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Commentary

Advancing health equity to improve health: the time is now

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Abstract

Health inequities, or avoidable inequalities in health between groups of people, are increasingly recognized and tackled to improve public health. Canada's interest in health inequities goes back over 40 years, with the landmark 1974 Lalonde report, and continues with the 2011 Rio Political Declaration on Social Determinants of Health, which affirmed a global political commitment to implementing a social determinants of health approach to reducing health inequities. Research in this area includes documenting and tracking health inequalities, exploring their multidimensional causes, and developing and evaluating ways to address them. Inequalities can be observed in who is vulnerable to infectious and chronic diseases, the impact of health promotion and disease prevention efforts, how disease progresses, and the outcomes of treatment. Many programs, policies and projects with potential impacts on health equity and determinants of health have been implemented across Canada. Recent theoretical and methodological advances in the areas of implementation science and population health intervention research have strengthened our capacity to develop effective interventions.

With the launch of a new health equity series this month, the journals *Canada Communicable Disease Report* and *Health Promotion and Chronic Disease Prevention in Canada* will continue to reflect and foster analysis of social determinants of health and focus on intervention studies that advance health equity.

Introduction

The World Health Organization (WHO) defines health inequity as ''avoidable inequalities in health between groups of people within and between countries.''[1] Not only is health equity an international and domestic concern, it is a fertile field of research and practice across disciplines, sectors and jurisdictions.

While a majority of Canadians enjoy good health, health inequalities persist and, in some areas, are growing.[2,3] But much can be done to address this. The objective of this introductory commentary is to review some key milestones in domestic and global health equity work, highlight recent advances and recommended actions in Canada, and assert that new evidence on inequalities and interventions can create promising opportunities for collaborative action across sectors to address health equity and improve health.

Key Milestones

Early days

The landmark 1974 Lalonde report, ''A New Perspective on the Health of Canadians,'' asserted that the quantity, quality and arrangement of acute health care systems explain only a fraction of why a population is healthy.[4] The ''health fields'' identified in the report (biology, individual choices, physical and social environments, and health care) were an early expression of what would become known as the ''social determinants of health.'' The Lalonde report was quickly followed by other key policy documents: the WHO Alma-Ata Declaration on Primary Health Care in 1978[5]; the Canadian Epp Report, *Achieving Health for All*,[6] and the WHO ''Ottawa Charter for Health Promotion'' in 1986.[7] Later key publications such as *Why Are Some People Healthy and Others Not? The Determinants of Health of Populations*[8] and *Strategies for Population Health: Investing in the Health of Canadians* in 1994[9] signalled a reframing of public health into a ''population health'' perspective, informed by social determinants of health.

Calls for global action

WHO Commission on Social Determinants of Health

In 2008, the WHO Commission on Social Determinants of Health made a clear link between the social determinants of health and health equity in its report *Closing the Gap in a Generation: Health Equity Through Action on the Social Determinants of Health*.[10] The Commission stated: ''inequities in health, avoidable health inequalities, arise because of the circumstances in which people grow, live, work, and age, and the systems put in place to deal with illness. The conditions in which people live and die are, in turn, shaped by political, social, and economic forces.''[10] The Commission's three overarching recommendations and related principles of action focus on:

- improving daily living conditions;
- tackling the inequitable distribution of power, money and resources—the structural drivers of the conditions of daily life; and
- measuring the extent of health inequities and assessing the health equity impact of policy and other actions.[10]

This renewed call for global action has supported efforts in Canada in the public health sector and across sectors. Reflecting

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growing urgency and better understanding of approaches to health that focus on social determinants and equity, another appeal for action was issued at the 2011 World Conference on Social Determinants of Health in Rio de Janeiro.

Rio Political Declaration on Social Determinants of Health

In May 2012, Canada and other United Nations Member States endorsed the Rio Political Declaration on Social Determinants of Health.[11] The declaration expresses global political commitment for the implementation of a social determinants of health approach to reduce health inequities. Aiming to build international momentum for the development of dedicated national action plans and strategies, the Declaration identified five action areas critical to addressing health inequities:

- adopt better governance for health and development;
- promote participation in policy making and implementation;
- reorient the health sector towards reducing health inequities;
- strengthen global governance and collaboration; and
- monitor progress and increase accountability.[11]

Canadian collaboration and action

The Chief Public Health Officer's Report

Addressing both health equity and the determinants of health, the Chief Public Health Officer's (CPHO) inaugural report[2] identified several priority areas and ways to address health inequalities in Canada:

- social investments (particularly for families with children living in poverty and for early childhood development);
- community capacity to address social determinants of health and health equity;
- integrated policies and joint action across sectors and jurisdictions;
- knowledge infrastructure to assess the health of subpopulations and the efficacy, adaptability and scalability of interventions; and
- leadership within and beyond the health sector.[2]

These priority areas remain relevant today as jurisdictions and sectors in Canada work together to address health inequities.

The Pan-Canadian Public Health Network

The Pan-Canadian Public Health Network (PHN) is a network of individuals from many sectors and levels of government, who effectively work together to strengthen public health in Canada. The PHN includes academics, researchers, public servants, members of non-governmental organizations and health professionals and is governed by a council of federal/provincial/territorial government representatives including the CPHO and senior public health officials from all jurisdictions. In 2010, the PHN council endorsed a set of *Indicators of Health Inequalities*[12] and recommended that the Public Health Agency of Canada (PHAC), the Canadian Institute for Health Information (CIHI) and Statistics Canada report on these indicators. This pan-Canadian initiative will provide baseline data on over 50 indicators of health outcomes (for both chronic and infectious diseases), health-related behaviours and social determinants of health inequalities (e.g. food security). These data will be stratified, where possible, by a wide range of variables related to identity and social location (including sex, socioeconomic status, Aboriginal identity, cultural and/or racial background, immigrant status, rural/urban residence and sexual orientation). Results from this initiative, expected in 2016, will provide new information to federal, provincial and territorial governments and civil society to support decision making, priority setting, development of effective interventions, and monitoring of health inequalities.

The Canadian Council on Social Determinants of Health

The Canadian Council on Social Determinants of Health (CCSDH) is a collaborative, multisectoral stakeholder group established by PHAC in 2005 (as the Canadian Reference Group) to support Canada's contribution to the WHO Commission on the Social Determinants of Health. Since then, its role has evolved in recognition of the importance of broad intersectoral engagement for effectively addressing health inequities. The current dual mandate of the CCSDH is to advise PHAC on implementing the Rio Political Declaration on Social Determinants of Health[11] and to facilitate and leverage action on the social determinants of health and health inequalities in Canada. CCSDH membership includes representatives from all levels of government, civil society, business, labour and academia and from among Aboriginal peoples; members have been selected for their expertise and experience in addressing the social determinants of health. The Council is co-chaired by a PHAC representative appointed by the CPHO.

From knowledge to action

While substantial progress has been made in tracking health inequalities, such knowledge alone does not improve health. Advances in health equity require complementary interventions at multiple levels (behavioural, organizational and societal/systemic) across different populations in different contexts.[13]

Recent advances

In the last five years, a range of programs, policies and projects on health equity and determinants of health have been implemented across Canada in various jurisdictions. Some of these actions are described in the Rio Political Declaration on Social Determinants of Health: A Snapshot of Canadian Actions 2015.[14]

In November 2015, the Canadian Institute for Health Information released a suite of products from its ''Trends in Income-Related Health Inequalities in Canada''[3] project. These products—including a technical report and an interactive online tool—examine changes in income-related health inequalities over the past decade. For 11 of 16 indicators (including both social determinants and health outcomes), the health gap between higher-income and lower-income groups did not change. However, for 3 indicators (smoking, hospitalization of adults for chronic obstructive pulmonary disease, and fair/poor self-rated mental health) the gap widened. While inequalities decreased for the remaining 2 indicators, this was the result of a ''levelling down'' effect, where health outcomes worsened among higher-income groups and remained the same in lower-income groups. This documentation of income-related trends in health inequalities makes an important contribution to Canadian evidence.

Future directions

Two important advances in applied research are particularly interesting in terms of our equity series: implementation science and population health intervention research.

Implementation science

Implementation science is the study of methods that promote the integration of research findings and evidence into health care policy and practice.[15] It addresses the challenges of implementation, applying advances from one area to another, and the scaling-up of interventions. Implementation science is informed by a range of research and practice disciplines, building on operations research, participatory action research, management science, quality improvement and impact evaluation.

Implementation science has been used to enhance equity in health in Canada and elsewhere. Participants in recent consultative meetings organized by the Alliance for Health Policy and Systems Research of the WHO, the United States Agency for International Development and the World Bank Group noted that implementation science should promote a culture of evidence-informed learning, engage stakeholders and improve decisions on policies and programs to achieve better health outcomes.[16]

An excellent example of implementation science improving a health outcome was one that addressed housing and HIV. Evidence shows that the lack of adequate housing is a barrier to HIV treatment and follow-up and is associated with an increased risk of forward transmission. [17] Housing assistance for people with HIV who were formerly homeless or inadequately housed was found to improve their outcomes.[17] In fact, adequate housing is linked to improved health for a number of health conditions.[18]

Population health intervention research

Population health intervention research (PHIR) is similar to implementation science in that it focusses on policies and programs (frequently outside the health sector) that have the potential to improve health equity and health at the population level.[19] However, the objective of PHIR is broader: it generates knowledge about whether specific interventions work, how they work, for whom and under what circumstances. It is also concerned with how classes and programs of interventions affect health and health equity in populations. PHIR concentrates on population health interventions, recognizing unique features of these interventions and the unique combination of tools required to study them. With this knowledge, we are better equipped to design interventions that can be effective for different populations across geographies and circumstances, and better equipped to advance health equity.

The challenges of this type of research are substantial, however, given ''the involvement of actors from diverse sectors, the multiplicity of interacting components, the unique characteristics of public health as a key delivery system, the need to take into account the influence of context on both intervention implementation and its effective mechanisms, and the specific ethical issues raised with population health interventions.''[20]

An excellent example of an upstream intervention that had significant effects on population health was the MINCOME social experiment, which aimed to alleviate poverty by providing residents of Dauphin, Manitoba with a guaranteed annual income (GAI). While the main objective of the original study (conducted from 1974–79) was to assess the impact of a GAI on the labour market, recent intervention research has focussed on the population health effects of the GAI. Results have shown that hospitalizations for accidents, injuries and mental health issues, as well as physician contact for mental health complaints, declined over the course of the experiment relative to a matched comparison group. Moreover, more adolescents involved in the experiment stayed on to complete high school, resulting in a variety of other health and social benefits that would have a significant impact over their life course.[21]

Conclusion

The goal of working on health equity and determinants of health is to improve the health of the population and to ensure that the conditions that support health are distributed fairly. Canada has been making important strides in measuring and monitoring health inequalities, strengthening data infrastructure, building open information systems, undertaking sophisticated analyses of health inequalities, as well as conducting and evaluating the effectiveness of interventions. These efforts are strengthening the capacity of public health and other sectors to tackle health inequities.

With the launch of a new health equity series this month, both the *Canada Communicable Disease Report* (CCDR) and the *Health Promotion and Chronic Disease Prevention in Canada* (HPCDP) welcome reports on applied research that assess strategies to mitigate inequity and improve health outcome while continuing to publish reports that track, monitor and analyze health inequities. The aim is to increase knowledge and capacity to act on social determinants, and rigorously evaluate our efforts to advance equity and improve health.

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Conflict of interest

None

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Socioeconomic gradients in cardiovascular risk in Canadian children and adolescents

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This article has been peer reviewed.

Abstract

Introduction: Cardiovascular disease (CVD) and its risk factors show clear socioeconomic gradients in Canadian adults. Whether socioeconomic gradients in cardiovascular risk emerge in childhood remains unclear. The objective of this study was to determine whether there are socioeconomic gradients in physiological markers of CVD risk in Canadian children and adolescents.

Methods: Using combined cross-sectional data from the Canadian Health Measures Survey 2007–2011, we examined the following cardiovascular risk markers: overweight (including obesity), aerobic fitness score (AFS), blood pressure (BP), blood lipids (total as well as HDL and LDL cholesterol and triglycerides), glucose metabolism and C-reactive protein (CRP) by sex in 2149 children (ages 6–11 years) and 2073 adolescents (ages 12–17 years). Multivariate linear and logistic regression analyses were used to identify patterns in cardiovascular risk across strata of household income adequacy and parental educational attainment, adjusting for age and ethnicity, and stratified by age group and sex.

Results: Young boys showed markedly higher prevalence of obesity than young girls (prevalence of 18.5%, 95% confidence interval [CI]: 15.6–21.5 vs. 7.7%, 95% CI: 5.2–10.3). However, negative SES gradients in adiposity risk were seen in young and adolescent girls rather than boys. Young and adolescent boys were more physically fit than girls (mean AFS of 541, 95% CI: 534–546 vs. 501, 95% CI: 498–505 in children; 522, 95% CI: 514–529 vs. 460, 95% CI: 454–466 in adolescents; $p < .001$). Although a positive income gradient in AFS was observed in both boys and girls, statistical significance was reached only in girls ($p = .006$). A negative gradient of parental education in BP was observed in young children. While we observed substantial sex differences in systolic BP, total and HDL cholesterol, fasting glucose and CRP in adolescents, sex-specific socioeconomic gradients were only observed for systolic BP, HDL and LDL cholesterol. Further studies with large samples are needed to confirm these findings.

Conclusion : This study identified important sex difference and socioeconomic gradients in adiposity, aerobic fitness and physiological markers of CVD risk in Canadian school-aged children. Population health interventions to reduce socioeconomic gradients in CVD risk should start in childhood, with a particular focus on preventing obesity in young boys of all SES and girls of low SES, promoting physical fitness especially in girls and in all ages of youth in low-SES groups, and increasing parental awareness, especially those with low educational attainment, of early CVD risks in their children.

Keywords: *socioeconomic gradients, socioeconomic status, cardiovascular risk, physical fitness, obesity, children and adolescents, Canadian Health Measures Survey*

Key findings

- Young boys had higher prevalence of obesity than young girls.
- Boys were more physically fit than girls.
- Canadian children and adolescents, particularly girls, show significant socioeconomic gradients in obesity, physical fitness and several physiological markers of risk of cardiovascular disease.

Introduction

Cardiovascular disease (CVD) is the leading cause of death in Canadian adults.[1] In adults, CVD and its risk factors show clear socioeconomic gradients.[2,3] Physiological and behavioural risk factors associated with CVD include overweight (as well as obesity), elevated blood pressure (BP), elevated low-density lipoprotein (LDL) and low high-density lipoprotein (HDL), elevated blood glucose, smoking, physical inactivity/low physical fitness and consuming a high fat diet.[4] Behavioural factors are known to be associated with many adverse health outcomes including the development of physiological risk factors of CVD.[5] Socioeconomic status (SES) is used as a proxy of physical environments in which children live and play. Emerging evidence suggests that several adverse health behaviours associated with CVD risk, such as unhealthy eating, physical inactivity and smoking, are disproportionally higher in youth with low SES.[6-8] Other research suggests children's long-term health can be affected by biological embedding

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of adversity during sensitive developmental periods and that children with low SES may be especially vulnerable to stressful influences.[9] We need further evidence to understand whether socioeconomic gradients in physical health develop in children exposed to various socioeconomic circumstances.

There is ample evidence that CVD risk factors originate in childhood and that low SES in childhood is associated with elevated risk of CVD and increased CVD mortality in later life.[10,11] SES is frequently measured as family income, parental education and occupational status. Family income and parental education, each representing a separate dimension of SES, are among the social factors most strongly associated with health.[12] Income may influence health most directly through access to material resources; education may be directly related to health through health behaviours and lifestyle choices and indirectly through income and psychosocial factors.[13] The direct and indirect contribution of material factors strongly predicts health in children. Intergenerational studies have found that parents' education can have a substantial impact on the health and education outcomes of their children.[14] Assessing the independent effect of income and education on health may help us understand the mechanisms through which they influence health and provide more options for policy development to reduce the risk of poor health outcomes for children.

Given the childhood obesity epidemics in many countries, a number of studies have demonstrated socioeconomic gradients in relation to obesity and, as a result, interventions aimed at reducing inequalities in childhood obesity have been created and implemented.[15,16] While recent evidence suggests a sex-specific pattern of SES gradients in overweight in Canadian adults, studies in children have not identified any such differences in SES-related overweight risk.[8,17] Few studies have addressed the early emergence of SES gradients in physiological CVD markers, and findings have been inconsistent partly due to differences in the SES indicators and in the age ranges of the study populations.[18-20] Understanding socioeconomic impacts on CVD risks in childhood may help identify high-risk groups to target for early CVD prevention programs that prevent lifelong inequalities in CVD.

The Canadian Health Measures Survey (CHMS),[21] a nationally representative survey with physical measures and blood and urine collection, provides a unique opportunity to examine the association of SES with physiological markers of CVD risk in Canadian children and adolescents.

Methods

Data source

The CHMS is a cross-sectional, comprehensive health measures survey that collects information on health status and risk factors in the Canadian household population. The survey represented 96.3% of the Canadian population aged 6 to 79 years living at home in the 10 provinces and 3 territories; it excludes people living on reserves or other Aboriginal settlements, certain remote areas and institutions, and full-time members of the Canadian Armed Forces. The Health Canada Research Ethics Board reviewed and approved all CHMS processes and protocols. Participation in the survey was voluntary and informed consent was obtained from each participant. We combined Cycle 1 (2007–2009) and Cycle 2 (2009–2011) for this study; the overall response rate for both cycles combined was 53.5%.[21]

The surveys consisted of a household interview that includes questions on sociodemographic characteristics and health and lifestyle and a visit to a mobile examination centre where physical measures are made and blood and urine samples collected. Parents/guardians answered all questions for participants aged 6 to 13 years; participants aged 14 years and older answered all questions on their own. Approximately half of the respondents were randomly selected to fast overnight before blood samples were taken. To increase statistical power, we combined CHMS Cycles 1 and 2 for a sample of 3799 (and a fasting sample of 1693) respondents aged 6 to 17 with data collected between 2007 and 2011.

Measures

Socioeconomic status

We used household income adequacy and parental educational attainments as indicators of SES. Household income adequacy is a variable derived by Statistics Canada, calculated using both total family income from all sources and total number of household members, and classified into lowest, lower-middle, upper-middle and highest income groups. Because only about 70% of the CHMS respondents reported their total household income, Statistics Canada used regression modelling techniques to impute missing values based on all or part of the following information: partial responses for the income range, nearest neighbour, collection site and household size.[22] We chose to only include respondents whose imputed income was based on fully or partially reported income range.

Parental educational attainment was based on the highest education attained by either parent, and categorized as less than secondary, secondary, some post-secondary and completed post-secondary education. Ethnicity was defined as White or non-White, the latter included Aboriginals living off-reserve. Respondents with missing values on these variables were excluded from the analysis, resulting in a total sample of 3591 and a fasting sample of 1645.

Cardiovascular outcomes

Birth weight of the CHMS participants aged 6 to 11 years was reported by their guardians. These children's physical activity was determined from their guardians' answers to the question ''Over a typical or usual week, on how many days was he/she physically active for a total of at least 60 minutes per day?'' Available answers included: (1) none; (2) 1 day; (3) 2 to 3 days; (4) 4 or more days. The child was considered physically active if the answer was (4); otherwise, they were considered inactive.[22] For adolescents aged 12 to 17, the physical activity module for adults was adopted and levels of activity classified as active, moderately active and inactive.[22,23] Cigarette smoking in adolescents was defined as current (combined current daily or occasional smoker) versus non-smoker (never smoked).

Body mass index (BMI) was calculated using measured standing height and weight, and weight status was defined according to World Health Organization growth reference for school-aged children and adolescents.[24] Waist circumference was measured at the mid-point between the highest point of the iliac crest and the last floating rib.[22]

Aerobic fitness or cardiorespiratory fitness levels were determined using the modified Canadian Aerobic Fitness Test (mCAFT) by recording participants' age-predicted maximal heart rate and calculating their predicted maximal aerobic power (VO_2 max).[25] The definition of poor aerobic fitness was based on a derived variable indicating aerobic fitness within a range that is generally associated with certain health risks in children. Details of the derivation of aerobic fitness score (AFS) and aerobic fitness norm are published in methodological papers and the CHMS Data Users' Guide.[22,26,27] Resting systolic BP and diastolic BP were measured according to the new protocol for standard BP measurement in surveys, described in our previous publication.[28]

All laboratory assays were conducted by Health Canada. Details of the standard laboratory procedures are available online.[22] Values for laboratory variables that were below the limit of detection were replaced by limit of detection divided by two. The dependent variables for CVD markers in the full sample included total cholesterol (mmol/L), HDL (mmol/L) and C-reactive protein (CRP; mg/L), while those based on the fasting sub-sample included LDL (mmol/L), triglycerides (mmol/L), glucose (mmol/L) and insulin (pmol/L). Insulin resistance was estimated by homeostasis model assessment of insulin resistance (HOMA-IR), a surrogate measure of insulin resistance in non-diabetic children, calculated by dividing the product of 0.1394*insulin (pmol/ml) and glucose (mmol/ml) by 22.5.[29] To preserve the size of the sample, respondents with missing values for a given dependent variable were excluded only from analyses involving that variable.

Statistical analyses

Respondents' SES and CVD risk characteristics were described for children and adolescents and compared between boys and girls using t-tests. We first examined the polychoric correlation between household income adequacy and parental educational attainment. Since they were only moderately correlated (correlation coefficient = 0.45), the independent effect of income adequacy and parental education on CVD risk were examined by (1) multivariate logistic regression for overweight (including obesity) and poor aerobic fitness, and (2) multivariate linear regression for BMI, waist circumference, AFS, BP, blood lipids (total, HDL and LDL cholesterol and triglycerides), fasting glucose, insulin and HOMA-IR, and CRP, adjusting for age and ethnicity, and stratified by sex. For the associations of SES with BP, models additionally adjusted for heart rate and height. Depending on the age range, we did not control for birth weight and physical activity in children, or smoking status and physical activity in adolescents because these variables may act as mediators so that the effects of SES on CVD risk may be underestimated.

Because of the complex sampling design of the CHMS and limited number of primary sampling units of Cycles 1 and 2, bootstrap weights were applied for variance estimation for proportions, means and parameters of regression models, with 24 degrees of freedom specified for combined Cycle 1 and 2 data. We used multivariate logistic and linear regression models to examine trends in CVD risks across all strata of income adequacy and parental education, adjusted for age and ethnicity. Satterthwaite-adjusted chi-square statistics were used to determine statistical significance ($p < .05$) of a linear trend.

All analyses were performed using SAS version 9.3 (SAS Institute Inc., Cary, NC, USA) and SUDANN version 10.0.1 (RTI International, Research Triangle Park, NC, USA).

Results

Table 1 shows SES indicators and CVD risk factors by sex in children aged 6 to 11 years. Boys were born significantly heavier than were girls. Prevalence of obesity in boys (18.5%; 95% confidence interval [CI]: 15.6–21.5) was more than double that of girls (7.7%; 95% CI: 5.2–10.3; $p < .001$). Similarly, average waist circumference was greater in boys than in girls (62.5 cm; 95% CI: 61.7–63.0 vs. 60.4 cm; 95% CI: 59.7–61.5; $p = .0004$).

Boys were physically fitter than girls, with a higher mean AFS (541; 95% CI: 534–546 vs. 501; 95% CI: 498–505; $p < .001$) and a lower prevalence of poor aerobic fitness (23.2; 95% CI: 18.5–28.7 vs. 30.6; 95% CI: 26.3–35.3; $p = .04$). No sex differences were found in most CVD physiological markers except for mean fasting insulin, which was higher in girls than boys (54.6 pmol/L; 95% CI: 50.0–59.2 vs. 43.4 pmol/L; 95% CI: 37.6–49.2; $p = .03$). We found no marked sex differences in the SES indicators.

Table 2 shows SES indicators and CVD risk factors by sex in adolescents aged 12 to 17 years. The sex difference in aerobic physical fitness persisted and increased in the adolescent population, with adolescent girls having even lower AFS (460; 95% CI: 454–466 vs. 522; 95% CI: 514–529 for adolescent boys; $p < .001$) and a higher proportion of poor aerobic fitness than adolescent boys (18.0; 95% CI: 14.7–21.9 for girls vs. 8.60; 95% CI: 5.97–12.3; $p = .002$). Moreover, we observed marked sex differences in multiple CVD markers such as unfavorable systolic BP, fasting glucose and HDL in adolescent boys, and unfavorable total cholesterol and CRP levels in adolescent girls. Again, no marked sex differences in SES indicators were observed.

Table 3 shows gradients of CVD risk in children according to income adequacy and parental educational attainment. For sufficient statistical power, we combined overweight and obese groups as a dependent variable in regression analyses. We found a significant income gradient in BMI (p for trend:.006) and overweight (p for trend:.01) in young girls, whereas non-White young boys showed a higher overweight risk than White boys (OR: 1.55; 95% CI: 1.03–2.32). Moreover, we also observed income and educational gradients in aerobic fitness (mean AFS and proportion of poor aerobic fitness) in young girls (p for trend:.006 and.003, respectively); a similar trend of income gradient in aerobic fitness was seen in young boys but did not reach statistical significance (p for trend:.11). Regardless of sex, there were negative educational gradients in elevated systolic BP and diastolic BP in young children. Non-White children had higher mean HDL than White children ($\beta = 0.11$; 95% CI: 0.04–0.18). Moreover, we also observed a positive parental educational gradient in HDL in young girls (p for trend:.047).

TABLE 1
Socioeconomic status and cardiovascular risk of Canadian children, 6–11 years, by sex

	Sample size, n[b]	Percentage or mean (95% CI)[a]			*p* value
		Total	Boys	Girls	
Income adequacy	2073				
Lowest	128	5.96 (4.31–8.19)	5.62 (3.74–8.37)	6.34 (4.21–9.44)	.62
Lower-middle	349	19.4 (16.1–23.2)	18.7 (14.1–24.4)	20.2 (16.4–24.5)	.62
Upper-middle	569	27.6 (24.6–30.8)	26.5 (23.4–29.8)	28.8 (24.0–34.1)	.40
Highest	1027	47.1 (41.9–52.3)	49.2 (42.8–55.6)	44.7 (39.4–50.1)	.12
Parental education	2073				
Less than secondary	80	3.89 (2.62–5.72)	3.55 (2.36–5.31)	4.27 (2.43–7.38)	.54
Secondary	178	8.75 (6.79–11.2)	8.02 (5.18–12.2)	9.59 (7.34–12.4)	.45
Some post-secondary	93	4.29 (2.84–6.45)	4.90 (2.64–8.92)	3.61 (2.16–5.97)	.46
Post-secondary	1722	83.1 (79.6–86.0)	83.5 (78.3–87.7)	82.5 (78.7–85.7)	.71
Ethnicity – White	1575	72.7 (62.5–81.0)	74.2 (63.7–82.5)	71.1 (60.2–79.9)	.23
Birth weight, g	2020	3378 (3338–3418)	3441 (3379–3503)	3312 (3264–3360)	.002
Physically active	1735	83.1 (81.0–85.2)	83.2 (79.9–86.5)	83.1 (80.6–85.5)	.71
Cardiovascular risk					
BMI, kg/m^2	2058	17.9 (17.7–18.1)	18.2 (17.9–18.4)	17.6 (17.4–17.9)	**.003**
Overweight	2058	20.4 (17.4–23.7)	18.8 (15.2–23.1)	22.1 (18.7–26.0)	.11
Obese	2058	13.4 (11.3–15.7)	18.5 (15.6–21.5)	7.7 (5.2–10.3)	< **.001**
Waist circumference, cm	2058	61.5 (61.0–62.2)	62.5 (61.7–63.0)	60.4 (59.7–61.5)	< **.001**
AFS	1302	518 (516–521)	541 (534–546)	501 (498–505)	< **.001**
Poor aerobic fitness	1302	27.0 (23.9–30.3)	23.2 (18.5–28.7)	30.6 (26.3–35.3)	**.04**
Systolic BP, mmHg	2063	93.9 (93.4–94.3)	93.6 (93.0–94.3)	94.2 (93.6–94.8)	.198
Diastolic BP, mmHg	2063	61.0 (60.5–61.6)	60.8 (60.0–61.6)	61.3 (60.7–61.8)	.29
C-reactive protein, mg/L	1791	0.96 (0.80–1.12)	1.05 (0.76–1.35)	0.86 (0.76–0.96)	.2
Total cholesterol, mmol/L	1816	4.23 (4.16–4.30)	4.22 (4.13–4.31)	4.25 (4.16–4.33)	.66
HDL cholesterol, mmol/L	1816	1.42 (1.39–1.45)	1.44 (1.40–1.47)	1.40 (1.36–1.44)	.11
LDL cholesterol, mmol/L	887	2.35 (2.28–2.43)	2.36 (2.26–2.46)	2.35 (2.22–2.47)	.88
Log-triglyceride, mmol/L	887	0.82 (0.77–0.87)	0.79 (0.73–0.85)	0.85 (0.79–0.92)	.12
Glucose, mmol/L	879	4.60 (4.51–4.69)	4.67 (4.53–4.81)	4.53 (4.46–4.60)	.05
Insulin, pmol/L	856	48.9 (45.2–52.6)	43.4 (37.6–49.2)	54.6 (50.0–59.2)	**.03**
HOMA-IR	851	1.56 (1.31–1.82)	1.46 (1.02–1.91)	1.67 (1.41–1.93)	.6

Source: 2007–2011 Canadian Health Measures Survey.

Abbreviations: AFS, aerobic fitness score; BMI, body mass index; BP, blood pressure; CI, confidence interval; HDL, high-density lipoprotein; HOMA-IR, homeostasis model assessment of insulin resistance; LDL, low-density lipoprotein.

[a] Population-weighted percentage or mean; 95% confidence interval.

[b] Depending on the variable, analysis excludes 76 to 333 non-respondents from the full sample and 26 to 62 non-respondents from the fasting sample.

Table 4 shows gradients in CVD risk in adolescents according to income adequacy and parental education. In contrast to the lack of SES gradient in risk of overweight in young boys, we observed a positive income gradient in adolescent boys, with boys living in the highest income households showing the highest risk. We observed a similar gradient in income adequacy when BMI was analyzed as a continuous outcome variable. There was an educational gradient in overweight risk in adolescent girls, with overweight prevalence higher in girls whose parents had low educational attainment. SES gradients in aerobic fitness persisted in girls into adolescence (*p* for trend:.05).

In terms of other physiological markers of CVD risk, we observed gradients of income adequacy in systolic BP and in total and LDL cholesterol in adolescent boys, with boys in highest income households demonstrating the highest risk, which is the same direction of income gradient observed with overweight. Furthermore, there were educational gradients in LDL cholesterol in adolescent boys and income gradient in HDL cholesterol in adolescent girls, with low-SES adolescents showing the higher risk. Similar to what we observed in younger children, non-White adolescents had higher mean HDL cholesterol than did their White counterparts ($\beta = 0.08$, 95% CI: 0.01–0.16). Even though we found significant trends in some of the CVD markers estimated

TABLE 2
Socioeconomic status and cardiovascular risks of Canadian adolescents, 12–17 years, by sex

	Sample size, n[b]	Percentage or mean (95% CI)[a]			*p* value
		Total	Boys	Girls	
Income adequacy	**1518**				
Lowest	73	5.05 (3.56–7.12)	4.22 (2.63–6.70)	5.99 (3.81–9.31)	.25
Lower-middle	215	14.7 (11.9–18.1)	17.0 (13.3–21.5)	12.1 (8.6–16.8)	.07
Upper-middle	443	27.7 (23.3–32.7)	27.6 (21.6–34.7)	27.8 (22.4–34.0)	.96
Highest	787	52.5 (46.3–58.6)	51.1 (43.8–58.4)	54.0 (46.5–61.3)	.47
Parental education	1518				
Less than secondary	52	3.83 (2.08–6.94)	3.03 (1.15–7.78)	4.74 (2.68–8.24)	.28
Secondary	129	9.38 (7.11–12.3)	10.2 (7.46–13.8)	8.45 (5.42–12.9)	.42
Some post-secondary	117	7.54 (5.58–10.1)	7.26 (4.75–11.0)	7.86 (5.58–10.9)	.74
Post-secondary	1220	79.2 (75.0–82.9)	79.5 (73.7–84.2)	79.0 (73.4–83.6)	.87
Ethnicity – White	1620	74.6 (65.4–82.0)	74.1 (64.6–81.9)	75.1 (64.7–83.3)	.76
Physically active	1505	30.1 (26.8–33.7)	31.5 (27.4–35.9)	28.6 (23.1–34.7)	.42
Daily smoker	1518	4.63 (2.98–7.12)	5.62 (2.95–10.5)	3.50 (2.23–5.45)	.28
Cardiovascular risk					
BMI, kg/m^2	1518	21.9 (21.4–22.5)	21.9 (21.2–22.6)	22.0 (21.3–22.7)	.86
Overweight	1518	18.3 (15.1–22.0)	17.9 (13.9–22.7)	18.8 (14.6–23.8)	.8
Obese	1518	14.3 (11.2–18.2)	13.9 (10.1–18.7)	14.8 (10.4–20.8)	.2
Waist circumference, cm	1518	75.2 (73.9–76.4)	75.9 (74.2–77.7)	74.2 (72.4–75.9)	.13
AFS	1518	492 (487–497)	522 (514–529)	460 (454–466)	**<.001**
Poor aerobic fitness	1518	13.1 (11.3–15.1)	8.60 (5.97–12.3)	18.0 (14.7–21.9)	**.002**
Systolic BP, mmHg	1514	98.0 (97.1–98.9)	99.7 (98.4–101.0)	96.1 (95.3–96.8)	**<.001**
Diastolic BP, mmHg	1514	61.8 (60.9–62.8)	62.4 (60.7–63.3)	61.6 (60.8–62.4)	.45
C-reactive protein, mg/L	1389	0.97 (0.83–1.12)	0.82 (0.70–0.94)	1.15 (0.87–1.42)	**.036**
Total cholesterol, mmol/L	1423	4.06 (3.98–4.13)	3.99 (3.91–4.08)	4.14 (4.04–4.23)	**.005**
HDL cholesterol, mmol/L	1423	1.31 (1.28–1.34)	1.26 (1.23–1.29)	1.37 (1.34–1.41)	**<.001**
LDL cholesterol, mmol/L	892	2.30 (2.21–2.39)	2.31 (2.20–2.43)	2.29 (2.18–2.40)	.72
Log-triglyceride, mmol/L	892	0.96 (0.91–1.02)	0.95 (0.88–1.03)	0.97 (0.90–1.04)	.69
Fasting Glucose, mmol/L	890	4.69 (4.61–4.77)	4.78 (4.69–4.87)	4.60 (4.52–4.68)	**<.001**
Insulin, pmol/L	869	70.2 (65.6–74.6)	69.5 (62.1–76.9)	70.8 (66.4–75.3)	.74
HOMA-IR	867	2.07 (1.91–2.23)	2.10 (1.87–2.32)	2.05 (1.89–2.21)	.65

Source: 2007–2011 Canadian Health Measures Survey.

Abbreviations: AFS, aerobic fitness score; BMI, body mass index; BP, blood pressure; CI, confidence interval; HDL, high-density lipoprotein; HOMA-IR, homeostasis model assessment of insulin resistance; LDL, low-density lipoprotein.

[a] Population-weighted percentage or mean; 95% CI.

[b] Depending on the variable, analysis excludes 132–227 non-respondents from the full sample and 22–45 non-respondents from the fasting sample.

using fasted samples, these results should be interpreted with caution because of the low sample sizes in low-SES groups.

Discussion

Our study examined whether there were SES gradients in physiological markers of CVD risk in a nationally representative sample of Canadian children and adolescents. We identified important sex and SES gradients in adiposity and aerobic fitness that emerge early in childhood. Young boys were twice as likely to be obese than young girls; however, decreasing risk of overweight with socioeconomic affluence was only seen in girls. More importantly, we found SES gradients in aerobic fitness throughout childhood, especially in girls. Educational gradients in BP emerged early in childhood. While adolescent boys in affluent families showed higher risk in some physiological markers of CVD, we found SES gradients in decreased HDL in adolescent girls and increased LDL in adolescent boys, with low-SES adolescents showing the higher risk.

Many of the studies that examined the relationship between SES and overweight in children and adolescents from developed countries have found an inverse gradient between SES and overweight.[30] Our finding on the inverse association of income adequacy with risk of overweight is congruent

TABLE 3
Multivariate linear or logistic regression analysis of socioeconomic gradients in cardiovascular risks in Canadian children, 6–11 years, by sex

	Sample, n	Income adequacy[a]				Highest parental education[a]			
		Lowest	Lower-middle	Upper-middle	*p for trend*	< Secondary	Secondary	Some post-secondary	*p for trend*
		β-coefficient (95% confidence interval)				β-coefficient (95% confidence interval)			
Boys									
Body mass index (kg/m^2)	860	0.68 (−0.82 to 2.19)	0.90 (−0.31 to 2.10)	0.47 (−0.50 to 1.45)	0.27	2.85 (−0.45 to 6.16)	0.63 (−1.07 to 2.34)	1.49 (−2.07 to 5.04)	0.15
Waist circumference (cm)	860	0.75 (−1.11 to 2.62)	0.82 (−0.36 to 2.01)	0.54 (−0.64 to 1.72)	0.36	3.18 (−0.25 to 6.61)	0.46 (−0.87 to 1.79)	1.71 (−2.62 to 6.04)	0.15
Aerobic fitness score	608	−17.1 (−38.8 to 4.75)	−10.1 (−26.5 to 6.35)	−7.33 (−23.6 to 8.93)	0.11	−18.3 (−49.8 to 13.3)	5.54 (−19.1 to 30.2)	18.8 (−8.08 to 45.7)	0.15
Systolic blood pressure (mmHg)[b]	855	−1.49 (−4.37 to 1.40)	0.49 (−1.74 to 2.72)	1.87 (−1.21 to 4.95)	0.18	**6.25 (1.25 to 11.2)**	**0.55 (−3.95 to 5.05)**	**0.99 (−5.65 to 7.64)**	**0.018**
Diastolic blood pressure (mmHg)[b]	855	−1.66 (−5.33 to 2.00)	0.68 (−2.02 to 3.37)	0.75 (−2.81 to 4.31)	0.34	**5.61 (0.70 to 10.5)**	**−0.03 (−5.06 to 4.99)**	**0.20 (−4.09 to 4.49)**	**0.02**
C-reactive protein (mg/L)	753	−0.08 (−0.98 to 0.82)	0.41 (−0.42 to 1.24)	1.21 (0.33 to 2.08)	0.5	1.37 (−0.34 to 3.08)	−0.32 (−0.85 to 0.22)	1.37 (−1.11 to 3.85)	0.36
Total cholesterol (mmol/L)	760	0.12 (−0.21 to 0.45)	−0.03 (−0.29 to 0.24)	0.11 (−0.05 to 0.26)	0.51	−0.10 (−0.50 to 0.29)	−0.24 (−0.50 to 0.01)	−0.06 (−0.28 to 0.17)	0.22
HDL cholesterol (mmol/L)	760	−0.02 (−0.20 to 0.15)	−0.03 (−0.15 to 0.08)	0.00 (−0.08 to 0.09)	0.72	0.05 (−0.16 to 0.26)	−0.08 (−0.18 to 0.01)	−0.09 (−0.21 to 0.02)	0.67
LDL cholesterol (mmol/L)	451	−0.13 (−0.70 to 0.43)	0.13 (−0.18 to 0.44)	0.23 (0.06 to 0.40)	0.56	0.02 (−0.34 to 0.38)	0.06 (−0.35 to 0.46)	−0.38 (−0.63 to −0.12)	0.41
Log-triglyceride (mmol/L)	458	0.17 (−0.07 to 0.40)	0.06 (−0.02 to 0.13)	0.04 (−0.00 to 0.08)	0.13	−0.05 (−0.22 to 0.11)	0.02 (−0.06 to 0.11)	−0.12 (−0.37 to 0.13)	0.93
Fasting glucose (mmol/L)	447	−0.09 (−0.36 to 0.17)	−0.22 (−0.51 to 0.06)	−0.19 (−0.48 to 0.11)	0.22	−0.07 (−0.35 to 0.21)	−0.01 (−0.38 to 0.37)	−0.03 (−0.25 to 0.20)	0.91
Fasting insulin (pmol/L)	443	37.0 (−18.2 to 92.2)	−1.62 (−13.3 to 10.0)	−2.88 (−14.5 to 8.75)	0.16	−11.5 (−41.6 to 18.5)	−7.42 (−26.0 to 11.2)	−15.2 (−32.6 to 2.28)	0.56
HOMA-IR	440	0.90 (−0.87 to 2.66)	−0.36 (−1.25 to 0.53)	−0.44 (−1.38 to 0.50)	0.28	−0.47 (−1.50 to 0.55)	−0.42 (−1.37 to 0.52)	−0.58 (−1.20 to 0.05)	0.43
		Odds ratio (95% confidence interval)				Odds ratio (95% confidence interval)			
Overweight/obese	860	1.40 (0.51 to 3.81)	1.35 (0.61 to 2.95)	1.47 (0.75 to 2.88)	0.56	2.65 (0.81 to 8.65)	1.82 (0.69 to 4.78)	1.95 (0.58 to 6.51)	0.18
Poor aerobic fitness	608	3.42 (0.93 to 12.4)	1.36 (0.50 to 3.44)	1.35 (0.52 to 3.37)	0.07	1.42 (0.26 to 8.11)	1.10 (0.16 to 7.99)	0.83 (0.25 to 2.82)	0.62
		β-coefficient (95% confidence interval)				β-coefficient (95% confidence interval)			
Girls									
Body mass index (kg/m^2)	833	**1.23 (0.12 to 2.34)**	**1.33 (0.70 to 1.97)**	**0.55 (−0.23 to 1.33)**	**0.006**	−0.97 (−2.35 to 0.41)	0.21 (−0.97 to 1.38)	0.13 (−1.04 to 1.30)	0.18
Waist circumference (cm)	833	0.95 (−0.53 to 2.42)	1.30 (0.57 to 2.03)	0.41 (−0.35 to 1.18)	0.09	−0.68 (−2.40 to 1.05)	0.02 (−1.15 to 1.20)	−0.03 (−1.20 to 1.14)	0.42
Aerobic fitness score	651	**−14.7 (−27.7 to −1.62)**	**−16.6 (−27.9 to −5.23)**	**−5.29 (−14.4 to 3.85)**	**0.006**	**−17.1 (−29.7 to −4.49)**	**−5.42 (−20.9 to 10.1)**	**7.38 (−11.3 to 26.0)**	**0.003**
Systolic blood pressure (mmHg)[b]	829	0.23 (−2.94 to 3.41)	0.29 (−1.37 to 1.96)	0.29 (−1.16 to 1.74)	0.88	**3.34 (−0.02 to 6.71)**	**1.29 (−1.27 to 3.86)**	**−0.35 (−3.53 to 2.82)**	**0.036**
Diastolic blood pressure (mmHg)[b]	829	**−2.42 (−4.62 to −0.21)**	**−1.29 (−2.85 to 0.27)**	**0.53 (−0.79 to 1.84)**	**0.005**	**3.64 (−0.16 to 7.45)**	**−0.10 (−2.83 to 2.64)**	**−0.89 (−3.65 to 1.87)**	**0.047**
C-reactive protein (mg/L)	712	0.28 (−0.71 to 1.26)	0.16 (−0.26 to 0.59)	0.16 (−0.23 to 0.55)	0.58	0.13 (−1.16 to 1.42)	−0.01 (−0.66 to 0.63)	0.24 (−1.03 to 1.50)	0.95
Total cholesterol (mmol/L)	725	−0.20 (−0.52 to 0.12)	0.00 (−0.34 to 0.35)	−0.06 (−0.27 to 0.15)	0.68	−0.22 (−0.60 to 0.16)	−0.09 (−0.38 to 0.14)	0.05 (−0.26 to 0.36)	0.21
HDL cholesterol (mmol/L)	725	−0.05 (−0.20 to 0.10)	−0.02 (−0.15 to 0.12)	−0.03 (−0.08 to 0.03)	0.82	**−0.12 (−0.28 to 0.04)**	**−0.13 (−0.24 to −0.02)**	**−0.03 (−0.11 to 0.06)**	**0.047**
LDL cholesterol (mmol/L)	440	0.10 (−0.25 to 0.44)	0.17 (−0.20 to 0.54)	0.08 (−0.15 to 0.32)	0.5	−0.03 (−0.49 to 0.43)	−0.07 (−0.40 to 0.26)	−0.16 (−0.60 to 0.28)	0.99
Log-triglyceride (mmol/L)	440	−0.01 (−0.12 to 0.09)	0.02 (−0.05 to 0.09)	0.00 (−0.06 to 0.06)	0.92	−0.06 (−0.17 to 0.05)	0.02 (−0.05 to 0.09)	−0.00 (−0.18 to 0.17)	0.37
Fasting glucose (mmol/L)	454	−0.10 (−0.36 to 0.16)	−0.23 (−0.53 to 0.07)	−0.18 (−0.47 to 0.11)	0.38	−0.07 (−0.33 to 0.19)	−0.00 (−0.37 to 0.36)	−0.02 (−0.24 to 0.19)	0.68
Fasting insulin (pmol/L)	424	4.57 (−14.8 to 23.9)	0.16 (−11.4 to 11.8)	−2.93 (−15.8 to 9.9)	0.57	−6.88 (−30.5 to 16.7)	10.9 (−16.9 to 38.7)	1.93 (−15.4 to 19.3)	0.74
HOMA-IR	422	−0.06 (−0.84 to 0.71)	−0.21 (−0.80 to 0.37)	−0.30 (−0.93 to 0.34)	0.93	−0.17 (−0.92 to 0.57)	0.29 (−0.60 to 1.18)	−0.07 (−0.70 to 0.56)	0.9

Continued on the following page

TABLE 3 (continued)

Multivariate linear or logistic regression analysis of socioeconomic gradients in cardiovascular risks in Canadian children, 6–11 years, by sex

	Sample, n	Income adequacy[a]				Highest parental education[a]			
		Lowest	Lower-middle	Upper-middle	*p for trend*	< Secondary	Secondary	Some post-secondary	*p for trend*
		Odds ratio (95% confidence interval)				Odds ratio (95% confidence interval)			
Overweight/obese	833	**2.46 (1.10 to 5.48)**	**2.63 (1.47 to 4.71)**	**1.48 (0.80 to 2.71)**	**0.01**	1.20 (0.25 to 5.78)	1.80 (0.74 to 4.38)	1.15 (0.35 to 3.82)	0.68
Poor aerobic fitness	651	2.12 (0.61 to 7.34)	2.46 (0.98 to 6.16)	1.28 (0.57 to 2.86)	0.11	**3.73 (1.22 to 11.4)**	**1.36 (0.44 to 4.24)**	**0.63 (0.14 to 2.89)**	**0.01**

Source: 2007–2011 Canadian Health Measures Survey.

Abbreviations: **HDL, high-density lipoprotein; HOMA-IR, homeostasis model assessment of insulin resistance; LDL, low-density lipoprotein.**

Note: The fully-adjusted model controls for household income adequacy, highest parental education, age (continuous) and ethnicity.

[a] Reference group: highest household income adequacy and parental post-secondary education.

[b] Model additionally controls for height and heart rate.

with other Canadian studies. A study of children in Grades 6 to 10 found that both individual- and area-level SES measures were associated with obesity,[7] and a study of Grade 5 students in a Nova Scotia school reported similar findings,[16] though Shields et al.[31] did not find such an association when using national survey data.

None of these studies of SES and overweight in children conducted sex-specific analyses. Our results—a sex-specific pattern, with gradients of income adequacy more strongly associated with overweight in young girls than in boys—mirror findings in recent Canadian studies of adults in which SES gradients in overweight risk were stronger in women than in men.[32,33] That children from families living in low income have limited access to material resources and are less able to afford leisure activities like organized sports is well understood; also accepted is that participating in organized sports promotes weight loss. In this study, we could not determine whether girls are more vulnerable to living in a disadvantaged socioeconomic environment or whether such an impact begins early in childhood or, indeed, if girls in high SES groups are more influenced and pressured by social norms to stay slim at an early age.[34] Furthermore, lack of SES gradients in overweight in young boys should not undermine the high prevalence of obesity in this population. The lack of SES gradient in overweight in young boys may be due to cultural/social attitudes that accept heavier weighted boys across all income groups in some ethnic/racial groups. Further evidence is shown by our finding that boys of non-White ethnicity were at higher risk of overweight independent of SES. Our finding supports federal/provincial/territorial governments' focus on initiatives to reduce childhood obesity and maintain healthy weight in children,[35] and further suggests that interventions to prevent childhood obesity and to reduce SES gradients in obesity should not only target low-SES girls but also boys of all SES groups.

Another key finding of this study is the striking sex differences and SES gradients in aerobic fitness in Canadian children and adolescents. We found a persistent SES gradient in aerobic fitness in Canadian girls throughout their childhood. Aerobic fitness, or the measured AFS we used in this study, is generally considered a physiological outcome of frequent physical activity and an objective marker of this behaviour.[20] The mechanisms by which low-SES may potentially affect the physical fitness of a child include, but are not limited to, gaps in health education, low-quality nutritive food and poor access to recreational facilities.

To the best of our knowledge, this is the first study to analyze the relationship between SES and physical fitness in Canadian youth. Our findings are similar to studies of US[34] and Swedish[36] youth that reported stronger SES gradients in physical fitness in girls than in boys. There is some evidence that boys are more likely to engage in vigorous leisure-time activity than girls, regardless of their SES, and that girls are more likely to engage in physical activity more in the form of organized sports, for which participation may be more encouraged and supported in high SES groups.[37,38] This hypothesis is supported by Canadian and other studies that showed that girls in low-SES neighbourhoods engaged in significantly more screen time than did girls who lived in high SES neighbourhoods, a relationship not observed in boys.[39,40] Note that some studies have suggested that organized sport is one of the best ways to encourage vigorous activity in adolescent girls,[41,42] and that vigorous physical activity is the best way to achieve cardiorespiratory fitness. Intervention research on effectively promoting physical fitness to reduce SES gradients in Canadian youth, and especially those in low-SES groups, is needed.

Our study identified SES gradients in biomarkers of CVD risk in Canadian children, that is, SES gradients in BP in young children and in HDL and LDL cholesterol levels in adolescents, suggesting the lifelong SES gradients in CVD risks are physiologically identifiable in childhood. Intergenerational research suggests that parents' education may affect children's health indirectly through income or by affecting a child's psychological well-being through poor parenting style or through chronic stress, which may be disproportionally high in children in socioeconomically disadvantaged families.[12] Our finding of an educational gradient in BP is consistent with the results of other studies that found that low parental education and

TABLE 4
Multivariate linear or logistic regression analysis of socioeconomic gradients in cardiovascular risks in Canadian adolescents (ages 12-17) by gender

	Sample, n	Income adequacy[a]				Highest parental education[a]			
		Lowest	Lower-middle	Upper-middle	*p for trend*	< Secondary	Secondary	Some post-secondary	*p for trend*
		β-coefficient (95% confidence interval)				β-coefficient (95% confidence interval)			
Boys									
Body mass index (kg/m^2)	845	**−2.72 (−4.19 to −1.25)**	**−1.58 (−2.93 to −0.23)**	**−0.03 (−1.12 to 1.07)**	**< 0.001**	−0.26 (−3.26 to 2.74)	0.79 (−1.34 to 2.91)	0.80 (−0.80 to 2.40)	0.87
Waist circumference (cm)	844	**−2.32 (−3.74 to −0.90)**	**−1.01 (−2.34 to 0.32)**	**−0.05 (−1.23 to 1.13)**	**0.0003**	0.89 (−1.41 to 3.19)	1.12 (−0.91 to 3.15)	1.28 (−0.41 to 2.98)	0.49
Aerobic fitness score	674	−13.2 (−44.6 to 18.2)	−1.60 (−13.7 to 10.5)	−5.83 (−20.1 to 8.45)	0.45	9.48 (−21.9 to 40.8)	−18.0 (−50.0 to 14.0)	−6.96 (−29.1 to 15.1)	0.73
Systolic BP (mmHg)[b]	843	**−2.30 (−5.27 to 0.66)**	**−0.98 (−3.91 to 1.94)**	**1.52 (−0.12 to 3.15)**	**0.04**	−2.31 (−8.37 to 3.76)	2.48 (0.03 to 4.93)	3.09 (−0.46 to 6.65)	0.41
Diastolic blood pressure (mmHg)[b]	843	−0.97 (−4.33 to 2.39)	−0.93 (−4.25 to 2.38)	0.31 (−1.40 to 2.01)	0.43	−5.31 (−13.5 to 2.92)	1.53 (−1.54 to 4.59)	1.78 (−1.26 to 4.81)	0.21
C-reactive protein (mg/L)	793	−0.23 (−0.99 to 0.53)	−0.19 (−0.51 to 0.13)	0.07 (−0.22 to 0.35)	0.24	0.60 (−0.21 to 1.40)	0.23 (−0.25 to 0.70)	0.19 (−0.41 to 0.79)	0.12
Total cholesterol (mmol/L)	806	**−0.27 (−0.52 to −0.01)**	**−0.02 (−0.33 to 0.29)**	**0.12 (−0.10 to 0.35)**	**0.02**	−0.36 (−1.21 to 0.48)	0.24 (−0.07 to 0.56)	−0.05 (−0.36 to 0.26)	0.54
HDL cholesterol (mmol/L)	761	−0.09 (−0.18 to 0.01)	0.03 (−0.05 to 0.11)	0.03 (−0.05 to 0.11)	0.1	−0.15 (−0.38 to 0.08)	−0.05 (−0.13 to 0.02)	−0.09 (−0.19 to 0.02)	0.17
LDL cholesterol (mmol/L)	395	**−0.45 (−0.84 to −0.05)**	**−0.08 (−0.43 to 0.27)**	**0.07 (−0.16 to 0.31)**	**0.02**	**0.56 (−0.18 to 1.31)**	**0.68 (0.02 to 1.34)**	**0.04 (−0.41 to 0.49)**	**0.04**
Log-triglyceride (mmol/L)	395	−0.07 (−0.16 to 0.01)	0.02 (−0.07 to 0.10)	0.04 (−0.02 to 0.11)	0.09	0.15 (−0.04 to 0.33)	0.06 (−0.09 to 0.21)	0.01 (−0.14 to 0.16)	0.08
Fasting glucose (mmol/L)	394	0.25 (0.03 to 0.48)	0.29 (−0.26 to 0.89)	0.15 (0.02 to 0.29)	0.06	**−0.43 (−0.72 to −0.06)**	**−0.20 (−0.49 to 0.09)**	**−0.10 (−0.33 to 0.13)**	**0.004**
Fasting insulin (pmol/L)	386	**−24.1 (−49.3 to 1.12)**	**−8.84 (−26.6 to 8.90)**	**15.9 (−8.53 to 40.3)**	**0.01**	36.7 (−16.6 to 90.1)	30.8 (−14.1 to 75.7)	21.5 (−17.8 to 60.8)	0.13
HOMA-IR	385	**−0.67 (−1.45 to 0.12)**	**−0.27 (−0.86 to 0.31)**	**0.52 (−0.20 to 1.24)**	**0.02**	0.91 (−0.72 to 2.98)	0.80 (−0.76 to 2.67)	0.75 (−0.44 to 1.94)	0.21
		Odds ratio (95% confidence interval)				Odds ratio (95% confidence interval)			
Overweight/obese	845	0.38 (0.11 to 1.31)	0.71 (0.38 to 1.27)	1.28 (0.84 to 1.94)	0.06	0.89 (0.11 to 7.97)	0.92 (0.34 to 2.48)	0.93 (0.49 to 1.78)	0.92
Poor aerobic fitness	674	1.14 (0.13 to 9.94)	0.55 (0.12 to 2.49)	0.91 (0.17 to 4.75)	0.96	2.57 (0.14 to 47.5)	—	2.60 (0.37 to 18.1)	0.29
		β-coefficient (95% confidence interval)				β-coefficient (95% confidence interval)			
Girls									
Body mass index (kg/m^2)	764	0.12 (−1.87 to 2.10)	0.62 (−0.79 to 2.03)	−0.12 (−1.10 to 0.86)	0.71	**2.84 (−0.43 to 6.10)**	**0.50 (−0.87 to 1.86)**	**−0.13 (−1.52 to 1.26)**	**0.05**
Waist circumference (cm)	761	0.57 (−2.39 to 3.53)	0.68 (−0.66 to 2.02)	−0.08 (−1.21 to 1.05)	0.57	2.45 (−0.93 to 5.83)	0.98 (−0.78 to 2.75)	−0.38 (−1.75 to 0.98)	0.07
Aerobic fitness score	634	**−17.5 (−41.1 to 6.10)**	**−22.5 (−32.1 to −12.9)**	**−3.02 (−13.6 to 7.52)**	**0.05**	0.35 (−19.1 to 19.8)	−13.7 (−31.5 to 4.11)	8.56 (−7.56 to 24.7)	0.46
Systolic blood pressure (mmHg)[b]	762	3.37 (−1.10 to 7.83)	0.62 (−1.97 to 3.21)	−0.22 (−1.92 to 1.47)	0.11	−0.70 (−5.51 to 4.11)	0.59 (−2.57 to 3.75)	−1.42 (−3.74 to 0.89)	0.99
Diastolic blood pressure (mmHg)[b]	762	2.64 (−1.05 to 6.33)	1.43 (−1.01 to 3.88)	0.45 (−0.89 to 1.79)	0.11	−0.83 (−6.81 to 5.14)	−1.58 (−3.35 to 0.18)	−2.00 (−3.98 to −0.03)	0.8
C-reactive protein (mg/L)	692	0.15 (−0.63 to 0.92)	0.22 (−0.44 to 0.88)	−0.10 (−0.71 to 0.51)	0.51	−0.59 (−1.17 to −0.02)	0.71 (−0.41 to 1.84)	−0.43 (−0.93 to 0.08)	0.55
Total cholesterol (mmol/L)	705	**−0.53 (−1.05 to −0.01)**	**−0.12 (−0.38 to 0.14)**	**0.11 (−0.14 to 0.37)**	**0.01**	−0.05 (−0.40 to 0.30)	0.02 (−0.24 to 0.29)	−0.12 (−0.40 to 0.16)	0.98
HDL cholesterol (mmol/L)	705	**−0.15 (−0.32 to 0.03)**	**−0.09 (−0.19 to 0.00)**	**−0.01 (−0.08 to 0.06)**	**0.045**	0.00 (−0.12 to 0.13)	−0.01 (−0.13 to 0.10)	−0.02 (−0.13 to 0.09)	0.93
LDL cholesterol (mmol/L)	352	−0.55 (−1.40 to 0.29)	0.02 (−0.26 to 0.30)	0.03 (−0.38 to 0.43)	0.17	−0.07 (−0.59 to 0.46)	−0.11 (−0.48 to 0.27)	−0.20 (−0.58 to 0.17)	0.89
Log-triglyceride (mmol/L)	352	0.03 (−0.06 to 0.12)	−0.06 (−0.16 to 0.05)	−0.01 (−0.08 to 0.06)	0.76	0.02 (−0.08 to 0.12)	0.05 (−0.03 to 0.13)	0.05 (−0.02 to 0.13)	0.75
Fasting glucose (mmol/L)	352	0.20 (−0.06 to 0.46)	−0.17 (−0.34 to −0.01)	−0.05 (−0.16 to 0.06)	0.25	0.03 (−0.17 to 0.23)	−0.21 (−0.39 to 0.03)	−0.06 (−0.20 to 0.06)	0.83
Fasting insulin (pmol/L)	341	11.1 (−27.3 to 49.4)	−8.65 (−27.0 to 9.69)	−6.49 (−19.1 to 6.12)	0.58	−8.44 (−31.3 to 14.4)	19.7 (−1.23 to 40.7)	−6.89 (−26.5 to 12.7)	0.96
HOMA-IR	341	0.38 (−0.69 to 1.46)	−0.32 (−0.90 to 0.27)	−0.24 (−0.64 to 0.15)	0.5	−0.26 (−0.93 to 0.41)	0.43 (−0.13 to 0.99)	−0.24 (−0.81 to 0.34)	0.89

Continued on the following page

TABLE 4 (continued)

Multivariate linear or logistic regression analysis of socioeconomic gradients in cardiovascular risks in Canadian adolescents (ages 12-17) by gender

	Sample, n	Income adequacy[a]				Highest parental education[a]			
		Lowest	Lower-middle	Upper-middle	*p for trend*	< Secondary	Secondary	Some post-secondary	*p for trend*
		Odds ratio (95% confidence interval)				Odds ratio (95% confidence interval)			
Overweight/obese	764	1.45 (0.44 to 4.76)	1.32 (0.57 to 3.07)	1.12 (0.58 to 2.18)	0.48	**3.60 (0.83 to 15.7)**	**2.10 (1.03 to 4.27)**	**0.90 (0.34 to 2.40)**	**0.02**
Poor aerobic fitness	634	2.13 (0.37 to 12.3)	2.53 (1.20 to 5.34)	1.22 (0.51 to 2.93)	0.25	1.28 (0.27 to 6.12)	2.52 (0.90 to 7.05)	0.39 (0.09 to 1.70)	0.29

Source: 2007–2011 Canadian Health Measures Survey.

Abbreviations: **HDL, high-density lipoprotein; HOMA-IR, homeostasis model assessment of insulin resistance; LDL, low-density lipoprotein.**

Note: The fully-adjusted model controls for household income adequacy, highest parental education, age (continuous) and ethnicity.

[a] Reference group: highest household income adequacy and parental post-secondary education.

[b] Model additionally controls for height and heart rate.

—: less than secondary and secondary parental education categories were combined because the sample size was insufficient.

harsh family environments explain some BP variability in children.[43,44]

Studies of the relationship between SES and lipids in children in other populations have produced inconsistent results.[45] Current evidence suggests that race/ethnicity, physical activity and body weight are important predictors for HDL and foods high in saturated fat, physical activity and body weight are among the determinants for LDL in children and adolescents.[46] Our finding of a relatively favorable HDL profile in non-Whites independent of SES suggests that some ethnic groups are more genetically protected than others from CVD. Both physical activity and body weight are among the determinants of cholesterol levels at an early age, reinforcing existing public health messages aimed at promoting physical activity and healthy diets and preventing overweight, particularly in socioeconomically disadvantaged children (and adolescents in particular) in order to prevent disparity in cardiovascular risk that could originate in childhood.

Strengths and limitations

Strengths of this study included our use of a nationally representative sample that provides reliable information on objective measures of CVD risks that allowed us to analyze early effects of SES on physiological markers of CVD risks. A population-based sampling strategy allows the results to be generalized to the Canadian population.

Limitations of our study are the small sample size, especially for low-SES groups and fasting samples, which may have prevented us from detecting an SES gradient or interpreting trends in subpopulations with large measurement variability, and the inability to examine potential interactions between SES indicators. Another limitation was biases associated with self-reported information on family income and parental education. Due to the cross-sectional design of the study, we were unable to assess changes (increases or decreases) of SES gradients in CVD risks over time throughout childhood. Some evidence suggests that childhood SES gradients in health track through adulthood,[47] whereas others found that SES gradients established in childhood do not persist through adolescence;[18] these different conclusions suggest the need for further research.

Conclusion

We sought to determine whether the clear SES gradient in CVD risk observed in Canadian adults is also seen in children and adolescents. We analyzed independent associations of income adequacy and parental education with physiological markers for CVD risks. Our study identified striking sex and SES gradients in adiposity and aerobic fitness in Canadian children, in particular in girls aged 6 to 17 years. Although an SES gradient in adiposity was not apparent in boys, and in fact, an inverse SES gradient in overweight was found in adolescent boys, young boys presented a considerably higher prevalence of obesity and abdominal obesity than do girls. This population should not be overlooked in the efforts to reduce CVD risk in childhood.

The educational gradients in some of the physiological markers that we observed in Canadian youth require further examination to confirm. However, health promotion should consider increasing awareness of early CVD risks in parents, especially less educated parents.

Our findings support current priorities to reduce childhood obesity and health inequalities in children.[35] Our study further suggests that public health interventions to prevent SES gradients in CVD risk should focus on reducing childhood obesity and promote physical fitness in all children, with special focus on girls and low-SES groups.

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Prevalence of metabolic syndrome and its risk factors in Canadian children and adolescents: Canadian Health Measures Survey Cycle 1 (2007-2009) and Cycle 2 (2009-2011)

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Abstract

Introduction: We investigated the prevalence of metabolic syndrome (MetS) and its risk factors, and the influence of socioeconomic status, in Canadian children and adolescents.

Methods: Canadian Health Measures Survey cycle 1 (2007–2009) and cycle 2 (2009–2011) respondents aged 10 to 18 years who provided fasting blood samples were included (n = 1228). The International Diabetes Federation (IDF) consensus definition for children and adolescents (10–15 years) and worldwide adult definition (≥ 16 years) were used to diagnose MetS. Prevalence of MetS and its risk factors were calculated and differences by socioeconomic status were examined using χ^2 tests.

Results: The prevalence of MetS was 2.1%. One-third (37.7%) of participants had at least one risk factor, with the most prevalent being abdominal obesity (21.6%), low HDL-C (19.1%) and elevated triglyceride levels (7.9%). This combination of abdominal obesity, low HDL-C and elevated triglyceride levels accounted for 61.5% of MetS cases. Participants from households with the highest income adequacy and educational attainment levels had the lowest prevalence of one or more MetS risk factors, abdominal obesity and low HDL-C.

Conclusion: The prevalence of MetS (2.1%) was lower than previously reported in Canada (3.5%) and the USA (4.2%–9.2%), potentially due to the strict application of the IDF criteria for studying MetS. One-third of Canadian children and adolescents have at least one risk factor for MetS. Given that the risk for MetS increases with age, these prevalence estimates, coupled with a national obesity prevalence of almost 10% among youth, point to a growing risk of MetS and other chronic diseases for Canadian youth.

Keywords: *Canadian Health Measures Survey, metabolic syndrome, health surveys, cardiometabolic risk factors, prevalence, adolescent, child*

Key findings

- Having metabolic syndrome (MetS) increases the risk for chronic disease—cardiovascular disease by two and type 2 diabetes by five.
- Only 2.1% of Canadian youth have MetS. However, one-third of Canadian youth have one or more risk factors for MetS.
- The biggest risk factor for MetS is abdominal obesity. As more youth are becoming obese, MetS will probably increase among Canadian youth.
- Risk of MetS increases with age. As a result, the risk for chronic diseases will probably increase as the Canadian population ages.
- Youth who live in better off or better educated households have the lowest risk for MetS.

Introduction

Chronic diseases constitute the leading cause of preventable death in Canada and the world as well as the largest avoidable burden on the public health care system.[1] The metabolic syndrome (MetS) is a constellation of cardiometabolic risk factors that are predictive for chronic disease and all-cause mortality.[2-4] It is estimated that risk of cardiovascular disease (CVD) doubles and the risk of type 2 diabetes increases fivefold if MetS is present.[3-6]

MetS is characterized by the presence of different combinations of risk factors including obesity, hypertension, elevated fasting triglycerides, insulin resistance, low total cholesterol, high low-density lipoprotein cholesterol, low high-density lipoprotein cholesterol (HDL-C), elevated apolipoprotein B, elevated C-reactive protein and elevated homocysteine.[7-9] These clinical features of MetS, if present together, tend to suggest a common etiology; the proposed mechanisms underlying MetS and its influence on health outcomes are discussed elsewhere.[7,10,11]

The global prevalence of obesity and diabetes has increased dramatically in the past quarter century.[12] This increase, in

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turn, has contributed to a higher prevalence of MetS.[13] Worldwide estimates of the prevalence of MetS range from 1.2% to 22.6% for youth and 9.0% to 35.0% for adults, depending on the definition of MetS used, the region, the study design, the years of the study, and the age group and study population.[13-16] In Canada, the prevalence of MetS among adults is between about 11.4% and 22.2%, which is greater than the prevalence estimates of 10% to 15% measured in adults in the early 1990s.[17-22] In comparison, the prevalence of MetS among adults in the USA is between about 22% and 34%.[23-26] It is widely accepted that the prevalence of MetS increases significantly with age.[17-20,23] The national prevalence among youth aged 12 to 19 years is 3.5% in Canada (based on a 2012 study using the Adult Treatment Panel III criteria for MetS) and 4.2% to 9.2% in the USA, with about 42% to 63% of youth in the USA having one or more MetS risk factors.[19,27-29] Further examination of national prevalence among youth will help us understand the progression of MetS and its risk factors among Canadians.

There is substantial evidence supporting an inverse relationship between socioeconomic status (SES) and CVDs, conditions that share some risk factors with MetS.[30-32] Studies examining the relationship between SES and MetS reveal a similar pattern in which people with a lower social status experience a significantly higher prevalence of MetS.[17,19,20,33,34] Canadian national studies have shown that the prevalence of MetS is significantly lower among people from households with postsecondary education compared to those with less education, a relationship that is particularly evident in women.[17,19,20] This inverse relationship remains consistent between household income and MetS, albeit less pronounced, with Canadian households with the lowest quartiles of income having a higher prevalence of MetS than households with average and higher incomes.[17,20]

A challenge in determining the prevalence of MetS has been the use of multiple criteria and definitions for identifying this condition. In response, the International Diabetes Federation (IDF) released the *IDF Consensus Worldwide Definition of the Metabolic Syndrome* as a single, universally accepted tool.[35] The IDF defines MetS as the presence of abdominal obesity (measured by waist circumference) and 2 or more of the following risk factors: low levels of HDL-C, hypertension, elevated fasting triglyceride levels and elevated glucose concentration.[6,36] Before the IDF consensus definition, the most recognized definitions were criteria established by the World Health Organization, the European Group for the Study of Insulin Resistance, and the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III criteria) (NCEP ATP III).[9,37,38]

Diagnosing MetS among children and adolescents proves particularly challenging given the difficulty in establishing accurate, meaningful and harmonized criteria for this population. Consequently, prevalence estimates of MetS among children and youth vary greatly depending on the adopted definition.[8] In 2007, the IDF released their *Consensus Definition of the Metabolic Syndrome in Children and Adolescents*.[36] This criterion provides an age- and sex-specific definition for youth aged 10 to 15 years. The IDF definition further stipulates that the worldwide adult definition of MetS should be applied for individuals aged 16 years or older and that MetS should not be diagnosed in children less than 10 years old.[36]

The main objectives of this study were to investigate the prevalence of MetS and its risk factors, and the influence of SES on these risk factors, in Canadian children and adolescents (10–18 years) using nationally representative data from the Canadian Health Measures Survey (CHMS). This study builds upon an earlier national analysis of Canadian youth

- by including those aged 10 and 11 years;
- by calculating the prevalence of one or more risk factors for MetS among youth;
- by examining the patterns of risk presentation; and
- by using data from two cycles of the CHMS.[19]

This is the first national study to strictly apply the IDF consensus definition of MetS in children and adolescents, the most current and universally accepted definition of MetS for youth; and to use Canadian age- and sex-specific waist circumference reference data to determine abdominal obesity in Canadian children and youth.

Methods

Data source

The CHMS is a nationally representative survey designed to collect information on the health of Canadians.[39-41] Conducted by Statistics Canada, the CHMS consists of an in-home interview and a physical assessment conducted at a mobile examination centre. The interview collects demographic, socioeconomic, family history and general health information. The physical assessment includes measures of anthropometry, spirometry, blood pressure, fitness and oral health and involves collecting biological specimens.[39-41] The survey covered Canadians living at home in the 10 provinces and 3 territories, although people living on reserves and other Aboriginal settlements, in institutions and in certain remote regions as well as full-time members of the Canadian Forces were excluded.[39-41] The CHMS cycle 1 (2007–2009) collected data on people aged 6 to 79 years, with cycle 2 (2009–2011) expanding to cover those aged 3 to 79 years.[39-41] In total, this represents 96.3% of the Canadian population.[39-41]

The CHMS produces reliable estimates at the national level by age group and sex through a multistage sampling strategy.[39-42] The selection of collection sites was informed by the Labour Force Survey sampling frame. A multitude of practices were used to minimize non-response; the combined response rate for home and clinic visits was 51.7% for cycle 1 and 55.5% for cycle 2.[39-42] Statistics Canada calculated the sampling weights by multiplying the selection weights for collection sites by the selection weights for dwellings, followed by a series of adjustments for non-response at the initial, interview and MEC stage.[42]

Study population

All 10- to 18-year-old CHMS respondents who provided fasting blood samples for

cycle 1 (2007–2009) or cycle 2 (2009–2011) were included (n = 1228). No participants were pregnant. Sample weights specific to the fasting subgroup were provided by Statistics Canada to ensure appropriate representativeness at the population level.

Criteria for diagnosing MetS

We applied the IDF consensus definition of MetS for children and adolescents to participants aged 10 to 15 years and the IDF worldwide adult definition adult criteria to participants aged 16 to 18 years.

The IDF consensus definition for children and adolescents defines MetS as having abdominal obesity (waist circumference equal or greater than the 90th percentile by age and sex) and the presence of two or more of the following clinical features: elevated triglycerides ($\geq$ 1.7 mmol/L); low HDL-C ($<$ 1.03 mmol/L); high blood pressure (systolic $\geq$ 130 mm Hg and/or diastolic $\geq$ 85 mm Hg and/or diagnosis of hypertension); and elevated glucose ($\geq$ 5.6 mmol/L and/or diagnosis of type 2 diabetes).[36]

The IDF worldwide adult criteria define MetS as having abdominal obesity and the presence of two or more of the following clinical features: high triglycerides ($\geq$ 1.7 mmol/L), low HDL-C ($<$ 1.03 mmol/L in males and $<$ 1.29 mmol/L in females); high blood pressure (systolic $\geq$ 130 mm Hg and/or diastolic $\geq$ 85 mm Hg and/or diagnosis of hypertension); and high glucose ($\geq$ 5.6 mmol/L and/or diagnosis of type 2 diabetes).[35]

We defined abdominal obesity using the 90th percentiles from the age- and sex-specific waist circumference reference data established from the 1981 Canadian Fitness Survey.[43] We applied the waist circumference cut-offs for 11-year-olds to those aged 10 to 11 years since this reference provided estimates for those aged 11 to 18 years only.

Variables for assessing demographic and socioeconomic status

A respondent's demographic and SES was assessed through the variables of household educational attainment, household income adequacy, Aboriginal status, and immigrant status. The use of household education and household income variables in this study is consistent with previous studies examining the relationship between SES and MetS.[17,19,20,26] Education is the most frequently used indicator of SES in epidemiological studies and, among indicators of SES, it tends to have the strongest and most consistent relationship with cardiovascular health.[20,31,44] Household income is another well-established SES indicator and determinant of health.[44-48] Statistics Canada calculated income adequacy by classifying each participant into categories based on total household income from all sources and the number of people living in the household.[39,40]

To allow for greater statistical power, we reclassified both the household educational attainment and income adequacy variables from 4 categories into 3. For income adequacy, we combined the ''lowest income'' and ''lower middle income'' categories, resulting in ''lowest and lower middle,'' ''upper middle'' and ''highest'' categories. For household educational attainment, we combined the ''less than secondary school graduation'' and ''secondary school graduation'' categories, resulting in ''secondary school graduation or less,'' ''some postsecondary'' and ''postsecondary graduation'' categories.

Statistical analysis

We conducted statistical analyses using SAS version 9.3 (SAS Institute Inc., Cary, NC, US) for data manipulation and variance estimation using the bootstrap method.[49] The prevalence of MetS and each risk factor were estimated and expressed as a frequency and a percentage with a 95% confidence interval (CI). χ^2 tests were used to examine differences in MetS, and each risk factor by gender, Aboriginal status, immigrant status, household education and income adequacy. The analyses were conducted using weighting and bootstrapping. Statistical significance was set at a p value of less than .05.

We obtained ethics approval for this project from the University of Ottawa's Research Ethics Board.

Results

Description of study sample

To be able to evaluate the criteria for MetS, of the original sample of child and adolescent respondents aged 10 to 18 years, we included in our study only those participants who provided fasting blood samples. This resulted in a final sample of 1228 participants. The sample included slightly more males (51.5%) than females (48.5%).

Table 1 shows an overview of the sample by demographic and SES.

Prevalence of MetS

Only 25 study participants were diagnosed with MetS, which represents 2.1% of participants (95% CI: 0.8–3.3)* (Table 2). This small number of participants with MetS prevented accurate disaggregation by sex, age or SES.

Prevalence of individual risk factors

Over one-third (37.7%; 95% CI: 33.8–41.6) of children and adolescents had at least one of the clinical features of MetS (1 or more risk factors) (Table 2). Risk factors in order of prevalence were abdominal obesity (21.6%; 95% CI: 16.6–26.7), low HDL-C (19.1%; 95% CI: 16.6–21.8), elevated triglycerides (7.9%; 95% CI: 4.8-11.0) and elevated glucose (1.7%; 95% CI: 0.7–2.8)†. The prevalence of elevated blood pressure was too low to provide an accurate statistical estimate. There were no gender differences for the prevalence of each risk factor.

Pattern of risk factor combinations

The most prevalent single risk factors were abdominal obesity (10.7%), low HDL-C (9.8%) and elevated triglycerides (2.7%) (Table 3). The most prevalent distinct combinations of two risk factors were abdominal obesity coupled with low HDL-C (5.1%) and abdominal obesity and elevated triglycerides (1.5%). Among distinct combinations of three risk factors, the most prevalent combination was abdominal obesity, low HDL-C

*This result is published with caution due to a coefficient of variation (CV) of 29.0.
†Due to small cell sizes, not all risk factors and SES categories could be reported.

TABLE 1
Sample profile, 10–18 years[a]

Characteristics	Study sample, n	Percentage of study sample, %
Demographic profile (n = 1228)		
Sex		
Male	632	51.5
Female	596	48.5
Age, years		
10	172	14.0
11	184	15.0
12	127	10.3
13	151	12.3
14	115	9.4
15	117	9.5
16	131	10.7
17	121	9.8
18	110	9.0
Socioeconomic profile		
Income adequacy (n = 1178)		
Lowest and lower middle	247	19.7
Upper middle	333	25.8
Highest	598	50.4
Household education (n = 1193)		
Secondary school graduation or less	126	11.0
Some postsecondary	81	6.4
Postsecondary graduation	986	78.3
Aboriginal origin or identity (n = 1227)		
Aboriginal	46	4.4
Not Aboriginal	1181	95.5
Immigrant status		
Immigrant	120	10.2
Not immigrant	1108	89.8

[a]Figures are based on raw data.

and elevated triglycerides (1.3%). This combination of three risk factors accounted for 61.5% of MetS cases (Table 3).

Associations between SES (household educational attainment and income adequacy) and risk factors

Participants from families with the highest incomes had the lowest percentage of one or more risk factor(s) (35.5%; 95% CI: 29.8–41.2), abdominal obesity (18.4%; 95% CI: 11.7–25.1) and low HDL-C (17.5%; 95% CI: 14.2–20.6) versus those from families with the lowest and lower middle incomes (Table 4). Educational attainment results showed that participants with a household member with postsecondary graduation had the lowest percentage of one or more risk factor(s) (35.3%; 95% CI: 31.0–39.6), abdominal obesity (19.8%; 95% CI: 14.6–25.0) and low HDL-C (17.5%; 95% CI: 14.8–20.2) versus those from households with some postsecondary education or secondary school graduation or less. Due to small cell sizes, the results could not be disaggregated by Aboriginal or immigrant status.

Discussion

The prevalence for MetS among children and adolescents (2.1%) was lower than previously reported in Canada (3.5%) and the USA (4.2%–9.2%).[19,27-29] Assuming our sample is representative of the Canadian population, this prevalence of 2.1% would be equivalent to about 64 832 children and adolescents. The prevalence of one or more risk factors (37.7%) among children and adolescents was also lower than reported in the USA (42%–63%).[28] In comparison to earlier national estimates on Canadian youth, our study's lower prevalence may be attributed to our applying the IDF definition of MetS, which has slightly more stringent criteria, including the required presence of abdominal obesity.[19,43,50,51] Furthermore, MetS is known to increase with age and our sample included younger ages (10–11 years) and had greater numbers of younger participants (n = 356 for 10–11 years) than older, adolescent participants (n = 231 for 17–18 years).[17,23]

The lower prevalence estimates we found compared to those in the USA may be attributable to several factors. Obese youth have a higher prevalence of MetS than do those of normal weight and the prevalence of obesity among youth is higher in the USA than in Canada.[52-54] The prevalence estimates in the USA were calculated using data from the National Health and Nutrition Examination Survey with variation in the periods of data collection (ranging from 1988–2006), the MetS definition (all variations of ATP III) and criteria for abdominal obesity. Our study followed a strict application of the IDF MetS definition including age- and sex-specific cut-offs. Finally, our study does not include Canadian residents living on reserve or in other Aboriginal settlements, populations shown to have a higher prevalence of MetS.[55-57]

Despite the overall low prevalence of MetS, note that one-third (37.7%) of study participants had at least one risk factor for MetS. This finding, coupled with a prevalence of obesity of almost 10% among Canadian children and youth, is disconcerting as the probability of MetS also increases with obesity.[26] Further, given that age is one of the most significant predictors for MetS, it is reasonable to assume that children and adolescents with one or more risk factors are more susceptible to MetS and, correspondingly, chronic disease as adults.[2-4] Evidence indicates that, in the long term, adults with MetS have an elevated risk of CVD-attributed mortality, although a moderate-to-high level of cardiorespiratory fitness has been shown to mitigate some of this risk.[53,58]

TABLE 2
Prevalence of metabolic syndrome and risk factors[a], 10–18 years[a] (n = 1228)

Condition	Total Sample		Male		Female		*p* value
	Frequency, n	% (95% CI) CV	Frequency	% (95% CI) CV	Frequency	% (95% CI) CV	
MetS	25	2.1 (0.8–3.3) 0.29[b]	—	—	—	—	—
Number of risk factors							
≥ 2	123	10.8 (7.4–14.2) 0.15	71	6.1 (3.5–8.7) 0.02	52	4.7 (2.8–6.5) 0.19	.3658
≥ 1	420	37.7 (33.8–41.6) 0.05	212	18.1 (15.4–20.8) 0.07	208	19.6 (16.4–22.9) 0.08	.3179
Abdominal obesity	240	21.6 (16.6–26.7) 0.11	130	10.6 (7.3–13.9) 0.15	110	11.0 (7.5–14.5) 0.16[b]	.7443
Low HDL–C	218	19.1 (16.6–21.8) 0.06	107	8.8 (6.6–11.0) 0.12	111	10.54 (8.4–12.3) 0.09	.2863
Elevated triglycerides	82	7.9 (4.8–11.0) 0.19[b]	42	4.7 (2.2–7.3) 0.26[b]	40	3.2 (1.7–7.2) 0.22[b]	
Elevated glucose	22	1.7 (0.7–2.8) 0.30[b]	—	—	—	—	—

Abbreviations: BP, blood pressure; CV, coefficient of variation; HDL-C, high-density lipoprotein cholesterol.

Note: Blank cells (—) indicate that the results cannot be published because of a cell size n < 10 and/or a CV ≥ 0.3306. The prevalence of elevated BP was too low to provide an accurate statistical estimate.

[a]These figures are based on weighted data.

[b]These figures are published with reservation as 0.16 ≤ CV ≥ 0.33.

Our findings support the conclusions of previous studies that abdominal obesity, low HDL-C and elevated triglycerides are the most prevalent risk factors of MetS among children and adolescents; [28] in fact, this combination accounted for 61.5% of all MetS cases in this study. The most prevalent risk factor was abdominal obesity (21.6%), which may be attributed to over one-quarter of Canadian youth being overweight or obese.[59] The IDF considers abdominal obesity as a prerequisite for MetS given that it is associated with an increased risk of cardiovascular disease and an independent predictor of insulin resistance, lipid levels and high blood pressure.[35,36,60] Our study defined abdominal obesity using age- and sex-specific reference data established from the 1981 Canadian Fitness Survey (90th percentile).[43] Domestic prevalence estimates of obesity among youth have almost doubled in the past 25 years, meaning that these predefined cut-offs represent norms for the Canadian population before this dramatic increase in body fat.[59,61]

Consistent with previous studies on youth, hypertension is not highly prevalent in the early onset of this syndrome.[28]

Participants from families in the highest income adequacy and household educational attainment groups had the lowest prevalence of one or more risk factors, abdominal obesity and low HDL-C, which is consistent with earlier findings between SES and MetS risk factors.[17,19,20,62] For abdominal obesity, a dose–response relationship was present for household education. The relationship between household education and prevalence of risk factors appeared to be more sensitive than household income, which is also consistent with previous findings.[17,20,62] This may be attributed to the influence of education on health literacy and behaviour, such as nutrition and physical activity, which are related to abdominal obesity and MetS.[22,63] Further, household education is considered

TABLE 3
Pattern of metabolic syndrome risk factor combinations[a]

Risk factor combination (n = 1228)	Frequency (%)
Presence of 1 risk factor	
Abdominal obesity	131 (10.7)
Low HDL-C	121 (9.8)
Elevated TG	33 (2.7)
Presence of 2 risk factors	
Abdominal obesity + low HDL-C	63 (5.1)
Abdominal obesity + elevated TG	19 (1.5)
Presence of 3 risk factors	
Abdominal obesity + low HDL-C + elevated TG	16 (1.3)
Risk factor combination in participants with MetS (n = 26)	**Frequency (%)**
Presence of 3 risk factors	
Abdominal obesity + low HDL-C + elevated TG	16 (61.5)

Abbreviations: HDL-C, high-density lipoprotein cholesterol; TG, triglycerides.

Note: Risk factor combinations with cell sizes n < 10 were not published as prevalence were too low to provide accurate statistical estimates.

[a]These figures are based on weighted data.

TABLE 4
Relationship between metabolic syndrome risk factors and socioeconomic status, fasting sub-sample ages 10–18 years[a]

Condition	Presence of ≥ 1 risk factor(s) % (95% CI) CV	Abdominal obesity % (95% CI) CV	Low HDL-C % (95% CI) CV
Income Adequacy (50 missing)			
Lowest and lower middle	35.9 (25.9–46.0) 0.14	21.4 (11.8–30.9) 0.22[b]	19.4 (12.7–26.1) 0.17[b]
Upper middle	41.8 (34.4–49.3) 0.09	28.1 (19.7–36.6) 0.15	20.2 (15.2–25.2) 0.12
Highest	35.5 (29.8–41.2) 0.08	18.4 (11.7–25.1) 0.17[b]	17.5 (14.2–20.6) 0.09
Household education (35 missing)			
Secondary school graduation or less	43.7 (29.4–58.0) 0.16	31.8 (17.6–46.1) 0.22[b]	19.3 (6.9–31.7) 0.31[b]
Some postsecondary	42.8 (32.4–53.2) 0.12	28.3 (13.7–42.9) 0.25[b]	26.1 (15.1–37.3) 0.21[b]
Postsecondary graduation	35.3 (31.0–39.6) 0.06	19.8 (14.6–25.0) 0.13	17.5 (14.8–20.2) 0.08

Abbreviations: BP, blood pressure; HDL-C, high-density lipoprotein cholesterol; SES, socioeconomic status.

Note: Small cell sizes prohibited further analysis of BP, glucose and triglyceride risk factors and Aboriginal and immigrant status SES factors.

[a]These figures are based on weighted data.

[b]These figures are being published with reservation as 0.16 ≤ CV ≥ 0.33.

to be more stable, and less influenced by health status, than household income over the life course.[30] More broadly, participants from households with lower education and income levels are more likely to experience unfavourable social, physical and economic environments that can contribute to poorer health outcomes, including a higher rate of mortality attributed to CVDs.[30,47] These results point to a need for interventions, including public policy, public education, research and medical care, that focus on mitigating the impact of lower levels of education and income on health outcomes. Research focussed on elucidating the causal pathways through which SES influences the risk for MetS and CVDs throughout the life course would be useful in designing effective, targeted interventions.

Future studies using more cycles of CHMS data may have the statistical power with which to examine MetS and its risk factors in Canadian children and adolescents in greater detail. The sex differences in MetS in relation to SES should be examined to better understand the sex-specific ways in which unfavourable socioeconomic conditions affect MetS outcomes. Further, regression analyses are needed to comprehensively examine the relationship between MetS, its risk factors, behaviour such as physical activity and sleep, and SES.

Strengths

This is the first national study to apply the IDF consensus definition of MetS to children and adolescents and to use Canadian age- and sex-specific waist circumference reference data for determining abdominal obesity to Canadian children and youth. Strictly applying the IDF criteria for studying MetS at the population level in Canada will allow for more accurate comparisons with future studies on MetS in children and adolescents.

This study was conducted using government survey data that is both high quality and representative of 96% of Canadians.

Limitations

Descriptive statistics was the only method we could use to examine MetS using this dataset of Canadian children and adolescents because the sample size was small; only those participants from whom fasting blood samples were taken were included. The sample size and low prevalence of MetS did not allow for an analysis of the relationship between each risk factor and MetS. The small sample size also prohibited a robust statistical analysis of the influence of demographic and SES variables on MetS and allowed only limited analysis of the influence of these variables on risk factors with no distinction by sex. It was not feasible to disaggregate by sex, age, Aboriginal status or immigrant status. Furthermore, the cross-sectional design of the CHMS limits inference about causal pathways underlying the observed relationships. Consequently, the study focussed on the prevalence of each MetS risk factor.

Nonetheless, the study results improve the understanding of the current landscape of cardiometabolic risks among Canadian children.

Conclusions

By investigating the prevalence of MetS and its risk factors among Canadian children and adolescents, this study highlights important health and socioeconomic considerations for Canada's child and adolescent population. The results affirm previous findings of a low prevalence of MetS among youth. The results also highlight important indicators of future health risk among Canadian youth by showing that one in three have at least one risk factor for MetS, one in five have abdominal obesity, and one in five have low HDL-C. Efforts to prevent, diagnose and treat MetS and its risk factors among youth are important to prevent type 2 diabetes, cardiovascular diseases and premature mortality.

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Release notice

Strengthening the evidence base on social determinants of health: measuring everyday discrimination through a CCHS rapid response module

In March 2014, Statistics Canada released new data on discrimination in Canada, the collection of which was funded by the Public Health Agency of Canada (PHAC). These data are now available to researchers across the country through the Canadian Research Data Centre Network (CRDCN).

A growing body of evidence indicates that discrimination is related to adverse health outcomes (including mental health,[1,2] family violence[3,4] and obesity[5]). Discrimination has a spectrum of effects across multiple levels: it can be expressed in ''micro-aggressions'' (assaults on dignity and social status) or in severe verbal or physical assaults, which may result in psychosocial effects such as stress and distress or in physical injury; it can also lead to negative outcomes for health and well-being by creating and reinforcing social inequalities (e.g. in income, housing, employment), which in turn, limit access to resources and opportunities.[6]

However, our capacity to measure discrimination in Canada has been limited. While national surveys have included questions on some aspect of interpersonal discrimination, several have been designed to focus only on a specific sub-population of Canadians. In order to strengthen the evidence base, help raise awareness, enhance our ability to communicate effectively about this issue and support better research and interventions on the links between discrimination and key health and social outcomes, the Social Determinants and Science Integration Directorate of PHAC funded a Rapid Response Module (RRM) in the 2013 Canadian Community Health Survey (CCHS). The RRM comprised questions adapted from the Everyday Discrimination Scale.[2]

The CCHS RRM provides unique information that has never been collected previously on a nationally representative sample in Canada. This information will add a new perspective on self-reported interpersonal discrimination, complementing data that have been collected by other Canadian surveys.

Links to CRDCN

To access Research Data Centre data: http://www.rdc-cdr.ca/research

To access the CCHS dataset: http://www.rdc-cdr.ca/datasets/cchs-canadian-community-health-survey

Links to Statistics Canada

Everyday Discrimination RRM Questionnaire (archived page): http://www23.statcan.gc.ca/imdb/pIX.pl?Function = showStaticArchiveHTML&a = 1&fl = http://www23.statcan.gc.ca/imdb-bmdi/instrument/3226_Q6_V1-eng.htm&Item_Id = 149959

Other Statistics Canada documentation on the Everyday Discrimination RRM (archived page): http://www23.statcan.gc.ca/imdb/pIX.pl?Function = showStaticArchiveHTML&a = 1&fl = http://www23.statcan.gc.ca/imdb-bmdi/document/3226_D80_T1-V1-eng.htm&Item_Id = 149987

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Other PHAC publications

Researchers from the Public Health Agency of Canada also contribute to work published in other journals. Look for the following articles published in 2015:

Canadian Task Force on Preventive Health Care. Recommendations on screening for cognitive impairment in older adults. CMAJ. 2015 Nov 30. [Epub ahead of print]

Foebel AD, Hirdes JP, **Lemick R, Tai JW**. Comparing the characteristics of people living with and without HIV in long-term care and home care in Ontario, Canada. AIDS Care. 2015;27(10):1343-53.