This observation was not statistically significant in the current study, possibly due to the small sub-samples of participants included in the analyses. Moreover, the MEC study and current study are different

by virtue of ethnicity of participants and sample size. Therefore, being aware of the presence of a specific risk factor or chronic disease may have an impact on dietary intake and those patients try to follow the recommendations. In fact, educating Canadian patients with health risk factors or chronic diseases could be very effective.

5.4.2 Predictors of metabolic syndrome

After adjusting the logistic model and testing all food group intake and socio-demographic characteristics, young individuals, physically active individuals, individuals who were in the highest quartile of nut consumption, in the second quartile of dietary fat intake, and individuals who consumed less diet soft drink had reduced risk of MetS.

The association between inactivity and increased risk of MetS has been investigated in several studies (Dunstan, et al., 2005; Ford, Kohl, Mokdad, & Ajani, 2005a; Grundy et al., 2005; Healy et al., 2008; Lakka & Laaksonen, 2007; Pan & Pratt, 2008; Rennie, McCarthy, Yazdgerdi, Marmot, & Brunner, 2003). According to NHANES data from 1999 to 2000, a cross-sectional study among 1626 men and women, demonstrated that not engaging in any moderate or vigorous physical activity approximately doubled the odds of having MetS compared to those engaging in ≥150 min/wk of such activity. Further, after multiple adjustment, sedentary activities including viewing television, videos, or a computer outside of work ≥4 h/d doubled the odds of having MetS over those with <1 h/d of such sedentary activity (Ford, et al., 2005b). In another population-based cross-sectional study of 6,241 Australian adults, total physical activity of ≥150 min/wk decreased the risk of MetS in both men and women after multiple adjustments against <150 min/wk of physical activity (Dunstan, et al., 2005). A cross-sectional study among the European population indicated the benefit of both vigorous and moderate physical activity in reducing the risk of having MetS after multiple adjustment (OR:0.52; CI: 0.40-0.67 and OR:0.78; CI: 0.63-0.96, respectively). However, Adjustment for BMI and resting heart rate substantially attenuated the association indicating the reduced BMI and increased cardiovascular fitness as important mediators (Rennie, et al., 2003).

Physical activity is an important component to manage MetS. Several mechanism related to the effect of physical activity on MetS were reviewed by Lakka and Laaksonen (Lakka & Laaksonen, 2007). Regular physical activity prevents gaining weight. Also exercise can improve

insulin sensitivity by increase in translocation of the GLUT 4 glucose transporter to the cell surface. Exercise, especially endurance exercise, can contribute to increase HDL-C. Aerobic exercise decreases systolic and diastolic blood pressure in normal and hypertensive individuals with slightly greater benefit in hypertensive individuals. Furthermore, according to cross-sectional studies, the concentration of inflammatory markers is lower in physically active or fit individuals than in inactive or unfit individuals. According to Canadian physical activity guidelines, 18-64 y adults and 5-17 y children and adolescents should participate in 150 and 60 minutes of moderate- to vigorous-intensity aerobic physical activity per week, respectively (Canadian Society for Exercise Physiology, 2011). For individuals with MetS the benefit of physical activity is greater when they go beyond the above recommendation. Sixty minutes or more of continuous or intermittent aerobic activity on almost every day, will improve risk factors such as obesity, elevated fasting plasma glucose, and high blood pressure (Thompson et al., 2003).

Consuming more diet soft drink significantly increased the risk of having MetS (OR:1.37). One reason might be due to greater consumption by individuals with known disease which was explained in the previous section. However, data were consistent with a cohort study of stroke incidence and risk factors (Gardener et al., 2012) as well as the Multi-Ethnic study of atherosclerosis. The corresponding studies evaluated risk of incident MetS, its components, and type 2 diabetes (Nettleton, et al., 2009). In the first article, those who drank diet soft drinks daily had a 43% increased risk of vascular events as compared to those who did not drink diet soft drinks adjusted for demographics, behavioral risk factors, daily diet, BMI, and vascular risk factors. In contrast, consumption of regular soft drinks was not associated with an increased risk for vascular events in multivariable-adjusted analyses. The corresponding article also suggests that individuals with elevated risk of vascular disease may consume diet soft drinks to reduce calories and sugar and lose weight (Gardener, et al., 2012). In the Multi-Ethnic study of atherosclerosis compared with non-consumers, the risk of MetS was 36% greater in those consuming ≥1 serving of diet soda daily after adjustment for demographic characteristics and energy intake. With further adjustment for waist circumference and BMI the association was no longer significant. Daily consumers of diet soda had a 67% elevated risk of type 2 diabetes compared with non-consumers which remained significant after adjustment for BMI and waist circumference. Data showed no significant associations between sugar-sweetened soda

consumption and risk of either MetS or type 2 diabetes (Nettleton, et al., 2009). Further, consuming ≥1 daily serving of diet soda had a significantly greater risk of developing high waist circumference e (≥102 cm if male and≥88 cm if female) or high fasting glucose (≥100 mg/dl) during follow-up. Despite the existence of these associations caution is required not to conclude causality between diet soda and diabetes, MetS, elevated TC/HDL ratio, and abdominal obesity. There are three hypotheses that need to be assessed: first, the calorie-free artificial sweeteners may increase desires for more energy-dense foods; second, substituting diet beverages for sugar-sweetened beverages may results in overconsumption of other foods or beverages; third, individuals with known chronic conditions or risk factors may adhere to consumption of less sugar-sweetened beverages. In fact, research on diet soda's relationship to diseases or risk factors raises more questions than it answers which requires further investigation.

In observational and intervention studies increased nut consumption has been associated with reduction in various mediators of chronic diseases. The mediators are oxidative damage to protein (Jenkins et al., 2006; Torabian, Haddad, Rajaram, Banta, & Sabaté, 2009), inflammation (Jiang et al., 2006), abdominal obesity, hypertension, low HDL-C, high fasting glucose and a lower prevalence of MetS (O'Neil, Keast, Nicklas, & Fulgoni, 2011), serum lipid in diabetics (Jenkins et al., 2011), insulin resistance (Casas-Agustench et al., 2011), and endothelial dysfunction (Ma et al., 2010). In two large independent cohorts of Nurses' Health study (1980–2010) and Health Professionals Follow-up study, the frequency of nut consumption was inversely associated with total and cause-specific mortality, independently of other predictors of death (Bao et al., 2013). However, In a randomized, parallel, controlled study design compared to the control diet both the walnut and the unsalted cashew nut intervention diets had no significant effect on the HDL-C, triglyceride, total cholesterol, LDL-C, serum fructosamine, serum high-sensitivity C-reactive protein, blood pressure and serum uric acid concentrations (Mukuddem-Petersen, Jerling, Hanekom, & White, 2007). The lack of effect was explained due to low total cholesterol and LDL-C concentrations of the subjects at baseline. In the current study consuming nuts ≥3 times/wk decreased the risk of having MetS by 64%. Casas-Agustench et al., 2011 found that daily intake of 30 g mixed nuts decreased lipid responsiveness in patients with MetS. In a systematic review consumption of 50-100 g (1.5-3.5 serving) of nuts ≥5 times/wk significantly decreased total cholesterol and LDL-C as heart related risk factors (Mukuddem-Petersen, Oosthuizen, & Jerling, 2005). In the current study consuming even ≥3 times/wk of

nuts in the Canadian population decreased the risk of having MetS. However, the amount of consumption is not fully comparable to other studies due to lack of reporting the amount of consumption in CHMS data. Further research reflecting the exact amount of consumption may give a more accurate estimation among Canadians.

Patients with MetS may have low antioxidant status (Ford, Mokdad, Giles, & Brown, 2003). Nuts have a protective effect against coronary heart disease and MetS due to its high antioxidant content (Davis et al., 2007). In fact, nuts are nutrient-dense foods and rich in unsaturated fatty acids, fiber, vitamins, minerals, and phenolic antioxidants and phytosterols (Bao, et al., 2013).

Dietary fat (mostly saturated and probably some unsaturated in salad dressings) in the second quartile decreased the risk of having MetS by 47% compared to the first quartile. The type of dietary fat intake cannot be fully stratified due to limitation in data which makes the interpretation difficult. Types of dietary fat are related to components of the metabolic syndrome. Studies indicated the association of saturated fatty acids and trans fatty acids with increase in the serum LDL-C concentration (Grundy, Abate, & Chandalia, 2002; Robitaille, Després, Pérusse, & Vohl, 2003) and insulin resistance (Riccardi, Giacco, & Rivellese, 2004; Vessby, 2003) as opposed to the association of unsaturated fatty acid with low prevalence of CVD and type 2 diabetes (Grundy, et al., 2002; Warensjö, Risérus, & Vessby, 2005). Since the association was not very strong even for the second quartile of intake in the current study further research with a valid dietary assessment tool is required among the Canadian population.

5.5   Strengths & Limitations

The fasted sub-sample used in our study was smaller than the whole CHMS sample. To generalize data to the Canadian population, we used the specific weights provided in CHMS for the fasted subsample. Although CHMS collected usual dietary intake through semi-quantitative questionnaire, the quantity of the consumption is not provided to obtain an exact measure of dietary intake.

5.6   Conclusion

The prevalence of MetS among Canadians age 12 to 79 y was 18.3%, with the lowest prevalence in the youngest group (3.5%) and the highest among those 60-79 y (41%). Abdominal obesity in all age groups and reduced HDL-C in adolescents were the major components to define MetS in the Canadian population. The prevention strategy for the Canadian population needs to consider those individuals who are less educated or who are less active or both. Additionally, to define abdominal obesity it would be more precise to implement ethno-specific cut points. According to previous studies and the current study nut consumption ≥3 times/wk may be beneficial to decreases the risk of having MetS. Further investigation is required with respect to the association between the increase risk of MetS and high diet soft drink consumption.

CHAPTER 6

STUDY 3. Prevalence of 10-Year Risk of Cardiovascular Diseases and Associated Risks in Canadian Adults: The Contribution of Cardiometabolic Risk Assessment Introduction

Cardiovascular disease is the main consequence of MetS. With Mets the risk of developing CVD doubles. Study 3 focused on how perfectly to predict 10-year risk of CVD in order to have a more accurate tool to assess the risk. In this study 10-year risk of CVD is obtained using the most recommended assessment tool, Framingham Risk Score as well as considering individuals with MetS. Changing the classification of individuals with MetS to a higher risk level is the new concept which has been explored at the population level in the current study. Dietary intake of individuals with different levels of CVD risk was also assessed.

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6.1   Introduction

Cardiovascular diseases (CVDs) are the leading cause of death in Canadian men and women (Public Health Agency of Canada, 2010). In 2008, CVD accounted for 29% of all deaths in Canada (28.0% in males and 29.7% in females) (Heart and Stroke Foundation, 2012). Over $20.9 billion is spent each year in Canada for physician services, hospital costs, lost wages, and decreased productivity related to heart disease and stroke. Population-based strategies in assessing the risk of CVD and cost-effective interventions would play a fundamental role in decreasing rates of morbidity and mortality from CVD. The Framingham Risk Score (FRS), developed through Framingham Heart Study in 1971 to 1974, is the most commonly recommended assessment tool for evaluating 10-year risk of CVD in the United States (Sheridan, et al., 2003).The National Cholesterol Education Program(Adult Treatment Panel III) recommends using FRS to assess 10-year risk of CVD (Wilson, D'Agostino, et al., 1998). The FRS has been validated for the Canadian population by a committee of clinicians and researchers (Sheridan, et al., 2003). However, increasing evidence has indicated that CVD risk algorithms such as FRS may underestimate an individual's actual risk and postpone the initiation of appropriate intervention probably due to not considering waist circumference and triglycerides (Leiter, et al., 2011a, 2011b). Metabolic syndrome (MetS) is a clustering of five chronic disease risk factors, including abdominal obesity, dyslipidemia (elevated triglycerides (TG) and reduced high-density lipoprotein cholesterol (HDL-C) level), hypertension, and elevated fasting plasma glucose (FPG) (Grundy, 2008). MetS is considered to be the main contributor to CVD and diabetes (Brien & Katzmarzyk, 2006; Grundy, 2008). Regardless of ethnic diversity, the risk of CVD doubles with MetS; also, the risk of diabetes increases fivefold (Dekker, et al., 2005; Galassi, et al., 2006; Gami, et al., 2007; Stern, et al., 2004). According to the previous study, 18.3% of Canadians aged from 12 to 79 y had MetS (Setayeshgar, et al., 2012). However, an increase in relative risk of CVD cannot be used to evaluate absolute risk. Furthermore, reported relative risk of CVD and its association with MetS are not comparable between studies as not all studies take into consideration potential confounders.

The new approach on assessing "cardiometabolic risk (CMR)" or "global cardiometabolic risk" considers the factors that go beyond the traditional risk factors. In 2009, the Canadian Cardiometabolic Risk Working Group suggested that CMR represents the

comprehensive catalogue of factors related to CVD and type 2 diabetes (Leiter, et al., 2011a, 2011b). Therefore, in evaluating CMR, both MetS and 10-year risk of CVD are considered. The calculation of FRS followed by the evaluation of the presence or the absence of MetS helps identify individuals whose risk might be underestimated. Therefore, in order to evaluate CMR, both the risk factors of MetS and the risk factors used to calculate 10-year risk of CVD are considered. This novel approach, CMR, has been used to identify the risk of CVD at the individual level; however, to my knowledge no nationally representative study has used this method to evaluate the risks at the population level. Moreover, it is not understood if calculating CMR could possibly change the prevalence of the 10-year risk of CVD at a population level. Different dietary patterns impact cardiometabolic components in different ways. The Dietary Approaches to Stop Hypertension (DASH) diet or Mediterranean diet are inversely associated with cardiometabolic abnormalities (Obarzanek et al., 2001; Panagiotakos, Pitsavos, & Stefanadis, 2006; Sacks et al., 2001). Similarly, coronary heart disease is positively associated with the Western diet as opposed to the prudent diet (Mente, de Koning, Shannon, & Anand, 2009). Previous findings from CHMS indicate that Canadians with MetS consumed less dairy products, sugar sweetened beverages, and dietary fat, but had a greater intake of diet soft drinks (Setayeshgar, et al., 2012). The dietary behavior of Canadians at different levels of CVD risk has yet to be explored. The first objective of the present study was to estimate the 10-year risk of CVD in Canadian adults and potential changes at the population level when CMR is taken into account. The second objective was to determine 10-year risk of CVD in Canadian adults by different socio-demographic characteristics. Further, the dietary intakes of Canadians in risk categories of 10-year risk of CVD were determined.

6.2   Materials and Methods

6.2.1   Study Population

Data from the Canadian Health Measures Survey (CHMS), Cycle 1, 2007–2009, conducted by Statistics Canada in partnership with Health Canada and the Public Health Agency of Canada was used. CHMS is a nationally representative survey collecting health indicators among a sample of approximately 5,500 Canadians aged 6 to 79 y (representative of 96.3% Canadians through multi-stage sampling strategy). The survey consists of two stages: the first

stage is self-reported data collection through interviews, and the second stage consists of taking direct physical measurements at Mobile Examination Centers (MEC). Individuals living on reserves or in other aboriginal settlements in the provinces, remote areas, institutional residents, and full time members of the Canadian Forces were excluded from the survey. The sampling weights, provided by Statistics Canada, were calculated by multiplying the selection weights for collection sites and the selection weights for dwellings (obtained from 2006 Census of Canada), adjusted for nonresponse (Giroux, 2007).The final individual weight was obtained after converting the household weights followed by adjustment for nonresponse at the interview stage and the MEC stage. For the purpose of the study, individuals who were either under the age of 30 years or over the age of 74 years, as well as non-fasting subjects, pregnant women, and those with confirmed diagnosis of heart disease were excluded. The final number of respondents for the current study was 1,293.

6.2.2   Creating Variables of Interest Using the CHMS Dataset

Using either LDL-C or Tchol is optional in assessing 10-year risk of CVD through FRS (Sheridan, et al., 2003). LDL-C could be found in only the fasted CHMS subsample; whereas, Tchol was available in both fasted and non-fasted subsamples. For the current study the fasted subsample was used and both LDL-C and Tchol were evaluated. Daily smokers, occasional smokers, and those who stopped smoking less than a year ago were classified as smokers. To recognize individuals with undiagnosed diabetes, Glycated Hemoglobin (HbA1c) $\geq 6.5\%$ was used as the criteria (The International Expert Committee, 2009). To obtain the total number of diabetics, cases of self-reported diabetes were added to the individuals with undiagnosed diabetes. Other variables such as age, HDL-C, and systolic and diastolic blood pressure were used to obtain the risk score. All variables were categorized according to the corresponding cutoffs defined in FRS. 10-year risk of CVD was reported using two approaches: (1) 10-year risk of CVD by implementing LDL-C and (2) 10-year risk of CVD by implementing Tchol. Both LDL-C and Tchol cut-points are specified in FRS.

6.2.3   Calculating CMR

To include the CMR in the estimation of 10-year CVD risk, first individuals with MetS were identified. The most recent unified definition established in 2005 by the International

Diabetes Federation (IDF), in collaboration with American Heart Association/National Heart, Lung, and Blood Institute (AHA/NHLBI), for adults was applied. The presence of at least three of the following five metabolic risk factors constitutes a diagnosis of MetS: abdominal obesity (**Table 2-1**), elevated TG level ($\geq$1.7mmol/L), reduced HDL-C level (<1.0 mmol/L in males; <1.3 mmol/L in females), elevated blood pressure (BP) (systolic $\geq$ 130 and/or diastolic $\geq$ 85mmHg), and elevated FPG level ($\geq$5.6mmol/L). IDF's recommendation by using ethno-specific cutoffs for waist measurement (Alberti, et al., 2009) was used. Individuals who have already been diagnosed as hypertensive, diabetic, or those who were using antihypertensive drugs were also included. In the next step, the 10-year CVD risk among individuals with MetS was multiplied by 1.5 (Leiter, et al., 2011a).

6.2.4   Prevalence of CMR by Levels of Socio-demographic Characteristics

The age- and sex-specific groups in the analysis were males and females aged 30–34 y, 35–39 y, 40–44 y, 45–49 y, 50–54 y, 55–59 y, 60–64 y, 54–69 y, and 70–74 y. Four levels of education based on the highest level achieved by any member of the household were defined as less than secondary school graduation, secondary school graduation, and some postsecondary, or postsecondary graduation. Four economic status levels were based on the total household income and the number of individuals in the household. Only two ethnic groups, that is, White and non-White, were created due to few numbers of non-White individuals in various ethnic groups in that category. Physical activity was measured in CHMS using a questionnaire which calculated the total daily leisure time energy expenditure (EE) values (kcal/kg/day) during leisure time activities. Respondents were subsequently categorized into "active" (EE $\geq$ 3), "moderate" (1.5 $\leq$ EE <3), or "inactive" (0 $\leq$ EE < 1.5) physical activity

6.2.5   Dietary Assessment in Three Levels of Risk

In CHMS, usual dietary intake was collected through a semi-quantitative food-frequency questionnaire. More explanation on the assessment of dietary intake can be found in "general methodology" section 3.2.2.

Tertile categorization was used for the achieved FRS scores. Participants were classified in three levels of risk: low, medium, and high. The low number of individuals at high risk level

resulted in wide confidence intervals for estimates. Thus, the comparison in dietary intake was interpreted between two categories of low and medium.

6.2.6   Data Analysis

To meet the first objective, the 95% confidence intervals (CI) of 10-year risk of CVD and CMR estimates were compared. No overlap in 95% CI of the estimates was considered a significant statistical difference at 0.05 (Schenker & Gentleman, 2001). The difference in 10-year risk of CVD by sex, age, and each socio-demographic characteristic was assessed by running independent sample t-tests or by looking at the overlap in 95% confidence intervals. At the population level, data were weighted and bootstrapped and comparisons across groups were performed using 95% CI overlap. To examine possible differences between dietary intakes across three levels of 10-year risk of CVD (Objective 3), the same method of using 95% weighted CI overlap was implemented. Data manipulation, cleaning, and creation of new variables were done using PASW Statistics 19. All statistical analyses were conducted by STATA/SE 11, StataCorp. As per Statistics Canada's recommendation, all analyses were weighted and bootstrapped in order to be representative of the Canadian population. The degrees of freedom of 11 in CHMS cycle 1 were limited due to sampling structure. Alpha was set at 0.05.

6.3   Results

6.3.1   10-Year Risk of CVD in Canadian Adults

The CHMS participants ($n = 1, 293$) represent 17,250,853 Canadians aged 30–74 y. Overall, the risk of CVD in Canadian adults using the LDL-C indicator and Tchol was 8.10%±8.89 (CI: 7.33–8.87) and 7.70%±8.75 (CI: 7.07–8.32), respectively. The corresponding risk when CMR was accounted for was 9.86%±12.93 (CI: 8.71–11.01) using the LDL-C indicator and 9.41%±12.66 (CI: 8.43–10.39) using the Tchol indicator. Using LDL-C or Tchol for either CMR or 10-year risk of CVD did not show any significant difference. The 10-year CVD risk was significantly greater using CMR compared to the 10-year risk of CVD based on only FRS (using Tchol indicator). However, the estimate was not significantly greater when LDL-C was used as an indicator. In the current study, the risk of CVD considering different

socio-demographic characteristics or dietary intake was reported using CMR with the LDL-C indicator.

6.3.2 Socio-demographic Characteristics of 10-Year Risk of CVD in Adults

The risk of CVD was not significantly different across sex groups (**Table** 6-1). Risk of CVD increased significantly by increase in age and decrease in the levels of education and physical activity (**Table 6-1**). Individuals from the highest-income level households had significantly less CVD risk compared to those from upper-middle income families. The risk was greater in inactive individuals compared with active individuals. In addition, the risk was greater in smokers and white Canadians compared to nonsmokers and non-white Canadians, respectively (**Table 6-1**).

Table 6-1 Weighted estimates of 10-year risk of CVD or Cardiometabolic risk (CMR) by socio-demographic characteristics of Canadians aged 30 to 74 y, Canadian Health Measures Survey, Cycle 1, 2007-2009 (population size=17,250,853)

Characteristics	10-year risk of CVD, percent±SE[†]	95% Confidence Interval
Sex		
Male	8.72±0.43	7.77-9.67
Female	10.92±1.07	8.56-13.28
Age[*1]		
1) 30-34 y	1.95±0.10	1.72-2.17
2) 35-39 y	2.95±0.40	2.07-3.84
3) 40-44 y	5.42±0.59	4.11-6.73
4) 45-49 y	6.02±0.39	5.15-6.88
5) 50-54 y	11.91±2.21	7.03-16.79
6) 55-59 y	14.32±1.80	10.34-18.30
7) 60-64 y	19.83±2.07	15.26-24.40
8) 65-69 y	20.68±0.94	18.60-22.75
9) 70-74 y	24.68±2.93	18.21-31.14
Education level[2]		
Less than secondary school graduation	19.35±3.69[*]	11.20-27.49
Secondary school graduation	14.85±1.52[*]	11.49-18.20
Some postsecondary	8.76±0.78[*]	7.04-10.49
Postsecondary graduation	8.50±0.44[*]	7.52-9.47
Income level		

Lowest income	11.10±3.04	4.39-17.81
Lower-middle income	12.06±1.72	8.26-15.86
Upper-middle income	11.34±0.81[*]	9.53-13.14
Highest income	8.10±0.49[*]	7.01-9.20
Physical activity		
Inactive	10.80±0.78[*]	9.06-12.54
Moderately active	9.66±0.66	8.19-11.13
Active	7.46±0.52[*]	6.30-8.63
Alcohol		
Never drink	10.74±2.42	5.41-16.08
Ever drink	9.81±0.48	8.74-10.87
Ethnicity		
Non-white	7.32±0.89[*]	5.35-9.30
White	10.32±0.53[*]	9.15-11.49
Smoking		
Non-smokers	8.87±0.32[*]	8.15-9.60
Smokers	13.18± 1.52[*]	9.83-16.53

[†] SE= Standard Error
[*] Statistically significant
[1] The risk of CVD in age group2 and 3 were significantly different. Age group 5, 6, 7, 8, and 9 were significantly different from age group 1, 2, 3, 4. In addition, age group 9 and 8 were significantly different from age group 5.
[2] The risk of CVD was significantly less among Canadians with some post-secondary education or postsecondary graduation compared to Canadians with less than secondary school graduation or secondary school graduation

6.3.3 Dietary Intake and 10-Year Risk of CVD

Most Canadian adults were in the low CVD risk level (94.4%) (**Figure 6-1**). The only significant association was observed in fruit and vegetable juice intake; Canadians at medium risk of CVD consumed less amounts of fruit and vegetable juice compared to their counterparts at low risk (**Table 6-2**).
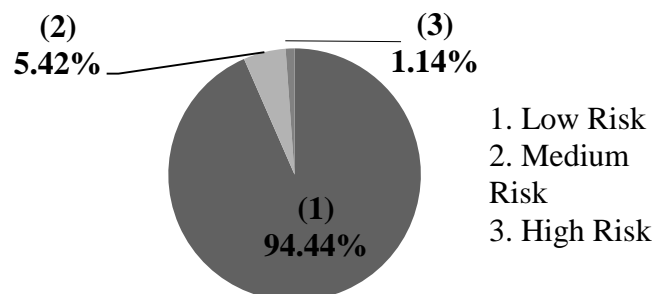
Figure 6-110-year risk of CVD in Canadian adults, 30-74 y, Canadian Health Measures Survey (CHMS), cycle 1, 2007-2009

Table 6-2 Dietary consumption among Canadians aged 30 to 74 y at different level of 10-year risk of CVD, Canadian Health Measures Survey, Cycle 1, 2007-2009 (population size=17,250,853)

Food and beverages times / day[1]	Low 10-year CVD risk (n=1190)	Medium 10-year CVD risk (n=84)	High 10-year CVD risk (n=19)
	Mean of intake±SE[2] 95% Confidence Interval		
Meat and Fish			
-Red meat, organs, hotdogs, sausage or bacon, seas foods, eggs, beans, and nuts	1.81±0.13 1.50-2.11	1.31±0.08 1.12-1.49	1.95±0.42 1.01-2.89
Grains, Fruit & Vegetable			
-Hot/cold cereal, white bread, brown bread, rice, pasta (grains)	2.87±0.13 2.56-3.18	3.76±1.24 1.02-6.50	3.35±1.84 -0.71-7.41
-Fruit and Vegetable	3.78±0.08 3.59-3.98	3.65±0.18 3.25-4.05	3.95±0.45 2.94-4.96
Milk & Dairy Products			
-Milk, cottage cheese, and yogurt or ice cream	1.52±0.05 1.41-1.63	1.59±0.18 1.17-2.00	1.44±0.22 0.94-1.95
Dietary Fat			
-Regular-fat salad dressing or mayonnaise and regular-fat potato chips, tortilla chips or corn chips	0.42±0.02 0.38-0.47	0.37±0.07 0.21-0.53	0.37±0.08 0.18-0.55
Water & Soft Drinks			
-Regular soft drink, sport drink, and fruit drink (sugar-sweetened beverages)	0.39±0.05 0.28-0.50	0.22±0.05 0.11-0.34	0.15±0.06 0.01-0.29
- Diet soft drink	0.16±0.01 0.12-0.20	0.31±0.11 0.06-0.55	0.39±0.17 0.01-0.77
-Fruit and vegetable juice	0.71±0.03[*] 0.64-0.79	0.43±0.09[*] 0.23-0.63	1.46±0.78 -0.26-3.19

[*] Statistically significant
[1] Frequency of consumption
[2] SE= Standard Error
[3] Confidence Intervals

6.4 Discussion

Overall the 10-year risk of CVD in Canadian adults was 9.86% considering individuals identified with MetS. The risk of CVD increased with age or by a decrease in the level of education (**Table 6-1**). The CVD risk was greater among upper-middle income households compared to higher-income households. Additionally, active Canadians with EE≥3 kcal/kg/day had significantly lower risk compared to their inactive counterparts with 0≤EE<1.5. Canadians with medium risk of CVD consumed significantly less fruit and vegetable juice compared to Canadians with low risk of CVD.

The introduction to the concept of Cardiometabolic Risk (CMR) and its risk factors which are not included in traditional risk assessment tools such as FRS, as well as the guidelines for identification and management, was published in a position paper by the Canadian Cardiometabolic Risk Working Group, in 2009 (Leiter, et al., 2011b). The authors believe that traditional risk assessment tools solely underestimated the absolute risk of CVD at an individual level. According to this concept, the absolute CVD risk can be obtained from the algorithms validated by the observational cohort studies (e.g. FRS) taking into account individuals identified with MetS. MetS imparts a relative increase in risk of CVD about 1.5- to 2-fold.

Evaluating the 10-year risk of CVD is usually conducted in prospective studies. Similar to the current study another study among American adults (Ford, Giles, & Mokdad, 2004) used a cross-sectional design which might be considered as a limitation. The estimate of 9.86% of 10-year CVD risk among the Canadian adult population in the current study was similar to American and European prospective studies (McNeill et al., 2005; Menotti, Lanti, Puddu, & Kromhout, 2000). Approximately 9.05% of the 12,089 black and white middle-aged individuals participating in the Atherosclerosis Risk in Communities (ARIC) study after 11 years of follow-up, developed ischemic stroke and Coronary Heart Disease (CHD) (McNeill, et al., 2005). The corresponding prevalence in the European cohorts comprised of men aged 40–59 y, after 10 years of follow-up, was 9.69% for fatal or non-fatal CVD (Menotti, et al., 2000). Additionally, the earlier study among Americans indicated 11.41% incidence of CHD after 12 years of follow-up through Framingham Heart Study which shows a decrease in the incidence of CVD among Americans over time (Wilson, D'Agostino, et al., 1998). Therefore, since the reports from

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prospective studies were similar to the current finding, it might be possible to use the estimate of 10-year risk of CVD through cross-sectional studies which are more feasible than prospective studies.

Findings suggest including CMR in assessing the risk of CVD not only at the individual level but also at the population level. The similar estimates using Tchol and LDL-C indicated the importance of a simple measure of Tchol in this assessment at the population level. Assessing CMR by using a simple measure of Tchol avoided the underestimation of CVD risk in individuals with MetS. A small overlap in the 95% confidence interval of the two estimates was observed when LDL-C was used, which likely relates to the small sample size.

Older age was considered as one of the strong factors in elevating the risk of CVD. The greater CVD risk in older adults is not surprising as this group usually have higher prevalence of most CVD defining components compared to other adults. A similar finding was seen among American adults 20 years and older who participated in a cross-sectional sample of 5,440 in the National Health and Nutrition Examination Surveys (NHANES) 1999-2004 (Wildman et al., 2008). According to NHANES the prevalence of metabolic abnormalities increased with age among all individuals. In this study they used elevated blood pressure; elevated triglyceride and glucose levels; insulin resistance; systemic inflammation; and decreased HDL-C level as cardiometabolic abnormalities which were comparable to the current study.

Physical activity in the current study was self-reported. The degree of activities were converted into energy expenditure during leisure time. Specifically, the significant difference in CVD risk between active and inactive individuals was similar to the finding of a Multiple Risk Factor Intervention Trial (Leon, Connett, Jacobs, & Rauramaa, 1987). Among 12,138 middle-aged men measuring self-selected Leisure-Time Physical Activity (LTPA), combined fatal and non-fatal major CHD events were 20% lower with high activity as compared with low LTPA. Moreover, better cardiometabolic characteristics such as lower heart rate, lower body mass index and a higher HDL-C level or lower risk of CHD have been observed in more active women from two cohort studies than less active ones (Dannenberg, Keller, Wilson, & Castelli, 1989). Although Lee et al 2001, indicated that vigorous intensity of physical activity had a significant impact on reducing the risk of CHD compared to lowest intensity, regular walking independently

predicted lower risk (Lee, Rexrode, Cook, Manson, & Buring, 2001). The significant role of physical activity is more evident when the impact has still remained after adjustment for potential confounders such as smoking status, diet, and alcohol use in most studies. Physical activity independently, regardless of obesity, could contribute to the development of CHD in women (Li et al., 2006).

Risk of CVD was about 1.5 times greater in smokers than non-smokers. In general, there is a non-linear relation between the risk of CVD and the number of cigarettes smoked daily (Burns, 2003; Willett et al., 1987). The mechanisms that cause acute cardiovascular events in smokers include increased hypercoagulability leading to thrombosis, endothelial dysfunction, and development of chronic inflammatory state by an increase in white cells and CRP values (Pyrgakis, 2009). In studies evaluating the impact of physical activity and risk of CVD, the interaction between physical activity and smoking has been reported. Among American women from the Women's Health Study, physical activity was inversely associated with CHD rate in current and past smokers but not in non-smokers (Lee, et al., 2005). Additionally, subjects from Framingham Offspring Study who were more active tended to smoke fewer cigarettes (Dannenberg, et al., 1989). On the other hand, the impact of physical activity and smoking on the risk of CVD cannot be reported hierarchically; that is, each has its own strong impact.

Others report current rather than childhood socio-economic status has more influence on most cardiometabolic risk factors: physical inactivity, HDL-C, triglycerides, post-load glucose, fibrinogen and smoking (Brunner, Shipley, Blane, Smith, & Marmot, 1999). Individuals in households with a high level of education and income had lower risk of CVD in the current study. Education had a greater impact on the risk of CVD; that is, a decreasing trend in the CVD risk was observed by raising the level of education. The greater impact of education might be explained by its unchangeable virtue (as occupation or income might). Thus, education is the most frequent measure used to evaluate the impact of socio-economic status and cardiometabolic abnormalities (Kaplan & Keil, 1993). According to a review by Kaplan and Keil, 1993, during 40 years of study there has been a consistent inverse relation between cardiovascular disease, primarily coronary heart disease, and many of the indicators of socio-economic status. Therefore, it would be necessary to control the impact of socio-economic status while evaluating the association between cardiometabolic risk and potential variables.

The dietary intake was compared only between individuals with medium risk of CVD and individuals with low risk of CVD, due to the small number categorized as high risk. The only significant difference in the dietary consumption was found in fruit and vegetable juice intake, with the medium risk group having a decreased intake of fruit and vegetable juice. This finding is likely due to the adherence of people with diagnosed disease such as diabetes to a special diet (Fitzgerald, et al., 2008). In the study on MetS among the Canadian population using CHMS data, diabetics significantly consumed less fruit and vegetable juice compared to individuals with no diagnosis of diabetes (Setayeshgar, et al., 2012). Diabetics are reported to pay more attention to food labels, especially to the sugar information, than those without diabetes. Diabetics in the Multiethnic Cohort Study (MEC) had lower consumption of juice (>10% difference) compared to those without diabetes (Nöthlings, et al., 2011). The Canadian Diabetes Association recommends to: "have vegetables and fruit more often than juice"

6.5   Strengths & Limitations

Although the findings in the current study and by Leiter et al. suggests including CMR in assessing the risk of CVD, several limitations need to be considered. Some of the risk factors are considered twice in obtaining CMR including HDL-C, blood pressure, and diabetes or fasting plasma glucose. These risk factors are among the risk factors required to obtain both MetS and 10-year risk of CVD. Furthermore, only fasted sub-sample can be used to obtain CMR due to the presence of fasting plasma glucose risk factor in order to define MetS.

The approach of determining the absolute risk considering relative factor of about 1.5 to 2 in individuals with MetS is not fully validated in longitudinal studies, rather it is based on studies reporting that the presence of the metabolic syndrome is associated with a 1.5-2 increased risk, above and beyond Framingham Risk Score. According to Louis et al, 2007, the prevalence of MetS or the risk of all-cause mortality is different using different combinations of MetS diagnostic criteria (Guize et al., 2007). However, this finding needs further investigation considering different factors such as ethnicity, lifestyle factors, and underlying disease. To be able to include all indicators, I had to use the fasted CHMS sub-sample. This along with the exclusion criteria made the sample size of the study smaller than the whole CHMS sample. Further cycles of CHMS in upcoming years are needed to compare the dietary intake between

three levels of CVD risk with a greater sample size, especially in high risk group. Although CHMS collected usual dietary intake through semi-quantitative questionnaires, the quantity of the consumption is not provided to obtain an exact measure of dietary intake. To generalize data to the Canadian population, specific weights provided in CHMS for fasted sub-sample was used. Moreover, since CHMS has provided fasted sub-sample the prevalence of 10-year risk of CVD using either LDL-C (present in fasted sub-sample) or Tchol (present in both fasted and non-fasted sub-sample) were compared.

6.6   Conclusion

The 10-year risk of CVD in the Canadian adult population aged 30 to 74 y significantly changed when CMR was considered (8.10% vs. 9.86%, respectively). In fact, the absolute CVD risk obtained from the validated traditional risk assessment tools might underestimate the10-year risk of CVD in the Canadian adult population. Compared to prospective studies, using cross-sectional studies may also give similar estimation of the 10-year risk of CVD. The 10-year risk of CVD was greater in smokers, individuals with low physical activity, or with low level of education. These factors need to be controlled when evaluating the association between cardiometabolic risk and potential factors. Dietary intake across three levels of 10-year risk of CVD showed a significant difference in fruit and vegetable juice intake between low and medium risk of CVD, with individuals at medium risk having less intake. However, larger studies are needed to evaluate the difference in dietary patterns among individuals at different levels of CVD risk.

CHAPTER 7

STUDY 4. Predicted 10-year risk of cardiovascular disease among Canadian adults using a modified Framingham Risk Score in association with dietary intake

The current study also has been focused on how to perfectly predict 10-year risk of CVD using the Framingham Risk Score. In the current research a different approach was implemented to achieve 10-year risk of CVD suggested by the Canadian Cardiovascular Society. This approach allowed me to include more subjects and all risk factors were considered only once.

In the current research the traditional approach is compared with the modified approach and the association between dietary intake and ≥10% of 10-year risk of CVD is evaluated.

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7.1 Introduction

Heart disease is the second leading cause of death in Canada after cancer (Statistics Canada, 2012). High incidence of CVD carries severe financial consequences in order to manage and treat the disease (The Conference Board of Canada, 2010). Initially, to identify individuals who most likely benefit from primary prevention, cardiovascular risk assessment should be conducted (S. L. Sheridan et al., 2010). The Framingham Risk Score (FRS), recommended by the National Cholesterol Education Program (Adult Treatment Panel III)(Wilson, D'Agostino, et al., 1998), is the most common assessment tool to evaluate 10-year risk of CVD in the United States (Sheridan, et al., 2003). FRS has also been validated in a Canadian cohort (Grover, Hemmelgarn, Joseph, Milot, & Tremblay, 2006) and recommended by the Canadian Cardiovascular Society (CCS) to estimate the 10-year risk of developing fatal and non-fatal cardiovascular events (Anderson, et al., 2013). FRS accurately predicts cardiovascular deaths for each Canadian individual in the Lipid Research Clinics Follow-Up Cohort (Grover, et al., 2006).

In 2012, CCS modified the FRS to increase the appropriate use of evidence-based CVD risk assessment in the management of dyslipidemia for reducing risk in the Canadian population. They suggested three main limitations for FRS to estimate 10-year risk of CVD. First, the estimation of CVD over the short term of 10 years is most likely targeted to older individuals for therapy. Second, the FRS is more accurate among younger individuals due to the strong association of the CVD risk factors with premature CVD. Third, the risk of other age-related diseases such as cancer increase in older individuals which decreases the accuracy of the FRS. Additionally, a population-based study indicated a high distribution of the individuals in the Low Risk (LR) category (risk <10%) over 10 years (Ford, Giles, et al., 2004). Therefore, two modifications were suggested. First they suggested considering the history of premature CVD in the fist-degree relatives of individuals 30-59y. Secondly, since a 10-year risk does not entirely account for the risk in younger individuals; CCS recommends calculating "cardiovascular age" for each individual (Anderson, et al., 2013).

Incorporating the two modifications in FRS to obtain more accurate estimate for 10-year risk of CVD was validated at the individual level by CCS. However, in order to estimate the risk at the national level it is essential to investigate whether the two modifications or either of them

could change the estimated risk significantly. Therefore, the main objective is to determine whether the estimate of the percent of 10-year risk of CVD obtained through FRS will change after implementing the two modifications and which modification has a stronger contribution.

Dietary modification has long been one of the main approaches in CVD prevention. The inverse relation between fruit and vegetables consumption and CVD risk has been supported by consistent evidence from epidemiologic studies with various designs (Dauchet, et al., 2006). According to the American Heart Association the detrimental effect of sugar on CVD risk can be reduced by having no more than 100 and 150 calories per day from added sugars, by American women and men, respectively (Johnson, et al., 2009). Additionally, the beneficial effects of omega-3 fatty acid from fish on CVD end points were evaluated (Psota, Gebauer, & Kris-Etherton, 2006). However, the association between diet and the predicted risk of CVD among Canadians has yet to be determined. The second objective is to determine the association between diet and the predicted 10-year risk of CVD while controlling for potential covariates including socio-demographic characteristics.

7.2 Materials and Methods

7.2.1 Study Population

Data from the Canadian Health Measures Survey (CHMS), cycle 1, 2007–2009 were used. More information on survey design and data collection can be found elsewhere (Statistics Canada, 2011a). For the purpose of the study, individuals who were either under the age of 30 or over the age of 74 were excluded. The final number of respondents for the current study was 2,730 (male=1279 versus female=1451) representative of 17,587,428 Canadians aged 30 to 74 y.

7.2.2 10-Year Risk of CVD

To estimate the 10-year risk of CVD, categorical charts and tables based on FRS were used (**Appendices 1 & 2**) (Wilson, D'Agostino, et al., 1998). Separate score sheets were available for each sex according to age, blood pressure, total cholesterol (Tchol), and high-density lipoprotein cholesterol (HDL-C) categories (Wilson, D'Agostino, et al., 1998). Further, the presence or absence of diabetes and smoking status were factored in the estimations. Daily smokers, occasional smokers, or those who stopped smoking in less than a year ago were

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classified as smokers. To identify individuals with undiagnosed diabetes, Glycated Hemoglobin (HbA1c) ≥6.5% was used as the criterion (The International Expert Committee, 2009). Cases of self-reported diabetes were added to the individuals with undiagnosed diabetes.

7.2.3 Modified FRS (mFRS)

Two modifications were implemented into tFRS (traditional FRS). Firstly the percent of 10-year risk of CVD was doubled in subjects aged 30-59 y without diabetes with the history of premature CVD present in a first-degree relative before 55 y for men and 65 y for women. For the second modification, to calculate the cardiovascular age, the formula suggested by Cooney et al. was used which seemed to be applicable at the population level (Cooney, et al., 2012). This is a generic equation to calculate risk age, from a Cox function, where age is included as a risk factor:

$$Risk\ Age = [(\beta chol \times chol) + (\beta sBP \times sBP) + (\beta smok \times currsmoking) + (\beta age \times age) - (\beta chol \times 4) - (\beta sbp \times 120)] \div \beta age$$

Where chol is cholesterol and sBP is systolic blood pressure. The β was achieved by running a multiple regression analysis with the percent of 10-year risk of CVD as the continuous dependent variable and total cholesterol, sBP, smoking (0=non-smokers, 1=smoker), and age (30-74 y) as independent variables. The β obtained for each variable through the regression equation was entered into the above equation to achieve the risk age or cardiovascular age. In the next step new 10-year risk of CVD or mFRS was re-created using two modifications.

7.2.4 Socio-demographic characteristics (covariates)

Four levels of education based on the highest level achieved by any member of the household were defined. Also, four economic status levels were based on the total household income and the number of individuals in the household. Only two ethnic groups, that is, white and non-white, were created due to few numbers of non-white individuals. Moreover, physical activity respondents were subsequently categorized into "active" (total daily leisure time energy expenditure (EE) ≥ 3 kcal/kg/day), "moderate" ($1.5 \leq EE < 3$), and "inactive" ($0 \leq EE < 1.5$). Alcohol intake was classified into "ever drinker" and "never drinker" due to insufficient data in

CHMS. To define abdominal obesity, the International Diabetes Federation's recommendation of using ethno-specific cutoffs for waist measurement was followed (Alberti, et al., 2009).

7.2.5 Classification of the risk

Although the risk categories have been arbitrarily chosen rather than by scientific evidence, they are commonly used internationally: Low Risk or LR, <10%, Intermediate Risk or IR, 10-19%, and High Risk or HR, ≥20% (Armstrong, Brouillard, & Matangi, 2011). The distributions were compared using either tFRS or mFRS.

7.2.6 The association of dietary intake with ≥10% of CVD risk

In CHMS, usual dietary intake was collected through a semi-quantitative food frequency questionnaire. Details regarding food groups in CHMS can be found elsewhere (Statistics Canada, 2013b). Dietary consumption data were converted into a daily frequency of consumption (times/day). In logistic regression model, in order to have a sensible intake for the food groups, quartiles of intake were created. The only exception is related to breakfast cereal intake in which tertile categorization was applied due to insufficient sample in the fourth quartile of intake.

Based on the distribution of the individuals into three risk categories scores, the majority of the subjects were identified in the LR (67.4%) category. Thus, in order to have a proper distribution to define the binary outcome variable in the logistic model, individuals were categorized as having <10% (outcome=0) of the risk or ≥10% (outcome=1).

7.2.7 Data Analysis

The 95% confidence intervals of the mean estimate obtained from tFRS and mFRS were compared as well as the significant difference in the distribution of the individuals in three risk categories. To differentiate the dietary intake of individuals with ≥10% and <10% of 10-year risk of CVD, "Regress" command was used to obtain the independent sample-t test in STATA. For the second objective, logistic regression analyses was utilized to build the best model in six stages. Data manipulation, cleaning, and creation of new variables were done using IBM SPSS statistics for windows, version 20 (Armonk, NY). All statistical analyses were conducted by

STATA/SE 11, StataCorp. As per Statistics Canada's recommendation, data were weighted and bootstrapped using specific command in STATA in order to be representative of the Canadian population.

7.3   Results

7.3.1   Descriptive modified 10-year risk of cardiovascular diseases

The sample (n=2730) used to investigate cardiovascular diseases (CVD) represents 17,587,428 Canadians aged 30-74 y. The mean for the percent of 10-year risk of CVD by tFRS and mFRS is displayed in **Table 7-1**. The estimated overall mean for the percent of 10-year risk of CVD was significantly greater based on the mFRS compared with tFRS. In order to realize which modification had more contribution in generating the significant difference, the mean of the risk was evaluated while using only one modification (**Table 7-1**). Cardiovascular risk age had a greater contribution in changing the overall CVD risk over 10 years. The mean of the percent risk of CVD was significantly greater using cardiovascular risk age compared with the traditional FRS. Although the confidence interval for the mean of the percent risk while modifying FRS among 30-59 y individuals with history of premature CVD in immediate family had overlap with tFRS, the overlap was very small.

Cardiovascular age modification had significantly greater contribution in increasing the risk among men compared to the second modification (**Table 7-1**). In women implementing either risk age or the presence of premature CVD history modifications separately did not change the percent of risk significantly, although using both modifications indicated significant alteration in the percent of risk (**Table 7-1**). Additionally, 10-year risk of CVD was significantly greater according to mFRS compared to tFRS among men and women, separately (**Table 7-1**).

Table 7-1 Weighted estimates of the mean for the percent of 10-year risk of cardiovascular diseases (CVD) based on using traditional FRS or modified FRS among Canadians aged 30 to 74 y (population size= 17,587,428)

10-year risk of CVD	percent of risk (mean)±SE[1] (Confidence Intervals)		
	All	Male	Female
Traditional FRS[2]	6.1%±0.2[*]	8.1±0.3[*]	4.1± 0.2[*]
	(5.66-6.74)	(7.38- 8.77)	(3.67- 4.54)
Modified FRS	8.6%±0.3[*]	11.9±0.6[*]	5.4±0.3[*]
	(7.89-9.43)	(10.66 -13.27)	(4.77- 6.11)
Modified FRS (considering cardiovascular risk age)	7.8%±0.3[*]	11.2 ±0.5[*]	4.6±0.2
	(7.21-8.52)	(9.96- 12.39)	(4.16- 5.11)
Modified FRS (considering 30-59 y individuals with the history of premature CVD in immediate family)	6.6%±0.2	8.6±0.3	4.7±0.26
	(6.16-7.08)	(7.89-9.25)	(4.14-5.31)

[*] Statistically significant
[1] SE= Standard Error
[2] FRS=Framingham Risk Score

The risk categories that are widely used internationally are low risk (5-9%), intermediate risk (10-19%), and high risk ($\geq$ 20%). **Table 7-2** and **Figure 7-1** indicate the distribution of Canadians 30-74 y into CVD risk categories based on traditional FRS and modified FRS. The distribution of Canadians in low, intermediate, and high risk of CVD displayed significant difference between traditional and modified RFS in low and high risk categories, 79.6% versus 67.3% (low risk) and 4.5% versus 13.7% (high risk), respectively. In fact using modified FRS increased the number of individuals categorized in high risk and decreased the number of individuals in the low risk category significantly. The same result was observed in Canadian men, however, the significant increase in the number of female individuals was observed only in the high risk category.

Table 7-2 Weighted estimates of the prevalence of 10-year risk of cardiovascular diseases (CVD) in three levels of category based on the traditional FRS and modified FRS among Canadians aged 30 to 74 y (population size= 17,587,428)

CVD risk categories	Traditional FRS[1]			Modified FRS		
	Estimated prevalence±SE[2] (Confidence Intervals)			Estimated prevalence±SE (Confidence Intervals)		
	All	**Male**	**Female**	**All**	**Male**	**Female**
Low Risk	79.6±1.4[*] (76.36-82.55)	69.7±2.3[*] (64.21-74.65)	89.3±1.65 (85.10-92.45)	67.3±1.8[*] (63.2-71.28)	53.5±3.1[*] (46.69-60.25)	80.8±2.3 (75.09-85.53)
Intermediate Risk	15.8±1.1 (13.53-18.4)	22.5±1.9 (18.60-27.03)	9.2±1.53 (6.40-13.20)	18.8±1.3 (16.13-21.91)	22.8±1.7 (19.28-26.90)	14.9±2.1 (10.86-20.2)
High Risk	4.5±0.4[*] (3.69-5.63)	7.8±0.8[*] (6.16-9.77)	1.4±0.2[*] (1.04-1.96)	13.7±0.9[*] (11.82-16.00)	23.6±1.7[*] (19.97-27.66)	4.2[†]±0.7[*] (2.81-6.28)

[*] Statistically significant
[1] FRS=Framingham Risk Score
[1] SE= Standard Error
[†] Data with a coefficient of variation from 16.6% to 33.3%.

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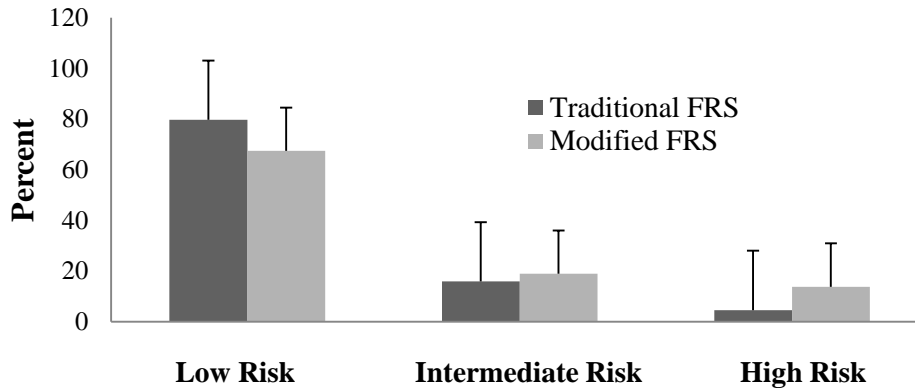
Figure 7-1 How traditional FRS and modified FRS differentiate the distribution of Canadians aged 30-74 y into 10-year risk of CVD category (population size= 17 587 428)

Table 7-3 indicates the mean for the percent of CVD risk by socio-demographic characteristics. The mean of the risk was significantly greater among Canadian men. The risk was significantly greater in older individuals. An ascendant increase in the mean of CVD risk was observed by decrease in the level of education. The mean of the risk was significantly greater in individuals from a family with at least a member with less than secondary school graduation compared with individuals from either a family with at least a member with some postsecondary or postsecondary graduation. Additionally, the mean of the risk was significantly greater among smokers compared to non-smokers as well as among abdominally obese individuals compared to not abdominally obese counterparts

Table 7-3 Weighted estimates of the mean for the percent of 10-year risk of cardiovascular diseases (CVD) by socio-demographic characteristics of Canadians aged 30 to 74 y (population size= 17,587,428)

Characteristics	CVD, estimated mean±SE[1]	Confidence Intervals
Sex		
Male	11.96±0.6[*]	10.66-13.27
Female	5.44±0.3[*]	4.77-6.11
Age group[2]		
30 – 34 y	3.50±0.5[*]	2.25-4.73
35 – 39 y	4.26±0.5[*]	3.17-5.36
40 – 44 y	7.43±0.9[*]	5.34-9.53
45 – 49 y	7.66±0.5[*]	6.57-8.74
50 – 54 y	11.38±0.8[*]	9.46-13.30
55 – 59 y	12.02±0.7[*]	10.44-13.61
60 – 64 y	11.69±0.6[*]	10.25-13.13
65 – 69 y	13.27±0.5[*]	12.03-14.50
70 – 74 y	12.32±0.6[*]	10.82-13.83
Education level[3]		
< secondary school graduation	11.61±0.7[*]	9.92-13.31
Secondary school graduation	10.33±0.9	8.26-12.38
Some postsecondary	8.46±0.6[*]	6.99-9.92
Postsecondary graduation	8.07±0.3[*]	7.42-8.73
Income level		
Lowest income	9.58±0.6	8.09-11.07
Lower-middle income	8.57±0.7	7.03-10.12
Upper-middle income	9.54±0.5	8.47-10.62
Highest income	8.11±0.3	7.32-8.89
Physical activity		

Inactive	9.33±0.5	8.23-10.44
Moderately active	7.88±2.3	6.70-9.06
Active	7.80±0.6	6.41-9.20
Alcohol		
Never drink	8.86±0.8	7.07-10.66
Ever drink	8.62±0.3	7.92-9.33
Ethnicity		
Non-white	8.23±1.1	5.83-10.63
White	8.73±0.3	8.08-9.38
Smoking		
Non-smokers	7.20±0.3[*]	6.52-7.89
Smokers	13.58±0.7[*]	11.87-15.28
Abdominal Obesity		
obese	9.85±0.4[*]	8.99-10.72
Not obese	7.80±0.3[*]	6.99-8.60

[*] Statistically significant
[1] SE= Standard Error
[2] 10-year risk of CVD was significantly lower in individuals 30-49 y compared with individuals 50-74 y.
[3] 10-year risk of CVD was lower in individuals with some postsecondary or postsecondary graduation compared with individuals with less than secondary school education

The vast majority of individuals were recognized at low risk therefore, the sample was categorized into two levels of <10% risk and ≥10% risk. The dietary consumption was assessed according to these two categories. Dietary consumption among Canadians aged 30 to 74 y with the CVD risk of <10% or ≥10% presented in **Table 7-4**. Canadians with ≥10% of CVD risk significantly consumed less dairy products and fruit and vegetables but greater SSBs compared to Canadians with <10% of CVD risk

Table 7-4 Dietary consumption among Canadians aged 30 to 74 y having 10-year risk of cardiovascular diseases (CVD) <10% or ≥10% (population size=17,587,428)

Food and beverages times/day[1]	Individuals with ≥10% CVD risk, Mean±SE[2] (CI[3])	Individuals with <10% CVD risk, Mean±SE[2] (CI[3])
Meat and Fish		
-*Red meat, organs, hotdogs, sausage or bacon, seas foods, eggs, beans, and nuts*	1.68±0.08 (1.49-1.87)	1.65±0.05 (1.53-1.77)
Grains, Fruit & Vegetable		
-*Hot/cold cereal, white bread, brown bread, rice, pasta (grains)*	3.56±0.29 (2.92-4.20)	3.00±0.13 (2.71-3.29)
-*Fruit and Vegetable*	2.90±0.11* (2.64-3.16)	3.59±0.09* (3.38-3.80)
Milk & Dairy Products		
-*Milk, cottage cheese, and yogurt or ice cream*	1.36±0.04* (1.27-1.46)	1.59±0.06* (1.45-1.73)
Dietary Fat		
-*Regular-fat salad dressing or mayonnaise and regular-fat potato chips, tortilla chips or corn chips*	0.40±0.01 (0.36-0.44)	0.42±0.01 (0.39-0.45)
Water & Soft Drinks		
-*Regular soft drink, sport drink, and fruit drink (sugar-sweetened beverages)*	0.43±0.02* (0.37-0.49)	0.32±0.02* (0.26-0.38)
- Diet soft drink	0.25±0.04† (0.15-0.34)	0.19±0.02 (0.14-0.25)
-*Fruit and vegetable juice*	0.67±0.04 (0.57-0.77)	0.70±0.03 (0.63-0.77)

* Intake is statistically significant between individuals with ≥10% risk and <10% risk based on independent sample t-tests.
[1] Frequency of consumption
[2] SE= Standard Error
[3] Confidence Intervals
* Independent samples t-test
† Data with a coefficient of variation from 16.6% to 33.3%.

7.3.2 Association between modified 10-year risk of CVD and dietary intake

7.3.2.1 Univariate analysis

The association between 10-year risk of CVD and dietary intake was assessed using logistic regression. The outcome is 10-year risk of CVD (≥10%=1 and <10%=0) and the independent predictors are different food groups and other covariates (socio-demographic

characteristics). **Table 9** and **Table 10** in **Appendix 3** indicate the univariate association between 10-year risk of CVD and each independent predictor. Socio-demographic variables and food groups were included in **Table 9** and **Table 10**, respectively.

Among socio-demographic characteristics in the full sample, abdominal obesity, low level of education, low income, smoking, and never drinking alcohol intake were associated with ≥10% 10-year risk of CVD based on the assumption of the p-value <0.2 for univariate analysis (**Table 9, Appendix 3).**

Since "sex" is one of the risk factor in 10-year risk of CVD and needed to be considered in obtaining the 10-year risk, it was not included in the logistic model. However, the analysis was run separately for males and females. Similarly, since smoking and age were considered to obtain 10-year risk of CVD, they were not included in the logistic model. The trend in the association between socio-demographic variables and 10-year risk of CVD for males and females were similar to what was obtained for the full sample (**Table 10, Appendix 3**). However, physically active individuals showed significant negative association based on the p-value<0.2 in males and females.

Based on the assumption of the p-value <0.2 for (**Table 10, Appendix 3**). In comparison to the first quartile, the odds of having ≥10% of CVD risk decreased by consuming more milk and dairy products, grains, dietary fat, and fruit and vegetables. However, compared to the first quartile, the odds of 10-year risk of CVD ≥10% increased by consuming more SSBs and potato.

Considering sex, dietary fat consumption and potato were no longer significant among females as well as milk and dairy products in males. Fruit and vegetable juice consumption in the 2nd quartile was associated with increased odds of having ≥10% of CVD in men. In addition, diet soft drink consumption was associated with increased risk in men.

7.3.2.2   Multivariate analysis

In the next step all significant univariate analysis were entered into a logistic model and multicollinearity was evaluated. The mean Vifs were equal to 1.87, 1.56, and 1.85 for the full sample, males and females, respectively. All three Vifs were less than 2.5 as the threat for

multicollinearity. **Table 7-5** indicates the best model of the association between the 10-year risk of CVD $\geq$10% and independent indicators after implementing specification error test.

Table 7-5 Logistic regression analysis indicating the association between $\geq$10% for 10-year risk of cardiovascular diseases (CVD) and the dietary intake after adjusting for the potential covariates[§] (population size=17,587,428)

Predictors	Odds Ratio(Confidence Interval) P-value		
	All	Males	Females
Abdominal Obesity[2]	1.41(1.06-1.87)	N/A	2.25(1.33-3.83)
	0.021		0.006
Ethnicity[2]	N/A[†]	N/A	2.54(1.13-5.71)
			0.027
Alcohol[3]	N/A	N/A	0.43(0.25-0.73)
			0.005
Education			
< secondary school graduation[4]			
Secondary school graduation	0.52(0.27-1.01)	0.56(0.20-1.53)	0.57(0.15-2.13)
	0.054	0.233	0.377
Some postsecondary	0.32(0.18-0.56)	0.46(0.23-0.90)	0.14(0.04-0.42)
	0.001	0.028	0.002
Postsecondary graduation	0.43(0.30-0.59)	0.48(0.23-1.00)	0.43(0.24-0.76)
	<0.001	0.053	0.007
Income			
Lowest income[4]			
Lower-middle income	0.84(0.47-1.53)	N/A	N/A
	0.558		
Upper-middle income	1.32(0.68-2.57)	N/A	N/A
	0.358		
Highest income	1.15(0.72-1.83)	N/A	N/A
	0.525		
Physical Activity			
Active[4]			
Moderately active	1.06(0.75-1.49)	0.99(0.67-1.45)	1.27(0.54-2.94)
	0.691	0.966	0.544
Inactive	1.01(0.67-1.52)	1.16(0.65-2.07)	1.35(0.58-3.15)
	0.947	0.561	0.447
Fruit and vegetable			
1st Q[4]			
2nd Q	0.84(0.53-1.33)	0.92(0.53-1.59)	0.88(0.50-1.56)

	0.443	0.748	0.644
3rd Q	0.53(0.34-0.80)	1.00(0.60-1.65)	0.43(0.18-0.98)
	0.007	0.995	0.047
4th Q	0.46(0.29-0.72)	0.58(0.28-1.19)	0.57(0.30-1.05)
	0.003	0.128	0.068
Potato[4]			
1st Q [4]			
2nd Q	1.18(0.77-1.79)	1.06(0.59-1.89)	N/A
	0.414	0.826	
3rd Q	1.58(1.09-2.31)	1.60(1.08-2.36)	N/A
	0.021	0.022	
4th Q	1.95(1.45-2.62)	2.21(1.34-3.64)	N/A
	<0.001	0.005	
Breakfast Cereal[5]			
1st tertile[4]			
2nd tertile	0.76(0.52-1.10)	N/A	N/A
	0.138		
3rd tertile	0.59(0.38-0.92)	N/A	N/A
	0.026		
Fruit & Vegetable Juice			
1st Q [4]			
2nd Q	N/A	1.59(1.04-2.43)	N/A
		0.035	
3rd Q	N/A	1.41(0.74-2.68)	N/A
		0.259	
4th Q	N/A	1.06(0.53-2.11)	N/A
		0.838	
Sugar Sweetened Beverages			
1st Q [4]			
2nd Q	N/A	0.80(0.48-1.33)	N/A
		0.369	
3rd Q	N/A	0.48(0.28-0.83)	N/A
		0.013	
4th Q	N/A	0.70(0.42-1.13)	N/A
		0.131	
Grains[5]			
1st Q [4]			
2nd Q	N/A	0.70(0.40-1.22)	N/A
		0.190	
3rd Q	N/A	0.60(0.34-1.03)	N/A

		0.065	
4th Q	N/A	0.60(0.43-0.84)	N/A
		0.007	
Diet Soft Drink[6]	N/A	1.60(0.73-3.45)	N/A
		0.216	

According to the "linktest" the p-value was <0.001 and 0.935 for variable _hat and variable _hatsq, respectively, indicate that the model fitted well for the full sample. Similarly, sex-specific p-values for _hat and variable _hatsq were <0.001 and 0.919 for males and 0.001 and 0.121 for females, respectively.
§ Covariates include age, sex, physical activity, ethnicity, education, income, smoking, abdominal obesity and alcohol intake.
†Not Applicable
[1]Abdominal obesity=1 (reference group) versus abdominal obesity=0
[1]White individuals=1 versus non-white=0
[2]Ever drinkers= 1 versus never drinkers=0
[3]Reference group
[4]Potato includes French fries, hash brown, boiled, backed, and mashed potato excluding sweet potato.
[5]For cereal since there were only 17 observations in category 4 therefore category 3 and 4 merged.
[6]Diet soft drink categorized into 2 categories (0 times/day and > 0 times/day).

The odds of having 10-year risk of CVD ≥10% was lower in individuals from a family with at least a member with some postsecondary and postsecondary graduation, respectively, compared with individuals from a family with at least a member of less than secondary school graduation. Consuming fresh fruit and vegetables in the amount of 3rd and 4th quartile and consuming cereal in the amount of 3rd tertile were associated with decreased odds of having 10-year risk of CVD ≥10% compared to the consumption in the amount of 1st quartile. However, being abdominally obese was associated with greater odds of having 10-year risk of CVD ≥10% compared to not being abdominally obese. Further, consuming potato in the amount of 3rd and 4th quartile was associated with increased odds of having 10-year risk of CVD ≥10% compared to the consumption in the amount of 1st quartile.

No interaction was observed among the variables. However, income was a confounder for all variables except abdominal obesity. Therefore, it remained in the final model although it was not significant. Further, physical activity remained in the model for the principal of parsimony. The percent of change in the estimate between the crude and adjusted coefficients (data not shown) for income are observed below which are all greater than 10% as the pre-set percentage of change.

Formula: │[crude coefficient – adjusted coefficient]/crude coefficient│X100
Potato consumption:
2nd quartile: │[0.25 – 0.16]/0.25│X100 = 36
Fruit and vegetable consumption

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3rd quartile: $\big| [-0.55 - -0.63]/-0.55 \big|$ X100 = 14.54

Cereal consumption

3th quartile: $\big| [-0.51 - -0.44]/-0.51 \big|$ X100 = 13.72

Education

Level 2: $\big| [-0.47 - -0.64]/-0.47 \big|$ X100 = 36.17

Level 3: $\big| [-0.92 - -1.12]/-0.92 \big|$ X100 = 21.74

Level 3: $\big| [-0.63 - -0.84]/-0.63 \big|$ X100 = 33.33

After adjusting for the potential covariates in the male sample, education, fruit and vegetable juice, potato, cereal, SSB, and grains consumption remained statistically significant in the model (**Table 7-5**). Among females education, abdominal obesity, ethnicity, fruit and vegetables, and alcohol consumption remained statistically significant in the model (**Table 7-5**).

Physical activity remained in both male and female model for the principal of parsimony. Further, fruit and vegetable intake as well as diet soft drink were confounders for fruit and vegetable juice, potato, and SSB intake in the male sample. Therefore, they remained in the final model although they were not significant. The percent of change in the estimate between the crude and adjusted coefficients (data not shown) are observed below which are all greater than 10% as the pre-set percentage of change.

Formula: $\big|$ [crude coefficient – adjusted coefficient]/crude coefficient $\big|$ X100

Potato consumption in the 2nd quartile:

Effect of fruit and vegetable: $\big| [0.11 - 0.14]/0.11 \big|$ X100 = 27.27

Effect of diet soft drink: $\big| [0.11 - 0.04]/0.11 \big|$ X100 = 63.63

Fruit and vegetable juice intake

Effect of fruit and vegetable in the 3rd quartile: $\big| [0.24 - 0.29]/0.24 \big|$ X100 = 20.83

Effect of diet soft drink in the 3rd quartile: $\big| [0.24 - 0.28]/0.24 \big|$ X100 = 16.66

Effect of fruit and vegetable in the 4rd quartile: $\big| [-0.09 - -0.02]/-0.09 \big|$ X100 = 77.77

Effect of diet soft drink in the 4rd quartile: $\big| [-0.09 - -0.03]/-0.09 \big|$ X100 = 66.66

Sugar-sweetened beverages

Effect of fruit and vegetable in the 2nd quartile: $\big| [-0.22 - -0.31]/-0.22 \big|$ X100 = 40.90

Effect of diet soft drink in the 2nd quartile: $\big| [-0.12 - -0.22]/-0.12 \big|$ X100 = 45.45

Effect of fruit and vegetable in the 3rd quartile: $\left| [-0.70 - -0.81]/-0.70 \right| \times 100 = 15.71$

Effect of diet soft drink in the 3rd quartile: $\left| [-0.70 - -0.61]/-0.70 \right| \times 100 = 12.85$

Effect of fruit and vegetable in the 4th quartile: $\left| [-0.37 - -0.48]/-0.37 \right| \times 100 = 29.73$

Effect of diet soft drink in the 4th quartile: $\left| [-0.37 - -0.24]/-0.37 \right| \times 100 = 35.13$

Table 7-6 indicates the conversion of each quartile of intake into the frequency of consumption per week. Each time of intake was considered as one serving of consumption for each food group according to Canada's food guide.

Table 7-6 The conversion of the quartile of food and beverages intake into weekly frequency of consumption

Quartile of Intake	consumption (times/week)		
	All	**Male**	**Female**
Fruit & Vegetable[1]			
1st Q	Intake ≤ 14		Intake < 17
2nd Q	14< intake<22		17≤ intake<25
3rd Q	22≤ intake≤30		18< intake<35
4th Q	Intake > 30		Intake ≥ 35
Cereal[2]			
1nd Q	Intake <1		
2rd Q	1≤ intake<4		
3th Q	Intake ≥ 4		
Potato[3]			
1st Q	Intake ≤ 1	Intake ≤ 1	
2nd Q	1< intake≤2	1< intake≤2	
3rd Q	2< intake<4	2< intake<4	
4th Q	Intake ≥ 4	Intake ≥ 4	
Sugar-sweetened beverages[4]			
1st Q		Intake=0	
2nd Q		0< intake≤1	
3rd Q		1< intake<4	
4th Q		Intake ≥ 4	
Grains[5]			
1st Q		Intake < 11	
2nd Q		11≤intake≤14	
3rd Q		14< intake≤18	
4th Q		Intake > 18	
Fruit and vegetable juice[6]			
1st Q		Intake < 1	

2nd Q	1≤intake<3.5
3rd Q	3.5≤ intake<7
4th Q	Intake > 7

[1] One serving is equivalent to 1 medium fruit or ½ cup cooked or one cup raw vegetable.
[2] One serving is equivalent to ¾ cup or 175 ml.
[3] One serving is equivalent to one hash brown or ½ cup baked potato, or 15 french fries.
[4] One serving is equivalent to 335 ml or one can.
[5] One serving is equivalent to one slice bread or ½ bagel or ½ pita or ½ cup rice or pasta etc.
[6] One serving is equivalent to ½ cup of 100% juice.

7.3.3   Summary

Using the modified FRS for predicting percent 10-year risk of CVD significantly increased the mean of the risk compared to the traditional approach, 8.66%±0.35 versus 6.06%±0.18. Similarly, the distribution of Canadians in low (<10%), and high risk (≥20%) categories of CVD displayed significant difference between traditional and modified RFS: 79.6% versus 67.3% (low risk) and 4.5% versus 13.7% (high risk), respectively. The risk was significantly greater among males (11.9% vs. 5.4%), less educated (<secondary school graduation (11.6%) vs. some postsecondary (8.4%) and postsecondary graduation (8.1%)), smokers (13.5% vs. 7.2%), and abdominally obese individuals (9.85% vs. 7.8%). The odds of having risk ≥10% was significantly lower in highly educated and Canadians with higher consumption of cereal and fruit and vegetable. However, the risk was significantly greater in individuals with abdominal obesity as well as individuals with high consumption of potato.

7.4   Discussion

7.4.1   Descriptive modified 10-year risk of cardiovascular disease

In overall the estimated percent risk of CVD over 10 years in Canadian adults aged 30-74 y (n=2730) increased from 6.06% to 8.66% after incorporating the two modifications suggested by CCS. Cardiovascular age had significantly greater contribution in such increase in the 10-year risk of CVD in Canadian men. However, among women implementing either modification alone did not change the risk significantly whereas using both modifications changed the risk significantly. The vast majority of Canadian adults were categorized at LR category (67.4%). Implementing the two modifications compared to not using them in traditional FRS (tFRS) significantly increased the number of individuals in the HR category in both sexes and decreased in the LR category in Canadian men.

Incorporating the two modifications in assessing the 10-year risk of CVD is recommended by CCS (Anderson, et al., 2013). In the current study, the modifications seemed to provide a more realistic estimate of CVD risk compared to the traditional approach by avoiding the underestimation of CVD risk especially in younger Canadians. Findings suggest that in order to have an accurate screening of at-risk individuals, considering both modifications and specifically cardiovascular age in Canadian men is necessary to estimate the 10-year risk of CVD.

The percent 10-year risk of CVD among Canadians is comparable to their American counterpart. According to the third National Health and Nutrition Examination Survey (NHANES III), 81.7% of Americans had a 10-year risk for coronary heart disease (CHD) of <10%, 15.5% of 10% to 20%, and 2.9% of >20% (Ford, W. H. Giles, & A. H. Mokdad, 2004) which are comparable with the estimates in Canadians. Findings indicated that a greater number of individuals are categorized in the high risk category and a lower number in the low risk category while considering mFRS rather than tFRS. Thus, the number of Canadian individuals who might benefit from lipid-lowering treatment may increase.

Individuals with ≥10% of CVD risk over 10 years had significantly greater SSBs consumption as opposed to lower intake of fruit and vegetable and milk and dairy product compared to individuals with <10% of CVD risk. Among these food groups, fruit and vegetable intake was also a predictor of the 10-year risk of CVD in the final logistic model among the Canadian population; this is discussed in the next section.

7.4.2   Predictors of ≥10% of 10-year risk of cardiovascular disease

Controlling for the potential covariates, the risk of having ≥10% 10-year risk of CVD was greater among individuals with low fruit and vegetable and breakfast cereal consumption and in individuals with greater potato consumption. Further, adult Canadians without abdominal obesity and individuals from families with at least one highly-educated member had lower risk of having ≥10% 10-year risk of CVD.

Low education level in the household was one of the strong predictors of having 10-year risk of CVD ≥10%. Certain diseases are more common in lower socio-economic classes such as

CVD and risk factors including smoking, obesity, physical inactivity, hypertension and hypercholesterolemia (Meier & Ackermann-Liebrich, 2005). Higher risk of CVD was associated with lower levels of education among Americans (Winkleby, Jatulis, Frank, & Fortmann, 1992). Further, education was associated with cardiovascular risk factors in German and Chinese people (Helmert, Shea, Herman, & Greiser, 1990; Z. Yu et al., 2000). Education is widely used as an indicator of socio-economic status because it is always available and invariant for all people and highly reliable (Helmert, et al., 1990). Further, respondents usually do not hesitate to respond to questions regarding education (Kaplan & Keil, 1993).

Excess in abdominal fat is strongly associated with the metabolic risk factors (Casanueva et al., 2010). Abdominal obesity was associated with increase in 10-year risk of CVD ≥10% in the full sample and among women. Abdominal obesity was the main defining component of metabolic syndrome (Setayeshgar, et al., 2012) and metabolic syndrome doubles the risk of developing CVD on its own (Alberti, et al., 2006). Therefore, abdominal obesity may increase the risk of CVD through its contribution to develop metabolic syndrome by affecting blood pressure, blood lipid level, and insulin sensitivity (Kahn & Flier, 2000).

Since the number of non-white individuals was very low, the ethnicity was categorized into two groups of "white" and "non-white". White individuals were at greater risk of CVD compared to non-white individuals. In the Third National Health and Nutrition Examination Survey (NHANES), 1988–1994, black and Mexican-American women and black men had greater risk for CVD than white individuals (Sundquist, Winkleby, & Pudaric, 2001). However, in a prospective study in UK, Afro-Caribbean individuals had lower incident of myocardial infarction (MI) than white people, whereas South Asian had similar incidents as white individuals (Group, 1998). Similar to the current study, white individuals in UK had the largest distribution. The ethnic groups are more diverse in the US compared to Canada or other countries which makes the US a more appropriate country to research different ethnic groups. Small number of non-white individuals confines the studies to have a picture of health status of various ethnicities (Clark et al., 2001).

Light to moderate consumption of alcohol intake is associated with reduced risk of multiple cardiovascular events (Ronksley, et al., 2011). In CHMS data amount of alcohol intake

and the type of alcohol consumed is not reflected. Classification of alcohol into "ever drinkers" and "never drinkers" was the achievable derived variable. In the current study ever drinking alcohol among women decreased the risk of having ≥10% of CVD compared with never drinking of alcohol. However, among Canadian men alcohol intake did not remain significant while controlling for the covariates. Ronksley et al performed a systematic review and meta-analysis in which the dose of 2.5–14.9 g alcohol (about ≤1 drink) per day was protective for CVD mortality, CHD mortality, stroke mortality, incident CHD events, and incident stroke events compared with no alcohol consumption (Ronksley, et al., 2011). In addition, according to the prospective Nurses' Health Study a U-shaped relation was strongly generated between alcohol intake and mortality in women (Fuchs et al., 1995). Light to moderate consumption, regardless of the type of alcohol (Cleophas, 1999; Fuchs, et al., 1995), compared to abstinence was significantly associated with reduced risk of fatal CVD. Further studies are required to observe the effect of alcohol consumption on cardiovascular events among Canadian men and women. The beneficial effect of light to moderate alcohol consumption may vary for each sex considering other potential covariates in Canadians.

The lower risk of CVD ≥10% with greater consumption of fruit and vegetables (≥22 times/week) in Canadians especially females, was similar to the results from a meta-analysis of nine cohort studies from US and Finland in which six cohorts reported an inverse association between fruit and vegetable intake and risk of coronary heart disease (CHD) (Dauchet, et al., 2006). In addition, according to the Nurses' Health Study and the Health Professionals' Follow-Up Study both men and women in the top quintile of fruit and vegetable intake had a 20% lower risk for CHD than did those in the lowest quintile (Joshipura et al., 2001). While the role of fruit and vegetable in reducing blood pressure, weight, and lipid profile is documented, the causal mechanism of association with CVD needs further investigation.

Potato consumption in the current study (>2 times/week) was an example of high caloric foods. The majority of the studies include potato in vegetable category. In the prospective Nurses' Health Study potato and french fries consumption were both positively associated with risk of type 2 diabetes (RR=1.14 and 1.21, respectively) after adjusted for age, dietary and non-dietary factors (Halton et al., 2006). A recent study in Iran reported significant positive associations between potato intake and metabolic syndrome, diabetes, HDL-C, hyperlipidemia

and fasting blood sugar level adjusted for age, sex, physical activity and dietary intakes (Khosravi-Boroujeni, Mohammadifard, et al., 2012). The high glycemic load of potato and high fat content of french fries are likely mechanism by which the risk of type 2 diabetes is increased.

Decreased risk of CVD was observed by greater consumption of breakfast cereal (≥4 times/week) in the full sample and greater consumption of the grains products among males (>18 times/week). Likewise, American postmenopausal women with intake of >3 g/1000 kcal of cereal fiber or >6 servings of whole grains per week had less progression of coronary atherosclerosis (Erkkilä, Herrington, Mozaffarian, & Lichtenstein, 2005). Additionally, cereal fiber consumption in elderly individuals >65 y after adjustment for socio-demographic characteristics, diabetes, fruit and vegetable fiber intake, and alcohol intake was associated with 21% lower CVD risk in the highest quintile of intake compared with the lowest quintile (Mozaffarian et al., 2003). Intake of cereal fiber may represent some other health-related behavior such as lower meat and saturated fat intake (Mozaffarian, et al., 2003). Further, Mozaffarian et al. reported the intake of cereal could slightly change blood pressure, serum lipid and glucose measures (Mozaffarian, et al., 2003).

Among Canadian males SSBs intake in the third quartile (1< intake<4) decreased the risk of having ≥10% of CVD risk compared to the first quartile significantly. The increased risk of cardiometabolic risks with high SSBs consumption has been evaluated in cohort studies, reviews and in epidemiological evidence (Dhingra, et al., 2007; Fung, et al., 2009; Hu & Malik, 2010; Malik, Popkin, Bray, Després, & Hu, 2010; Malik, et al., 2006). The U-shaped association between SSBs intake and having ≥10% of CVD risk was observed in the current study. Further prospective studies with greater sample size are needed to evaluate the impact of SSBs intake on 10-year risk of CVD among Canadians.

7.5   Strengths & Limitations

Data from our nationally representative cross-sectional study do not indicate any causal relationship. Also, conducting a follow-up study with endpoint cardiovascular events and mortality to find out if the higher predicted risk better predicts the future events may be beneficial. The adopted approach for determining "cardiovascular age" appears applicable to use at the population level although further validation of the formula might be needed. The dietary

intake assessment in CHMS is not a complete dietary assessment and the quantity of the consumption is not provided. However, our population based study is innovative in utilizing mFRS at the population level.

7.6 Conclusion

The estimated 10-year risk of CVD in Canadian adult population aged 30-74 y significantly increased after applying the two modifications suggested by CCS (8.66% versus 6.06%). Considering both modifications and specifically "cardiovascular age" in men is beneficial. Identifying high-risk Canadian individuals at younger age by calculating their cardiovascular age and considering the history of premature CVD in their family is substantial. These individuals can achieve the long-term advantage of risk factor modification and prevention strategies. Low intake of fruit, vegetable, and breakfast cereal and greater intake of potato along with low education level and abdominal obesity were the predictors of increasing 10-year risk of CVD ≥10% among Canadian adults. These findings indicate the need for community-based CVD primary prevention programs in lower educated population groups.

CHAPTER 8

GENERAL DISCUSSION

This chapter contains three parts: first a discussion of the descriptive dietary intakes of Canadians aged 6-79 y; second, an examination of the status of the health outcomes related to metabolic syndrome and diseases arising from it, among Canadian adolescents and adults; and lastly, an evaluation of the associations between all these health outcomes and dietary intake.

8.1   Dietary Intake among Canadians aged 6-79 y

In this thesis, dietary intakes of Canadians were reported by socio-demographic characteristics. Notable findings were that adolescents (12-19 y) had the greatest consumption of SSBs and fat compared to other age groups of Canadians. Fruit and vegetable juice intake was greatest among both children and adolescents compared to other groups. Grains and milk product consumption was lowest among middle-aged individuals (40-59 y). These findings are expected as similar results have been reported using the 2004 Canadian Community Health Survey (CCHS) (Garriguet, 2007, 2008a, 2008b). According to the CCHS data children had the greatest intake of fruit juice and adolescents had the greatest intake of SSBs (Garriguet, 2008b). Additionally, the average amount of milk consumption dropped with advancing age; however, the average increased after age 71 (Garriguet, 2008a). These findings using CCHS data are similar to what is reflected in **Figure 4-3** in this thesis although the age categorization is different. Making the right dietary and lifestyle choices early in life will help young people develop health-promoting behaviors that they can follow throughout life.

Active individuals had more milk and dairy product, fresh fruit and vegetable, and fruit and vegetable juice compared to inactive Canadians. In this research adjusting for energy intake was not possible; therefore, it is probable that a greater total intake (kcal) of physically active individuals may be one explanation for this finding. There is little research on evaluating the dietary pattern of physically active individuals compared to inactive. Among the elite athletes dairy products were the most important energy source after grains and cereals (van Erp-Baart, Saris, Binkhorst, Vos, & Elvers, 1989). In a cross-sectional study among postmenopausal women, active individuals had greater whole dairy products compared to inactive individuals

(Silva, Alves, Maturana, & Spritzer, 2013). However, fruit and vegetable intake was not different between groups in that study. Further research should be undertaken to determine the dietary pattern of physically active Canadians while adjusting for energy intake.

Canadians from low-income households reported having greater amounts of SSBs and lower amounts of fresh fruit and vegetable compared to Canadians from high-income households. Expensive and cheap supply of fruit and vegetable, and SSBs, respectively, might have an impact on the amount of intake. According to CCHS data, more children having the dietary (beverage) pattern dominated by SSBs lived in households with low income compared to children in other beverage groups (Danyliw, 2010). Six barriers were investigated to explain why there is poorer access among low-income households to fruit and vegetables: cost, transportation, quality, variety, changing food environment, and changing societal norms on food (Haynes-Maslow, et al., 2013). Cost of food has been found as the most important factor that could prevent or encourage low income households to purchase food (Hendrickson, et al., 2006; Herman, et al., 2006). Poor families or communities are the ones surrounded most by unhealthy diets all around the world. Fat and especially sugar in the form of beverages are the cheapest and easiest source to meet calorie requirement (World Heart Federation, 2014). Additionally, the vast marketing of SSBs and fast foods make them even more accessible than fruit and vegetable for low income communities.

Not only income but also education has remarkable impact on food choices. Individuals with "some postsecondary education" had the greatest intake of SSBs compared to individuals with other levels of education. Individuals with "postsecondary graduation" had lower intake of SSBs compared to low educated individuals (**Table 4-2**). Danyliw et al. in their research on Canadian children did not find a difference in soft drink intake with education (Danyliw, 2010). However, in other research using CCHS data on adults, individuals whose beverage intake were dominated by SSBs did not have high level of education compared to individuals who were in a different cluster of beverage intake (Nikpartow, et al., 2012). Education and income both need to be considered in generating strategies or changing policies. Three factors should be targeted: First, policies towards regulating food prices need to make healthy foods cheaper and more accessible. Second, legislations should focus on reducing the promotion of unhealthy foods, especially to adolescents and children. Third, school health programs can educate children,

parents, and teachers about the impact of diet on health and help them to make healthy choices (World Heart Federation, 2014).

8.2   Status of health outcomes related to metabolic syndrome in Canada

The estimated overall prevalence of abdominal obesity (12-79 y), elevated TC/HDL-C ratio (20-79 y), MetS (12-79 y), and diabetes (20-79 y) among Canadians were 35.7%, 20.5%, 18.3%, and 7.5%, respectively. The prevalence of abdominal obesity was significantly greater among Canadian women which was similar to Spanish women (Gutiérrez-Fisac, et al., 2012), Portuguese women (Sardinha, et al., 2012), Chinese women (Wang, Liu, et al., 2012), English women (Howel, 2012), and American women (Ford, et al., 2010). Elevated TC/HDL-C ratio was significantly greater among Canadian men and in middle-aged individuals (40-59 y) similar to previous research (Prospective Studies Collaboration, 2007).

Abdominal obesity appeared to associate with several health outcomes as evidence has indicated (Folsom et al., 2000; Onat et al., 2007; Zhang, Rexrode, van Dam, Li, & Hu, 2008). It is the most prevalent component to develop MetS. Additionally, abdominal obesity was the strong predictor associated with risk of diabetes, elevated TC/HDL-C ratio and having 10-year risk of CVD ≥10% (Balkau, et al., 2007; Hamdy, et al., 2006; Nieves, et al., 2003).

Among the components to develop MetS, reduced HDL-C was as prevalent as abdominal obesity in Canadian adolescents similar to American adolescents (Duncan, et al., 2004). Further research is required to explore the causation. However, one possible reason might be due to biologically reduced HDL-C levels especially among boys over 12 y during puberty (Morrison, et al., 1979; Porkka, et al., 1991). Abdominal obesity and reduced HDL-C as the most prevalent components in adolescents could help to bring about strategies to decrease the prevalence of MetS in adulthood by focusing earlier in the lifecycle for prevention.

The average 10-year risk of CVD was 8.66%. Two approaches were implemented: modifying FRS or considering individuals with MetS (Anderson, et al., 2013; Leiter, et al., 2011a). Both approaches significantly increased the overall 10-year risk of CVD among Canadians. Data on CMR indicated using either LDL-C or Tchol makes no difference in obtaining 10-year risk of CVD. However, in calculating CMR several limitations are present.

Three risk factors were considered twice: reduced HDL-C, high blood pressure, and diabetes. Additionally, only the fasted sub-sample can be used in CMR due to the presence of the fasting plasma glucose risk factor to define MetS. However, there are no such limitations when FRS is modified considering risk age and the history of premature cardiovascular event in the first-degree relative of the individual. Thus, modifying FRS may give more accurate estimation of the 10-year risk of CVD.

After modifying FRS the majority of the Canadians were classified in the low risk category of CVD (67.4%), a rate similar to Americans (Ford, W. H. Giles, et al., 2004). The distribution categorized in the low risk decreased (79.6% to 67.4%) whereas the distribution categorized in the high risk increased (4.5% to 13.4%) significantly after modifying FRS. Of the two modifications, risk age had the greater contribution in changing the overall 10-year risk of CVD especially among men. Therefore, in situations where obtaining the history of premature cardiovascular event in the first-degree relative of a person is difficult, calculating risk age using the CVD charts should be enough to estimate a precise 10-year risk of CVD at the individual level.

8.3   Association between dietary intake and health outcomes

All the chronic diseases and their risk factors examined in the thesis were interrelated. Abdominal obesity, and elevated TC/HDL-C ratio were the risk factors for MetS, and diabetes and CVD can result from MetS. The significant predictors in the final model were similar for abdominal obesity and MetS. This might be due to abdominal obesity being the greatest risk factor for developing MetS among Canadians.

A high consumption of diet soft drink was associated with risk of abdominal obesity, MetS, diabetes, and elevated TC/HDL-C ratio. Two population-based prospective studies found diet soft drink consumption is associated with risk of vascular events (Gardener, et al., 2012; Nettleton, et al., 2009). Consuming ≥ 1 daily serving of diet soda gave a significantly greater risk of having high waist circumference or having a higher fasting glucose (Nettleton, et al., 2009). One suggested reason was that diet soft drink might be consumed in greater amounts by those individuals who were aware of their metabolic disorders such as abdominal obesity or diabetes

(Gardener, et al., 2012). Further research is required to determine e causality between diet soda and specific health outcomes.

Low intakes of fruit and vegetables and of cereals or high potato consumption were associated with ≥10% risk of CVD for 10 years. The findings on cereal and fruit and vegetable intake were examined in several studies (Dauchet, et al., 2006; Erkkilä, et al., 2005; Joshipura, et al., 2001; Mozaffarian, et al., 2003). Cereal and fruit and vegetable intakes are usually representative of positive health-related behaviours such as having lower meat and saturated fat intakes (Mozaffarian, et al., 2003). Thus, individuals with greater consumption of fiber from cereal and fruit and vegetable may adhere to an overall healthy diet and follow other lifestyle recommendations. Additionally, evidence indicates a positive biologic effect of dietary fibers such as changing blood pressure, serum lipid, glycemia and insulin sensitivity. Potato mainly represented high calorie food in this thesis. In two studies potato consumption in the form of french fries or baked was associated with increased risk of type 2 diabetes probably due to high glycemic load of potato (Halton, et al., 2006; Khosravi-Boroujeni, Mohammadifard, et al., 2012). Further evidence is required evaluating the impact of potato consumption on the risk of chronic diseases. Further longitudinal research is warranted considering different foods made with potato.

Intake of nuts appeared to be associated with a reduction in the risk of MetS among Canadians. Several studies indicated the association between nuts and reduced risk of chronic diseases. Nuts could decrease oxidative damage to protein (Jenkins, et al., 2006; Torabian, et al., 2009), inflammation (Jiang, et al., 2006), prevalence of risk factors for MetS (O'Neil, et al., 2011), serum lipid in diabetics (Jenkins, et al., 2011), insulin resistance (Casas-Agustench, et al., 2011), and endothelial dysfunction (Ma, et al., 2010). In this thesis and other studies, consuming nuts ≥ 3 times/week was beneficial. Research is still required to determine the optimal amount to consume as well as the type of nuts in association with risk of diseases.

Considering the factors entered to the models, the risk of abdominal obesity, MetS, and diabetes were associated with the level of physical activity. Also, level of education was a significant predictor of abdominal obesity. There is evidence that in order to properly manage abdominal obesity, intensity and duration of physical activity as well as dietary intake needs to

be monitored. In fact, only increasing physical activity does not reduce abdominal obesity in a dose-response manner (Ross & Janssen, 2001).

Concern is growing about the increasing rate of obesity as it is distributed differently across social groups by levels of education. Evidence indicated that educated individuals are able to make better use of health related information than those who are less educated (Kemna, 1987). The lack of education about energy contents of foods has also been suggested to influence social class on obesity (Cutler & Lleras-Muney, 2006). Diet and physical activity are always the two important contributors to prevent and manage chronic diseases and risk factors. Educating at risk individuals might be another effective contributor.

The health outcomes evaluated in this research share common risk factors. These modifiable factors are influenced and shaped by societal, economic and physical conditions. Changing health behaviors and biological factors have the potential to reduce the risk of these health outcomes in the Canadian population. The factors which remained significant were physical activity, being educated, consuming fibers from fruit and vegetable and cereals, having less diet soft drink, and consuming more nuts as snacks.

8.4   Strengths & Limitations

The strength of the present studies in this thesis is the use of CHMS which is a large nationally representative survey of Canadian men and women. Using CHMS data is advantageous due to having direct health measurement in order to identify individuals with undiagnosed diabetes, elevated TC/HDL-C ratio, increased risk of CVD, and MetS. To my knowledge there is no study using a population-based sample in this scale to evaluate the association between different food groups and specific health outcomes in Canada adjusting for potential factors. Identification of MetS in Canadian adolescents using the age and sex specific criteria was the first ever conducted, as reported in this thesis. Furthermore, novel approaches were implemented for study 3 and 4. Calculating CMR using FRS and cardiovascular risk age using a specific formula at the population level were first applied in studies in this thesis.

However, several limitations are present in this research. Similar to other studies based on large survey data, the studies presented in this thesis may have some limitations due to the

study design in survey data and analytical approaches. Data from the current nationally representative cross-sectional study do not indicate any causal relationship. Moreover, the effect of potential confounders, which were not possible to consider in the current thesis such as total energy intake, cannot be ignored.

Although CHMS collected usual dietary intake through semi-quantitative questionnaire, the quantity of the consumption was not provided, therefore it was not possible to obtain an exact measure of dietary intake. The errors in the measure of diet are likely to bias associations toward the null hypothesis (no association) which could attenuate the estimates of disease relative risks (Kipnis et al., 2003). This limitation might be stronger for some food and beverage groups. It appeared that the results on beverage intake such as SSBs, diet soft drink, and fruit and vegetable juice need further research. Also, data on dietary fat intake appear to need more investigation.

The fasted subsample used in the study of MetS and CMR was smaller than the whole CHMS sample. The approach of determining the absolute risk of CVD considering relative factor of about 1.5 to 2 in individuals with MetS is not fully validated in longitudinal studies, rather it is based on studies reporting that the presence of the metabolic syndrome is associated with a 1.5–2 increased risk, above and beyond Framingham Risk Score. Additionally, the formula to obtain risk age needs to be validated at the population level.

8.5   Future Research

Data from CHMS are innovative in recognizing MetS cases, abdominally obese individuals, undiagnosed diabetics, individuals with elevated TC/HDL-C ratio, and individuals with great risk of CVD over 10 years. Further cycles of CHMS in upcoming years are needed with a greater sample to obtain a comprehensive picture and statistical model of each chronic diseases or risk factors among Canadians.

The adopted approach for determining "cardiovascular age" or CMR appears applicable to use at the population level although further validation of such approaches is required.

Further research is required on overall dietary patterns using data with a more comprehensive dietary assessment to improve the understanding of the numerous modifiable

determinants of disease risk that may guide the development of innovative, focused, and individualized preventative strategies.

Studies suggest many approaches with significant potential to reduce the risk of chronic diseases. Tobacco use, physical inactivity, and unhealthy diet are the three important risk factors to develop chronic disease. According to this thesis education programs to specific age and sex group could also be beneficial. Managing these factors is primary prevention of chronic diseases and risk factors. We may suggest that public health practitioners focus more on environmental changes that promote healthy eating such as type of food available in schools, access to healthier foods such as fruit and vegetable, cereals, and nuts, public education especially for at risk individuals such as abdominally obese individuals. Promoting physical activity through appropriate environmental and policy approaches would help. Increase access to facilities, building walking or biking trails, or focusing on recreational activities could be beneficial approaches.

Finally in order to develop appropriate recommendations to identify gaps in existing knowledge and to establish priorities for preventative strategies future research should be directed toward improved longitudinal studies in order to make the best of primary prevention of CVD or MetS and the risk factors among specific age, sex, and risk groups. Longitudinal research should mostly target adolescents as their knowledge is limited and any effort to change their diet and lifestyle could sustain into adulthood.

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APPENDICES

Appendix 1

Risk prediction chart, Framingham Risk Score (FRS) for males

Calculating Global CHD Risk in Men

Step 1: Age

Years	Points	Years	Points
30 to 34	-1	55 to 59	4
35 to 39	0	60 to 64	5
40 to 44	1	65 to 69	6
45 to 49	2	70 to 74	7
50 to 54	3		

Step 2: LDL or TC Level

LDL

mg per dL	mmol per L	Points
< 100	< 2.59	-3
100 to 129	2.59 to 3.35	0
130 to 159	3.36 to 4.13	0
160 to 190	4.14 to 4.92	1
> 190	> 4.92	2

TC

mg per dL	mmol per L	Points
< 160	< 4.14	-3
160 to 199	4.14 to 5.16	0
200 to 239	5.17 to 6.20	1
240 to 279	6.21 to 7.23	2
≥ 280	≥ 7.24	3

Step 3: HDL Level

mg per dL	mmol per L	Points (if LDL used in step 2)	Points (if TC used in step 2)
< 35	< 0.91	2	2
35 to 44	0.91 to 1.15	1	1
45 to 49	1.16 to 1.28	0	0
50 to 59	1.29 to 1.54	0	0
≥ 60	≥ 1.55	-1	-2

Step 4: Blood Pressure

Systolic (mm Hg)	Diastolic (mm Hg) < 80	80 to 84	85 to 89	90 to 99	≥ 100
< 120	0 points				
120 to 129		0 points			
130 to 139			1 point		
140 to 159				2 points	
≥ 160					3 points

NOTE: When systolic and diastolic pressures provide different point scores, use the higher score.

Step 5: Diabetes Mellitus

Present?	Points
No	0
Yes	2

Step 6: Smoking

Smoker?	Points
No	0
Yes	2

Step 7: Total Points

Step 1: Age	___
Step 2: LDL or TC level	___
Step 3: HDL level	___
Step 4: Blood pressure	___
Step 5: Diabetes mellitus	___
Step 6: Smoking	___
Total points	___

Step 8: CHD Risk

Total points	10-year risk if LDL used in step 2 (%)	10-year risk if TC used in step 2 (%)
≤ -3	1	—
-2	2	—
-1	2	2
0	3	3
1	4	3
2	4	4
3	6	5
4	7	7
5	9	8
6	11	10
7	14	13
8	18	16
9	22	20
10	27	25
11	33	31
12	40	37
13	47	45
≥ 14	≥ 56	≥ 53

Step 9: Comparative Risk

Age (years)	Average 10-year CHD risk (%)	Average 10-year risk of hard event* (%)	Low 10-year CHD risk† (%)
30 to 34	3	1	2
35 to 39	5	4	3
40 to 44	7	4	4
45 to 49	11	8	4
50 to 54	14	10	6
55 to 59	16	13	7
60 to 64	21	20	9
65 to 69	25	22	11
70 to 74	30	25	14

*—Hard events exclude angina pectoris.
†—Low risk as calculated for a man of the same age who does not smoke or have diabetes, and has optimal blood pressure, an LDL level of 100 to 129 mg per dL or TC level of 160 to 199 mg per dL, and an HDL level of 45 mg per dL.

Key

Color	Relative risk	Color	Relative risk
Green	Very low	Orange	High
White	Low	Red	Very high
Yellow	Moderate		

Appendix 2

Risk prediction chart, Framingham Risk Score (FRS) for females

Calculating Global CHD Risk in Women

Step 1: Age

Years	Points	Years	Points
30 to 34	−9	55 to 59	7
35 to 39	−4	60 to 64	8
40 to 44	0	65 to 69	8
45 to 49	3	70 to 74	8
50 to 54	6		

Step 2: LDL or TC Level

LDL

mg per dL	mmol per L	Points
< 100	< 2.59	−2
100 to 129	2.59 to 3.35	0
130 to 159	3.36 to 4.13	0
160 to 190	4.14 to 4.92	2
> 190	> 4.92	2

TC

mg per dL	mmol per L	Points
< 160	< 4.14	−2
160 to 199	4.14 to 5.16	0
200 to 239	5.17 to 6.20	1
240 to 279	6.21 to 7.23	1
≥ 280	≥ 7.24	3

Step 3: HDL Level

mg per dL	mmol per L	Points (if LDL used in step 2)	Points (if TC used in step 2)
< 35	< 0.91	5	5
35 to 44	0.91 to 1.15	2	2
45 to 49	1.16 to 1.28	1	1
50 to 59	1.29 to 1.54	0	0
≥ 60	≥ 1.55	−2	−3

Step 4: Blood Pressure

Systolic (mm Hg)	Diastolic (mm Hg) < 80	80 to 84	85 to 89	90 to 99	≥ 100
< 120	−3 points				
120 to 129		0 points			
130 to 139			0 points		
140 to 159				2 points	
≥ 160					3 points

NOTE: When systolic and diastolic pressures provide different point scores, use the higher score.

Step 5: Diabetes Mellitus

Present?	Points
No	0
Yes	4

Step 6: Smoking

Smoker?	Points
No	0
Yes	2

Step 7: Total Points

Step 1: Age	_____
Step 2: LDL or TC level	_____
Step 3: HDL level	_____
Step 4: Blood pressure	_____
Step 5: Diabetes mellitus	_____
Step 6: Smoking	_____
Total points	_____

Step 8: CHD Risk

Total points	10-year risk if LDL used in step 2 (%)	10-year risk if TC used in step 2 (%)
≤ −2	1	1
−1	2	2
0	2	2
1	2	2
2	3	3
3	3	3
4	4	4
5	5	4
6	6	5
7	7	6
8	8	7
9	9	8
10	11	10
11	13	11
12	15	13
13	17	15
14	20	18
15	24	20
16	27	24
≥ 17	≥ 32	≥ 27

Step 9: Comparative Risk

Age (years)	Average 10-year CHD risk (%)	Average 10-year risk of hard event* (%)	Low 10-year CHD risk† (%)
30 to 34	< 1	< 1	< 1
35 to 39	< 1	< 1	1
40 to 44	2	1	2
45 to 49	5	2	3
50 to 54	8	3	5
55 to 59	12	7	7
60 to 64	12	8	8
65 to 69	13	8	8
70 to 74	14	11	8

*—Hard events exclude angina pectoris.
†—Low risk as calculated for a woman of the same age who does not smoke or have diabetes, and has optimal blood pressure, an LDL level of 100 to 129 mg per dL or TC level of 160 to 199 mg per dL, and an HDL level of 55 mg per dL (1.42 mmol per L).

Key

Color	Relative risk	Color	Relative risk
Green	Very low	Orange	High
White	Low	Red	Very high
Yellow	Moderate		

Appendix 3, Tables of univariate binary logistic analyses for five health outcomes

Table 1 Separate binary logistic regression analysis for each socio-demographic predictor of abdominal obesity (population size: all=26 483 831, male=13 171 095, female=13 312 736)

Predictors	Odds Ratio(95% Confidence Interval) P-value		
Age Groups	**All**	**Male**	**Female**
12-19 y[1]			
20-39 y	2.77(1.71-4.47)	3.13(1.13-8.69)	2.57(1.68-3.92)
	<0.01*	0.031*	<0.001*
40-59 y	4.77(3.07-7.39)	6.23(2.48-15.66)	3.93(2.55-6.07)
	<0.001*	<0.01*	<0.001*
60-79 y	9.03(6.03-13.51)	10.81(4.27-27.37)	8.04(5.40-11.98)
	<0.001*	<0.001*	<0.001*
Education			
< secondary school graduation[1]			
Secondary school graduation	0.63(0.41-0.97)	0.54(0.28-1.05)	0.75(0.46-1.23)
	0.038*	0.070*	0.231
Some postsecondary	0.54(0.31-0.94)	0.56(0.32-0.98)	0.56(0.25-1.24)
	0.033*	0.043*	0.139*
Postsecondary graduation	0.41(0.28-0.58)	0.42(0.25-0.70)	0.41(0.26-0.65)
	<0.001*	<0.01*	<0.01*
Income			
Lowest income[1]			
Lower-middle income	1.45(1.09-1.94)	1.84(0.58-3.55)	1.37(0.98-1.90)
	0.016*	0.064*	0.060*
Upper-middle income	0.91(0.63-1.32)	1.06(0.56-1.99)	0.95(0.64-1.43)
	0.609	0.840	0.813
Highest income	0.79(0.53-1.16)	1.05(0.41-2.66)	0.75(0.47-1.18)
	0.206	0.909	0.189*
Physical activity			
Active[1]			
Moderately active	1.77(1.16-2.72)	1.72(1.12-2.63)	1.72(0.98-3.01)
	0.013*	0.018*	0.056*
Inactive	2.72(2.03-3.56)	2.85(2.00-1.06)	2.44(1.69-3.54)
	<0.01*	<0.001*	<0.001*
Smoking[2]	0.80(0.66-0.99)	0.67(0.44-1.02)	0.97(0.78-1.23)
	0.042*	0.059*	0.831
Ethnicity[3]	1.06(0.70-1.61)	0.93(0.44-1.95)	1.17(0.75-1.84)

	0.750	0.834	0.446
Alcohol intake[4]	0.96(0.71-1.28)	1.06(0.64-1.75)	0.94(0.67-1.30)
	0.742	0.798	0.674

* Significantly different from the reference group
[1]Reference group
[2]Smokers=1(reference group) versus non-smokers=0
[3]White individuals=1(reference group) versus non-white=0
[4]Ever drinkers= 1 (reference group) versus never drinkers=0

Table 2 Separate binary logistic regression analysis between each food group and abdominal obesity (population size: all=26 483 831, male=13 171 095, female=13 312 736)

Predictors	**Odds Ratio(95% Confidence Interval)**		
	P-value		
Meat & Fish	**All**	**Male**	**Female**
1st Q[1]			
2nd Q	0.90(0.66-1.22)	0.89(0.54-1.49)	0.93(0.64-1.37)
	0.461	0.654	0.702
3rd Q	0.78(0.63-0.98)	0.72(0.46-1.13)	0.92(0.72-1.19)
	0.037*	0.138*	0.511
4th Q	0.75(0.49-1.14)	0.73(0.47-1.14)	0.85(0.46-1.56)
	0.162*	0.153*	0.573
Grains			
1st Q[1]			
2nd Q	0.86(0.68-1.11)	0.73(0.47-1.11)	1.02(0.65-1.61)
	0.235	0.130*	0.916
3rd Q	0.67(0.53-0.89)	0.50(0.30-0.82)	0.86(0.61-1.23)
	<0.01*	0.011*	0.376
4th Q	0.77(0.56-1.05)	0.79(0.52-1.18)	0.79(0.53-1.16)
	0.098*	0.226	0.205
Fruit & Vegetable			
1st Q[1]			
2nd Q	0.91(0.97-1.23)	0.88(0.60-1.28)	0.88(0.61-1.26)
	0.515	0.472	0.456
3rd Q	0.93(0.70-1.24)	0.94(0.66-1.34)	0.81(0.51-1.26)
	0.597	0.714	0.318
4th Q	0.79(0.55-1.13)	0.59(0.39-0.88)	0.74(0.45-1.22)
	0.178*	0.014*	0.216
Dietary Fat			
1st Q[1]			
2nd Q	0.77(0.30-1.01)	0.77(0.57-1.04)	0.80(0.52-1.24)
	0.074*	0.089*	0.289
3rd Q	0.70(0.38-1.12)	0.80(0.64-1.00)	0.64(0.48-0.83)

	Col1	Col2	Col3
	<0.001*	0.054*	<0.01*
4th Q	0.64(0.48-0.83)	0.68(0.41-1.13)	0.68(0.47-1.00)
	<0.01*	0.124*	0.053*

Milk & Dairy Products

	Col1	Col2	Col3
1st Q [1]			
2nd Q	0.84(0.61-1.15)	0.78(0.48-1.26)	0.90(0.63-1.25)
	0.253	0.284	0.460
3rd Q	0.83(0.66-1.03)	0.80(0.64-1.00)	0.91(0.62-1.32)
	0.092*	0.054*	0.581
4th Q	0.64(0.48-0.83)	0.40(0.24-0.67)	0.85(0.56-1.28)
	<0.01*	<0.01*	0.398

Sugar Sweetened Beverages

	Col1	Col2	Col3
1st Q [1]			
2nd Q	0.75(0.56-1.02)	0.68(0.39-1.20)	0.82(0.56-1.18)
	0.063*	0.167*	0.250
3rd Q	0.61(0.34-1.04)	0.54(0.40-0.73)	0.78(0.53-1.10)
	<0.01*	<0.01*	0.183*
4th Q	0.52(0.27-0.61)	0.47(0.26-0.83)	0.68(0.46-1.01)
	<0.01*	0.015*	0.053*
Diet Soft Drink[2]	1.46(1.20-1.79)	1.65(1.27-2.16)	1.30(0.94-1.79)
	<0.01*	<0.01*	0.102*

Fruit & Vegetable Juice

	Col1	Col2	Col3
1st Q [1]			
2nd Q	0.76(0.63-0.92)	0.67(0.44-1.03)	0.86(0.66-1.13)
	<0.01*	0.068*	0.254
3rd Q	0.68(0.55-0.84)	0.45(0.33-0.62)	0.94(0.60-1.47)
	<0.01*	<0.001*	0.775
4th Q	0.73(0.56-0.95)	0.55(0.37-0.81)	0.98(0.72-1.34)
	0.022*	0.006*	0.915

Potato consumption

	Col1	Col2	Col3
1st Q [1]			
2nd Q	0.81(0.60-1.11)	0.78(0.46-1.30)	0.89(0.61-1.32)
	0.185*	0.315	0.544;
3rd Q	0.86(0.69-1.07)	0.79(0.51-1.22)	1.05(0.81-1.37)
	0.165*	0.263	0.677
4th Q	1.24(0.97-1.61)	1.10(0.72-1.66)	1.59(1.15-2.19)
	0.080*	0.623	<0.01

*Statistically significant from the reference group
[1]Reference group
[2] Diet soft drink categorized into 2 categories (0 times/day versus > 0 times/day (reference group)).

Table 3 Separate binary logistic regression analysis for each socio-demographic predictor of elevated TC/HDL-C ratio (population size: all=23 135 787, male=11 440 786, female=11 695 001)

Predictors	Odds Ratio(95% Confidence Interval) P-value		
Age Groups	**All**	**Male**	**Female**
20-39 y[1]			
40-59 y	1.86(1.42-2.45) <0.01*	1.81(1.20-2.73) 0.008*	2.25(1.30-3.89) 0.008*
60-79 y	1.38(1.05-1.83) 0.025*	1.32(0.93-1.88) 0.106*	1.76(0.99-3.11) 0.050*
Education			
< secondary school graduation[1]			
Secondary school graduation	0.90(0.63-1.28) 0.549	0.84(0.45-1.57) 0.564	0.93(0.37-2.36) 0.870
Some postsecondary	0.66(0.37-1.15) 0.132*	0.83(0.36-1.90) 0.634	0.37(0.14-0.96) 0.042*
Postsecondary graduation	0.73(0.44-1.20) 0.194*	0.86(0.28-0.58) 0.468	0.46(0.20-1.07) 0.069*
Income			
Lowest income[1]			
Lower-middle income	1.09(0.54-2.23) 0.777	0.90(0.25-3.18) 0.861	1.15(0.31-4.22) 0.816
Upper-middle income	1.04(0.51-2.12) 0.911	0.91(0.24-3.35) 0.873	0.83(0.22-3.09) 0.765
Highest income	1.26(0.56-2.79) 0.541	1.12(0.30-4.22) 0.850	0.82(0.26-2.57) 0.705
Physical activity			
Active[1]			
Moderately active	1.22(0.81-1.83) 0.296	1.27(0.73-2.20) 0.364	1.72(0.62-3.46) 0.056*
Inactive	1.27(0.86-1.86) 0.192*	1.37(0.78-2.39) 0.241	2.44(0.79-3.01) <0.001*
Smoking[2]	1.05(1.05-1.92) 0.025*	0.67(0.68-1.62) 0.791	0.97(0.20-0.78) 0.831
Ethnicity[3]	1.22(0.65-1.53) 0.955	0.98(0.55-1.75) 0.953	1.17(0.67-2.22) 0.446
Alcohol intake[4]	0.53(0.27-1.05) 0.068*	0.45(0.16-1.23) 0.112*	0.94(0.67-1.30) 0.674

*Statistically significant from the reference group
[1]Reference group
[2]Smokers=1(reference group) versus non-smokers=0

Table 4 Separate binary logistic regression analysis between each food group and elevated total/HDL cholesterol ratio (population size: all=23 135 787, male=11 440 786, female=11 695 001)

Predictors	Odds Ratio(95% Confidence Interval) P-value		
Meat & Fish	**All**	**Male**	**Female**
$1^{st} Q^1$			
$2^{nd} Q$	0.95(0.64-1.40)	0.94(0.52-1.69)	0.84(0.43-1.62)
	0.788	0.824	0.572
$3^{rd} Q$	0.91(0.64-1.29)	0.84(0.47-1.48)	0.71(0.47-1.05)
	0.562	0.513	0.082*
$4^{th} Q$	0.79(0.55-1.13)	0.79(0.49-1.28)	0.45(0.29-0.69)
	0.180*	0.314	0.002*
Grains			
$1^{st} Q^1$			
$2^{nd} Q$	1.25(0.78-1.99)	1.34(0.722.51)	1.05(0.57-1.94)
	0.305	0.323	0.864
$3^{rd} Q$	1.09(0.78-1.53)	0.95(0.53-1.71)	1.42(0.74-2.71)
	0.563	0.868	0.261
$4^{th} Q$	1.17(0.80-1.73)	1.06(0.66-1.71)	1.25(0.68-2.28)
	0.372	0.786	0.429
Fruit & Vegetable			
$1^{st} Q^1$			
$2^{nd} Q$	0.97(0.68-1.39)	1.01(0.79-1.72)	1.09(0.71-1.67)
	0.872	0.958	0.654
$3^{rd} Q$	0.91(0.64-1.29)	1.32(0.89-1.95)	0.74(0.31-1.78)
	0.569	0.145*	0.469
$4^{th} Q$	0.58(0.44-0.78)	0.66(0.44-0.98)	0.88(0.54-1.43
	0.002*	0.043*	0.584
Dietary Fat			
$1^{st} Q^1$			
$2^{nd} Q$	0.98(0.30-1.01)	0.95(0.57-1.04)	0.85(0.52-1.24)
	0.923	0.865	0.319
$3^{rd} Q$	1.09(0.38-1.12)	0.71(0.64-1.00)	1.25(0.48-0.83)
	0.431	0.043*	0.410
$4^{th} Q$	1.36(0.48-0.83)	0.78(0.41-1.13)	1.69(0.47-1.00)
	0.020*	0.168*	0.015*
Milk & Dairy Products			
$1^{st} Q^1$			

2nd Q	0.76(0.57-1.01)	0.68(0.42-1.11)	1.13(0.73-1.73)
	0.058*	0.115*	0.553
3rd Q	1.25(0.80-1.59)	1.21(0.89-1.06)	1.67(1.01-2.76)
	0.070*	0.203	0.045*
4th Q	0.77(0.51-1.16)	1.02(0.62-1.67)	0.73(0.48-1.11)
	0.191*	<0.938	0.130*
Sugar Sweetened Beverages			
1st Q [1]			
2nd Q	1.02(0.68-1.53)	1.14(0.68-1.88)	0.81(0.44-1.46)
	0.900	0.588	0.450
3rd Q	1.35(1.02-1.80)	0.92(0.66-1.29)	1.33(0.75-2.36)
	0.040*	0.621	0.296
4th Q	1.44(1.05-1.96)	0.84(0.60-1.18)	1.67(0.93-2.98)
	0.025*	0.286	0.078*
Diet Soft Drink[2]	1.24(0.98-1.57)	1.54(1.16-2.05)	1.00(0.66-1.51)
	0.066*	0.006*	0.973
Fruit & Vegetable Juice			
1st Q [1]			
2nd Q	1.03(0.70-1.52)	1.11(0.71-1.75)	0.91(0.50-1.65)
	0.832	0.614	0.728
3rd Q	0.90(0.60-1.35)	0.80(0.53-1.19)	1.09(0.57-2.10)
	0.586	0.246	0.759
4th Q	1.24(0.72-2.17)	1.03(0.65-1.62)	1.54(0.65-3.67)
	0.396	0.892	0.295

* Statistically significant from the reference group
[1] Reference group
[2] Diet soft drink categorized into 2 categories (0 times/day and > 0 times/day (reference group)).

Table 5 Separate binary logistic regression analysis for each socio-demographic predictor of diabetes (population size=22 674 040)

Predictors	Odds Ratio	P-value	95% Confidence Interval
Sex[1]	1.18	0.319	0.83-1.70
Age Groups			
20-39 y[2]			
40-59 y	8.55	<0.001[*]	5.02-14.58
60-79 y	20.22	<0.001[*]	11.36-36.01
Education			
< secondary school graduation[2]			
Secondary school graduation	0.44	0.025[*]	0.22-0.88
Some postsecondary	0.27	<0.01[*]	0.11-0.64
Postsecondary graduation	0.31	<0.01[*]	0.17-0.56
Income			
Lowest income[2]			
Lower-middle income	0.94	0.867	0.49-1.84
Upper-middle income	0.55	0.115[*]	0.25-1.18
Highest income	0.48	0.057[*]	0.22-1.03
Physical activity			
Active[2]			
Moderately active	1.38	0.154[*]	0.87-2.21
Inactive	2.27	<0.01[*]	1.43-3.60
Smoking[3]	0.72	0.126[*]	0.46-1.11
Ethnicity[4]	0.81	0.548	0.38-1.70
Alcohol intake[5]	0.39	<0.001[*]	0.26-0.59

[*] Statistically significant from the reference group
[1] Male considers 1 versus female zero
[2] Reference group
[3] Smokers=1(reference group) versus non-smokers=0
[4] White individuals=1 (reference group) versus non-white=0
[5] Ever drinkers= 1(reference group) versus never drinkers=0

Table 6 Separate binary logistic regression analysis between each food group and diabetes (population size=22 674 040)

Food groups	Odds Ratio	P-value	95% Confidence Interval
Meat & Fish			
1st Q^1			
2nd Q	0.83	0.315	0.56-1.22
3rd Q	0.84	0.507	0.48-1.46
4th Q	0.84	0.583	0.43-1.63
Grains			
1st Q^1			
2nd Q	1.02	0.909	0.63-1.67
3rd Q	0.93	0.810	0.48-1.79
4th Q	1.15	0.530	0.71-1.86
Fruit & Vegetable			
1st Q^1			
2nd Q	1.12	0.678	0.60-2.11
3rd Q	1.41	0.095	0.93-2.14
4th Q	0.93	0.750	0.57-1.51
Dietary Fat			
1st Q^1			
2nd Q	0.58	0.142[*]	0.27-1.23
3rd Q	0.75	0.055[*]	0.56-1.00
4th Q	0.55	0.097[*]	0.27-1.13
Milk & Dairy Products			
1st Q^1			
2nd Q	0.68	0.018[*]	0.50-0.92
3rd Q	1.05	0.869	0.52-2.12
4th Q	0.69	0.288	0.34-1.42
Sugar Sweetened Beverages			
1st Q^1			
2nd Q	0.36	<0.01[*]	0.20-0.66
3rd Q	0.36	<0.01[*]	0.19-0.66
4th Q	0.22	<0.01[*]	0.10-0.45
Diet Soft Drink[2]	2.49	<0.001[*]	1.72-3.59
Fruit & Vegetable Juice			
1st Q^1			
2nd Q	0.46	<0.01[*]	0.28-0.76
3rd Q	0.67	0.224	0.35-1.32
4th Q	0.68	0.085[*]	0.44-1.06

[*] Statistically significant from the reference group
[1] Reference group
[2] Diet soft drink categorized into 2 categories (0 times/day and >0 times/day(reference group)).

Table 7 Separate binary logistic regression analysis for each socio-demographic predictor of metabolic syndrome (population size=27 043 753)

Predictors	Odds Ratio	P-value	95% Confidence Interval
Sex[1]	0.87	0.315	0.65-1.16
Age Groups			
12-19 y[2]			
20-39 y	2.32	0.161[*]	0.68-7.94
40-59 y	7.51	<0.01[*]	2.39-23.63
60-79 y	18.82	<0.001[*]	6.48-54.65
Education			
< secondary school graduation[2]			
Secondary school graduation	0.68	0.290	0.32-1.45
Some postsecondary	0.18	<0.01[*]	0.06-0.53
Postsecondary graduation	0.26	<0.01[*]	0.13-0.50
Income			
Lowest income[2]			
Lower-middle income	1.70	0.084[*]	0.92-3.15
Upper-middle income	1.03	0.934	0.50-2.12
Highest income	0.65	0.235	0.32-1.37
Physical activity			
Active[2]			
Moderately active	2.15	<0.01[*]	1.26-3.65
Inactive	3.10	<0.001[*]	1.98-4.86
Smoking[3]	0.95	0.697	0.73-1.23
Ethnicity[4]	0.80	0.315	0.50-1.27
Alcohol intake[5]	1.05	0.881	0.51-2.17

[*] Statistically significant from the reference group
[1] Male considers 1 versus female zero
[2] Reference group
[3] Smokers=1(reference group) versus non-smokers=0
[4] White individuals=1(reference group) versus non-white=0
[5] Ever drinkers= 1 (reference group) versus never drinkers=0

Table 8 Separate binary logistic regression analysis between each food group and metabolic syndrome as the outcome indicator (population size=27 043 753)

Food groups	Odds Ratio	P-value	95% Confidence Interval
Meat & Fish			
1st Q[1]			
2nd Q	0.93	0.767	0.57-1.53
3rd Q	0.61	0.035[*]	0.38-0.95
4th Q	0.49	<0.01[*]	0.32-0.73
Grains			
1st Q[1]			
2nd Q	0.84	0.459	0.50-1.40
3rd Q	0.78	0.272	0.49-1.25
4th Q	0.70	0.099[*]	0.45-1.08
Fruit & Vegetable			
1st Q[1]			
2nd Q	1.61	0.019[*]	1.10-2.35
3rd Q	1.31	0.291	0.76-2.25
4th Q	1.05	0.854	0.59-1.87
Dietary Fat			
1st Q[1]			
2nd Q	0.55	0.053[*]	0.30-1.01
3rd Q	0.66	0.111[*]	0.38-1.12
4th Q	0.54	0.021[*]	0.33-0.89
Milk & Dairy Products			
1st Q[1]			
2nd Q	0.65	0.057[*]	0.41-1.01
3rd Q	0.94	0.758	0.60-1.47
4th Q	0.47	0.013[*]	0.27-0.83
Sugar Sweetened Beverages			
1st Q[1]			
2nd Q	0.64	0.096[*]	0.37-1.10
3rd Q	0.60	0.064[*]	0.34-1.04
4th Q	0.40	<0.01[*]	0.27-0.61
Diet Soft Drink[2]	1.31	0.012[*]	1.07-1.60
Fruit & Vegetable Juice			
1st Q[1]			
2nd Q	0.81	0.556	0.37-1.75
3rd Q	0.62	0.148[*]	0.32-1.21
4th Q	0.81	0.490	0.43-1.54

[*] Statistically significant from the reference group
[1] Reference group

[2] Diet soft drink used as a continuous variable because about 69% had no consumption of drink which made it impossible to categorize the variable into four or even 2 categories.

Table 9 Separate binary logistic regression analysis for each socio-demographic predictor of 10-year risk of CVD (population size: all=17 587 428, male=8 909 397, female=8 529 394)

Predictors	Odds Ratio(95% Confidence Interval)		
	P-value		
	All	**Male**	**Female**
Abdominal Obesity[1]	1.51(1.12-2.05)	1.53(1.11-2.11)	2.35(1.34-4.12)
	0.011*	0.013*	<0.01*
Education			
< secondary school graduation[2]			
Secondary school graduation	0.56(0.25-1.25)	0.61(0.25-1.50)	0.46(0.12-1.76)
	0.141*	0.258	0.234
Some postsecondary	0.35(0.23-0.54)	0.48(0.27-0.85)	0.11(0.03-0.37)
	<0.001*	0.018*	<0.01*
Postsecondary graduation	0.40(0.24-0.66)	0.42(0.21-0.85)	0.28(0.13-0.59)
	<0.01*	0.021*	<0.01*
Income			
Lowest income[2]			
Lower-middle income	0.67(0.45-1.01)	0.60(0.17-2.03)	0.72(0.35-1.49)
	0.055*	0.381	0.356
Upper-middle income	0.88(0.60-1.30)	1.03(0.37-2.86)	0.59(0.31-1.16)
	0.497	0.946	0.116*
Highest income	0.64(0.48-0.84)	0.60(0.26-1.39)	0.42(0.23-0.77)
	<0.01*	0.210	<0.01*
Physical activity			
Active[2]			
Moderately active	1.11(0.78-1.58)	1.06(0.66-1.70)	1.63(0.77-3.46)
	0.507	0.766	0.177*
Inactive	1.37(0.86-2.21)	1.42(0.83-2.43)	1.97(0.97-3.97)
	0.163	0.169*	0.056*
Smoking[3]	3.43(2.53-4.65)	4.33(2.42-7.74)	3.14(2.11-4.68)
	<0.001*	<0.001*	<0.001*
Ethnicity[4]	1.12(0.73-1.71)	0.95(0.56-1.59)	1.87(0.93-3.77)
	0.555	0.834	0.073*
Alcohol intake[5]	0.81(0.61-1.08)	0.93(0.49-1.78)	0.49(0.27-0.87)
	0.144*	0.829	0.021*

* Statistically significant from the reference group
[1] Abdominal obese=1 (reference group) versus not obese=0
[2] Reference group
[3] Smokers=1(reference group) versus non-smokers=0
[4] White individuals=1 (reference group) versus non-white=0
[5] Ever drinkers= 1 (reference group) versus never drinkers=0

Table 10 Separate binary logistic regression analysis between each food group and percent 10-year risk of cardiovascular diseases (CVD) (population size: all=17 587 428, male=8 678 030, female=8 909 398)

Food groups	Odds Ratio(95% Confidence Interval) P-value		
Meat & Fish	**All**	**Male**	**Female**
$1^{st} Q^1$			
$2^{nd} Q$	1.11(0.82-1.50)	1.15(0.74-1.80)	0.87(0.40-1.88)
	0.457	0.490	0.707
$3^{rd} Q$	0.87(0.66-1.15)	0.81(0.49-1.32)	0.65(0.38-1.09)
	0.324	0.361	0.094^*
$4^{th} Q$	0.99(0.68-1.45)	0.96(0.52-1.78)	0.70(0.40-1.23)
	0.999	0.914	0.196^*
Grains			
$1^{st} Q^1$			
$2^{nd} Q$	0.81(0.58-1.12)	0.68(0.40-1.15)	0.84(0.50-1.41)
	0.183^*	0.139	0.477^*
$3^{rd} Q$	0.66(0.43-1.02)	0.60(0.34-1.06)	0.70(0.41-1.20)
	0.061^*	0.077^*	0.177^*
$4^{th} Q$	0.82(0.58-1.16)	0.69(0.48-0.99)	0.65(0.33-1.27)
	0.243	0.047^*	0.189^*
Fruit & Vegetable			
$1^{st} Q^1$			
$2^{nd} Q$	0.78(0.50-1.24)	0.85(0.49-1.48)	0.86(0.47-1.55)
	0.275	0.564	0.596
$3^{rd} Q$	0.50(0.32-0.78)	0.86(0.54-1.38)	0.37(0.17-0.83)
	$<0.01^*$	0.513	0.020^*
$4^{th} Q$	0.37(0.22-0.63)	0.53(0.24-1.15)	0.44(0.23-0.86)
	$<0.01^*$	0.101^*	0.021^*
Dietary Fat			
$1^{st} Q^1$			
$2^{nd} Q$	0.86(0.62-1.21)	0.83(0.47-1.47)	0.73(0.47-1.14)
	0.382	0.507	0.159^*
$3^{rd} Q$	0.97(0.78-1.21)	0.93(0.63-1.37)	1.00(0.60-1.68)
	0.797	0.713	0.974
$4^{th} Q$	0.82(0.62-1.08)	0.80(0.49-1.29)	0.55(0.32-0.93)
	0.147^*	0.330	0.030^*
Milk & Dairy Products			
$1^{st} Q^1$			
$2^{nd} Q$	0.74(0.53-1.03)	0.78(0.48-1.26)	0.90(0.63-1.25)

	0.074[*]	0.284	0.460
3rd Q	0.74(0.51-1.09)	0.70(0.47-1.05)	0.91(0.62-1.32)
	0.124[*]	0.079[*]	0.581
4th Q	0.52(0.31-0.86)	0.40(0.24-0.67)	0.85(0.56-1.28)
	0.017[*]	<0.01[*]	0.398
Sugar Sweetened Beverages			
1st Q [1]			
2nd Q	1.12(0.71-1.76)	0.83(0.50-1.38)	1.14(0.52-2.48)
	0.594	0.450	0.706
3rd Q	1.09(0.75-1.58)	0.57(0.35-0.92)	1.09(0.66-1.79)
	0.618	0.028[*]	0.696
4th Q	1.64(1.17-2.29)	0.81(0.48-1.33)	1.64(0.90-3.00)
	<0.01[*]	0.374	0.095[*]
Diet Soft Drink[2]	1.24(0.65-2.36)	1.58(0.78-3.19)	0.94(0.37-2.35)
	0.466	0.174[*]	0.886
Fruit & Vegetable Juice			
1st Q [1]			
2nd Q	1.11(0.90-1.36)	1.46(0.93-2.29)	0.74(0.45-1.23)
	0.290	0.091[*]	0.226
3rd Q	0.90(0.63-1.27)	1.16(0.71-1.89)	0.63(0.37-1.07)
	0.514	0.515	0.085
4th Q	0.93(0.61-1.42)	0.89(0.52-1.52)	0.82(0.45-1.47)
	0.715	0.649	0.477
Potato consumption			
1st Q [1]			
2nd Q	1.31(0.60-1.11)	1.09(0.63-1.88)	1.17(0.71-1.92)
	0.124[*]	0.315	0.490
3rd Q	1.79(0.69-1.07)	1.40(0.93-2.11)	1.57(0.82-3.00)
	<0.01[*]	0.263	0.152[*]
4th Q	2.30(0.97-1.61)	2.08(1.27-3.39)	2.00(1.07-3.72)
	<0.001[*]	0.623	0.032[*]

[*] Statistically significant from the reference group
[1] Reference group
[2] Diet soft drink categorized into 2 categories (0 times/day and > 0 times/day (reference group)).

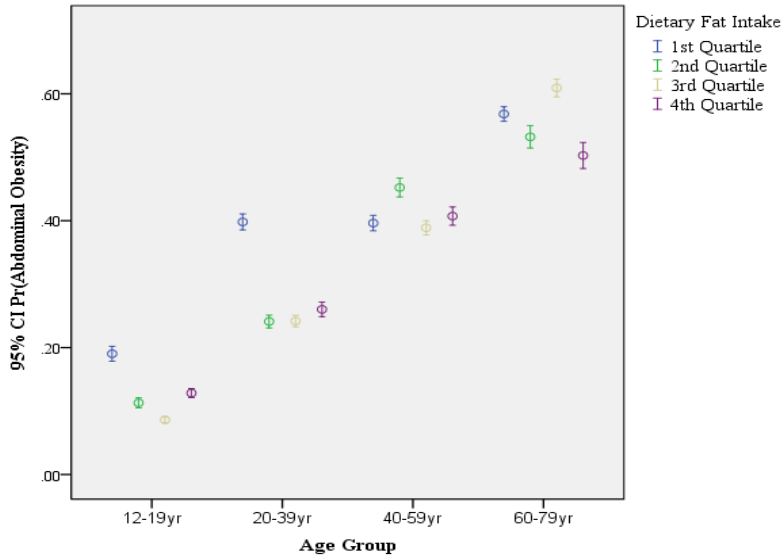## Appendix 4, Figures of interaction

Figure 1. The interaction graph between dietary fat intake and age groups in all population (population size=26 483 831). A significant interaction was observed between the 2nd quartile of fat intake with age group 20-39 y and between the 3rd quartile of fat intake and age group 60-79 y compared to age group 12-19 y.
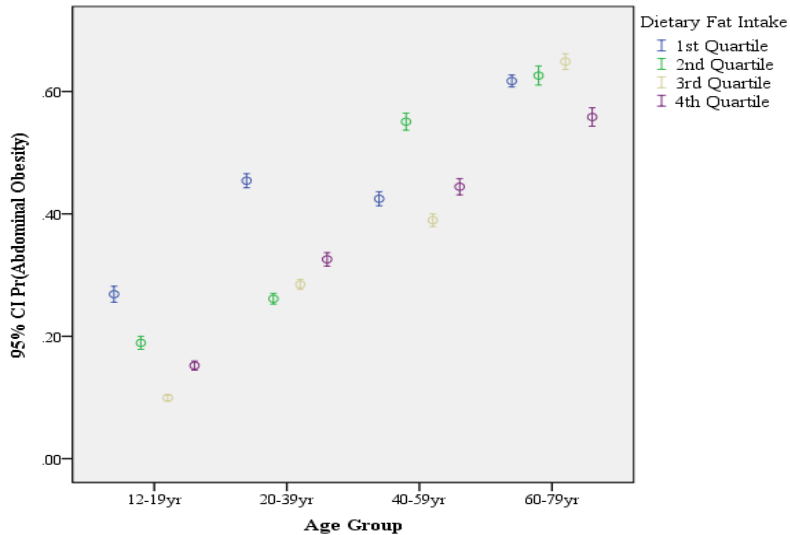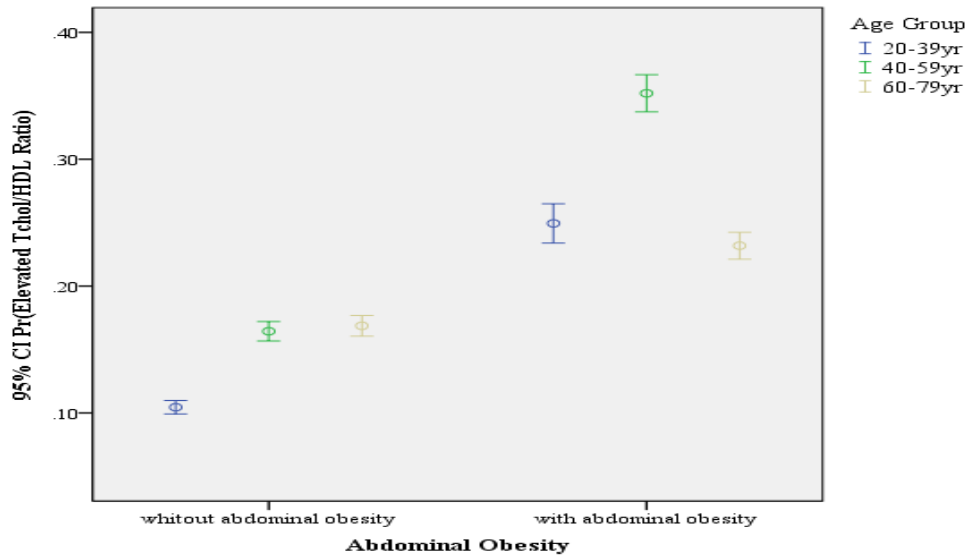

Figure 2. The interaction graph between dietary fat intake and age groups in females (population size=13 312 736). A significant interaction was observed between the 2nd quartile of fat intake with age group 20-39 y and between the 3rd quartile of fat intake and age group 60-79 y compared to age group 12-19 y.

Figure 3. The interaction graph between abdominal obesity and age groups in all population (population size=23 135 787). A significant interaction was observed between abdominal obesity and age group 60-79 y compared to age group 20-39 y.
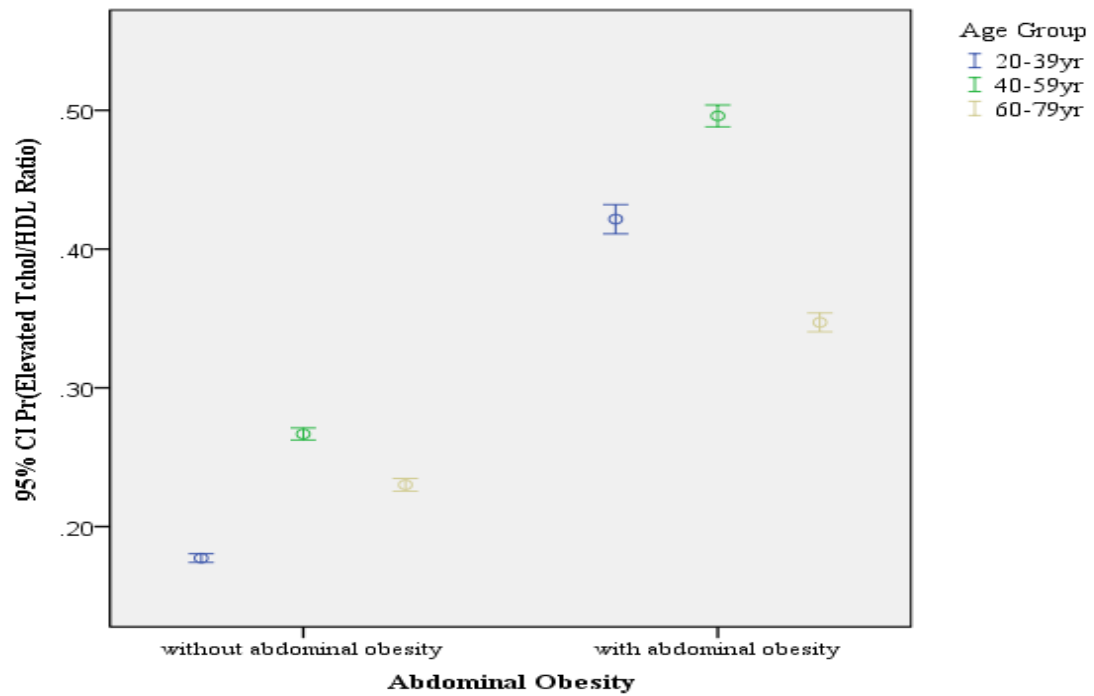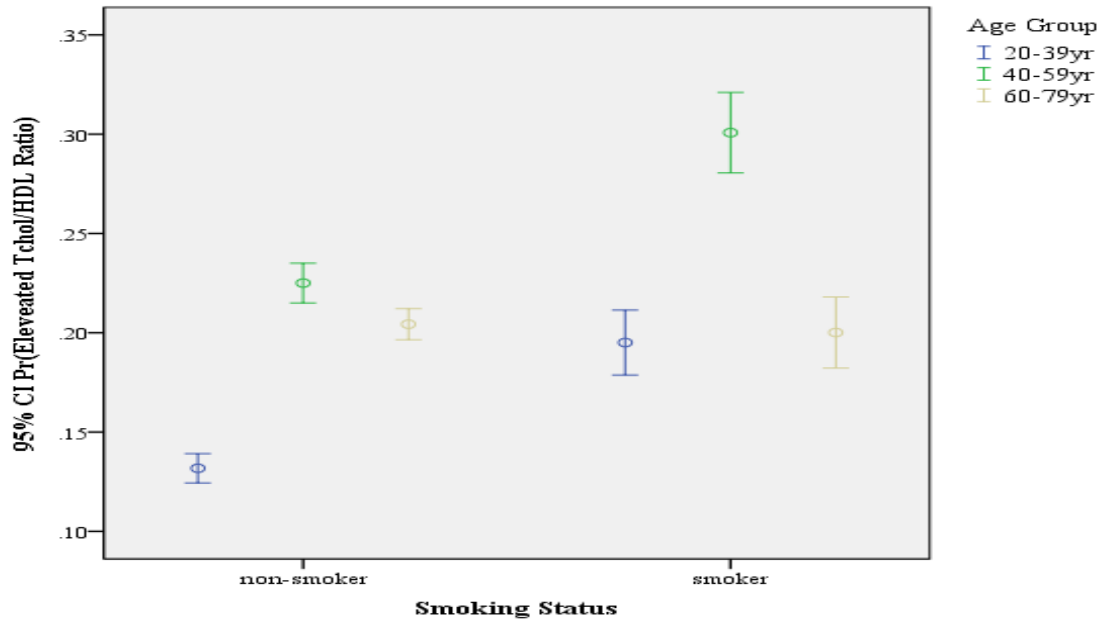

Figure 4. The interaction graph between abdominal obesity and age groups in males (population size=11 440 786). A significant interaction was observed between abdominal obesity and age group 60-79 y compared to age group 20-39 y.

Figure 5. The interaction graph between age group and smoking all population (population size=23 135 787). A significant interaction was observed between smoking and age group 60-79 y.
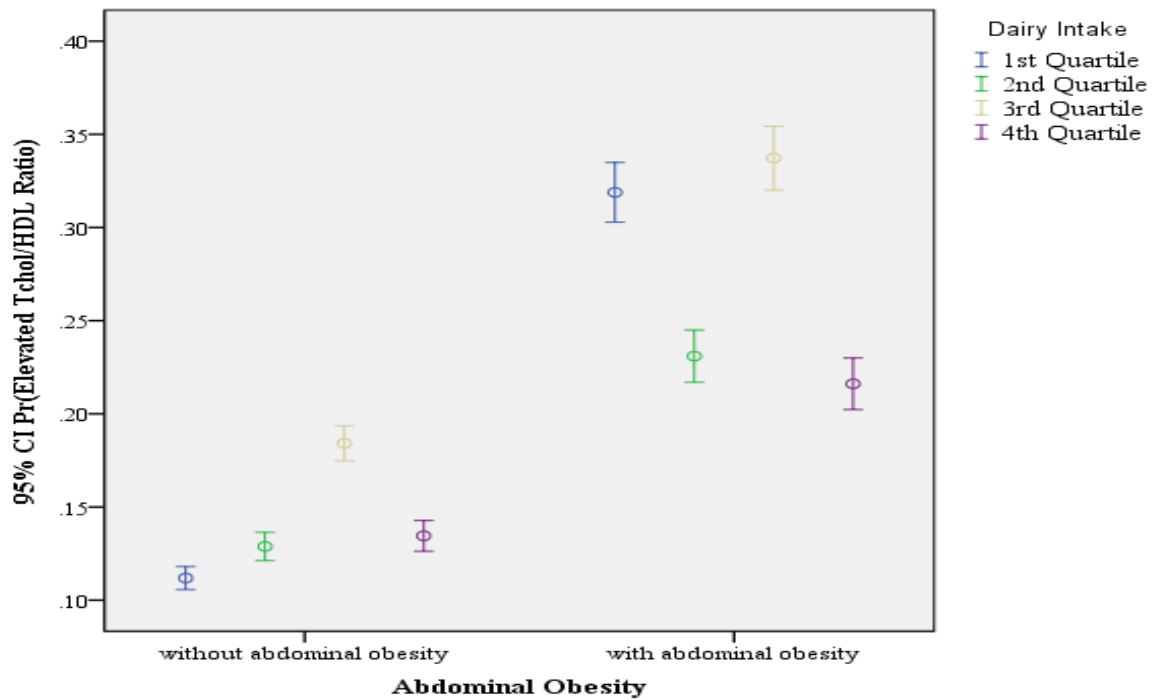

Figure 6. The interaction graph between abdominal obesity and milk and dairy product consumption in all population (population size=23 135 787). A significant interaction was observed between abdominal obesity with 2nd and 4th quartile compared to 1st quartile.
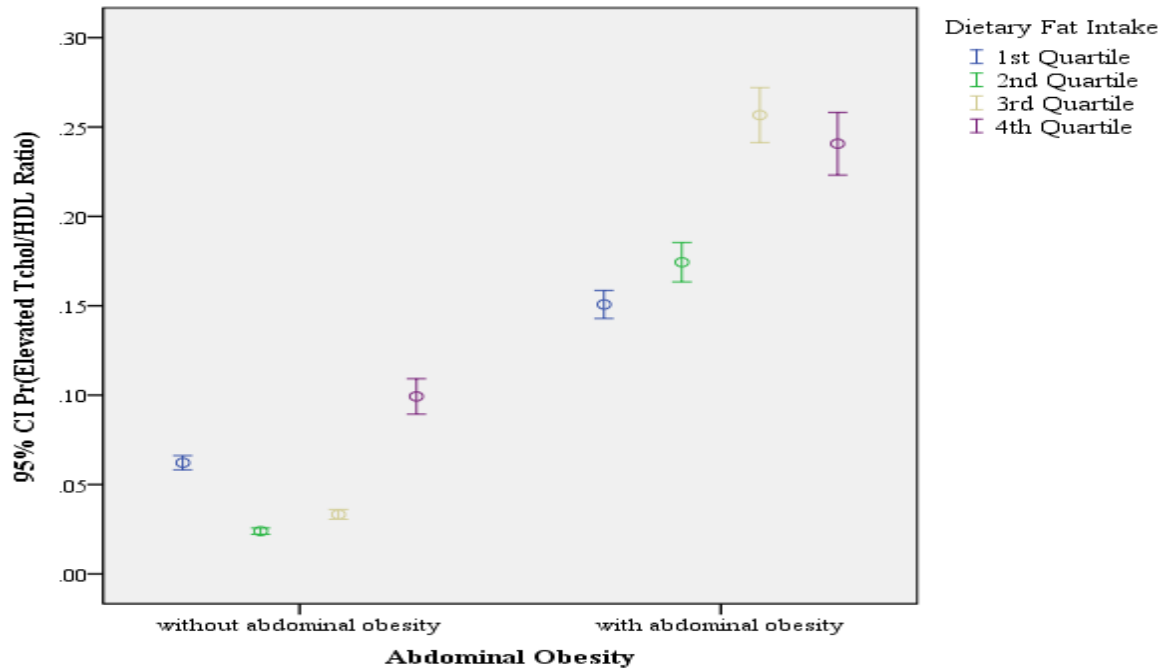
Figure 7. The interaction graph between abdominal obesity and dietary fat consumption among females (population size=11 695 001). A significant interaction was observed between abdominal obesity and 3rd compared to 1st quartile.